

Coeliac Artery Thrombosis: An Unusual Cause of Acute Abdomen

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Abstract

Coeliac artery thrombosis with ischaemia is a rare condition, which usually presents with symptoms suggestive of severe peptic ulcer disease. It is usually associated with risk factors for thrombosis or embolism. It is rare because of large number of collaterals between the coeliac and superior mesentery artery. Early detection and intervention is required to prevent the progression to its complications, that includes gastric ischaemic necrosis.

Splenic infarction is a relatively uncommon diagnosis and this clinical presentation can mimic other causes of acute abdominal pain. Cardiologic and haematologic disorders are common reasons for this entity. There have been a few series and single case reports of splenic infarction published in peer-reviewed medical journals.

We here present a 48-year-old female patient with coeliac artery thrombosis, resulting in complete splenic and partial liver infarction. The importance of this case, without any aetiological predisposing factors, is that this kind of clinical situation should be considered in the differential diagnosis of acute abdominal pain.

Introduction

Thrombosis of the coeliac artery trunk is a rare cause of acute abdominal pain. Conditions that increase the tendency of thrombosis such as atherosclerosis, collagen vascular disorders, coagulation abnormalities, and malignancies are the leading causes of coeliac artery thrombosis¹. Computed tomography angiography is the gold standard for diagnosis.

The purpose of treatment is to reestablish blood flow in the mesenteric vessels and to prevent end-organ ischaemic damage and infarction. Percutaneous angioplasty and surgical treatment are the preferred methods of treatment for coeliac artery thrombosis. It is usually associated with other cardiovascular diseases. Although there is substantial advancement in the diagnosis and treatment of coeliac artery thrombosis, hospital mortality is still at 59 - 93%².

The successful treatment depends on early diagnosis and elective intervention with thrombolysis followed by either surgical or endovascular intervention to re-establish blood flow and surgical resection of necrotic parts and good intensive care.

Case report

A 48-year-old female was admitted in her home country (Iraq) for severe epigastric pain, nausea and vomiting associated with left lower limb swelling. She was provisionally diagnosed as pancreatitis with T2DM

(controlled) and left lower limb DVT. Patient was a known case of hypertension from 1 year (on erratic treatment).

At the time of admission to our hospital, she was drowsy and febrile (temperature = 100.4° F). Her BP was 140/90 mmHg, HR - 120/min, RR - 32/min, SPO₂ = 86% on room air. Breath sounds were diminished in bilateral basal areas. Abdomen was distended and tender in left hypochondrium and epigastrium. Bilateral lower limb swelling and redness was present, more on right foot.

Patient was shifted to ICU in view of drowsiness and electrolyte imbalance. On investigations her haemoglobin was 9.4 g/dl, TLC = 23,500/mm³, platelets - 5.06 Lakh/mm³ and ESR = 58 mm/hr. There was hyponatraemia (Na⁺ = 125 meq/l), K⁺ = 3.1 meq/l and creatinine - 0.6 mg/dl. Liver enzymes were elevated (SGOT - 181 U/L, SGPT - 142 U/L, GGT - 228 U/L, ALP - 167 U/L) and serum albumin was 2.0 g/dl. Serum bilirubin, PT and INR were within normal range. Pancreatic enzymes were raised (amylase - 2,625 U/L, lipase - 407 U/L). Her RBS was 154 mg/dl and HbA1c = 6.1%. Thyroid function test and lipid profile were within normal range. Urine and blood culture were sterile.

Protein C, protein S and antithrombin III levels were normal. Anticardiolipin antibodies (IgG and IgM) and lupus anticoagulant were normal.

Chest X-ray revealed haziness in lower zones of both lungs with blunting of bilateral CP angles. CT-pulmonary angiogram showed normal study. USG abdomen showed enlarged liver (18.8 cm longitudinal span) and grade 2 fatty changes. Mild-

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to-moderate free fluid was seen in peritoneal cavity.

Abdominal CT angiogram showed evidence of complete occluded coeliac axis with hypodense intraluminal filling defect (Fig. 1 arrow) and non opacified common hepatic artery along with hepatic branches, splenic artery and gastro

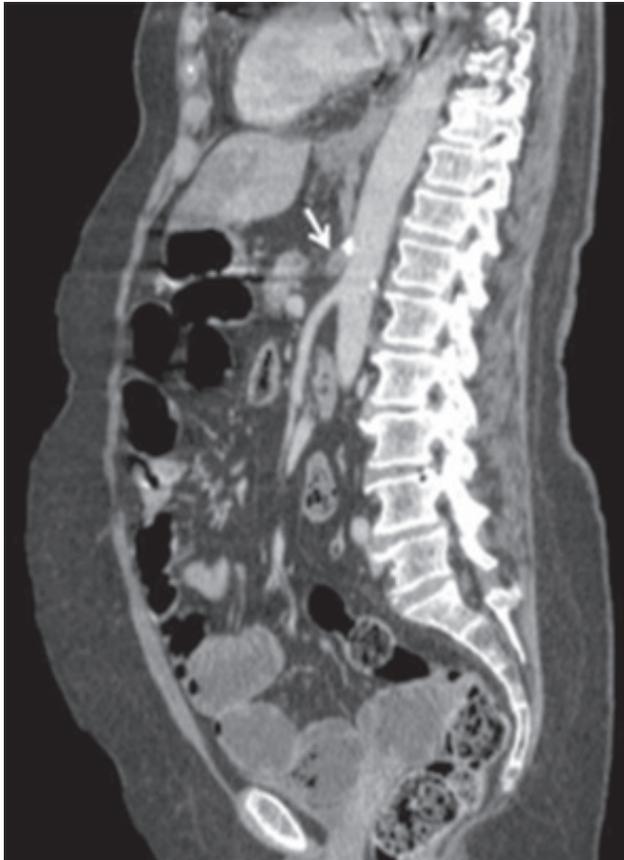


Fig. 1:

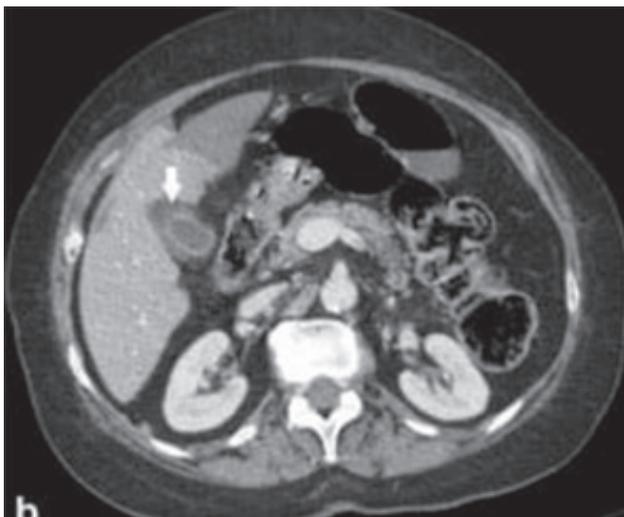


Fig. 2:

duodenal artery s/o thromboembolism (Fig.2 arrow). There was evidence of acute infarct involving portions of the liver and whole of the spleen (Fig. 3 arrow and asterisk). Bilateral lower limb doppler showed no evidence of DVT but DPA thrombosis was seen in left leg.

Intervention radiology team and gastro-surgery consultation was taken in view of extensive thrombosis of coeliac artery and hepato-splenic infarct and was advised to continue systemic anticoagulation along with antiplatelets, statin, anti hypertensive, insulin, anti-fungal, intravenous antibiotics and other supportive measures.



Fig. 3:

Discussion

Coeliac artery thrombosis and splenic infarction is a relatively uncommon diagnosis. O'Keefe *et al*³ reviewed a large autopsy series and found that only 10% of splenic infarctions had been diagnosed ante-mortem. Once a splenic infarction is identified, the cause of infarction should be elicited such as haematologic, metabolic, or thromboembolic disease⁴.

The aetiology of thromboembolism is commonly cardiac disorders including atrial fibrillation, ventricular aneurysm and heart valve diseases⁵. Other aetiological disorders can be acute pancreatitis, antiphospholipid syndrome, malignancies, atherosclerosis, oral contraceptive drugs, diseases related with hypercoagulability and surgical trauma⁶.

Pancreatitis is a common aetiological factor in patients with coeliac artery thrombosis. Kumaran *et al*⁷ reported a case with isolated coeliac trunk thrombosis caused by local complication of acute pancreatitis. They performed

laparotomy due to clinical deterioration. Total gastrectomy and pancreatic debridement was carried-out for visceral necrosis and the patient recovered well after six months.

Serck and Cogbill⁸ reported a 59-year-old female with sigmoid colon carcinoma who had acute visceral ischaemia originating from the coeliac trunk and superior mesenteric artery and, extensive visceral infarction ensued and died. Also coeliac and splenic arteries thrombus in a 14-year-old female who had received oral contraceptive agent for menorrhagia was reported by Arul *et al*⁹. She was well at follow-up only with anticoagulant therapy and more aggressive therapy was not sought.

Another cause of acute coeliac occlusion is coeliac artery dissection. Matsuo *et al*¹⁰ reported a 59-year-old male with isolated dissection of the coeliac artery, with likely splenic infarction. They observed this patient without any treatment modalities because of well developed collateral vessels. Furthermore, some authors applied transcatheter arterial embolisation or surgical reconstruction because their patients became symptomatic with clinical deterioration in the follow-up period^{11,12}. These treatment methods resulted in successful outcome in these patients.

CW Kim and JW Kim¹³ reported a case of coeliac artery thrombosis, and splenic infarction which was surgically treated, and was related to protein S deficiency. In our case, there was thrombosis of the coeliac trunk along with complete splenic infarct and portion of hepatic infarct, along with metabolic syndrome – like features, which were hard to treat surgically, and there was no deficiency of protein C, S.

An uncomplicated splenic infarction can be managed safely with medical treatment, but early surgical intervention is necessary when the complications of infarct such as abscess and rupture occur. Our case was diagnosed in the early period and anticoagulant therapy was started within a short

time, so the result was successful. This clinical situation should be considered in the differential diagnosis of abdominal pain without any aetiological, predisposing factors.

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