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Chiropractic management of a geriatric patient with idiopathic neuralgic amyotrophy: a case report

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Objective: *The purpose of this paper is to describe chiropractic management of a patient with neuralgic amyotrophy (NA) and to provide discussion regarding presentation, differential diagnosis, management and prognosis of idiopathic NA.*

Case presentation: *An 85 year old Caucasian male presented to a chiropractic clinic with right periscapular and lateral rib cage pain. The patient had previously sought evaluation and treatment from multiple health care providers and underwent multiple interventions without relief.*

Intervention and outcome: *The patient was managed with a course of chiropractic care and an ongoing home exercise program was carried out. The patient reported*

Objectif : *Ce document a pour objectif de décrire la prise en charge chiropratique d'un patient atteint d'amyotrophie névralgique (AN), ainsi que de discuter de la présentation, du diagnostic différentiel, de la prise en charge et du pronostic d'AN idiopathique.*

Exposé de cas : *Un homme blanc de 85 ans se présente à une clinique de chiropratique en se plaignant de douleur périscapulaire droite et latérale à la cage thoracique. Le patient s'était* déjà fait évalué *et traité par nombre de fournisseurs de soins de santé et avait subi de nombreuses interventions, sans soulagement.*

Intervention et résultat : *Le patient a reçu des soins chiropratiques et on lui a créé un programme d'exercices à domicile. Le patient a déclaré une*

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spontaneous resolution of pain approximately 14 months post onset.

Summary: *NA is a poorly known clinical entity amongst health care providers and poses challenges in timely and proper diagnosis. Recognition of NA is important for patients to be best managed and for more optimal patient outcomes to be achieved.*

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KEY WORDS: chiropractic, neuralgic amoytraophy, Parsonage-Turner syndrome, brachial neuritis

disparition spontanée de la douleur environ quatorze mois après l'apparition des symptômes.

Résumé : *L'AN est une entité clinique mal connue des fournisseurs de soins de santé, ce qui complique le fait de parvenir à un diagnostic exact avec rapidité. Il est important de reconnaître l'AN pour assurer une prise en charge optimale des patients et obtenir des résultats optimaux.*

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MOTS CLÉS : chiropratique, amyotrophie névralgique, syndrome de Parsonage-Turner, névrite brachiale

Introduction

Neuralgic amyotrophy (NA) is a marked sudden onset disorder which may present in otherwise healthy individuals characterized by abrupt symptoms of severe neuropathic pain of the upper extremity and subsequent neuromusculoskeletal dysfunction of the shoulder girdle.[1,2] Though NA is also known as Parsonage-Turner syndrome, it may additionally be referred to as acute brachial neuritis, acute brachial plexitis, acute brachial neuropathy, Kiloh-Nevin syndrome, brachial plexus neuropathy, idiopathic brachial plexopathy, idiopathic brachial neuritis, localized neuritis of the shoulder girdle, multiple neuritis of the shoulder girdle, paralytic brachial neuritis, serum neuritis, shoulder girdle neuritis, or shoulder girdle syndrome.[3] The hallmark symptom of NA is sudden onset of intense shoulder girdle pain with no precipitating traumatic event. The initial onset of pain may last up to several weeks[3] and subsequently transition to varying presentations of local paresis, sensory deficit, progressive weakness, or atrophy of the shoulder girdle and upper extremity musculature.[2] The variety of interchangeable symptoms reported with NA which may overlap with more commonly known disorders, multiple specialists often consulted by patients suffering from NA, and the lack of recognition of NA amongst health care providers can lead to lengthy differential diagnosis, delayed diagnosis, misdiagnosis and mismanagement.[3]

This syndrome was first reported in the 1880s by Dreschfeld with many subsequent cases described over the next half century.[4] Parsonage and Turner introduced a description of clinical characteristics associated with neuralgic amyotrophy in a series of 136 cases in 1948.[4,5] Though Parsonage and Turner referred to this disorder as neuralgic amyotrophy, it has become commonly referred to as Parsonage-Turner syndrome as well. The exact etiology of NA is unknown and may occur as a sporadic condition, though it has also been shown to present as an autosomal dominant hereditary trait known as hereditary neuralgic amyotrophy (HNA).[6] Though infectious and malignant causes have been recently discredited, there is some discussion of possible autoimmune origin.[7] It is possible that NA may be a clinical entity which manifests itself as a collection of multiple underlying mechanisms, phenotypes, and prognoses, and not as a single disorder.[6] A lumbosacral variant of NA exists and is known as lumbosacral radiculoplexus neuropathy which is more commonly known to occur in patients with mild type 2 diabetes.[7] Traditionally the prevalence of NA has been thought to be 2-4 cases per 100,000; though recent reports suggest an incidence rate of 1 per 1,000 per year.[7] NA is found to be more common in males than females, and most often presents in the second, third, and seventh decade of life.[8,9] While it seems NA is mostly known in the adult population, incidence of NA presenting in children and infants have been reported.[7] Recurrences are not uncommon and may appear in either the same extremity or an entirely new region. Manifestation of symptoms and symptom patterns may even emerge differently in reoccurrences as well.[7] Diagnosis of NA can be difficult due to the varying degree of patient presentation including severity and lo-

cation of pain, paresis, and atrophy. The differential diagnosis requires clinicians to distinguish NA from peripheral neuritis, radiculopathy, shoulder pathology, complex regional pain syndrome, rotator cuff injury, acute calcific tendonitis, adhesive capsulitis, cervical spine disorders, peripheral nerve compression, tumor, acute poliomyelitis, amyotrophic lateral sclerosis, posterior interosseous nerve palsy[10,11] and costovertebral/costotransverse joint irritation. The goal of treatment largely focuses on pain management with additional attention paid to maintaining functionality of the affected limb, most commonly the shoulder and arm.

As reported above, the prevalence of NA is quite low; furthermore the number of documented cases presenting to the chiropractic clinician is even smaller. To our knowledge, there are only two preceding reports of NA published in the chiropractic literature.[9,12] Both cases involved male patients in their thirties with an idiopathic acute onset of symptoms and electrodiagnostic evidence of neurologic involvement. Rix and colleagues described a patient that was improving with a combination of reassurance, range of motion and strength exercises, though this patient was lost to follow-up.[12] Charles described a patient that had failed surgical pronator teres release and was subsequently treated with cervical manipulation, deep tissue therapy, and hand/finger exercise.[9] This latter case resulted in full functional and pain recovery after 12 treatment sessions. An improved ability for health care providers to recognize the distinct clinical manifestations of NA, formulate differential diagnosis, appreciate best management strategies, and understand prognoses associated with NA is needed. The objective of this article is to describe a patient with NA presenting to a private chiropractic practice and further discuss clinical presentation, differential diagnosis, management, and prognosis of idiopathic NA.

Case presentation

An 85-year-old Caucasian male presented to a private chiropractic clinic with a chief complaint of right periscapular and lateral rib cage pain. The area of greatest pain was localized about the right inferolateral scapular border. Excluding this condition, he appeared to be in good health, ambulatory, and maintained an active lifestyle. The patient denied family history of related musculoskeletal disease or symptoms. The patient reported an unremarkable health history including no history of smoking, cardiovascular disease and a healthy weight throughout life. Past medical history was unremarkable.

The patient's pain began with sudden onset of severe pain while lying in bed prior to waking in the early morning period. His pain gradually decreased over the next few hours to a tolerable level. The following night the pain returned to a severe level and persisted for approximately 2 days. The patient presented to the local Emergency Department and was evaluated by electrocardiogram (EKG) which was unable to establish a cardiovascular causation. The patient was discharged with instructions to follow up with his primary care provider. Upon primary care evaluation, further diagnostic studies were performed, including a second EKG and computed tomography (CT) of the chest which the patient reported revealed no abnormal findings. Subsequent referral to a cardiologist was given. Upon cardiology evaluation, the patient was determined to have vascular blockage, despite having no typical associated symptomatology, and a coronary artery bypass graft (CABG) surgery was recommended. CABG surgery was completed 4 months after initial complaint. The patient began cardiac rehabilitation with a local physical therapist following surgery. At this time the patient reported that his chief complaint of right periscapular and lateral rib cage pain remained the same. The patient was referred to interventional pain medicine and spinal magnetic resonance imaging (MRI) of unknown regions was performed. These investigations revealed findings consistent with the patient's age and no definitive causation of pain was determined. The patient underwent two thoracic nerve block procedures which yielded no substantial benefit with relief lasting only a few days. The patient was prescribed opioid medication which provided appreciable relief; however, the patient did not like taking this medication and wished to avoid taking them long term. The patient was subsequently referred for chiropractic care following a cardiac rehabilitation session in which the physical therapist noticed prominent right scapular winging.

The patient presented for initial chiropractic evaluation 8 months following initial onset. Pain was reported to fluctuate between 5-8/10 on a numerical pain rating scale. The patient reported pain constant in nature which intensified in the evening. Provocative activities included raising the right arm and applying direct pressure to the

shoulder blade region, such as sitting against the back of a chair. The patient reported noticing discomfort while seated against the back of a chair soon after initial onset of pain, and described that prominent right scapular winging was noticed by a family member as well at this time.

Physical examination revealed thoracic spine ranges of motion grossly within normal limits with pain noted at right rotational end range. Cervical ranges of motion were reduced in bilateral lateral flexion, extension, flexion, and bilateral rotation. Shoulder ranges of motion were grossly within normal limits bilaterally. Palpation revealed tension and tenderness in the cervical and thoracic paraspinal musculature, greatest at the right levator scapula and right serratus anterior regions. Passive joint motion palpation revealed restricted mobility in the lower cervical and upper thoracic spine regions. Sensory examination of the upper extremity was grossly intact to light touch. Gross motor strength testing of the upper extremity was within normal limits. Scapular movements were determined to be reduced and more difficult to achieve on the right compared to left. Visual inspection revealed winging of the right scapula measured at 2 inches on the right compared to ½ inch on the left. (Figure 1) Attempts to obtain past medical records and diagnostic studies were unsuccessful.

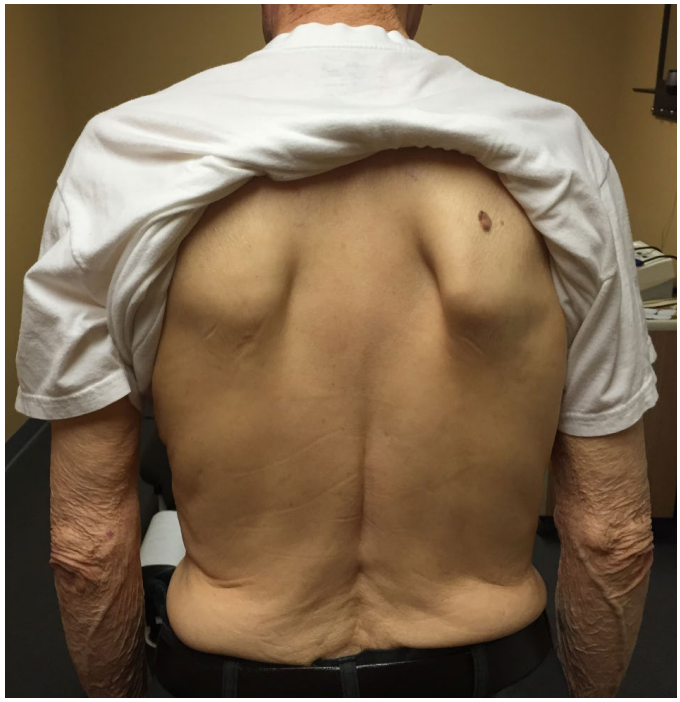

Figure 1.
Appearance of marked winging right scapula observed upon initial chiropractic examination. The patient presented for chiropractic evaluation approximately 8 months post onset of symptoms.

Clinical diagnosis

A working diagnosis was established with multifactorial consideration, including history and physical examination findings. The patient was determined to be in the chronic musculoskleletal and paretic phase of neuralgic amyotrophy[1]with residual biomechanical pain contributed via paresis of the right serratus anterior. A thorough history and physical examination provided classic criteria indicating the diagnosis of NA, such as sudden onset of severe cervicothoracic/scapular pain initiating at night[1], lack of benefit with various musculoskeletal related interventions[1], presence of winging scapula[1], lack of benefit with cardiac intervention, lack of appreciable sustained benefit from pain management interventions and a reported lack of abnormal findings of visceral origin upon diagnostic studies (in this instance, MRI, CT, and EKG).

Chiropractic management and outcome

The patient was placed on a treatment plan of 3 visits per week for duration of 4 weeks with treatment consisting of spinal manipulation administered to the thoracic spine, myofascial release therapy, and medium frequency electrical stimulation of the serratus anterior with an aim to reduce subjective pain levels. Additionally, scapular function rehabilitation exercise therapy was included in the treatment plan with an objective to reduce functional limitations. A home exercise program (HEP) was established. A lumbar chair support was dispensed and prescribed to be used to encourage proper spinal posture and reduce discomfort associated with sitting in a chair. TENS device was also dispensed to the patient for consideration as a self-care palliative relief strategy. The patient was provided with educational materials regarding the benign nature and treatment options for the management of neuralgic amyotrophy. The patient was referred to his primary care physician for co-management of the patient, particularly in relation to the patient's desire to discontinue his prescribed pain medication. The aforementioned trial of chiropractic care was carried out with which the patient

Table 1.
Comparison of hallmark features and case specific features of neuralgic amyotrophy

Hallmark features of neuralgic amyotrophy	**Case Descriptors**
Males more common than females[8]	Male
Most often presents in 2nd, 3rd, 7th decades of life[8,9]	85 years of age
Initial onset characterized by abrupt severe pain often at night[1,2]	Initial onset of severe pain in early morning hours while in bed
May present in otherwise healthy individuals[2]	Past medical history unremarkable; patient reported healthy lifestyle
Patchy paresis often in serratus anterior and/or trapezius with secondary musculoskeletal asymmetries and faulty movement patterns[1]	Prominent winging scapula and scapular dyskinesia
Sensory deficits presenting in non-dermatomal pattern atypical in location in relation to nerve distribution associated with involved muscle paresis which may last for weeks to months.[1,7]	No sensory deficits appreciated at time of chiropractic evaluation at 8 months post-onset
No gold standard laboratory, diagnostic, or specific imaging test to give a definitive diagnosis[7]	EKG, CT, MRI revealing no causation
Best treatments focus on manual and rehabilitation therapy, NSAIDs, opioids[7]	Patient failed to show appreciable relief of chief complaint with CABG surgery and two thoracic spine nerve block procedures; Patient obtained reduction in pain complaints with pain medication and manual/rehabilitation treatments

was willing and compliant. Upon completion of the initial treatment plan, a re-evaluation was performed revealing no appreciable change in objective clinical findings. Numerical pain rating scale revealed improvement in pain levels, recorded at 0-1/10 at time of re-evaluation; though the patient reported daily fluctuation of pain, which some days reached a 7/10 rating. The patient expressed subjective value in pain relief and functional ability at this time, reporting ongoing treatment and HEP compliance provided adequate pain relief comparable to relief obtained from previous opioid medication usage. The patient was recommended continued care for palliative relief aligning with the patient's values of maintaining non-prescriptive pain relief interventions. The patient elected to continue chiropractic care on an as needed basis with continuation of a daily HEP. The patient adhered to this treatment plan, presenting for an additional 18 chiropractic visits, and reported abrupt spontaneous resolution of symptoms six months after beginning chiropractic care.

Discussion

Presentation

This case supplements two other known cases reported in the literature of chiropractic management of NA. Our case is unique in that it exemplifies the elusiveness of proper and timely diagnosis of NA and illustrates the potential of multiple and possibly unnecessary interventions for this particular syndrome. The patient in this case presented with symptoms often typical to initial onset of NA such as severe acute onset of pain located in the shoulder girdle and thorax/rib area which began in the early morning hours while in bed. Patients presenting with NA report cervical, shoulder, and/or arm pain in 96% of all cases. Pain levels are high and 90% of patients grade their initial onset of pain at 7 or greater on a Numerical Rating Scale.[1] Pain most commonly is reported at night and often there is no means of palliative relief at this time, with their pain spontaneously lessening. The pain can also present

with an intermittent onset and can take up to 3 weeks to transition out of the acute onset phase.[1,7] During the acute phase, pain fails to respond well to traditional palliative treatments.[7]

Once the acute onset stage is over, paresis and subsequent local musculoskeletal dysfunction and possible atrophy most often will occur.[1] While NA patients often have several symptoms including pain, paresis, shoulder dysfunction, and atrophy, they are not all necessarily consistently present at the same juncture[1] or periods of time in any given presentation. When present, shoulder girdle dysfunction results from scapular instability due to the functional loss of the serratus anterior, rhomboid, or the trapezius muscles.[1] The majority of patients will experience additional periods of pain once the initial onset is over.[1] These subsequent pain experiences are thought to be the result of two consequences of the NA syndrome process. First, the irritated or injured nerves in the plexus can result in amplified mechanical sensitivity, producing neuropathic pain in the affected nerves' regions from increased strain on them with extension, abduction or elevation of the arm. This may last from a few weeks up to months in any given case.[1] Secondly, due to the dysfunction associated with local neuromsuculoskeletal tissue such as local shoulder girdle muscular paresis, proper biomechanics may be strained and place excessive stress on compensating musculature resulting in ongoing pain throughout the progression of NA.[1]

Differential diagnosis

A thorough physical exam should be specific and complete to help distinguish between the multiple conditions which may present with similar features as NA. One of the hallmark signs of NA presenting in the upper extremity is substantial scapular winging and scapular dyskinesia. Though serratus anterior paresis is commonly thought to be the main offended musculature resulting in musculoskeletal asymmetry/abnormalities, other muscles of the shoulder girdle may be affected resulting in presentations. For example, trapezius paresis may cause an appearance of a depressed shoulder and scapular sagging.[1] Many patients will subsequently develop musculoskeletal related pain in the region of the compensating musculature, often including periscapular region.[1]

Sensory examination of NA patients may reveal a non-specific pattern that is not consistent with localization of pain or regular dermatomal distribution related to the paresis of affected musculature[7], though could show pain with palpation of the affected peripheral nerve. However it is important to recognize that sensory deficits and hypersensitivity may dissipate in weeks to months post-onset.[1]

NA is a clinical diagnosis and does not have a gold standard laboratory, diagnostic, or specific imaging test to give a definitive diagnosis[7] though some studies suggest utilization of electroneuromyography and magnetic resonance imaging (MRI) may be helpful in confirming diagnosis.[4] Previous studies have reported appreciable change in electroneuromyography studies identified as early as three weeks post onset of symptoms.[4,13] These alterations typically entail acute denervation and suggestion of situational axonal degeneration.[4] Though these studies are sensitive for identifying denervation, assuming NA will lead to denervation might only be partially accurate as sampling error leading to a negative study is possible.[7]

Sensory nerve conduction studies have also been shown to be a poor study in assessing for the presence of NA. These studies have been reported to fail to show abnormalities in 80% of patients with NA, even when the affected nerves are examined.[7] Thus, a normal sensory nerve conduction study should not eliminate the inclusion of NA in the differential diagnosis.[7] MRI findings associated with NA include intramuscular edema and muscle atrophy, which may show associated fatty infiltration.[4,14,15] It is important, though, to emphasize the notion that diagnostic studies indicating these findings should be viewed as only one piece of the entire clinical picture. These diagnostic studies results should not be used to rule out the clinical diagnosis of NA, but can be used as additional rationale for the confirmation of the diagnosis.[16]

In this case, the patient reported prior diagnostic studies, though could only recall the use of electrocardiogram, computed tomography, and magnetic resonance studies. It is not known if an electroneuromyography study was administered. It appears that diagnostic studies utilized in this case provided ability to aid in confirmation of NA diagnosis.

In spite of the many conditions that may be considered part of the differential diagnosis, a meticulous history and physical exam should help to distinguish NA from other considerations.[7] Nonetheless, NA and its diagnosis seem

to be relatively foreign to many health care providers[7] and delayed diagnosis, misdiagnosis and mismanagement are not uncommon. In this case, the patient completed multiple treatment options for several months with minimal relief. The patient in this case identified as presenting with common features associated with NA and responded well to a subsequent course of chiropractic treatments and self-care treatments for ongoing management of symptoms.

Management and Prognosis

Failure to properly identify neuralgic amyotrophy, particularly in the acute phase, can lead to substantial mismanagement and potential for suffering for several weeks. Approaches to care may focus primarily on conservative treatment options including manual and rehabilitation therapy, as well as traditional medical treatment options including long acting non-steroidal anti-inflammatory medications, opioids, and corticosteroid injection.[7,17,18] During the initial unrelenting pain presentation there is only pharmaceutical options and corticosteroid injection that have been shown to potentially manage symptoms and hasten recovery.[1,7,17,18]

Manual and rehabilitation therapy may be indicated due to the biomechanical changes that ensue in the latter phase of NA. The objective of these treatments is to stabilize the musculoskeletal system about the area of insult. Often NA presents in the lower neck and shoulder region and thus typical therapy involves stabilizing the scapula by strengthening the various muscles around the shoulder girdle.[1] The serratus anterior muscle is frequently a site of local paresis and presents a hurdle for therapy creating an inability to produce and maintain proper shoulder mechanics and muscular endurance. Thus, a graded progression must be utilized to allow for strengthening while not fatiguing the muscles to the point that they contribute to further shoulder and scapular dysfunction. Improving stabilization about the shoulder girdle and reducing the presence of scapular winging is important in the long-term success of patient suffering from NA. Potential goals of treatments aim to restore faulty movement patterns and reduce biomechanical imbalances that may have resulted from adaptive alterations from NA.

Various other treatment options have shown some indication for continued study to further assess for their ability to aid in the management of NA patients. There is some thought of autoimmune influence in the manifestation of NA and thus some treatment efforts have been focused on associated management with immunomodulants such as prednisone, intravenous immunoglobulins and other prophylactic immunotherapies.[7] However, few studies have been conducted in this area of NA management and to date there is no conclusive evidence to recommend this mode of intervention. Surgical intervention for NA patients suffering from substantial nerve injury, consisting of hourglass constriction (shown by intraoperative visualization or nerve ultrasound), has shown some preliminary success.[7,19,20] Future studies are still needed, though, to determine if surgical intervention is a viable routine treatment option.[7]

Patients generally regain strength in affected paretic muscles, though roughly one-third of NA patients still report some form of dysfunction or symptom even six years post initial presentation.[1,7] Previous studies have shown no well-defined link between neurological considerations and functional outcomes or a specific natural course; though studies have identified a strong association between persistent pain, fatigue and the presence of scapular instability.[7,21,22]

Limitations

This is a case report which only describes the findings of this individual case. The findings of this study cannot be generalized to other patients or the general public. Resolution of symptoms in this case may not have been related to chiropractic care and may have been due to the natural progression of NA. In this case there was no availability of past medical records or diagnostic studies for review. This case relied heavily on communication with the patient and obtaining a thorough patient history, which is subject to potential recall bias.

Summary

This case features the importance of timely recognition and proper diagnosis of NA. Recognition of NA can be difficult due to the nature of NA as a clinical diagnosis which does not have a gold standard laboratory, diagnostic or specific imaging test to establish a definitive diagnosis, and the fact that it mimics so many other etiologies. Prompt recognition of common features associated with NA and differentiation from other diagnostic considerations can be appreciated with a thorough history and examination, which identifies the classic criteria for a

diagnosis of NA. Prognosis is commonly characterized by fragmented recovery from symptoms associated with NA, with a potential for an extended length of time for resolution and recurrence and continued reports of some degree of residual dysfunction are common even up to several years after initial onset.

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