Case Report

Gastrosplenic Fistula and Coeliac Artery Occlusion

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ABSTRACT

Gastrosplenic fistula (GSF) is a very rare complication of several disease processes and can lead to catastrophic bleeding, necessitating emergent treatment. Splenic or gastric lymphomas are the predominant causes, with trauma and gastric surgery also implicated in several case reports. We present a case of a gastrosplenic fistula resulting from occlusion of the coeliac artery. To our knowledge, this is the first reported case of a GSF resulting from severe intra-abdominal arterial disease.

A 60-year-old male initially presented to the emergency department with epigastric pain. He had an extensive medical history, including dialysis-dependent end-stage renal failure, atrial fibrillation, coronary artery disease, and multiple previous abdominal surgeries. Investigation with CT angiography revealed calcified occlusion of the coeliac artery as well as extensive calcification throughout his aorta and arterial tree. A diagnosis of mesenteric angina was made, but due to his poor functional status, he was not suitable for surgical or transcatheter interventions. He was treated symptomatically, but a month later developed sudden worsening of his epigastric pain, followed by large volume haematemesis. CT angiography showed a GSF with extensive gastric necrosis. Due to his poor functional status and rapid deterioration, he opted for palliation and passed away two days later.

It has been postulated that GSF develops from the invasion of malignant tissue from the stomach to the spleen or vice versa, and subsequent necrosis of this tissue results in fistula formation. This case demonstrates that the invasion of an adjacent organ may not be necessary; necrosis itself can cause erosion that ultimately results in fistula formation.

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Background

Gastrosplenic fistula (GSF) is an abnormal communication between the stomach lumen and the splenic parenchyma. It was first described by De Scoville et al. in 1967 [1]. It is extremely rare and commonly associated with complications of several disease processes. It is most commonly described to be related to lymphoma, particularly diffuse large B-cell lymphoma (DLBCL). Other aetiologies such as splenic abscess, trauma, bariatric surgery and Crohn’s disease have been reported to cause GSF [2-4]. We present a case of a gastrosplenic fistula resulting from occlusion of the coeliac artery. To our knowledge, this is the first reported case of a GSF resulting from severe intra-abdominal arterial disease.

Case Report

A 60-year-old male initially presented to the emergency department with sudden onset epigastric pain. He had an extensive medical history, including dialysis-dependent end-stage renal failure, atrial fibrillation, coronary artery disease, and multiple previous abdominal surgeries. He also had a significant history of vascular disease, whereby he had femoral endarterectomy as well as femorofemoral crossover bypass graft done for peripheral vascular disease. CT abdomen on presentation was unremarkable, other than a small amount of free fluid surrounding the spleen. Ultrasound hepatobiliary system noted features suggestive of cholecystitis. In view of his co-morbidities, he was treated non-operatively with intravenous antibiotics.

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He complained of persistent abdominal pain after one week of treatment. Progress CT with angiogram revealed calcified occlusion of the coeliac artery as well as extensive calcification throughout his aorta and arterial tree. There was gas within the liver, but it was unclear whether this was portal venous gas or gas within the biliary tree. A diagnosis of mesenteric angina was made. However, due to his poor functional status, he was not suitable for surgical or transcatheter interventions and was treated symptomatically. He was clinically improving and was stepped down for rehabilitation.

A few weeks later, he developed a sudden worsening of his epigastric pain, followed by large volume haematemesis. He was haemodynamically unstable. Repeat CT angiography showed a gastrosplenic fistula with extensive gastric necrosis (Figures 1A, 1B & 1C). Due to his poor functional status and rapid deterioration, he opted for palliation and passed away two days later.

Discussion

Gastrosplenic fistula is extremely rare. Frenkel et al. reported a median age of 55 years for patients with GSF, and they were predominantly male, at approximately 80% [2]. The most common presentations for GSF are abdominal pain and constitutional symptoms such as fever, weight loss and fatigue [2, 3]. Acute upper gastrointestinal (GI) bleed was reported only in less than a quarter of the patients; however, if it occurs, it can be life-threatening.

DLBCL is the most common cause of GSF. It can originate from splenic lymphoma, gastric lymphoma or even extensive lymphoma involving both spleen and stomach [2, 3]. There were cases reported to show association with sleeve gastrectomy, Crohn’s disease and splenic abscess [2-4]. Idiopathic spontaneous GSF has also been described in a 16-year-old boy [5]. To our knowledge, there have not been any reported cases of GSF caused by coeliac artery occlusion.

CT abdomen is most useful to confirm GSF. CT angiogram can also be included to exclude any active arterial bleed. If present, transcatheter embolization can be considered. Upper GI endoscopy is both diagnostic and therapeutic. It is helpful to evaluate the extent of disease and allow endoscopic haemostasis. Surgical resection is the treatment of choice for most reported cases, which includes gastrectomy, splenectomy or both. However, successful treatment with chemotherapy with radiotherapy has been reported for GSF secondary to DLBCL [6]. In our case, he was palliated due to his co-morbidities.

It has been postulated that GSF develops from the invasion of malignant tissue from the stomach to the spleen or vice versa, and subsequent necrosis of this tissue results in fistula formation [3]. This case demonstrates that the invasion of an adjacent organ may not be necessary; necrosis itself can cause erosion that ultimately results in fistula formation.

Conflicts of Interest

None.

REFERENCES

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