

Peripheral Arterial Embolism from the Mural Abdominal Aortic Thrombosis

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Received: 11 Jan 2021
Accepted: 27 Jan 2021
Published: 30 Jan 2021

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Keywords:

Aortic dilatation; Abdominal aortic aneurysm; Critical limb ischemia; Blue toe syndrome; Duplex ultrasound; Combined antithrombotic therapy

Citation:

Musil D. Peripheral Arterial Embolism from the Mural Abdominal Aortic Thrombosis. *Ann Clin Med Case Rep.* 2021; V5(9): 1-3.

1. Abstract

We present two men suffering from critical limb ischemia (Fontaine gr. IV.) due to mural aortic thrombosis with peripheral arterial embolism. Duplex ultrasound was adequate for the diagnosis in both cases. CTA only confirmed the ultrasound findings. Anticoagulation and vasodilation, combined in the second case with antiplatelet therapy, was successful. During the first 4 months the thrombus dissolved/diminished, and symptoms and signs of limb ischemia improved in a few weeks.

2. Introduction

The most frequent sources of peripheral arterial embolism are left heart sections, aneurysms of the abdominal aorta, aorto-arterial thrombosis and venous thromboembolism in the *foramen ovale apertum*. A less frequent cause of peripheral embolism is mural aortic thrombosis [1] (Reber, 1999). In such cases, the thrombus is present on exulcerated atherosclerotic plaques or as atypical mural aortic thrombosis, non-atherosclerotic/non-aneurysmatic, which is a heterogeneous group of disorders related to acute aortitis, malignant diseases, infections, surgery, Ormond's disease, hypercoagulable states, TBC, autoimmune disorders and other triggering factors [2-4] (Vaideeswar, 2001, Taglietti, 2008, Dieskemann, 2009).

3. Case Reports

We present the case histories of two old men suffering from critical limb ischemia due to peripheral arterial embolism. The source of the embolisation was atherosclerotic, abdominal aorta. Anticoagulation and vasodilation, combined in the second case with antiplatelet therapy, was successful and symptoms and signs of limb ischemia improved in a short time. Anticoagulation combined with antiplatelet therapy in the second case was successful.

3.1. Case 1

An 80-year-old man presented with a 3-month history of rest and night pain in both feet. The pain started about a month after coronary artery bypass graft (CABG) for coronary artery multivessel disease. The

previous month, the pain was so intense that he could not sleep, waking up every hour and for relief he took down the legs of the bed. The pain was not exacerbated by walking. Analgesics did not relieve the problem and the signs progressed to small skin necrosis on the toetips. The man was a no smoker but he had an eight-year history of type 2 diabetes mellitus (T2DM) with diabetic nephropathy, neuropathy and chronic renal insufficiency, arterial hypertension, dyslipidemia, coronary artery disease with acute myocardial infarction 16 months previously with PTCA and reinfarction four months beforehand with CABG, when dual antiplatelet therapy (clopidogrel 75 mg/day plus acetylsalicylic acid 100 mg/day) was started.

On physical examination the patient was hemodynamically stable. Both feet were cold. He had cyanotic toes sensitive to gentle palpation. On the tips of the left big toe and second toe and right big toe, second, third and fourth toe were seen minor skin necroses (Figure 1). Peripheral pulses were palpable. The ankle-brachial index (ABI) was 1.1 on the left ankle and 1.2 on the right ankle. Duplex ultrasound (DUS) of the lower extremities showed no evidence of arterial occlusion or hemodynamically significant stenosis. Ultrasound examination of the abdominal aorta revealed diffuse calcified sclerotic plaques and mild dilatation (20-25 mm) of the aorta, 35-45 mm in length with mural thrombosis. Enlargement of the abdominal aorta did not meet the diagnostic criterion for an aneurysm (Figure 2). A CT angiography (CTA) using contrast confirmed mild ectasia of the subrenal abdominal aorta with irregular mural thrombosis and leakage of blood into the thrombosis (Figure 3).

The man was hospitalized for *bilateral critical limb ischemia as a result of peripheral arterial embolism from the mural aortic thrombosis*. Dual antiplatelet therapy was supplemented by anticoagulation (subcutaneous nadroparin 0.6 ml b.i.d.) and vasodilating infusions (*procaini hydrochloridum* 0.2 % plus *pentoxifyllinum* 100 mg) for 15 days. Oral vasodilator therapy (*naftidrofuryli* 600 mg/day followed by 400 mg/day) together with anticoagulation (subcutaneous nadroparin 0.6 ml b.i.d.)

were continued on an outpatient basis. Resting night pain in both feet disappeared within a week and in six weeks the defects on the toetips healed. On ultrasound examination of the abdominal aorta 4 months after

the initiation of treatment the mural thrombosis was not apparent, hence anticoagulation could be changed to antiplatelet therapy (acetylsalicylic acid 100 mg/day) after 12 months and routine one year surveillance of the abdominal aorta using ultrasound has been started.

3.2. Case 2

A 73-year-old-male presented for medical attention owing to cyanosis and skin defects on his feet. Three months previously resting night pain and cyanotic discoloration of both feet first appeared. A month later, minor skin toetips defects were noticed. The man was a smoker (20 cigarettes a day) and had a history of coronary artery disease with acute myocardial infarction seven years previously, arterial hypertension and hypercholesterolemia. He was not diabetic. The patient was on antiplatelet therapy (acetylsalicylic acid 100 mg/day) for several years.

On physical examination inspection of the abdomen revealed no palpable mass. The toes on both feet were cold, cyanotic and sensitive to palpation. Minor skin necrosis on the tip of the second and third toe on the left foot and the third toe on the right foot was present. The pulses of the femoral, popliteal and tibial arteries were palpable. ABI was 0.95 on the left ankle and 1.0 on the right ankle. DUS revealed no occlusion or hemodynamically significant stenosis of the iliac arteries and the arteries of both legs. An ultrasound examination of the abdomen confirmed the presence of the abdominal aortic aneurysm (AAA) of 40 mm maximal diameter, with an echogenic mural thrombosis up to 14 mm wide (Figure 4). CTA showed dilatation of the subrenal abdominal aorta of 47-51 mm with bland soft sheets and peripheral calcifications (Figure 5).

Oral vasodilator therapy (*naftidrofuryli* 400 mg/day) and anticoagulation (subcutaneous nadroparin 0.8 ml o.d.) were started on an outpatient basis for 3 months together with acetylsalicylic acid 100 mg/day. The patient was then transferred onto oral warfarin while vasodilation and antiplatelet therapy continued. Resting pain and cyanosis in both feet disappeared within two weeks and in eight weeks the acral, skin necroses on the toetips healed. Control ultrasound examination of the abdominal aorta 3 months after the treatment initiation, showed the mural thrombosis was significantly reduced. The combined antithrombotic therapy (warfarin and acetylsalicylic acid) continued for 12 months, when endovascular aneurysm repair (EVAR) was performed.

4. Discussion

Peripheral arterial embolism in patients with an unknown source of embolization is still associated with significant morbidity and mortality. Embolising aortic thrombi are suspected in peripheral or cerebral vascular ischemic events. Clinical manifestations of the thrombosis of



Figure 1: Cyanotic toes with minor acral skin and subungual necroses on both feet. Very dry and peeling skin caused by diabetic neuropathy.

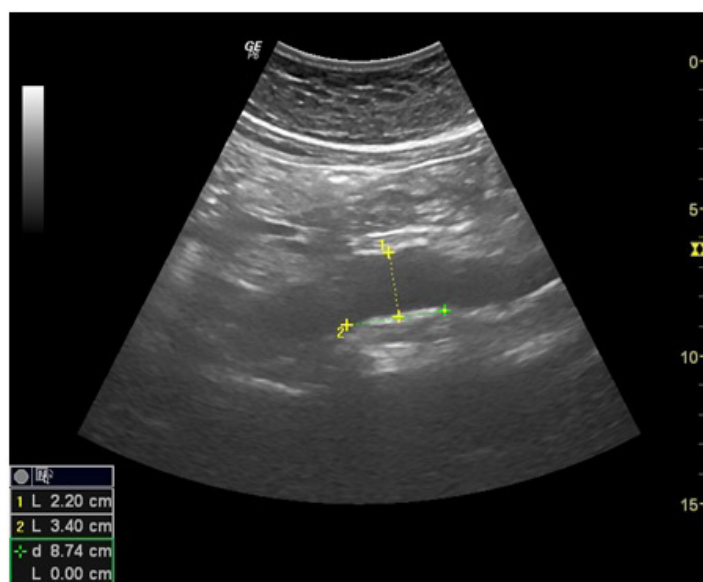


Figure 2: Ultrasound examination of the slightly dilated abdominal aorta with diffuse calcified sclerotic plaques and mural thrombosis.



Figure 3: CT angiography of the mild ectatic subrenal abdominal aorta.



Figure 4: Ultrasongraphy of the abdominal aortic aneurysm of 40 mm maximal diameter, with an echogenic mural thrombosis 14 mm wide.

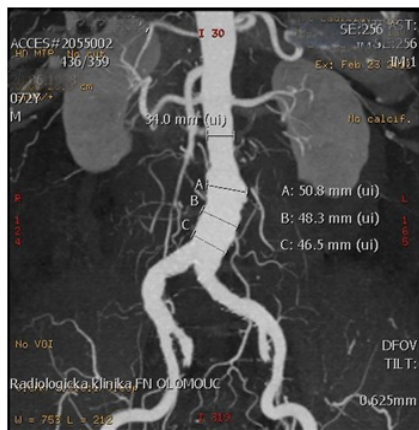


Figure 5: CT angiography of the subrenal abdominal aortic aneurysm.

an abdominal aortic aneurysm are well known and can range from acute interruption of blood flow through the aorta with acute pain, pallor, coldness, paraplegia and absent femoral pulses to critical limb ischemia with resting leg pain and acral skin necrosis. Asymptomatic complete occlusion of an abdominal aortic aneurysm has also been described [5] (Moulakakis, 2010).

A much less common cause of peripheral limb ischemia is mural aortic thrombosis with embolisation. In a group of 89 patients with acute embolic events who had undergone an extensive diagnostic workup, mural aortic thrombi were identified in only eight of them (9 %, median age 63 years, bilateral or repetitive distal embolisations). All patients had several risk factors for atherosclerosis [1] (Reber, 1999).

We have presented here, two cases of mural abdominal aortic thrombosis with repetitive peripheral embolic events in both legs. Patients were over 70 years with serious clinical manifestations of atherosclerosis in other areas and several risk factors for atherosclerosis (dyslipidemia, arterial hypertension, smoking, age). In both cases the symptoms started as the resting and night pain of the acral parts of legs with cyanosis and minor toetips necroses. Bilateral critical limb ischemia with normal ABI values, and without ultrasound identification of any occlusion or hemodynamic significant stenosis in the lower limb arteries, cause us to do an ultrasound examination of the abdominal aorta. Arterial emboli arise from proximal ulcerated plaques in first case in aortic dilatation and in second in AAA in a process called *aorto-arterial embolisation*.

Although arteriogenic macroemboli may pose a threat to foot or limb, microemboli present with more limited pathology such as skin necrosis or digital ischemia. In both cases, the thrombus was present on exulcerated atherosclerotic plaques, in the first case identified as a mobile thrombus, in the second case as an older, echogenic thrombosis. In both cases described, *peripheral arterial embolism led to the bilateral critical limb ischemia (Fontaine gr. IV), digital ischemia and minor, acral, skin necroses (blue toe syndrome).*

The advent of DUS, CTA and magnetic resonance imaging (MRI) has led to identification of mural aortic thrombi as a source of distal embolisation in a much higher proportion of patients than previously. DUS was adequate for the diagnosis in our patients. The CTA only confirmed the ultrasound findings.

All patients with mural aortic thrombosis should be first treated with heparin. In case of failure, thrombectomy may be undertaken in younger

patients. Owing to the highly invasive nature of the procedure, careful work-up including transesophageal echocardiography (TEE) should be performed to rule out any other cause of embolism and to determine if the lesion presents a high potential for embolization. The follow-up must include long-term anticoagulation and routine surveillance using ultrasound (DUS, TEE) or MRI [6] (Choukroun, 2002).

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