CASE REPORT

Ruptured Popliteal Artery Aneurysm

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ABSTRACT

Popliteal aneurysms commonly present with acute limb ischemia or symptoms due to compression of adjacent structures. Ruptured popliteal aneurysms are uncommon due to its superficial location leading to early diagnosis, before it reaches huge size, vulnerable for rupture. Unless there is a high index of suspicion, ruptured popliteal aneurysms may masquerade as deep vein thrombosis; sometimes a concomitant presentation due to compression of the popliteal vein. We describe a case of ruptured popliteal artery aneurysm with deep vein thrombosis and how expeditious management led to good outcome.
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INTRODUCTION

Popliteal aneurysms classically manifest in one of several ways. Approximately 37% of the aneurysms are asymptomatic and are detected by palpation or ultrasonography. About half the patients present acutely with limb ischemia from thrombosis (approximately 32%) or embolization (approximately 20%). Less commonly they present with symptoms arising from compression of adjacent structures (approximately 10%), with deep vein thrombosis or associated neurological symptoms (1,2). By contrast, rupture is rare. In the 16 series encompassing a total of 1910 aneurysms, only 40 ruptures were reported, yielding an overall incidence of 2.1% (3).

We describe an interesting case of an elderly man who presented with severe leg swelling and was diagnosed as femoro-popliteal deep vein thrombosis by the general practitioner. A missed diagnosis resulted in delay in proper management. A high index of suspicion led to CT arteriography and clear demonstration of ruptured popliteal artery aneurysm.

CASE REPORT

A 70-year-old male developed a sudden onset of pain and swelling over the right knee after a seemingly trivial non vigorous exercise. Apparently he went for a long walk in the park and while he rested and stretched his leg, he experienced sudden onset of severe pain behind the knee, like 'something broke'. He had thought it was due to a stretched muscle and managed himself with elevation and cold compresses. Over a period of ten days, the swelling increased in size and make walking difficult and he began to have fever. He consulted a general practitioner who made a diagnosis of deep vein thrombosis. This was supported by an elevated D-dimer. The patient had history of hypertension, diabetes and hyperlipidemia. He was on Telmisartan 20mg od, Amiodarone 200mg od, Linagliptin 2.5 mg plus metformin 500mg bd, Gliclazide MR 30mg od.

He was not on any antiplatelets or anticoagulation. When he was examined at the emergency room, he was on a wheelchair and was unable to walk. His PR was 90 per minute, in sinus rhythm and blood pressure was 130/90 mmHg. He was having a low grade temperature of 37.5 degree Celcius. His leg was in 30-degree flexion deformity (Figure 1). The right knee was swollen and tense. The swelling was more pronounced over the medial aspect of the knee. The swelling was not pulsatile. The calf was also swollen, tense and tender. The popliteal pulse and foot pulses were absent.

Our clinical impression at that time was an infected haematoma of the popliteal fossa, either due to muscle tear or a ruptured aneurysm with popliteal vein thrombosis.

His Hb was 14.3 g/dL, Tw was 8.5x 103 uL, Hb A1c was 5.6%.C-Reactive protein was 215 mg/L, ESR was 105mm/H

Mass CKMB was 7.2 ug/L. The renal profile, Liver

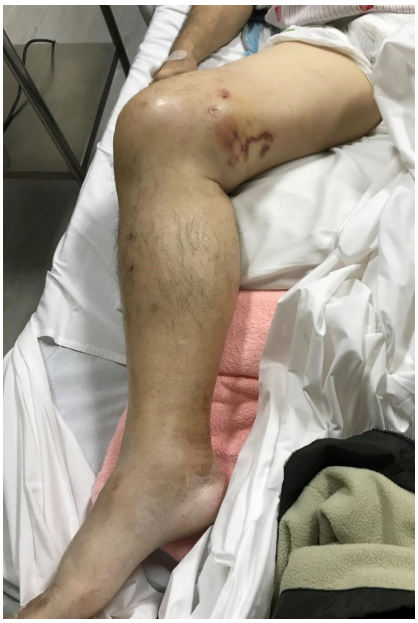

Figure 1: The swelling at the right knee and calf with ecchymosis over the skin due to hot compresses. The knee was in 30-degree flexion deformity

Function Tests, and Thyroid Function Tests were normal. The blood culture did not show any growth.

Duplex scan showed a large haematoma at the popliteal fossa with compression of the Popliteal vein, giving rise to Popliteal vein and Superficial Femoral vein (SFV). CT arteriography confirmed aneurysm of the Popliteal artery measuring 2.5-cm and likely an area had ruptured giving rise to the large haematoma (Figure 2 and 3). The Popliteal and SFV were thrombosed. The contralateral popliteal artery showed irregular wall and presence of a small aneurysm.

The patient was admitted to the ward and was started on IV Ceftriaxone 2 g stat and 12 hourly. He was posted for surgery under general anaesthesia- for evacuation of clot, excision of the popliteal artery and a Femoro-Popliteal bypass using contralateral great saphenous vein.

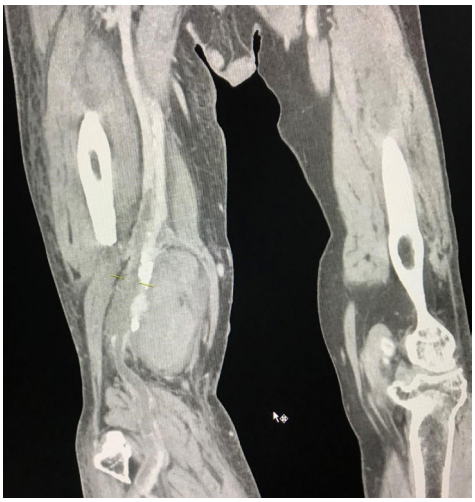

Figure 3: Reconstructed view of the right popliteal artery and popliteal vein. The thrombus is seen within the popliteal vein extending upwards to the superficial femoral vein (blue arrow) due to compression by the haematoma (red arrow)

Intraoperatively, after proximal control of the superficial femoral artery at the adductor canal, nearly 1 litre of clot was evacuated. The aneurysmal Popliteal artery was isolated and a tear was noted in the atherosclerotic Popliteal artery (Figure 4). This segment of the artery was ligated and excised. GSV from the contralateral thigh was harvested and was used as a conduit for bypass. The infected haematoma cavity was drained. Post operatively, the foot pulses returned. The knee flexion and extension improved.

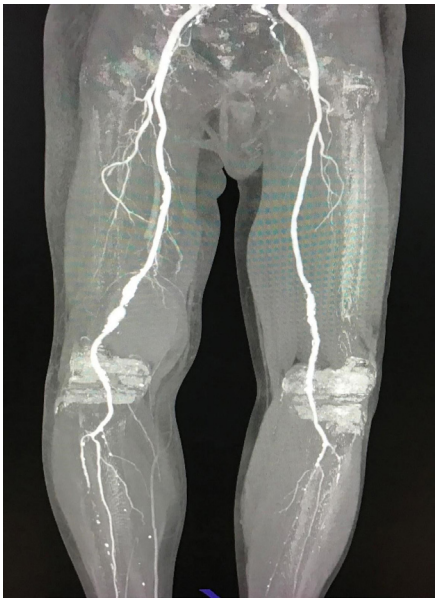

Figure 2: The reconstructed CTA showing right Popliteal artery aaneurysm and a large soft tissue haematoma. The contralateral popliteal artery also showed irregularity in the wall

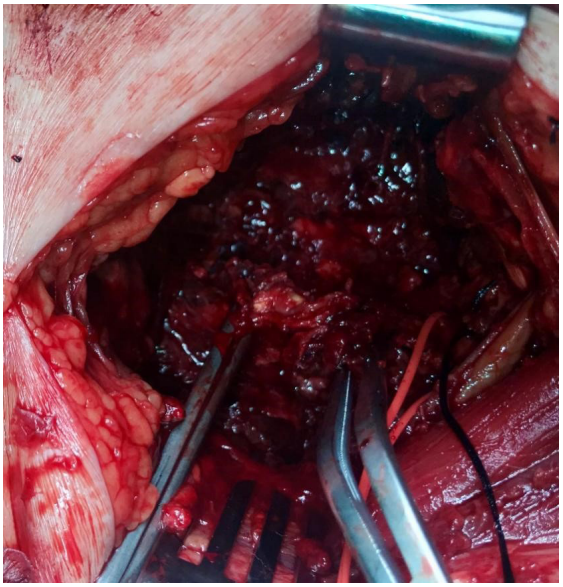

Figure 4: Popliteal artery isolated in the 'sea of haematoma'. The hole (blue arrow) was identified, and the proximal and distal end was ligated close to the GSV bypass site

The histopathology confirmed atherosclerotic Popliteal artery with a thinned out wall and fibrin clots. The tissues that were sent for culture grew gram negative rods, suggestive of Salmonella species (non-typhoid) and was sensitive to Ceftriaxone, Ciprofloxacin, Ampicillin, Trimethoprim-Sulfamethoxazole.

The Ceftriaxone was continued at 1 g twice a day for a week and subsequently switched to oral Ciprobay 500mg bd for a period of 6 weeks. After evacuation of haematoma, he was started on SC Clexane 60mg bd and the anticoagulation was continued for a period of 6 months using Apixaban 5mg bd.

The patient was followed up for 6 months. He has resumed his regular walks in the park and is able to bend his knees and squat. His anticoagulation was stopped after 6 months and he was switched to oral Aspirin, alongside his regular medications.

DISCUSSION

The prevalence of popliteal artery aneurysms (PAAs) is approximately 1% in men aged 65 to 80 years old but accounts more than 80% of all peripheral aneurysms. (4) Most of them are athero-sclerotic in origin, other causes include infection, trauma, or those associated with Marfan's and Behcet's disease. Rupture of popliteal artery aneurysm is extremely rare. The reason why rupture is rare is unknown. It could be early diagnosis due to the superficial location of popliteal artery or the limited space in the popliteal fossa to allow the aneurysm to expand to enormous size with risk of rupture.

In this patient, diagnosis was delayed due to the late presentation and the misdiagnosis of deep vein thrombosis. Over-reliance on D-Dimer in a patient with leg swelling, can lead to a wrong diagnosis. This patient did have deep vein thrombosis but unless the underlying cause of ruptured popliteal artery aneurysm is managed, the deep vein thrombosis will not improve or resolve.

Ruptured PAAs carries a high morbidity and mortality and is challenging to manage. Factors like patient's frailty, comorbidities and good vessel run off will affected the treatment decision. Although endovascular repair is both safe and effective, open surgical repair still remains a treatment of choice as it improves long-term outcome for PAAs (5). There is little evidence to prove which technique of repair has the best outcome due to the small number of reported cases with relatively short-term outcome data (5). Early screening and routine monitoring might reduce incidence of ruptured PAAs and early planning for surgery has a good outcome.

In our patient, the haematoma grew Salmonella species, which suggest a differential diagnosis of ruptured mycotic aneurysm of the popliteal artery. However, the insidious onset, presence of atherosclerotic changes of popliteal artery with thinned out wall on histology and the presence of contralateral popliteal aneurysm, were suggestive of ruptured atherosclerotic popliteal artery aneurysm. Ligation of the ruptured segment and a saphenous vein bypass resulted in a good outcome. He has been followed up for 6 months and is ambulant and has returned to his normal activities.

CONCLUSION

Popliteal artery aneurysms rarely present as rupture. There may be a concomitant deep vein thrombosis due to a tense haematoma compressing on the popliteal vein. Early diagnosis with expeditious repair results in good outcome.

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