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SPORT RELATED COMPRESSION SYNDROMES – REVIEW OF THE LITERATURE

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Abstract

Sport related compression syndrome can be defined as the various symptoms related to compression of nerves and vessels by surrounding tissues which appearance or exacerbation is associated with physical activity. A great variety of abnormal compressions of nerves, arteries and rarely veins may appear in athletes and other physically active individuals. Most frequent syndromes are: thoracic outlet syndrome, popliteal artery entrapment syndrome, chronic exertional compartment syndrome, acute exertional compartment syndrome, upper extremity exertional compartment syndrome. They result in exercise-induced pain, swelling of the limb, paresthesias, ischemia and intermittent claudication that can significantly affect athletic performance. Therefore the importance of a careful clinical investigation and early diagnosis should be emphasized to provide appropriate conservative or surgical treatment and prevent further complications. The purpose of this review is to present the anatomy, patophysiology, diagnosis and treatment of theese particular pathologies.

***Key words**: athletes, TOS, popliteal artery entrapment syndrome, compartment syndrome*

Introduction

Sport related compression syndrome can be defined as the various symptoms related to compression of nerves and vessels by surrounding tissues which appearance or exacerbation is associated with physical activity. An abuse injury resulting in pain and muscle stiffness can emerge in areas where the space available for tissues (e.g. muscles, nerves, and blood vessels) is limited. During exercise the tissues become swollen and pressed against another structure and becomes ischemic, deinnervated, inflamed or damaged. Compression of the nerve result in an inability to transmit impulses due to direct damage of compressed nerve fibres or indirect destruction caused by restriction in oxygen supply. Compression of the veins leads to angioneurotic disorders, characterized by edema and cyanosis, which appear as a result of reduced blood outflow from restricted area while arteries remain intact. Compression of the arteries is exciting cause of acute or chronic ischemia as a result of inadequate blood flow to the affected region or the local complications of artery including aneurysm or distal microembolisation. A great variety of abnormal compressions of nerves, arteries and seldom veins may appear in athletes and other highly physical active individuals, including the most frequent diseases such as: thoracic outlet syndrome, popliteal artery entrapment syndrome, chronic exertional compartment syndrome, acute exertional compartment syndrome, upper extremity exertional compartment syndrome. The purpose of this review is to present the anatomy, pathophysiology, diagnosis and treatment of theese particular pathologies.

Thoracic outlet syndrome

Aetiology

Thoracic outlet syndrome (TOS) consists of a group of distinct disorders produced by compression of the vessels and nerves of the upper limbs when they pass into the arms from the neck [1]. TOS is an often misdiagnosed cause of neck, shoulder and arm ailments [2, 3]. Although the term thoracic outlet syndrome and its definition (state with compression of neurovascular bundle in the thoracic outlet region) was proposed in 1956 by Peet, its proper diagnostic and therapeutic management still remains controversial [2-5]. The complexity of TOS arises from the statement that many different disorders such as cervical rib syndrome, Naffziger's syndrome - anterior scalenus muscle syndrome, costoclavicular syndrome, hyperabduction, clavicle fracture, pectoral minor muscle syndrome, head of humeral bone syndrome, exertional axillary vein thrombosis, result in TOS appearance [1].

Pathophysiology

The stage, level and type of compression or the stretching of the neurovascular bundle is responsible

for visible clinical symptoms [2, 6–8]. Patients suffering from TOS can be divided into four groups presenting only neurological, arterial, venous or mixed-aethiology symptoms. Approximately 95% of patients suffer from neurological symptoms but most of them present also vascular symptoms [1]. Neurogenic type, the most frequent type of TOS is usually connected with dysesthesia (hyperesthesia, hypoesthesia, paresthesia), vasomotoric disturbances and pain which can affect each part of the upper limb or radiates to the neck and can also imitate the stenocardial pain (pseudoangina). The remarkable muscular atrophy can be observed as a consequence of progression of this condition. The venous type is connected with limb oedema involving the shoulder girdle and hand or finger cyanosis appearing usually in association with physical effort. The arterial type is described as acute or chronic ischemia of the upper limbs or the presence of local complications including subclavian artery aneurysm or distal microembolisation [1, 9, 10].

The appearance of TOS symptoms are related to the certain positions of the body (physiological dropping of the shoulder girdle, faulty posture: anteversion of the pelvis with lumbar lordosis abolition, reduction of pectoral kyphosis) or to upper limb position and movement. Exertional elevation and abduction of the arm as well as prolonged forced upper extremity position which can occur during sleep are considered to be of great significance for the syndrome's appearance (nerve and artery compression, Paget-Schroeter syndrome) while restitution of neutral position is associated with pain relief and reduction of other symptoms. TOS occurs mainly in young athletes, especially these practicing strength disciplines (body-building, weight lifting, rowing) and sports requiring wide range of movements in the shoulder joint (gymnastics, swimming) [9, 11-14].

Diagnostic

The diagnosis of TOS consists of clinical tests, noninvasive imaging (X-rays, Duplex-Doppler ultrasounds) and invasive methods (phlebography, arteriography, electromyography) in selected cases. Clinical tests consist of provocation of the compression by abduction of the shoulder (Wright's test), abduction and external rotation (AER test) or directing the face on the opposite side and upwards (Adson's test). The positive test result manifests with dimminishing of the palpable pulse on the radial artery on examined arm due to compression of the subclavian artery. The results can be obiectivised by use of the Duplex-Doppler sonography during the examination. Theese tests are adequqte only for arterial pathology. The clinical tests should be always accompanied by diagnostic imaging apropriate for suspected pathology.

Management

Both conservative and surgical treatment can be used for the TOS. Conservative treatment consisting of rehabilitation, painkillers or physiotherapy should precede invasive surgical intervention [15].

The standard surgical treatment includes the first rib resection and excision of other bone (i.e. additional cervical rib) or fibromuscular anomalies, such as scalenectomy. The transaxillar access provides the best cosmetic results which is quite important especially for women. The complications of compression of the artery require decompressive surgery and vascular reconstruction. The necessity of vascular anastomoses determines the over and/or subclavicular access. In the case of aneurysm the vascular prosthesis or saphenous vein interposition graft can be used. Because of the risk of compression there is an indication for reinforced prosthetic grafts. There are reports of endovascular procedures of treating the aneurysms and stenoses of subclavian artery in TOS. Such procedures consist of percutaneous implantation of the covered stent (wallstent) into the aneurysm or can be preeceded by angioplasty stabilised by traditional metal stent [1-5,7-9]. The same method can be used in case of postthrombotic stenosis of the subclavian or axillar vein [22]. Open surgery decompression of the subclavian vein prevents the restenosis. Currently the venous thrombectomy is very rarely performed because of the risk of the intraoperative pulmonary artery embolisation or recurrent thrombosis. Indications for surgical treatment in neurological TOS include anomalies in nerve conduction or somatosensory evoked potentials disturbances. The surgery prevents the irreversible brachial plexus degeneration and enables fast recovery and immediate return to previous sport activity [16-24].

Popliteal artery entrapment syndrome

Aethiology

Popliteal artery entrapment syndrome (PAES) is an important although infrequent cause of serious disability and lower-extremity claudication among young adults with low cardiovascular risk and athletes associated with abnormal anatomic relationships between the popliteal artery and surrounding musculotendinous structures. Hypertrophy of the calf muscles induced by recurring sudden and forceful muscle contractions related to sport activities especially team sports, such as soccer, rugby, basketball, and martial arts constitutes the most frequent reason for PAES development [25]. Originally PAES was described by Anderson Stuart in 1879 [26]. The first operative decompression of an entrapped popliteal artery at Leyden University in the Netherlands was performed by Hamming and Vink in 1959 [27]. But the term "popliteal artery entrapment syndrome" was introduced by Love and Whelan of Walter Reed General Hospital in the United States in 1962 [28].

Pathophysiology

The knowledge of popliteal fossa anatomy is necessary to understand the possible reasons of PAES development. The popliteal fossa is a diamond-shaped depression limited by biceps femoris tendon superolaterally, semimembranosus muscle superomedially, and medial and lateral heads of the gastrocnemius muscle inferiorly. The popliteal artery normally courses between the medial and lateral heads of gastrocnemius and thus might be entrapped by adjacent muscles and tendons due to embryological variations of the myogenesis and arteriogenesis [29]. Due to the complexity of embryological development, anatomical abnormalities that can cause PAES are classified into various types associated with: atypical course of the popliteal artery from the medial or lateral head of the gastrocnemius muscle; compression of normally running artery by anomalous musculotendinous formations lying between the two gastrocnemius heads (usually compression by the aberrant head of the gastrocnemius, or compression by the excessively hypertrophied soleus, plantaris, or popliteus muscle); or a combination of a dislocated artery and muscular abnormalities [25].

The most accepted Whelan classification, modified by Rich, consists of five types of PAES such as:

Type 1 – the medial head of the gastrocnemius muscle is normal, and the popliteal artery is displaced medially around and deep to the muscle.

Type 2 – the medial head of the gastrocnemius muscle arises from an abnormal lateral position. The popliteal artery descends normally but passes medial to and deep to the muscle. Type 3 – the popliteal artery is compressed by an abnormal slip of gastrocnemius muscle

Type 4 – the popliteal artery is entrapped by a fibrous band or by the popliteus muscle.

Type 5 – any of the four preceding types that include the popliteal vein.

Recently, a "functional" PAES has been described in patients with normal anatomy (type 6) [30]. Patients with PAES are typically young (60% <30 years old), healthy males (15:1 male predilection). In young athletes with calf claudication, 60% of cases may be due to PAES. Bilateral popliteal artery involvement has been reported in 22–67% of patients presenting with PAES. Turnipseed found that patients with functional PAES are younger than those with the anatomic types (mean age 24 vs 43 years) and are more commonly female (60% vs 28% of cases) [30].

Diagnostic

Symptoms include transient tingling, coldness in the foot, leg swelling, numbness, blanching, aching pain, pain at rest, and tiredness or cramping of the calf with subsequent intermittent claudication. Recurrent chronic compression of the popliteal artery may lead to chronic vascular microtrauma of the arterial wall with intramural hematoma or thrombus, episodes of distal embolization, aneurysm, dissection, and further thrombosis with acute distal ischemia particularly characteristic for young patients with inadequate collateral circulation [31]. The diagnosis of PAES is both uncommon and difficult and therefore awareness of this entity seems to be of great significance for correct therapeutical management. The validity of careful medical history and physical examination should be emphasized. Due to the absence of typical signs and symptoms in resting state at the initial stages of entrapment syndrome, provocative tests are needed for early diagnosis. Diagnostic delay may result in irreversible arterial damage, which can impair viability of the affected limb. Duplex ultrasonography constitutes the most common noninvasive technique used for the PAES examination. Arteriography may be performed for the confirmation of diagnosis.

Management

Surgery is recommended for symptomatic PAES treatment. Posterior S-shaped incision in the popliteal fossa is considered to be the best surgical approach, which enables complete exposure of the popliteal artery and its surrounding structures. The release of the popliteal artery by division of the anomalous musculotendinous tissue is the treatment of choice in the case of popliteal artery patency. Popliteal artery occlusion both, stenotic or aneurysmal, requires additional vascular reconstruction. Thrombendarterectomy is difficult to perform in the entrapped segment of popliteal artery, because no plane of cleavage exists in the wall of the fibrous, cord-like narrow artery. When decompression of the popliteal artery by division of the anomalous muscle is insufficient, autologous vein graft interposition is also necessary. The contralateral bigger saphenous vein is the material of choice. The ipsilateral bigger and small saphenous veins are usually spared because of their importance in this area, in which the circulation already has been compromised [25].

Compartment syndromes

Compartment syndromes are defined as conditions in which increased pressure within a limited space compromises the circulation and function of the tissues within that space. They may be acute or chronic. They can affect the vessels and nerves running inside the fascia due to arising intrafascial pressure.

Chronic exertional compartment syndrome (CECS)

Aetiology

CECS is a condition in which tissue pressure within the limited space (compartment) bounded by bone or

fascia is increased during physical exercise [32]. This elevation in pressure reduces local blood flow, causing ischemia which results in pain and neuromuscular dysfunction, usually improved by rest. Chronic elevation of compartment pressure causes thickening of the compartment fascia, which further exacerbates the condition, resulting in the need for surgery in most cases for relief of symptoms [33]. Most commonly, it occurs in the lower leg, but has been reported in the thigh, foot, upper extremity, hand, forearm and erector spinae musculature (34). The incidence of CECS in the general population is unclear, but the number of CECS in men and women is similar [35, 36]. The first published report of chronic exertional compartment syndrome (CECS) was by Mavor in 1956 [37]. He reported bilateral anterior leg pain in a professional soccer player during exercise, which was successfully treated with surgery. It is thought that an even earlier account came from Dr. Edward Wilson's description of his own symptoms in 1912 [38].

Traditionally the calf has been divided into four distinct myofascial compartments: anterior, lateral, superficial posterior, and deep posterior. Additionally, it has been shown that the tibialis posterior muscle is contained within its own fascial compartment and may be considered the "fifth" compartment of the lower leg [39]. The anterior compartment contains the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius. The neurovascular structures course anterior to the interosseous membrane and include the anterior tibial artery and vein and the deep branch of the common peroneal nerve. The lateral compartment contains the peroneus longus and peroneus brevis muscles and the superficial branch of the common peroneal nerve. The superficial posterior compartment contains the soleus, plantaris, and gastrocnemius muscles and the sural nerve. In the deep posterior compartment lie the flexor digitorum longus, flexor hallicus longus, and proximally, the popliteus. The neurovascular structures include the posterior tibial artery and vein and the tibial nerve. There are no neurovascular structures within the compartment of the tibialis posterior.

Pathophysiology

In CECS, tissue pressures are elevated, but not to the same degree as in acute compartment syndrome and do not lead to irreversible ischemic changes. During strenuous exercise, there can be up to a 20% increase in muscle volume and weight due to increased blood flow and edema. In addition, the normal muscular hypertrophy that occurs over time with chronic exercise reduces the reserve volume available within the fascial compartment. Chronic fascial thickening may also contribute to compartment noncompliance. A normal fascial compartment can accommodate increased muscular volume during strenuous exercise, however, in CECS, a relatively noncompliant compartment leads to abnormally elevated tissue pressures. Research on CECS has shown that elevated tissue pressures lead to relative ischemia within the intrafascial compartment [40]. The anterior and deep posterior compartments are primarily affected. It is postulated that increased compartment pressure obstructs venous outflow and leads to a reduced arteriovenous gradient and a decrease in local blood flow. This results in ischemia of both muscle tissues and nerves leading to pain and paresthesias.

Diagnostic

Evaluation of the athlete with chronic exercised-induced lower leg pain should begin with a thorough history and physical examination. Patients with CECS will often complain of pain that starts as a dull ache, usually within the first 30 minutes after the initiation of exercise. Burning, cramping, or aching pain and tightness develop as exercise is continued. Almost all patients complain that the pain adversely affects their athletic performance. The pain is usually located over the involved compartments and may radiate to the ankle or foot or limited to the involved compartments. Usually, the pain ceases when the provocative activity is stopped. Symptoms occur bilaterally in up to 80% to 95% of patients [41, 42].

Physical examination of the resting asymptomatic patient is typically benign and may only be helpful to rule out other etiologies. Because CECS may be associated with other stress-related conditions, further diagnostic testing is often required to confirm the presumptive diagnosis of CECS. Presently, the diagnosis of CECS is usually confirmed by direct measurement of compartment pressure. Measurements of resting pressures should be taken before and after exercise, muscle contraction and during muscle relaxation. However, compartment pressure measurements may also be normal in the presence of typical symptoms of CECS. These considerations indicate the need for a reliable noninvasive test to complement clinical assessment and compartment pressure measurements to diagnose CECS. More studies are needed to define threshold values for the diagnosis of CECS. Two recent MRI studies have shown a statistically significant increase in T2-weighted signal intensity in the anterior compartment of patients with chronic anterior compartment syndrome [43, 44].

The only nonoperative treatment that is certain to alleviate the pain of CECS is the cessation of causative activities. Unfortunately, this is an unappealing option for the competitive and recreational athlete. Overall, however, nonoperative treatment for CECS has been generally unsuccessful in 100% of patients [33]. For patients who wish to continue their same exercise

regimen, fasciotomy of the involved compartments is the treatment of choice, successful in 60% to 100% of patients [45]. Postoperatively, a compressive dressing should be applied for the first two to three days. Patients should be encouraged to begin early range of motion exercises and weightbearing to help prevent scarring and adhesions. Physical therapy may aid in mobilization in the first and second postoperative week. After three to four weeks, patients may begin full activities as tolerated. Complications as high as 11% have been reported after surgical treatment [41]. These include wound infections, hematomas, arterial and nerve injury, lymphocele, and deep vein thrombosis. In addition to formal open procedures, endoscopic techniques for fasciotomies have recently been described [46, 47]. While these techniques allow for less soft tissue damage and possibly a quicker return to full activities, there are no long-term results to determine their efficacy. Recurrence rates after fasciotomy vary anywhere from 3% to 12% [48]. Recurrence of elevated compartment pressures and pain upon exertion is most likely due to inadequate release of the involved compartment, or post-surgical fibrosis formation leading to a reduction in the space available for muscular expansion during exercise.

Upper extremity exertional compartment syndrome

Chronic exertional compartment syndrome in the upper extremity is considered rare and has been reported in both, the volar and dorsal compartments of the forearm, and in the thenar, hypothenar, and interosseous musculature of the hand [34]. As in lower extremity CECS, patients will complain of pain and swelling over the involved compartments after a period of intensive muscle use and may experience distal parasthesias. It is important to methodically examine the patient to rule out other causes of these symptoms. The differential diagnosis includes vascular claudication, herniated cervical disc, compressive neuropathies, chronic tenosynovitis, and angina with referred pain. The same diagnostic criteria proposed by Pedowitz and colleagues for diagnosis of lower extremity CECS can be applied to the upper extremity [35]. Patients should be comprehensively examined to rule out more common etiologies, like ulnar stress fracture. Surgical release of the involved compartments is the mainstay of treatment for upper extremity CECS.

Acute exertional compartment syndrome (AECS)

Acute, atraumatic, exercise-induced compartment syndrome is an infrequently, but if unrecognized, can be devastating. It can present in the nonathlete who engages in strenuous, lengthy and unaccustomed physical activity; or it can present in athletes with a prior history of CECS as an acute-on-chronic type. Understanding the pathogenesis, diagnosis, and treatment of AECS is of paramount importance in preventing complications. The pathophysiology of acute exertional compartment syndrome is identical to compartment syndromes caused by trauma, burns, electrocution, and drug overdose, and is caused by an increase in intracompartmental pressure within a closed tissue space. This increased compartment pressure, when exceeding capillary perfusion pressure, leads to decreased tissue perfusion, soft tissue ischemia, and, if not treated in time, cell death. The greater the pressure and the more extensive the duration, the more severe the effects on muscle and nerve [49]. The clinical diagnosis of compartment syndrome is often described as consisting of the five "p's": pain, pallor, pulselessness, paresthesia, and paralysis. However, full-blown AECS can occur even with normal pulses and capillary refill, and paralysis and paresthesia are usually not noted until ischemia has been present for greater than one hour. Therefore, if pulselessness, paralysis, and paresthesia are present, the compartment syndrome has been longstanding and the diagnosis delayed. The most important clinical diagnostic sign of acute compartment syndrome is pain. It is a severe, unrelenting pain, out of proportion to the clinical examination, unrelieved with rest. Undiagnosed compartment syndrome carries with it disastrous consequences. Foot drop or clawing of the toes and equinovarus foot deformities significantly compromise functional use of the extremity. Further, compartment syndrome can lead to acute renal failure, which is caused by rhabdomyolysis. If suspected, compartment pressures should be rapidly obtained and involved compartments should undergo emergency fasciotomies. AECS should be included in the differential diagnosis of any athlete with leg pain. Prompt diagnosis can help avoid the serious consequences associated with the condition [50].

Summary

Compression syndromes are considered to be of great significance for neurovascular symptoms in athletes and other physically active individuals. They result in exercise-induced pain, swelling, paresthesias, ischemia and intermittent claudication that can significantly affect athletic performance. Therefore the importance of a careful clinical investigation and early diagnosis should be emphasized so as to provide appropriate conservative or surgical treatment and prevent further complications. The differential clinical diagnosis of compression syndromes in active sports participants should include muscle rupture, tendinopathies, popliteal artery adventitial cystic disease, stress fractures, medial tibial stress syndrome (MTS), fascial defects, effort-induced venous thrombosis, nerve impingement, lumbar disc herniation. Compression syndromes of the upper and lower extremity should

be comprehensively examined to rule out mentioned-above etiologies.

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