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Extensive Foregut Ischaemia in a Patient With Type 1 Diabetes Following Hip Fracture Surgery

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Abstract

Acute compromise of the coeliac trunk is a rare but potentially serious cause of visceral ischaemia. Due to extensive collateral circulation, foregut necrosis is uncommon even in the presence of significant coeliac artery compromise. We present a case of a man in his 40s with type 1 diabetes mellitus who developed severe foregut ischaemia following surgical fixation of an intertrochanteric femoral fracture. His postoperative course was complicated by sepsis, abdominal distension and increasing oxygen requirements. Computed tomography demonstrated gastric pneumatosis, prompting emergency laparotomy, which revealed extensive ischaemia involving the distal oesophagus, stomach, spleen and gallbladder, with preservation of the duodenum. A subtotal cholecystectomy and venting gastrostomy were performed, with the remaining affected viscera managed conservatively. Subsequent CT angiography demonstrated focal coeliac trunk compression consistent with median arcuate ligament compression morphology. Following specialist vascular review, this finding was not considered clinically significant or to warrant intervention. The precise aetiology of the ischaemia remained uncertain, with coeliac artery thrombosis considered a possible, but unconfirmed, underlying mechanism in the context of diabetes mellitus. The patient was managed with gastric decompression, anticoagulation and supportive care, with subsequent clinical and radiological improvement. This case highlights the severity of foregut ischaemia despite extensive collateral circulation and the importance of multidisciplinary management when no clear target for vascular intervention is identified.

Categories: General Surgery, Cardiac/Thoracic/Vascular Surgery, Gastroenterology
Keywords: acute mesenteric ischaemia, coeliac artery thrombosis, foregut ischaemia, gastric ischaemia, hip fracture surgery, postoperative thrombosis, type 1 diabetes

Introduction

The coeliac trunk provides the primary blood supply to the foregut, supplying the distal oesophagus, stomach, liver, spleen, pancreas and proximal duodenum [1]. Acute occlusion of this vessel is relatively rare because of the abundance of collateral circulation between the coeliac and superior mesenteric arteries [2]; therefore, clinically significant foregut ischaemia is extremely rare. Compromised coeliac artery blood supply can result in severe visceral ischaemia with high morbidity and mortality [3].

The causes of compromise to the coeliac artery are primarily atherosclerotic disease causing in situ thrombosis, thromboembolic events and external compression (e.g., median arcuate ligament syndrome). Trauma or iatrogenic vascular injury may also lead to compromise of the coeliac artery. In addition, systemic factors such as long-standing diabetes mellitus contribute to atherosclerosis and macrovascular disease, thereby increasing the risk of arterial thrombosis [3].

This is illustrated through a case in which a patient with type 1 diabetes mellitus developed extensive foregut ischaemia following orthopaedic trauma and surgery. Although the precise underlying vascular mechanism remained uncertain, coeliac artery thrombosis was considered a possible explanation in the context of diabetes mellitus. The anatomical distribution of organ involvement was suggestive of compromise of the coeliac vascular territory. This case highlights the importance of considering visceral ischaemia in patients with unexplained postoperative abdominal deterioration, as well as the role of multidisciplinary assessment in establishing the possible underlying cause and guiding management.

Case Presentation

A man in his 40s with a background of type 1 diabetes mellitus was brought to hospital by ambulance after sustaining a fall while answering his door. He had a history of recurrent falls attributed to diabetic neuropathy.

Radiological imaging demonstrated a displaced intertrochanteric fracture of the right proximal femur, for which he underwent intramedullary nail fixation under the orthopaedic team.

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On the first postoperative day, he developed epigastric abdominal pain with tenderness on palpation and was noted to have an acute kidney injury, which was managed with intravenous fluid resuscitation.

Over the following 48 hours, he developed increasing oxygen requirements and respiratory distress. His clinical condition deteriorated further with features of sepsis, including hypotension, tachycardia and tachypnoea. His National Early Warning Score 2 (NEWS2) [4] was 9, indicating a high risk of clinical deterioration and the need for urgent clinical assessment (respiratory rate 25 breaths per minute, oxygen saturation 96% on 2 L oxygen, heart rate 120 beats per minute, blood pressure 106/58 mmHg, temperature 38.7°C and alert on the Alert, Voice, Pain, Unresponsive (AVPU) scale). He was commenced on empirical intravenous broad-spectrum antibiotics.

Laboratory investigations obtained approximately one hour after the onset of abdominal pain demonstrated leukocytosis, markedly elevated inflammatory markers, anaemia and acute kidney injury. The laboratory results are summarised in Table *1*.

Laboratory parameter	Patient result	Reference range
White blood cell count	16.6 × 10^9/L	4.0-11.0 × 10^9/L
C-reactive protein	432 mg/L	0-5 mg/L
Lactate	1.56 mmol/L	0.5-2.2 mmol/L
Haemoglobin	70 g/L	130-180 g/L
Creatinine	202 µmol/L	60-110 µmol/L
Estimated glomerular filtration rate	27 mL/min/1.73 m^2	≥90 mL/min/1.73 m^2

TABLE 1: Laboratory Investigations During Clinical Deterioration

Reference ranges represent typical adult laboratory values and may vary between institutions. Estimated glomerular filtration rate is expressed per 1.73 m^2 of body surface area.

His abdomen became distended and diffusely tender with guarding, and he had not opened his bowels for five days. The general surgical team reviewed the patient and requested an urgent CT scan of the abdomen and pelvis to identify a potential intra-abdominal source of sepsis.

Imaging demonstrated gastric intramural gas with suspected perforation adjacent to the greater curvature, with differential diagnoses including gastric emphysema and emphysematous gastritis (Figure *1*). Given the patient's clinical deterioration, an ischaemic intra-abdominal process was also considered.

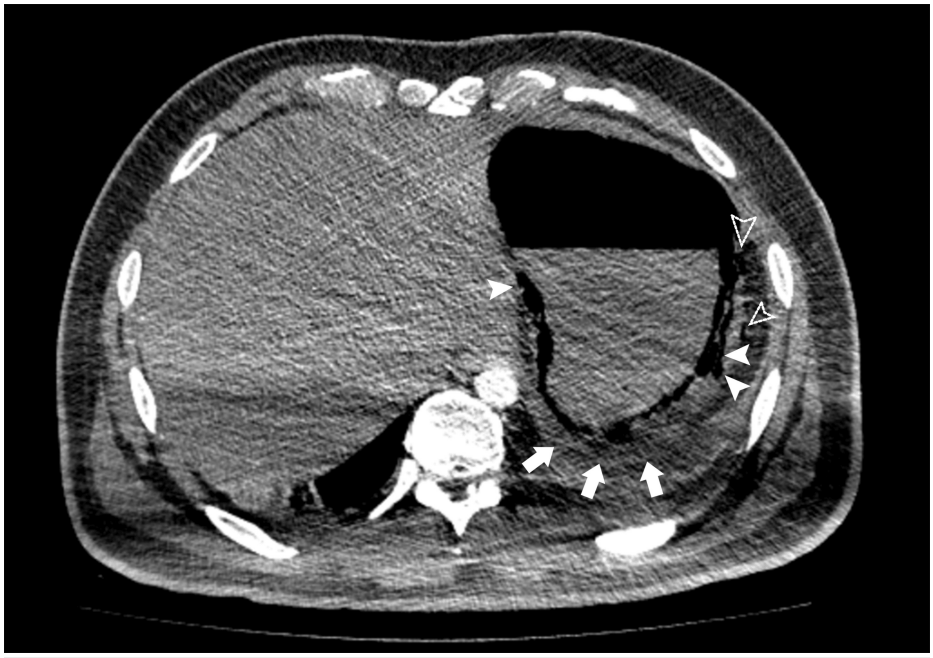


FIGURE 1: CT Abdomen and Pelvis Demonstrating Gastric Pneumatosis With Suspected Ischaemic Changes

Axial contrast-enhanced CT of the abdomen and pelvis demonstrates intramural gas along the greater curvature of the stomach (solid white arrowheads), with subtle adjacent extraluminal gas locules (open white arrowheads with dotted outlines). Reduced mural enhancement of the gastric fundus (solid white arrows) raises concern for gastric ischaemia, with the extraluminal gas suggesting possible focal perforation.

Due to ongoing clinical deterioration, the patient underwent emergency exploratory laparotomy approximately six hours following CT imaging.

At laparotomy, extensive visceral ischaemia was identified. The stomach appeared globally pale and ischaemic from the gastro-oesophageal junction to the first part of the duodenum. The gallbladder was gangrenous, and patchy splenic necrosis was also observed. The duodenum appeared viable. The distal oesophagus could not be adequately visualised intraoperatively.

Given the extent of disease, intraoperative discussion with a tertiary centre was undertaken regarding possible oesophagectomy and gastric resection. However, the patient was haemodynamically unstable and required inotropic support, making major resection prohibitively high risk.

A damage-control strategy was therefore adopted, and the patient underwent subtotal cholecystectomy, venting gastrostomy insertion and placement of multiple intra-abdominal drains.

Because the distal oesophagus could not be directly assessed during laparotomy, intraoperative oesophagogastroduodenoscopy was subsequently performed. This demonstrated sloughing mucosa from the distal oesophagus approximately 30 cm from the incisors and extensive necrotic mucosa throughout the stomach, while the duodenum appeared viable.

Postoperatively, the patient was transferred to intensive care and commenced on intravenous unfractionated heparin anticoagulation in view of the suspected underlying thrombotic mechanism.

Given the intraoperative findings of extensive foregut ischaemia without a clear aetiology, CT angiography was performed one day following laparotomy to assess for a vascular cause. This demonstrated severe focal compression of the coeliac trunk by the diaphragmatic crus, consistent with median arcuate ligament compression morphology (Figure *2*). Although this raised the possibility of a vascular contribution to the ischaemia, following review by the vascular surgery team, the compression was not considered sufficiently clinically significant to warrant vascular intervention, and conservative management was recommended.



FIGURE 2: CT Angiography Demonstrating Focal Coeliac Trunk Compression With Median Arcuate Ligament Compression Morphology

Sagittal reformatted CT angiography demonstrates extrinsic compression of the superior aspect of the proximal coeliac trunk by the median arcuate ligament (solid black arrow), resulting in focal stenosis near its origin (solid black arrowhead). Associated post-stenotic dilatation and the characteristic hooked configuration of the proximal coeliac artery are demonstrated (open white arrowheads with dotted outlines).

In the absence of a surgically correctable large-vessel occlusion, and given the patient's background of long-standing diabetes mellitus, the pattern of widespread foregut ischaemia raised suspicion of coeliac artery thrombosis, potentially occurring on a background of diabetic macrovascular disease. However, no definitive thrombus was identified on imaging, and the precise underlying mechanism remained uncertain.

Following stabilisation in intensive care and vascular surgical input, anticoagulation was transitioned from

intravenous heparin to treatment-dose enoxaparin, and the patient was subsequently stepped down to the surgical ward. Serial lactate measurements throughout his intensive care admission remained at or below 2.2 mmol/L.

The patient experienced a prolonged hospital course complicated by intra-abdominal collections and a bile leak following subtotal cholecystectomy, requiring interventional radiology drainage and endoscopic retrograde cholangiopancreatography (ERCP) with biliary stent placement. He gradually improved clinically. The intra-abdominal drains were sequentially removed prior to discharge as collections resolved. He was discharged with a venting gastrostomy in situ, with a plan for removal at approximately eight weeks.

At an outpatient review seven days following discharge, the patient reported fevers, prompting repeat CT imaging. This demonstrated significant interval improvement in the previously identified gastric pneumatosis (Figure *3*); however, a gallbladder fossa abscess was also identified (Figure *4*). The collection was deemed too small for percutaneous drainage by interventional radiology and was therefore managed conservatively with antibiotics, resulting in clinical improvement.



FIGURE 3: Abdominal and Pelvic CT Demonstrating Interval Resolution of Gastric Pneumatosis and Improvement in Gastric Mural Enhancement

Follow-up axial contrast-enhanced CT of the abdomen and pelvis, obtained approximately four weeks after the initial examination, demonstrates restoration of normal mural enhancement within the gastric fundus (solid white arrows) and resolution of the previously demonstrated gastric pneumatosis along the greater curvature (solid white arrowheads). No residual perigastric extraluminal gas or significant surrounding inflammatory fat stranding is identified.



FIGURE 4: Abdominal and Pelvic CT Demonstrating Postoperative Gallbladder Fossa Abscess

Axial contrast-enhanced CT of the abdomen and pelvis obtained following subtotal cholecystectomy demonstrates a fluid collection within the gallbladder fossa (black arrow), with peripheral rim enhancement (solid white arrowheads) and internal gas locules (open white arrowhead with dotted outline), consistent with a postoperative abscess. Minimal surrounding inflammatory fat stranding is also present.

At a subsequent presentation four weeks following discharge from his initial hospital admission, the patient reported fever and leg pain and was found on CT pulmonary angiography to have bilateral pulmonary emboli. He was subsequently commenced on long-term anticoagulation with a direct oral anticoagulant.

At follow-up eight weeks after gastrostomy insertion, the patient continued to recover well. The venting gastrostomy was successfully removed as planned, and he remained clinically stable on anticoagulation.

Discussion

Foregut vascular anatomy

Foregut vascular anatomy is important for understanding the distribution of organ ischaemia. The coeliac trunk is a major branch of the abdominal aorta, arising at approximately the level of the 12th thoracic vertebra. Structures derived from the embryological foregut receive their arterial supply predominantly from branches of the coeliac trunk [1].

Typically, the coeliac trunk divides into three major branches: the left gastric artery, splenic artery and common hepatic artery.

The left gastric artery supplies the distal oesophagus and lesser curvature of the stomach. The splenic artery supplies the spleen and contributes to the blood supply of the stomach through the short gastric and left gastroepiploic arteries. The common hepatic artery gives rise to the proper hepatic artery and the gastroduodenal artery, which contributes to the blood supply of the stomach and duodenum [5].

Collateral circulation protects the foregut organs from ischaemia. An important collateral pathway is the pancreaticoduodenal arcade, which provides communication between the coeliac and superior mesenteric artery (SMA) circulations [5,6].

Consequently, isolated occlusion of the coeliac trunk does not frequently result in severe ischaemia because of these collateral pathways. However, acute compromise of coeliac arterial flow may exceed the capacity of the collateral circulation, particularly in the presence of reduced systemic perfusion or extensive thrombotic disease.

The patient's distribution of ischaemia, involving the distal oesophagus, stomach, gallbladder and spleen with sparing of the duodenum, is consistent with compromise of the coeliac arterial circulation. Preservation of the duodenum may be explained by its dual arterial supply and preserved collateral circulation from the SMA via the pancreaticoduodenal arcade [5,6], as illustrated by the foregut arterial anatomy in Figure *5*.

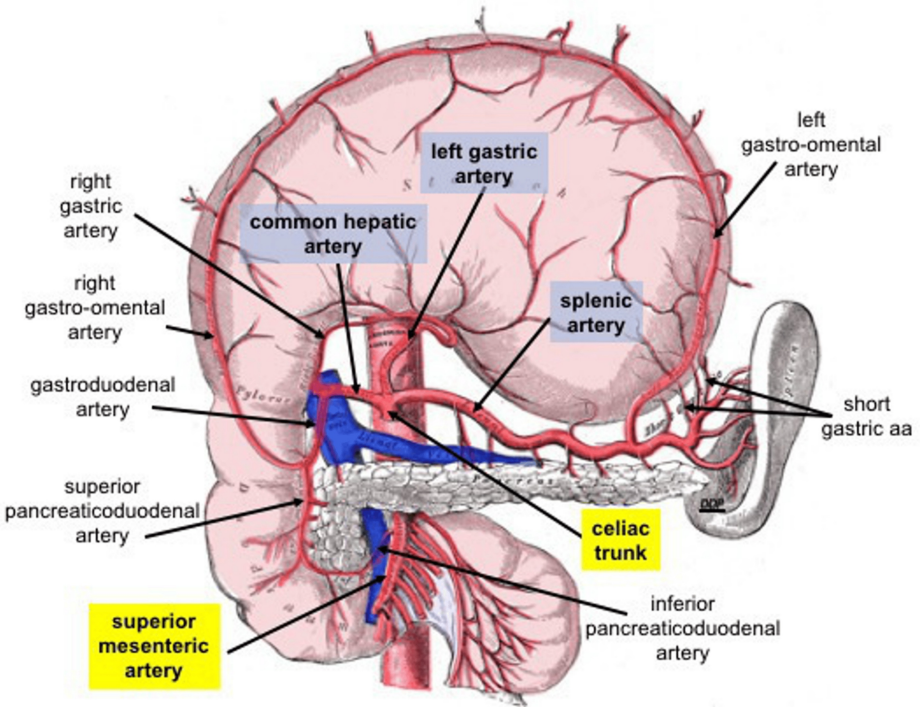


FIGURE 5: Foregut Arterial Supply

Arterial supply of the foregut demonstrating the coeliac trunk and its major branches: left gastric, splenic and common hepatic arteries [7].

Image by Dennis M DePace, PhD, based on original artwork by Henry Vandyke Carter from Gray's Anatomy. Reproduced from Wikimedia Commons under the Creative Commons Attribution-ShareAlike 4.0 International (CC BY-SA 4.0) licence. No changes were made.

Aetiology and pathophysiology

Although no definitive coeliac artery thrombus was identified on imaging, the clinical and anatomical findings raised the possibility of coeliac artery thrombosis, potentially associated with underlying diabetic macrovascular disease. Diabetes is associated with accelerated atherosclerosis and endothelial dysfunction, both of which impair perfusion and increase the risk of arterial thrombosis [8]. Given that baseline vascular reserve was potentially compromised secondary to diabetic macrovascular disease, it is possible that collateral pathways were unable to adequately compensate for the acute reduction in coeliac arterial flow, resulting in widespread foregut ischaemia.

Additional factors likely contributed to a prothrombotic state. The recent traumatic injury and subsequent surgery are recognised to induce a hypercoagulable state [9], increasing the risk of both arterial and venous thrombosis [10]. The combination of possible underlying diabetic macrovascular disease and acute physiological stress may therefore have increased the risk of coeliac axis thrombosis.

Fat embolism following long bone fracture was also considered as a potential contributing factor. Long bone fractures and subsequent internal fixation can result in the release of marrow fat into the circulation. While fat embolism predominantly affects the pulmonary vasculature, systemic embolisation has been described [11]. However, the absence of classical clinical features, combined with the distribution of ischaemia observed, made this less likely than thrombosis secondary to diabetic macrovascular disease.

Median arcuate ligament compression

Imaging demonstrated focal compression of the coeliac trunk by the diaphragmatic crus, consistent with median arcuate ligament compression morphology. Median arcuate ligament syndrome typically presents as a chronic condition with postprandial abdominal pain and weight loss [12]. Vascular surgical opinion was

that this finding was unlikely to be clinically significant and did not warrant intervention. It is therefore unlikely to represent the primary cause of ischaemia. Consequently, management remained focused on supportive care, gastric decompression and anticoagulation rather than vascular intervention.

Clinical presentation and diagnosis

Foregut ischaemia can be difficult to diagnose due to a non-specific initial presentation. A key radiological finding in this case was the presence of intramural gastric gas on CT. This may represent gastric emphysema, emphysematous gastritis or ischaemia, and in the context of clinical deterioration should prompt urgent surgical assessment [13].

Notably, the patient's lactate levels remained within the normal range despite extensive foregut ischaemia, highlighting that a normal lactate level does not exclude severe gastrointestinal ischaemia and should not delay further investigation when clinical suspicion remains high [14].

Management

Management of extensive foregut ischaemia is technically challenging and depends on the extent of ischaemia and the patient's clinical condition [15]. In critically ill or haemodynamically unstable patients requiring surgery for acute intestinal ischaemia, a damage-control approach may be appropriate, allowing physiological stabilisation and subsequent reassessment of tissue viability [16,17]. In this case, subtotal cholecystectomy, venting gastrostomy and drainage were performed to stabilise the patient and allow for subsequent recovery.

This case is notable for the extent of foregut ischaemia despite the presence of normally protective collateral circulation. It highlights how acute physiological stress, in combination with underlying macrovascular disease, can precipitate catastrophic vascular events.

Conclusions

Acute foregut ischaemia is rare due to the extensive collateral circulation of the foregut but should be considered in patients who develop unexplained abdominal deterioration following major surgery. Intramural gastric gas on CT in the context of clinical deterioration should raise concern for ischaemia and prompt urgent surgical assessment. The distribution of gastrointestinal ischaemia, including preservation of the duodenum, may help identify the vascular territory involved.

This case demonstrates extensive foregut ischaemia following orthopaedic surgery in a patient with type 1 diabetes mellitus, with a suspected but unconfirmed underlying vascular mechanism. It highlights the importance of multidisciplinary assessment and individualised management when extensive visceral ischaemia is identified. In haemodynamically unstable patients, a damage-control surgical approach may allow initial stabilisation rather than immediate definitive resection.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Mehtab Mann, Noureldine Nagui, Simon Fisher, Isha Wadhwa

Acquisition, analysis, or interpretation of data: Mehtab Mann, Yuuki Na

Drafting of the manuscript: Mehtab Mann, Noureldine Nagui, Isha Wadhwa

Critical review of the manuscript for important intellectual content: Mehtab Mann, Simon Fisher, Isha Wadhwa, Yuuki Na

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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