Unusual Causes of Intermittent Claudication: Popliteal Artery Entrapment Syndrome, Cystic Adventitial Disease, Fibromuscular Dysplasia, and Endofibrosis

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Current Treatment Options in Cardiovascular Medicine 2009, **11:**156–166 Current Medicine Group LLC ISSN 1092-8464 Copyright © 2009 by Current Medicine Group LLC

Opinion statement

In the general population, vascular causes of exercise-induced limb discomfort are most often the result of peripheral artery disease (PAD) due to atherosclerosis. However, several other clinical entities can often mimic the symptoms of atherosclerotic PAD of the lower extremities, particularly among younger patients with fewer risk factors for atherosclerosis, who often are more athletically fit than patients with PAD. Treatment for these entities often requires percutaneous or surgical intervention. This article reviews four uncommon vascular causes of exercise-induced limb discomfort: popliteal artery entrapment syndrome, cystic adventitial disease of the popliteal artery, fibromuscular dysplasia of the lower-extremity arteries, and endofibrosis of the iliac artery.

Introduction

Atherosclerosis is the most common cause of vascular insufficiency to the lower limbs. In addition to atherosclerosis, uncommon nonatherosclerotic vascular disorders may present with exercise-induced limb discomfort, including popliteal artery entrapment syndrome (PAES), cystic adventitial disease (CAD) of the popliteal artery, fibromuscular dysplasia (FMD) of the lower-extremity

arteries, and endofibrosis of the iliac artery. Careful attention to presenting features, clinical examination, and diagnostic testing can help differentiate these unusual causes of claudication from atherosclerosis (Table 1). Treatment and prognosis are specific to each syndrome. These entities are described in detail in the following text.

Popliteal artery entrapment syndrome

PAES is a clinical syndrome resulting from the compression of the
popliteal artery within the popliteal fossa due to extrinsic anatomic
entrapment by muscular or ligamentous structures. In young, athletically fit patients presenting with intermittent claudication, PAES may
account for as many as 60% of cases [1]. Although several subtypes
of PAES have been described, the majority of cases are a result of an
anatomic abnormality resulting in malpositioning of the popliteal

Table 1. Diagnosis and management of unusual causes of claudication						
Parameter	Popliteal artery entrapment	Cystic adventitial disease	Fibromuscular dysplasia	Endofibrosis		
Demographics	Usually < 60 years old Male > female	40–50 years old Male > Female	Female > male (extrapolated from renal FMD)	30- to 50-year-old competitive athletes		
Clinical presentation	Distal calf claudication Rarely critical limb ischemia or distal embolization	Sudden onset of claudication, may be waxing and waning	Claudication	Proximal limb claudica- tion in cyclists or other high-perfor- mance athletes		
Physical diagnostic maneuvers	Diminished pulses with plantar/dorsiflexion	Ishikawa's sign: loss of pedal and/or popliteal pulses with knee flexion	None	Iliac fossa bruit after provocative exercise		
Diagnostic testing	Exercise PVR or ABI PVR or duplex ultrasound with foot plantar and dorsiflexion MR or CT angiography with foot plantar and dorsiflexion Invasive angiography with foot plantar and dorsiflexion	MR Duplex ultrasound Angiography may demonstrate hourglass or scimitar sign	Duplex ultrasound CT angiography Contrast angiography	Duplex ultrasound Exercise PVR or ABI CT angiography Contrast angiography with thigh hyperflexion		
Treatment	Surgical release of entrapment Surgical bypass with interposed saphenous vein graft	Percutaneous aspiration with CT or ultrasound guidance Surgical excision Surgical bypass	Percutaneous trans- catheter angioplasty	Surgical endarterectomy and vein patch Surgical resection or arterial shortening Arterial release Percutaneous trans- luminal angioplasty with stent		
ABI—ankle-brachial indices; FMD—fibromuscular dysplasia; PVR—pulse volume recording.						

- artery with respect to the gastrocnemius muscle, popliteus muscles, or a fibrous band. The prevalence of these anatomic abnormalities was reported to be as high as 3.5% in a postmortem study [2], but the incidence of the clinical syndrome was 0.165% in a series of young men entering the Greek military [3].
- In general, patients are young, healthy, athletic males, but those with other anatomic variants may present later in life or with sedentary lifestyles [4]. In type I PAES, the popliteal artery is displaced medially to the normally located medial head of the gastrocnemius muscle. In type II, the medial head of the gastrocnemius muscle arises from an abnormal lateral origin as the popliteal artery passes medially. In type III, the artery is compressed by an accessory muscle slip arising from the gastrocnemius head. The type IV variant results from a fibrous band across the popliteus muscle compressing the popliteal artery. Any involvement of the popliteal vein in combination with the previous types is considered type V or popliteal venous entrapment syndrome [5]. A subgroup of patients with functional PAES (type VI) has been described with normal anatomy and a hypertrophied gastrocnemius, soleus, or popliteus muscle. In contrast to those with types I through V, these patients are younger (mean age 24 years), more commonly female, and almost exclusively well-conditioned athletes or highly physically active [4].
- PAES usually presents with intermittent claudication of the calf with extreme exertion in an athletically fit patient. However, with time, repeated

- trauma to the artery may result in true stenosis or even complete occlusion of the artery, potentially even resulting in chronic or acute critical limb ischemia. Poststenotic dilatation may result in aneurysm formation and resultant distal embolization [1]. On physical examination, pulses may diminish with plantar or dorsiflexion of the foot, although this finding is not specific to PAES.
- Diagnosis may be achieved with pulse volume recording (PVR), anklebrachial indices (ABI) with segmental pressures, or duplex ultrasonography. These tests should be performed by a skilled vascular technologist with the patient's knee extended and his or her foot in the neutral, forced plantar-flexed, and dorsiflexed positions. Symptom-limited "stress testing" with ABI or PVR after exercise (eg, treadmill testing at 2.0 miles/h, 10%–12% grade) may demonstrate an absolute reduction in arterial pressure in the affected limb.
- Axial imaging with CT angiography (CTA) or magnetic resonance angiography (MRA) with the knee extended and foot in the neutral, plantarflexed, and dorsiflexed positions will demonstrate arterial narrowing and identify the particular PAES anatomy and subtype. However, the gold standard for diagnosis remains diagnostic contrast angiography with provocative maneuvers. Turnipseed [4] proposed a useful diagnostic algorithm based on his evaluation of 240 patients. In patients less than 55 years of age presenting with intermittent claudication, testing should begin with ABI/PVR with exercise. If abnormal, vascular imaging should be performed with contrast angiography or MRA with dynamic plantar and dorsiflexion of the foot. If initial exercise PVR is normal, duplex ultrasound or PVR with dynamic flexion should be performed, with any abnormalities proceeding to vascular imaging with MRA or contrast angiography as described earlier.

Surgery

- Surgery is recommended in all patients presenting with types I to IV PAES, because the natural history of PAES often results in arterial degeneration, occlusion, or distal embolization that necessitates more complex surgical intervention [1].
- In patients with functional (type VI) PAES, cessation of strenuous physical activity generally results in improvement or resolution of symptoms. Surgical management for functional PAES is controversial and best reserved for patients with severe and classic symptoms [4].
- A few cases of catheter-based embolectomy, catheter-directed thrombolysis, and percutaneous transcatheter angioplasty (PTA) have been described before muscle release in patients presenting with thrombosis or occlusion due to PAES, potentially sparing the use of surgical vein grafts [6]. However, this technique has not been adopted widely and is not recommended because of a lack of long-term follow-up data and concerns over arterial damage persisting despite release.

Standard procedure

In cases of type I, II, or III PAES involving a healthy underlying artery, release of the entrapment is achieved with surgical division of the medial head of the gastrocnemius muscle with exploration for abnormal muscle slips. In these patients, long-term patency is excellent.

If the artery has deteriorated or is thrombosed, or if an aneurysm has developed, saphenous vein interposition grafting is advised in addition to the release procedure.

Contraindications	
	Inability to undergo vascular surgery because of anatomic or comorbid medical conditions.
Complications	
	Complications arising from vascular surgery and anesthesia.
Cost/cost-effectiveness	
	Complete transport is relatively expressive but yoursely definitive. There are

Surgical treatment is relatively expensive but usually definitive. There are no acceptable alternatives for comparison.

Cystic adventitial disease

- CAD is a rare cause of intermittent claudication, usually affecting men in the fifth decade of life. It results in progressive obliteration of the arterial lumen by mucoid cysts that form within the adventitia. The exact nature and etiology of the cysts are controversial. Four theories have been proposed: the synovial or ganglion hypothesis, developmental or embryologic theory, local trauma, or systemic disease. The first two theories have received the most support in the literature [7•]. The synovial hypothesis derives from the histologic and biochemical similarities to ganglia, and holds that these cysts arise from synovial structures that enlarge and track during development or later to involve the adventitia of nearby arteries [8]. The embryologic theory proposes that mesenchymal-derived, mucin-secreting cells from nearby joint structures become incorporated into the adventitia of nearby vessels during development [8].
- The popliteal artery is affected in 85% of cases. Other reported sites of involvement include the external iliac, femoral, radial, and ulnar arteries [5]. Clinically, patients presenting with CAD do not have typical risk factors for peripheral artery disease (PAD) due to atherosclerosis, and they often describe sudden onset of limb discomfort after vigorous athletic activity. Symptoms may be waxing and waning in nature and may even transiently resolve, only to reappear months later [9]. The classic finding on physical examination is Ishikawa's sign, which is the loss of palpable pedal and/or popliteal pulses with knee flexion [7•]. Diagnosis is made by duplex ultrasound or MRI, which shows arterial stenosis with adventitial cysts in the vessel wall [5]. Angiography may demonstrate arterial stenosis; however, the classic arteriographic finding is the "hourglass" or "scimitar" sign within the lumen. Without this finding, arteriography cannot distinguish CAD from other causes of arterial stenosis.

Interventional procedures

Percutaneous transcatheter angioplasty

• Because stenosis results from external compression due to a localized, highpressure cystic structure, PTA is reported to be minimally effective in treating CAD and is not recommended if other alternatives are available [10,11].

Standard procedure

In the case of popliteal CAD, contralateral retrograde femoral access or ipsilateral antegrade femoral access is obtained. The stenotic area is crossed with a guidewire, and angioplasty balloons are inflated at the area of stenosis.

Contraindications	To delta control de la desta del del del del del de
	Inability to lie flat for the procedure, coagulopathy, renal insufficiency.
Complications	
	Rupture of cyst contents, resulting in thrombus and distal embolization; failure to improve stenosis and distal arterial flow; arterial dissection or embolization; access site complications including bleeding, hematoma, pseudoaneurysm formation, and arteriovenous fistula formation.
Cost/cost-effectiveness	
	Given the expense of PTA and the low likelihood of its success, it is not a cost-effective option.
Percutaneous aspiration	
·	• A series of nine patients treated with ultrasound-guided cyst aspiration resulted in technical success and improvement in clinical symptoms in all patients, with no reported complications. Follow-up for a mean of 14.8 months with a maximum of 32 months demonstrated no cases of recurrence [12].
	 Recurrence after percutaneous aspiration has been reported, therefore requiring close ultrasonographic follow-up [13].
Standard procedure	
·	Ultrasound- or CT-guided percutaneous aspiration of popliteal CAD may be performed with the patient in the prone position, and yields a gelatinous material. Doppler ultrasound may be used to confirm resolution of the cyst and normalization of arterial flow in real time.
Contraindications	
	Inability of the patient to lie prone for the procedure.
Complications	
·	Damage to the popliteal artery is a concern, although it has not been reported in the literature. The cyst may recur, with reaccumulation of the mucoid contents.
Special points	
	Surveillance duplex ultrasonography is recommended at 3, 6, and 12 months after the procedure. Aspiration does not preclude future surgery if there is recurrent disease.
Cost/cost-effectiveness	
	Aspiration is less expensive than open surgical procedures but is balanced with a higher recurrence rate, requiring greater monitoring and possibly repeat procedures. Ultrasound guidance is significantly less expensive than CT guidance. There are no direct comparisons with surgical management.
Surgery	
Standard procedure	
	If the artery is not totally occluded, the preferred technique is open evacuation with excision of the adventitial cyst and preservation of the vessel intima and media. In the case of total occlusion or arterial degeneration, autologous vein bypass grafting is performed [7•]. Some surgeons

ation, autologous vein bypass grafting is performed [7•]. Some surgeons

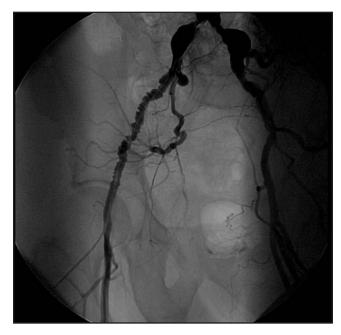


Figure 1. Medial fibroplasia variant of fibromuscular dysplasia of the right external iliac artery. Note the bilateral common iliac artery aneurysms.

prefer bypass grafting in all patients because it is more definitive and may be associated with a lower recurrence rate [14].

	Inability to undergo vascular surgery because of anatomic conditions or serious medical comorbidities.
Complications	
	Complications arising from vascular surgery and anesthesia.
Special points	
	The recurrence rate for open cystic evacuation with excision is reported to be 10% [7•].
Cost/cost-effectiveness	
	There are no direct comparisons with percutaneous aspiration. Although

There are no direct comparisons with percutaneous aspiration. Although surgical management may entail more initial costs than aspiration, this expense is offset by lower recurrence rates.

Fibromuscular dysplasia

• FMD is an idiopathic, nonatherosclerotic, noninflammatory process of the peripheral, abdominal, and cerebrovascular arteries. The renal arteries and extracranial cerebrovascular arteries are most commonly affected. Rarely, FMD may affect other vascular territories, including the iliac, femoral, or popliteal arteries, and result in intermittent claudication and critical limb ischemia, largely as a result of distal embolization. Histopathologically, FMD is routinely subdivided into four subtypes based on the location of the lesion: medial fibroplasia (Fig. 1), perimedial fibroplasia, intimal fibroplasia, and adventitial (or periarterial) hyperplasia [15].

- Within the renal arteries, the vast majority of cases are of the medial fibroplasia variant, consisting of multiple alternating stenotic and aneurysmal segments, resulting in the classic "string of beads" appearance on angiography [16•]. In the medial fibroplasia variant, the beads are larger than the arterial lumen. Perimedial fibroplasia is characterized by one or multiple focal stenoses, often with a robust collateral network [17]. The intimal fibroplasia variant appears as a focal stenotic segment or tubular stenosis. Adventitial hyperplasia may have several different morphologies, including focal or tubular stenoses [17]. There are no data regarding the distribution of histologic subtypes within the lower-extremity arteries. Likewise, epidemiologic and demographic data are limited in patients with FMD affecting the lower extremities. However, most patients with symptoms related to FMD are younger, are female, and do not classically possess the risk factors commonly associated with atherosclerotic PAD.
- The diagnosis of FMD should be entertained in a patient presenting with symptoms consistent with claudication but without classic atherosclerotic risk factors, particularly in a female patient. FMD has been associated with Ehlers-Danlos syndrome (type IV), Alport's syndrome, pheochromocytoma, Marfan's syndrome, and Takayasu's arteritis [17]. Although the gold standard for diagnosis of FMD remains invasive contrast angiography, duplex ultrasonography and multidetector CTA may provide adequate images to confirm the diagnosis. MRA may not have sufficient spatial resolution to allow a definitive diagnosis of FMD. It is generally accepted that patients with renal artery FMD be evaluated for potential treatment benefit. However, in patients with carotid FMD, there is a risk of intracranial aneurysms; therefore, imaging of the intracranial arterial circulation should be strongly considered.

Interventional procedures

Percutaneous transluminal angioplasty with or without stent placement

- Although FMD in the iliac and popliteal arteries is a rare condition with few published clinical reports of treatment with PTA, PTA is considered the first-line therapy. Largely, this is because of PTA's reported efficacy as primary therapy for FMD involving the renal arteries, which has demonstrated low recurrence rates with long-term follow-up [18].
- In the renal arteries, treatment with PTA alone yields excellent results. Stent placement in the renal and extrarenal arteries should be reserved for cases with elastic recoil causing severe residual stenosis, or PTA resulting in flow-limiting arterial dissection.
- Recurrent stenosis after PTA may be treated with repeat PTA with or without stent placement.

Standard procedure

Usually performed via ipsilateral femoral arterial access. In the case of dissection, recoil, or persistent gradient, a balloon-expandable stent may be deployed at the site of stenosis.

Contraindications

Inability to lie flat for the procedure, coagulopathy, renal insufficiency.

Complications

Inability to dilate the artery because of elastic recoil, perforation, dissection, and restenosis; access site complications including bleeding, hematoma, pseudoaneurysm formation, and arteriovenous fistula formation.

Cost/cost-effectiveness

Although no direct comparisons with surgery are available, PTA likely is significantly less expensive, with a shorter hospital stay.

Surgery

• Given the excellent short- and long-term success rates for PTA, surgery for FMD should be reserved for rare cases in which PTA is technically impossible.

Standard procedure

Surgical revascularization may be performed with interposed saphenous vein graft or synthetic material (likely reserved for cases in which the distal anastomosis will be in the femoral or above-knee popliteal arteries). Other options, such as patch angioplasty or arterial dilation, may be available on an individual case basis depending on the location and extent of involvement.

Contraindications

Inability to undergo vascular surgery because of anatomic abnormalities or serious comorbid medical conditions.

Complications

Complications arising from vascular surgery and anesthesia.

Cost/cost-effectiveness

There have been no direct comparisons with PTA or stent placement; however, surgery likely has significantly higher procedural costs and a longer length of hospital stay.

Endofibrosis of the iliac artery

- Endofibrosis, a rare condition resulting in iliac artery stenosis, classically occurs among competitive cyclists aged 30 to 50 years. It has been reported in other athletes, including runners [19,20•,21]. Endofibrosis is caused by intimal hyperplasia, which is similar histologically to the intimal fibroplasia variant of FMD. However, in contrast to FMD, endofibrosis often is bilateral, is more diffuse in nature, and almost exclusively involves the external iliac arteries [17].
- A recent report of 19 histopathologic specimens of endofibrosis demonstrated loose connective tissue with moderate to high cellularity, loose collagen fibers, and a dearth of inflammatory cells compared with atherosclerotic specimens [22]. Endofibrosis is thought to be a result of repeated trauma to the vessel caused by stretching and compression of the external iliac artery at the level of the hip, compression by the psoas muscle, tethering from psoas muscle arteries, and shear forces due to enhanced blood flow to the lower extremities [23]. Patients often complain of claudication, a sensation of swelling, or neurologic symptoms in the proximal lower limb at the point of maximal exertion [24].
- Although physical examination is frequently normal, a bruit may be audible in the iliac fossa after exercise. As an initial screening test, exercise ankle-brachial indices appear to have good sensitivity for

endofibrosis but provide limited information about the specific level or type of arterial lesion. Patients with endofibrosis of the iliac artery typically have normal rest ABI with a significant decrease in ABI after exercise [24]. Duplex ultrasonography performed by an experienced ultrasonographer may be useful in confirming the diagnosis of endofibrosis, and is best used in concert with exercise ABI, pulse volume recordings, and segmental pressures [20•]. Contrast angiography long has been considered the gold standard for diagnosis, and the sensitivity of this test has been reported to be as high as 100% when the patient is imaged with thigh hyperflexion in the "racing position" [25]. CT and MRA may prove to be useful modalities, particularly if imaging can be performed with thigh flexion; however, data on diagnostic accuracy are limited.

Interventional procedures

Percutaneous transcatheter angioplasty with or without stent placement

- Because the affected patient population involves highly trained and often competitive athletes, the appeal of PTA as a minimally invasive therapeutic modality is evident.
- Several authors have raised concerns about the ability to acutely dilate the fibrotic artery, the possibility of dissection resulting from PTA, and the long-term durability of angioplasty. Cases have been reported of successful PTA, with short- and medium-term follow-up showing symptomatic improvement without recurrence [19,24].
- One case of balloon-expandable stent deployment was reported with symptomatic improvement and patency as determined by duplex ultrasonography to 3 months [20•]. However, because of the risk of fracture with stent deployment in an area subject to mechanical stress, the routine use of stenting for endofibrosis is not recommended.

Standard procedure

For iliac artery involvement, ipsilateral retrograde femoral access is obtained. The stenotic area is crossed with a guidewire, and angioplasty balloons are inflated at the area of stenosis. In the case of dissection, recoil, or persistent gradient, a balloon-expandable or self-expanding stent may be deployed at the site of stenosis.

Contraindications

Inability to lie flat for the procedure, coagulopathy, renal insufficiency.

Complications

Inability to dilate the artery because of elastic recoil, dissection, and restenosis; access site complications including bleeding, hematoma, pseudoaneurysm formation, and arteriovenous fistula formation.

Cost/cost-effectiveness

Although no direct comparisons with surgery are available, PTA likely is less expensive, with a shorter hospital stay.

<u>Surgery</u>

• Despite the concerns over subjecting a high-performance athlete to a surgical procedure that at minimum requires a significant recovery time, most cases in the literature have been treated surgically.

Standard procedure	
	Several surgical procedures to treat endofibrosis have been described, including endarterectomy and vein patch placement; resection of the stenotic portion with saphenous vein interposition graft; shortening of the artery, which is useful if the underlying problem is related to kinking; and arterial release, which is useful if there is functional kinking and no luminal stenosis of the artery. Arterial release was demonstrated to be highly successful and safe in a prospective study of 23 carefully selected patients [26].
Contraindications	
	Inability to undergo vascular surgery because of anatomic abnormalities or comorbid medical conditions.
Complications	
	Complications arising from vascular surgery and anesthesia.
Cost/cost-effectiveness	
	Although there have been no direct comparisons with PTA or stent placement, surgery likely has higher procedural costs and a longer length of hospital stay.

Disclosures

No potential conflicts of interest relevant to this article were reported.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance
- Levien LJ, Veller MG: Popliteal artery entrapment syndrome: more common than previously recognized. J Vasc Surg 1999, 30:587–598.
- 2. Gibson MH, Mills JG, Johnson GE, Downs AR: Popliteal entrapment syndrome. *Ann Surg* 1977, 185:341–348.
- 3. Bouhoutsos J, Daskalakis E: Muscular abnormalities affecting the popliteal vessels. *Br J Surg* 1981, 68:501–506.
- 4. Turnipseed WD: Popliteal entrapment syndrome. *J Vasc Surg* 2002, 35:910–915.
- Wright LB, Matchett WJ, Cruz CP, et al.: Popliteal artery disease: diagnosis and treatment. Radiographics 2004, 24:467–479.
- 6. Steurer J, Hoffmann U, Schneider E, et al.: A new therapeutic approach to popliteal artery entrapment syndrome (PAES). *Eur J Vasc Endovasc Surg* 1995, 10:243–247.
- 7.• Ortiz MW, Lopera JE, Gimenez CR, et al.: Bilateral adventitial cystic disease of the popliteal artery: a case report. Cardiovasc Intervent Radiol 2006, 29:306-310.

Case report and thorough discussion of the literature of CAD of the popliteal artery.

- 8. Levien LJ, Benn CA: Adventitial cystic disease: a unifying hypothesis. *J Vasc Surg* 1998, 28:193–205.
- Cassar K, Engeset J: Cystic adventitial disease: a trap for the unwary. Eur J Vasc Endovasc Surg 2005, 29:93–96.
- 10. Fox RL, Kahn M, Adler J, et al.: Adventitial cystic disease of the popliteal artery: failure of percutaneous transluminal angioplasty as a therapeutic modality. *J Vasc Surg* 1985, 2:464–467.

- Khoury M: Failed angioplasty of a popliteal artery stenosis secondary to cystic adventitial disease—a case report. Vasc Endovasc Surg 2004, 38:277–280.
- 12. Do DD, Braunschweig M, Baumgartner I, et al.: Adventitial cystic disease of the popliteal artery: percutaneous US-guided aspiration. *Radiology* 1997, 203:743–746.
- 13. Sieunarine K, Lawrence-Brown MM, Kelsey P: Adventitial cystic disease of the popliteal artery: early recurrence after CT guided percutaneous aspiration. *J Cardiovasc Surg* (*Torino*) 1991, 32:702–704.
- di Marzo L, Peetz DJ Jr, Bewtra C, et al.: Cystic adventitial degeneration of the femoral artery: is evacuation and cyst excision worthwhile as a definitive therapy? Surgery 1987, 101:587–593.
- Stanley J: Renal artery fibrodysplasia. In Renal Vascular Disease. Edited by Novick A, Scoble J, Hamilton G. London: WB Saunders; 1996:21–23.
- 16.• Plouin PF, Perdu J, La Batide-Alanore A, et al.: Fibromuscular dysplasia. Orphanet J Rare Dis 2007, 2:28.

A thorough recent review of renal and extrarenal FMD.

- 17. Slovut DP, Olin JW: Fibromuscular dysplasia. N Engl J Med 2004, 350:1862–1871.
- 18. Davies MG, Saad WE, Peden EK, et al.: The long-term outcomes of percutaneous therapy for renal artery fibromuscular dysplasia. *J Vasc Surg* 2008, 48:865–871.
- Ford SJ, Rehman A, Bradbury AW: External iliac endofibrosis in endurance athletes: a novel case in an endurance runner and a review of the literature. Eur J Vasc Endovasc Surg 2003, 26:629–634.

20.• Maree AO, Ashequl Islam M, Snuderl M, et al.: External iliac artery endofibrosis in an amateur runner: hemodynamic, angiographic, histopathological evaluation and percutaneous revascularization. Vasc Med 2007, 12:203-206.

Case report of endofibrosis in a runner, with a review of the literature and representative histopathology.

- Walder J, Mosimann F, Van Melle G, Mosimann R: Iliac endofibrosis in 2 cycling racers [in French]. Helv Chir Acta 1985, 51:793–795.
- 22. Vink A, Bender MH, Schep G, et al.: Histopathological comparison between endofibrosis of the high-performance cyclist and atherosclerosis in the external iliac artery. *J Vasc Surg* 2008, 48:1458–1463.
- 23. Chevalier JM, Enon B, Walder J, et al.: Endofibrosis of the external iliac artery in bicycle racers: an unrecognized pathological state. *Ann Vasc Surg* 1986, 1:297–303.
- 24. Alimi YS, Accrocca F, Barthelemy P, et al.: Comparison between duplex scanning and angiographic findings in the evaluation of functional iliac obstruction in top endurance athletes. Eur J Vasc Endovasc Surg 2004, 28:513–519.
- Kral CA, Han DC, Edwards WD, et al.: Obstructive external iliac arteriopathy in avid bicyclists: new and variable histopathologic features in four women. J Vasc Surg 2002, 36:565–570.
- 26. Schep G, Bender MH, van de Tempel G, et al.: Detection and treatment of claudication due to functional iliac obstruction in top endurance athletes: a prospective study. *Lancet* 2002, 359:466–473.