Giant splenic artery mycotic aneurysm presenting with massive hematemesis

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We report a 40-year-old man with rheumatic heart disease who presented with abdominal pain for three weeks and hematemesis for 24 hours. CT scan showed a large splenic artery aneurysm without evidence of pancreatitis. Mycotic aneurysm due to infective endocarditis was considered and confirmed by echocardiogram, which showed aortic and mitral valve regurgitation and vegetations. He was managed successfully with coil embolization of the aneurysm and antibiotics. [Indian J Gastroenterol 2003;22:147-148]

Key words: Infective endocarditis

Mycotic aneurysms are uncommon complications of infective endocarditis (IE), occurring in 2%-10% of cases. They result from septic embolization of vegetations to the arterial vasa vasorum or the intraluminal space with subsequent spread of infection through the intima and outward through the vessel wall. They occur frequently in intracranial arteries, followed by visceral arteries and arteries of the upper and lower extremities.1

A 40-year-old man, non-addict, presented with low-grade fever for three months, episodes of severe left upper quadrant abdominal pain and back pain for three weeks, and two episodes of massive hematemesis over 24 hours. He was earlier diagnosed to have rheumatic heart disease, but had discontinued anti-rheumatic prophylaxis. He was given a course of antibiotics after negative blood cultures for his fever, without any avail. On examination, he was pale and there was a pulsatile mass in the epigastric and left hypochondrial region, around 7 cm in diameter.

CT scan done earlier had shown a large splenic artery aneurysm. Emergency angiogram showed an aneurysm from the splenic artery measuring 4 cm in size and approximately 4 cm from the origin of the splenic artery (Fig). The distal splenic artery was re-formed through collaterals from the superior mesenteric, left gastric and hepatic arteries with no direct continuity beyond the aneurysm. The splenic artery proximal to the aneurysm was embolized using coils and no filling of the aneurysm was seen on check angiogram.

The next day gastroscopy showed no fresh bleed, and CT angiogram showed a 10-cm aneurysm without any contrast filling. CT also showed splenic infarcts, but no evidence of periarterial inflammatory disease. The disparity in the size of the aneurysm between CT and angiogram was due to a thrombus partially filling the aneurysm.

Transabdominal and transesophageal echocardiogram revealed vegetations in the aortic valve with aortic and mitral valve regurgitation, confirming the diagnosis of IE. Though his blood cultures were negative, he became afebrile on antibiotics within three weeks and hematemesis did not recur. On the basis of thromboembolic phenomena and absence of pancreatitis, a diagnosis of mycotic aneurysm was made.

Splenectomy aneurysms are the most common visceral artery aneurysms, accounting for 60% of cases.
The causes are arterial fibrodysplasia, multiple pregnancies, penetrating trauma, septic emboli, and perianterial inflammatory diseases like chronic pancreatitis. Less than 10% are caused by infective endocarditis. The importance lies in their potential for rupture or erosion into an adjacent viscus, leading to life-threatening hemorrhage.

Development of myotic aneurysm in patients with IE has been associated with high mortality, with surgery as the only option in the past. Recently, endovascular treatment has been established as a safe and feasible alternative to surgery, and is the choice of treatment in the management of visceral artery aneurysms irrespective of etiology and location. Tihansky et al. reported successful transcatheter embolization of multiple myotic splenic artery aneurysms. There are only six published case reports of myotic aneurysm of splenic artery, of which only one has been managed with transcatheter embolization.

References

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Received January 23, 2003. Accepted February 16, 2003

Superior mesenteric artery aneurysm presenting with epigastric pain and raised platelet count

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A 90-year-old man was transferred to a district general hospital for recuperation following heart failure. He had a past history of myocardial infarction, atrial fibrillation, congestive heart failure, epilepsy and colonic polyps.

On initial investigations he was found to have normocytic anemia with hemoglobin 9.8 g/dL, MCV 98, urea 14 mmol/L, creatinine 161 µmol/L and ESR 120. He had low serum iron content with raised platelet count of 760,000/mm³. He had increased gamma globulin fraction on serum electrophoresis and weak Bence Jones protein in urine. Multiple myeloma was ruled out by normal bone marrow examination.

Ultrasonography revealed a simple right renal cyst. Echocardiogram revealed moderately impaired left ventricular function but no evidence of vegetation. A few days after admission he complained of post-prandial epigastric pain, but abdominal examination was normal. Considering the raised platelet count and past history of colonic polyps, but no evidence of infective or inflammatory process, gastroscopy and colonoscopy were requested to rule out gastrointestinal bleeding.

He became acutely ill with acute abdomen about 2 weeks after admission while waiting for the investigations. On examination he was tachycardic, hypotensive, pale and sweaty. Examination revealed a distended abdomen with guarding and rigidity and absent bowel sounds. Hernial orifices were normal. An initial diagnosis of perforated viscus was made though an erect chest X-ray revealed no free gas under the diaphragm.

Hemoglobin dropped from 11 g/dL to 7 g/dL overnight. A diagnosis of intra-abdominal bleeding was made and he was transfused, but in view of his age and poor prognosis he was treated conservatively. He died in a few hours.

Post-mortem examination revealed a large hemorrhagic mass in the mesentery of size 8 cm x 5 cm x 4 cm. A large amount of partially clotted blood was present in the abdominal cavity. Histological examination of the mass showed ruptured superior mesenteric artery aneurysm.

The incidence of superior mesenteric artery aneurysms is approximately 1 in 12,000 in multiple necropsy series. They are difficult to detect when the patient is asymptomatic. By the time they bleed and present as acute abdomen it is sometimes too late.

Causes include mycosis (30%-60%), atherosclerosis (20%), connective tissue disease, congenital fibromuscular dysplasia, cystic medial necrosis, trauma and idiopathic. Myotic aneurysms are related to intravenous drug abuse or subacute bacterial endocarditis and are usually caused by non-hemolytic streptococci.

Clinical presentations may include abdominal pain, palpable abdominal mass, audible bruit, cardiac murmur with fever, weight loss, or acute abdomen with evidence of intra-abdominal bleeding. Complications include rupture, thrombosis, dissection with occlusion, or superior mesenteric artery-to-portal vein fistula. Diagnosis is based on clinical suspicion and imaging techniques including Doppler ultrasonography, CT scan and angiography (with or without CT or MR guidance).

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