SHORT REPORT

Ruptured Mycotic Aneurysm of Peroneal Artery

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A 61-year-old lady, with history of aortic and mitral valve regurgitation developed infective endocarditis due to acinetobacter following tooth extraction. While on antibiotic treatment, she developed pain and swelling of left calf. Duplex scan was done with a suspicion of deep vein thrombosis. It showed a leaking pseudo aneurysm of the peroneal artery, which was confirmed on angiography. Surgical repair of the defect on the peroneal artery with excision of pseudo aneurysm was done. She recovered on continued antibiotic therapy. Isolated pseudo aneurysm of peroneal artery following infective endocarditis is rare and mimics calf vein thrombosis. Surgical repair may give good results.

Keywords: Pseudoaneurysm; Peroneal artery; Infective endocarditis.

Introduction

A mycotic aneurysm by definition is an acquired aneurysm caused by an infection in the vessel wall. These aneurysm have become rare since the advent of antimicrobial therapy and now they are almost exclusively limited to patients who use intravenous drugs.¹ The vast majority of reported cases of mycotic aneurysms involve major axial vessels proximal to popliteal artery. However, there are case reports of mycotic aneurysms at the popliteal artery level and some single reported cases that involve the tibial and peroneal vessels.² We report a case of a ruptured mycotic aneurysm of peroneal artery.

Case Report

A 61-year-old lady, previously diagnosed to have aortic and mitral valve regurgitation was admitted in another hospital with features of infective endocarditis following tooth extraction. A 2D echocardiogram revealed vegetations in the left ventricular outflow track. Her blood culture grew acinetobacter. She was started on crystalline penicillin, ceftriaxone and metrogyl. During the subsequent hospital stay she developed pain and swelling in the left calf. Mild pitting oedema was present over the left leg. Duplex scan of the left lower limb performed for suspected deep vein thrombosis revealed focal aneurysmal dilatation of the peroneal artery measuring 1.5×2 cm² with no evidence of leak. There was no deep vein thrombosis. She was started on antibiotics. However, 48 h later, her pain over the left lower limb increased and the swelling enlarged considerably in size and became pulsatile. Repeat duplex scan revealed ruptured aneurysm of the posterior segment of the peroneal artery with dampened distal flow (Fig. 1) and she was referred to our centre. Peripheral angiogram was done which was suggestive of a saccular pseudoaneursym of 55×35 mm² in size arising from the tibioperoneal trunk bifurcation projecting posteriorly with narrow neck. Peroneal artery filling was antegrade but sluggish. Posterior tibial artery was filling in retrograde through the plantar arch up to the aneurysm. There were focal signs of rupture with secondary sacculation of 50 mm posterior to primary sac (Fig. 2). The leg was viable. She was taken up for emergency surgery. At surgery, there was a leaking aneurysm involving the peroneal artery with a surrounding haematoma. After initial vascular control of suprageniculate popliteal artery, the haematoma was evacuated. This revealed the
defect in the peroneal artery measuring around 1 cm, immediately after its origin from the tibioperoneal trunk. As the peroneal arterial wall itself surprisingly appeared healthy, this was repaired with 6–0 prolene suture. The surrounding muscles, however, appeared nonviable probably due to the compartmental pressure form the haematoma. On table duplex scan following the repair confirmed restoration of flow through the peroneal artery and posterior tibial artery. A conservative debridement of nonviable muscles in the posterior compartment was done. Cultures obtained from evacuated haematoma were negative. Postoperative period was uneventful and she was discharged on 10th postoperative day. She has recovered completely on a 6-week course of antibiotics.

Discussion

In 1885, Sir William Osler described the first infected aneurysm of the aorta complicating bacterial endocarditis. He described that 80% of patients with bacterial endocarditis developed mycotic embolisation with the potential for aneurysm formation in those arteries weakened by the infectious process. Morbidity and mortality related to mycotic aneurysms, has dramatically decreased with advances for antimicrobial therapy. Currently, the incidence of these aneurysms following an episode of endocarditis is quite rare and their location is dependent upon the peripheral lodgement site of mycotic emboli. These peripheral embolic events usually involve the lower extremities with most emboli lodging at the common femoral artery bifurcation. Involvement of the arterial system below the femoral artery level has been described only in case reports. Several pathogenic mechanisms have been proposed to explain the origin of spontaneous mycotic aneurysms. Posttraumatic mycotic aneurysms of the peripheral arteries are more common than the spontaneous variety and currently they represent 80% of all mycotic aneurysms.

In addition to a change in associated morbidity, microbial etiology of mycotic aneurysms has shifted over time. Prior to the widespread use of antibiotics, the involved organisms were typically staphylococci, streptococci and pneumococci. Gram-negative organisms such as Pseudomonas aeruginosa are frequently cultured in addition to Gram-positive cocci. Fungi are also involved in a small but significantly percentage of mycotic aneurysms and despite their infections nature, up to 25% of mycotic aneurysms can be culture negative. The fact that acinetobacter was cultured in this case is quite unusual.

Acinetobacter is a Gram-negative coccobacillus that is a common commensal of the soil, water and sewage. Isolation in the hospital setting usually represents colonization rather than invasive infection. Common sites of acinetobacter infections are the respiratory tract and the urinary tract. Endocarditis and pseudoaneurysm formations due to this organism is rare.

A high index of suspicion and early diagnosis are the most important factors in the successful surgical management of mycotic aneurysms. Clinical presentation usually includes fever and painful pulsatile mass in the vicinity of major blood vessels, a thrill or bruit may be associated. As in our case a duplex scan usually confirms the diagnosis and may also demonstrate leakage from the aneurysm. Angiography is required to plan for surgery.

Management of mycotic aneurysms is less clear because of paucity of cases reported in the literature. Surgical treatment is directed to control hemorrhage. Debridement of all infected tissue and restoration of circulation is the subsequent aim. Hemorrhage can be controlled by clamping the vessel proximally and distally. Debridement should include evacuation of
infected haematoma and excision of all necrotic tissues, which usually includes the arterial wall itself. Restoration of circulation in such cases is typically achieved with bypass grafting using autogenous vein. In our case, as the arterial wall itself was unexpectedly healthy, we did a primary repair of the arterial wall. This has not caused any problem in this patient so far. Endovascular options such as coil embolisations/thrombin glue injection, stenting appear attractive alternatives to surgery. We did not consider them in our case, as the patient had signs of compartmental compression due to the large calf haematoma. Additionally, the evidence of such interventions in mycotic aneurysm is limited.

**Conclusion**

Ruptured mycotic aneurysm of peroneal artery arising from bacterial endocarditis are rare. High index of suspicion is crucial in early surgical management.

**References**


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