Exercise related transient abdominal pain: a case report and review of the literature

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Exercise-related transient abdominal pain (ETAP) is more commonly known to athletes as a runner’s stitch. Many athletes also report shoulder tip pain (STP) associated with the ETAP. Although widely known, ETAP remains under analyzed and under reported in the medical literature. Often thought of as benign and self-limiting, ETAP has been shown to be very detrimental to the performance of many athletes from novice to elite. This case report of an elite triathlete with ETAP and subsequent review of literature, outlines the various theories about the etiology of ETAP, the epidemiology associated with it, some differentials to consider, and how chiropractic care may benefit those suffering from ETAP.

(JCCA 2009; 53(4):251–260)

**Key Words:** exercise, abdominal pain, stitch, cramp, runner’s stitch, runner’s abdominal pain, subcostal pain

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**Introduction**

Exercise-related transient abdominal pain (ETAP) is more commonly known to athletes as a stitch, stitch in the side, side cramp, side ache and subcostal pain.\(^1,2\) Many athletes also report shoulder tip pain (STP) associated with the ETAP.\(^1\) Although widely known, ETAP remains under analyzed and under reported in the medical literature.\(^2\)

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**Case report**

**History**

A 20 year old triathlete presented to the clinic complaining of a 1.5 to 2 year history of right sided abdominal pain that only occurred with training. He has been a triathlete for 4 years and has always been aware of some cramping but never this severe. The onset was unclear

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but the athlete felt it might have been due to a new bike that wasn’t set up for him properly and seemed to force him into a more kyphotic posture in the thoracic spine. The cramping was aggravated by his heart rate getting over 170 bpm which occurred frequently during “intensity training,” the impact of running, oblique exercises during gym workouts and performing a side bridge exercise. He stated that the pain would start during the swim portion of the triathlon and worsened with the bike and run. The pain was always localized to the upper right quadrant of his abdomen in the area immediately inferior to the ribs. He related that the pain would progress to the right diaphragm on occasion but did not mention any related shoulder tip pain. During an episode, the pain would start as a “tightening” and progress to a “sharp pain.” He also reported trouble breathing when the pain would get intense. He could push himself through the pain without the pain getting any worse but, on two occasions, was unable to complete races; one during the run phase in 2003 and one during the swim phase in 2004. The intensity of the pain would often reach a 9 on a 10 point scale (10 being the worst pain) with a varied time in onset during training.

Other associated symptoms included tight “hips” and cramping in his right posterior ribs with running. He has had an x-ray and an ultrasound of his abdomen which were read as normal. His previous treatment for this injury included acupuncture, ART to his psoas and iliacus, and a core stability program with little success.

The patient was a student and he did not report having the abdominal pain with any of his normal activities of daily living.

Physical examination

A physical exam revealed bilateral hip tightness particularly of the hip flexors, extensors and abductors. Muscle testing revealed inhibition in the psoas bilaterally (right > left), gluteus medius bilaterally (right > left), and the right external rotators of the hip. (Added note by author: in this case inhibition was used to describe non-painful, non-neurologic weakness of these muscles.) Motion palpation revealed restrictions in the thoracic spine and thoracolumbar junction. In particular, the T9 facet on the right and its corresponding costovertebral joint recreated a milder version of the pain of chief complaint in the abdomen. Tender points revealed by digital palpation were found in the psoas, rectus femoris, gluteus medius, spinal erectors, latissimus dorsi and iliacus bilaterally, and the right TFL, QL, internal oblique, and transverse abdominus. None of these tender points recreated the pain of the chief complaint.

Diagnosis and treatment

The patient was subsequently diagnosed with exercise-related transient abdominal pain (ETAP) with associated lower kinetic chain myofascial dysfunction as well as thoracic and thoracolumbar joint dysfunction. He was subsequently treated with ART®, spinal manipulative therapy and exercise to affected areas for four treatments over the course of a month. Exercise therapy included psoas, rectus femoris and QL stretches, core stability exercises on the stability ball, gait and balance training on a balance beam and running drills stressing proper gait mechanics (eg. walking A’s).

The patient was asked to keep a log of activity, training intensity and ETAP intensity over the course of the month. His training intensity remained high while training 5–6 days per week. His ETAP intensity varied from a low of 2/10 to a high of 8/10 (on two training days) and was consistently below 5/10. The patient was extremely happy with the decrease in pain with treatment (9/10 to 5/10 on average with a low of 2/10) but was unable to continue treatment due to extensive travel in order to comply with the plan of management.

Discussion

Epidemiology

It has been reported that “GI symptoms such as nausea, vomiting, belching, bloating, heartburn, chest pain, GI cramps, side ache, and diarrhea are experienced by 20–50% of athletes.”3 They are receiving more and more attention by athletes, coaches and medical practitioners due to the detrimental affect these symptoms can have on performance.3 In their study, Peters et al3 found that runners experienced more lower GI symptoms than upper (71% versus 36%) while cyclists had both upper (67%) and lower (64%). This was confirmed by the triathletes in their study who experienced more lower (79%) than upper (54%) while running and both upper (52%) and lower (45%) while cycling.3 Side ache was considered to be a part of the lower GI symptoms.
Morton and Callister, in a study of 965 athletes in 6 sports found that 61% of athletes had experienced ETAP over the period of 1 year. Broken down by sport, swimmers reported having had ETAP the most (75%), followed by runners (69%), horseback riding (62%), aerobics (52%), basketball (47%) and cycling (32%). In another study by Morton, Richards and Callister, it was found that during a 14 km community walk/run 31% of the participants had experienced ETAP and 42% of those said it negatively affected their performance. ETAP does not seem to affect the athlete during every exercise session. Morton and Callister found that 52% of the athletes reported it occurred less than 10% of the time while 82% said it occurred in no more than 20% of their exercise sessions.

Although often thought of as mild and benign, in those athletes that experienced ETAP, 84% of them reported that ETAP caused them to reduce their intensity level (72%) or stop the activity altogether (12%). These results indicate that over half of the athletes polled felt that ETAP had a deleterious effect on their performance. Our athlete described several incidents where he was forced to slow down as a result of ETAP and in a couple of instances had to stop completely.

Shoulder tip pain (STP) has been found to be a component of ETAP for some athletes but not all. Morton and Callister defined the STP as non-injury related that localized to the lateral 1/3 of the trapezius border extending to the acromion consistent with referred pain from the diaphragm. They found that 34% of the athletes had non-injury related shoulder pain but only...
47% of those localized it to the aforementioned part of the trapezius. This constituted 14% of the total respondents. Again broken down by sport, runners experienced the most STP at 17%, followed by swimming (15%), aerobics (11%), basketball (10%) and cycling (4%) (horseback riding was not included). In our case, the athlete did not report any STP associated with his ETAP.

ETAP and STP are also not mutually exclusive. Most often those with STP had ETAP (18%) but there were some athletes that only reported STP (8%).

Location and character
It is a commonly held clinical belief that ETAP occurs mostly on the right side and is a benign cramping sensation relieved after a few seconds of rest but this is not entirely true for every athlete with ETAP.

ETAP is well localized in 79% of the cases and in the same spot in 62%. The most common area for ETAP is the right middle third of the abdomen (just adjacent to the umbilicus) in 58% of athletes followed by the left middle third (43%) and umbilical area (21%)(percentages were >100% due to people experiencing ETAP in more than 1 area). ETAP, however was experienced to a lesser degree in all areas of the abdomen. Our athlete consistently reported the location of his ETAP to be in the right upper quadrant of the abdomen.

Using Melzack and Torgersons 10 pain descriptors, Morton and Callister found that the pain was described most often as sharp (35%), cramping (27%), stabbing (15%), aching (9%) and pulling (6%). In terms of severity, they found that ETAP was most severe at 4.7 +/- 0.1 on a 10 point numerical rating scale with stabbing being the most severe followed by sharp, aching, cramping, and pulling.

Those athletes who reported their ETAP to be the most severe experienced ETAP more frequently, had more residual soreness and felt that the pain took longer to resolve.

Factors affecting ETAP and STP
Very few studies have looked at the factors that affect ETAP and STP. It has been thought that ETAP is more pronounced in younger athletes and those athletes that are less fit. In an attempt to elucidate these and other factors affecting ETAP, Morton and Callister in their survey of 965 athletes, asked for subjective data on height, weight, age, and training volume and frequency. Their findings are summarized below.

- **Age:** Consistent with clinical observation, the prevalence, severity and frequency of ETAP and STP were both significantly decreased with increasing age. Although the severity of the pain on the 10 point numerical scale was found to significantly decrease with age, the descriptors used for the pain sensation were not significantly affected by age. The reason for the changes seen due to age were not easily explained using the current theories of the cause of ETAP and STP.
- **BMI:** The prevalence and frequency of ETAP and STP were not affected by BMI although those with a higher BMI had more localized ETAP and reported more severe STP but not ETAP.
- **Gender:** Few gender differences with respect to ETAP and STP were found. Men were more likely to report ETAP as “aching” compared to women.
- **Training:** Training more frequently significantly decreased the frequency of ETAP in those surveyed but had little effect on the severity and prevalence. These two measures also seemed unrelated to the number of years in the sport or training volume. Morton and Callister concluded that, contrary to the popular belief that novice athletes who were less fit would experience more severe ETAP and STP, athletes of all levels were just as susceptible.

Provoking factors
Morton and Callister have also surveyed athletes with respect to those factors that may provoke ETAP. Because of its transient nature, many athletes, in an attempt to explain their symptoms, link certain “things or conditions” with their ETAP. The following is a list of the subjective factors that the athletes reported as provoking their ETAP:

- eating (52%) and drinking (38%) – these could be broken down further into specific types of food and drink – high sugar content (37%), water (30%), fatty foods (16%), fruit and fruit juices (15%), and dairy products (10%);
- poor fitness level;
- high exercise intensity; competition;
- lack of warm-up;
- exercising in cold conditions.
Peters attempted to determine if the osmolality of supplements administered to triathletes during bouts of running and cycling increased their gastrointestinal symptoms. Each athlete completed three trials with different supplementation separated by one week. The three supplements included a hypertonic (higher osmolarity) high energy drink of 40 mg of carbohydrate in 100 ml of water, a semi-solid supplement mixture of banana, white bread, marmalade, and water and an equal volume of a placebo fluid with flavouring similar to the high energy drink. Following the ingestion of the supplement prior to each bout of exercise, a hypotonic thirstquencher (4 mg of carbohydrate (orange juice) and 100 ml of water) was administered at fifteen minute intervals during each bout. The placebo group received a thirstquencher flavoured placebo in a similar time interval. With respect to ETAP (side ache in the study) and the osmolality of the supplement, there was a significant difference found between the 1st running bout and the 2nd cycling bout for the high energy and semi-solid supplements ($P < 0.01$) as well as the placebo supplement ($P < 0.05$). There was also a significant difference for the high energy supplement and side ache ($P < 0.01$) between the 1st cycling bout and the 1st running bout. This suggests that hypertonic fluids may be slightly more provoking factor in ETAP during running compared to cycling but all three supplements did provoke ETAP.$^{2,6}$. There was no significant difference between the three supplements and the occurrence of ETAP. Peters, in his discussion, suggested that the supplements may have been “mildly hypertonic” during the exercise bouts due to the mixing of the hypotonic thirstquencher throughout each trial. This may have contributed to the insignificant findings.

Plunkett also attempted to determine if osmolality played a role in the development of ETAP. He found during progressive bouts of exercise, hypertonic fluids caused a significantly greater amount of ETAP compared to no fluids and hypotonic fluids. The fluids used in order of increasing osmolarity were no fluids, water, Exceed (a commercial energy drink), Coca-cola, and Duphalac (a solution of the sugar lactulose). This suggests that hypertonic fluids may play a role in ETAP during sustained bouts of exercise.

More research is needed to determine if the osmolality of food or fluids has an influence on ETAP and/or its intensity.

### Etiology – proposed theories

#### Diaphragmatic ischemia

One of the traditional causative explanations of ETAP is diaphragmatic ischemia due to the shunting of blood from the respiratory muscles to the gut or to the muscles involved in movement.$^2$

Origins of this theory are rooted in the referral of the diaphragm to tip of the shoulder, the subjective association of eating and drinking and increasing intensity (increased respiration) with ETAP, and the most common location of ETAP being the periumbilical/sub-diaphragmatic regions.$^{2,5,7}$

This theory is unlikely because of the pain of ETAP being as low as the iliac and hypogastric regions and the low incidence level of STP (14%) compared to ETAP (61%).$^5$ A study done by Roussos and Macklem refuted the shunting theory in part by showing that during decreased cardiac output, the respiratory muscles may actually deprive the rest of the body of blood. This makes sense from a survival standpoint – breathing is the most essential component of life. Plunkett reported on another study that showed that following the ingestion of a large meal, there was no change in diaphragmatic movement under fluoroscopy with those runners experiencing ETAP. Although the diaphragm may be involved in ETAP, this suggests that diaphragmatic ischemia was not the cause.

#### Visceral ligament stress

Another early competing theory for the cause of ETAP and STP was stress on the visceral ligaments (gastrophrenic, lienophrenic and coronary ligaments) connecting the diaphragm to the abdominal organs.$^{2,7}$ Early anecdotal evidence suggested that ETAP may be caused by the vertical jolting in such sports as camel riding, horseback riding and driving vehicles “off road.”$^7$ Morton and Callister, and Plunkett report that Sinclair was the first to propose the theory of visceral ligament stress. Visceral ligament stress could account for STP referral from the diaphragm, the varied location of ETAP in the abdomen which Sinclair felt was due to referred pain from the ligaments themselves, and the subjective increase in ETAP following meals.$^{2,7}$ Plunkett also report-
ed a case of a conscious patient who reported ETAP-like pain during abdominal surgery when there was traction on the mesentery.

Although the above observations lend credence to the visceral ligament theory, the fact that abdominal pain is usually along the midline and described as dull and not well localized is in contrast to the pain of ETAP. ETAP is usually well localized, all over the abdomen and can be sharp or stabbing in character.²

Plunkett⁷ attempted to test the first two theories of ETAP by having athletes digest fluid of varying absorbability and use various physical methods in an attempt to increase or decrease the amount of ETAP. Their results, although in a small sample size, showed little evidence for the diaphragmatic ischemia theory. More of their outcomes pointed in the direction of the visceral ligament stress theory although conflicting results still left some doubts as to this theory’s applicability.

**Muscle cramp**

A considerable number of athletes (27%) describe ETAP as a cramping sensation² and in turn feel that a muscular cramp may be the root cause. Although the varied location of ETAP and good localization may be explained by the muscle cramp theory, its ability to explain the cause of STP is not. Muscle cramping is not highly regarded in the literature as a possible theory of ETAP and even its own cause is not well understood.²

Morton and Callister⁹ recorded surface EMG in the area of pain during an episode of ETAP in 14 symptomatic individuals. There was no change in EMG activity immediately post exercise following the onset of ETAP compared to immediately after the pain had subsided. Both recordings were taken with the subject semi-reclined and with them holding their breath in order to reduce the activity of the abdominals and diaphragm respectively. They also recorded EMG during specific tests designed to activate the respiratory musculature which was detected at the site of ETAP yet was not recorded during an episode of ETAP. This suggests that respiratory muscle cramping was not responsible for the pain associated with ETAP.⁹

Morton and Callister¹⁰ compared spirometry results before and after an episode of ETAP in 14 subjects that developed symptoms during a treadmill test. They found that there were no significant changes in spirometry values suggesting that the diaphragm is not directly related to the cause of ETAP. This further reinforces that diaphragmatic ischemia and respiratory muscle cramping are not involved in the etiology of ETAP.

**Parietal peritoneum irritation**

The irritation of the parietal peritoneum is another competing theory of the etiology of ETAP.² The parietal peritoneum consists of the layers of tissue that line the abdominal wall and the pelvic cavity. The visceral peritoneum is the layer of tissue lining the abdominal organs separating them from the parietal peritoneum by a potential space known as the peritoneal cavity. This cavity is filled with a serous fluid to alleviate friction between the two layers.¹¹

In their study in 2000, Morton and Callister² felt that ETAP may be a form of exertional peritonitis secondary to friction between the parietal and visceral folds. Their argument is summarized below:

- the multi-level innervation of the viscera explaining the varied location of ETAP;
- the innervation of the subdiaphragmatic portion of the parietal peritoneum by the phrenic nerve could, potentially, cause STP;
- the highly localized and sharp pain with irritation of the abdominal part of the parietal peritoneum;
- the distension of the stomach with a large meal may increase the friction between the surfaces;
- the friction between the surfaces could be affected by the osmotic gradient in the viscera explaining the subjective changes in ETAP with hypertonic food and drink;
- the fact that the parietal peritoneum is sensitive to movement when irritated;
- the change in sensation in ETAP with increasing intensity of movement which would be consistent with an increase in the amount of friction.

These points led them to the conclusion that ETAP was linked to the irritation of the parietal peritoneum.¹,²

**Thoracic facet referral**

Morton and Aune¹² reported on a case of ETAP in an elite 25 year old middle distance runner that experienced increasingly frequent bouts of increasingly severe ETAP
following an injury to the thoracic spine. The exact pain of ETAP for this patient was recreated with palpation of the thoracic facet that corresponded to the dermatomal level of innervation to the painful area. Following this discovery, they examined 17 other runners to further investigate this theory. They found that in 47% of the athletes, their exact sensation of ETAP was reproduced with palpation of the thoracic facets from T8 to T12 and in another 35% they reported pain referral toward the site of ETAP. They also reported that an increased thoracic kyphosis is provocative of ETAP.12 This theory is consistent with the findings of ETAP with jolting activities such as horseback riding, all terrain driving/racing and camel racing. The dermatomal innervation of the abdomen by the various levels of the thoracic spine11 may also explain ETAP’s varied locations and its consistent localization. Thoracic facet referral does not explain STP however.

In our case, the athlete reported a similar sensation (just less intense) to his ETAP with palpation of his right mid-thoracic spine facet and costovertebral area. This suggests that his ETAP in part was associated with some thoracic spine dysfunction.

**Psoas major and Quadratus lumborum**

The medial arcuate ligament of the diaphragm forms a fascial arch covering the psoas major muscle and blending with its fascia.11,13 The lateral arcuate ligament of the diaphragm has a similar attachment with the quadratus lumborum muscle.11,13 These muscles play an important role in the segmental stabilization of the lumbar spine.14,15 It is conceivable that tightness/dysfunction in these muscles could result in the subdiaphragmatic pain of ETAP and STP secondary to the attachment to the diaphragm. This could explain the prevalence of ETAP in those sports that cause vertical jolting (horse back riding, camel racing) and those that require repetitive hip flexion (running, cycling, swimming, aerobics). It also explains the increase in ETAP during intense intervals with these sports because of the increase in the static and dynamic activation of the psoas and quadratus lumborum muscles and the increase in diaphragm activation secondary to increased oxygen demand. It does not, however, explain the various locations of pain in the abdomen with ETAP.

Our athlete had considerable tightness, dysfunction and inhibition in his psoas and QL bilaterally (right greater than left) which may have contributed to his ETAP.

**Differential diagnoses**

Eichner16 outlines several differential diagnoses that need to be considered when an athlete presents with symptoms of ETAP. Also, because ETAP has a relatively consistent localization and character of pain, a change in location and intensity in an athlete with a previous diagnosis of ETAP must be re-examined and other differentials must be ruled out based on the location of the pain. Although there are many causes of abdominal pain, the following outlines a few differential diagnoses in which symptoms are increased with exercise including:16

- **upper left quadrant:** Splenic enlargement may cause upper left quadrant pain and is common in mononucleosis. Splenic infarction must be considered in athletes with sickle cell trait when training or competing at altitude.
- **flank pain:** An athlete with acute, severe flank pain especially with radiation into the groin may be experiencing renal colic due to kidney stones. If the flank
pain is acute and bilateral, acute renal failure must be considered as well as rarely diagnosed exercise-induced loin pain syndrome.

- **Upper quadrant pain above the costal margin**: Especially in rowers, a stress fracture of the ribs or intercostal muscle strain must be entertained with stabbing pain above the costal margin.
- **Upper medial quadrant**: With midline pain in the upper quadrant, abdominal wall muscle strain may mimic ETAP and must be ruled out.
- **Lower quadrant**: If the pain presents in the lower quadrant pain, exercise associated intestinal ischemia could be the cause.

Eichner\textsuperscript{16} reminds us of the old adage, depending on what part of the world you are from, when you hear hoof beats you think horses but it may also be zebras. The following include several zebras easily mistaken for classic ETAP:

### Chronic constipation

Anderson\textsuperscript{16,17} reported the case of a 14 year old female runner who reported ongoing, well localized, periumbilical pain that was worsening over the period of 4–5 months prior to examination. The pain was initially described as a dull ache that progressed to a sharp pain when running (rated as 6/10) but always resolved within a few minutes of stopping. On presentation, her pain had increased in frequency and severity (now rated as 9/10) and was forcing her to stop running. Her physical exam and laboratory studies were within normal limits. An x-ray showed a moderate amount of stool located diffusely throughout her colon but was within normal limits. On consultation with a gastroenterologist, he suggested the jarring from running may be provoking a clinically silent and relatively mild constipation. The patient was prescribed psyllium and within 10 days her pain with running had resolved. On follow-up 6 months later, she had continued to maintain her fibre intake and was still pain free with running.

### Adhesion of ascending colon to anterior abdominal wall

Lauder and Moses\textsuperscript{16,18} reported the case of a 28 year old elite, male triathlete with no previous history of abdominal pain who suffered an acute attack of sharp right-sided abdominal pain during the run portion of a triathlon. After the first episode, he continued to have a similar pain during training while running and while swimming to a lesser degree. His pain continued to be severe during other races and he subsequently underwent further evaluation that revealed adhesions between the ascending colon and the anterior abdominal wall. The adhesions were surgically lysed and the athlete was able to resume training and subsequent races in a pain free state.

### Chronic cholecystitis

Dimeo et al\textsuperscript{16,19} reported on a 29 year old elite, male, long distance runner that had had a 12 year complaint of severe, stitching type, upper right quadrant abdominal pain with strenuous, intense training and racing. The pain had worsened a few months prior to presentation and in the prior few weeks had progressed to a similar upper right quadrant pain following eating fatty meals. Laparoscopic abdominal surgery was performed and revealed congenital adhesions anteriorly between the gallbladder and liver to the abdominal wall and posteriorly between the gallbladder, transverse colon and liver. The adhesions were sectioned and the gallbladder was removed revealing a chronic cholecystitis on histological examination. No post-operative complications occurred and on two year follow-up, he had raced several times with no return of symptoms.

### Median Arcuate Ligament Syndrome

Desmond and Roberts\textsuperscript{20} reported on a case of ETAP in a 21 year old elite middle distance runner. He reported recurrent upper abdominal discomfort and diarrhea with increased intensity of training. He had had symptoms since he was 15 years old. He was found to have compression of the celiac artery by the median arcuate ligament. The median arcuate ligament is a tendinous band uniting the medial portion of the two crura of the diaphragm.\textsuperscript{11} His symptoms of diarrhea and ETAP were completely relieved by surgery and he was able to return to competitive running.

### Treatment

**Thoracic mobilization/manipulation**

In their case study, Morton and Aune\textsuperscript{12} reported that mobilization of the affected thoracic facet along with mobi-
lizing exercises over a four week period reduced the pain of ETAP and allowed the patient to return to competitive running.

In a pilot study by Schiller\textsuperscript{21}, spinal manipulative therapy of the thoracic spine was found to be more effective than placebo treatment for mechanical thoracic spine pain. This suggests that SMT may be effective in the treatment of ETAP if thoracic spine symptoms are present. DeFranca and Levine\textsuperscript{22} also found some success with manipulation of the thoracic spine for T4 syndrome.

Our patient was manipulated in the thoracic spine as part of his treatment regimen which may have contributed to his reduction in symptoms.

**Soft tissue therapy and stretching**

Our patient was treated with ART\textsuperscript{®} of his psoas and quadratus lumborum specifically as well as other gluteal and hip musculature for a period of 1 month. Follow-up stretching of the QL and psoas were prescribed for the patient. Treatment and stretching of the patient’s psoas and QL may have contributed to his reduction of symptoms.

**Dietary modifications**

A diet log and food/beverage intake schedule during training may reveal any consistent triggers of ETAP and lead to subsequent dietary alterations in an attempt to prevent the onset of ETAP. Hypertonic supplements (food or fluid) may produce more ETAP then hypotonic supplements although more research needs to be done in this area.\textsuperscript{6,7} By monitoring intake during training, athletes with food or fluid triggers may be able to identify and avoid these triggers thus avoiding a decrease in performance due to ETAP. It is also suggested that non-symptomatic supplementation during training be maintained during race days.

Our athlete did not report any food or drink triggers.

**Other**

In their survey, Morton and Callister\textsuperscript{2} asked how those athletes that suffered from ETAP treated it. Their treatments included bending over forward (18%), stretching the affected site (22%), deep breathing (40%), and pushing on the affected area (31%). Deep breathing and bending forward were reported as the most effective by those that that suffered ETAP more severely.\textsuperscript{2} In the study by Plunkett,\textsuperscript{7} three methods of reducing stitch were employed including: contraction of the abdominals; modified breathing; and tightened abdominal belt. They found that stitch was relieved rapidly and significantly with these maneuvers and its intensity was restored within 30–60 seconds of them being stopped.

Our athlete did not report any self-treatment methods.

**Conclusions**

ETAP is a very common condition known to most athletes. Although most often thought of as minor annoyance during training and competition, Morton and Callister\textsuperscript{2} have shown that it can be severe enough to cause an athlete to stop completely. Its cause at this time is unknown but several theories have been put forward. This suggests that more time should be spent on elucidating its cause and subsequent treatment in an effort to reduce its impact on those athletes that suffer from ETAP.

**Limitations and directions for future study**

One of the limitations of this case study was the inability of the patient to continue treatment. It is feasible, although unlikely due to continued training, that symptom resolution was due to natural history or other training modifications. With continued treatment longer than 1 month, the ETAP intensity may have returned or resolved completely. An analysis of his swimming, running and cycling biomechanics may have been beneficial to elucidate training errors but was not undertaken.

Future study is needed to determine the underlying cause of ETAP, in particular whether it can be explained by one unified theory or if various subtypes of ETAP exist. Many injuries are the result of a combination of factors including training errors, poor biomechanics, muscular imbalance and poor nutrition. To find the individual cause in a multisport athlete can be like trying to find several needles in the proverbial haystack but finding them could help further direct treatment and prevention strategies.

**Acknowledgement**

The author acknowledges and thanks Dr. Larry Bell for his assistance in the diagnosis and treatment of this athlete. Without his help and guidance this case report would not have been possible.
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