Obstructive Jaundice: an Unusual Presentation of a Superior Mesenteric Artery Aneurysm

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Superior mesenteric artery (SMA) aneurysms are rare, comprising only 8% of all splanchnic artery aneurysms and most of them have been reported in the surgical literature.1,2 These aneurysms are usually asymptomatic and prone to rupture. We present a case of an atherosclerotic superior mesenteric artery aneurysm causing obstruction of the distal common bile duct, resulting in obstructive jaundice.

Case Report

A 47-year-old man was admitted with intermittent episodes of jaundice over a period of two months and pain in the right hypochondrium for the past two weeks. His biochemical assays showed a direct bilirubin of 15.01 mg%, an SGPT of 200 IU/L and alkaline phosphatase to be 758 IU/L. His initial ultrasound showed a possible lesion in the region of the head of pancreas and a diagnosis of carcinoma of the head of pancreas was given. An ERCP was performed that showed an abrupt cut-off at the level of the distal common bile duct and a stent was introduced into the CBD bypassing the obstruction. Since the radiographic appearances of the CBD on ERCP were not those commonly seen in association with a carcinoma of the head of pancreas and the gastroenterologist asked for a CT scan to be performed on the patient. The spiral (helical) CT scan showed the presence of a large aneurysm arising from the trunk of the superior mesenteric artery. The aneurysm had a broad neck along the right lateral part of the trunk of the superior mesenteric artery. Its thrombosed portion was extending up to the second part of the duodenum and compressing the distal common bile duct. The aneurysm measured 7.2 x 6.1 centimeters, the aneurysmal sac measured 3.1 x 3.0 centimeters while the thrombosed portion measured 4.1 x 3.1 centimeters. Extensive mural calcification was also seen along the whole length of the abdominal aorta. A digital subtraction angiography was performed on this patient. The celiac arteriogram showed the common hepatic, left hepatic and the left gastric arteries to be normal. The right hepatic artery was not opacified. The gastroduodenal artery was opacified up to its proximal extent. The gastroduodenal artery was then selectively catheterized which showed it to be slightly narrowed due to an extrinsic mass effect. The catheter was then introduced into the superior mesenteric artery and a large bilobed aneurysm was seen arising from the trunk of the main artery. A replaced right hepatic artery was also visualized and was seen to arise just proximal to the neck of the aneurysm. The inferior mesenteric artery was occluded. The whole length of the abdominal aorta was also irregular and the lumen of the aorta distal to the superior mesenteric artery was found to be narrowed. Twelve hours post angiography the patient developed a leak in the aneurysm and the patient died before he could be taken to the operating theater.

Discussion

Aneurysms of the superior mesenteric artery are very rare and an uncommon finding, having an incidence of one in 12,000 autopsies performed.3 The majority of superior mesenteric artery aneurysms are mycotic in origin, other causes implicated include atherosclerosis, inflammatory as in pancreatitis, collagen vascular disease, aortic dissection and trauma. Very rarely are they congenital in origin. The diagnosis of an SMA aneurysms is usually on clinical assessment or they may be an incidental finding as was the case in our patient. It can be diagnosed with confidence on gray scale and color Doppler sonography, showing the presence of color flow within the true lumen of the aneurysm while the thrombosed segment appears as a hypoechoic area in close proximity to the color flow visualized on the Doppler imaging.4 Computed tomography has been shown to play a major role in the evaluation and confirmation of SMA aneurysms.5,6 The introduction of helical computed tomography using power injectors for the delivery of contrast has been made it possible to view the presence of contrast in the arterial and venous phase within the splanchnic vascular system. This enables visualization of the contrast distended vessel and any other associated abnormality. CT is thus able to provide excellent anatomic detail of the vessel involved and its surrounding structures.

Although the diagnostic capabilities of detection of vascular lesions on helical computed tomography are excellent, arteriography still remains the final imaging method used in the diagnosis and evaluation of SMA aneurysms since it alone can provide proper information for further surgical management.7 The successful treatment of SMA aneurysms has always been surgical, involving a broad range of managements including vessel ligation with or without excision, revascularisation with primary anastomosis and obliterative aneurysemorrhapy. The future holds even exciting prospects of treatment including transluminally placed endovascular grafts with the combined efforts of the vascular surgeon and interventional radiologist and placement of covered stents.8
Motor Neuronopathy associated with Adenocarcinoma of Esophagus

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Neurologic paraneoplastic syndromes (NPS) are remote neurologic effects, except metastasis, of systemic cancers. These are a rare group of disorders, commonly associated with small cell lung carcinoma (SCLC). Various NPS have been described, including motor neuronopathy. The motor neuronopathy has commonly been described with small cell carcinoma of lung, breast cancer and lymphoproliferative disorders. We report a case of motor neuronopathy in association with large cell adenocarcinoma of esophagus. To our knowledge this is the first reported case of motor neuronopathy associated with adenocarcinoma of the esophagus.

Case Report

A 57-year old man, smoker, presented with a 2-months history of progressive walking difficulty. He was emaciated and his neurological examination was significant for absent gag reflex, mild proximal limb weakness (power of 4/5 on MRC scale), impaired proprioception and ataxic gait. Chest, abdominal and cardiovascular examination was normal. There was no lymphadenopathy. His complete blood counts, serum electrolytes, liver function tests, CPK were normal. Motor nerve conduction studies of right median, ulnar, posterior tibial and peroneal nerves revealed mildly slow conduction velocities. However, there was no conduction block. Sensory nerve conduction studies of bilateral sural, right median and right ulnar nerves were normal. The EMG findings were suggestive of anterior horn cell disorder (motor neuronopathy). He developed complaints of dysphagia. An upper GI endoscopy revealed a fungating and ulcerated growth at distal esophagus, confirmed on histopathology to be large cell adenocarcinoma. Later patient developed mental status changes and blindness. MRI brain showed multiple hemorrhagic metastasis to bone (sternum). An esophageal stent was placed and patient was managed conservatively. Later he developed recurrent pneumonia and died two months after the initial presentation.

Discussion

Neurologic paraneoplastic syndrome was first described by Oppenhiem in 1888. The exact incidence of neurologic paraneoplastic syndrome is not known, however incidence in patients with SCLC, the most commonly associated tumor with the syndromes, is less than 3%. The mechanism of development of neurologic paraneoplastic syndrome is not fully understood; however, there is evidence that this is an autoimmune phenomenon. In addition to autoimmunity, role of opportunistic viral infections has been proposed as underlying mechanism for motor neuropathy. The paraneoplastic syndrome can effect any part of nervous system including spinal cord/anterior horn cells. The paraneoplastic motor neuron disease (motor neuronopathy) has commonly been reported in association with SCLC, breast cancer and lymphoproliferative disorders. To our knowledge there are only two reports of motor neuronopathy in association with esophageal cancers and none of these was adenocarcinoma. We reported motor neuronopathy in association of large cell esophageal adenocarcinoma. EMG findings of our patient are suggestive of anterior horn cell disorder/motor neuronopathy. Our patient did have evidence of reinnervation in three body regions and denervation in only two regions. Furthermore, normal sensory nerve conduction studies, normal CMAP amplitudes, normal distal motor latencies and widespread reinnervating motor unit potentials and patchy active denervation suggest anterior horn cell disorder. Mildly slow conduction velocities could also be seen in axonal neuropathy and multifocal motor neuropathy with conduction block (MMNCB) but preserved compound muscle action potential and absence of conduction block makes these diagnoses unlikely in our

References