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Gurusamy Kapilan
Senior Registrar in Vascular
and Transplant Surgery,
National Hospital of Sri Lanka,
Colombo, Sri Lanka

Sanjaya Iddamalgod
Registrar in Vascular and
Transplant Surgery, National
Hospital of Sri Lanka,
Colombo, Sri Lanka

Joel Arudchelvam
Consultant Vascular and
Transplant Surgeon, National
Hospital of Sri Lanka,
Colombo, Sri Lanka

Corresponding Author:
Joel Arudchelvam
Consultant Vascular and
Transplant Surgeon, National
Hospital of Sri Lanka,
Colombo, Sri Lanka

Ruptured mycotic abdominal aortic aneurysm presenting with spinal cord ischaemia: A very rare presentation

Gurusamy Kapilan, Sanjaya Iddamalgod and Joel Arudchelvam

Abstract

Mycotic abdominal aortic aneurysms (AAA) are rare. This case report describes a 59 year old female who presented with a history of sudden onset abdominal pain and bilateral lower limb paralysis. She presented after 24 hours of the onset of symptoms. Bilateral distal lower limb pulses were absent. Computerized tomographic (CT) scan of abdomen showed a leaking, saccular, juxtarenal AAA. There was a thin layer of thrombus on the posterior wall of the aneurysm. The lumbar arteries were not visualised, indicating that they were thrombosed. On bilateral fasciotomy of the legs all muscles were viable. Therefore acute lower limb paralysis due to leaking AAA and thrombosis of the spinal arteries leading to spinal cord ischaemia was clinically diagnosed. Patient underwent repair of the AAA with antibiotic coated tube polyester graft. Salmonella was isolated from the aortic wall tissue. Patient was treated with prolonged course of antibiotics. Hemo-dynamically she improved but her lower limb muscle and sensory functions did not improve.

Keywords: Mycotic aneurysm, abdominal aortic aneurysm, ruptured aneurysm, spinal cord ischemia, spinal artery thrombosis.

Introduction

Mycotic Aortic Aneurysm (MAA) arises as a result of infection of the aortic wall. It is rare, occurring in about 0.6% of all aortic aneurysms [1]. Thrombosis of Abdominal Aortic Aneurysms (AAA) is also a rare event [2]. Further spinal cord ischemia and paralysis following rupture and wall (and spinal artery) thrombosis of AAA is a very rare event. This case report describes a very rare presentation of ruptured mycotic AAA with wall thrombosis and spinal cord ischemia.

Case

59 year old female presented with a history of sudden onset bilateral lower limb paralysis for one day duration. She had a history of moderate back pain for two weeks but she did not have fever. On the day of developing paralysis she also developed abdominal pain, but she did not have faintishness. She was a patient with type 2 Diabetes mellitus and hypertension. She was not known to have AAA previously. On examination bilateral lower limb pulses were absent and bilateral feet were cold. On abdominal examination, there was a tender pulsatile mass in the epigastric region. There were absent movements and impaired sensations of both lower limbs. The sensory level was at the 3rd lumbar (L3) level and the motor level was at the 4th lumbar level (L4). Patient was haemodynamically stable. But the haemoglobin level was 5 g/dl and the C- reactive protein level was 122 mg/l. An initial clinical diagnosis of leaking AAA with thrombosis of the lower aorta resulting in acute lower limb ischaemia was made. Computerized tomography scan (CT) of the abdomen showed a juxta renal saccular AAA with active leak in the retroperitoneal area with haematoma extending posterior to the right kidney. (Fig 2).

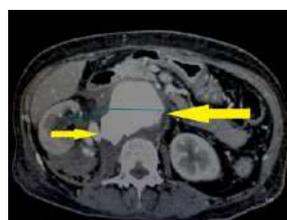


Fig 1: Image showing mycotic aneurysm sac with thickened wall and the area of rupture and leak

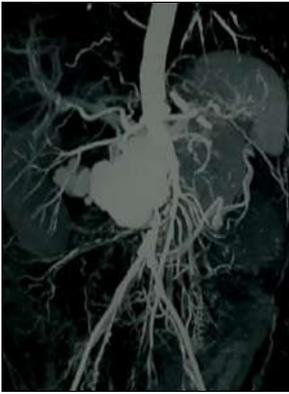


Fig 2: Reconstructed image showing juxta renal mycotic aneurysm

There was thickening of the aneurysm wall. The maximum diameter of the aneurysm was 71.2 mm. Occlusion of the lower end of the aorta was also demonstrated. There was also a layer of thrombus on the posterior wall of the aneurysm sac, the lumbar arteries posterior to the aneurysm was not patent. An immediate fasciotomy of the legs and laparotomy was planned. On fasciotomy all four compartments were found to be viable. On laparotomy an AAA with thick inflamed tissues around was found. (Fig 3).

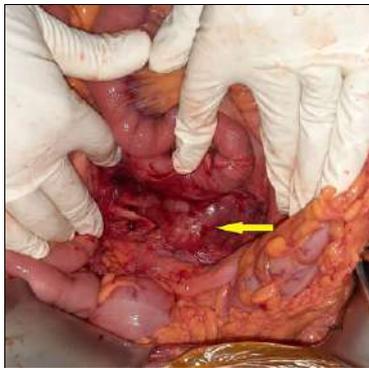


Fig 3: Intra operative image showing mycotic aneurysm sac with inflamed wall

Retroperitoneal haematoma was found. The duodenum, root of the mesentery and the left renal vein were adhered to the sac. Supra-coeliac aorta was mobilised to achieve proximal control temporarily. Root of the mesentery was partially mobilised. Mobilisation of the duodenum was not possible due to the adhesions. Supra-coeliac clamp was applied. Then left renal vein was divided distal to the suprarenal and gonadal vein junction. Aneurysm neck at the juxtarenal level was dissected. Then a suprarenal clamp was applied and the supra-coeliac clamp was released. The aneurysm sac was opened. The infected thrombus was removed. No bleeding from the lumbar artery orifices was noted. The duodenum was mobilised away with part of the aneurysm wall. Renal arteries were controlled by Fogarty balloon inflation inside the lumen of the arteries. A 16mm tube antibiotic soaked polyester graft was used for repair. *Salmonella Enteritidis* species was isolated from the aortic wall tissue. Patient had oliguria on postoperative day 0 and day 1. Subsequently urine output gradually improved. She was on inotropes until postoperative day 2. Subsequently her haemodynamic functions improved. Distal pulses appeared on both lower limbs, but the motor and sensory functions did not improve.

Discussion and Conclusion

MAA is caused by infection of the diseased aortic wall (atherosclerosis or an already existing aneurysm) resulting in degradation of the wall and aneurysm formation. Mycotic aneurysms are saccular in shape. Infection results from septic emboli from infective endocarditis and other causes of bacteraemia.

Common bacteria associated with MAA are salmonella species, *Staphylococcus*, *Streptococcus* and *Klebsiella* [3]. *Salmonella Enteritidis* was isolated from the aneurysm sac of the current patient. *Salmonella* species bacteria are gram negative and anaerobic. *Salmonella Enteritidis* is acquired through contaminated food mainly poultry products and meat. Patients develop fever, diarrhoea and abdominal pain which usually settle spontaneously. Bacteraemia can occur in 3% to 8% of patients resulting in localisation of the organisms in various parts of the body e.g. heart, gallbladder, kidneys and atherosclerotic plaques. Infection of the atherosclerotic plaques in the aortic wall results in disruption of the wall and aneurysm formation.

This patient presented with sudden onset bilateral lower limb weakness, with bilateral absent distal pulses suggesting an acute limb ischemia. But fasciotomy revealed that all the muscles were viable in both legs despite more than 24 hours of ischaemia. Therefore a clinical diagnosis of bilateral lower limb paralysis due to spinal cord ischemia as a result of thrombosis of the aneurysm wall with thrombosis of the spinal arteries was made. CT scan also showed a thin layer of thrombus on the posterior wall of the aneurysm and the lumbar arteries were not visualised confirming the thrombosis of the lumbar arteries.

Spinal cord has a segmental blood supply. It has one anterior spinal artery (ASA) and two posterior spinal arteries (PSA). All of the spinal arteries originate from the vertebral artery. The ASA supplies the anterior 2/3 of the spinal cord. These spinal arteries (ASA and PSA) anastomose with the branches of the segmental spinal arteries (SPA) arising at regular intervals from the neck vessels and the aorta. These segmental spinal arteries enter the spinal canal through the intervertebral foramen along the spinal nerve roots. And then SPA divides into an Anterior Radiculomedullary Artery (ARA) and a Posterior Radiculomedullary Artery (PRA). ARA anastomoses with the ASA thus contributing to the blood supply of the anterior 2/3 of the spinal cord. PRA anastomose with the PSA. Even though the SPA arises at regular intervals, only few of them are a large and dominant to supply a significant portion of the spinal cord [4]. One such SPA is the artery of Adamkiewicz. This usually arises from the aorta at the level of T9 - L2 often from the left side. But this level can vary. Occlusion to this artery during aortic procedures is known to cause spinal cord ischemia and lower limb paralysis. Also the aneurysm of this patient started at first lumbar vertebral (L1) level. Therefore it is possible that the artery of Adamkiewicz was thrombosed. But thrombosis of the AAA is a rare event, spinal cord ischemia as a result thrombosis of the AAA and lumbar arteries is even rarer presentation [2]. The annual rupture rate of AAA of more than 7cm is 6.3% - 22.3% [5-6]. Biomechanical studies have found that the wall stress on the saccular aneurysm is greater than the fusiform aneurysm [7]. Therefore the rupture rate of saccular aneurysm is greater than that of fusiform aneurysm. Therefore repair of the saccular aneurysm is recommended

for any size In addition infected aneurysms are more prone to rupture than other aneurysms [8].

Standard treatment for MAA is ligation of the aorta and excision of the aneurysm sac and an extra anatomical axillo bifemoral bypass. But other methods like antibiotic coated graft replacement like in the above described case, biological grafts, autologous femoral vein grafts are also described [9]. At present endovascular aneurysm repair (EVAR) is also described [10]. Antibiotic treatment is given for a prolonged period for salmonella infection e.g. at least 6 weeks.

Therefore this case report describes a very rare presentation of ruptured MAA and aneurysm wall (and spinal artery) thrombosis resulting in acute spinal cord ischemia leading to bilateral lower limb paralysis. However exclusion of acute lower limb ischemia in such cases by performing urgent fasciotomy and confirmation of the AAA leak and wall thrombosis with urgent CT scan are very important in the management of such patients.

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