Association chiropratique canadienne | Canadian Chiropractic Association

CANADIAN CHIROPRACTIC ASSOCIATION

Chair — Dr. Gerald Olin, BSc, DC

JCCA STAFF

Editor — Kent Stuber, DC, MSc
Division of Graduate Education & Research
Canadian Memorial Chiropractic College, Calgary, Alberta

Editor Emeritus — Allan Gotlib, C.M., DC
Toronto, Ontario

Associate Editors — Jeffrey Quon, DC, PhD
School of Population & Public Health
Faculty of Medicine, University of British Columbia, Vancouver, British Columbia

André Bussières, DC, FCCS(C), PhD
Faculty of Medicine, McGill University, Montréal, Québec
Département chiropratique, Université du Québec à Trois-Rivières, Trois-Rivières, Québec

Dana J. Lawrence, DC, MMedEd, MA
Parker University, Dallas, Texas

Assistant Editors — Gregory N. Kawchuk, DC, PhD
University of Alberta, Edmonton, Alberta

Mohsen Kazemi, RN, DC, MSc, PhD, FRCCSS(C), FCCPOR(C)
Faculty of Clinical Education, Graduate Studies and Research,
Canadian Memorial Chiropractic College, Toronto, Ontario

Production Co-ordinator — John Doiron

Advertising — Editor, Journal of the Canadian Chiropractic Association
186 Spadina Avenue, Suite 6, Toronto, Ontario M5T 3B2
Tel: 416-585-7902 877-222-9303 Fax: 416-585-2970

Email: Dr. Kent Stuber<kstuber@jcca.ca>
Website: www.jcca-online.org

TYPESETTING

Thistle Printing
35 Mobile Drive, Toronto, Ontario M4A 2P6

JCCA
Journal of the Canadian Chiropractic Association

Published by the Canadian Chiropractic Association and issued quarterly

EDITORIAL AND EXECUTIVE OFFICES,
186 SPADINA AVENUE, SUITE 6, TORONTO, CANADA M5T 3B2

General Information: The Journal of the Canadian Chiropractic Association is the official publication by the Canadian Chiropractic Association. The JCCA is published by the Canadian Chiropractic Association as a medium of communication between the Association and its members and is a forum for fair comment and discussion of all matters of general interest to the chiropractic profession and the Association. Readers are invited to comment and express their opinions on relevant subjects. Views and opinions in editorials and articles are not to be taken as official expression of the Association's policy unless so stated. Publication of contributed articles does not necessarily imply endorsement in any way of the opinions expressed therein and the Journal and its publisher does not accept any responsibility for them. Business correspondence should be addressed to: the Editor of JCCA, 186 Spadina Avenue, Suite 6, Toronto, Canada M5T 3B2.

INDEXING SERVICES

JCCA is indexed by PubMed Central, Scopus, CINAHL (Cumulative Index to Nursing and Allied Health Literature), MANTIS (formerly CHIROLARS), AMED, PASCAL, Index to Chiropractic Literature, and selectively by SPORTDiscus.

Contents

JCCA Vol 63 No 3 ISSN 0008-3194 (Print) and ISSN 1715-6181 (Electronic)

Editorial Board

JCCA December 2019 Sports Chiropractic Special Issue: 11th Edition

Mohsen Kazemi, RN, DC, MSc, FRCCSS(C), FCCPOR(C), PhD[1]
Assistant Editor

(JCCA. 2019;63(3):143)

KEY WORDS: sports, chiropractic

MOTS CLÉS : sports, chiropratique

It is hard to believe that it has been 21 years since I was the first graduate from the Sports Sciences full time residency program at the Canadian Memorial Chiropractic College (CMCC). Twenty years ago, I received my first Fellowship from the Royal College of Chiropractic Sports Sciences Canada, RCCSS(C). Reflecting on the program at the time, what I learned, and the opportunities it provided me, I would do it again without hesitation. The program has evolved since, courses and placements have been added. Original research papers and systematic reviews and case studies produced by the chiropractic sports residents and Fellows have been improving and increasing year after year. It is wonderful to see the chiropractic sports residents becoming Fellows and leaders nationwide. Eleven years ago, to provide a conduit for our research output we started the Sports Issue of the JCCA and it has been a great success. This year is no exception. The 11th annual sports issue includes practical case reports and original research papers. I hope you enjoy the content presented in this issue.

I would like to encourage you to get involved in sports chiropractic research in any way that you can. If you have a dataset, an interesting case, or an innovative research idea and need help developing it, please do not hesitate to contact me, or any member of our editorial board or the RCCSS(C).

[1] Canadian Memorial Chiropractic College

Corresponding author: Mohsen Kazemi, 6100 Leslie Street, Toronto, ON M2H 3J1
E-mail: mkazemi@cmcc.ca
Tel: 416-482-2340

The author has no disclaimers, competing interests, or sources of support or funding to report in the preparation of this manuscript.

ISSN 0008-3194 (p)/ISSN 1715-6181 (e)/2019/144–153/$2.00/©JCCA 2019

The effect of a six-week plyometric training on dynamic balance and knee proprioception in female badminton players

Raana Alikhani, MSc[1]
Shahnaz Shahrjerdi, PhD[1]
Masod Golpaigany, PhD[1]
Mohsen Kazemi, RN, DC, FRCCSS(C), FCCPOR(C), MSc, PhD[2]

Objective: *Non-contact anterior cruciate ligament (ACL) injury is one of the most common severe injuries among female badminton players. Dynamic balance (DB) and knee proprioception (KP) are critical in preventing this injury. The purpose of this study was to investigate the effect of a six-week plyometric training (PT) program on DB and KP in female badminton players.*

Methods: *Twenty-two healthy beginner female badminton players were randomly assigned to either control (CG) or experimental group (ExG). The ExG went through PT for six weeks. Pre- and post-intervention Y balance and photography tests were used to assess DB and KP, respectively.*

Results: *There was no difference between groups prior to PT in DB (p=0.804) and KP (at 45°, p=0.085 and at 60°, p=0.472 angles; p>0.05). However, after the PT only ExG improved significantly in DB (p=0.003)*

Objectif : *La rupture sans contact au ligament croisé antérieur (LCA) est l'une des blessures graves les plus courantes chez les joueuses de badminton. Un équilibre dynamique (ED) et une proprioception du genou (PG) sont essentiels dans la prévention de cette blessure. L'objectif de cette étude était d'évaluer les effets d'un entrainement pliométrique d'une durée de six semaines sur l'ED et le PG chez les joueuses de badminton.*

Méthodologie : *Vingt-deux joueuses de badminton novices et en bonne santé ont été réparties au hasard entre le groupe de contrôle (GC) et le groupe expérimental (GE). Les joueuses du groupe expérimental ont suivi un entrainement pliométrique pendant six semaines. Des tests d'équilibre Y avant et après l'intervention et des tests photographiques ont été utilisés pour évaluer l'ED et le PG, respectivement.*

Résultats : *Aucune différence n'a été constatée entre les groupes avant un EP dans l'ED (p=0,804) et le PG (à 45°, p=0,085 et à 60°, p=0,472) Toutefois, après l'EP seul le GE a fait état d'une amélioration de manière*

[1] Department of Physical Education and Sport Sciences, Arak University
[2] Department Graduate Studies and Research, Canadian Memorial Chiropractic College

Corresponding author: Mohsen Kazemi, 6100 Leslie Street, Toronto ON, M2H 3J1
Tel: 416-482-2340
E-mail: mkazemi@cmcc.ca

The authors have no disclaimers, competing interests, or sources of support or funding to report in the preparation of this manuscript. Ethics approval was provided by the Ethics Committee of Arak University, Medical Sciences division.

and KP (at 45°, p=0.004 and at 60°, p=0.010 angles; p<0.05).

Conclusion: *Female badminton players' dynamic balance and knee proprioception improved significantly after plyometric training (PT). These results may be important in preventing non-contact anterior cruciate ligament (ACL) injury, which requires further investigation.*

(JCCA. 2019;63(3):144-153)

KEY WORDS: badminton, anterior cruciate ligament, dynamic balance, knee proprioception, plyometric training, female players

significative en ED (p=0,003) et PG (à 45°, p=0,004 et à 60°, p=0,010 angles ; p<0,05).

Conclusion : *L'équilibre dynamique et la proprioception du genou des joueuses de badminton se sont améliorés de manière significative après un entrainement pliométrique. Ces résultats peuvent être considérables dans la prévention des ruptures sans contact du ligament croisé antérieur (LCA) qui nécessitent un examen plus approfondi.*

(JCCA. 2019;63(3):144-153)

MOTS CLÉS : badminton, ligament croisé antérieur, équilibre dynamique, proprioception du genou, entraînement pliométrique, joueuses

Introduction

Participation by women in intercollegiate and international competitions has increased significantly in recent years. Female athletes involved in jumping and cutting (quick direction maneuvers) activities are at higher risk of non-contact anterior cruciate ligament (ACL) injury than their male counterparts.[1] Approximately 250,000 people injure their ACL each year in the United States alone, with the most important potential mechanism being reduced knee proprioceptive ability.[2] Mechanoreceptors (i.e. muscle spindles or the fascial system) have been found in ACL tissue.[3,4] Interestingly, the deep fascia has also been shown to have a prominent role in proprioception and peripheral motor coordination.[4] ACL rupture, the most common severe knee ligament injury, is more prevalent among young athletes (15 to 25 years of age), with 70% due to non-contact injury mechanism.[5] The rate of this injury is higher in female athletes compared to male athletes due to the intrinsic factors such as anatomical, hormonal, neuromuscular, and biomechanical differences.[6] In addition to the high cost of treatment, ACL injury can lead to the loss of athletic participation and other complications such as osteoarthritis, physical, and psychological problems.[7]

Badminton is one of the most popular racket sports in the world that attracts many recreational and competitive athletes. Badminton is a sport suitable for all people of all ages and levels. Badminton requires frequent jumps, lunges, quick changes of direction, rapid arm movements, rapid eye-hand coordination, and adequate body position sense.[8,9] Høy *et al*. reported that 5% of all sports injuries are reported in badminton.[10] Among them, ACL is likely the most common severe knee ligament injury in badminton.[11,12] Kimuraet *et al*. reported that these injuries accounted for 37% of all badminton injuries.[11] In the London Summer Olympic Games in 2012, badminton was one of the most injurious non-contact sports, with non-contact ACL injury as the most common injury reported.[13] In the Rio Summer Olympic Games in 2016, the most injured region in badminton players was the lower limb (mostly knee joint and non-contact ACL injury).[14] The most common injury mechanism in badminton was a single-limb landing after an overhead stroke (smash or clear shot), whereas sudden deceleration with change of direction (plant-and-cut) was the second most common injury mechanism.[10,15] Reeves *et al*. reported intrinsic factors to be the main causes of this injury in badminton.[15]

Badminton players require a high level of dynamic balance during quick movements around the court to perform well and prevent injury.[16] The capability to maintain the body's base of support with minimal movement when standing (static balance) and during movement (dynamic balance) is defined as balance, whereas dynamic balance involves some levels of expected movement around the center of gravity projection.[17] The regulation of balance depends on the sources of the visual, vestibular, and proprioceptive stimulus.[18] The proprioceptive system includes mechanoreceptors (i.e. muscle spindles, Golgi tendon organs, Ruffini nerve endings, Paccini corpuscles, Meissner's corpuscles, nociceptors, and Merkel's discs[19]) which are located in muscles, tendons, joints, fascia[20], and skin. Muscle spindles, as the most important source of proprioception, provide important sensory information regarding muscle tension or length of muscle fibers and the velocity of change of muscle displacement.[21] Golgi tendon organs usually have a protective function against excessive tensile loads in the muscle.[22] Recent research shows that fascia exhibits a high density of mechanoreceptors. The fascial system is rich in proprioceptors, particularly the Ruffini and the Paccini corpuscles, which play a significant role in conveying mechanical tension, in order to control an inflammatory environment.[20] Mechanoreceptors are specialized sensory neurons that respond to mechanically applied pressure or tissue deformation.[23] The mechanoreceptors provide important afferent information, regarding position (static) and movement (dynamic), to the central nervous system by transforming the mechanical energy created by physical deformation of the joint and muscles to electrical energy of nerve action potential for processing.[24,25] This system is an important component of balance.[18] The contribution of visual cues is essential during the establishment of static balance, while the contribution of proprioceptive input is necessary in dynamic balance.[26] Generally, proprioception is referred to as the ability to know where the body is in space without using the eyes, either consciously or unconsciously.[27,28] Proprioception can be divided into two components: joint movement (kinaesthesia) (the dynamic sense of movement, including joint acceleration, force, and velocity) and joint position sense (the static sense of movement).[29] Awareness of posture, movement, joint position, limb velocity, changes in balance and weight, and resistance of objects in relation to the body increases by proprioception and their associated neurological systems.[27,28] Knee joint proprioception contributes to joint stability, postural and motor control through conscious or unconscious information arising from proprioception receptors in the capsules, ligaments, and muscle spindles.[30]

Several different ACL injury prevention programs have been attempted and each is based on different design concepts and emphasize different components of a preventive program. They include plyometric, strengthening, balancing, endurance, and stability programs.[31] Hewett *et al*. reported that the effectiveness of preventive programs in different athletes can differ as it is dependent on enhancing neuromuscular control and preventing ACL injuries.[32] Plyometric training can be introduced as an effective training modality in improving joint awareness, balance, and neuromuscular properties.[33] Plyometrics are a type of exercise that uses the stretch-shortening cycle of musculotendinous tissue which uses the energy stored during the eccentric loading phase and stimulation of the muscle spindles to facilitate maximum power production during the concentric phase of movement known as reactive neuromuscular training.[31] Multiple studies demonstrate that plyometric training may have a significant effect on knee stabilization and prevention of non-contact ACL injury among female athletes.[22,34,35] Corina *et al*. introduced stretching, proprioception coordination, and plyometric exercises to prevent common musculoskeletal injuries (sprain and strain) in badminton. Power is an important factor in performance and success in badminton.[12] Plyometric training improves athletic power and develops neuromuscular adaptability.[36] It improves athletic performance and may also prevent knee injury by increasing neuromuscular adaptations and correcting faulty jumping or cutting mechanics by developing suitable landing technique, helping to increase knee-flexion angles, and decreasing hip adduction/knee valgus angles at landing.[22,37] Plyometric training may facilitate neural adaptations that enhance proprioception, kinesthesia, and muscle performance characteristics.[38] These adaptations are created by the repeated stimulation of mechanoreceptors near the end range of motion. In addition, the relationship between plyometric training and balance has been attributed to development of neuromuscular adaptability.[39] Hence, due to the prevalence of non-contact ACL injuries in female badminton players and the significance of dynamic balance and knee proprioception in preventing this injury, this study aimed to investigate the effect of six-week plyometric training on the dynamic balance and knee proprioception of female badminton players.

Methods

Participants

This semi-experimental study recruited 22 healthy beginner female badminton players, ranging from 15 to 25 years of age, through purposive and convenience sampling. Beginner players were defined as having maximum three years of experience in playing badminton at club level with irregular exercises. Inclusion criteria were participants with less than three years of irregular participation in club level badminton, with no history of lower limb, knee joint, and particularly ACL injuries. Exclusion criteria were the presence of any injuries, experimental group participants could not have taken part in plyometric training in the four consecutive weeks prior to the study, and control group participants could not engage in plyometric training during the six-week period of the study.

Participants were randomly divided into experimental (N=12) and control groups (N=10) (Table 1). The experimental group received plyometric training 20 minutes per session, three sessions per week for six weeks; whereas the control group continued with their own routine exercises. The Ethics Committee of Arak University, Medical Sciences division approved the tests and training program. The subjects signed a consent form prior to participation. To minimize learning effects, three days prior to the intervention, participants viewed an instructional video about dynamic balance and knee proprioception tests and then practiced the tests. The examiners also viewed the instructional video to standardize the test assessment. To eliminate the effect of fatigue, participants were asked to avoid intense exercises 48 hours before performing the tests. The dominant limb was used to perform tests (dynamic balance and knee proprioception) and for limb length measurement. When the six-week training period was completed, the tests were recorded one-day after the last session of training.

Table 1.
Baseline physical characteristics

Variable	Group	N	Mean + SD
Age (y)	Experimental Control	12 10	22.00± 1.30 22.00 ± 0.84
BMI (kg/m^2)	Experimental Control	12 10	22.95 ± 3.07 22.60 ± 1.98
Sport experience (y)	Experimental Control	12 10	2.50 ± 1.00 3.00 ± 0.94

Dynamic balance assessment

Pre- and post-intervention the Y balance test was conducted in both groups to assess dynamic balance. This test has been shown to be a reliable measure and has validity as a dynamic test to predict risk of lower extremity injury.[17,40] The Y balance test device consists of a stance platform from which three pieces of polyvinylchloride pipe project in the anterior, posteromedial, and posterolateral reach directions. The participant stands on a center footplate to perform the test. While maintaining single-limb stance on the dominant limb, the participant reaches with the other limb in the anterior, posteromedial, and posterolateral directions in relation to the stance foot by pushing a reach indicator as far as possible. The participant pushes the reach indicator along the pipe with their dominant limb, and the reach indicator remains over the tape measure after performance of the test to allow for easy measurement. The test was completed in the order of anterior, posteromedial, and lastly posterolateral direction. Three consecutive trials were performed to push the reach indicator in each direction and a short rest break (10 to 15 seconds[41]) was allowed to reduce fatigue. Attempts were discarded and had to be repeated if the participant failed to maintain unilateral stance on the platform or failed to maintain reach foot contact with the reach indicator in the target area while the reach indicator was in motion, used the reach indicator for stance support, or failed to return the reach foot to the starting position under control. Each participant was allowed maximum of six attempts to obtain three successful trials for each direction. Maximum and average distance (to the nearest 0.5 cm) over the three trials were recorded and analyzed for the dominant limb in all directions. The participant's lower limb reach was normalized to limb length. The limb length was measured from the anterior superior iliac spine to the most distal portion of the medial malleolus. The normalized value was calculated as the reach distance, divided by the limb length, and then multiplied by 100%, which was to express reach distance as a percentage of limb length. Total reach distance was the sum of the three successful reach directions divided by three times limb length, and at the end, multiplied by 100%.

Knee proprioception assessment

Pre- and post-intervention photography was conducted for both groups to assess knee proprioception. The

Table 2.
Plyometric training protocol used in experimental group.

Phase 1	Sets	Reps	Phase 2	Sets	Reps	Phase 3	Sets	Reps
Wall jumps	1	15	Wall jumps	1	15	Wall jumps	1	15
Squat jumps	1	15	Squat jumps	1	15	Tuck jumps	1	15
180° jumps	1	30	Tuck jumps	1	15	180° jumps – speed	1	15
Bounding	1	15	180° jumps	1	15	Triple broad – vert	2	5
Front/back jumps	1	15	Front/back jumps	1	15	Hop, hop, hop-stick	2	6
Side/side jumps	1	15	Side/side jumps	1	15	Crossover hop, hop, hop-stick	2	6
Broad jumps	1	5	Broad jumps – stick	1	15	X-hops	2	6
Triple broad – vert	1	5	Triple broad – vert	1	5	Scissor jumps	2	6
Scissor jumps	1	6	Hop, hop, hop and stick	2	6	Box jumps	2	6
Hop, hop, hop and Stick	1	6	Crossover hop, hop, hop and Stick	2	6	Box drops	2	6
Box jumps	1	6	180° jumps – ball catch	1	6	Depth jumps	2	6
			Scissor jumps Box jumps Box drops	1 2 2	6 6 6	Box-depth-180° – box-depth-vertical	1	6

Phase – 1 low level of difficulty; Phase 2 – intermediate level of difficulty; Phase 3 – advanced level of difficulty (for detailed explanations of each exercise please refer to Appendix 1).

photography method consisted of a digital photograph (Nikon D3300), non-reflective markers, and AutoCAD software.[42] This test has high validity and reliability to assess knee proprioception.[43] Having been introduced to the test method, the participants were asked to wear shorts to paste the markers on the desired points. The markings were made in a sitting position, four skin markers were attached to the external aspect of the dominant limb. Participants' limb length was measured with a standard tape measure. Four red square markers (4x4 cm) were attached to dominant limb at the following locations: over the proximal 1/4 distance between the tip of greater trochanter and the lateral knee joint line, the neck of fibula, and over the proximal of lateral malleolus. Then the participant bent her knee at 90º angle, and the fourth marker was attached over the iliotibial tract adjacent to the superior border of the patella in this position. The locations of markers were based on previous studies.[44-47] The authors chose mid-knee joint range of motion as the goal angle (40–80˚of flexion) to measure knee proprioception since most of the performance of muscle spindles is in the midrange of joint motion.[48] Accordingly, in the present study, knee flexion angles of 45° and 60° were used to measure knee proprioception in the sitting position (sitting position is preferred to a prone position[49]). After skin marking, the participants were asked to sit on the seat with their eyes closed. The seat height was set so that participant's limb did not touch the ground. Initially, the examiner set each participant's knee at 45° flexion using a goniometer (MSD model) without changing the ankle position, the participant was then asked to hold the position for five seconds. In this position, the first photograph was taken of the lateral side

Table 3.
Independent samples test statistical analysis of change in dynamic balance and knee proprioception of training and control group over a 6-week.

Variable	Time	Mean + SD	t	p
Dynamic balance	Pre–test – experimental group	91.32 ± 7.56	0.252	0.804
	Pre–test – control group	90.52± 7.28		
	Post–test –experimental group	99.12 ±7.60	3.357	0.003*
	Post–test –control group	88.20 ± 7.59		
45° knee flexion	Pre–test –experimental group	2.41 ± 1.06	–1.813	0.085
	Pre–test –control group	3.55 ± 1.83		
	Post–test –experimental group	1.54 ± 1.03	–3.477	0.004*
	Post–test –control group	3.90 ± 1.92		
60° knee flexion	Pre–test–experimental group	2.83 ± 1.15	–.733	0.472
	Pre–test –control group	3.20 ± 1.18		
	Post–test–experimental group	2.00 ± 1.10	–2.826	0.010*
	post–test –control group	3.45 ± 1.30		
* = significant difference (p<0.05).				

of the knee. The participants were asked to return their knees to the resting position. After seven seconds of rest, the participants were asked to actively reconstruct the knee flexion at 45° angle at a desired speed and hold the angle. This process was repeated three times and photographed at each time. In order to eliminate the possible effects of reconstruction, as well as fatigue, the participant was asked to walk at an angle of 45° for one-minute. The whole process was repeated to measure knee proprioception at a 60° angle. The photos were then transferred to a computer. The angle of each photograph was calculated by AutoCAD software and compared with the target angle (target angle was the angle that the examiner set the participant's knee at 45° flexion using a goniometer). Finally, the composite angular difference (angle difference between target angles and reconstruction angles), at three times' repeat and regardless of the sign ± of scores, was recorded as the knee articular angle reconstruction absolute error of each participant.

Training protocol

Initially an educational video of the plyometric training program was shown to the experimental group. In addition, all the exercises were demonstrated and explained by a trainer. All movements of plyometric training protocol were performed and recorded by a plyometric trainer. Athletes were verbally encouraged to increase knee-flexion angles and decrease hip adduction/knee valgus angles at landing. To prevent potential injury and gradual improvement of participants, the training was designed in three phases from beginner to advanced levels of difficulty (Table 2, Appendix 1). The training protocol was also explained to the control group so that they would not engage in the same exercises. The experimental group performed 20 minutes of plyometric training, with 10 minutes warm up and cool down, three times per week for six weeks. The control group continued with their usual badminton practice and training.

Statistical analysis

Data were analyzed using SPSS software version 22, with a 95% confidence level ($p \leq 0.05$). A test of normal distribution (Shapiro-Wilk) was conducted on all data before the analysis ($p<0.05$). All data were normally distributed. An Independent Sample T test was used to test the difference between groups (experimental and control; $p<0.05$).

Results

The dynamic balance was significantly improved in the experimental group versus the control group post intervention (p=0.003) (Table 3). The knee articular angle reconstruction absolute error was significantly improved in the experimental group compared to control after plyometric training (at 45°, p=0.004 and at 60°, p=0.010 angles) (Table 3).

Discussion

Dynamic balance

The aim of the present study was to test the effects of a six-week plyometric training intervention on the dynamic balance and knee proprioception in female badminton players. Six weeks of plyometric training produced significant positive changes in dynamic balance of female badminton players. Plyometric training has an important role in improving lower-body stability.[12] These training regimens develop suitable landing techniques and improve dynamic control of the center of mass (COM), which ultimately develops neuromuscular adaptability. Myer *et al*. showed that female high-school volleyball players decreased their medio-lateral center of pressure after seven weeks of plyometric training.[50] Majeed *et al*. reported a significant difference in the dynamic balance of male badminton players after six weeks of plyometric training.[51] Cherni *et al*. observed that eight weeks of in-season plyometric training by top-level female basketball players reduced the risk of falls and injuries by improving dynamic postural control.[52] However, Arazi and Asadi found no significant improvement in the dynamic balance of semi-professional male basketball players following eight weeks of high-intensity plyometric training.[39] These discrepancies could be due to differences in intensity of training, number of contacts, plyometric drills, methods of assessment of dynamic balance, sex, age, and years of experience of the sample population. Improving dynamic balance has been reported to enhance functional adaptations, feed-forward adjustments that activate appropriate muscles before landing and proprioceptive input, as well as reduction of lower extremity injury risk.[52]

Knee proprioception

Our findings suggest that a six-week plyometric training program improves the knee proprioception of female beginner badminton players in active flexion angles of 45° and 60°. Zamani *et al*. found that eight weeks of plyometric training enhanced the knee proprioception of male college students at 30°, 45°, and 60° angles.[53] Byoung-Do *et al*. reported considerable improvements after a period of lower extremity plyometric training on the proprioception and postural stability of collegiate soccer players with postural instability.[54] Other studies have reported improved proprioception after plyometric training.[33,35,55] Plyometric training reduces the sensitivity of Golgi tendon organs against excessive tensile loads in the muscle allowing the elastic components of muscles to undergo greater stretch.[22] Lephart *et al*. believe that decreasing the sensitivity of Golgi tendon organ results in an increase in the performance of muscle spindles and consequently improves proprioception.[56] Plyometric training, through enhanced neural recruitment of motor unit or neural firing frequency, enhances reflex potentiation, and/or changes elastic properties of the muscle and connective tissue, which in turn increases neuromuscular adaptability.[57] Cug *et al*. tested the effect of a four-week dynamic balance training program on recreationally active participants (male and female) and contrary to the present study, found no significant influences on the ankle and knee joint position sense.[58] This disagreement may be due to the differences in duration and/or type of training. On the other hand, plyometric training consists of concentric and eccentric contractions with a high level of tension and force which may cause injury. As such one may assume that the best time to train polymetrically and avoid injury would be during preseason training. However, Michaelidis *et al*. stated that in-season plyometric training was more effective than the preseason training in ACL injury prevention.[59]

Conclusion

Badminton players require significant dynamic balance and knee proprioception for satisfactory performance and prevention of musculoskeletal injuries, especially non-contact ACL injuries. The results of this study demonstrated that a six-week plyometric training program improved dynamic balance and knee proprioception in beginner female badminton players. Hence, plyometric training can be utilized by badminton coaches and players to improve dynamic balance and knee proprioception, which in turn may reduce non-contact ACL injuries. Further investigation of the effect of plyometric training on reduction of injuries in badminton at various skill levels, is highly recommended.

References

1. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer: NCAA data and review of literature. Am J Sports Med. 1995;23(6): 694-701.
2. Relph N. The measurement of knee joint position sense [PhD thesis]: University of Salford; 2015.
3. Barrack RL, Lund PJ, Skinner HB. Knee joint proprioception revisited. J Sport Rehabil. 1994;3(1): 18-42.
4. Stecco C, Macchi V, Porzionato A, Duparc F, De Caro R. The fascia: the forgotten structure. Ital J Anat Embryol. 2011;116(3): 127-138.
5. Griffin LY, Albohm MJ, Arendt EA, Bahr R, Beynnon BD, DeMaio M, et al. Understanding and preventing noncontact anterior cruciate ligament injuries: a review of the Hunt Valley II meeting, January 2005. Am J Sports Med. 2006;34(9): 1512-1532.
6. Hewett TE, Ford KR, Hoogenboom BJ, Myer GD. Understanding and preventing ACL injuries: current biomechanical and epidemiologic considerations-update 2010. North Am J Sports Phys Ther. 2010;5(4): 234-251.
7. Mandelbaum BR, Silvers HJ, Watanabe DS, Knarr JF, Thomas SD, Griffin LY, et al. Effectiveness of a neuromuscular and proprioceptive training program in preventing anterior cruciate ligament injuries in female athletes. Am J Sports Med. 2005;33(7):1003-1010.
8. Shariff A H GJ, Ramlan A A. Musculoskeletal injuries among Malaysian badminton players. Singapore Med J. 2009;50(11):1095-1097.
9. Wang J, Moffit J. Teaching badminton based on student skill levels. Strategies. 2009;22(6):14-18.
10. Høy K, Lindblad B, Terkelsen C, Helleland H. Badminton injuries--a prospective epidemiological and socioeconomic study. Br J Sports Med. 1994;28(4): 276-279.
11. Kimura Y, Ishibashi Y, Tsuda E, Yamamoto Y, Tsukada H, Toh S. Mechanisms for anterior cruciate ligament injuries in badminton. Br J Sports Med. 2010;44(15): 1124-1127.
12. Corina P, Mihaela O. The prevalence of musculoskeletal lesions in badminton players–a study regarding the primary and secondary prevention strategies. Gymnasium. 2017;18(1):124.
13. Engebretsen L, Soligard T, Steffen K, Alonso JM, Aubry M, Budgett R, et al. Sports injuries and illnesses during the London Summer Olympic Games 2012. Br J Sports Med. 2013;47(7):407-414.
14. Soligard T, Steffen K, Palmer D, Alonso JM, Bahr R, Lopes AD, et al. Sports injury and illness incidence in the Rio de Janeiro 2016 Olympic Summer Games: a prospective study of 11274 athletes from 207 countries. Br J Sports Med. 2017;51(17):1265-1271.
15. Reeves J, Hume PA, Gianotti S, Wilson B, Ikeda E. A retrospective review from 2006 to 2011 of lower extremity injuries in badminton in New Zealand. Sports. 2015;3(2): 77-86.
16. Hassan IHI. The effect of core stability training on dynamic balance and smash stroke performance in badminton players. Intl J Sports Sci Phys Educ. 2017;2(3): 44.
17. Gribble PA, Hertel J, Plisky P. Using the star excursion balance test to assess dynamic postural-control deficits and outcomes in lower extremity injury: a literature and systematic review. J Athl Train. 2012;47(3): 339-357.
18. Subasi SS, Gelecek N, Aksakoglu G. Effects of different warm-up periods on knee proprioception and balance in healthy young individuals. J Sport Rehabil. 2008;17(2):186-205.
19. Richards J, Selfe J. Clinical principles of kinesiology. Mercer's Textbook of Orthopaedics and Trauma Tenth edition: CRC Press; 2012: 224-240.
20. Bordoni B, Zanier E. Clinical and symptomatological reflections: the fascial system. J Multidiscip Healthcare. 2014;7:401-411.
21. Röijezon U, Clark NC, Treleaven J. Proprioception in musculoskeletal rehabilitation. Part 1: Basic science and principles of assessment and clinical interventions. Man Ther. 2015;20(3): 368-377.
22. Chimera NJ, Swanik KA, Swanik CB, Straub SJ. Effects of plyometric training on muscle-activation strategies and performance in female athletes. J Athl Train. 2004;39(1): 24-31.
23. Coast JR. Handbook of Physiology. Section 12. Exercise: regulation and integration of multiple systems. Med Sci Sports Exerc. 1997;29(3): 424.
24. Lephart SM. Proprioception and neuromuscular control in joint stability. Human Kinetics. 2000:405-413.
25. Stillman BC. Making sense of proprioception: the meaning of proprioception, kinaesthesia and related terms. Physiother. 2002;88(11): 667-676.
26. Paillard T. Vieillissement et condition physique: Ellipses; 2009.
27. Andrews JR, Harrelson GL, Wilk KE. Physical Rehabilitation of the Injured Athlete: Expert Consult-Online and Print: Elsevier Health Sciences; 2012.
28. Nagai T, Schilaty ND, Strauss JD, Crowley EM, Hewett TE. Analysis of lower extremity proprioception for anterior cruciate ligament injury prevention: current opinion. Sports Med. 2018;48(6):1303-1309.
29. Ogard WK. Proprioception in sports medicine and athletic conditioning. Strength Condition J. 2011;33(3):1111-1118.
30. Daneshjoo A, Mokhtar AH, Rahnama N, Yusof A. The effects of comprehensive warm-up programs on proprioception, static and dynamic balance on male soccer players. PloS One. 2012;7(12):e51568.
31. Asadi A, de Villarreal ES, Arazi H. The effects of

plyometric type neuromuscular training on postural control performance of male team basketball players. J Strength Condition Res. 2015;29(7):1870-1875.
32. Hewett TE, Myer GD, Ford KR. Anterior cruciate ligament injuries in female athletes: part 1, mechanisms and risk factors. Am J Sports Med. 2006;34(2): 299-311.
33. Arazi H, Asadi A. The effect of aquatic and land plyometric training on strength, sprint, and balance in young basketball players. J Human Sport Exerc. 2011;6(1).
34. Hewett TE, Stroupe AL, Nance TA, Noyes FR. Plyometric training in female athletes: decreased impact forces and increased hamstring torques. Am J Sports Med. 1996;24(6): 765-773.
35. Sadoghi P, von Keudell A, Vavken P. Effectiveness of anterior cruciate ligament injury prevention training programs. J Bone Joint Surg. 2012;94(9): 769-776.
36. Brown TN, Palmieri-Smith RM, McLean SG. Comparative adaptations of lower limb biomechanics during unilateral and bilateral landings after different neuromuscular-based ACL injury prevention protocols. J Strength Condition Res. 2014;28(10):2859-2871.
37. Willadsen EM, Zahn AB, Durall CJ. What is the most effective training approach for preventing noncontact ACL injuries in high school aged female athletes? J Sport Rehabil. 2017;28(1): 94-98.
38. Patel NN. Plyometric training: a review article. Intl J Curr Res Rev. 2014;6(15):33.
39. Arazi H, Asadi A. Effects of high-intensity plyometric training on dynamic balance, agility, vertical jump and sprint performance in young male basketball players. J Sport Health Res. 2012;4(1): 35-44.
40. Shaffer SW, Teyhen DS, Lorenson CL, Warren RL, Koreerat CM, Straseske CA, et al. Y-balance test: a reliability study involving multiple raters. Military Med. 2013;178(11):1264-1270.
41. Cote KP, Brunet ME, II BMG, Shultz SJ. Effects of pronated and supinated foot postures on static and dynamic postural stability. J Athl Train. 2005;40(1):41-46.
42. Nasseri N, Hadian MR, Bagheri H, Olyaei STG. Reliability and accuracy of joint position sense measurement in the laboratory and clinic; utilising a new system. Acta Medica Iranica. 2007;45(5):395-404.
43. Naylor JM, Ko V, Adie S, Gaskin C, Walker R, Harris IA, et al. Validity and reliability of using photography for measuring knee range of motion: a methodological study. BMC Musculoskel Dis. 2011;12(1):77.
44. Lafortune M, Lambert C, Lake M. Skin marker displacement at the knee joint. J Biomech. 1993;26(3):299.
45. Cappozzo A, Catani F, Della Croce U, Leardini A. Position and orientation in space of bones during movement: anatomical frame definition and determination. Clin Biomech. 1995;10(4):171-178.
46. Tully E, Stillman B, editors. A revised model for 2D kinematic analysis of supine hip and knee motion in the sagital plane. Proceedings of the 12th International Congress of the World Confederation for Physical Therapy; Washington; 1995.
47. Lamoreux L. Coping with soft tissue movement in human motion analysis. Human motion analysis: Current applications and future directions New York: Institute of Electrical and Electronic Engineers. 1996: 43-70.
48. Larsen R, Lund H, Christensen R, Røgind H, Danneskiold-Samsøe B, Bliddal H. Effect of static stretching of quadriceps and hamstring muscles on knee joint position sense. Br J Sports Med. 2005;39(1):43-46.
49. Bouët V, Gahéry Y. Muscular exercise improves knee position sense in humans. Neurosci Letters. 2000;289(2):143-146.
50. Myer GD, Ford KR, Brent JL, Hewett TE. The effects of plyometric vs. dynamic stabilization and balance training on power, balance, and landing force in female athletes. J Strength Condition Res. 2006;20(2):345-353.
51. Majeed A, Nizar K, Latheef A, Nishad M. Effects of plyometric training on agility and dynamic postural control in badminton players. Intl J Sports Sci Fitness. 2016;6(2).
52. Cherni Y, Jelid MC, Mehrez H, Shephard RJ, Paillard TP, Chelly MS, et al. Eight weeks of plyometric training improves ability to change direction and dynamic postural control in female basketball players. Frontiers Physiol. 2019;10:726.
53. Zamani J, Rahnama N, Khayambashi K, Lenjannezhad S. The effects of 8 weeks plyometric training on knee proprioception. Br J Sports Med. 2010;44(Suppl 1):i12-i13.
54. Seo B-D, Shin H-S, Yoon J-D, Han D-W. The effect of lower extremity plyometric training on the proprioception and postural stability of collegiate soccer players with postural instability. Korea J Sport Biomech. 2010;20:1-23.
55. Potteiger JA, Lockwood RH, Haub MD, Dolezal BA, Almuzaini KS, Schroeder JM, et al. Muscle power and fiber characteristics following 8 weeks of plyometric training. J Strength Condition Res. 1999;13(3):275-279.
56. Lephart SM, Pincivero DM, Giraido JL, Fu FH. The role of proprioception in the management and rehabilitation of athletic injuries. Am J Sports Med. 1997;25(1):130-137.
57. Vissing K, Brink M, Lønbro S, Sørensen H, Overgaard K, Danborg K, et al. Muscle adaptations to plyometric vs. resistance training in untrained young men. J Strength Condition Res. 2008;22(6):1799-1810.
58. Cuğ M, Wikstrom EA. 4-weeks Dynamic Balance training fails to improve ankle and knee joint position sense. Cumhuriyet Med J. 2018;40(3).
59. Michaelidis M, Koumantakis GA. Effects of knee injury primary prevention programs on anterior cruciate ligament injury rates in female athletes in different sports: a systematic review. Phys Ther Sport. 2014;15(3):200-210.

Appendix 1.
Glossary of exercises training

1. Wall jumps: With knees slightly bent and arms raised overhead, jump up and down off toes.
2. Squat jumps: Standing jump raising both arms overhead, land in squatting position touching both hands to floor then takes off into a maximum vertical jump.
3. 180° jumps: Two-footed jump. Rotate 180° in mid-air. Hold landing for 2 seconds and then repeat in reverse direction.
4. Bounding: Start bounding in place and slowly increase distance with each step, keeping knees high.
5. Front/back jumps: Two-footed jump on mattress, tramp or other easily compressed device. Perform front–to back.
6. Side/side jumps: Two-footed jump on mattress, tramp or other easily compressed device. Perform side-to-side
7. Broad jumps: Two-footed jump as far as possible. Jumping horizontally and vertically to achieve maximum horizontal distance.
8. Triple broad-vert: Three broad jumps with vertical jump immediately after landing the third broad jump.
9. Scissor jumps: Start in stride position with one foot well in front of other.
10. Hop, hop, hop and Stick (Hold): Single-legged hop (Three successive jumping). Stick second landing for 5 seconds. Increase distance of hop as technique improves.
11. Broad jumps-stick (Hold): Two-footed jump as far as possible. Hold landing for 5 seconds.
12. Crossover hop, hop, hop and Stick (Hold): Start on a single leg and jumps at a diagonal across the body, lands on the opposite leg with the foot pointing straight ahead, and immediately redirects the jump in the opposite diagonal direction.
13. Tuck jump: From standing position jump, and bring both knees up to chest as high as possible. Repeat quickly.
14. 180° jumps-ball catch: Two-footed jump. Rotate 180° in mid-air and catch of ball which thrown towards her.
15. Box drops: Landing portion of a depth jump. Step from a box and stick the landing.
16. 180° jumps-speed: Two-footed jump. Rotate quick 180° in mid-air.
17. X-hops: Begins faces a quadrant pattern stands, on a single leg. Hops diagonally, lands in the opposite quadrant, maintains forward stance and holds the deep knee flexion landing for 3 seconds and then hops laterally into the side quadrant and again holds the landing. Next hops diagonally backward and holds the jump. Finally, hops laterally into the initial quadrant and holds the landing.
18. Depth jumps: Stand on a box, step off, hit the ground, and immediately jump up as high as possible at ground contact.

ISSN 0008-3194 (p)/ISSN 1715-6181 (e)/2019/154–161/$2.00/©JCCA 2019

Use of the Mulligan concept in the treatment of lateral ankle sprains in the active population: an exploratory prospective case series

Lucas Bianco, DAT, MS, LAT, ATC, CMP[1]
Smokey Fermin, DAT, MS, LAT, ATC[2]
Robert Oates, DAT, LAT, ATC[3]
James May, DAT, LAT, ATC[4]
Scott W Cheatham, PhD, DPT, OCS, ATC[5]
Alan Nasypany, EdD, LAT, ATC[4]

Introduction: *Patients classified with ankle sprains are commonly treated with a multimode intervention approach. Currently, protection and tissue healing are the most accepted forms of care for these patients.*

Introduction: *Les patientes classées comme présentant une entorse à la cheville sont généralement traitées avec une approche d'intervention multimodale. À l'heure actuelle, la protection et la guérison des tissus représentent les formes de soins les plus largement acceptées pour ces patientes.*

1 Longwood University
2 Whitworth University
3 Heidelberg University
4 Department of Movement Sciences, University of Idaho
5 Division of Kinesiology, California State University Dominguez Hills

Corresponding author: Lucas Bianco, 201 High Street, Tabb Building, Farmville, VA 23909
Tel: (434) 665-7996
Email: lucas.bianco@biokinetix.com

List of Abbreviations
FMWM – Fibula Mobilization-with-Movement
MFMWM – Modified Fibula Mobilization-with-Movement
LAS – Lateral Ankle Sprain
NRS – Numeric Rating of Pain Scale
PSFS – Patient-Specific Function Scale
FAAM – Foot and Ankle Ability Measure
GRoC – Global Rating of Change
DPAS – Disablement of Physically Active Scale
WBLT – Weight-Bearing Lunge Test
YBT – Y-Balance Test

The authors have no disclaimers, competing interests, or sources of support or funding to report in the preparation of this manuscript.
The involved patients provided consent for case publication.

Case presentation: *Six patients (4 male, 2 female) 20.2 ± 1.3 years of age were classified with acute grade I lateral ankle sprains (LAS). Each patient was treated with either the Fibular Mobilization with Movement (FMWM) or Modified Fibular Mobilization with Movement (MFMWM).*

Management and outcome: *The clinical outcomes for the patients treated with both fibula MWM improved and patients returned to activity levels at about three days after three treatments.*

Discussion: *As medicine continues to advance and explore new theories for rehabilitative clinical practice it is necessary to assess interventions on patients. This prospective exploratory case series was written to share a clinical intervention, Mulligan Concept, and the outcomes that occurred in the patients with a lateral ankle sprain.*

(JCCA. 2019;63(3):154-161)

KEY WORDS: athletic training, manual therapy, mobilizations with movement, Mulligan, sports medicine

Exposé de cas : *Six patients (4 hommes et 2 femmes) âgés de 20,2 ± 1,3 ans présentaient une entorse latérale de la cheville aiguë de grade I. Chaque patient a été traité soit avec la mobilisation fibulaire avec mouvement (FMWM) ou la mobilisation fibulaire modifiée avec mouvement (MFMWM).*

Gestion et résultats : *Les résultats cliniques pour les patients traités avec les deux mobilisations fibulaires avec mouvement se sont améliorés et les patients retournent à leurs niveaux d'activité environ trois jours après trois traitements.*

Discussion : *Au fur et à mesure que la médecine avance et explore de nouvelles théories pour la pratique clinique de réadaptation, il est nécessaire d'évaluer les interventions sur les patients. Cette série de cas exploratoires prospectifs a été rédigée dans le but de partager une intervention clinique, le concept Mulligan, ainsi que les résultats obtenus chez les patients présentant une entorse latérale de la cheville.*

(JCCA. 2019;63(3):154-161)

MOTS CLÉS : entraînement sportif, thérapie manuelle, mobilisations avec mouvement, Mulligan, médecine sportive

Introduction

Many therapeutic interventions are indicated when providing care to patients diagnosed with a Lateral Ankle Sprain (LAS). Current health care standards are often based on protocols for pain management, which focus on protection and tissue healing. Protection, Rest, Ice, Compression, and, Elevation, along with stretching, therapeutic modalities, therapeutic exercise, and medication, are often prescribed for LAS injuries.[1-3] However, these best practices for LAS injuries may be limited in effectiveness due to a primary model of focus on apparent tissue-healing.[3-5]

The Mulligan Concept (MC) includes a treatment technique termed "mobilization-with-movement" (MWM) to correct a hypothesized anterior positional fault of the distal fibula for patients classified with a LAS injury.[6] Kaminski *et al*. recommends joint mobilizations to create arthrokinematic changes to help restore function but, states that MWM need further research.[7] The MC approach can be used to address the positional fault that may be indirectly responsible for the symptoms and functional limitations reported by the patient.[1, 3-6]

Improved patient-oriented evidence and disease-oriented evidence has been highlighted in patients treated with the MC, and improvement occurred in shorter time frames than would be expected for tissue healing to occur.[8,9] However, further investigation is necessary in order to clarify and validate the potential benefits.[7] Research on outcomes of clinically applicable patient care to introduce clinicians to the MC for LAS are needed. Accordingly, the purpose of this study was to examine the effects of two different MC MWM in the non-weight bearing position on active patients classified with a grade I LAS.

Table 1.
Patient demographics, injury history, and Mulligan concept techniques applied. (FMWM: Fibula Mobilization with Movement; MFMWM: Modified Fibula Mobilization with Movement)

Patient	Age	Sex	Onset	Time to Tx	Mechanism	Treatment
1	19	Male	Acute	<24 Hours	Noncontact	FMWM
2	21	Male	Acute	<24 hours	Noncontact	FMWM
3	22	Female	Acute	<24 hours	Contact	MFMWM
4	19	Male	Acute	48-72 hours	Noncontact	FMWM
5	19	Male	Acute	<24 hours	Contact	MFMWM
6	21	Male	Acute	<24 hours	Noncontact	MFMWM

Table 2.
Ankle sprain grading system[10]

Clinical grade	Description of grade level
Grade I (Mild)	Minimal swelling (edema) and tenderness; minimal or no function loss; no mechanical joint instability
Grade II (Moderate)	Moderate pain, swelling, and tenderness over involved structures; Yes some loss of joint motion; joint stability is mild to moderately impaired
Grade III (Severe)	Complete ligament rupture with evident swelling, hemorrhage, and tenderness over involved structures, function lost; joint motion and instability evident as abnormal

Table 3.
Study inclusion and exclusion criteria.

Inclusion criteria	Exclusion criteria
18-40 years of age	Acute fracture to lower extremity
150 minutes a week of physical activity	Syndesmotic or medial ankle sprains
Evaluation and classification in ≤3 days	Lower extremity surgery over past 12 months
Grade I LAS Classification	Gross laxity in the lateral ankle ligaments

Case presentation

A total of six patients met inclusion criteria for the current study (Table 1). The convenience sample of participants, who were seeking evaluation and treatment of ankle pathology, were informed of the study guidelines and consent was obtained prior to being included in the study. A detailed history, observation, palpation, and orthopedic tests were completed during evaluation to determine the severity of their LAS injury, with an ankle sprain grading system being used to classify each patient (Table 2).[10] Further study criteria was met before patients were accepted into the study (Table 3).

After each patient met inclusion criteria and consented to participate in the study, each was instructed to complete an intake packet (i.e. NRS, PSFS, FAAM, GRoC and DPAS) and the weight bearing lunge test (WBLT) was then conducted (Figure 1).[11-16] The protocol used to assess the WBLT was based on previous research modeled by Vicenzino *et al*.[9,10,17-20] The WBLT was used due to high reliability reported in various literature examining the WBLT test.[8, 16]

Next, patients completed bilateral lower extremity Y-Balance testing (YBT). The protocol for YBT assessment in this study was based on work by Plisky et al; interrater reliability ranged from 0.99 to 1.00.[21] After the completion of the WBLT and YBT, a modified PSFS score was used to assess the patient's perception of function during each test.[21]

Prior to data collection, Institutional Review Board approval was granted at the work site of the treating clinician (TC). The Institutional Review Board accepted this a priori study design based on the details that all care provided was considered to be part of an appropriate treatment plan. The TC had completed the Mulligan Concept Lower Extremity Course and had been signed off on the MWM to the ankle joint by a Certified Mulligan Practitioner and Instructor. The TC later went on to be Certified as a Mulligan Practitioner.

Figure 1.
Weight-bearing lunge test start and stop positions.

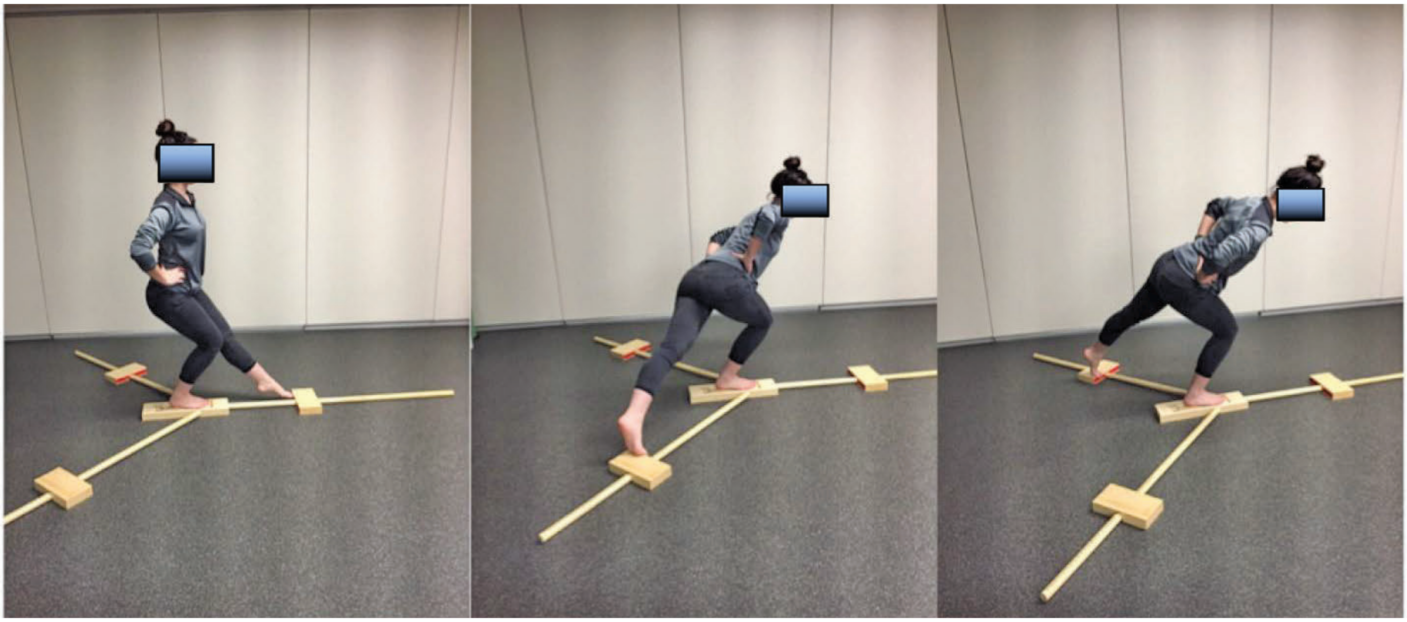

Figure 2.
Y-balance anterior, posteromedial, and posterolateral testing directions.

Figure 3.
FMWM treatment application.

Figure 4.
MFMWM treatment application.

Management and outcome

All patients were initially treated with the FMWM technique to glide the distal fibula anterior-cranially to posterior to correct the hypothesized positional fault (Figure 3). If a Pain-free Immediate Long-Lasting (P.I.L.L.) effect occurred within three attempts, the FMWM treatment was continued; however, if the P.I.L.L. effect was not achieved after three attempts, the TC attempted the MFMWM technique, with a more proximal contact to achieve the anterior-cranial to posterior glide of the fibula to correct a positional fault (Figure 4), to best meet P.I.L.L. standards.[6]

Application

While receiving treatment, patients were restricted from sports-related activities but were allowed to continue with activities of daily living. From the initial treatment session until discharge criteria was met, each patient was treated with the MC. During immediate care (first 48 hours) compression, elevation, and ice was used. At the time of the data collection, the use of Protection, Rest, Ice, Compression, and, Elevation was considered the accepted standard of care. The authors believe it is worth stating that these patients were not treated with ice as a patient preference, cryotherapy was not administered or withheld from any patients. At the time this manuscript was submitted, to the best of their knowledge, PRICE was still considered a standard method of treatment; however, there have been several papers published, related to appropriate treatment using PRICE, that information has now shown different findings regarding the use of PRICE in acute injuries. There is evidence in the literature that supports this decision.[22-24] Three of the patients (2,3, and 6) were then treated with ice, compression, and elevation for about five minutes, one time on the first day following the mechanism of injury, for pain relief. Patients were then returned to activity and discharged based on the TC evaluation and previously established norms for the collected outcome measures. Discharge criteria included, NRS score of 2 or less, and a DPA score of 23 or less.[11-13] In regards to the FAAM a score of 90 or above for daily activities and a score of 80 or above for sports activities were required prior to discharge.[15] With the YBT participants had to be within 4 cm compared bilaterally.[21] The WBLT had to be within 1.5 cm compared bilaterally.[16, 17]

Comparative outcomes

Results are reported to illustrate the initial effects of MC, effects following the complete treatment protocol, and follow-up measures in the six patients of this case series. Three patients reported a P.I.L.L. effect with the FMWM and the other three patients exhibited a P.I.L.L. effect with

Table 4.
Outcomes from initial to discharge to one month follow-ups (has met established MCID or MCD**)*

	Intake	Post-Treatment		Discharge		One-Week Follow-Up		One-Month Follow-Up	
	Mean	Mean	Mean Difference	Mean	Mean Difference	Mean	Mean Difference	Mean	Mean Difference
NRS	3.33 ± 1.03	.83 ± 1.33	2.5*	0 ± 0.0	3.33*	0 ± 0	0	0 ± 0.37	0
PSFS WBLT	6.5 ± 0.84	8 ± 1.26	1.5	9.67 ± 0.82	3.17*	9.83 ± 0.37	0.17	10 ± 0	0
PSFS YBT	6.33 ± 0.82	7.5 ± 1	1.2	9.67 ± 0.83	3.33*	9.83 ± 0.37	0.17	10 ± 0	0
WBLT	7 ± 3.1 cm	8.9 ± 3.35 cm	1.9 cm	10.42 ± 3.06 cm	3.42**	10 ± 2.78	0.42	10 ± 2.80 cm	0.42
YBC	81.78 ± 11.15 cm	85.3 ± 6.9 cm	3.5	88 ± 9.53 cm	6.25	87.7 ± 8.2	0.33	89.2 ± 6.3 cm	1.21
DPA	22.67 ± 9.7	N/A	N/A	5.33 ± 9.5 cm	17.33*	3.8 ± 5.4	1.5	1.5 ± 3.4	3.83
GRoC	1.33 ± 2.34	N/A	N/A	6 ± 0	4.67*	6.2 ± 0.4	0.17	7 ± 0	1
FAAM ADL	82.5 ± 14.27	N/A	N/A	99.5 ± 0.8	17*	98.2 ± 3.7	1.7	100 ± 0	3.5
FAAM Sport	65 ± 23	N/A	N/A	88.67 ± 14.14	23.67*	95 ± 5	0.83	99.83 ± 0.37	4

the MFMWM. Initial change in NRS met MCID standards and positive trends were reported with PSFS, WBLT, and YBT with the initial pre-treatment assessment to the initial post-treatment assessment (Table 4). Each patient returned to unrestricted activity after meeting discharge criteria within an average of 2.33 treatments completed over an average of 2.83 days (Table 5). At the follow-up dates of one-month and three-month, the patients retained the improvements that were present at discharge to unrestricted activities.

Table 5.
Patient discharge information.

Patient #	# of Treatments	Days to Discharge
1	2	3
2	2	2
3	2	2
4	2	2
5	3	5
6	3	3

Discussion

The results of this prospective exploratory study are similar to those seen in existing literature that have suggested the use of the FMWM and MFMWM to improve reported pain, function, and disability in patients classified with acute grade I LAS injuries as in this case series.[18-20] For the current study being presented, immediate improvement in clinical outcomes were present as well as at discharge, and follow-up time points. As indicated in Table 4, MCID were achieved for the NRS, the DPA scale, the GRoC, both subsections of the FAAM, and on both modified PSFS. A MDC improvement occurred for the WBLT and YBT over the course of treatment.[16, 21] For all measures, these changes were maintained for one-week and one-month follow ups, suggesting that both FMWM and MFMWM are potentially viable options to treat patients classified with grade I LAS.[21,25-29]

The MCID for the NRS is considered ≥ 1.7 points on the ten-point scale.[11] In patients with acute injury, the MCID for the DPA scale has been reported at nine points.[12] For the PSFS the MCID is considered two points of change for the average of three activities.[13] The MCID for the FAAM-ADL and FAAM-Sport has been established as eight and nine respectively.[15] Lastly, MCID has been established for the GRoC of two points.[14] Each measurement was taken pre-intervention, post-intervention, at discharge, and at a 1-month follow-up. This return to unrestricted activity occurred at a quicker timeframe than is traditionally expected with grade I LAS.

Currently, several studies highlight the benefit of immediate and meaningful changes in pain and function in patients classified with LAS.[3-5, 7-10, 17-19] Through MWM, the patients benefited from both a joint mobilization and an emphasis on pain-free early motion. This case series supports earlier studies that have utilized the early intervention and joint mobilizations with movement in the management of ankle sprains.[17-19] Within the MC, several techniques, not only the FMWM technique has been used to treat patients classified with LAS.[25-27] Overall, the findings for this study were consistent with other established literature on the FMWM and MFMWM at reducing pain and increasing function.[17-19]

The clinical outcomes for the patients treated with both fibula MWMs improved and patients return to activity levels at about three days after three treatments. Current recovery standards for acute grade I LAS injuries are 11.86 to 20 days from onset to return to play, while some patients continue to report symptoms such as pain, instability, etc. after returning to activity.[30,31] About 30% of patients with previous ankle sprain develop chronic ankle instability (CAI) and 78% of the patients with CAI develop osteoarthritis.[5] No long term outcomes were recorded but, at the one-month follow-up all patients maintained their improved outcome scores. The treatment was guided through the use patient-oriented outcomes instead of solely objective functional testing and/or tissue healing biomarkers or diagnostic tests (MRI, ultrasound, Anterior Draw Test, Inversion Stress Test, etc.). Therefore, when the immediate changes in initial pain and function lasted over time the patient continues functional progressions toward unrestricted activities and the TC continues the treatment plan. Based on the theory of the MC, it is possible that the improvements may be based on modulated neurophysiological pathways versus apparent tissue disruption.[32,33] The limitations of this study include sample size, the multimodal intervention approach, and patients being physically active individuals.

Summary

The clinical importance of this prospective exploratory study is the immediate and follow-up outcomes observed during the assessments at specific time points. This study will add to other research studies on the use of MC in clinical practice, specifically for the treatment of grade I LAS injuries.

References

1. Hertel J. Functional anatomy, pathomechanics, and pathophysiology of lateral ankle instability. J Athl Train. 2002;37(4): 364-375.
2. McGovern RP, Martin RL. Managing ankle ligament sprains and tears: current opinion. Open Acces J Sports Med. 2016;7: 33-42.
3. O'Connor SR, Bleakley CM, Tully MA, McDonough SM. Predicting functional recovery after acute ankle sprain. PloS One. 2013;8(8):e72124.
4. Kobayashi T, Gamada K. Lateral ankle sprain and chronic ankle instability: a critical review. Foot Ankle Spec. 2014;7(4): 298-326.
5. Wikstrom EA, Hubbard-Turner T, McKeon PO. Understanding and treating lateral ankle sprains and their consequences. Sports Med. 2013;43(6):385-393.
6. Mulligan BR. Mobilisations with movement (MWM'S). J Man Manip Ther. 1993;1(4): 154-156.
7. Kaminski TW, et al. National Athletic Trainers' Association Position Statement: Conservative Management and Prevention of Ankle Sprains in Athletes. J Athl Train. 2013 48(4): 528-545.
8. Denegar CR, Hertel J, Fonseca J. The effect of lateral ankle sprain on dorsiflexion range of motion, posterior talar glide, and joint laxity. J Orthopaed Sports Phys Ther. 2002;32(4): 166–173.
9. Vicenzino B, Branjerdporn M, Teys P, Jordan K. Initial changes in posterior talar glide and dorsiflexion of the ankle after mobilization with movement in individuals with recurrent ankle sprain. J Orthopaed Sports Phys Ther. 2006;36(7): 464–471.
10. Beynnon BD, Renström PA, Haugh L, Uh BS, Barker H. A prospective, randomized clinical investigation of the treatment of first-time ankle sprains. Am J Sports Med. 2006;34(9): 1401-1412.
11. Salaffi F, Stancati A, Silvestri CA, Ciapetti A, Grassi W. Minimal clinically important changes in chronic musculoskeletal pain intensity measured on a numerical rating scale. Euro J Pain. 2004; 8(4): 283-291.
12. Vela LI, Denegar CR. The disablement in the physically active scale, part II: the psychometric properties of an outcomes scale for musculoskeletal injuries. J Athl Train. 2010;45(6):630-641.
13. Abbott JH, Schmitt J. Minimum important differences for the patient-specific functional scale, 4 region-specific outcome measures, and the numeric pain rating scale. J Orthopaed Sports Phys Ther. 2014;44(8): 560-564.
14. Kamper SJ, Maher CG, Mackay G. Global rating of change scales: a review of strengths and weaknesses and considerations for design. J Man Manip Ther. 2009;17(3): 163-170.

15. Martin RL, Irrgang JJ, Burdett RG, Conti SF, Swearingen JM. Evidence of validity for the Foot and Ankle Ability Measure (FAAM). Foot Ankle Intl. 2005;26(11): 968-983.
16. Bennell K, Talbot R, Wajswelner H, Techovanich W, Kelly D, Hall AJ. Intra-rater and inter-rater reliability of a weight-bearing lunge measure of ankle dorsiflexion. Austral J Physiother. 1998; 44(3):175-180.
17. O'Brien T, Vicenzino B. A study of the effects of Mulligan's mobilization with movement treatment of lateral ankle pain using a case study design. Man Ther. 1998;3(2): 78-84.
18. Mau H, Baker RT. A modified mobilization-with-movement to treat a lateral ankle sprain. Int J Sport Phys Ther. 2014;9(4): 540-548.
19. Hudson R, Baker RT, May J, Reordan D, Nasypany A. Novel treatment of lateral ankle sprains using the Mulligan concept: an exploratory case series analysis. J Man Manipulative Ther. 2017; 25(5): 251-259.
20. May JM, Nasypan A, Paolino J, Baker R, Seegmiller J. Patient outcomes utilizing the Mulligan concept of MWM to treat intercollegiate patients diagnosed with lateral ankle sprain: an a priori case series. J Sport Rehabil. 2016; 26(6): 486-496.
21. Plisky PJ, Gorman PP, Butler RJ, Kiesel KB, Underwood FB, Elkins, B. The reliability of an instrumented device for measuring components of the star excursion balance test. North Am J Sports Phys Ther. 2009; 4(2): 92–99.
22. Hubbard TJ, Denegar CR. Does cryotherapy improve outcomes with soft tissue injury? J Athl Train. 2004;39(3): 278-279.
23. Nemet D, Meckel Y, Bar-Sela S, Zaldivar F, Cooper DM, Eliakim A. Effect of local cold-pack application on systemic anabolic and inflammatory response to sprint-interval training: a prospective comparative trial. Eur J Appl Physiol. 2009;107(4): 411-417.
24. Prado MP, Mendes AA, Amodio DT, Camanho GL, Smyth NA, Fernandes TD. A comparative, prospective, and randomized study of two conservative treatment protocols for first-episode lateral ankle ligament injuries. Foot Ankle Intl. 2014;35(3): 201-206.
25. Vicenzino B, Hing W, Rivett D, Hall T. Mobilisation With Movement: The Art and Science. Edinburgh, Australia: Elsevier Australia; 2011.
26. Baker RT, Nasypany A, Seegmiller JG. The mulligan concept: mobilizations with movement. Intl J Ath Ther Tr. 2013;(1):30-34
27. Vicenzino B, Wright A. Effects of a novel manipulative physiotherapy technique on tennis elbow: a single case study. Man Ther. 1995;1(1): 30-35.
28. Slatyer MA, Hensley MJ, Lopert R. A randomized controlled trial of piroxicam in the management of acute ankle sprain in Australian Regular Army recruits: the Kapooka Ankle Sprain Study. Am J Sports Med. 1997;25(4): 544–553.
29. Vicenzino B, Branjerdporn M, Teys P, Jordan K. Initial changes in posterior talar glide and dorsiflexion of the ankle after mobilization with movement in individuals with recurrent ankle sprain. J Orthopaed Sports Phys Ther. 2006;36(7): 464-471.
30. Cross KM, Worrell TW, Leslie JE, Van Veld Khalid R. The relationship between self-reported and clinical measures and the number of days to return to sport following acute lateral ankle sprains. J Orthopaed Sports Phys Ther. 2002;32(1): 16-23.
31. Aiken AB, Pelland L, Brison R, Pickett W, Brouwer B. Short-term natural recovery of ankle sprains following discharge from emergency departments. J Orthopaed Sports Phys Ther. 2008;38(9): 566-571.
32. Sterling M, Jull G, Wright A. Cervical mobilisation: concurrent effects on pain, sympathetic nervous system activity and motor activity. Man Ther. 2001;6(2): 72-81.
33. Zusman M. The absolute visual analogue scale (AVAS) as a measure of pain intensity. Austral J Physiother. 1986;32(4): 244-246.

ISSN 0008-3194 (p)/ISSN 1715-6181 (e)/2019/162–170/$2.00/©JCCA 2019

Isolated musculocutaneous neuropathy: a case report

Jaclyn A. Kissel, BSc, DC, FRCCSS(C)[1]
Cristina Leonardelli, BScKin, DC, MHK[2]

Objective: *To present the diagnostic and clinical features of musculocutaneous neuropathy, propose possible conservative management strategies, and create awareness of this rare condition.*

Case presentation *We present the case of a 24-year old competitive soccer athlete, who sought care for an unrelated lower extremity complaint. Upon examination, significant wasting of the right biceps was noted. The patient reported right arm pain and weakness that began six months prior, following a long sleep with his arm beneath him. Neurological examination revealed an absent deep tendon reflex of C5 on the right, diminished sensation on the right anterolateral forearm, and significant weakness in muscle testing of the biceps brachii on the right. The patient was referred to a neurologist to confirm suspicion of a musculocutaneous nerve injury. Electromyography and magnetic resonance imaging confirmed the diagnosis of musculocutaneous neuropathy and ruled out other differential diagnoses. The patient is currently awaiting confirmation to*

Objectif : *Présenter les caractéristiques diagnostiques et cliniques de la neuropathie musculo-cutanée, proposer des stratégies de prise en charge conservatrices possibles et sensibiliser la population à cette maladie rare.*

Présentation de cas : *Nous présentons le cas d'un athlète de 24 ans pratiquant le soccer en compétition qui a reçu des soins pour une douleur d'un membre inférieur sans lien avec sa pratique sportive. L'examen a révélé une perte importante du biceps droit. Le patient a rapporté des douleurs et des faiblesses localisées au bras droit qui se sont manifestées six mois auparavant, après un long sommeil avec son bras positionné sous son corps. L'examen neurologique a révélé l'absence d'un réflexe tendineux profond de niveau C5 du tendon droit, une diminution de la sensation sur l'avant-bras antérolatéral droit et une faiblesse significative dans le test musculaire. Le patient a été envoyé chez un neurologue pour confirmer les soupçons d'une lésion du nerf musculo-cutané. L'électromyographie et l'imagerie par résonance magnétique ont confirmé le diagnostic de neuropathie musculo-cutanée et exclu d'autres diagnostics différentiels. Le patient attend actuellement*

1 Canadian Memorial Chiropractic College
2 Graduate Student, Sport Sciences RCCSS (C) SSRP program

Corresponding author: Jaclyn Kissel, 6100 Leslie St., Toronto, Ontario, M2H 3J1
Phone: (647) 300- 0121
E-mail: jkissel@cmcc.ca

The authors have no disclaimers, competing interests, or sources of support or funding to report in the preparation of this manuscript.
The involved patient provided consent for case publication.

determine if he is a surgical candidate for a nerve transfer.

Summary: *Musculocutaneous neuropathy is a rare condition. Recognition of the clinical presentation of this condition is important for early diagnosis and prompt intervention.*

(JCCA. 2019;63(3):162-170)

KEY WORDS: chiropractic, atrophy, biceps, musculocutaneous nerve, neuropathy, weakness

une confirmation pour déterminer s'il est un candidat chirurgical à la réalisation d'un transfert de nerfs.

Résumé : *La neuropathie musculo-cutané est une maladie rare. La reconnaissance de la présentation clinique de cette condition est importante pour un diagnostic précoce et une intervention rapide.*

(JCCA. 2019;63(3):162-170)

MOTS CLÉS : chiropratique, atrophie, biceps, nerf musculo-cutané, neuropathie, faiblesse

Introduction

Isolated injury to the musculocutaneous nerve is a rare occurrence. Associated signs and symptoms of an isolated musculocutaneous neuropathy may include weakness in elbow flexion or shoulder flexion, atrophy of the biceps brachii, and pain or paresthesia at the lateral forearm. Previous case studies have reported this condition following strenuous activity, trauma, in athletes who engaged in repetitive or contact sports, caused by anatomical anomalies, and following surgery to an unrelated body part.[1–8] We report a case of musculocutaneous neuropathy following prolonged position of the arm beneath the body during sleep. It is important to understand the clinical presentation and diagnostic criteria to allow early diagnosis and intervention in this condition.

Case presentation

A healthy 24-year-old university student and competitive soccer player presented to the chiropractic clinic to undertake rehabilitation after dislocating his ankle which occurred six months prior while playing soccer. During this visit, the patient also complained of right shoulder and arm pain which began shortly after the ankle injury. At the time of the dislocation, he presented to a local emergency room department where the ankle was reduced. He was sent home with narcotic analgesics (acetaminophen with codeine). No other follow-up was suggested or arranged. He reported that he fell asleep on the couch lying on his right side with his right arm underneath his body. The following morning, he woke up after a 10-hour sleep with intense burning, tingling, numbness, and pain in the entire upper limb. He was unable to move his arm due to pain. He also reported significant neck pain and stiffness. He had no significant history of previous neck or upper limb injuries, and denied any prior neurological conditions. In the weeks following his injury, some of the symptoms improved – tingling and burning subsided, the intensity of the pain diminished, and numbness localized to the lateral aspect of the forearm primarily. He recovered some movement in his right upper limb at the elbow, but complained of weakness especially with lifting and push-ups. The patient had not noticed any muscle atrophy.

On observation, he was a tall lean male with visible atrophy of the right biceps, which was noticed on the first visit. There was no other muscle wasting noted elsewhere on the arm or forearm. The patient had full range of motion at the elbow. The deep tendon reflex (DTR) at the elbow over the biceps tendon was absent on the right, while all remaining upper limb DTR's were found to be 2+. During sensory examination, he was unable to distinguish sharp and dull sensation located to the right anterolateral forearm, over the territory of the right lateral antebrachial cutaneous nerve, a branch of the musculocutaneous nerve. Active and passive range of motion of the elbow was full. There was significant weakness during manual muscle testing of the biceps, which was tested with the elbow flexed to ninety degrees and with the forearm supinated. Using McGee's grading system for manual muscle testing, the patient was able to generate grade 4/5 strength in elbow flexion but it appeared to come entirely from brachioradialis contraction, as there was no fasciculation noted in the biceps muscle during testing.

Following this examination, the patient was referred to his family physician for consideration of a neurologist

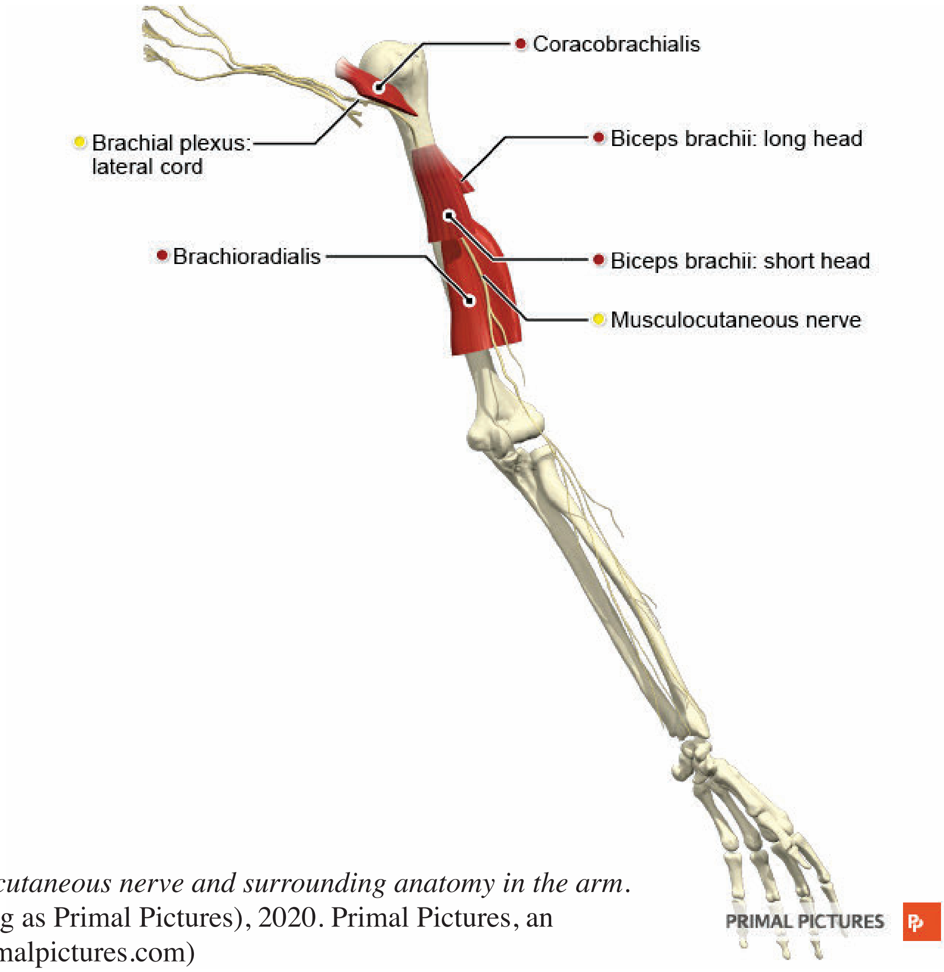

Figure 1.
Illustration of the musculocutaneous nerve and surrounding anatomy in the arm.
(© Informa UK Ltd (trading as Primal Pictures), 2020. Primal Pictures, an informa business www.primalpictures.com)

referral, to confirm our suspicion of a musculocutaneous nerve lesion as the chiropractor believed manual therapy and rehabilitation for the nerve would have been futile due to the advanced findings. The family physician referred the patient to a neurologist who confirmed our physical examination findings and ordered an electromyography (EMG) test and an MRI of the cervical spine and brachial plexus. The nerve conduction study showed an absent lateral antebrachial sensory response and during needle examination the right biceps showed no voluntary activity, but frequent fibrillation potentials. MRI of the cervical spine and brachial plexus was unremarkable. Based on these results the neurologist diagnosed this patient with a right musculocutaneous neuropathy. At the time of writing this case, the patient was waiting to determine if he was a surgical candidate for a possible nerve transfer. The time frame in which this diagnosis was reached, following the referral to the neurologist, took approximately six months. This was one year from the original insult.

Discussion

Anatomy and etiology

The musculocutaneous nerve is the terminal branch of the lateral cord of the brachial plexus. It receives fibres from the C5, C6, and C7 nerve roots. It branches off the lateral cord just distal to the coracoid process and exits the axilla by piercing the coracobrachialis (see Figure 1).[9] It descends between the biceps brachii and brachialis, supplying both muscles. At the cubital fossa, specifically at the lateral margin of the biceps aponeurosis, it pierces the cutaneous fascia and continues as the lateral cutaneous nerve of the forearm, supplying sensory innervation to the skin of the lateral forearm.[1]

In the current literature, there are few reports of isolated musculocutaneous nerve injury. The clinical presentation of musculocutaneous nerve lesions in the literature is quite variable. Most case reports documented the following physical examination findings: grade 3 or 4 weakness in elbow flexion with motor testing, atrophy of the right biceps brachii, diminished deep tendon reflexes at C5, and paresthesia and/or sensory deficit at the anterolateral aspect of the forearm.[1–3,6–8,10,11]

Physical examination

Neurodynamic testing challenges the physical capabilities of the peripheral nervous system using multi-joint movements of the limbs in order to alter the length of the nerve.[12] A "positive" test may suggest increased mechanosensitivity in neural tissues and the following have been proposed to distinguish a positive response: reproduction of the patient's symptoms with movement of a distal body segment, differences (i.e. aching, burning, tingling, etc.) between involved and uninvolved side, and alteration in resistance perceived by the examiner.[12] Positioning of the patient for nerve tension testing of the musculocutaneous nerve would include the following: shoulder girdle depression, elbow extension, shoulder extension, ulnar deviation of the wrist with thumb flexion, and either medial or lateral rotation of the arm. Palpation of the nerve (sustained or unsustained) and isometric contraction of a muscle supplied by the nerve may also identify enhanced mechanical sensitivity in neural structures.[12–14]

Manual muscle strength testing (MMST) can be a useful diagnostic tool to determine impairments in the strength of muscles that are innervated by the musculocutaneous nerve, including the biceps brachii, brachialis, and coracobrachialis. MMST is graded on a scale of 0 – 5 and descriptors of each grade are described as follows.[15] A grade of 5 is assigned when the therapist cannot break the patient's hold position when applying maximum resistance. Grade 4 designates a muscle that is able to complete a full range of motion against gravity but not able to hold the position against maximum resistance. A grade of 3 is assigned when the muscle can complete a full range of motion against gravity, but not additional resistance. Grade 2 designates muscle strength in a muscle that can complete a full range of motion in a position that minimizes gravity. A grade of 1 means some contractile activity is visible or palpable, however movement is not generated. Lastly, a grade 0 muscle is completely inert on palpation.

Tinel's test is commonly used as a provocative test in peripheral neuropathy, however there is no specific literature on this test in musculocutaneous nerve entrapments. Tinel's sign is the sensation of tingling felt at the site of the lesion or distally along the course of the nerve when it is percussed, particularly in the corresponding cutaneous distribution of the nerve.[16] In musculocutaneous neuropathy, if the nerve is percussed at the suspected lesion site (e.g. where it pierces the coracobrachialis), tingling at the anterolateral forearm would indicate a positive Tinel's sign.

Mechanism of injury

The mechanism of injury for musculocutaneous neuropathy described in the literature varies. Some describe iatrogenic causes secondary to prolonged positioning of the arm during an unrelated surgery, or direct injury to the nerve during surgery.[1,17] Repetitive, vigorous upper extremity activity (e.g. lifting, throwing, or carrying), or a single forceful extension of the upper extremity (e.g. pushing or wrestling) are more commonly cited mechanisms of injury.[2–4,6,7,10,18] Most reported injuries to the musculocutaneous nerve occur proximal to the biceps and brachialis muscles, most commonly at the coracobrachialis muscle, due to hypertrophy or strong contraction of the muscle, resulting in mechanical and/or ischemic nerve injury.[3,4,10]

Peripheral nerve injuries range in severity from neuropraxia to axonotmesis to neurotmesis.[19] Neuropraxia injuries are most common, and involve a conduction block of motor or sensory function without damage to the neural elements and with intact connective tissue sheaths. This usually occurs due to compression, mild crush, traction, or local ischemia.[20] Axonotmesis results in axonal injury and distal axon degeneration with an intact connective tissue sheath, and usually follows a more severe trauma which causes a crush injury.[19] Neurotmesis is complete disruption of the nerve and nerve sheath caused by a transection or laceration of the nerve.[19,21]

Nerve regeneration and repair processes occur differently depending on the severity of the injury. With lesions that involve less than 20-30% of the axons, collateral sprouting from surviving axons occurs over 2-6 months.[22] When more than 90% of axons are injured, nerve tissue regenerates from the injury site, and rate of recovery depends on the distance to the injury site. Proximal injuries can be problematic due to the longer distance to re-innervate distal muscles, and the regeneration and repair phases continue for months.[22] Peripheral nerve regeneration typically occurs at a rate of 1-2mm per day.[23,24]

Differential diagnoses

Differential diagnoses for musculocutaneous nerve injuries include biceps tendon injury or rupture, strain or tear of the biceps or brachialis muscles, C5 or C6 radiculopathy, and brachial plexus injury.[2,3,6,11,21] There are some specific signs and symptoms to be aware of to aid in differentiating the listed potential diagnoses. Biceps tendon injuries would present without sensory changes.[2,6] Loss of contour of the biceps muscle belly, possible bunching of the muscle belly superiorly, and absence of the tendon in the antecubital fossa would also help differentiate a biceps tendon rupture.[7] A diagnosis of C5 or C6 radiculopathy would present with pain and/or weakness in the sensory and motor distribution of the musculocutaneous nerve, but would also include weakness of other muscles supplied by the C5 or C6 nerve root, including serratus anterior, pectoralis major, and deltoid as well as neck pain.[25] In addition, radicular pain may be exacerbated by manoeuvers that irritate the involved nerve root, such as coughing, sneezing, Valsalva, and certain movements and positions of the cervical spine.[25] A brachial plexus injury would typically present with a wider distribution of sensory and motor involvement in the upper extremity, which could be confirmed with electrophysiological testing.

Diagnostic testing

While upper extremity entrapment neuropathies are typically diagnosed during the clinical assessment, a variety of diagnostic testing modalities can help to confirm clinical suspicion.[26] Electrophysiological studies such as electromyography and nerve conduction studies can usually determine the location and severity of the injury, however they are mildly invasive, operator dependent, and are unable to determine structural causes of denervation.[26,27] Ultrasonography and magnetic resonance imaging may be used to confirm the location and etiology of the nerve compression, or establish an alternative diagnosis.[26]

Motor and sensory nerve conduction studies (NCS) of the musculocutaneous nerve have been described in the literature. Standard techniques involve the measurement of nerve response amplitude and conduction velocity along the course of the nerve.[28] Needle electromyography (EMG) involves inserting a fine needle electrode into the muscle. EMG may identify the presence of denervated muscles, showing absence or reduction of motor unit recruitment during voluntary movement, and fibrillation potentials at rest.[29]

Electrodiagnostic studies (NCS and EMG) should be performed 10-21 days after a peripheral nerve injury. At this time, the extent of axonal loss can be assessed and the extent of the lesion can be distinguished.[28] Electrodiagnostic findings in musculocutaneous neuropathy may include absent lateral antebrachial cutaneous nerve and musculocutaneous motor responses and EMG abnormalities at the biceps brachii and brachialis, as well as coracobrachialis depending on the location of the lesion.[5]

Magnetic resonance imaging (MRI) can useful in demonstrating specific muscle denervation patterns, as it can identify muscle edema within 24-48 hours of denervation as well as map out denervated muscles in order to localize entrapment or compressive neuropathies.[30] With chronic denervation, fatty atrophy of the muscle is demonstrable months later.[31] In musculocutaneous nerve injuries, the nerve itself likely will not be visualized, however edema in the coracobrachialis, brachialis, and/or biceps brachii muscles would suggest involvement of the nerve.[26] With muscle edema, MRI findings include increased signal intensity on T2-weighted images superimposed on an otherwise normal appearance of the involved muscle.[32] Fatty infiltration is usually seen in association with muscle atrophy. MRI findings include increased quantities of fat with characteristic signal intensity within the involved muscle, usually with a decreased volume of muscle tissue.[32] T1-weighted images are most reliable in revealing chronic muscle denervation changes.[32] Due to the rarity of isolated musculocutaneous nerve injury, the literature on imaging findings is limited. However, we may be able to extrapolate findings from different peripheral nerve injuries.

Ultrasound is a cost-efficient, quick, non-invasive modality. It allows for dynamic evaluation and visualization of long nerve segments, however it can only depict superficially located nerves. [26] It can also be used to either demonstrate or rule out biceps tendon injury.

Magnetic resonance neurography (MRN) is a relatively new imaging modality that has been used to confirm clinical suspicion of peripheral neuropathy by clearly depicting peripheral nerve anatomy and pathology.[33] MRN can demonstrate a nerve abnormality, nerve entrapment or impingement lesions, and diffuse peripheral nerve lesions, as well as detect incidental lesions that mimic neuropathy symptoms, or exclude a neuropathy diagnosis by showing normal nerves and muscles.[33,34] MRN can reveal the morphological characteristics of nerves, such as calibre, contour, and continuity as well as relationships to other nerves or other non-neural structures such as muscle or bone.[34] The technique uses a combination of two- and three-dimensional and diffusion imaging pulse sequences in order to image nerves directly.[35] Injured nerves typically exhibit a hyperintense signal on T2-weighted images within 24 hours of the insult and the pattern is different for Sunderland ClassI-V injuries.[35,36] Class I-III injuries show a uniform hyperintense signal, Class IV injuries show a heterogenous nerve signal with focal fascicular abnormality, and Class V injuries show a discontinuous nerve.[35] Muscle denervation typically occurs distal to the site of the injury, resulting in edema and atrophy, important findings in the diagnosis of neuropathy.[35,37]

Abnormally high signals in nerve fascicles correlate well with abnormal EMG/NCS studies that demonstrated nerve injuries such as traumatic injuries, compression neuropathies, or peripheral nerve tumours.[34] These findings may significantly influence the management of peripheral neuropathies, as more severe injuries/conditions will likely be unresponsive to conservative care and should be referred to a specialist. While there is very limited literature on MRN in musculocutaneous neuropathy, likely due to the rarity of the condition, there is existing MRN research in other peripheral neuropathies that is promising.

Treatment

Similarly, there is very limited evidence for best management practices in the treatment of musculocutaneous neuropathies. Several treatment strategies, both surgical and non-surgical, have been used in cases of musculocutaneous nerve injury. Conservative management is typically used first, and surgical interventions follow when there is no improvement with conservative treatment. Non-operative management techniques include recommendations for relative rest and reduced activity, pharmacological agents (including non-steroidal anti-inflammatory drugs or anticonvulsants), local nerve inhibition methods (such as injection of corticosteroids or botulinum toxin), or physical therapy.[2,3,6,7,10] Many cases in the literature reported full recovery with conservative management as described above. None of the cases reviewed for this condition described specific physical therapy techniques that were employed in order to treat musculocutaneous neuropathy. However, we may be able to extrapolate from the literature on peripheral nerve entrapments to propose possible treatment techniques that can be performed by manual/physical therapists.

Conservative management

Effective conservative management for peripheral neuropathies may include: education, soft tissue interventions, and neurodynamic mobilization techniques.[12] Educating patients about the mechanism, presentation, and clinical behaviour of their pain can reduce the fear associated with their experience of pain, as well as influence emotional and cognitive components of pain. [12,38,39] It is also important to ensure that adjacent nonneural structures are functioning optimally in order to reduce nociceptive input.[40] This may include interventions such as mobilization and/or manipulation of local joints, soft tissue therapy, taping to unload neural structures, or neuromuscular control retraining.[12] Myofascial therapy techniques such as Active Release Technique (ART) ® may be effective in treating peripheral nerve entrapments, however much of the literature is low-level evidence comprising of case reports. ART® is described as a hands-on touch and case-management system that allows a practitioner to diagnose and treat soft-tissue injuries.[41] It is performed by taking the target tissue from a shortened position to a lengthened position while the practitioner maintains tension longitudinally along the soft tissue fibres.[41] One suggested mechanism of the ART® protocol in treating nerve entrapments is the movement of the tissues in such a way that causes relative motion between the entrapped nerve and the surrounding tissue.[41] While not specifically reported in musculocutaneous neuropathy, the utilization of ART® as a therapeutic intervention has been reported in cases of pudendal nerve entrapment, carpal tunnel syndrome, saphenous nerve entrapment, and posterior interosseous nerve entrapment.[42–46] It should be noted that more research is needed in order to fully understand the mechanism of myofascial therapy techniques in the treatment of peripheral nerve entrapments. Finally, neural tissue mobilizations have been hypothesized to have a positive impact on symptoms, however more research needs to be done in this area.[12] Neural tissue mobilization techniques involve passive or active movements that focus on restoring the nervous system's ability to tolerate compression, friction, and tension forces that are associated with daily activities.[12] Nee and Butler proposed several neural mobilization techniques that may improve patients' self-reported pain and disability, as well as physical signs of mechanosensitivity.[12] Gliding techniques attempt to produce a sliding movement between neural tissues and adjacent tissues.[40,47] Tensile loading techniques involve oscillatory movements of the limb in a fashion that puts tension on both the distal and proximal end of the nerve. The goal of these techniques is to improve the neural tissue tolerance to tension and lengthening.[40]

Operative management

If musculocutaneous nerve (or other peripheral nerve) injuries fail to respond with time and conservative treatment, then numerous surgical options have been described in the literature. These include epineurotomy, decompression, nerve graft, and nerve transfer, which will be described in this section. Surgical intervention is usually considered when there is no clinical or EMG evidence of reinnervation, typically within six months of the injury.[22] The literature lacks a specific description of a release procedure for proximal musculocutaneous nerve injuries. Yilmaz et al. described a surgical epineurotomy procedure, where the epineurium was incised and interfascicular release was performed at the hyperemic and edematous segment of the nerve. This resulted in complete symptom relief following surgery and the patient was still symptom-free at one-year follow up.[4] Surgical decompression may be advised if symptoms continue to persist 12 weeks after injury.[1,11] This intervention involves the resection of a small triangular wedge of aponeurosis that overlies the nerve. Surgical management may be a more successful option depending on the type of nerve injury, the severity of the lesion, and time of delay in repair.[48] Bhandari and Deb compared the outcomes of nerve grafts versus nerve transfers in the operative management of isolated musculocutaneous nerve injuries.[49] It should be noted that all of the subjects in their study had neurotmesis injuries, and the authors acknowledge that most neuropraxia injuries improve with conservative management. Conventional nerve grafting involves replacing the damaged portion of the nerve with a graft from a sensory nerve elsewhere in the body, typically the sural nerve.[49] Conversely, nerve transfer involves reconstructing the nerve by transferring fascicles from other nerves (such as the ulnar and median nerves) directly onto the motor branches of the musculocutaneous nerves. Bhandari and Deb reported that distal nerve transfer resulted in better functional outcomes than conventional nerve grafting.[49] In this study, electromyography revealed the first sign of biceps reinnervation at 10 weeks in the nerve transfer group, compared with 20 weeks in the grafted group.[49]

Summary

The clinical presentation of the isolated musculocutaneous neuropathy in this case was typical of many reported cases in the literature. However, the mechanism of injury we report has not yet been described. Although this condition is rare, timely diagnosis is necessary in order to initiate proper treatment as soon as possible to avoid further insult to the nerve and associated muscle atrophy. The role of healthcare providers in a rare case such as this, is to recognize the signs and symptoms as early as possible, determine if a trial of conservative care is appropriate as a first-line treatment, and/or initiate referral to a specialist for further diagnostic testing and imaging. Electrodiagnostic studies such as electromyography and nerve conduction studies may aid in confirming the diagnosis. In most cases of neuropraxia, conservative management is successful and may include neurodynamic mobilization techniques, pain education, and treatment of adjacent nonneural structures using joint manipulation/mobilization, soft tissue therapy, taping, or muscle control training. However, when conservative management is unsuccessful, surgical intervention is required. In conclusion, early diagnosis of musculocutaneous neuropathies along with an appropriate intervention will help ensure the best possible outcome for the patient.

References

1. Besleaga D, Castellano V, Lutz C, Feinberg JH. Musculocutaneous neuropathy: case report and discussion. HSS J. 2010;6(1): 112–116.
2. Pecina M, Bojanic I. Musculocutaneous nerve entrapment in the upper arm. Int Orthop. 1993;17: 232–234.
3. Papanikolaou A, Maris J, Tsampazis K. Isolated musculocutaneous nerve palsy after heavy physical activity. Inj Extra. 2005;36(11): 486–488.
4. Yilmaz C, Eskandari MM, Colak M. Traumatic musculocutaneous neuropathy: a case report. Arch Orthop Trauma Surg. 2005;125(6):414–416.
5. Merrell CA, Merrell KL. A variation of musculocutaneous neuropathy: implications for electromyographers. PM&R. 2010;2(8):780–782.
6. Henry D, Bonthius DJ. Isolated musculocutaneous neuropathy in an adolescent baseball pitcher. J Child Neurol. 2011;26(12):1567–1570.
7. Chabra T, Tahbildar P. Case report on isolated musculocutaneous nerve injury following a wrestling match. J Med Dent Sci. 2015;14(3):97–99.
8. Juel VC, Kiely JM, Leone KV, Morgan RF, Smith T, Phillips LH. Isolated musculocutaneous neuropathy caused by a proximal humeral exostosis. Neurology. 2000;54(2): 494.
9. Moore KL, Dalley AF, Agur AM. Clinically Oriented Anatomy. 7th ed. Baltimore: Lippincott Williams & Wilkins; 2013.
10. DeFranco MJ, Schickendantz MS. Isolated musculocutaneous nerve injury in a professional fast-pitch softball player: a case report. Am J Sports Med. 2008;36(9): 1821–1823.
11. Davidson JJ, Bassett FH, Nunley JA. Musculocutaneous nerve entrapment revisited. J Shoulder Elbow Surg. 1998;7(3): 250–255.
12. Nee RJ, Butler D. Management of peripheral neuropathic pain: Integrating neurobiology, neurodynamics, and clinical evidence. Phys Ther Sport. 2006;7(1): 36–49.
13. Novak CB, Mackinnon SE. Evaluation of nerve injury and nerve compression in the upper quadrant. J Hand Ther. 2005;18(2): 230–240.
14. Elvey RL. Physical evaluation of the peripheral nervous system in disorders of pain and dysfunction. J Hand Ther. 1997;10(2): 122–129.
15. Hislop H, Avers D, Brown M. Daniels and Worthingham's Muscle Testing-E-Book: Techniques of Manual Examination and Performance Testing. Elsevier Health Sciences; 2013.
16. Tinel J. "Tingling" signs with peripheral nerve injuries. J Hand Surg. 2005;30(1): 87–89.
17. Abbott KM, Nesathurai S. Musculocutaneous nerve palsy following traumatic spinal cord injury. Spinal Cord. 1998;36(8): 588.
18. Sander HW, Quinto CM, Elinzano H, Chokrouerty S. Carpet carrier's palsy: musculocutaneous neuropathy. Neurology. 1997;48(6):1731–1732.
19. Seddon H. Surgical Disorders of the Peripheral Nerves. Edinburgh, Scotland: Churchill Livingstone; 1972.
20. Dahlin LB, Wiberg M. Nerve injuries of the upper extremity and hand. EFORT Open Rev. 2017;2(5):158–170.
21. Duralde XA. Neurologic injuries in the athlete's shoulder. J Athl Train. 2000;35(3):316–328.
22. Campbell WW. Evaluation and management of peripheral nerve injury. Clin Neurophysiol. 2008;119(9):1951–1965.
23. Sunderland S. Rate of regeneration in human peripheral nerves: Analysis of the interval between injury and onset of recovery. Arch Neur Psych. 1947;58(3):251–295.
24. Grinsell D, Keating C. Peripheral nerve reconstruction after injury: a review of clinical and experimental therapies. BioMed Res Int. 2014;698256: 1–13.
25. Abbed KM, Coumans J-VCE. Cervical radiculopathy pathophysiology, presentation, and clinical evaluation. Neurosurgery. 2007;60(suppl1): S28-S34.
26. Linda DD, Harish S, Stewart BG, Finlay K, Parasu N, Rebello RP. Multimodality imaging of peripheral neuropathies of the upper limb and brachial plexus. Radiographics. 2010;30(5): 1373–1400.
27. Andreisek G, Burg D, Studer A, Weishaupt D. Upper extremity peripheral neuropathies: role and impact of MR imaging on patient management. Eur Radiol. 2008;18(9): 1953–1961.
28. Quan D, Bird SJ. Nerve conduction studies and electromyography in the evaluation of peripheral nerve injuries. Univ Pa Orthop J. 1999;12: 45–51.
29. Arancio O, Cangiano A, De Grandis D. Fibrillatory activity and other membrane changes in partially denervated muscles. Muscle Nerve. 1989;12(2):149–153.
30. Kim S-J, Hong SH, Jun WS, Choi J-Y, Myung JS, Jacobson JA, et al. MR imaging mapping of skeletal muscle denervation in entrapment and compressive neuropathies. Radiographics. 2011; 31(2): 319–332.
31. Andreisek G, Crook DW, Burg D, Marincek B, Weishaupt D. Peripheral neuropathies of the median, radial, and ulnar nerves: MR imaging features. Radiographics. 2006 ;26(5): 1267–1287.
32. May DA, Disler DG, Jones EA, Balkissoon AA, Manaster BJ. Abnormal signal intensity in skeletal muscle at MR imaging: patterns, pearls, and pitfalls. Radiographics. 2000;20(suppl1): S295–S315.
33. Chhabra A, Andreisek G, Soldatos T, Wang KC, Flammang AJ, Belzberg AJ, et al. MR neurography: past, present, and future. Am J Roentgenol. 2011;197(3): 583–591.
34. Du R, Auguste KI, Chin CT, Engstrom JW, Weinstein PR. Magnetic resonance neurography for the evaluation of peripheral nerve, brachial plexus, and nerve root disorders. J Neurosurg. 2010;112(2): 362–371.

35. Chhabra A, Madhuranthakam AJ, Andreisek G. Magnetic resonance neurography: current perspectives and literature review. Eur Radiol. 2018;28(2): 698–707.
36. Sunderland S. A classification of peripheral nerve injuries producing loss of function. Brain. 1951;74(4): 491–516.
37. Filler AG, Kliot M, Howe F., Hayes CE, Saunders DE, Goodkin R, et al. Application of magnetic resonance neurography in the evaluation of patients with peripheral nerve pathology. J Neurosurg. 1996;85: 299–309.
38. Moseley G. A pain neuromatrix approach to patients with chronic pain. Man Ther. 2003;8(3): 130–140.
39. Shacklock MO. Central pain mechanisms: a new horizon in manual therapy. Aust J Physiother. 1999;45(2): 83–92.
40. Butler D. The sensitive nervous system. Adelaide, Australia: Noigroup Publications; 2000.
41. Leahy M. Active Release Techniques® Soft Tissue Management Systems for the Upper Extremity - 2nd edition. 2008.
42. Durante JA, MacIntyre IG. Pudendal nerve entrapment in an Ironman athlete: a case report. J Can Chiropr Assoc. 2010;54(4):276-281.
43. George JW, Tepe R, Busold D, Keuss S, Prather H, Skaggs CD. The effects of active release technique on carpal tunnel patients: a pilot study. J Chiropr Med. 2006;5(4): 119–122.
44. Robb A, Sajko S. Conservative management of posterior interosseous neuropathy in an elite baseball pitcher's return to play: a case report and review of the literature. J Can Chiropr Assoc. 2009;53(4):300-310.
45. Settergren R. Conservative management of a saphenous nerve entrapment in a female ultra-marathon runner. J Bodyw Mov Ther. 2013;17(3): 291–301.
46. Saratsiotis J, Myriokefalitakis E. Diagnosis and treatment of posterior interosseous nerve syndrome using soft tissue manipulation therapy: a case study. J Bodyw Mov Ther. 2010;14(4): 397–402.
47. Shacklock MO. Clinical neurodynamics: A new system of musculoskeletal treatment. Edinburgh, Scotland: Elsevier/ Butterworth Heinemann; 2005.
48. Osborne AWH, Birch RM, Munshi P, Bonney G. The musculocutaneous nerve: results of 85 repairs. Bone Jt J. 2000;82(8): 1140–1142.
49. Bhandari PS, Deb P. Management of isolated musculocutaneous injury: comparing double fascicular nerve transfer with conventional nerve grafting. J Hand Surg. 2015;40(10): 2003–2006.

ISSN 0008-3194 (p)/ISSN 1715-6181 (e)/2019/171–177/$2.00/©JCCA 2019

Clinical presentation of an adolescent female synchronized swimmer with a simple bone cyst in the proximal humerus: a case report

Mitchell Savic, BHSc (Hons), DC, FRCCSS(C)[1]
Alexander Lee, BSc (Hons), DC, FRCCSS(C)[2]
Ashley Hook, HBSc, DC, DACBR, MSc[2]

Objective: *This case raises awareness for healthcare practitioners who may not suspect a simple bone cyst (SBC) in a pediatric athlete with a high risk of pathological fracture.*

Case summary: *SBC's are often considered as asymptomatic lesions which are commonly found incidentally on plain film radiographs. Presented here is a case of a 14-year-old competitive synchronized swimmer with a SBC in the proximal humerus with no MRI evidence of soft tissue pathology or pathological fracture, presenting clinically with refractory posterior shoulder pain.*

Summary: *Early detection of a SBC in a pediatric athlete is essential so an interdisciplinary care approach can be employed to ensure the appropriate management in those where there is high risk of pathological fracture. Clinicians should be aware of the risk factors for the*

Objectif : *Ce cas sensibilise les professionnels de la santé qui ne soupçonnent pas la présence d'un kyste osseux essentiel (KOE) chez un jeune athlète présentant un risque élevé de fracture pathologique.*

Résumé du cas : *Les KOE sont souvent considérés comme des lésions asymptomatiques que l'on trouve souvent par hasard sur les radiographies ordinaires. On présente ici le cas d'une adolescente âgée de 14 ans pratiquant la natation synchronisée, souffrant d'un KOE dans l'humérus proximal et présentant cliniquement une douleur réfractaire à l'épaule postérieure. L'IRM n'a pas mis en évidence de pathologie des tissus mous ni de fracture pathologique.*

Résumé : *La détection précoce d'un KOE chez un jeune athlète est essentielle pour qu'une approche de soins interdisciplinaires puisse être mise en œuvre afin d'assurer une prise en charge appropriée chez ceux qui présentent un risque élevé de fracture pathologique. Les cliniciens doivent être conscients des facteurs de risque*

[1] Private practice, Mississauga, ON
[2] Canadian Memorial Chiropractic College

Corresponding author: Dr. Mitchell Savic, Velocity Sports Medicine & Rehabilitation, 167 Lakeshore Road West, Mississauga, Ontario
E-mail: drmitchsavic@gmail.com
Tel: 289-338-7277

The authors have no disclaimers, competing interests, or sources of support or funding to report in the preparation of this manuscript.
The involved patient provided consent for the use of clinical information and imaging with respect to this case report.
The CMCC Research and Ethics Board have approved this case report.

development of a pathological fracture, and for other potential complications that may arise thereafter in those with a SBC.

(JCCA. 2019;63(3):171-177)

KEY WORDS: chiropractic, benign tumour, simple bone cyst, swimmer, synchronized

de fracture pathologique et des autres complications potentielles qui peuvent survenir par la suite chez les personnes atteintes d'un KOE.

(JCCA. 2019;63(3):171-177)

MOTS CLÉS : chiropratique, tumeur bénigne, kyste osseux essentiel, nageuse, synchronisée

Introduction

A simple bone cyst (SBC), also referred to as a unicameral bone cyst, is the most common benign tumour that is seen in children and adolescents (up to 85% of all cases).[1,2] These bone cysts represent approximately 3% of all tumours of the bone and have been reported to occur more frequently in boys than girls (2:1).[2] The average age of diagnosis is nine years, with a peak incidence between the ages of three and 14 years.[1,2] They are cystic, fluid filled cavities that can develop in tubular and flat bones, most typically found in the proximal humerus and femur, which account for greater than 90% of all cases.[1–4] These cysts do have a tendency to expand and weaken the bone they reside in, despite not being a true neoplasm.[1]

A definite pathogenesis of a SBC to this day remains controversial. The most widely accepted etiology is a venous outflow obstruction in the cancellous bone causing an increase in intraosseous pressure. This increased pressure results in elevation of inflammatory protein markers released by endothelial cells along the wall of the cyst, causing subsequent osteoclastic activation and bone resorption.[2–5] Other suggested mechanisms of pathogenesis include: a disturbance in local bone growth, intramedullary (post-traumatic) hemorrhage, and trapped intraosseous nests of synovial cells.[2]

Although SBC's are common and often considered asymptomatic[2,6], it has been reported that approximately two thirds of these lesions will at some point endure pathological fracture[7]. This should be of particular concern in the pediatric athletic population due to the increased load and torque forces that may be placed through the humerus.[8] This suggests that even in a non-contact scenario, the humerus may be at risk for fracture in the presence of a cyst.[8] In sports where body contact with others is more probable, this risk of fracture is only expected to increase. This raises further concern in such sports particularly if early detection has not been made.

We present an atypical clinical presentation of an adolescent competitive synchronized swimmer who experienced refractory shoulder pain despite a lack of MRI evidence of soft tissue pathology or pathological fracture of a SBC in the proximal humerus. We will also highlight possible complications and risk factors associated with SBC to raise awareness of a condition that may possibly go undiagnosed in young athletes.

Case presentation

A 14-year-old female, competitive synchronized swimmer presented with a four day history of localized, right-sided, posterior shoulder pain sustained while performing a manoeuvre referred to as sculling. Sculling is an action in synchronized swimming, whereby the swimmer treads water solely with their arms, while inverted as they are performing out of water movements with their legs. This action involves heavily resisted and repetitive internal and external rotation of the shoulders (Figure 1).

Upon presentation, the patient complained of a dull ache in the posterior shoulder at rest, which became sharp when she performed sculling. The patient did not report any previous shoulder injuries in her past. On examination, there was full, pain-free right shoulder range of motion (ROM) with no signs of swelling, erythema, or bony malalignment. Empty can, Hornblower's test and manual muscle testing of external rotation strength at zero degrees shoulder abduction were positive for pain and were rated 4/5 on the right for muscle strength. Palpation of the infraspinatus and teres minor insertions at the posterior aspect of the humeral head reproduced the patient's chief complaint. An evaluation of generalized joint hypermobility (GJH) using the Beighton Test[9] revealed a

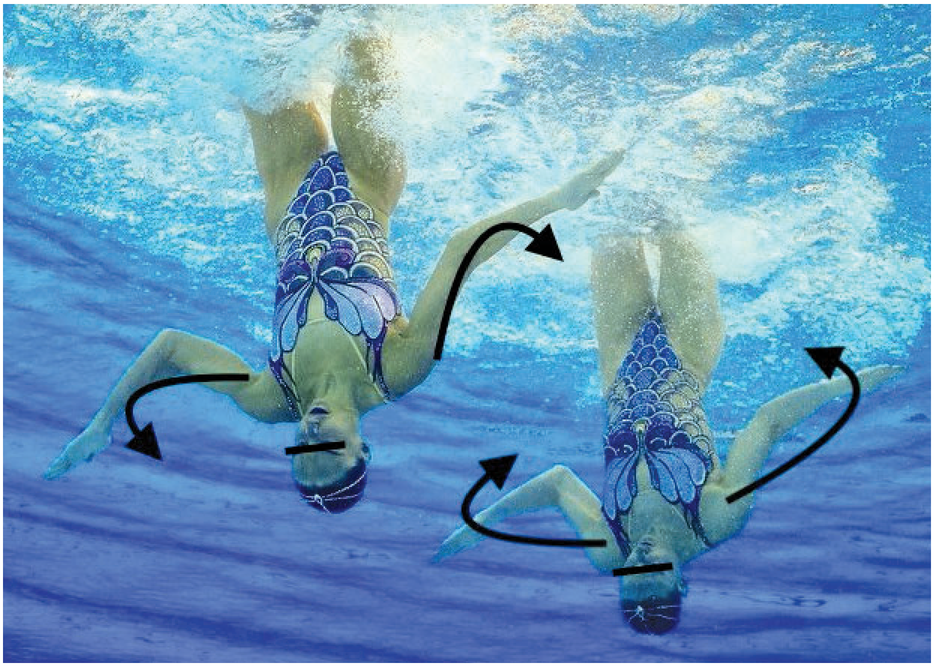

Figure 1.
Sculling is the underwater action used by synchronized swimmers to hold position and propel their bodies in different directions while inverted by repetitively and forcefully internally and externally rotating their shoulders. This is similar to treading water with your legs, known as "egg-beating".

score of 3/9 (+ bilateral hyperextension of 5th metacarpophalangeal joint >90° & + palm of hands touching flat on the ground when knees extended).

An assessment of shoulder instability using the Feagin Test, Load and Shift Test or sulcus sign was not made. Cervical spine range of motion was full and pain free, and foraminal encroachment tests of the cervical spine were negative. Sensation and motor testing of the C5-T1 levels were within normal limits bilaterally and deep tendon reflexes of C5-7 were 2+ bilaterally.

The patient was given a clinical diagnosis of an acute, right-sided rotator cuff tendinopathy and a six-week conservative plan of management consisting of Active Release Therapy (ART®) and a progressive rotator cuff and scapular stabilizer strengthening rehab protocol was implemented. The patient was treated with this approach for 9 visits over 6 weeks. Following this treatment plan, the patient showed minimal clinical change in her symptomatology and was unable to perform sculling due to pain.

A radiograph of the right shoulder was ordered (Figure 2) which revealed a well-defined, osteolytic lesion. Differential diagnoses based off the radiographic findings included: a SBC, aneurysmal bone cyst (ABC), and a chondroid lesion, such as an enchondroma. The patient was

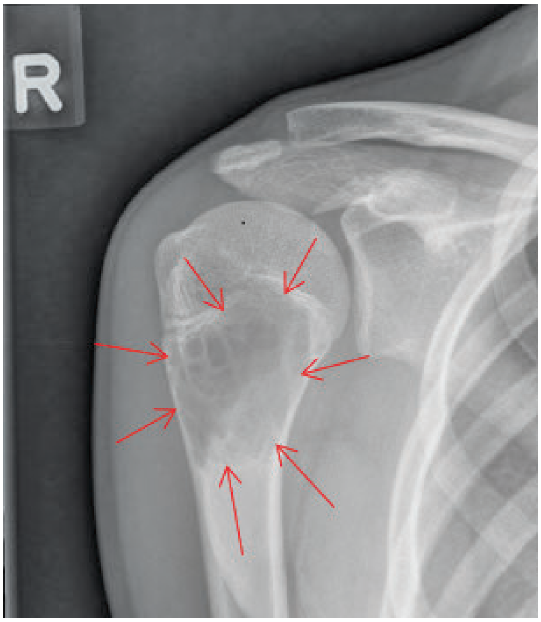

Figure 2.
AP plain film radiograph of the right shoulder revealing a well-defined, slightly expansile, osteolytic lesion (outlined by arrows), located centrally within the metaphysis abutting the epiphyseal growth plate with no signs of periosteal reaction, fracture, or soft tissue mass.

immediately referred to an orthopedic surgeon with a specialty in bone tumours of the upper extremity, who sent the patient for a magnetic resonance imaging (MRI) of the right shoulder (Figure 3) which demonstrated a slightly expansile, T2 hyper-intense and T1 hypo-intense intramedullary unilocular cystic lesion in the proximal humeral metaphysis causing endosteal scalloping and cortical thinning with no evidence of fracture. All rotator-cuff and long head of biceps tendons appeared to be intact with no sign of pathology. An orthopedic surgeon confirmed the diagnosis of a SBC based off the aforementioned MRI features which are pathognomonic for this lesion, along with the consideration of the patient's age which is also consistent with its clinical presentation. One possible inconsistency with the clinical presentation of this suspected SBC is that they are not typically painful.

At this point in time (six to eight weeks post initial presentation), the patient was still experiencing intermittent, refractory dull, achy pain along the posterior shoulder, despite her diligence with pain-free rehabilitation exercises, and avoiding all aggravating factors including synchronized swimming. The orthopedic surgeon handling the patient's case prescribed a conservative "wait and see" approach which simply involved a follow-up up MRI, activity restriction, and to continue rehab for the rotator cuff and scapular stabilizers at a recommended physiotherapy clinic.

The patient was contacted a year later, reporting that she had stopped participating in synchronized swimming. She has no pain at rest or during any of her activities of daily living. By this point, the patient decided to not follow-up with any radiographs and/or MRI, and as such, the status of the SBC is currently unknown. Any further follow-up with the patient was lost as she no longer attends the clinic where the assessment and treatments were conducted.

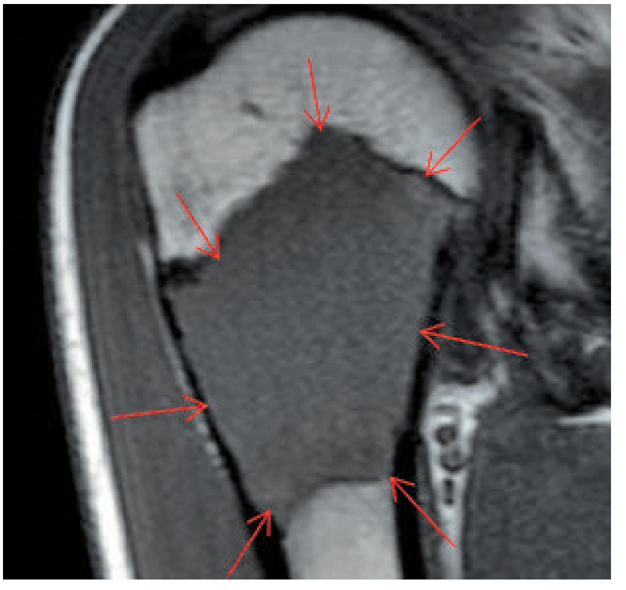

Figure 3.
T1 weighted, coronal slice MRI of the right shoulder revealing a 4.5 x 3.6 x 3.3 cm slightly expansile, hypo-intense intramedullary unilocular cystic lesion (outlined by arrows), in the proximal humeral metaphysis causing endosteal scalloping and cortical thinning with no evidence of fracture.

Discussion

The diagnosis of a SBC can normally be made with the use of plain film radiographs, however, confirmation can be made with advanced imaging (such as MRI) in cases where there is a strong resemblance to other benign bony lesions. An example of such a lesion, and a strong differential diagnosis is an ABC, which is at higher risk of gross expansion and pathological fracture.[1] Although generally not as expansile as an ABC, SBC's have the capability to expand and thin the cortex that it surrounds. This may compromise the cortex possibly resulting in a pathological fracture, which is not uncommon in the athletic population.[10] SBC's also reside centrally within the medullary cavity, unlike ABC's which have a more eccentric presentation. The more central the location, the smaller the likelihood of fracture risk and soft tissue involvement.[2] Ten percent of those that fracture will present with a fallen fragment sign on plain film radiographs, which represents a small portion of the fractured bone which can separate and sink to the bottom of the cyst.[2,11]

With that said, it is important to understand risk factors which may further increase fracture risk. A retrospective study by Urakawa *et al*.[12] identified 155 subjects with a SBC, 141 of which exceeded six-month follow-up. The majority of subjects (35%) had a SBC which resided in the proximal humerus, followed by five other locations in the lower extremities. In this study, a multivariate analysis revealed that ballooning of the bone, a cyst located in a long bone, male sex, and multilocular cysts were significant risk factors in increasing the incidence of pathological fractures.[12] Kaelin and MacEwan[13] proposed a cyst index formula (cyst area divided by the diaphyseal diameter^2) and cut-off score to help determine fracture risk. A cyst index greater than 3.38 was predictive of pathological fracture in this particular study. Similar studies have reported other risk factors associated with increased incidence of pathological fracture, which include: a high cyst index, high percentage of bone occupied by the cyst in the transverse plane, thin cortical thickness, and those active phase cysts residing in the upper limb.[13–16]

Furthermore, active phase cysts are a potential risk factor for fracture. It has generally been accepted throughout the literature that cysts residing within 1cm of the growth plate are considered active[2], minimizing the amount of normal bone between them. Haidar *et al*.[17] underwent a retrospective analysis whereby a measurement of two centimeters from the growth plate was determined as the cut-off point between active versus latent cysts. The destined progression in the natural history of the cyst is to migrate down into the diaphysis through endochondral ossification where it eventually becomes latent (non-active). These cysts are not commonly seen in adults, owing to the fact that they spontaneously fill and ossify on their own sometime after skeletal maturity is reached. Since SBC's are typically asymptomatic in the absence of pathological fracture, there is no inquiry for imaging and thus go undetected during pre-adulthood prior to ossification, making natural history difficult to document.[2,3,18]

Analysis of the present patient's SBC on MRI (Figure 2) reveals many of the risk factors mentioned above. The cyst's location is in a long bone, it is showing properties of expansion (ballooning), a high percentage of the bone is occupied by the cyst in the transverse plane, there is presence of thinning cortex, and it clearly appears to be in an active phase, as it's shown abutting the epiphyseal growth plate. Furthermore, using the equation proposed by the Kaelin and MacEwan[13], the patient had a cyst index well above the cut-off score (4.5cm x 3.6cm = 16.2/1.32cm^2 = 9.30). The only fracture risk factors that do not appear to exist in the present patient's case are the fact that the cyst is not multilocular in nature, and she is not of male sex. It was also described in the study by Urakawa *et al*.[12] that males in the study were more likely to be physically active, which may be the main contributor to the increased incidence of fracture within this cohort. The presence of multiple risk factors suggest that the present patient is of high risk of developing a pathological fracture.

As for management, Urakawa *et al*. suggested that high risk cysts should either be considered for surgery, or at the very least, careful observation.[12] Due to the fact that SBC's are self-limiting and have a tendency to heal without any intervention, it is common to adopt a more conservative plan of management, especially in children who have asymptomatic, latent cysts.[5] Educating and restricting activity in those athletes with a SBC who are at high risk of fracture should be of utmost importance in those who are following this conservative approach. In a recent survey of members of the European Pediatric Orthopedic Society (EPOS) and Pediatric Orthopedic Society of North America (POSNA)[19] surgery was highly recommended only in those children with symptomatic SBC's under 10 to 12 years of age (by 71% of members) and in those children with asymptomatic, high risk of fracture SBC's (by 53% of members). The most commonly reported surgical options used include: curettage with or without bone grafting, percutaneous decompression, corticosteroid injections, or autologous bone marrow injections. It is important to note that many of these interventions have high rates of recurrence, with improved outcomes seen with cysts measuring >2cm from the growth plate.[19]

As a clinician working with pediatric athletes, not only is it important to detect the presence of a bone cyst, or any other bony lesions, it is also important to understand the possibility or risk of other complications that may arise. In the presence of a pathological fracture, one such potential complication is the development of avascular necrosis (AVN) of the humeral head. Urakawa *et al*. reported one subject with a femoral neck SBC who developed AVN after a displaced fracture.[12] There are other reports in the literature that have described similar instances of AVN of the femoral head as a complication after pathological fracture.[12] Brooks *et al*. specify that AVN of the humeral head may result after either displaced fractures, or fracture dislocations.[20] Humeral head AVN has not been reported in SBC cases, likely due to the fact that, to the authors' knowledge, there presently is no report of displaced humeral SBC pathological fractures in the literature.

Another possible complication is growth arrest, especially in those active cysts which are likely communicating with the epiphyseal growth plate. Although extremely rare, growth arrest is a possibility, especially when a displaced femoral neck fracture is involved. Other possible mechanisms for growth arrest may include: the hypothetical instance where a pathological fracture involves the disruption of the growth plate, or through accidental damage of the growth plate through invasive surgical techniques.[5] Fortunately, there are no reported cases of malignant transformation of SBCs.[3]

It is not entirely understood as to the source of the present patient's pain. SBC's that have not fractured are typically asymptomatic, however, there is suggestion in the literature that pain could arise from fissuring of the cyst throughout its growth and migration.[1,3] It is not known if this bears any relationship to endosteal scalloping which was reported in the patient's imaging. Given that there was no evidence of other shoulder pathology in the patient's MRI, it is plausible that this fissuring mechanism may have played a factor in the patient's experience of refractory shoulder pain. It is also important to note that the patient's pain was aggravated by certain movements and can be elicited with palpation of muscular structures, which would suggest either pain of musculo-tendinous origin or local hyperalgesia as a compensatory response from the cyst. Entertaining the idea of pathology present within the surrounding musculo-tendinous structures (i.e rotator cuff) as a source of the patient's pain may still be warranted considering the biomechanical joint stresses involved with certain sport-specific movements, such as sculling. Although rotator cuff tears in the general pediatric population account to less than 1% of all total rotator cuff tears[21], those who participate in repetitive and large rotational torqueing movements of the glenohumeral joint increase the risk of rotator cuff tears through tensile shearing forces[21]. Furthermore, there is always the possibility for false-negative MRI results considering the sensitivity of detecting tendinopathy and partial tears. A meta-analysis identified the pooled sensitivity for detecting partial rotator-cuff tears and tendinopathy (0.67-0.83) is much lower than for full tears (>0.90) using either MRI or ultrasound (US).[22] Having said that, it is plausible to assume that the patient may have suffered from rotator cuff pathology despite a normal MRI. Another explanation to her musculoskeletal pain should include the possibility of GJH resulting in instability of the humeral head within the glenoid. Competitive swimmers may have this propensity to hypermobility[23] and resultant glenohumeral subluxation which may negatively influence adjacent soft tissue structures (i.e. rotator cuff) through repetitive abutment. There seems to be a positive correlation between GJH, measured using the Beighton Test[9], and an increased active horizontal shoulder abduction (AHSA) score in 10 to 15 year old competitive swimmers[23]. The patient in this case scored a 3/9 on the Beighton Test, suggesting an absence of GJH (+ score is 4/9 or >).[9] MRI of the patient's shoulder also revealed insignificant soft tissue injury findings (no presence of edema, tendinopathy or tear). Despite these results, assessing for GJH is an important aspect of the physical exam in athletes who participate in activities which require excessive and repetitive joint ROM. Perhaps one factor as to why there appears to be a strong correlation between GJH and an increased AHSA in pediatric competitive swimmers is genetic selection. Genetic variability may make it easier for some individuals to participate in sports which require certain sport-specific movements, particularly at a high level which may pose as an injury risk. Instead of focusing on soft tissue related diagnoses (i.e rotator cuff tendinopathy), it may be beneficial to shift the clinical focus to movement-related deficiencies in those who are hypermobile. From a rehabilitation perspective, a rationale to compensate for deficits in the passive structures surrounding the glenohumeral joint is to retrain the dynamic shoulder stabilizers in order to maintain glenohumeral joint centration.[24]

As a treating practitioner, early detection of SBCs is central to their management. When identified early, decisions relating to activity restriction (or elimination), and early referral to an orthopedic surgeon can be undertaken. This will help to not only decrease the likelihood of developing a pathological fracture, but will also allow for the development of a plan of management that will be more conducive to treating structures around a weakened bone. That being said, practitioners must be cognizant when treating soft tissue structures surrounding the cyst, in order to help decrease the risk of iatrogenic fracture which could plausibly be caused by manual therapy.

Summary

This case highlights the importance of early detection of a SBC in a pediatric synchronized swimmer. She presented with symptoms consistent with rotator cuff pathology, but was determined to have a SBC that was classified as a potential high risk for pathological fracture. As illustrated by this case, it is important to determine the risk factors for the development of a pathological fracture, and for other potential complications that may arise thereafter in those with a SBC.

References

1. Pretell-Mazzini J, Murphy RF. Unicameral bone cysts: general characteristics and management. J Am Acad Orthop Surg. 2014;22(5): 295–303.
2. Noordin S, Allana S, Umer M, Jamil M, Hilal K, Uddin N. Unicameral bone cysts: current concepts. 2018;34: 43–49.
3. Mascard E, Gomez-Brouchet A, Lambot K. Bone cysts: Unicameral and aneurysmal bone cyst. Orthop Traumatol Surg Res. 2015;101(1): S119–S127.
4. Baig R, Eady JL. Unicameral (simple) bone cysts. South Med J. 2006;99(9): 966–977.
5. Jg Z, Ding N, Wj H, Wang J, Shang J, Zhang P. Interventions for treating simple bone cysts in the long bones of children. Cochrane Database Syst Rev. 2014;(9).
6. Garceau G, Gregory C. Solitary unicameral bone cyst. J Bone Jt Surg. 1954;(36):267.
7. Wilner D. Radiology of Bone Tumors and Allied Disorders. Philadelphia: WB Saunders. 1982.
8. Teoh KH, Watts AC, Chee Y-H, Reid R, Porter DE. Predictive factors for recurrence of simple bone cyst of the proximal humerus. J Orthop Surg. 2010;18(2):215–219.
9. Grahame R, Bird HA CA. The revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS). J Rheumatol. 2000;7:1777–1779.
10. Rowe L, Brandt J. Simple bone cysts in athletes. Chiro Sport Med. 1988;(2):33.
11. Reynolds J. The "fallen fragment sign" in the diagnosis of unicameral bone cysts. Radiology. 1969;(92):949.
12. Urakawa H, Tsukushi S, Hosono K, Sugiura H, Yamada K, Yamada Y, et al. Clinical factors affecting pathological fracture and healing of unicameral bone cysts. BMC Musculoskelet Disord. 2014;15: 159.
13. Kaelin A, MacEwan G. Unicameral bone cysts. Natural History and the risk of fracture. Int Orthop. 1989;13(4):275–282.
14. Ahn J, Park J. Pathological fractures secondary to unicameral bone cysts. Int Orthop. 1994;18(1):20–22.
15. Zamzam M, Abak A, Bakarman K, Al-Jassir F, Khoshhal K, Zamzami M. Efficacy of aspiration and autogenous bone marrow injection in the treatment of simple bone cysts. Int Orthop. 2009;33(5):1353–1358.
16. Tey I, Mahadev A, Lim K, Lee E, Nathan S. Active unicameral bone cysts in the upper limb are at greater risk of fracture. J Orthop Surg. 2009;17(2):157–160.
17. Haidar SG, Culliford DJ, Gent ED, Clarke NMP. Distance from the growth plate and Its relation to the outcome of unicameral bone cyst treatment. J Child Orthop. 2011;5(2):151–156.
18. Jaffe H, Lichtenstein L. Solitary unicameral bone cyst, with emphasis on the roentgen picture, the pathologic appearance and the pathogenesis. Arch Surg. 1942;44:1004.
19. Farr S, Hahne J, Bae DS. Current trends and variations in the treatment of unicameral bone cysts of the humerus: a survey of EPOS and POSNA members. J Pediatr Orthop. 2019;00(00).
20. Brooks C, Revell W, Heatley F. Vascularity of the humeral head after proximal humeral fractures. J Bone Jt Surg. 1993;75:132–136.
21. Perez JR, Massel D, Barrera CM, Baraga MG, Pretell-mazzini J, Kaplan LD, et al. Rotator cuff tears in the pediatric population: comparing findings on arthroscopic evaluation to pre-operative magnetic resonance imaging. J Clin Orthop Trauma. 2018;9:S123–S128.
22. Roy J, Braën C, Leblond J, Desmeules F, Dionne CE, Macdermid JC, et al. Diagnostic accuracy of ultrasonography, MRI and MR arthrography in the characterisation of rotator cuff disorders: a systematic review and meta-analysis. Br J Sport Med. 2015;49:1316–1328.
23. Junge T, Henriksen P, Andersen HL, Byskov LD, Knudsen HK, Juul-kristensen B. The association between generalized joint hypermobility and active horizontal shoulder abduction in 10 – 15 year old competitive swimmers. BMC Sports Sci Med Rehabil. 2016;4–9.
24. Watson L, Balster S, Hons B, Lenssen R, Hoy G, Pizzari T. The effects of a conservative rehabilitation program for multidirectional instability of the shoulder. J Shoulder Elb Surg. 2018;27(1):104–111.

ISSN 0008-3194 (p)/ISSN 1715-6181 (e)/2019/178–186/$2.00/©JCCA 2019

Conservative management of Morel-Lavallée lesion: a case study

Trevor Vander Doelen, BSc, MSc, DC[1]
Adam Manis, BSc, MSc, MD, Dip Sport Med[1]

Objective: *To illustrate the conservative clinical management of Morel-Lavallée lesion.*

Clinical features: *A 34 year-old male presented with a forced flexion induced right knee injury that was diagnosed as a Morel-Lavallée lesion by way of Magnetic Resonance Imaging (MRI).*

Intervention and outcome: *The patient was co-managed by a sports medicine physician and a chiropractor with a combination of percutaneous aspiration, dry needling, soft tissue therapy, extracorporeal shockwave therapy (ESWT), and corrective exercise.*

Summary: *The patient experienced improved function throughout chiropractic care. Almost complete resolution was achieved at 18 weeks after injury and maintained at 35 weeks after injury. Further research is needed to understand if this improvement in function is better than the natural history of this condition.*

(JCCA. 2019;63(3):178-186)

KEY WORDS: chiropractic, extracorporeal shockwave therapy, conservative management, electroacupuncture, fascial abrasion tool, Morel-Lavallée lesion

Objectif : *Illustrer la prise en charge conservatrice clinique de la lésion de Morel-Lavallée.*

Caractéristiques cliniques: *Un homme de 34 ans s'est présenté avec une blessure au genou droit provoquée par une flexion forcée qui a été diagnostiquée comme une lésion de MorelLavallée grâce à l'imagerie par résonance magnétique (IRM).*

Intervention et résultats : *Le patient a été pris en charge conjointement par un médecin du sport et un chiropraticien avec une combinaison d'aspiration percutanée, d'aiguilles sèches, de thérapie des tissus mous, de thérapie par ondes de choc extracorporelles et d'exercices correctifs.*

Résumé : *L'état du patient s'est amélioré tout au long des soins chiropratiques. Une guérison presque complète a été atteinte 18 semaines après la blessure et 35 semaines après la blessure, le patient été guéri. D'autres recherches sont nécessaires pour comprendre si cette amélioration de la fonction est meilleure que l'histoire naturelle de cette condition.*

(JCCA. 2019;63(3):178-186)

MOTS CLÉS : chiropratique, thérapie par ondes de choc extracorporelles, prise en charge conservatrice, électro-acupuncture, outil d'abrasion du fascia, lésion de MorelLavallée

[1] Private practice, Toronto ON

Corresponding author: Trevor Vander Doelen, 4–1209 King Street West, Toronto, ON M6K 1G2
E-mail: dr.vanderdoelen@gmail.com

The authors have no disclaimers, competing interests, or sources of support or funding to report in the preparation of this manuscript.
The involved patient provided consent for case publication.

Figure 1. *Inferomedial thigh bruising present five days after trauma.*

Figure 2. *Lower leg bruising present five days after trauma.*

Introduction

The Morel-Lavallée lesion (MLL) is a closed soft tissue injury usually sustained from trauma.[1,2] It can be described as a degloving injury, as the injury involves shearing forces and separation of the hypodermis from the underlying fascia, similar to removing a glove.[2] This causes disruption of the vasculature and lymphatic structures resulting in hemolymphatic fluid collection between the tissue layers. This injury needs to be diagnosed, preferably with Magnetic Resonance Imaging (MRI), and managed in a timely fashion as to prevent infection, pseudocyst formation, cosmetic deformity, and musculoskeletal dysfunction.[1,2] There is a gap in the academic literature in regards to the conservative management of this condition and no clinical guidelines currently present. The purpose of this case study is to present the successful results of a multimodal conservative treatment approach that focuses on both the local lesion and the movement compensations associated with it.

Case presentation

A 34 year-old male presented to a chiropractic clinic with right knee pain and bruising of the inferomedial aspect of their thigh and lower leg. The patient had not seen a health care provider prior to this visit. The patient stated that this pain and bruising began after a traumatic sliding maneuver that involved sudden flexion of his right knee playing American football five days prior. There was no audible "pop" or "snap" with this sudden flexion. He was unable to continue playing due to pain. Swelling and bruising began the next day. The patient described their pain as dull, with a severity of 5/10, localized to the medial aspect of the knee and was causing a limp while walking. There was no distal pain, paresis, or paresthesia described. Since the injury, but prior to the physical examination, the patient had travelled on an airplane and attempted to play golf, but was unable to continue due to knee pain. The patient had not taken any medication and the patient's systems review, family history, and medical history were unremarkable

On gait examination, the patient's gait was antalgic but was able to weight bear with minimal pain. The patient was unable to extend their right hip and right knee during the gait cycle. Right ankle dorsiflexion was also limited as compared to the asymptomatic side during the gait assessment. Observation revealed bruising on the inferomedial thigh and medial ankle (see Figures 1 and 2) but no edema. Minimal tenderness was noted on palpa-

Table 1.
Lower Extremity Functional Scale (LEFS) results

Visit	Date	LEFS Score
Initial Visit	Feb 6, 2018	10/80
Follow Up 1	March 2, 2018	56/80
Follow Up 2	March 21, 2018	68/80
Follow Up 3	June 14, 2018	79/80
Follow Up 4	October 10, 2018	77/80

tion of the bruised area, however there was tenderness of the right semitendinosis tendon and medial joint line. The pedal pulse was palpated bilaterally. Lower limb sensory, motor, and reflex testing revealed normal neurological findings bilaterally. Range of motion testing revealed active and passive knee flexion were reduced, as compared to the asymptomatic side. Active knee flexion range of motion (ROM) was measured at 110° and passive knee flexion ROM at 115°. This limitation was due to superficial tension in the anterior quadricep musculature as described by the patient. Muscle testing of the trunk, hip, and thigh musculature was performed isometrically in a supine position and revealed a weak right gluteus maximus (3/5) when tested in a neutral hip position with 90 degrees of knee flexion as well as a weak right quadratus lumborum (3/5). Left hip, leg, and trunk musculature tested strong (5/5) with isometric muscle testing with the patient in a supine position.

Orthopedic testing of the right knee included Lachman, McMurray, anterior drawer, posterior drawer, Thessaly's, pivot-shift, varus, and valgus stress tests were within normal limits. A lower extremity functional scale (LEFS) outcome measure was completed at each visit (see Table 1). A score of 10/80 was recorded at the initial visit indicating severe disability.

Due to the significant ecchymosis, the patient was referred to a sports medicine physician (SMP). The patient was seen by the SMP the same day and was referred for a right knee radiographic series and diagnostic ultrasound. The imaging results were found to be negative two days later and the patient was then sent by the SMP for an MRI. The results of the MRI performed nine days later revealed:

- No meniscal, cruciate, or collateral ligament tears but potential low grade sprain of proximal portion of ACL and MCL
- Large subcutaneous fluid collections (10 cm x 12 cm) superficial to fascia and within the subcutaneous compartments in keeping with the degloving MLL
- Question raised of deep vein thrombosis of the popliteal vein

Upon receiving these results and an urgent call from the radiologist, the SMP ordered a venous doppler of the right lower extremity to rule out deep venous thrombosis, which the patient had performed the same day. The results of the venous doppler received two days later revealed no evidence of deep vein thrombosis above or below the right knee. Two days after the venous doppler, and approximately three weeks post-injury, the patient presented to the SMP and it was noted that the area was not red or hot. The SMP explained the risks and benefits of needle aspiration of the area to remove fluid and reduce pressure. The patient consented to treatment and the SMP removed 100 ml of non-purulent blood. The patient was advised to follow up in five days and in the meantime wear a compressive sleeve, monitor the area for signs and symptoms of infection, and begin rehabilitation with the initiating chiropractor. That week the patient was unable to see the chiropractor due to work travel.

The patient presented to the SMP five days later and reported reduced pain and increased ROM. The patient was advised to begin riding a stationary bike and see the chiropractor for manual therapy and corrective exercise. The patient was again advised to monitor the area for signs and symptoms of infection.

Upon follow-up with the chiropractor 8 days after the aspiration and approximately four weeks post-injury, the patient was more functional with a LEFS score of 56/80. Their pain had decreased and passive knee flexion ROM was 125 degrees, an improvement of 10 degrees since the first visit. There was slight tenderness superficially on palpation of the patella and bruised area but no heat or erythema present. Superficial fluid was still present proximal and medial to the right patella. The chiropractor used soft tissue therapy and a fascial abrasion tool (FAT tool) to reduce tension in the hip flexor myofascia, anterior and posterior thigh myofascia, and calf myofascia for the pur-

Box 1. *Exercise prescription*

- Hip flexor stretching in a lunge position
- Two legged glute bridge
- Box squat
- Sliding lunge
- Retlouping (see Figure 3)
- Kettlebell swings (20 lbs)
- Kettlebell front squats (20 lbs)
- Goblet squat (20 lbs)
- Sumo deadlift
- Bulgarian split squat (no weight)

pose of improving hip extension ROM and ankle dorsiflexion ROM that were found to be reduced during initial gait assessment. The chiropractor also used dry needling with electrical stimulation at a current frequency of 2 Hz into the right hip capsule, anterior portion of gluteus medius, tensor fascia latae, right vastus lateralis, right vastus medialis, right saphenous nerve, right common peroneal nerve, and the right recurrent genicular nerve for the purpose of trigger point release and pain reduction.[3-6] The chiropractor avoided needling areas of effusion or bruising. The patient was given common corrective exercises to be performed in between visits (Box 1). One set of ten repetitions of half of the prescribed exercises were to be done every other day. The lesser known retlouping exercise (Figure 3), typically used for postsurgical knee patients, was performed to improve hamstring flexibility and knee extension ROM. Sliding lunges were performed in all planes of movement to a ROM within the patient's pain tolerance.

Four days later the patient presented to the SMP and the right knee ROM was now full with only slight pain at full flexion. Cycling and corrective exercises had been well tolerated. A superficial edema region measuring 3 cm in diameter just above the patella was noted. No tenderness, heat, or erythema noted. A follow up visit in two weeks was advised.

The patient presented to the chiropractor for their second follow up visit at seven weeks post-injury and four weeks post-aspiration, and reported no pain in the knee with day-to-day activity and had now attempted jogging, the elliptical machine, lunging, and squats successfully without pain. A LEFS score of 68/80 was recorded. The chiropractor again used soft tissue techniques including manual therapy and a FAT tool to reduce tension in the hip

Figure 3.
Retlouping exercise.

flexor myofascia, anterior and posterior thigh myofascia, and calf myofascia. The chiropractor also used dry needling with electrical stimulation at a current of 2 Hz into the right hip capsule, anterior portion of gluteus medius, tensor fascia latae, right vastus lateralis, vastus medialis, right saphenous nerve, right common peroneal nerve, and the right recurrent genicular nerve. The chiropractor still avoided needling areas of effusion or bruising. The patient was encouraged to gradually increase the load with his strength training and was prescribed trunk and hip focused exercises including the dead bug exercise, bird dog exercise, and front and side planks.

The third follow up visit was approximately 19 weeks post-injury and 16 weeks post-aspiration. At this visit the patient presented with almost complete function and a LEFS score of 79/80. There was still lingering sensitivity on the superficial aspect of the patella, but the patient reported they were very functional with activities of daily living. The chiropractor again used soft tissue techniques including manual therapy and a FAT tool to reduce tension in the hip flexor myofascia, anterior and posterior thigh myofascia, and calf myofascia. The chiropractor again used dry needling with electrical stimulation at a current of 2 Hz into the right hip capsule, anterior gluteus medius, tensor fascia latae, right vastus lateralis, vastus medialis, right saphenous nerve, right common peroneal nerve, and the right recurrent genicular nerve.

The patient presented to the chiropractor 17 weeks later (36 weeks post-injury and 33 weeks post-aspiration) with slight discomfort proximal to the patella and a LEFS score of 77/80, a possible slight regression since the previous visit. However this slight regression was less than the minimum clinically important difference of 9 points for the LEFS.[7] The distal quadricep musculature slightly proximal to the patella was palpably hardened as compared to the left quadricep. Due to suspected "scarring" of the underlying myofascia, the chiropractor advised using Extracorporeal Shockwave Therapy (ESWT) in the region and the patient consented. An ESWT session (Storz MP100 unit) of 2000 hits at 15 Hz with an intensity of 2 bars was used. The patient reported slight discomfort during the ESWT application but insisted it was manageable. Long-term outcomes after this visit and beyond 17 weeks post-injury were not available for the purposes of this study.

Management

Treatment interventions for this condition were performed by the chiropractor and the SMP and included percutaneous fluid aspiration, soft tissue manual therapy, instrument-assisted soft tissue mobilization, dry needling with electrical stimulation, corrective exercise and radial extracorporeal shockwave therapy.

Percutaneous Fluid Aspiration

Percutaneous aspiration of 100 ml of fluid was performed three weeks post injury by the SMP.[8]

Soft-Tissue Manual Therapy

Myofascial soft tissue therapy was applied to the hip myofascia, anterior and posterior thigh myofascia, and calf myofascia bilaterally to improve ROM of these tissues and the associated joints.[9,10]

Instrument-Assisted Soft Tissue Mobilization (IASTM)

IASTM has been shown to be an effective tool for reducing the pain pressure threshold of a myofascial trigger point which involves pain, weakness, and dysfunction.[11] A FAT tool was used on the anterior thigh and calf musculature on the ipsilateral side of the lesion for the purpose of improved myofascial ROM and pain reduction.[11,12]

Dry Needling With Electrical Stimulation

Dry needling involves the insertion of acupuncture needles without injectate into or around nerves, muscles, and or connective tissue for the management of pain and dysfunction in neuromusculoskeletal conditions by way of neurohumoral response.[3-6] Dry needling was not used around the bruised lesion or in areas with edema, but was used proximal and distal to this region. Needle insertion sites during the initial visit included the proximal quadriceps on the ipsilateral side for pain control and muscular tension reduction. Subsequent visits involved needle insertion into the right hip capsule, anterior gluteus medius, tensor fascia latae, right vastus lateralis, vastus medialis, adjacent to the right saphenous nerve, right common peroneal nerve, and the right recurrent genicular nerve. These dry needling points were chosen as they displayed hypertonicity on palpation, reflective of trigger points.[6]

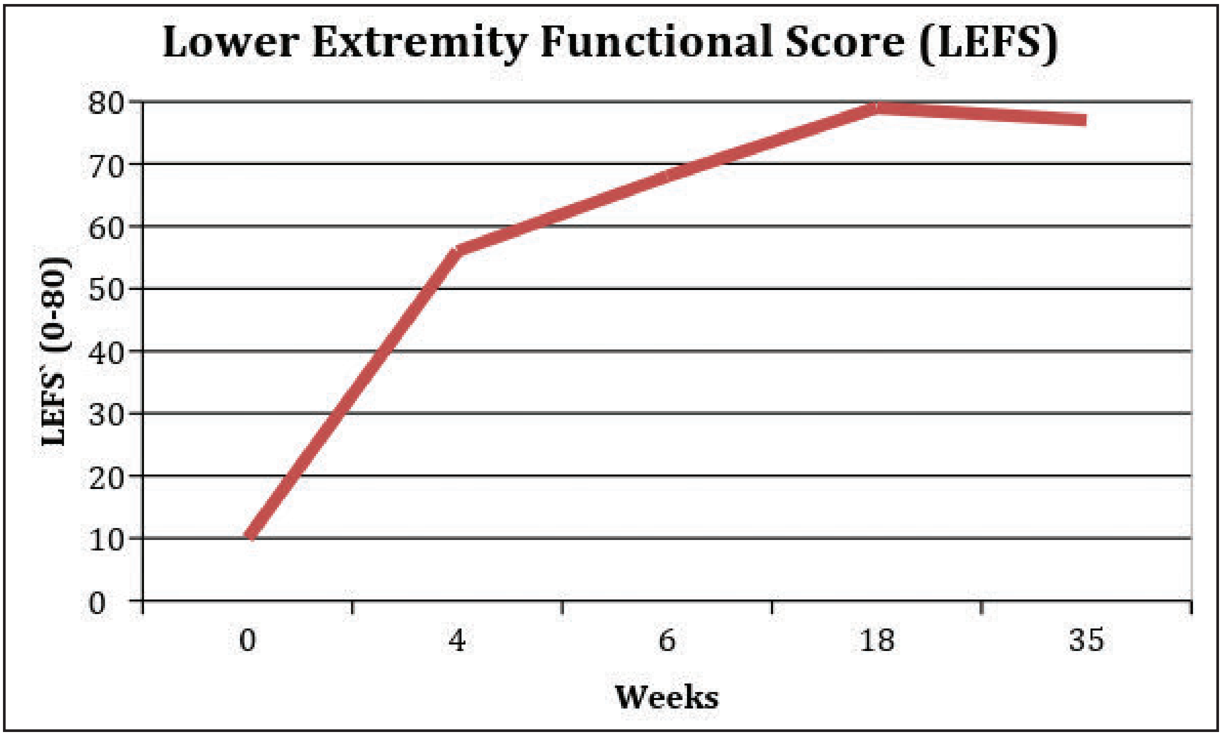

Figure 4.
Lower Extremity Functional Scale.

Corrective Exercise
A corrective exercise approach with the goal of returning to right lower extremity function involved trunk, hip, knee, and foot mobility and stability exercises. These were used in conjunction with a gradual progression of cycling, elliptical, and running.[13]

Extracorporeal Shockwave Therapy (ESWT)
Radial ESWT was applied to the lesion during the final visit (35 weeks after the initial visit) as the lesion had been chronic at this stage and ESWT can significantly improve the healing process of chronic wounds.[14]

Follow-up and Outcomes
The LEFS was used as an outcome measure. The questionnaire was completed at the start of each chiropractic visit. The long-term follow-up beyond 35 weeks was not included in this document as it was still ongoing (see Figure 4).

Discussion

Epidemiology
MLL is uncommon and has been reported to be involved in 8.3% of pelvic traumas.[15] It is often associated with acetabulum and pelvic fractures but can also be related to knee trauma, which is less severe.[15-17] However, over the period of 14 NFL seasons, one team reported 27 MLLs in 24 American football athletes, which may suggest the injury is more common than once thought.[17]

Pathology
MLL occurs due to a shearing of superficial subcutaneous tissue away from underlying fascial layers (see Figure 4). Areas of mobile overlying skin and tough underlying fascia like the proximal lateral thigh and quadriceps fascia proximal to the knee are more prone to this condition.[16,17] Following tissue separation, blood and lymph tissue are able to travel more freely and often pool in between lay-

ers of tissue.[1,2, 15-17] Blood within these cavities begins to resorb leaving a seroanguinous fluid surrounded by a haemosiderin layer which induces inflammation and leads to a fibrous capsule. If not intervened promptly, this capsule will lead to a chronic MLL.[16]

Signs and symptoms

MLL typically presents as enlarged, tight, and tender tissue that may contain ecchymosis, erythema, and surface abrasion of the skin. An important clinical feature is the presence of fluctuance within the lesion. Potential differential diagnoses to consider include haematoma, fat necrosis, haemangioma, soft tissue sarcoma, early myositis ossificans, or bursitis. MLL can typically be discerned based on the mechanism of injury and the progression of symptoms.

Investigations

When trauma and the signs and symptoms mentioned above are present, MLL should be ruled out with diagnostic imaging.[16,18] Diagnostic ultrasound is an inexpensive initial investigative tool and typically demonstrates the lesion as anechoic relative to hyperechoic mass.[19] MRI is the imaging modality of choice and should be ordered urgently, as done in this case. Diagnostic ultrasound is less useful but Doppler ultrasound can be used to rule out a suspicion of vascular compromise such as deep vein thrombosis, as done in this case.[16] Computed Tomography (CT) does not provide value in the assessment of MLL.[18]

Classification

Mellado and Bercandino[20] have proposed a classification system for MLL based on size, MRI characteristics, and capsular presence. Shen *et al*. have more recently suggested using a more simplified classification system identifying MLL as either acute or chronic. According to Shen *et al*., MLL can be diagnosed as chronic when the lesion contains a capsule.[8]

Complications

Since the mechanism of injury is commonly traumatic, bacterial infection can be a concern in MLL due to an open lesion of the skin. While infection rates with MLL are inconclusive, due to the severity of the potential complications with infected tissue, this should be the primary concern with this lesion.

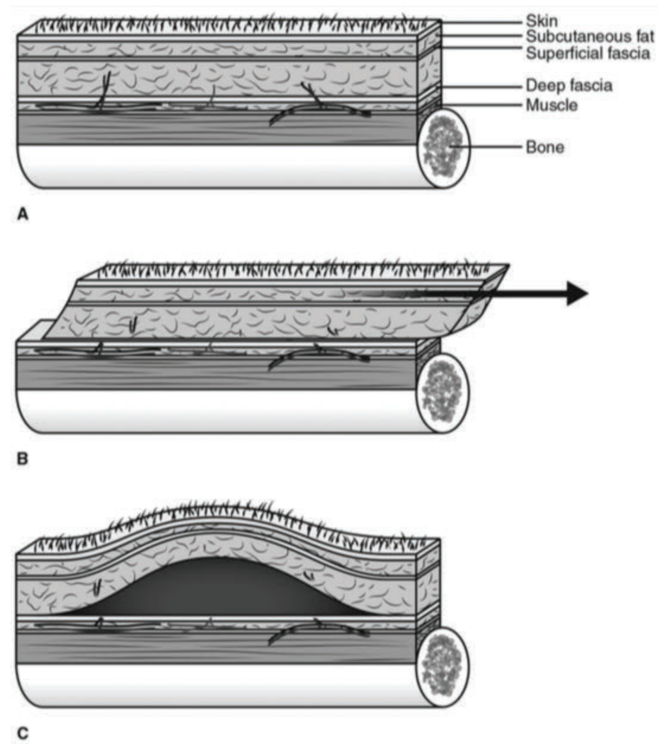

Figure 5.
Cross-section of tissue from skin to bone (image reproduced with permission of Scolaro et al.[2]).

Management

Management decisions for MLL are based on the volume of the cavity created in the lesion, as well as the chronicity of the lesion. There are currently no management guidelines for the management of MLL. Many small cohort studies have investigated the efficacy of conservative management, percutaneous aspiration, sclerodesis, and open surgery, but there is currently no high quality evidence to support any one treatment strategy over the other.[1,2,15,16] As for conservative management measures, compression is thought to flush out the inevitable edema in the area and has shown good effects in knee related MLL.[8,17,18] High recurrence rates of MLL have been shown after percutaneous aspiration of the lesion. MLL with a volume greater than 50 ml has a higher likelihood of recurring than those with less than 50 ml of fluid.[8] This patient had 100 ml aspirated, was informed of this higher likelihood of recurrence and yet did not present with a recurrence while under care. Sclerodesis to close off the cavity has been successfully used in MLL. The sclerodesis agents cause cell destruction within the periphery of the lesion, which then induces fibrosis.[8] Doxycycline as a sclerodesis agent has shown the best results and is recommended in a recent review.[1,21] Sclerodesis has been shown to be efficacious in lesions up to 700 ml in volume, with the mean volume of treated lesions being approximately 400 ml.[22] Surgical intervention reduces recovery time as compared to compression therapy on its own, other than in cases of acute MLL around the knee.[12] Open drainage and mass resection are also options that can be used if there is a larger volume or if not responding to more conservative strategies. The clinical decisions made by the SMP were to use minimally invasive strategies like aspiration and compression and the outcomes along with chiropractic care were successful.

The treatment interventions chosen by the chiropractor and described in the management section above were with the goals of first managing pain, mobilizing the soft tissue structures above and below the MLL lesion, preventing scarring of the lesion, increasing muscle strength, and improving function.[4-12]

Summary

The MLL is an uncommon soft tissue degloving injury that should be considered as a differential diagnosis for any patient who has sustained a serious traumatic injury. A wide variety of treatment interventions have been proposed in the literature, yet none considering soft tissue conservative management including dry needling with electrical stimulation, soft tissue manual therapy, instrument-assisted soft tissue therapy, corrective exercise and ESWT. The treatment(s) chosen by a health care practitioner for a patient presenting with MLL should be made on an individualized basis based on the size and chronicity of the lesion. This case study illustrates that conservative soft tissue treatment options may be considered for patient's presenting with MLL if the lesion is relatively small (less than or equal to 100 ml) and in the acute stage. The successful conservative management of this patient, more prone to recurrence due to a percutaneous aspiration greater than 50 ml, demonstrated the potential for improving function and preventing recurrence with a multimodal conservative treatment approach. Long term follow-up with this patient, beyond 35 weeks, would allow for comment on longer term efficacy of the interventions. Future research is warranted to consider if these treatment strategies would also be helpful to improve function, reduce symptoms and prevent recurrence for larger MLL lesions or those in the chronic stage, either on their own, or in addition to less conservative options such as percutaneous aspiration or sclerodesis.

References

1. Singh R, Rymer, B, Youssef B, Lim, J. The Morel-Lavallée lesion and its management: a review of the literature. J Orthopaed. 2018; 15: 917-921.
2. Scolaro JA, Chao T, Zamorano DP. The Morel-Lavallée lesion: diagnosis and management. J Am Acad Orthop Surg. 2016;24(10): 667-672.
3. Dunning, J, Butts R, Mourad F, Young I, Flannagan S, Perreault T. Dry needling: a literature review with implications for clinical practice guidelines. Phys Ther Rev. 2014: 19(4): 252-265.
4. Huang C, Wang Y, Han JS, Wan Y. Characteristics of electroacupuncture-induced analgesia in mice: variation with strain, frequency, intensity and opioid involvement. Brain Res. 2002;945(1): 20-25.
5. Ulett GA, Han S, Han JS. Electroacupuncture: mechanisms and clinical application. Biologic Psychiatr. 1998;44(2): 129-138.
6. Gattie E, Cleland JA, Snodgrass S. The effectiveness of trigger point dry needling for musculoskeletal conditions by physical therapists: a systematic review and meta-analysis. J Orthopaed Sports Phys Ther. 2017;47(3): 133-149.
7. Binkley JM, Stratford PW, Lott SA, Riddle DL. The lower extremity functional scale (LEFS): scale development, measurement properties, and clinical application. Physical Ther. 1999; 79: 371-383.
8. Shen C, Peng J, Chen X. Efficacy of treatment in peri-pelvic Morel-Lavallée lesion: a systematic review of the literature. Arch Orthop Trauma Surg. 2013: 133(5): 635-640.
9. Tak S, Lee Y, Choi W, Lee G. The effects of active release technique on the gluteus medius for pain relief in persons with chronic low back pain. Physical Ther Rehabil Sci. 2013; 2(1): 27-30.
10. Piper S, et al. The effectiveness of soft-tissue therapy for the management of musculoskeletal disorders and injuries of the upper and lower extremities: a systematic review by the Ontario Protocol for Traffic Injury Management (OPTIMa) Collaboration. Manual Therapy 2015; 21: 18-34.
11. Gulick D. Instrument-assisted soft tissue mobilization increases myofascial trigger point pain threshold. J Bodywork Movement Ther. 2018; 22(2): 341-345.

12. Cheatham SW, Lee M, Cain M, Baker R. The efficacy of instrument assisted soft tissue mobilization: a systematic review. J Can Chiropr Assoc. 2016; 60(3): 200-211.
13. Kristensen J, Franklyn-Miller A. Resistance training in musculoskeletal rehabilitation: a systematic review. Brit J Sports Med. 2012; 46(10): 719-726.
14. Zhang L, Weng C, Zhao Z, Fu X. Extracorporeal shock wave therapy for chronic wounds: a systematic review and meta-analysis of randomized controlled trials. Wound Repair Regen. 2017: 25: 697-706.
15. Nickerson T, Zielinski M, Jenkins D, Schiller H. The Mayo Clinic experience with Morel-Lavallée lesion: establishment of a practice management guideline. J Trauma Acute Care Surg. 2014; 76(2): 493-497.
16. Bonilla-Yoon I, Masih S, Patel D, et al. Morel-Lavallée lesion: pathophysiology, clinical presentation, imaging features, and treatment options. Emerg Radiol. 2014; 21(1): 35-43.
17. Tejwani S, Cohen S, Bradley J. Management of Morel-Lavallée lesion of the knee: twenty-seven cases in the national football league. Am J Sports Med. 2007; 35(7): 1162-1167.
18. Nair A, Nazar P, Sekhar R, Ramachandran P, Moorthy S. Morel-Lavallée lesion: a closed degloving injury that requires real attention. Indian J Radiol Imag. 2014; 24(3): 288-290.
19. Gilbert BC, Bui-Mansfield LT, Dejong S. MRI of a Morel-Lavallee Lesion. Am J Roenten. 2004;182: 1347-1348.
20. Mellado JM and Bencardino JT. Morel-Lavallée lesion: review with emphasis on MR imaging. Magn Reson Imaging Clin N Am. 2005;13: 775-782.
21. Bansal A, Bhatia N, Singh A, Singh A. Doxycycline sclerodesis as a treatment option for persistent Morel-Lavallée lesions. Injury. 2013; 44(1): 66-69.
22. Penaud A, Quignon R, Danin A, Bahe L, Zakine G. Alcohol sclerodhesis: an innovative treatment for chronic Morel-Lavallée lesions. J Plast Reconstr Aesthetic Surg. 2011; 64(10): 262-264.

ISSN 0008-3194 (p)/ISSN 1715-6181 (e)/2019/187–196/$2.00/©JCCA 2019

Exploring the role of microinstability of the hip: an atypical presentation of femoroacetabular impingement (FAI) and labral tear in a collegiate endurance athlete: a case report

Mitchell Savic, BHSc(Hons), DC, FRCCSS(C)[1,2]
Kevin D'Angelo, BSc(Hons), DC, FRCCSS(C)[2]

Objective: *This case is designed to aid practitioners in understanding the potential role of hip microinstability as a possible underlying source of hip pain and dysfunction.*

Case presentation: *A 25-year-old female collegiate cross-country athlete presented with a 2-year history of progressive left hip and groin pain. Extensive clinical examination and imaging confirmed the presence of cam-type femoroacetabular impingement, a labral tear and gluteal tendinopathy. Despite multiple intra and extra-articular pathologies, understanding the role of hip instability and implementing a rehabilitation exercise program focused on hip joint centration alleviated the patient's symptoms at rest and during activity.*

Summary: *A robust history and physical exam of the hip is essential with the addition of imaging when*

Objectif : *Ce cas est conçu pour aider les praticiens à comprendre le rôle potentiel de la microinstabilité de la hanche comme source sous-jacente possible de douleur et de dysfonctionnement de la hanche.*

Exposé de cas : *Une athlète de cross-country collégiale âgée de 25 ans présente des antécédents médicaux de deux ans d'une douleur progressive située au niveau de la hanche gauche et de l'aine. Un examen et une imagerie cliniques approfondis ont confirmé la présence d'un conflit fémoroacétabulaire de type came, d'une déchirure du labrum et d'une tendinopathie glutéale. Malgré de multiples pathologies intra et extra-articulaires, la compréhension du rôle de l'instabilité de la hanche et la mise en œuvre d'un programme d'exercices de réadaptation axé sur l'articulation de la hanche ont atténué les symptômes du patient au repos et au cours de l'activité.*

Résumé : *Une anamnèse robuste et un examen physique de la hanche sont essentiels avec l'ajout de*

[1] Division of Graduate Studies, Canadian Memorial Chiropractic College, Toronto, Ontario
[2] Private practice, Burlington, Ontario

Corresponding author: Mitchell Savic, Canadian Memorial Chiropractic College, 6100 Leslie Street, Toronto, Ontario, M2H 3J1
E-mail: drmitchsavic@gmail.com
Tel: 289-338-7277

The authors have no disclaimers, competing interests, or sources of support or funding to report in the preparation of this manuscript. The involved patient provided consent for case publication. The CMCC Research and Ethics Board have approved this case report.

testing criteria is positive. Clinicians should be aware of the role hip microinstability plays and its clinical implications when in the presence of other contributing factors such as generalized joint laxity, and/or intra-articular pathology.

(JCCA. 2019;63(3):187-196)

KEY WORDS: chiropractic, femoroacetabular impingement, FAI, hip, labral tear, microinstability

l'imagerie lorsque les critères d'examen sont positifs. Les cliniciens devraient être conscients du rôle que joue la micro-instabilité de la hanche et de ses implications cliniques lorsqu'elle est en présence d'autres facteurs contributifs tels que l'hyperlaxité généralisée et la pathologie intra-articulaire.

(JCCA. 2019;63(3):187-196)

MOTS CLÉS : chiropratique, conflit fémoro-acétabulaire, CFA, hanche, déchirure du labrum, micro-instabilité.

Introduction

The hip is a complex region owing to the fact of several overlapping intra and extraarticular anatomical structures, and several nearby musculoskeletal regions which may refer to and mimic hip pathology.[1-8] Some of the structures that can manifest clinically as hip pain either may not be well recognized as a source of hip pain, or perhaps the assessment of such structures is not sufficient to make a correct diagnosis. This may be owing to the lack of validated tests and deficiencies in the literature on this specific area.[1-7] Hip microinstability, a functionally progressive condition which typically presents alongside multiple hip pathologies can also play a role, particularly in the young athletic population. Although both traumatic (macro) hip instability and microinstability have been described to cause symptomatic supraphysiological translation of the femoral head in the acetabulum, microinstability is not as objectively obvious and typically presents after a long history of repetitive axial or rotational loading of the hip joint resulting in progressive development of symptoms in the absence of a clear underlying etiology.[9-12] For these reasons, diagnosing hip pathology is a diagnostic challenge.[1-3,7] The clinical utility of physical examination tests for diagnosing hip pathology have been criticized, which is one of the primary reasons why radiographic and special imaging modalities are used as a primary means to diagnose.[7]

The purpose of this case study is to create a diagnostic tool for clinicians by highlighting an endurance athlete displaying a unique presentation of hip pain. This case will systematically facilitate a complete list of hip differential diagnoses and recommended examination and/or imaging tests to help guide manual practitioners in the assessment and diagnosis of hip pain/pathology.

Case presentation

A 25-year-old female runner presented to a sport specialist chiropractor with a two-year history of progressive left hip and groin pain. She was a former collegiate cross-country athlete and this was her first year removed from her varsity career. She reported her pain began in the last year of her competitive running season and came on approximately 45 minutes into her training runs and would progressively get worse. Her left hip pain was described as a heavy fatigue feeling which began in the left gluteal region and would radiate into the posterolateral and anterolateral thigh with distance. While at university, she was overseen by her assigned athletic therapist who had treated her injury as a hip flexor and gluteal strain. She had also saw a medical doctor during her last year of competition who also believed her hip pain was muscular in origin and suggested she continue with physical therapy. After leaving university, she briefly competed in both half and full marathons but had to give up running in the last year due to the onset of sharp left hip and groin pain during her runs. Unlike the previous pain, her symptoms would initially present around the 10-km mark into her runs and got worse with increasing distance to the point where she had to stop her training and running sessions. The pain this time became sharper into the groin area during the runs and would present as a deep-seated dull pain in the groin and posterior hip following activity such as running. There were no episodes of radiating leg pain or give-way sensations reported during her initial presentation of pain. The patient also reported no previous history of imaging, trauma, effusion, hospitalizations or co-morbidities.

Over the past year, the patient had sought treatment by several physiotherapists, massage therapists and chiropractors who were treating her under the diagnosis of a chronic hip flexor tendinopathy using with soft tissue techniques, laser, and exercises. She reported the treatment offered her temporary relief and allowed her to perform her activities of daily living (ADLs) and resistance-based training workouts with minimal discomfort. However, in the last six months, her pain in the hip became sharp during squats, lunges, and with sitting longer than two hours. She reported becoming anxious as she feared she would have to start avoiding her active lifestyle at the expense of her increasing hip pain. The pain was reported on a visual analogue scale (VAS) as a 3/10 at rest and as a high as an 8/10 when preforming tasks such as lunges, squats and attempting to run. She had not received imaging at this point but voiced her concerns and was waiting to see her medical doctor in a few weeks for her hip pain. Based on the recommendation from a former teammate, she presented to sport specialist chiropractor.

Upon initial observation, the patient demonstrated an externally rotated foot and tibia on the left during single leg stance, double leg stance and the stance phase of the gait cycle. Performance of both overhead and two-legged squats revealed left hip and groin pain at approximately 90 degrees of hip flexion. Lumbar spine range of motion testing (active, passive and resisted) was unremarkable bilaterally. Noting her extreme ease to place both palms of her hands on the floor during lumbar flexion, a Beighton's Scale exam[13] was performed where she scored 7/9 (positive for lumbar flexion, bilateral elbow and knee hyperextension, bilateral 1st phalanx extension). Visual examination of the low back and both hips demonstrated no signs of effusion, ecchymosis or deformities.

Physical examination of both hips revealed a dull pain in the groin on end-range flexion and internal rotation. When performing external rotation in full hip flexion on the left, the patient reported sharp pain along both the posterolateral aspect of the hip and groin. This became more pronounced when taking the same (left) hip into extension and external rotation. Both the left hip scour and flexion-adduction-internal-rotation (FADDIR) tests were painful and created similar dull groin pain with no noticeable catching or reproduction of mechanical locking. The left hip FADDIR test was deemed a hard end feel at end range in comparison to the right hip. Performance of the posterior hip impingement test and external de-rotation tests on the left hip recreated her characteristic hip and sharp groin pain.[7,10] Despite pain in the external de-rotation test, there was no notable differences in strength when compared to the right side. Active straight leg-raise (SLR), lumbar Kemp's, thigh thrust, and both sacroiliac joint compression and distraction tests were negative. Hip log roll, hip axial loading and valsalva tests were negative. When this same battery of testing was performed on the right hip, all tests were negative. The basis of this clinical hip exam and its justification has been noted in previous published literature[1,7,14] and the clinical rationale to this method will be highlighted in the discussion. Manual palpation of the left quadratus femoris recreated her chief groin and posterolateral hip pain while the capsular portion of her iliopsoas (common) tendon created the

Table 1. *Differential diagnoses for hip pain.*[1,15]

- Intra-articular pathology
  - o Femoroacetabular bony morphologic changes (FAI)
  - o Labral tear
  - o Osteoarthritis
  - o Inflammatory arthritis
  - o Avascular necrosis (AVN)
  - o Dysplasia
  - o Adhesive capsulitis
  - o Synovitis/inflammation
  - o Laxity (traumatic and atraumatic)
- Extra-articular pathology
  - o Snapping hip (internal/external)
  - o Greater trochanteric pain syndrome (trochanteric bursitis)
  - o Ischial & psoas bursitis
  - o Osteitis pubis
  - o Sports hernia (athletic pubalgia)
  - o Piriformis syndrome
  - o Lumbar spine/Sacroiliac joint pathology referral
  - o Obturator internus/gemelli complex tendinopathy/tear
  - o Hip flexor, extensor, abductor, adductor tendinopathy/tear
  - o Sacral stress fracture
  - o Ischiofemoral impingement
  - o Sciatic & pudendal Neuropathy

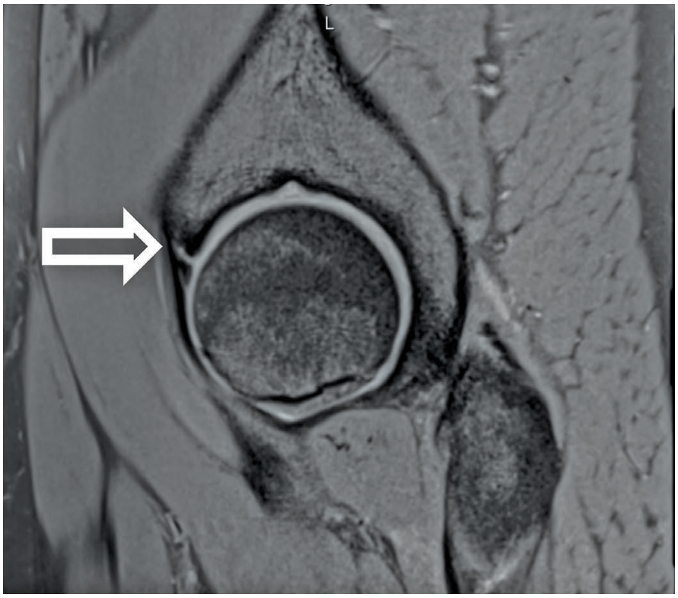

Figure 1.
A T1-weighted sagittal MR image of the left hip demonstrating an acetabular labral tear (white arrow).

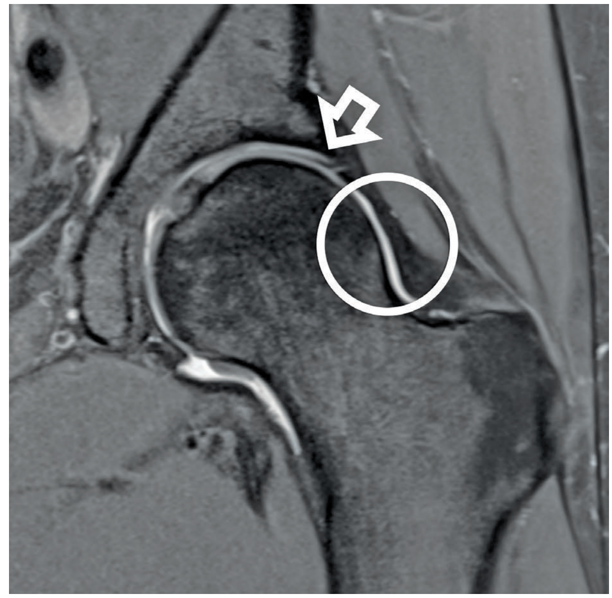

Figure 2.
A T2 fat-suppressed MRI of the left hip demonstrating a mild cam-type morphology (white circle) with an acetabular labral tear (white arrow).

dull, deep left groin pain. Other notable hypertonic and tender areas included the proximal adductor magnus tendon, gluteus minimus and medius, piriformis, and greater sciatic notch on the left. No radicular pain or paresthesia was recreated during palpation and the reported orthopedic tests.

Given her history and clinical exam findings, the patient was diagnosed with suspected left posterior hip (ischiofemoral) impingement and subsequent quadratus femoris irritation. Due to the generalized joint laxity throughout the joints in her body as indicated from the high score on the Beighton's scale, there was also a clinical suspicion of atraumatic hip instability.[15,16] In order to rule out competing intra-articular and extra-articular differential diagnoses (Table 1), she was referred back to her physician with a request for both plain film and MR imaging. Initial plain film imaging suggested a possible cam-type morphology of the left hip and the subsequent MRI confirmed these findings in addition to an acetabular labral tear in the left hip. There was also evidence of edema in the insertional tendon of the left quadratus fem-

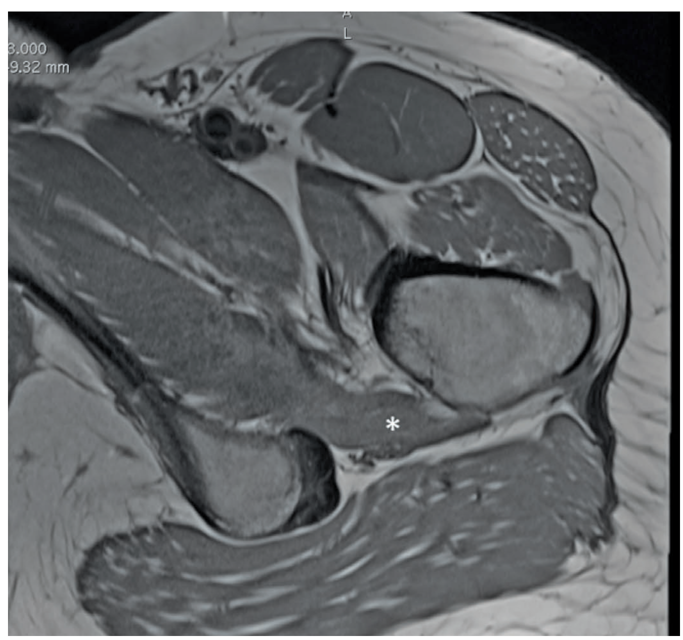

Figure 3.
A T1-weighted axial MR image of the left hip demonstrating edema in the insertional tendon of the quadratus femoris (white asterisk). No active bony edema is present in the left trochanter or ischial tuberosity.

oris with no marrow edema in the ischial tuberosity and greater trochanter (see Figures 1-3). No other findings including partial or full-thickness tears to the surrounding hip musculature was noted. No specific imaging protocols were administered for atraumatic instability of the hip. Previous work[14,17] has discussed these imaging findings in detail for allied healthcare practitioners.

Once the diagnosis was confirmed with advanced imaging, she was scheduled for a consult with an orthopedic surgeon to evaluate her hip. While this consult was pending, the patient was placed on a multimodal plan of management including soft tissue therapy, electroacupuncture, joint mobilizations, and rehabilitation exercise over the course of two months: two times a week in the first three weeks, and once a week for the following three weeks, and one follow up over a two-week period. This plan of management and detailed rehabilitation program has been previously described in the literature[14] and will be explored in further detail in the Discussion (Table 2). When the patient was able to see the orthopedic surgeon three months later, they informed her that she was not a surgical candidate at this time due to her high function and lack of joint related hip pain and was encouraged to keep going with her plan of non-surgical management. Following this visit with the orthopedic surgeon, the patient was seen for treatment twice (two weeks apart) before leaving for a new job out of province. Since she was pain free for most of her activities of daily living and able to complete her desired exercise training, she was given a discharge and encouraged to keep up with her rehabilitation exercises two to three times per week. The one activity she did not return to at this time point was long distance running

Table 2. *Rehabilitation exercise program, adapted from MacIntyre et al*.[14]

Phase	Exercises/Stretches	Reps	Sets	Time
Phase 1	Posterior hip capsule stretching	15	2	30 sec
	Dynamic hip capsule stretching	15	2	
	Potato squat with 5lb medicine ball	12	2	30 sec
	Modified curl-up	12	3	
	Side bridge track	2	3	
	Bird dog track	12	3	
	Pelvic (supine) bridge with theraband at knees	12	3	45 sec
	Side-lying hip abduction	15	2	
	Deadbug with isolated single limb movement	15	3	
	Single leg balance on disc/pillow		3	
	BOSU squats	15	2	
	Goblet Squat with 20lb dumbbell	12	2	60 sec
Phase 2	Deadbug (contralateral movement + exercise ball)	15	3	
	Pallof press track (split stance to upright posture)	15	3	15
	Exercise ball plank to stir-the-pot	2	3	sec
	Single leg squat (BOSU)	15	3	
	Step ups & lateral cross-over step up	15	3	
	Multi-angle lunge (clock lunge)	15	3	
	Multi-plane monster walks	15	3	
	Split squats	10	2	
Phase 3	Slideboard multi-angle lunges	15	3	
	Slideboard lateral slides	15	3	
	Single leg box squat	12	3	
	Pallof press on BOSU/stability disc	15	3	
	Zigzag bounds	10	3	
	Front & lateral shuffles (ladder agility drills)	Max	3	5 min
	Tuck jumps to tuck holds on BOSU	10	3	

(limited to <10 km distances) as this was advised by the surgeon on the consult.

Discussion

Epidemiology

The hip is a complex structure to examine, as there are a multitude of different pain generators that clinically speaking, could all present similarly (Table 1). Femoroacetabular pathology is a common cause of anterior hip (groin) pain. Although under-recognized as a possible cause of posterior hip pain, studies have presented this atypical symptomatology.[1] Femoroacetabular impingement (FAI) in particular, a well-documented anatomical bony variant within the hip, should be considered in patients with posterior hip pain, especially in cases where anterior hip (groin) pain is also created or exacerbated with hip motion. FAI has also shown to cause chondral and labral injury, and may stimulate early osteoarthritic development in affected individuals.[1,5,6,14,22] A retrospective study of 51 patients with hip pathology who participated in fluoroscopically guided intra-articular hip joint injections identified 71% of patients who had posterior hip pain. In approximately 20% of those with either a labral tear or early osteoarthritic change also experienced posterior hip pain in another retrospective hip arthroscopy study. The vast majority of patients in the latter study also had coexisting anterior hip pain.[8,23]

With respect to this case, the patient presented with transient hip pain that worsened over time. Although a battery of FAI-related orthopedic testing was inconclusive, the posterior distribution of her pain may have been attributed to the cam-type FAI. Interestingly, FAI, labral tears, periarticular muscular weakness and ligamentous or capsular laxity are known contributing factors to microinstability of the hip[9-12,16,24,25] which can further exacerbate conditions such as ischiofemoral impingement and extra-articular hip tendinopathy causing anterior and/or posterior hip pain[10,12,15,16]. Not only can microinstability be the cause of such factors, but it may also be the consequence.[9] As previously mentioned, FAI has the potential to provoke labral injury through the abnormal, premature abutment between the femoral head and acetabular rim at certain extreme hip ranges of motion.[10,16,17] In the presence of an acetabular labral tear, a negative pressure seal once produced by the labrum is now compromised, increasing the potential likelihood of subtle postero-inferior subluxations of the femoral head via a levering moment created by the abnormal bony morphology, consistent with microinstability.[10,16,17,26] The labral seal may also play a role in lubricating the hip joint, adequately attenuating forces in a uniform manner, and preventing direct cartilage contact.[26] A loss of such a seal may be a strong contributor in the development of chondral damage within a hip joint consistent with FAI and a labral injury findings on advanced imaging.[26]

Clinical assessment

A systematic review and meta-analysis examining the diagnostic accuracy of hip FAI/labral tear (LT) orthopedic tests revealed interesting results.[7] Due to vast amount of heterogeneity, variable reference standards and bias seen throughout the examined published articles, only nine studies qualified for meta-analysis examining two orthopedic tests. The flexion-adduction-internal rotation (FADDIR) test and flexion-internal rotation (Flex-IR) test are the only two that are recommended to be used for the purposes of screening for FAI/LT pathology (Table 3). As

Table 3.
Diagnostic properties of physical examination tests for FAI/LT and hip microinstability.[7,18]

Clinical Examination Test	Sensitivity (SN)	Specificity (SP)
FADDIR	96.5% (95% CI, 94% to 99%)	7% (95% CI, 5.0 % to 9.0%)
Flex-IR	97.5% (95% CI, 96% to 99%)	25% (95% CI, 1.0 % to 81.0%)
AB-HEER	80.6% (95% CI, 70.8% to 90.5%)	89.4% (95% CI, 80.5% to 98.2%)
Prone Instability	33.9% (95% CI, 22.1% to 45.7%)	97.9% (95% CI, 93.7% to 100.0%)
HEER	71.0% (95% CI, 59.7% to 82.3%)	85.1% (95% CI, 74.9% to 95.3%)

with all hip examination tests, these two mentioned tests are not specific.[7] It has therefore been a recommendation to include a battery of such tests during clinical examination.[1,7,14]

A recent paper examined the diagnostic accuracy of three physical examination tests in the assessment of hip microinstability using intraoperative identification of instability as a reference standard in 194 patients.[18] Tests of interest included the abduction-hyperextension-external rotation (AB-HEER) test, the prone instability test, and the hyperextension-external rotation (HEER) test (Table 3). The AB-HEER test was determined to have the highest clinical utility with the highest sensitivity of 80.6% and specificity of 89.4%. Although the prone instability test had a very low sensitivity of 33.9%, it had the highest specificity of 97.9%, which would raise the clinical suspicion of hip microinstability assuming a positive test. Lastly, the HEER test scored mediocrely with a sensitivity and specificity of 71.0% and 85.1% respectively. Luckily, not only did all three tests yield a high positive predictive value ranging from 86.3% to 95.5%, there was a 95.0% likelihood that a patient had hip microinstability when all three tests were positive. Although other studies need to be published in order to further validate these tests, they can aid to substantiate this relatively overlooked clinical diagnosis in an area of the body where diagnostic accuracy of physical examination tests are typically low. Both authors feel that this information would have been useful in the case presented to help make clinical hip testing more conclusive and establish a more appropriate differential diagnosis list. Since generalized joint laxity could be a contributor to microinstability, an assessment using the Beighton's Physical Examination Criteria[13] is warranted and had value in the case presented[11].

It is first important to understand that every case is different, each with their own varying degrees of severity and complexities. In the case presented, the patient had a MRI confirmed cam-type FAI and acetabular labral tear in the left hip. There was also evidence of edema in the insertional tendon of the left quadratus femoris suggesting a tendinopathy. The Warwick Agreement, an international consensus meeting consisting of 22 panel members from nine countries and five different medical specialties came to substantial agreement for 6 different statements regarding the diagnosis and management of patients with FAI syndrome (FAIS).[19] It was concluded that FAI should be diagnosed clinically as a motion related syndrome, which includes a consideration of presenting symptoms, clinical signs, and imaging findings. Cam and/or pincer bony morphologic changes in the hip may be present on imaging modalities in those who experience no symptoms. A systematic review[20] reported the prevalence of FAI based off imaging findings of a total of 2,114 asymptomatic hips. Cam morphology was identified in 37% of the sample. There was a 3:1 prevalence of cam morphology favouring athletes versus the general population. Although pincer morphology was more poorly defined, it represented 67% prevalence of the entire sample. Lastly, 68.1% of all asymptomatic hips studied revealed labral injury on MRI. These results support the decision that FAIS should be diagnosed clinically with an assessment integrating various planes of movement.[20]

Management

Treatment options for FAIS varies based on the severity of injury, involvement of surrounding soft tissue structures (i.e. labral tear), age, activity level, and symptoms. Currently, there is no high-level evidence to support a definitive treatment for FAIS. The Warwick Agreement recommends and supports the consideration of three possible treatment options for the management of FAIS which include: conservative care, rehabilitation, and/or surgery. A multidisciplinary, shared-decision making approach between the patient and a team of healthcare professionals is recommended.[19] Conservative care options may include patient education, lifestyle and activity modification, non-steroidal anti-inflammatory drugs, intra-articular steroid injections, and/or watchful waiting. The goals of rehabilitation are to improve: hip stability, neuromuscular control, strength, range of motion, and movement patterns of the lumbo-pelvic region. Combined conservative and rehabilitative approaches which have been detailed previously in the literature[14,21] attempt to address the neuromusculoskeletal insufficiencies in the surrounding soft tissues despite the presence of structural abnormalities (i.e. labrum) or osseous structures (i.e. femoral head). In cases where a non-surgical plan of management fails to adequately restore functionality, an arthroscopic technique with labral debridement/repair or an open technique can be used to address the bony and soft tissue defects which are suspected to be the source of dysfunction in those with FAI and labral tears.[14,19] Regardless of the course of action, whether conservative or surgical, rehabilitation is used as a primary means to address the lumbo-pelvic or lower kinetic chain deficiencies that are suspected to be associated with the hip dysfunction and symptomatology.[14,19] Postoperative rehabilitation protocols have been described, although their value is uncertain due to a lack of high-level clinical studies.

In the case presented, the labral tear in the hip can be considered true hip (macro) instability by definition. However, without advanced imaging and the atypical presentation during clinical assessment, entertaining the possibility of hip microinstability with the presence of multiple hip pathologies would seem plausible to allow implementation a rehabilitation program focusing on joint centration and stability.[11,12] This shifts clinical focus to resolving the largest movement-related deficiency versus focusing on the subtleties of the interrelated or nonrelated tissue specific diagnoses. It is in the authors' opinions that this would result in the most favourable results for a patient and addresses the movement-related issues with hip conditions such as FAIS. Essentially, if the passive structures of the hip are compromised (as they are for the patient in this case), there is a dysfunctional and compensatory over-recruitment of the local deep musculature. These active stabilizers of the hip include the gluteus minimus and medius, quadratus femoris, the gemelli complex, obturator internus and externus, iliocapsularis and the deep fibres of iliopsoas, which all act to reduce the shear forces placed upon the hip joint when recruited sufficiently.[11,12]

Macintyre *et al*.[14] proposed an eight-week rehabilitation program in a case of an elite level ice hockey goaltender with MRI confirmed cam-type FAI and acetabular labral tear who was treated non-surgically. The rehab program consisted of three phases: neuromuscular facilitation, functional training, and sport specific training which were created to progressively improve muscle coordination, strength endurance, and improve lumbo-pelvic stability. The patient also received passive treatment one to two times per week for a total duration of six weeks (total eight treatments) which consisted of Active Release Therapy (ART)®, spinal manipulative therapy (SMT), Mulligan hip Mobilizations with Movement (MWM), and contemporary medical acupuncture. After six weeks of treatment the patient was pain free during rest and exercise, with symptoms only exacerbated with the hip scour and FADDIR tests on the affected hip. By eight weeks the patient returned to play pain free. It is important to highlight this case as it demonstrates a promising outcome with regards to improvements in symptoms and function despite the presence of a cam-type FAI and labral tear which were not treated surgically. This suggests that neuromuscular structures are strongly involved in those with FAI and labral tears, and addressing their dysfunction, alongside the presence of any biomechanical limitations remain a strong predictor in clinical, non-surgical outcomes.[14] Although it is not known as to whether this hockey goaltender had developed hip microinstability, the prescribed exercise program that was implemented would theoretically promote hip joint centration and stability which would be of benefit for our patient, returning her to a pain free state during exercise. Using this case as a guide, a similar plan of manual therapy and rehabilitation exercise was used for the case presented and followed the course of two months: two times a week in the first three weeks, and once a week for the following three weeks, and one follow up over a two-week period. The rehabilitation protocol followed can be found in Table 2.

Revisiting the case

Typically, at risk patients for hip microinstability are young females participating in sports which require excessive hip joint ranges of motion, such as gymnastics, dance, or yoga.[9] Although the patient did not partake in any such sport, she did happen to be a young female who tested positive for generalized joint laxity (Beighton's score of 7/9). Her MRI confirmation of cam-type morphologic changes of the femoral head (FAI) along with a labral tear was also evidence for localized hip laxity and instability. With all these things considered, along with a history of progressively declining symptoms in the absence of a single traumatic event, it is likely that hip microinstability was an active contributor to her hip pain during the evolution of her injury.[9,11,12,24] Considering this patient was an experienced endurance runner with these intra- and extra-articular hip pathologies present, they all could have been exploited during repetitive single leg axial loading. This may have been responsible for episodes of shifting location and character of pain and atypical testing during routine clinical examinations.[10,12] As highlighted in previous sections, it becomes important to have a robust and patient focused clinical examination to best formulate a specific plan of management that addresses the goals of the patient. Understanding the movement-related deficiencies in such a case is a significant factor to tailor a targeted and progressive exercise regimen to increase overall joint stabilization and lower body strength. Furthermore, having the ability to recognize more serious joint pathology in the differential diagnosis list and utilizing a model of multidisciplinary management for advanced imaging and medical management is critical for best patient outcomes.

Summary

This case highlights the necessity for an exhaustive and complete physical exam when assessing pain in the hip, which goes far beyond simply assessing the hip, especially considering the lack of robust hip examination tests. Not only are there a multitude of intra and extraarticular hip structures that can cause hip pain, several distant anatomical regions exist which can refer pain to the same area. Hip microinstability should be on the radar for clinicians, especially in those who are at high risk with multiple hip pathologies. An awareness of hip pain differential diagnoses, an understanding of assessment, and directions to plan of management will help guide practitioners when dealing with cases presenting in a similar fashion.

References

1. Battaglia PJ, D'Angelo K, Kettner NW. Posterior, lateral, and anterior hip pain due to musculoskeletal origin: a narrative literature review of history, physical examination, and diagnostic imaging. J Chiropr Med. 2016;15(4): 281–293.
2. Beckmann JT, Safran MR, Abrams GD. Impingement and trochanteric-pelvic. Oper Tech Sports Med. 2015;23(3): 184–189.
3. Martin HD, Khoury A, Schroder R, Palmer IJ. Ischiofemoral impingement and hamstring syndrome as causes of posterior hip pain: where do we go next? Clin Sports Med. 2016;35(3): 469–486.
4. Lee S, Kim I, Lee SM, Lee J. Ischiofemoral impingement syndrome. Ann Rehabil Med. 2013;37(1): 143–146.
5. Cheatham SW. Extra-articular hip impingement: a narrative review of the literature. J Can Chiropr Assoc. 2016;60(1): 47–56.
6. Nakano N, Yip G, Khanduja V. Current concepts in the diagnosis and management of extra-articular hip impingement syndromes. Int Orthop. 2017;41(7): 1321–1328.
7. Reiman MP, Goode AP, Cook CE, Hölmich P, Thorborg K. Diagnostic accuracy of clinical tests for the diagnosis of hip femoroacetabular impingement/labral tear: a systematic review with meta-analysis. Br J Sports Med. 2015;49(12):811–811.
8. Lesher JM, Dreyfuss P, Hager N, Kaplan M, Furman M. Hip joint pain referral patterns: a descriptive study. Pain Med. 2008;9(1): 22-25.
9. Dangin A, Tardy N, Wettstein M, May O, Bonin N. Microinstability of the hip: a review. Orthop Traumatol Surg Res. 2016;102(8): S301–S309.
10. Kalisvaart MM, Safran MR. Microinstability of the hip--it does exist: etiology, diagnosis and treatment. J Hip Preserv Surg. 2015;2(2): 123–135.
11. Bolia I, Chahla J, Locks R, Briggs K, Philippon MJ. Microinstability of the hip: a previously unrecognized pathology. Muscles Ligaments Tendons J. 2016;6(3): 354–360.
12. McNeill W, Scott S. Treatment of hip microinstability and gluteal tendinopathies involves movement control and exercise. J Bodyw Mov Ther. 2016;20(3): 588–594.
13. Grahame R, Bird HA CA. The revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS). J Rheumatol. 2000;7: 1777–1779.
14. Macintyre K, Gomes B, Mackenzie S, Hons B. Conservative management of an elite ice hockey goaltender with femoroacetabular impingement (FAI): a case report. J Can Chiropr Assoc. 2015; 59(4): 398-409.
15. Shindle MK, Ranawat AS, Kelly BT. Diagnosis and management of traumatic and atraumatic hip instability in the athletic patient. Clin Sports Med. 2006;25(2): 309–326.
16. Shu B, Safran MR. Hip Instability: Anatomic and clinical considerations of traumatic and atraumatic instability. Clin Sports Med. 2011;30(2): 349–367.
17. Emary P. Femoroacetabular impingement syndrome: a narrative review for the chiropractor. J Can Chiropr Assoc. 2010;54(3): 164–176.
18. Hoppe DJ, Truntzer JN, Shapiro LM, Abrams GD, Safran MR. Diagnostic accuracy of 3 physical examination tests in the assessment of hip microinstability. Orthop J Sport Med. 2017;5(11):1–6.
19. Griffin DR, Dickenson EJ, O'Donnell J, Agricola R, Awan T, Beck M, et al. The Warwick Agreement on femoroacetabular impingement syndrome (FAI syndrome): an international consensus statement. Br J Sports Med. 2016;50(19): 1169–1176.
20. Frank JM, Harris JD, Erickson BJ, Slikker W, Bush-Joseph CA, Salata MJ, et al. Prevalence of femoroacetabular impingement imaging findings in asymptomatic volunteers: a systematic review. Arthrosc - J Arthrosc Relat Surg. 2015;31(6): 1199–1204.
21. Pierce CM, LaPrade RF, Wahoff M, O'Brien L, Philippon MJ. Ice hockey goaltender rehabilitation, including on-ice progression, after arthroscopic hip surgery for femoroacetabular impingement. J Orthop Sport Phys Ther. 2013;43(3): 129–41.

22. Levy DM, Cvetanovich GL, Kuhns BD, Greenberg MJ, Alter JM, Nho SJ. Hip arthroscopy for atypical posterior hip pain: a comparative matched-pair analysis. Am J Sports Med. 2017;45(7):1627–1632.
23. Prather H, Hunt D, Fournie A, Clohisy JC. Early intra-articular hip disease presenting with posterior pelvic and groin pain. PM&R. 2009;1(9): 809–815.
24. Han S, Alexander JW, Thomas VS, Choi J, Harris JD, Doherty DB, et al. Does capsular laxity lead to microinstability of the native hip? Am J Sports Med. 2018;46(6): 1315–1323.
25. Dumont GD. Hip instability: current concepts and treatment options. Clin Sports Med. 2016;35(3): 435–447.
26. Ayeni OR, Adamich J, Farrokhyar F, Simunovic N, Crouch S, Philippon MJ, et al. Surgical management of labral tears during femoroacetabular impingement surgery: a systematic review. Knee Surg Sports Traumatol. 2014; 22(4): 756-762.

ISSN 0008-3194 (p)/ISSN 1715-6181 (e)/2019/197–204/$2.00/©JCCA 2019

Pubic stress fracture presenting as a strain of adductor longus in a 16-year-old elite soccer player with Crohn's disease: a case report

Cameron Marshall, DC, FRCCSS(C)[1]
Robert Gringmuth, DC, FRCCSS(C), FCCPOR(C)[2,3]

Background: *Adductor strains are the most commonly reported muscle injuries in adolescent soccer players and the second most common muscle injuries in adult players. Health practitioners should be aware of possible differential diagnoses, such as a pubic stress fracture or pubic apophysitis when athletes present with chronic groin pain.*

Purpose: *To present a rare case of a unilateral pelvic stress fracture of a 16-year old elite soccer player with a history of Crohn's disease.*

Study design: *Retrospective case report (n=1).*

Methods: *Case notes of two sports-based practitioners were reviewed and compiled retrospectively.*

Results: *Following activity restriction, a period of rest, conservative care, and progressive rehabilitation, this athlete was able to achieve a pain-free state with near equal iso-kinetic strength bilaterally as measured*

Contexte : *Les entorses des adducteurs sont les blessures musculaires les plus fréquemment signalées chez les joueurs de soccer adolescents et les deuxièmes blessures musculaires les plus fréquentes chez les joueurs adultes. Les professionnels de la santé devraient connaitre les diagnostics différentiels possibles, comme une fracture de stress du pubis ou une apophysite pubienne lorsque les athlètes présentent des douleurs chroniques à l'aine.*

Objectif : *Présenter un cas rare de fracture de stress pelvien unilatéral chez un joueur de soccer d'élite de 16 ans ayant des antécédents de maladie de Crohn.*

Plan d'étude : *Cas clinique rétrospectif (n=1).*

Méthodologie : *Les notes de cas de deux praticiens du sport ont* été *examinées et compilées rétrospectivement.*

Résultats : *Après une période de restriction d'activité, une période de repos, des soins conservateurs et une rééducation progressive, cet athlète a pu atteindre un* état *indolore avec une force isocinétique bilatérale presque* égale, *mesurée par le test musculaire Cybex 6000 (Cybex International Inc., Medway,*

[1] Complete Concussion Management Inc, Oakville, ON
[2] Canadian Memorial Chiropractic College
[3] Sports Injury & Rehabilitation Centre, Inc, Oakville, ON

Corresponding author: Robert Gringmuth, 7601 Martin Grove Road, Vaughan, ON L4L 9E4
Tel: 416-420-8153
E-mail: info@sportsinjuryrehab.com

The authors have no disclaimers, competing interests, or sources of support or funding to report in the preparation of this manuscript.
The involved patient provided consent for case publication.

by Cybex 6000 (Cybex International Inc., Medway, MA, USA) muscle testing. Full activity was resumed 10 months after initial presentation and the athlete was able to return to playing professional soccer.

Summary: *This case report presents a rare diagnosis of a unilateral pubic stress fracture presenting as a strain of adductor longus. Although quite rare, differential diagnoses such as a potential underlying stress fracture should be considered when presented with chronic or recurrent groin pain.*

(JCCA. 2019;63(3):197-204)

KEY WORDS: chiropractic, stress fracture, pubic bone, groin pain, soccer injury

MA, USA). L'activité sportive complète a repris 10 mois après la présentation initiale et l'athlète a pu recommencer à jouer au soccer à un niveau professionnel.

Résumé : *Ce cas clinique présente un diagnostic rare de fracture de stress pubien unilatéral se présentant sous la forme d'une lésion du moyen adducteur. Bien qu'ils soient assez rares, les diagnostics différentiels tels qu'une fracture de stress sousjacente potentielle devraient* être *pris en considération dans le cas d'une douleur chronique ou récurrente à l'aine.*

(JCCA. 2019;63(3):197-204)

MOTS CLÉS : chiropractie, fracture de stress, os pubien, douleur à l'aine, blessure au soccer

Introduction

According to the Federation Internationale de Football Association (FIFA), soccer is the most popular sport in the world with over 265 million players worldwide. In FIFA's 2011 "Big Count" study, Canada ranked 22nd in terms of athlete participation in soccer with an estimated 2.7 million participants. The United States ranked second, behind China, in athlete participation with 24 million (fifa.com). The sport of soccer involves running, kicking, jumping, and cutting - resulting in a number of lower limb injuries.[1-3] Groin injuries account for 5% to 18% of all soccer injuries and frequently present diagnostic challenges for practitioners.[2,4-5] Strains of any of the three adductor muscles represent the most common cause for soccer-related groin injury. A 10-year prospective cohort study of elite French adolescent (age 14 to 16) soccer teams, representing 237,600 hours of exposure time, demonstrated that muscle strains were the third most common injury behind contusions and sprains (mostly of the ankle). Of these muscle strains, the groin was the most common location of injury making it a very common and likely diagnosis for an athlete presenting with groin pain.[3,6-8] Possible differential diagnoses for groin pain include osteitis pubis, inguinal disruption[9], apophysitis and in rare cases pelvic stress fractures.

Stress fractures can be divided into the following types: fatigue fracture, insufficiency fracture, and a combined type – fatigue and insufficiency fracture. Fatigue fractures are thought to develop through an accumulation of micro trauma and the inability of the bone to keep up with skeletal repair.[1] Insufficiency fractures develop when there is inadequate bony remodeling in response to normal stressors[1,6-8] and these insufficiency fractures tend to occur with Relative Energy Deficiency in Sport (RED-S) previously called the Female Athlete Triad[10], metabolic disease and osteoporosis. In sports medicine, stress fractures of the pelvis are far less common than lower extremity stress fractures, and only account for 1% to 7% of stress fractures.[3,6-8] These fractures are considered low-risk and have been reported mainly in long-distance runners and female military recruits.[3,6-8] There are many factors that contribute to the development of stress fractures. Extrinsic factors include training regimen, footwear, training surface, and type of sport.[1,6-8] Intrinsic factors include biomechanics, anatomy, hormones, and nutrition.[1] The third type of stress fracture is a combined fatigue and insufficiency fracture, and pertains to the following case.

This is a case report of a sixteen-year-old national level male soccer player presenting with a seemingly recurrent episode of adductor longus strain. The player had been diagnosed with Crohn's disease six months prior to presentation. Advanced diagnostic imaging later revealed a stress fracture of the left superior pubic ramus with associated evidence of inguinal disruption. Our motivation for

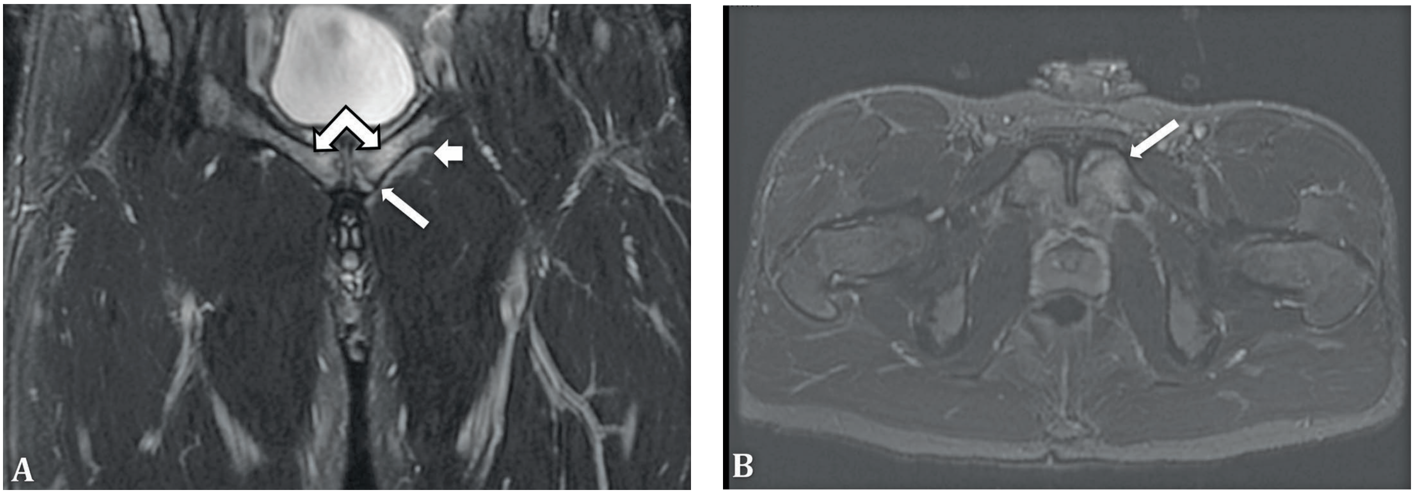

Figure 1.
Sagittal (A) and Axial (B) T2 weighted MR images displaying bilateral bone edema on both sides of the pubic symphysis (double arrow) with a small amount of fluid in the joint (Osteitis Pubis). There is a dark signal cleft indicating a non-displaced fracture of the left superior pubic ramus (thin arrow). There is also increased signal intensity within the left adductor longus tendon (thick arrow) and tendon thickening (Tendinopathy). MRI taken in December of 2011.

this study is to provide clinical awareness to healthcare providers of the rare, but possible differential diagnosis of pubic stress fractures in athletes with chronic groin pain.

Case presentation

A sixteen year-old male national level soccer player presented to a sports medicine clinic with left groin pain one week following an injury sustained during a soccer game in which the player was tackled. He was able to walk off the field without assistance following the tackle. The pain on presentation was located over the region of the left groin and radiated around to the gluteal region on the ipsilateral side. He also described paresthesia in the posterior left thigh. He had injured his left groin several times with repeated strains in the past. All resolved successfully with conservative management. The patient reported no pain with coughing, sneezing, or straining. His medical history included a diagnosis of Crohn's disease by his family physician six months earlier, for which he was taking low-dose oral methotrexate (once a week). He was in a period of remission at the time of presentation.

Physical examination revealed full range of motion of the left hip with pain only at the end ranges of abduction as well as hip extension. There was also pain with manual resisted testing of adduction as well as internal rotation of the left hip. Examination of the low back was unremarkable as was neurological evaluation of the lower limb. Palpation revealed tenderness over the pectineus and adductor longus muscles. Isokinetic muscle testing was performed using the Cybex 6000 (Cybex International Inc., Medway, MA, USA). The patient reported being right leg dominant. Testing was performed at 30 degrees/second. Five repetitions were performed and the average of these five repetitions was recorded as 110 foot pounds of force on the right for adduction and 98 foot pounds of force on the left for adduction. In comparing the force generated from the right to the left leg a 22% strength deficit on the left with adductor strength testing during the initial assessment was identified. Isokinetic testing of the hamstrings and quadriceps using the Cybex 6000 did not reveal strength deficits comparing right to left.

The patient was diagnosed with a suspected strain of the left adductor longus and conservative treatment was initiated consisting of relative rest, interferential current (IFC), cluster laser, and ice. He was also referred to a general surgeon who specializes in sports injury management two days later for a second opinion and a diagnostic ultrasound. Following his examination, the surgeon concurred

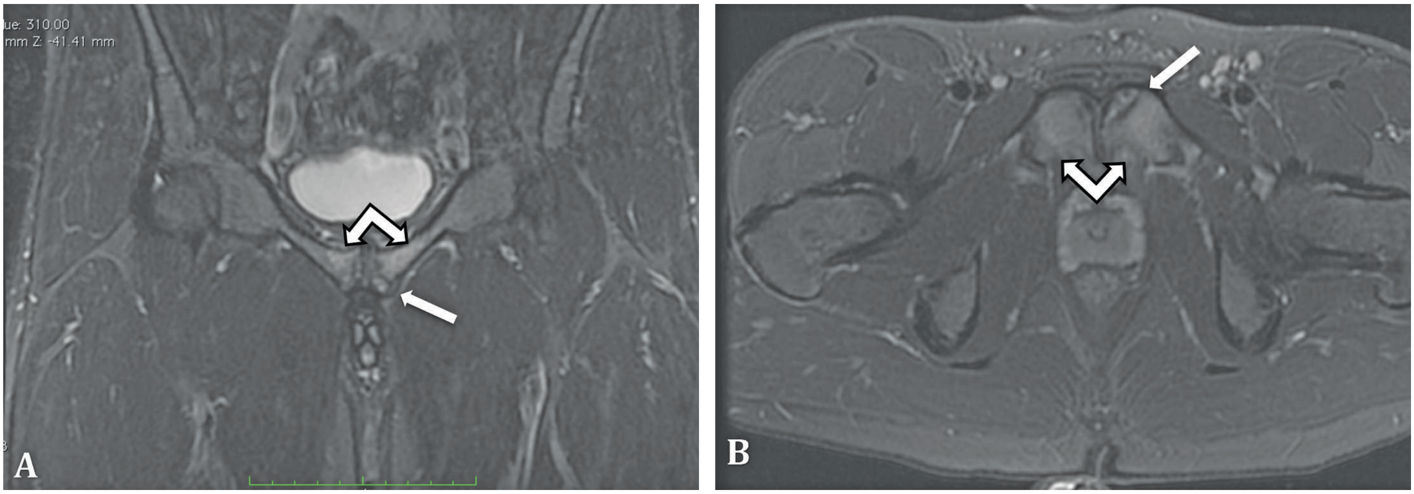

Figure 2.
Second MRI taken March 2012. Image A is the sagittal view and Image B is the axial view. There is again prominent T2 signal involving the pubic symphysis on bilaterally (double arrow). There is a dark signal cleft identified in the left aspect of the pubic symphysis (single arrow) which the radiologist considered suspicious for an apophysitis. There is no evidence of significant tendinopathy or tears within either groin.

with the suspected diagnosis and ordered a bilateral diagnostic ultrasound of the groin region. Ultrasound examination revealed elongated lymph nodes in the left groin region with no sign of muscle tear or hernia. Due to operator dependency of diagnostic ultrasound as an imaging modality, the patient was still suspected to have a minor tear of the adductor longus and was referred back to the sports specialist chiropractor for continued therapy.

Treatment continued consisting of IFC, laser, ice, as well as active rehabilitation of the pelvic musculature for approximately three weeks at which point the patient reported approximately 75% improvement in symptoms, but was still having trouble with full speed running and kicking the ball. A follow-up appointment was scheduled with the general surgeon who then ordered a follow-up diagnostic musculoskeletal ultrasound. Once again, results showed no evidence of a tear or hernia. MR imaging was subsequently ordered and scheduled for one month later. Despite continued therapy and rehabilitation, the patient plateaued at 75% improvement and saw no change over the following month.

MR imaging of the pelvic and groin regions revealed a non-displaced fracture of the left superior pubic ramus extending to the symphysis pubis. This was associated with bone edema of both pubic rami and a small amount of fluid in the symphysis pubis joint which was consistent with osteitis pubis. There was also increased signal within the left adductor longus tendon and tendon thickening in keeping with a tendinopathy. There was no definite tear identified in the adductor or abdominal musculature (Figure 1). Upon receiving the results from the radiologist, all attempts at sport activity and rehabilitative exercises were ceased. The patient was placed on limited weight bearing with crutches to ambulate and continued receiving conservative treatment for his injury.

Follow-up

Following the MRI diagnosis of stress fracture the patient was removed from all sports activity. The general surgeon opted for a non-surgical method of care and the patient continued with conservative care from the sports chiropractor and a certified kinesiologist. Conservative management consisted of rest, electromagnetic therapy (Theramend, Nortek Medical Devices) using a setting for osteoporosis and bone healing, interferential current (IFC), clustered infrared laser, and home icing. Treatment was delivered at a frequency of three times per week for twelve weeks, at which point the patient was reporting significant improvements. Follow-up MR imaging was done at this time and compared to the previous MRI.

Multiplanar multisequence MR imaging of the pelvis revealed a high T2 signal within the pubic symphysis bilaterally. There was a dark signal cleft identified in the left aspect of the pubic symphysis which was considered suspicious for an apophysitis at the pubic symphysis by the reporting radiologist. There was no evidence of tendinopathy or tear within the groin or abdominal musculature (Figure 2).

Due to incomplete healing at this time, the patient continued with conservative care under the advice of the general surgeon. Cybex strength testing was performed one month later and the patient was showing significant improvements having only minimal deficits comparing right to left in adductor strength. Using the same parameters as on the initial Isokinetic Cybex assessment the values were 138 foot pounds generated on the right and 124 foot pounds generated on the left showing a residual 10% deficit on the left. Treatment and rehabilitation continued for the next month at which point the patient had no pain and no deficits in strength and was permitted to fully return to play. The full recovery for this patient from presentation to full return to play was approximately ten months. This patient currently just finished his first full season of professional soccer in Europe with no re-aggravation of his condition and no reports of groin pain.

Discussion

The groin is a common source of pain in rotational type sports that require large amounts of force transmission through the abdominal and adductor musculature via the pubic symphysis. The symphysis pubis is an amphiarthrodial, fibrocartilaginous joint charged with the task of dissipating tensile, shear, and compressive forces during the gait cycle.[5,11] Sports in which players twist, turn, and kick, such as soccer, magnify these types of forces across the symphysis potentially leading to injury.[12] Common differential diagnoses for groin pain in the athlete are muscle strain, osteitis pubis, and inguinal disruption. Inguinal disruption is a commonly misunderstood term in the sports medicine literature and is often misdiagnosed as osteitis pubis, sports hernia, adductor strain, or abdominal wall strain. Inguinal disruption[9] is considered to be a syndrome resulting from muscular imbalances across the symphysis pubis causing exertional pubic or groin

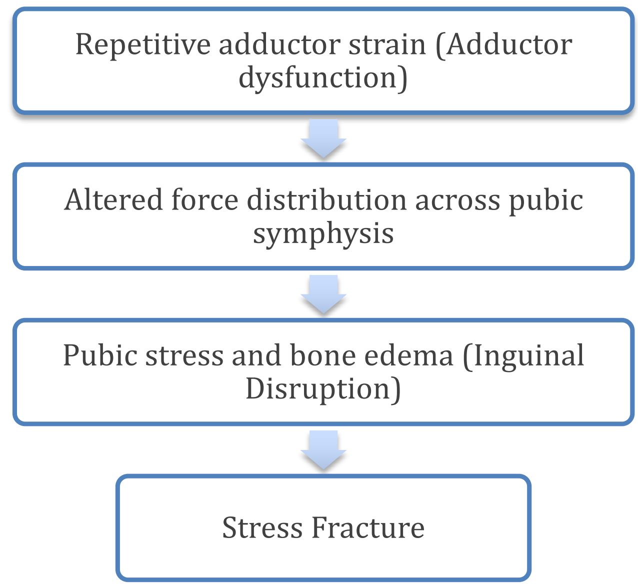

Figure 3.
Proposed progression of injury.

pain[5,13]. The sequelae of inguinal disruption[9] includes adductor dysfunction, and/or rectus abdominis dysfunction, along with osteitis pubis.[13-15] 'Sports hernia', a term often used synonymously with inguinal disruption[9], is actually a tear of the transversalis fascia of the abdomen, resulting in eventual incompetency of the posterior inguinal wall, and may, or may not, involve osteitis pubis[16]. The athlete in this particular case showed evidence of having both osteitis pubis and adductor longus tendinopathy. It is therefore assumed that this particular athlete represents a case of inguinal disruption[9] that has progressed to the point of stress fracture.

One mechanism that has been proposed for inguinal disruption[9] is repetitive stressing of the adductor and abdominal muscles during the kicking motion potentially leading to altered symphysis force distribution[4]. A magnetic resonance study examining the pelvis of professional and amateur soccer players with chronic groin pain (greater than three months) demonstrated inflammatory changes at the symphysis pubis in 97/100 subjects. In comparison, none of the 100 control subjects, soccer players and rowers, demonstrated any inflammatory changes at the symphysis (χ^2= 188.34, $p<0.001$). Inflammatory changes found in the chronic groin pain group include isolated adductor micro tears in 48.4% of subjects, isolated osteitis pubis in 9.3% of subjects, and combined adductor micro tears and osteitis pubis in 42.3% of subjects. The increased prevalence of isolated adductor micro tears as well as combined micro tears with osteitis pubis in comparison to findings of isolated osteitis pubis, led the authors to hypothesize that adductor micro tears may be the precipitating event in this syndrome. Theoretically, dysfunction of the adductor musculature leads to an altered distribution of forces across the symphysis pubis causing increased stress and subsequent bone edema through this joint.[14] To our knowledge there have not been any prospective studies done to examine this theory, but in the case of an athlete with a history of repetitive groin strains, this represents a logical progression of injury (Figure 3).

A number of similar MRI studies have demonstrated the high incidence of pathological changes surrounding the symphysis pubis in athletes with chronic groin pain. Not represented by a high incidence in these studies however, are associated fractures of the pubic bones.

Review papers examining the findings of inguinal disruption[9] through advanced imaging make note of the potential for stress fractures to occur in conjunction with these injuries, however no indication of prevalence or incidence is reported[5,17]. Stress fractures are believed to be a result of chronic stress that limits the bone's ability to remodel itself. Associated risk factors include being female, having nutritional deficits, and changes in training regiments that place greater stress on the bone.[17,23-24]

One MRI study examined 52 athletes with groin pain of greater than 3 months compared with six asymptomatic control athletes. Findings in the groin pain group consisted of osteitis pubis, adductor dysfunction, and the pre-hernia complex, which consisted of sports hernia, conjoint tendon tear, external oblique tear, and rectus abdominis sheath tears. There were no fractures reported in this study.[18] Another study of 141 patients diagnosed with either inguinal disruption or sports hernia demonstrated numerous findings of bone edema, secondary cleft signs (osteitis pubis), rectus abdominis, and adductor tendon injuries.[9] Unlike the previous study, this particular study had one incident of pubic ramus fracture; however no demographic information was given for this patient.[13] Further examination of the literature revealed only three case reports and one case-series of non-traumatic pubic fractures in an athletic population indicating an apparent rarity of these injuries. In total, these four papers discussed six pubic fractures; four were avulsion injuries of gracilis[19-20], one was an avulsion injury of adductor longus[21], and one was a combined pubic bone stress fracture with an associated adductor longus avulsion.[22] Furthermore, a prospective longitudinal cohort study of 54 elite male soccer teams (2379 players) followed over eight consecutive seasons (1,180,000 hours of exposure) found that stress fractures accounted for a mere 0.5% of all injuries, with only 6% of these injuries located in the pelvis, thus indicating an extreme rarity of pelvic stress fractures in elite soccer players.[23]

While the patient in this case was male with no recent change in his training, he was diagnosed with Crohn's disease six months prior to presentation which may have led to undiagnosed nutritional deficiencies. Crohn's disease is a relapsing inflammatory bowel disease (IBD) that can affect anywhere along the digestive tract, although most commonly affecting the terminal ileum (45%) and colon (32%). This disease is typically thought to occur in adulthood however over the past few decades there has been a general increase in pediatric onset inflammatory bowel disease (IBD) with approximately 20-30% being diagnosed before 18 years of age.[25] While there is no specific known cause of Crohn's disease it has been attributed to a combination of genetic, immunobiologic, and environmental factors.[26-27] The presentation of Crohn's typically consists of gastrointestinal (GI) symptoms such as abdominal pain, constipation or diarrhea, with the passage of blood, mucus, or both. Depending upon location of the disease process, Crohn's disease can also cause malabsorption of various nutrients related to bone and general health leading to numerous extra intestinal disorders such as anemia, arthropathy, and osteoporosis.[26]

Numerous population based studies have shown that patients diagnosed with IBD have a 21% to 40% increased risk of fracture.[28-33] A number of factors have been suggested to increase the fracture risk in Crohn's patients including nutritional deficiencies, malabsorption, systemic inflammation, increased disease severity, and corticosteroid use; however, the evidence is lacking on a single causative factor.[28-33] The patient in the current case was diagnosed with Crohn's disease a mere six months prior to presentation and had never been prescribed corticosteroids, however, there is evidence to suggest decreased bone density in newly diagnosed patients. A recent study of 60 adolescents (mean age 12.2±2.1 years) with newly diagnosed IBD examined bone mineral density as well as serum 25-hydroxy vitamin D at the time of diagnosis and compared them to fifty-six age and sex-matched children without IBD. Serum level of 25-hydroxy vitamin D was significantly lower in the IBD group compared to controls (p=0.04) and age-matched bone density Z-scores had a mean of -1.4±0.9 in the IBD group indicating osteopenia in these newly diagnosed adolescents.[16]

Childhood and adolescence are critical periods for bone mineralization and growth. Malabsorption due to IBD may hinder proper bone formation thereby increasing the risk of fracture in this population.[31] While it cannot be definitively known for certain whether or not IBD has contributed to the fracture in this case, it is likely to have played a role. Another relevant differential diagnosis in this case is pubic apophysitis. A retrospective case series compared an age matched symptomatic adductor related groin pain group to asymptomatic controls and found that all symptomatic subjects demonstrated stress-related physical changes of the pubic apophysis versus their counterparts.[34] The changes to the symptomatic apophysis included widening, asymmetry and small rounded cyst-like expansions according to these authors. Although a stress fracture in this case is likely due to the added bony burden of Crohn's disease, pubic apophysitis should be considered especially since the conservative management would be very similar.[34]

Summary

Although quite rare, stress fractures of the pelvis are important considerations for the diagnosis of chronic and recurrent groin pain. Although these injuries can be quite painful and may require a lengthy recovery, they are not usually associated with significant complications. These injuries rarely require surgery and generally respond quite well to symptomatic treatment, temporary immobilization and rehabilitation.[12] The injury sustained by the athlete in the present case was likely a result of previous groin injuries leading to an altered force distribution, inguinal disruption[9], and subsequent stress fracture. This may have also been the product of overtraining combined with an underlying nutritional deficiency caused by Crohn's disease. Stress fractures of the pelvis should be considered as a potential differential diagnosis of chronic or recurring groin pain particularly with female athletes. Especially when seen following recent and sudden increases in training volume, or with athletes presenting with underlying nutritional deficiencies.

Acknowledgments

The authors wish to acknowledge Michael Liang, Rebecca Muzos, and Frank Markus for their contributions to this case.

References

1. Dauty M, Collon S. Incidence of injuries in French professional soccer players. Int J Sports Med. 2011;32(12): 965-969.
2. Ibrahim A, Murrell GA, Knapman P. Adductor strain and hip range of movement in male professional soccer players. J Orthop Surg. 2007;15(1):46-49.
3. Le Gall F, Carling C, Reilly T, Vandewalle H, Church J, Rochcongar P. Incidence of injuries in elite French youth soccer players: a 10-season study. Am J Sports Med. 2006;34(6): 928-938.
4. Charnock BL, Lewis CL, Garrett Jr WE, Queen RM. Adductor longus mechanics during the maximal effort soccer kick. Sports Biomech. 2009;8(3): 223-234.
5. Mullens FE, Zoga AC, Morrison WB, Meyers WC. Review of MRI technique and imaging findings in athletic pubalgia and the "sports hernia". Eur J Radiol. 2012;81(12): 3780-3792.
6. Pepper M, Akuthota V, McCarty EC. The pathophysiology of stress fractures. Clin Sports Med. 2006;25(1):1-6.
7. Snyder RA, Koester MC, Dunn WR. Epidemiology of stress fractures. Clin Sports Med. 2006; 25(1):37-52.
8. Hosey RG, Fernandez MM, Johnson DL. Evaluation and management of stress fractures of the pelvis and sacrum. Orthopedics. 2008;31(4): 383-385.
9. Sheen AJ, Stephenson BM, Lloyd DM, Robinson P, Fevre D, Paajanen H, De Beaux A, Kingsnorth A, Gilmore OJ, Bennett D, Maclennan I. 'Treatment of the Sportsman's groin': British Hernia Society's 2014 position statement based on the Manchester Consensus Conference. Br J Sports Med. 2013: 1079-1087.
10. Mountjoy M, Sundgot-Borgen J, Burke L, Carter S, Constantini N, Lebrun C, Meyer N, Sherman R, Steffen K, Budgett R, Ljungqvist A. The IOC consensus statement: beyond the female athlete triad—Relative Energy Deficiency in Sport (RED-S). Br J Sports Med. 2014;48(7): 491-497.
11. Becker I, Woodley SJ, Stringer MD. The adult human pubic symphysis: a systematic review. J Anat. 2010;217(5):475-87.
12. Macintyre J, Johnson C, Schroeder EL. Groin pain in athletes. Curr Sports Med Rep. 2006;5(6):293-299.
13. Zoga AC, Kavanagh EC, Omar IM, Morrison WB,

Koulouris G, Lopez H, Chaabra A, Domesek J, Meyers WC. Athletic pubalgia and the "sports hernia": MR imaging findings. Radiology. 2008;247(3):797-807.
14. Cunningham PM, Brennan D, O'Connell M, MacMahon P, O'Neill P, Eustace S. Patterns of bone and soft-tissue injury at the symphysis pubis in soccer players: observations at MRI. Am J Roentgenol. 2007;188(3):W291-296.
15. Rabe SB, Oliver GD. Athletic pubalgia: recognition, treatment, and prevention: a review of the literature. Athl Train Sports Health Care. 2010;2(1):25-30.
16. Gerhardt MB, Brown JA, Giza E. Occult groin injuries: athletic pubalgia, sports hernia, and osteitis pubis. Practical orthopedics, sports medicine and arthroscopy, ed. DA Johnson and RA Pedowitz. Baltimore, MD: Lippincott Williams & Wilkins. 2006: 531-545.
17. Omar IM, Zoga AC, Kavanagh EC, Koulouris G, Bergin D, Gopez AG, Morrison WB, Meyers WC. Athletic pubalgia and "sports hernia": optimal MR imaging technique and findings. Radiographics. 2008;28(5): 1415-1438.
18. Robinson P, Barron DA, Parsons W, Grainger AJ, Schilders EM, O'Connor PJ. Adductor-related groin pain in athletes: correlation of MR imaging with clinical findings. Skeletal Radial. 2004;33(8): 451-457.
19. Major NM, Helms CA. Pelvic stress injuries: the relationship between osteitis pubis (symphysis pubis stress injury) and sacroiliac abnormalities in athletes. Skeletal Radiol. 1997;26(12): 711-717.
20. Wiley JJ. Traumatic osteitis pubis: the gracilis syndrome. Am J Sports Med.1983;11(5): 360-363.
21. Vogt S, Ansah P, Imhoff AB. Complete osseous avulsion of the adductor longus muscle: acute repair with three fiberwire suture anchors. Arch Orthop Trauma Surg. 2007;127(8): 613-615.
22. Tehranzadeh J, Kurth LA, Elyaderani MK, Bowers KD. Combined pelvic stress fracture and avulsion of the adductor longus in a middle-distance runner: a case report. Am J Sports Med. 1982;10(2): 108-111.
23. Ekstrand J, Torstveit MK. Stress fractures in elite male football players. Scand J Med Sci Sports. 2012;22(3): 341-346.
24. Kaeding CC, James RY, Wright R, Amendola A, Spindler KP. Management and return to play of stress fractures. Clin J Sport Med. 2005;15(6):442-447.
25. Abraham BP, Mehta S, El-Serag HB. Natural history of pediatric-onset inflammatory bowel disease: a systematic review. J Clin Gastroenterol. 2012;46(7): 581-589.
26. Baumgart DC, Sandborn WJ. Crohn's disease. Lancet. 2012; 380(9853): 1590-1605.
27. Thia KT, Sandborn WJ, Harmsen WS, Zinsmeister AR, Loftus EV. Risk factors associated with progression to intestinal complications of Crohn's disease in a population-based cohort. Gastroenterology. 2010;139(4):1147-1155.
28. Bernstein CN, Blanchard JF, Leslie W, Wajda A, Yu BN. The incidence of fracture among patients with inflammatory bowel disease: a population-based cohort study. Ann Intern Med. 2000;133(10): 795-799.
29. Bernstein CN, Leslie WD. Osteoporosis and inflammatory bowel disease. Alimentary Pharmacol Ther. 2004;19(9): 941-952.
30. Dear KL, Compston JE, Hunter JO. Treatments for Crohn's disease that minimize steroid doses are associated with a reduced risk of osteoporosis. Clinical Nutr. 2001;20(6): 541-546.
31. El-Matary W, Sikora S, Spady D. Bone mineral density, vitamin D, and disease activity in children newly diagnosed with inflammatory bowel disease. Dig Dis Sci. 2011;56(3): 825-829.
32. Leslie WD, Miller N, Rogala L, Bernstein CN. Vitamin D status and bone density in recently diagnosed inflammatory bowel disease: the Manitoba IBD Cohort Study. Am J Gastroenterol. 2008;103(6):1451-1459.
33. Cooper C, Brusse LS, Leufkens H, Javaid MK, Arden NK. Inflammatory bowel disease and the risk of fracture. Gastroenterology. 2003;125(6): 1591-1597.
34. Pubic apophysitis: a previously undescribed clinical entity of groin pain in athletes. Sailly M, Whiteley R, Read JW, Giuffre B, Johnson A, Hölmich P. Br J Sports Med. 2015;49(12): 828-834.