SADDLE EMBOLISM OF AORTA IN RHEUMATIC HEART DISEASE

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Abstract:
Rheumatic heart disease is an important cause of systemic arterial embolism. Acute aortic occlusion is an infrequent, but potentially catastrophic condition with a high early mortality rate. It calls for immediate revascularization in order to salvage the limbs and save life. We hereby report a 25 year old female with rheumatic heart disease in atrial fibrillation with thromboembolic occlusion of the infrarenal aorta at the bifurcation, known as a saddle embolus, with a favourable outcome.

Keyword: saddle embolism, aortoiliac occlusion, rheumatic heart disease, atrial fibrillation, aortography, embolectomy, anticoagulant therapy

Introduction:
Systemic arterial embolisations occur commonly in rheumatic heart disease especially mitral stenosis. It has an approximate incidence of 20%. Among them acute aortoiliac occlusion (“saddle embolism”) is one of the rare events which is detrimental to life. Physicians must be sufficiently familiar with the characteristic clinical manifestations of saddle embolus in order to reach the diagnosis promptly, recommend appropriate therapy, and avoid irreversible ischemic injury. Time is of the essence in the successful treatment of patients with this disease1. We hereby present a 25 year old female with acute aortoiliac occlusion.

Case Report
A 25 year old female presented to us with sudden onset of severe bilateral lower limb pain associated with numbness of 1 day duration, followed by difficulty in moving the toes of her left lower limb. She also noticed mild bilateral leg swelling. She gave history of palpitations at rest and breathlessness at rest which was more on lying supine, of 1 day duration. Patient was a known case of rheumatic heart disease with mitral stenosis. She had undergone closed mitral commissurotomy 10 years ago and was on penicillin prophylaxis. She also had a cerebrovascular accident, an infarct involving the left middle cerebral artery 3 years ago. She was advised oral anticoagulation therapy for left atrial appendage clot, however she was non compliant. She had no other comorbidities and had regular menstrual cycles with normal flow, and no habits.
On clinical examination, patient was dyspnoeic at rest, minimally pale. There was no central cyanosis or clubbing or lymphadenopathy. Pulse rate was 110/min, irregularly irregular with an apex pulse deficit of more than ten/min. BP was 110/70 mmHg. On examining both the lower limbs, limbs were pale, cold with cyanotic hue. There was minimal pitting pedal edema. Left foot drop was present. Bilateral femoral, popliteal, posterior tibial and dorsalis pedis pulses were not palpable. On examination of the cardiovascular system, jugular venous pressure was elevated with absent ‘a’ waves. First heart sound was varying in intensity and the pulmonary component of the second heart sound was loud. A mid diastolic murmur and a grade 3/6 pansystolic murmur were heard at the apex, the pansystolic murmur radiated to the axilla. Respiratory system examination revealed bilateral basal fine crackles. On examination of the abdomen, mild congestive hepatomegaly was noted. Central nervous system examination showed residual right hemiparesis with right upper motor neuron facial nerve palsy and left foot drop. Routine investigations revealed mild leukocytosis (WBC-13,100/ cumm) and elevated ESR (30mm/hr). Biochemical investigations were within normal limits. Coagulation parameters were normal (PT-12.4s, aPTT 38s, INR 1.2).

**Fig.1: ECG showing atrial fibrillation**

ECG (fig.1) showed evidence of atrial fibrillation and, chest x-ray showed features of LA enlargement with prominent pulmonary arteries. ECHO revealed features of moderate mitral stenosis (MVA 1.1cm$^2$) with mild mitral regurgitation and pulmonary hypertension with a clot in left atrial appendage. There was no evidence of infective endocarditis. Hand Doppler examination of the lower limbs showed biphasic flow in both popliteal, right posterior tibial and dorsalis pedis arteries; and venous flow in left posterior tibial and dorsalis pedis arteries. 64 slice CT angiogram of abdominal aorta and both lower limb run off (fig.2, fig.3, fig.4) revealed intraluminal acute thrombus within aortic bifurcation, contiguously propagating into right common iliac artery completely occluding and into left common iliac artery narrowing the lumen with another long segment thrombus within left common femoral and superficial femoral arteries. The final diagnosis of rheumatic heart disease, post mitral commissurotomy status, with old MCA infarct, in atrial fibrillation, in cardiac failure, with left atrial appendage clot with acute thromboembolic aortoiliac occlusion ("saddle embolism") with bilateral lower limb ischemia was made.
Fig. 2: Multislice CT angiography of abdominal aorta and both lower limb run off showing aortoiliac occlusion along with another long segment thrombus within left common femoral and superficial femoral arteries.

Fig. 3: Multislice CT abdominal angiography showing intraluminal thrombus within aortic bifurcation contiguously propagating into right common iliac artery completely occluding and into left common iliac artery narrowing the lumen.

Fig. 4: Multislice CT angiography of abdominal aorta and both lower limb run off showing aortoiliac occlusion along with another long segment thrombus within left common femoral and superficial femoral arteries.

Medical management was started with intravenous heparin 5000 units, diuretics, digoxin and antibiotics. Immediate revascularization surgery was done. After anaesthetic fitness, under local anaesthesia, bilateral transfemoral embolectomy was done using Fogarty’s catheter. Intraoperative findings included saddle embolus of the aorta with left femoral thrombus. Post operatively hand Doppler examination revealed triphasic flow in both right and left popliteal, posterior tibial and dorsalis pedis arteries. Patient was continued on heparin which was switched over to oral anticoagulation therapy with nicoumalone sodium 2 mg once daily, monitoring INR. Patient was discharged on tenth day of admission with minimal residual left foot drop and was given foot drop splint. Her INR at the time of discharge was 2.4 and she was advised to comply with oral anticoagulant drug therapy and to serially monitor INR.

Discussion:
System arterial embolism is common in rheumatic heart disease; especially mitral stenosis (in upto 20% cases). This is usually due to blood stasis and thrombus formation in left atrial cavity or appendage. Most often these patients are in atrial fibrillation, however in 20% of cases embolic events occur in sinus rhythm. This may be attributed to the loss of atrial contractile function which favours thrombus formation, despite electrical evidence of sinus rhythm. Infective endocarditis also contributes. The risk of embolism directly correlates with age and the size of left atrium and inversely with cardiac output. Sites of
embolism include cerebral (50%), coronary, renal and peripheral vessels including saddle embolism. Acute aortoiliac occlusion is a life-threatening event. It can be due to embolic occlusion of infrarenal aorta at its bifurcation ("saddle embolus")³, dissection, trauma or acute thrombosis of the abdominal aorta superimposed on atherosclerotic or aneurysmal disease. The origin of embolism is usually from the left side of the heart, typically as a thrombus from the left atrium secondary to atrial fibrillation, particularly in the setting of rheumatic mitral stenosis or from the left ventricle, secondary to myocardial infarction⁴, aneurysm or dilated cardiomyopathy; other sources include prosthetic valve thrombus; infective endocarditis⁵; atrial myxoma⁶; or paradoxical embolism from a right to left intracardiac shunt from a peripheral venous source. Thrombotic occlusion of the aorta most commonly develops at a point of atherosclerotic narrowing (in 75-80%); wherein acute limb ischemia is often prevented by collateral circulation. Symptomatology includes sudden onset severe bilateral lower extremity pain, numbness, paraesthesia and weakness²⁷. Prolonged ischemia may lead to myonecrosis, acute renal failure, acidosis, hyperkalemia and death. Less common presentations include sudden onset bilateral lower extremity weakness, abdominal pain due to mesenteric ischemia, and severe hypertension from renal artery involvement. On examination lower limbs are cold, pale, cyanotic with a mottled appearance; and it may lead to gangrene. Pulses are absent below the abdominal aorta²⁷. Signs of ischemic neuropathy may be present which the physician may mistakenly diagnose as spinal cord infarction or compression. The diagnosis is confirmed by aortography especially to evaluate renal and mesenteric arterial involvement in those with concomitant hypertension, anuria or abdominal pain. Some clinicians advocate that patient should be taken for immediate surgical intervention without angiography to avoid irreversible ischemic damage to the limbs in the event of delay². Management includes initiation of intravenous heparin therapy followed by immediate revascularization surgery. A transfemoral arterial embolectomy using Fogarty balloon tipped catheter under local anaesthesia is the procedure of choice to remove a saddle embolus⁸⁹; if not, a direct transabdominal aortotomy is undertaken. Direct aortic reconstruction or revascularization with aortofemoral or axilofemoral bypass is advocated for thrombotic occlusions. The operative mortality rate is 31-40% in acute cases; more so (85%) in those with severe left ventricular dysfunction or hypercoagulable state. In 98% of cases limb can be salvaged. In order to prevent recurrent emboli, life long anticoagulant therapy must be advocated.

References:


4 Lawrence Gould, Frank Migliorelli. Aortic saddle embolus in acute myocardial infarction: Successful removal by a Fogarty catheter technique.


