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Case report

Exercise-related bilateral leg atypical claudication in female olympic taekwondo player: A case report

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Abstract
We report the case of an Olympic taekwondo athlete with an atypical bilateral intermittent claudication that represented a handicap in her performance during competition fight. Diagnosis of chronic compartment syndrome was established by confirmation of compartment hypertension and the patient was submitted to fasciectomy. Recurrence of symptoms like numbness or tingling after 6 months raised the suspicion of deep compartmental hypertension that, once confirmed, was treated by a repeat deep fasciectomy. New symptoms appeared 4 months after surgery but no hypertension in the compartment was detected. Functional popliteal artery entrapment syndrome (FPAES) was suspected, an unusual form of this syndrome, in which a hypertrophic plantaris tendon as the cause of the entrapment was observed. Resection of the band was performed in a third operation. The patient evolved favourably, maintaining high competition level.

Key words: Entrapment syndrome, popliteal artery, compartment pressure, lower extremity claudication.

Introduction

Taekwondo is an Olympic sport in which legs are of paramount importance and the slightest movement can be decisive in the result of a competition. We report the case of an Olympic taekwondo athlete with an atypical bilateral intermittent claudication that represented a handicap in her performance during competition fight. Although tendon and muscular injuries are the most common cause of problems in such circumstances, it may be difficult to make an accurate diagnosis and choose an effective treatment. Functional popliteal artery entrapment syndrome (FPAES) should be considered when symptoms like numbness or tingling are present, with or without paresthesias.

PAES is an uncommon cause of lower extremity claudication, first described in 1879 by Stuart (Stuart, 1879), which is usually attributed to an anomalous anatomic relationship between the popliteal artery and the medial head of the gastrocnemius muscle. We present an unusual form of this syndrome caused by hypertrophy of the plantaris muscle tendon that produces the entrapment.

Case report

We report the case of an Olympic female taekwondo player who, at the age of sixteen, developed progressive pain in the gastrocnemius muscle. She presented with atypical lower extremity claudication with continuous race for 5-10 minutes and hardening of the muscle that prevented her from pursuing the race. Physical examination revealed pulses present without any significant alteration. Ultrasound study did not show any change in muscular structure. Forced plantar flexion generated pain that improved with rest. Compartment pressure was measured according to the modified criteria of Pedowitz (Pedowitz et al., 1990). Abnormal compartment pressure (22 mmHg) was observed in the left leg in the superficial posterior compartment and anterior compartment at rest 5 minutes after exercise.

In the right lower extremity, abnormal compartment pressure (26 mmHg) was observed at rest in the superficial posterior compartment, and in the anterolateral compartment (30 mmHg). These values were maintained after exercise (30 mmHg in the superficial posterior compartment, and 30 mmHg in the anterior compartment). Deep posterior compartment pressure was normal at rest, reaching 20 mmHg after exercise (Table 1).

Fasciectomy of anterolateral and posterior compartments (superficial and deep) of both legs was performed.

Table 1. Measurements of compartment pressure in lower extremity.

	First visit					
	Anterolateral Compartment		Superficial Posterior Compartment		Deep Posterior Compartment	
	At Rest	5 min After Ex	At Rest	5 min After Ex	At Rest	5 min After Ex
RIGHT	30 mmHg	30 mmHg	26 mmHg	30 mmHg	15 mmHg	20 mmHg
LEFT	14 mmHg	22 mmHg	22 mmHg	22 mmHg	14 mmHg	19 mmHg
	Six months after first fasciectomy					
RIGHT					22 mmHg	40 mmHg
LEFT					20 mmHg	24 mmHg
	Four months after second fasciectomy					
RIGHT					14 mmHg	
LEFT					15 mmHg	

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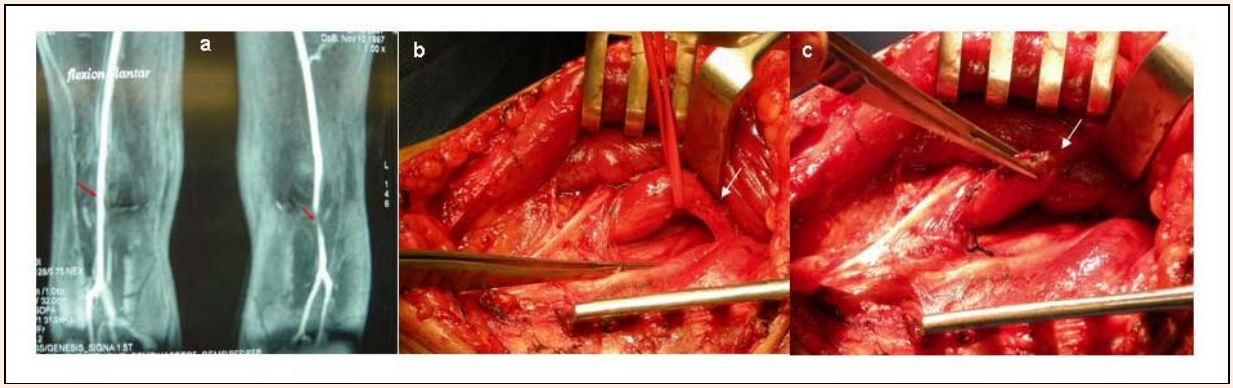

Figure 1. a) Functional magnetic resonance angiography (fMRA) with forced plantar flexion, showing bilateral arteriovenous collapse, most evident in the left lower extremity (red arrows). No anatomic abnormality of muscle or ligament insertion. b) Surgical exposure with posterior approach shows a hypertrophied plantaris muscle (with red vessel loop) compressing the vascular bundle (white arrows). c) Section of the hypertrophied plantaris muscle and neurovascular release of adhesions (white arrows).

No postoperative complications were observed but symptoms did not completely disappear as discomfort persisted with long-term exercise. Magnetic resonance imaging (MRI) study showed no significant alterations. Measurement of compartment pressures was repeated six months after surgery, showing pathological values at rest and after effort in both deep posterior compartments (Table 1). A repeat fasciectomy of deep posterior compartment was performed, extended to muscle fascia (epimysium) and tibialis posterior muscle.

The patient had a favourable evolution, returned to her full activity and won a national competition. Nevertheless, pain reappeared four months after surgery. We reviewed the compartment pressure values that appeared to be within normal limits.

Doppler ultrasonography showed a reduction of arterial flow with forced plantar flexion with collapse of the popliteal artery and vein. Ankle brachial indices decreased from 1 to 0.6 bilaterally after five minutes exercise. Functional magnetic resonance angiography (fMRA) with forced plantar flexion showed bilateral arteriovenous collapse, more evident in the lower left limb. Detailed study of MRI results did not show any anatomic abnormality of muscle or ligament insertion, and the patient was diagnosed of FPAES (Figure 1a).

Surgery of the left leg was performed via a posterior approach in the popliteal fossa according to the method of Trickey. The proximal third of the hypertrophied tendon of plantaris muscle compressing the vascular bundle was excised, leaving the muscle belly into place (Figure 1b and Figure 1c).

The patient evolved favourably with complete resolution of symptoms and was able to resume high level sporting activity.

Atypical claudication symptoms persisted in the contralateral non-operated leg (right) and, following the good results obtained with surgery in the left leg, the same procedure was used, hypertrophied plantaris muscle was identified and excised.

The patient evolved favourably, maintaining high competition level and remained asymptomatic at one year follow-up.

Discussion

Lower limb claudication is considered a disease of elderly patients with associated cardiovascular risk factors. When atypical claudication develops in young vigorous individuals and athletes, tendon or muscle injury is usually suspected. Nevertheless, when symptoms include numbness and tingling with or without paresthesia on the plantar or dorsal surface of the foot with physical exercise (Stuart, 1879; Turnipseed, 2002), PAES should be considered (Rignault et al., 1985). This syndrome is relatively more frequent in men (Elias et al., 2003). In recent years, however, the incidence rate of PAES in women has risen significantly, probably due to their growing participation in highly competitive sports (Turnipseed, 2009).

The aetiology of PAES may be functional (FPAES) or anatomic (PAES). The latter may threaten limb viability whereas patients with functional entrapment syndrome do not appear to be at risk of developing acute ischemia or thrombotic complications (Rignault et al., 1985). It may be symptomatic and cause atypical claudication when repetitive overuse produces muscular hypertrophy and a competition for anatomic space (Chernoff et al., 1995; Rignault et al., 1985; Turnipseed and Posniak, 1992).

FPAES is an uncommon disease that is frequently misdiagnosed and overlooked (Turnipseed, 2002). Dynamic tests such as Doppler ultrasonography and fMRA, with plantar flexion and dorsiflexion of the foot, showed a lateral neurovascular displacement and compression between soleal band and medial gastrocnemius and plantaris muscle. fMRA proved to be the best test for diagnosing popliteal entrapment that also allowed us to rule out other aetiologies of the compression (Love and Whelan, 1965; Turnipseed, 2004). In our patient, MRI did not detect any anatomic extrinsic compression of popliteal artery by adjacent structures, but Doppler ultrasonography and fMRA, revealed vascular collapse and interruption of vascular flow during forced plantar flexion.

Surgical treatment should only be indicated when PAES results from an anatomical mechanical cause and in

cases of symptomatic functional entrapment (Love and Whelan, 1965; Turnipseed, 2004). The method of Trickey allowed us to explore the popliteal artery in the popliteal fossa and visualise the structure that compressed it, as the complementary explorations performed did not indicate anatomical alterations.

The series published by Turnipseed (2004) showed that patients with symptomatic functional popliteal entrapment treated surgically yielded very good results, without complications and with complete resolution of symptoms in all cases (Love and Whelan, 1965; Turnipseed, 2004).

Conclusion

Functional popliteal artery entrapment syndrome is an uncommon cause of lower extremity claudication. When atypical claudication develops in young vigorous individuals and athletes, tendon or muscle injury is usually suspected.

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Key points

- We need a thorough clinical examination to reach a satisfactory clinical diagnosis.
- FPAES is an uncommon disease that is frequently misdiagnosed and overlooked.
- Dynamic tests are essential for diagnosis de FPAES.

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