CASE REPORT

# Celiac Artery Trunk Thrombosis Presenting as Acute Liver Failure

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## ABSTRACT

Acute mesenteric ischemia is a life-threatening vascular emergency that requires early diagnosis and intervention to adequately restore mesenteric blood flow and to prevent bowel necrosis and patient death. While, almost always superior and inferior mesenteric arteries are involved, we report a 57-year-old male with an unusual celiac artery trunk thrombosis leading to gastero-duodenal and hepato-splenic infarction, and presenting an acute liver failure.

**Key words:** *Mesenteric ischemia. Celiac artery. Acute liver failure. Thrombosis.*

## INTRODUCTION

Acute mesenteric ischemia (AMI) refers to an abrupt reduction in blood flow to the intestinal circulation of sufficient magnitude to compromise the metabolic requirements and potentially threaten the viability of the affected organs. AMI is a life-threatening vascular emergency with mortality rates still ranging between 60 and 80%.[1-3] The clinical setting of acute mesenteric ischemia is determined by combination of a rather rare, difficult (and often enough overlooked) diagnosis, high fatality rates and the need for rapid and aggressive diagnostic and therapeutic interventions in an often elderly and multi-morbid patient; thus making clinical management a challenging and dangerous task.

AMI arises primarily from problems in the superior mesenteric artery (SMA) circulation or its venous outflow. The inferior mesenteric artery (IMA) seldom is the site of lodgment of an embolus. Only small emboli can enter this vessel because of its small lumen. We report an unusual case of celiac artery trunk occlusion causing gasteroduodenal, hepatic and splenic infarction.

## CASE REPORT

A 57-year-old man was admitted to the Internal Medicine Emergency Department with acute abdominal pain of 2 weeks duration. Abdominal pain was vague and intermittent and was associated with anorexia. The patient was obese with history of Diabetes and hyper-lipidemia which was under treatment with glibenclamide and gemfibrozil. No history of arrhythmia, vavular heart disease or an antecedent embolic event, weight loss, postprandial abdominal pain (food fear), smoking or alcohol abuse was noted. Surgical consultation was prompted by aggravation of abdominal pain and deterioration of general condition. On examination patient was confused and haemodynamically unstable. Physical examination revealed temperature of 36°C, pulse rate of 125 beats/minute, respiratory rate of 23/minute and blood pressure of 80/55 mmHg. Abdominal findings were generalized tenderness and rebound tenderness with marked distention and guarding. The patient was mildly icteric and had an episode of haematemesis; digital rectal examination showed a bloody stool.

Laboratory studies revealed a haemoglobin value of 14.7 g/dl and a white blood cell count of 15500/µl, with 81% neutrophils. Biochemistry revealed a blood sugar level of 265 mg/dl, blood urea nitrogen at 137 mg/dl, serum creatinine at 2.6 mg/dl, serum amylase level of 25 U/l, lipase level of 60 U/l, and elevated C-reactive protein at 192 mg/l (+++).

Severe coagulopathy with partial thromboplastin time (PTT) of 44.1 seconds, prothrombin time (PT) of 34.2 seconds and international normalization ratio (INR) of 6.28 was noted. Liver enzyme analysis showed serum glutamic-oxaloacetic transaminase (SGOT) level of 1784 U/L, serum glutamic-pyruvic transaminase (SGPT) of 1780 U/L, alkaline phosphatase (ALP) of 378 U/L, lactate dehydrogenase (LDH) of 2785 U/L compatible with acute hepatic failure.

Arterial blood gases (ABG) analysis revealed a compensated metabolic acidosis with pH of 7.41, $PCO_2$ of 31 mmHg, $PaO_2$ of 63.5 mmHg, $HCO_3$ of 14.7 mmol/L and base excess of -7.5 mmol/L.

Plain radiographs were non-diagnostic. An urgent CT scan was performed which showed infarction of liver and spleen along with edematous wall of stomach and small intestine (Figure 1). Mesenteric CT angiography did not show any flow in celiac artery trunk, with abrupt cutoff of contrast in the vicinity of its origin and normal trunk of superior and inferior mesenteric arteries (Figure

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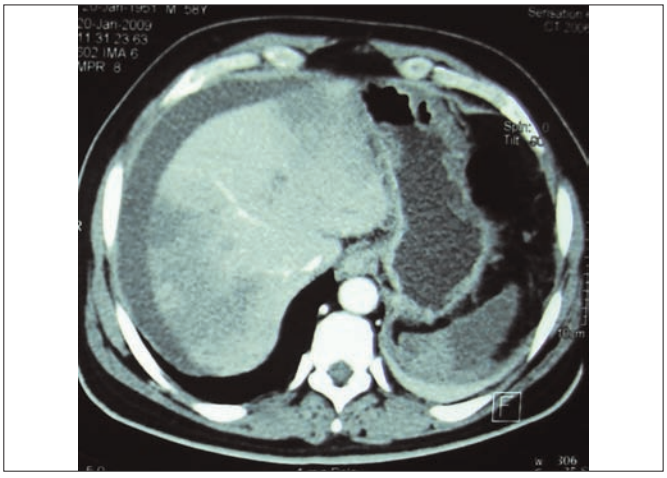

**Figure 1:** A contrast-enhanced CT scan demonstrating thickened gastric wall associated with massive liver and splenic infarction.

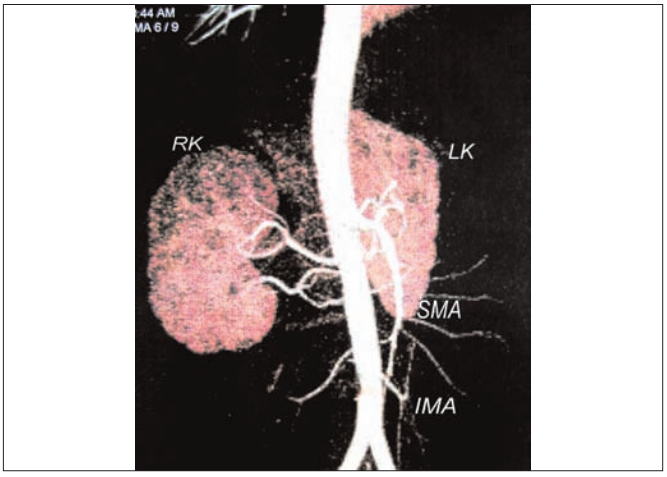


**Figure 2:** CT angiography showing normal superior mesenteric artery (SMA), inferior mesenteric artery (IMA), right (RK) and left (LK) kidneys and non-enhancement of celiac artery trunk.

2).

Decision was taken to proceed to emergency laparotomy for high probable acute mesenteric ischemia, which revealed completely gangrenous stomach, first and second portion of duodenum, omentum and diffuse ischemic changes and infarction of liver and spleen. Visible thrombosis was seen upon vessel transection.

Thus, the operative findings confirmed the suspicion of acute mesenteric ischemia. However, due to extensive organ damage, the patient was found to be inoperable and died soon after the operation.

## DISCUSSION

As the average life expectancy increases, acute bowel ischemia represents one of the most life-threatening abdominal crises in elderly patients.[4] Because of its relative infrequency, the incidence of AMI has been underestimated and there are no established guidelines for its diagnosis and treatment based on the evidence of randomized control trials.

Clinical presentation is non-specific in most cases and can be characterized by an initial discrepancy between severe abdominal pain and minimal clinical findings. Complications such as ileus, peritonitis, pancreatitis, and gastro-intestinal bleeding may also mask the initial signs and symptoms of AMI.[5,6] As bowel ischemia rapidly progresses to irreversible bowel necrosis, severe metabolic derangements ensue, leading to a series of events that culminate in multiple organ dysfunction and death. Establishing an early diagnosis of acute bowel ischemia depends on the identification of persons at risk of development of ischemic bowel and timely use of diagnostic and therapeutic methods to quickly restore blood flow.[2]

Arterial emboli are the most frequent cause of AMI and are responsible for approximately 40-50% of cases.[6] Most of mesenteric emboli originate from a cardiac source. Myocardial ischemia or infarction, arterial tachyarrythmias, endocarditis, cardiomyopathies, ventricular aneurysms, and vavular disorders are risk factors for the development of mural thrombus, which can subsequently embolize to mesenteric arteries.[7] Most visceral arterial emboli preferentially lodge in the superior mesenteric artery (SMA) because it emerges from the aorta at an oblique angle. Whereas 15% of arterial emboli occur at the origin of the SMA, 50% lodge distally to the origin of the middle colic artery, which is the first major branch of the SMA.[7] The celiac axis, the superior and the inferior mesenteric artery supply the forgut, midgut and hindgut, respectively and arterial occlusions may lead to infarction and necrosis of the organs supplied by them.

Two cases of celiac artery trunk thrombosis have previously been reported as a consequence of acute and chronic pancreatitis causing gastric infarction in both, and splenic infarction in later case, requiring total gastrectomy and splenectomy.[8,9] We report a case of celiac artery trunk thrombosis leading to gastro-duodenal, splenic and massive liver infarction presenting as abdominal pain and acute liver failure, combination not encountered in literature before. Although arterial thrombosis has been reported, affecting various important major vessels in close proximity to the pancreas[9,] but in this case no evidence of pancreatitis were found neither in the operating field, nor in pathologic study.

The consequences of celiac artery trunk thrombosis are variable. While stomach and spleen are more susceptible to ischemia and infarction, but it seems that dual blood supply and superior collateral flow ensure that the liver is resilient to the effects of thrombosis. In the patient reported here, propagation of thrombosis to distal branches and collateral circulation was the reason of liver infarction presenting as fulminant hepatic failure. During exploratory laparotomy, the patient found to be inoperable because of extensive gastro-duodenal and

hepato-splenic necrosis and died soon after surgery due to multi-organ failure.

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