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Arterial pathologies in athletes

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Abstract

Identification and treatment of arterial pathologies in athletes can present a significant challenge because symptoms may be nonspecific and standard examination is often unremarkable. Sportspeople frequently experience long delays to diagnosis, during which time their ability to exercise and their quality of life may be severely impacted. Though the conditions that affect this young patient group may not be widely seen in the general population, knowledge of their existence and recogniton of the need for specialist referral can aid early identification and appropriate management. Due to the relatively low number of reported cases, there is a paucity of robust evidence regarding optimal management of these conditions and treatment recommendations are therefore based largely upon expert consensus. It is hoped that wider use of international registries will help to address this deficit and inform future guidelines.

Key words: Arterial pathology; athlete; claudication

Introduction

The onset of exercise-induced upper or lower limb pain in young, otherwise healthy individuals can often lead to diagnostic difficulty, particularly when those affected are high-performance athletes. Since this group is inevitably

exposed to the risk of physical injury during training or competition, such symptoms are commonly attributed to musculoskeletal causes. It is often only when symptoms fail to resolve despite standard physiotherapy regimens that other potential causes are sought.

Though non-atherosclerotic arterial arterial disease is well described in athletes it remains relatively rare and management of this group of conditions can present a significant challenge.

Lower limb

Iliac Artery Endofibrosis (IAE)

IAE has most frequently been described in cyclists though it has also been reported in many other forms of athlete, including speed skaters, endurance runners, triathletes, rugby players, soccer players, cross country skiers and body builders. It typically affects individuals of less than 40yrs of age and some studies have suggested that prevalence (by haemodynamic criteria) may be as high as 10-20% amongst elite cyclsits.[1] Nearly 90% of those affected are male. Symptoms of IAE are generally unilateral and only become apparent at near-maximal exercise. For reasons that remains unclear, patients present with a unilateral left sided lesion in 80-90% of patients,[1 2] and less than 15% of patients have bilateral flow-limitation at time of presentation.[1 3 4]

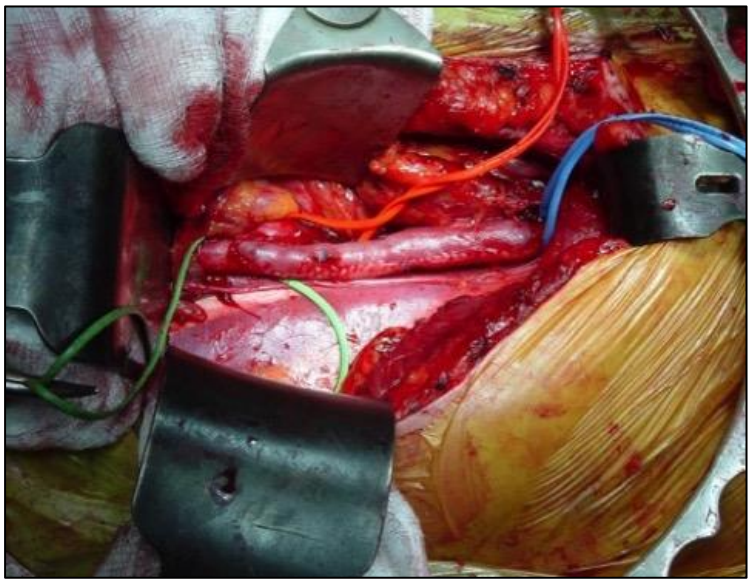
The history may often be nonspecific, though recent expert consensus has concluded that thigh pain, leg weakness and resolution of symptoms within 5 minutes of ceasing exercise are the points of history most suggestive of endofibrosis. [5] Physical examination is often unremarkable at rest and all pulses are typically present. A bruit may be audible over the iliac or femoral vessels, especially after exercise or with the hip in flexion.[6] Sensitivity and specificity of physical examination can be significantly improved by using provocative exercise tests. Ankle-brachial pressure index (ABPI) is usually normal at rest, but when ABPI is recorded immediately after ceasing maximal exercise (ideally within 1 minute) it may be possible to identify endofibrotic flow-limitation with sensitivity and specificity of up to 100%[7-10], though the absolute pressure drop required to make the diagnosis remains controversial. Nonetheless, there seems to be general agreement that a between-leg ABPI difference of over 21mmHg (in patients with unilateral symptoms) can be considered diagnostic. [5]

Many mechanisms have been implicated in the development of endofibrosis and whilst the majority of these are local mechanical factors, systemic factors may also be involved. Many of the sports in which IAE is most common involve repetitive hyperflexion of the hip joint that may results in stretching and deformation of the iliac arteries, stimulating endofibrotic change. It is also postulated that this hyperflexion could stimulate psoas hypertrophy, which may cause displacement or deviation of the iliac artery, exaggerating arterial kinking, particularly if there are additional arterial branches to the hypertrophied muscle that might be tethering the vessel abnormally. Finally, it has been suggested that

affected individuals may have elongated iliac arteries that are abnormally tortuous and increase the likelihood of vessel kinking during hip flexion.

Since the symptoms of IAE are dependent not only on anatomical abnormalities, but also on the physiological changes associated with intense exercise, identifying endofibrosis with standard imaging can be challenging. As with ABPI, Duplex ultrasound often appears normal at rest. However, some patients will have ultrasound detectable thickening of the intima-media thickness of the common femoral or external iliac arteries at rest. Ultrasound is also highly user-dependent and sonographers will often have little experience of endofibrosis. Magnetic resonance angiography (MRA) may be useful in identifying arterial elongation and kinking, though can suffer from significant movement artefact and therefore has greatest sensitivity when used in *combination* with duplex ultrasound. However, MRA is not readily available in many units in the UK and with improvements in CT resolution and 3D reconstruction, CT-angiography is likely to be the most useful means of obtaining detailed anatomical information on the affected arterial segment prior to intervention. Due to the two-dimensional nature of digital subtraction angiography (DSA), this appears to be of less use than cross-sectional imaging in assessing IAE. Finally, some groups have reported that near infrared spectroscopy (NIRS) may be useful in identifying patients with decreased thigh muscle perfusion and that reduced power in the affected limb (as measured by cycle ergometer crank-torque) may be a useful surrogate for poor iliac flow.[11 12] Larger, prospective studies are undoubtedly needed to establish the true efficacy of all of these techniques.

Pharmacological therapies do not seem to provide any benefit in the treatment of IAE and endovascular intervention is generally considered to be inappropriate due to poor reported results with angioplasty alone and the potential for fracture or displacement of any stents placed.[5] Surgical endarterectomy with patch-plasty of the affected arterial segment is therefore the mainstay of treatment for IAE, with vein as the preferred patch material. Following surgery, patients are typically advised not to return to the provocative sport for at least 6-8 weeks, after which time exercise testing can be undertaken to confirm whether treatment has been successful.



Intraoperative image showing patch-plasty of previously endofibrotic arterial segment

Popliteal Entrapment Syndrome

Popliteal entrapment syndrome (PES) describes a group of conditions in which compression of the popliteal artery, popliteal vein and tibial nerve (either singly or in combination) in the popliteal fossa by surrounding musculoskeletal

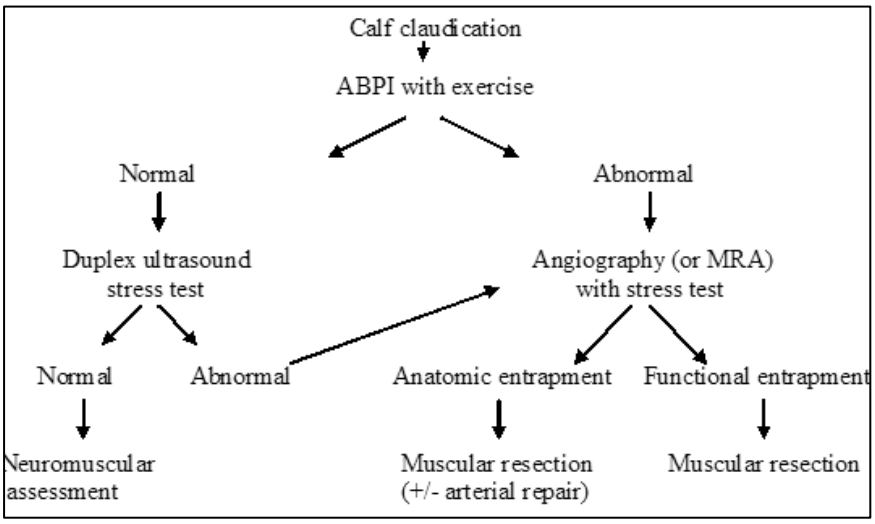
structures occurs to a degree sufficient to cause vascular or neurogenic symptoms. The predominant symptom is that of calf claudication (unilateral in >60% of cases) and popliteal artery entrapment syndrome (PAES) is believed to be responsible for a significant proportion of intermittent claudication in young patients, with patients presenting at a mean age of 32yrs. [13] It is more common amongst men than women (9:1) and is most often identified in those who undertake strenuous physical activity on a regular basis such as athletes and military personnel. The mean duration of symptoms prior to diagnosis is 24 months, though it is important that the condition is recognised and treated appropriately, as there have been reports of progressive injury to the popliteal artery with subsequent limb loss. Indeed, more than 10% of patients with PAES may present with acute limb ischaemia due to localized popliteal thrombosis.

Though a number of anatomical classification systems have been suggested for the various types of popliteal entrapment, the most unified is perhaps the consensus classification system offered by the Popliteal Vascular Entrapment Forum, which identifies a total of six different subtypes of PAES.[14] In Type I, the popliteal artery passes more medially than usual, and runs medial to the medial head of gastrocnemius (which is in the normal anatomical position) whilst the popliteal vein remains in the normal position. In Type II the medial head of gastrocnemius inserts more laterally than usual (in between popliteal artery and vein) and displaces the artery medially. In Type III, some fibres of the medial head of gastrocnemius are inserted more laterally than usual such that they envelop the artery and the artery appears to run through the body of the medial head, where it is compressed. In Type IV, the gastrocnemius muscle is generally

normal but the artery passes deep to popliteus where it may be compressed. Type V is uncommon, but refers to any of the other 4 anatomical subtypes, with the addition feature of popliteal vein entrapment. In Type 6 PAES the abnormality is functional rather than anatomical: Whilst structure and position of muscle and vessels are both normal, hypertrophy of the gastrocnemius leads to extrinsic compression of the artery.

As with iliac endofibrosis, clinical signs may be subtle and difficult to discern for those not accustomed to the condition. A popliteal bruit may be audible on auscultation of the popliteal fossa, particularly during active plantarflexion. Similarly, foot pulses may also be diminished during plantarflexion.

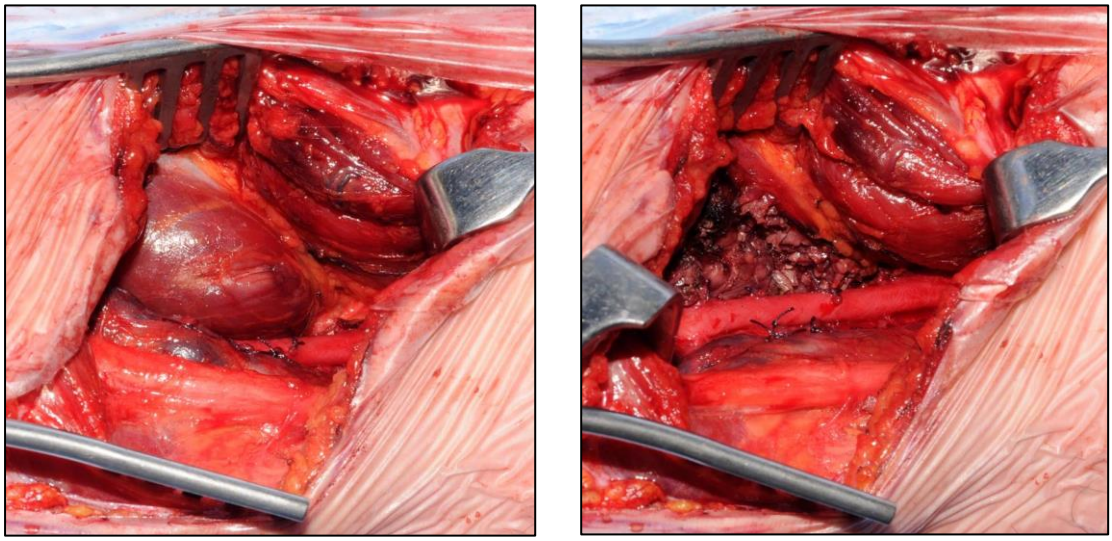
ABPI should be measured both at rest and after exercise and may be found to be lower post-exercise (although frequently there is no detectable pressure drop especially in patients without arterial damage and esp in type 6). Arterial Duplex should be performed and may be capable of identifying PAES with a sensitivity of over 80%,[13] though significant stenosis or occlusion are typically apparent during plantarflexion. Magnetic resonance imaging (MRI) can also be used and is generally preferable to CT-angiography, as it can more accurately identify abnormal anatomy/extrinsic compression as well as arterial stenosis, though there have not been any studies directly comparing the sensitivity of these modalities. DSA may also be of benefit – particularly when MRA is not readily available - as it allows more dynamic assessment of the degree of arterial stenosis and images can be captured before and during plantarflexion.



Proposed algorithm for the diagnosis and management of popliteal artery entrapment syndrome

Open surgical repair should be undertaken in all cases of proven PAES. Whilst angioplasty and stenting can usefully be used to address arterial stenosis caused by other disease processes, they are not appropriate in PAES as the primary objective of intervention is to relieve extrinsic compression. Indeed, stenting may result in stent displacement or fracture – particularly amongst athletes. Arterial release can be achieved via medial or posterior approaches to the popliteal fossa. The medial head of gastrocnemius can then be divided to release the artery. Once this has been done, a nerve stimulator can be used to elicit gastrocnemius contraction intraoperatively with duplex ultrasound positioned distally to confirm that contraction no longer causes diminished flow. Following arterial release, persistent arterial stenosis can be corrected with arterial patch-plasty or excision of the stenosed segment and interposition grafting – ideally with vein.

After intervention, patients should undergo focused rehabilitation after 48hrs of bed rest. Crutches should be used for the first 3-4 days before beginning non-impact physiotherapy for 1 week. Patients should then gradually increase activity but be restricted to low-impact running for 4-6 weeks. Although robust follow-up data are sparse, there is evidence to suggest that surgical correction may lead to resolution of symptoms in over 70% of patients. [13]



Images showing popliteal artery before and after release
(with division of medial head of gastrocnemius)

Chronic exertional compartment syndrome (CECS)

CECS is a form of transient compartment syndrome in which the fascial compartments of the affected limb are not large enough to permit the natural increase in muscle volume that occurs during exercise. This results in poor tissue perfusion and cramping pain in the affected limb. It is most common in the lower leg (especially the anterior and lateral compartments) and typically affects long-distance runners - when symptoms may mistakenly be diagnosed as 'shin

splints' - though it is also seen in other sports including skiing, football, rugby and tennis.

Whilst symptoms are only felt during exercise, what distinguishes CECS from the other forms of exertional lower limb pain described above, is that the pain is most often felt in the anterior or lateral aspect of the leg. Depending on whether anterior, lateral or deep posterior compartments are involved, examination may reveal weakness of dorsiflexion, eversion or toe flexion respectively. Paraesthesia in the relevant distribution may also be a feature. When symptoms are present, the affected compartment may also feel obviously tense on palpation.

Initial investigation should be aimed at excluding other causes of anterior leg pain and plain radiography or MRI can be used to rule out tibial stress fracture. Similarly, duplex ultrasound can be used to exclude arterial disease. The most established diagnostic test for CECS is measurement of intracompartmental pressures immediately after exercise, though there remains some debate about whether all four compartments of the leg should be assessed and whether both legs should be tested regardless of symptoms. [15] Similarly, there is no overall consensus about the absolute post-exercise increase in compartment pressure required to make the diagnosis of CECS, though a pressure >30mmHg at 1 minute after cessation of exercise is a commonly used threshold.[16] Despite the high specificity of measuring intracompartmental pressure, it remains an invasive test and other investigative modalities have therefore been sought. Near infrared spectroscopy is the most promising of these. When NIRS is used to

assess tissue oxygenation in the affected compartment, a tissue oxygen saturation of less than 50% may be able to identify CECS with a specificity of 67% and a sensitivity of 78%, though this is typically only available in highly specialist centres. MRI has also been suggested as a possible means of identifying CECS, by comparing intensity of T2-weighted images before and after exercise, but this seems to be less reliable than either NIRS or measurement of compartment pressure.[17]

Once the diagnosis has been made, conservative measures such as reducing (or temporarily stopping) the provocative exercise and using anti-inflammatory drugs can be tried. However, these measures are usually ineffective and resumption of full activity leads to recurrence of symptoms. For those with persistent symptoms, surgical treatment involves fasciotomy of the affected compartment. This may achieve symptomatic relief in over 80% of patients and appears to be most effective for those with symptoms in the anterior or lateral compartments.[18] Targeted postoperative physiotherapy is also important to ensure early mobilisation of the affected limb and prevent formation of excessive scar tissue that might cause further muscle constriction.

Cystic adventitial disease (CAD)

Though perhaps less common than the other conditions described above, CAD should always be considered in the differential diagnosis for young patients with symptoms of non-atherosclerotic arterial disease. Mucinous material accumulates in the adventitial layer of arteries, particularly the popliteal artery,

though it has also been reported in the iliac, femoral, radial, brachial and axillary arteries. The cause of this accumulation of proteoglycans is unclear, though it has been suggested that there may be four main underlying aetiologies: *traumatic*, with repetitive trauma causing separation of the adventitia and media, allowing accumulation of mucin; *degenerative*, as part of a systemic syndrome of mucinous degeneration; *developmental*, with incorporation of mucin-secreting mesencymal cells into the adventitia leading to cyst formation; and *synovial*, where there is an abnormal connection between the adventitial layer of an artery and the synovial cavity of an adjacent joint. [19]

Men are affected more often than women (ratio 5:1), symptoms are almost always unilateral, and patients present with gradually worsening claudication in the affected limb in the absence of atherosclerotic risk factors. Pulses may well be normal when the leg is examined in extension, but flexion causes obliteration of the distal pulses (Ishikawa's sign).[20]

Duplex ultrasonography is usually sufficient to identify CAD, as cystic lesions are readily visible and luminal narrowing and increased flow velocities can also be demonstrated. Nonetheless, cross-sectional imaging with MRI or CT can be useful to exclude other pathologies such as popliteal entrapment or Baker's Cyst. Though DSA is rarely necessary to assess CAD per se, if it is performed it may reveal the typical 'scimitar' appearance, with a smooth, curvilinear narrowing of the artery. Where lesions are multiple or circumferential, the alternative 'hourglass' appearance may be seen.

Though the indications for surgery are not clear cut for CAD, lesions are usually progressive and since most patients are relatively young and active, intervention is likely to be necessary to maintain their quality of life. Cyst aspiration is not generally considered effective due to the high rates of cyst reaccumulation and recurrence of symptoms. Angioplasty and stenting are also inappropriate because stenosis is the result of extrinsic compression rather than an intra-arterial lesion. Open surgery is therefore the optimal treatment option for CAD. Though simple cyst aspiration is usually unsuccessful, evacuation of the cyst with simultaneous excision of the cyst wall is far more successful and this is therefore the preferred approach if there is short isolated segment of disease.[21] When there is evidence of more extensive arterial degeneration, the affected segment should be excised with subsequent interposition grafting.

Persistent sciatic artery

Persistence of the sciatic artery is an embryological abnormality in which this artery – originally a continuation of the internal iliac artery - fails to involute as it normally would during development. In so-called 'complete' persistence of the sciatic artery, this is the only blood supply to the lower limb and the femoral arteries are absent. In 'partial' persistence, the external iliac artery and superficial femoral artery may be present but are hypoplastic. Though this anatomical anomaly is often identified as an incidental finding, patients with a persistent sciatic artery may also present with claudication during exercise and the diagnosis must therefore be considered when young patients present with these symptoms.

On examination, patients classically have absence of a femoral pulse despite having palpable popliteal and pedal pulses. Aneurysmal degeneration of the sciatic artery is also common and this may result in a palpable pulsatile mass in the buttock region. Patent sciatic arteries can usually be identified on duplex ultrasound, though it is important to differentiate any aneurysm of the sciatic artery from aneurysms of the gluteal artery (or other small vessels), for whilst smaller vessels may be safely ligated, ligation of a persistent sciatic artery can result in acute limb ischaemia. Though incidentally identified persistent sciatic arteries do not need to be treated, surgery should be undertaken when claudication is impairing quality of life or if there is evidence of aneurysmal dilatation. This typically involves ligation of the sciatic artery with simultaneous femeropopliteal bypass.

Fibromuscular dysplasia (FMD)

Fibromusclular dysplasia is a rare condition that may occasionally affect athletes and primarily affects women between 20 and 40 years of age. Though the exact aetiology of FMD remains unclear, it is a non-inflammatory, non-atherosclerotic condition in which intra-arterial fibrotic webs develop in the intima, media or adventitia of affected arteries, causing progressive stenosis. It most commonly occurs in the renal arteries with associated hypertension, though may also occur in other vascular beds, including the arteries of the lower limb, leading to claudication. Clinicians should have a high index of suspicion in any young patients with claudication and synchronous hypertension, though diagnosis is usually made with angiography that may reveal a classical 'beaded' appearance.

The natural history of FMD is poorly understood, but since patients typically only present when they become symptomatic, intervention should usually be considered. Where the lesion is non-occlusive, angioplasty may be effective, though surgical bypass should be undertaken for occlusive disease.

Upper limb

Although arterial problems most commonly affect the lower limbs of athletes, they can also occur in the upper limb. In addition to the arterial trauma that frequently results from fracture or dislocation injuries to the arm, non-traumatic conditions are also seen. The most common of these is arterial thoracic outlet syndrome.

Thoracic Outlet Syndrome (TOS)

Thoracic outlet syndrome is a condition in which arteries, veins or nerves are compressed as they exit the thoracic cavity to supply the arm. Compression can be caused by a variety of congenital anatomical abnormalities such as cervical rib, elongated transverse processes of the cervical vertebrae, aberrant soft tissues or fibrous bands. In athletes, however, muscle hypertrophy - particularly of the scalenus, subclavius and pectoralis minor muscles - may be sufficient to cause compression of neurovascular structures. It is therefore most prevalent in those who take part in sports that involve repetitive, forceful overhead movements, such as weightlifters, swimmers and baseball players.

Patients with *arterial* TOS typically present with cramping 'claudication' type pain in the affected arm during exercise. This may progressively worsen as exercise continues and be relieved by rest. In more severe cases, patients may also report that the arm becomes cool, pallid, or even cyanosed. Whilst patients often describe that the affected arm feels 'heavy', power and sensation are usually intact. On rare occasions, presentation may be with symptoms of acute limb ischaemia or even embolic stroke.

As with other forms of arterial disease in athletes, clinical examination findings may be relatively nonspecific and clinicians must therefore be mindful of the possibility of TOS when investigating young patients with exertional upper limb pain. Initial examination should seek to exclude musculoskeletal causes of pain such as subacromial impingement, adhesive capsulitis and rotator cuff injury. Neurological assessment should also be made to exclude cervical radiculopathy and neuropathy.

Brachial and radial pulses should be compared between the affected and non-affected arms and blood pressure measured in both. All of these are frequently normal in the anatomical position, though an inter-arm difference in blood pressure of more than 20mmHg may be considered significant.[22] Provocative tests can also therefore be used. The test most commonly used for detection of arterial TOS is the Wright hyperabduction test, which involves abducting and externally rotating the arm whilst palpating for loss of the radial pulse. [23] This test can be enhanced by auscultating for a bruit in the supraclavicular fossa. Adson's test can also be used, during which the patient is asked to extend their

neck and turn their head towards the affected limb. The patient's arms are then held in adduction and the radial pulse assessed during deep inspiration. Notably, none of these tests has been shown to be particularly reliable.

In the absence of sensitive or specific examination techniques, imaging is clearly important and plain radiography of the chest and cervical spine can be used to exclude any bony abnormality. Duplex ultrasonography can then be used to make a dynamic assessment of flow in the subclavian and axillary arteries. This is typically done with the arm at 0, 90, 120 and 180 degrees of abduction. Whilst this can be very useful for identifying changes in flow during provocative manoeuvres, it remains an indirect measure of more proximal arterial compression and detailed cross-sectional imaging is generally necessary to clarify the site of stenosis. DSA is generally avoided as it fails to provide any information about the cause of impingement. CT angiography can be used effectively to demonstrate the site of arterial stenosis if imaging is performed with arms in both neutral and hyperabducted positions and can clarify any bony cause of extrinsic compression. However, MRI is undoubtedly the optimum imaging modality in athletes as it allows clear identification of aberrant or hypertrophied muscles and abnormal fibrous bands.

Cessation of the provocative sport may be sufficient to alleviate symptoms in some individuals, though for many athletes this is not a practical option. Surgical correction typically involves resection of the first rib with simultaneous excision of any aberrant fibrous tissue, with arterial reconstruction (with interposition grafting or bypass) should also be undertaken if there is evidence of significant

aneurysmal degeneration of the subclavian artery at time of surgery. For athletes however - in whom there may be no bony abnormality – selective supraclavicular scalenectomy may be sufficient to relieve symptoms and associated with significantly lower morbidity. Duplex ultrasonography can then be used to confirm that there is no residual arterial compression following surgery. Athletes should then make a gradual return to activity, though there is no clear consensus on how soon they should return to competitive sport.[24]

Take home messages

- **Though these conditions are rarely seen in the general population, they may be more common in athletes than is generally acknowledged**
- **They can be hard to identify as there may be no detectable abnormality on examination at rest**
- **Formal diagnosis usually requires specialist tests and all athletes with suspected arterial pathology should be referred to a specialist vascular centre for full evaluation**
- **There is little robust evidence relating to treatment and management of these conditions due to the relatively small numbers of cases. Current recommendations are therefore based largely on expert consensus. It is hoped that large-scale registries may help to address this in future.**

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