Case Report

Fibromuscular Dysplasia of the Lower Extremities

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Fibromuscular dysplasia (FMD) is a nonatherosclerotic, non-inflammatory vascular disease that mainly affects the renal and internal carotid arteries. Involvement of other sites, including arteries of the extremities, is uncommon, and only a few histologically confirmed cases have been reported. FMD of the arteries of the extremities can result in ischemia requiring surgical or endovascular reconstruction. In the present report, two cases of FMD are described: one case of femoropopliteal artery occlusive disease, and one case of nonsymptomatic progression of external iliac artery dissection, both with histological confirmation of FMD. Clinical presentation, treatment, outcome and histological findings of previously reported cases are reviewed. FMD should be considered as a cause of occlusion, stenosis, dissection or aneurysm of the peripheral arteries: although rare, it can lead to limb-threatening ischemia or life-threatening aneurysm rupture.

Key words: fibromuscular dysplasia, femoral artery, iliac artery, dissection, aneurysm, extremity

INTRODUCTION

Fibromuscular dysplasia (FMD) is a nonatherosclerotic, non-inflammatory systemic occlusive arteriopathy of unknown etiology that mainly affects renal, carotid, and splanchnic arteries. Since it was first described in 1938,¹⁾ numerous reports have appeared describing the disease both in renal arteries and extra-renal vessels.²⁾ Involvement of the arteries of the extremities is uncommon, and only a few histologically confirmed cases have been reported. FMD-related lesions in the extremities occasionally cause limb ischemia through luminal stenosis or peripheral thrombus. Aneurysm formation caused by FMD may also cause limb ischemia through peripheral thrombus. This ischemia can be resolved with arterial reconstruction.

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REPORT OF CASES

Case 1

Clinical findings

A 63-year-old woman presented with disabling left leg claudication, along with hypertension and hypercholesterolemia treated by oral medication. Physical examination revealed a pulsating mass in the lower abdomen, normal pulses over the femoral arteries, a bilateral pulsating mass on the popliteal arteries, and no pulsation over the left pedal artery. The left foot was cold with skin pallor. Ankle-brachial pressure indices were 0.37 on the left and 0.75 on the right. Laboratory values upon admission included serum cholesterol of 396 mg/dl and triglycerides of 674 mg/dl. Three-dimensional computed tomography (CT) revealed bilateral common iliac artery aneurysms (measuring 5.9 cm in the left and 4.0 cm in the right), and serial stenoses altering with mural aneurysms in bilateral superficial femoral and popliteal arteries (the classic “string-of-beads” appearance) (**Fig. 1**). Popliteal arteries presented with aneurysms (measuring 17 mm in the left and 20 mm in the right) with luminal thrombi. The left popliteal artery was occluded above the trifurcation, and only the peroneal artery was contrast enhanced through collateral vessels. On the right side,

Fig. 1 Computed tomographic angiography of case 1. “String-of-beads” appearance on bilateral the femoral and popliteal arteries.

the lumen of the right popliteal aneurysm was patent, the tibioperoneal trunk was occluded, and only the anterior tibial artery was patent. There were no abnormal findings on the renal or visceral arteries, although the “string-of-beads” appearance was observed bilaterally in the internal carotid arteries. From the characteristic “string-of-beads” appearance, FMD of bilateral femoropopliteal and internal carotid arteries, and bilateral common iliac and bilateral popliteal artery aneurysms were diagnosed.

The iliac aneurysms were successfully excised and reconstructed with a Y graft. After 1 month, left leg ischemia was treated with a femoral to peroneal artery bypass with a reversed great saphenous vein. The superficial femoral artery was ligated at its origin, and its short segment was excised for histological examination. The postoperative course was uneventful.

Histological findings

The medial layer of the femoral artery was thickened by marked collagen fibrosis and muscular hyperplasia. Smooth muscle fibers were focally disorganized, and internal elastic membrane was partially fragmented. Intima contained marked fibrous thickening and a focus of atheromatous gruel (**Fig. 2**). Findings were compatible

Fig. 2 Femoral artery of case 1 in cross-section. Medial layer is focally thickened with marked collagen fibrosis and disorganized muscular hyperplasia. Internal elastic membrane is partially fragmented. Intima also shows a marked fibrous thickening and focus of atheromatous gruel. (elastic van Gieson stain, original magnification $\times 40$)

with medial fibroplasia with associated atherosclerotic change caused by aging and hyperlipidemia.

Case 2

Clinical findings

A 37-year-old man presented with sudden-onset back pain. No clinical features suggestive of Marfan or Ehlers-Danlos syndrome were present. Although the patient reported no history of vascular events, he had a family history of maternal death at 60 years of age by rupture of an aneurysm. Because CT revealed the presence of a retroperitoneal hematoma surrounding the body of the pancreas, acute pancreatitis was diagnosed and the treatment with intravenous administration of protease inhibitor was initiated. Thereafter, detailed CT findings suggested dissection and rupture and spontaneous thrombus of the celiac artery (**Fig. 3**). Percutaneous transluminal coil embolization of the partially thrombosed celiac trunk was successfully conducted. Systemic vascular investigation revealed a short segment of dissection in the right external iliac artery. The renal, cerebral, and peripheral arteries appeared normal. No symptoms of lower leg ischemia were present initially. After follow-up by three-dimensional CT for 17 months, the right iliac dissected lumen was enlarged, and a new dissection appeared in the left external iliac artery (**Fig. 4**). Resection and interposition of bilateral external iliac arteries with ePTFE prosthetic grafts were performed.

Fig. 3 Computed tomographic angiography of case 2. Dissected celiac artery with segmental thrombosis. Partially patent lumen of splenic artery was contrast enhanced through posterior pancreatic artery. CA, celiac artery; SA, splenic artery; PHA, proper hepatic artery; GDA, gastroduodenal artery; LGA, left gastric artery; DPA, dorsal pancreatic artery

Fig. 4 Computed tomographic angiography of case 2. Dissections in right (A) and left (B) external iliac arteries.

Fig. 5 Macroscopic luminal view of resected external iliac artery in case 2. Dissection and unusual corrugations by serial wall thickening and the intervening crease are visible.

Luminal visualization of the resected external iliac arteries revealed dissection and unusual corrugations by serial wall thickening and an intervening crease (**Fig. 5**). The postoperative course was uneventful.

Histological findings

External iliac artery cross-section revealed both intimal and medial thickening and medial dissection. Intimal layer had irregularly arranged mesenchymal cells present in a loose matrix of fibrous connective tissue. No lipid or inflammatory components were present. The inner media was thickened with disoriented smooth muscle fibers, and the internal elastic membrane was partially fragmented. A dissected channel was present at the outer third of the media, dividing the medial layer into a fibroplastic inner

layer and an almost intact paramedial layer. External elastic membrane and adventitia were almost intact (**Fig. 6**). Findings were compatible with medial dissection with concomitant secondary intimal fibroplasia.

DISCUSSION

Fibromuscular dysplasia is a group of nonatherosclerotic, non-inflammatory arteriopathy that most commonly involve renal and carotid arteries, though it has been described in almost every arterial bed in the body. The clinical presentation may vary from an asymptomatic condition to a limb-threatening ischemia or life-threatening hemorrhage, depending on the involvement of the arterial bed, degree of stenosis, and pathological

Fig. 6 External iliac artery of case 2 in cross-section. A dissected channel is present at the outer third of media, dividing the medial layer into a fibroplastic inner part and an almost intact perimedial part. Intima is also thickened with irregularly arranged mesenchymal cells. (elastic van Gieson stain, original magnification $\times 40$)

types of FMD.

A pathological classification of FMD was proposed by Harrison and McCormack according to the vessel layer involved; i.e., medial, intimal, and adventitial.³⁾ Medial fibromuscular dysplasia is most common (85%) and has been defined further into four subgroups: medial fibroplasia, perimedial fibroplasia, medial hyperplasia, and medial dissection.

Medial fibroplasia occurs most frequently and is well known because of the “string-of-beads” appearance on angiography. This appearance is the result of focal areas of stenosis with intervening areas of aneurysmal dilatation. Stenotic areas are due to thickened fibromuscular ridges, in which the arterial muscle is completely replaced by fibroplasia with loose collagen. The mural aneurysm results from thinning or complete loss of smooth muscle. The internal elastic membrane is deficient in the aneurysms.

In medial hyperplasia, the stenosis is produced primarily by hyperplasia of arterial smooth muscle cells.

In medial dissection, a channel is located on the outer third of the media. The media displays zones of fibroplasia replacing degenerated elastic fibrils, particularly around the channel.

Perimedial fibroplasia is characterized by marked, intense fibroplasia in the outer half of the media which usually replace the external elastic membrane. The fibroplastic layer varies in thickness, so considerable irregularity of the lumen is produced.

Intimal fibroplasia occurs in less than 10% of patients

with FMD and is characterized by an accumulation of loose, moderately cellular fibrous tissue without atherosclerotic or inflammatory components. Blunt trauma, catheter-induced intraluminal injury, or luminal stenosis of the vessel may cause “secondary” intimal fibroplasia.

Adventitial fibroplasia is the least common in the renal FMD and, to our knowledge, has never been reported in extremity arteries.

Using the Harrison and McCormack classification, case 1 was diagnosed as medial fibroplasia with added atherosclerotic change; case 2 was diagnosed as medial dissection with secondary intimal fibroplasia. Without a histological diagnosis, we are not certain if the celiac arterial dissection in case 2 was caused by FMD, although FMD could occasionally occur in visceral arteries, and there are some reports describing celiac artery dissection caused by FMD, with a histological diagnosis.⁴⁾

The etiology of FMD has been the subject of much speculation. Cigarette smoking is associated with an increased risk of this disease; however, the mechanisms by which smoking contributes to the development of FMD have not been elucidated. The predominance of the disease in middle-aged women has led to the suggestion that it is caused by a hormonal influence on smooth muscle. Mechanical vessel wall stress is another classical hypothesis, as FMD of the renal arteries often co-presents with ptosis of the right kidney. Sang et al. analyzed these environmental factors in a case-control study of 33 patients with FMD. The number of pregnancies, oral contraceptives, gynecological disorders, and incidence of abnormal renal mobility did not differ between patients with FMD and matched controls.⁵⁾ Genetic factors may also play a role in FMD, and since the disease is more common among first-degree relatives,⁶⁾ an autosomal dominant transmission of the disease has been suggested.^{7, 8)} Some inheritable connective tissue disorders, such as Ehlers-Danlos syndrome (type IV)⁹⁾ and Marfan syndrome,¹⁰⁾ are associated with multiple aneurysms and arterial dissection and have been associated with FMD. Bofinger et al. reported a significantly higher frequency of angiotensin-converting enzyme I allele in FMD patients than in controls¹¹⁾ and suggested that a polymorphism of the renin-angiotensin system may cause defective remodeling in the muscular layer of arteries and subsequent development of FMD. Autoimmunity associated with HLA Drw6 was also suggested in a limited study.⁵⁾ Because of the lack of large cohorts, these studies on the possible causes of FMD have not been confirmed.

Numerous reports have described FMD occurring in

Table 1 FMD in femoral and popliteal arteries

Author (year)	Age	Sex	Site of disease	Appearance	Presentation	Treatment	Outcome	Histology
Prince and Vawter (1971)	1	M	Bilateral femoral arteries	Beaded	Ischemia	Amputation	Amputation	Medial
	16	M	Right popliteal artery	Aneurysm	Thrombosis	Resection	Good	Medial
Tisnado et al. (1982)	69	F	Bilateral femoral to popliteal arteries	Beaded	Microemboli of toes	Conservative	good	N/A
Iwai et al. (1985)	65	F	Bilateral popliteal arteries	Beaded	Claudication	Angioplasty Sympathectomy	Good	Medial
	25	F	Right popliteal artery	Beaded	None	None	Good	N/A
	48	M	Right deep femoral artery	Beaded	Raynauds syndrome	Sympathectomy	Good	N/A
Stinnet et al. (1987)	10	F	Bilateral popliteal arteries	Aneurysm	Claudication	Bypass	Good	Medial hyperplasia
Dungen et al. (1990)	15	F	Right superficial femoral artery	Occlusion	Claudication	Bypass	Good	Perimedial fibroplasia
	21	M	Left superficial femoral artery	Occlusion	Claudication	Sympathectomy and bypass	Amputation	Medial dissection
	36	M	Left popliteal artery	Occlusion	Claudication	Bypass	Good	Intimal
Fiche et al. (1991)	20	M	Right popliteal artery	Aneurysm	Knee effusion	Interposition	Good	Medial
Neukirch et al. (1996)	39	F	Left popliteal artery	Aneurysm	None	Resection and anastomosis	Good	Media
present case (2009)	63	F	Bilateral femoral to popliteal arteries	Beaded	Claudication	Bypass	Good	Medial

FMD, fibromuscular dysplasia

renal arteries and extrarenal vessels. In 1982, Mettinger and Ericson reviewed reports concerning 1197 patients with FMD.¹²⁾ Renal arteries were involved in 58%, cervicocranial arteries in 32%, visceral arteries in 25%, and iliac arteries in 25% of cases. Of these nearly 1200 cases, only one was located in an infrainguinal artery of the lower extremity.

To the best of our knowledge, only 13 cases of FMD of the femoral or popliteal artery have been reported in the literature, including case 1 in the present report (**Table 1**).^{13–19)} Of these 13 cases, seven were female and six were male, the mean age was 33 years, and the age range was 1–69 years. Angiographically, six cases appeared beaded, four were single aneurysms, and three were total occlusions. Eight cases of FMD of the femoral or popliteal artery had histological FMD diagnoses: seven were medial and one intimal.

Although FMD of the iliac artery (the third most common arterial site) with a beaded appearance or with

aneurysm formation has been well documented, iliac artery dissection caused by FMD has been rarely reported. To our knowledge, only 12 cases of iliac artery dissection associated with FMD have been reported, including case 2 in this report (**Table 2**).^{20–26)} Of these cases, seven were male and five were female. The mean age was 42 years, and the age range was 21–56 years. Most patients presented with leg ischemia or local pain, underwent bypass or resection and interposition, and had good outcomes. Two patients involved rupture and hypovolemic shock; and, in both cases, the patients died post-operatively. Seven reported cases of FMD of the iliac artery have had histological diagnoses; all were medial FMD. Although our case was asymptomatic, an operative indication was decided from the progressive enlargement of the dissected pseudolumen, newly developed contralateral iliac dissection, and possible disastrous consequences of ruptured dissecting aneurysm.

It is generally not considered difficult to differentiate

Table 2 Iliac artery dissection associated with FMD

Author (year)	Age	Sex	Site of dissection	Symptom	treatment	Outcome	histology
Burri et al. (1983)	45	F	Left external iliac artery	Inguinal pain	Bypass	Good	medial
Sauer et al. (1990)	56	F	Right common iliac artery	Claudication	Conservative	Good	N/A
Patel et al. (1990)	39	M	Left external iliac artery	Inguinal pain	Bypass	Good	medial
Thevernet et al. (1992)	45	F	Right external iliac artery	Leg ischemia	Bypass	Good	medial
	51	M	Bilateral external iliac arteries	Claudication	Bypass	Good	N/A
	47	F	Right external iliac artery	Abdominal pain	Bypass	Good	N/A
	29	F	Left external iliac artery	Abdominal pain	Interposition with vein	Good	N/A
	53	M	Right external iliac artery	Claudication	Bypass	Good	N/A
Luck et al. (2002)	45	M	Right external iliac artery	Claudication	Endarterectomy	Good	medial
Honjo et al. (2004)	30	M	Left common iliac artery	Shock, rupture	Bypass	Dead	medial
Yoshioka et al. (2005)	21	M	Left common iliac artery	Abdominal pain, rupture	Bypass	Dead	medial
present case (2009)	37	M	Bilateral external iliac arteries	none	Interposition with ePTFE	Good	medial

FMD, fibromuscular dysplasia

atherosclerosis from FMD. Atherosclerosis typically occurs in older patients with typical cardiovascular risk factors. On the other hand, FMD usually occurs in younger patients who have few cardiovascular risk factors. Lacking specific markers or effective non-invasive tests, angiographic imaging is almost essential for the diagnosis of FMD. Recently, computed tomographic angiography or gadolinium-enhanced magnetic resonance angiography have emerged as less invasive diagnostic imaging compared to contrast-enhanced digital subtraction angiography. Although the “string of beads” appearance is well known as a specific angiographic feature of FMD, FMD subtypes other than medial fibroplasia can present with a wide variety of angiographic appearances. For example, intimal fibroplasia and medial hyperplasia may present as a smooth or focal stenosis. Perimedial fibroplasias also may present with “string-of-beads” appearance. However, the beads in perimedial fibroplasia do not exceed the original arterial lumen. Kincaid et al. reported that “string-of beads” appearances were observed in 78 (62%) of 125 patients with renal FMD.²⁷⁾ In cervicocranial vessels, Mettinger et al. reported that 26 (62%) of 37 patients had a “string-of-beads” appearance.²⁸⁾ In cases of infrainguinal femoral and popliteal arteries, 6 (46%) of 13 had a “string-of-beads” appearance (**Table 1**). Therefore, the “string-of-beads” appearance can be specific to medial fibroplasia, but is not sensitive

enough to diagnose all cases of FMD. Moreover, these angiographic signatures are only visible at the early stage of disease. Once an occluded vessel takes on atherosclerotic and/or inflammatory features, it can be difficult to distinguish FMD from atherosclerosis or arteritis without histological confirmation. The collection of tissue for histological analysis may not be possible from occluded peripheral arteries treated with bypass surgery. Therefore, a fair amount of peripheral arterial occlusion caused by fibrodysplastic disease may be misdiagnosed as a common atherosclerotic disease.

FMD of lower extremities may cause both acute and chronic leg ischemia by luminal stenosis, thrombosis and distal embolism. It may present with cold legs, intermittent claudication, ischemic ulcer or limb gangrene and may require arterial reconstruction.

Treatment of FMD of the extremities includes anticoagulation, lumbar sympathectomy, bypass surgery, aneurysm resection, and interposition, depending upon the arterial segments involved. Recently, successful percutaneous transluminal angioplasty and endovascular therapy have also been reported.^{29–31)}

In conclusion, fibromuscular dysplasia should be considered as a potential cause of arterial occlusion, stenosis, dissection or aneurysm in the peripheral arteries. Although it is a rare cause of such pathologies, FMD may lead to life-threatening hemorrhage or limb-threat-

ening ischemia.

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