Endovascular Management of a Gastric Artery Aneurysm Rupture

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Abstract

Visceral artery pseudoaneurysms or aneurysms (VAPA) can form secondary to a variety of congenital, traumatic and inflammatory pathologies such as pancreatitis. Massive haemorrhage into the gastrointestinal tract or the peritoneal cavity from visceral artery pseudoaneurysms or aneurysms can result in death in 40% of cases. Gastroduodenal artery aneurysm (GDA) rupture is a rare, life-threatening condition and bleeding into the gastro-intestinal tract is the most rapidly fatal complication of an arterial visceral aneurysm. They represent 1.5% of all visceral artery aneurysms and are classified into true and pseudo aneurysms depending on their aetiology. They are challenging to diagnose and may prove fatal if they rupture. They can be managed with a surgical, endovascular or a combined approach. The authors present the case of a 63 year old female presenting with hypotension and abdominal pain. A ruptured aneurysm of her gastroduodenal artery was subsequently found. She was successfully treated with transcatheter coiled embolization. The aetiology, clinical presentation and management of a gastroduodenal artery aneurysm are discussed. This case acts as a cautionary reminder of considering a ruptured GDA in the differential diagnosis in patients presenting to the Emergency Department with hypovolaemic shock and an acute abdomen.

Keywords: Endovascular; Gastric artery; Aneurysm rupture; VAPA; Pseudoaneurysms

Case Presentation

A 63 year old female was found collapsed by her husband at home. There was a witnessed loss of consciousness of 3 minutes. She was transferred by ambulance to the Emergency Department. She was hypotensive (BP 79/61 mmHg) and tachycardic (heart rate 105 beats per minute). She denied a history of chest pain or palpitations, but described generalised abdominal pain and feeling dizzy. Her medical history included hypertension and hyper cholesterolaemia. She was diagnosed with microcytic anaemia, endoscopy and colonoscopy were normal and she commenced treatment with ferrous fumarate. Her surgical history included a hysterectomy for a symptomatic uterine fibroid. Her medications included perindopril 10mgs od, amlodipine 10 mgs od and ferrous fumarate 100mgs od. Her BMI was 22 kg/m². She reported two episodes of loose stools and nausea. She was passing flatus. Her GCS was 15/15 and her observations were as follows: BP 75/60 mmHg, HR 120 bpm, RR 16, SpO2 96% on air and temperature 36.70C. Physical examination confirmed good air entry bilaterally and her heart sounds were normal with
no audible murmur. Her abdomen was mildly distended with generalised tenderness on palpation. Bowels sounds were present on auscultation. Fluid resuscitation with 2 litres of plasmalyte was initiated.

**Investigations**

Her 12 lead ECG showed sinus tachycardia, with a heart rate of 120 beats per minute. Her venous blood gas confirmed a lactate of 2.1mmol/L and a pH of 7.35. Haematological investigations showed a microcytic anaemia and a leucocytosis (Table 1). Her clotting and liver function tests were normal.

**Table 1**: Laboratory investigations confirmed a leucocytosis and a microcytic anaemia.

<table>
<thead>
<tr>
<th>Complete Blood Count</th>
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<tbody>
<tr>
<td>WCC</td>
<td>17.42x10^9/L</td>
</tr>
<tr>
<td>Platelets</td>
<td>257x10^9/L</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Differential Count</th>
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<tbody>
<tr>
<td>Neutrophils</td>
<td>12.70%</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>1.40%</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>82 g/L</td>
</tr>
<tr>
<td>MCV</td>
<td>79 Fl</td>
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<table>
<thead>
<tr>
<th>Renal</th>
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<tbody>
<tr>
<td>Sodium</td>
<td>138 mmol/L</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.4 mmol/L</td>
</tr>
<tr>
<td>Urea</td>
<td>7.6 mmol/L</td>
</tr>
<tr>
<td>Creatinine</td>
<td>125 umol/L</td>
</tr>
<tr>
<td>Egfr</td>
<td>48</td>
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Her chest radiograph showed no evidence of consolidation or a pleural effusion. Her abdominal radiograph showed a normal distribution of the bowel gas. There was some air in the left hypochondrium suggestive of a pneumoperitoneum. She sustained a witnessed collapse in the emergency department with no loss of cardiac output. She was hypotensive (BP 80/68 mmHg) and tachycardic (110 beats per minute). Her peripheries were cool. Her abdomen was becoming increasingly distended. A CT Abdomen with contrast demonstrated a severe, diffuse haemoperitoneum and a large haematoma in the right hemi-abdomen measuring 130x119mm. The haematoma was causing a mass effect over the adjacent structures, including the inferior vena cava. Active flush of contrast in the arterial phase was observed, in keeping with active bleeding (Figure 1). The origin of the bleeding was difficult to establish but was thought to be coming from the gastroduodenal artery. The major haemorrhage protocol was activated. She was resuscitated with blood components, 1 gram of tranexamic acid and 10mmol of calcium chloride. The patient had a prohibitive high risk for surgery.
Differential Diagnosis
A diagnosis of haemorrhagic shock due to intra-abdominal bleeding was rendered. After haemodynamic stabilisation with blood component transfusion, a repeat CT abdomen and pelvis was performed. It demonstrated an occlusion of the coeliac artery with collateral blood flow via the inferior anterior and inferior posterior pancreatoduodenal branches of the superior mesenteric artery.

![Figure 1: Active flush of contrast in the arterial phase was observed, in keeping with active bleeding.](image1)

Treatment
She proceeded to rapid sequence intubation. She was sedated with remifentanil and propofol prior to transfer to interventional radiology for embolization. She remained cardiovascularly stable without inotropic support. Severe occlusion of her coeliac axis was observed. Coiled embolization of her gastroduodenal artery via her superior mesenteric artery was performed (Figure 2). She was haemodynamically stable at the end of the procedure and was transferred to ICU for further management and monitoring for signs of abdominal compartment syndrome.

![Figure 2: Coiled embolization of her gastroduodenal artery via her superior mesenteric artery was performed.](image2)

Outcome
An abdominal pressure of <20 mmHg was aimed for, measured every 2 hours. Her oxygen requirements increased from 30 to 50% FiO\textsubscript{2}. Furthermore, her haemoglobin dropped from 94 to 75 g/dl. Her heparin infusion was held as an intra-abdominal bleed.
or an infected haematoma of the retroperitoneal space was suspected. Her abdominal pressure remained stable at 16 mmHg. Further investigation with an USS showed no evidence of free fluid. Her chest radiograph showed bi-basal shadowing suggestive of ventilator associated pneumonia. Treatment with IV tazobactam was initiated. Her acidosis and hypothermia were corrected. Her hypoxia persisted, therefore, a CT was performed. Emboli were observed at the distal aspect of the right main pulmonary artery, extending into the right upper, middle and lower lobes as well as the segmental arteries. Small bilateral pleural effusions were noted. Hypo-enhancement in both hepatic lobes was suggestive of infarcts and multi-focal infarcts were also noted in her spleen. Her haematoma in the right abdomen had reduced in size to 10x6cm and there was a reduction in size in the overall volume of haemoperitoneum.

She commenced treatment dose dalteparin. As her inflammatory markers remained high, she received vancomycin and meropenem on microbiology advice. She had a failed extubation secondary to laryngeal oedema. She was reviewed by ENT and following administration of IV dexamethasone, she was successfully weaned. She was transferred to the surgical ward and her recovery was uneventful. Two days following her discharge, she presented to the emergency department with increasing left sided abdominal pain. Her inflammatory markers were raised (WCC 18.36x10⁹/L, neutrophils 14.51 and CRP 237 mg/L). A CT abdomen and pelvis with contrast demonstrated a reduction in size of her haematoma from 11.6x6.6cm to 9.6x6.8cm. The haematoma demonstrated uniform peripheral enhancement. The loculated fluid around the right lobe of the liver showed peripheral enhancement and had marginally increased in volume. The peripheral enhancement in both of these collections was non-specific and probably represented normal change in a resolving haematoma. As her inflammatory markers remained raised despite treatment with intravenous amoxicillin and metronidazole, US guidance of her peri-hepatic collection was performed. A 12F drain was inserted and placed on free external drainage. Her drain was removed after 48 hours and she described severe right upper quadrant abdominal pain. Therefore, a CT abdomen and pelvis with contrast was performed. The previously demonstrated mesenteric haematoma had marginally reduced in volume, although the larger perihepatic fluid collection had not changed in size significantly. No new intra-abdominal fluid collection was demonstrated. The left pleural effusion had marginally increased in volume, although the right pleural effusion had almost completely resolved. The patchy bibasal lung atelectasis remained unchanged. As her inflammatory markers were within normal range, she was discharged home with oral co-amoxiclav. She remains under review.

Discussion

Gastroduodenal Artery aneurysm (GDA) was first reported by Starlinger in 1930, since then there have been increasing reports due to improved radiological techniques [1]. Visceral artery aneurysms and pseudoaneurysms are defined as a >1.5 fold increase in the normal diameter of the celiac, superior or inferior mesenteric arteries and their branches. It excludes aneurysms of the aortoiliac axis. They are a rare occurrence with an incidence of 0.1-0.2%. Aneurysms can be divided into true aneurysms or pseudoaneurysms, also known as a false aneurysm. True aneurysms involve all layers of the wall and result from vessel wall abnormalities secondary to atherosclerosis. Pseudoaneurysms occur after vascular injuries or inflammatory processes such as pancreatitis causing a tear in the vessel wall mediated by pancreatic proteolytic enzymes. It can also be related to iatrogenic injury occurring during instrumentation, for example, during a gastrectomy for duodenal ulcer. They can also occur following a cholecystectomy. Other causes include alcohol excess, liver cirrhosis and syndromes such as Marfan’s or Ehlers Danlos. Other predisposing factors include fibromuscular dysplasia, polyarteritis nodosa and septic emboli. Pseudoaneurysms more commonly involve the splenic, renal, hepatic and pancreaticoduodenal arteries. They are most commonly found between 50 and 58 years of age. They have a male predilection (m:f 4.5:1). The mean size is 3.6 cm [2]. Only 1.5% of all visceral artery aneurysms

Involvement of the gastroduodenal artery (GDA) [3]. However, it has a 75% risk of rupture. Atherosclerosis of the superior mesenteric artery or the celiac artery is the pathophysiology underlying the development of a true GDA with subsequent stenosis. It is also rarely caused by congenital absence of the celiac axis [4]. Gastrointestinal bleeding is the presenting feature in >50% and patients may present with haematemesis, melena or haemobilia. Further bleeding into the peritoneum will cause haemorrhagic shock and severe abdominal pain, the second most common presenting symptom. The mortality rate with rupture is 40% with the highest mortality rate coming from rupture into the duodenum [5]. A pulsatile abdominal mass with or without a bruit on auscultation could be the sole warning sign. Unruptured GDAs may be asymptomatic in 7.5% or may cause upper abdominal pain, obstructive jaundice or microcytic anaemia. In rare cases, the GDA may rupture into the superior mesenteric vein and present with bleeding varices [6]. It is important to establish a history of recent pancreatitis, vascular or laparoscopic intervention. Gold standard investigation is visceral angiography which has 100% sensitivity, followed by computed tomography (67%) and ultrasonography (50%) [7]. Transcatheter coil embolization and stenting are endovascular options in uncomplicated stable aneurysms [8]. Percutaneous endovascular embolization achieves haemostasis in 80-95% of emergency cases [9]. Associated risks include coil or stent migration causing gastric outlet obstruction, aneurysm dissection or rupture and emboli. Narrow necked PA or aneurysms are best treated via direct delivery of coils into the sac. Wide necked, large diameter vessels can be treated by stent insertion [10]. Low-flow VPAs can be treated with percutaneous thrombin injection alone. Other surgical strategies include revascularization, vessel ligation or aneurysmal sac exclusion.

**Conclusion**

Complications include haemorrhage, wound infection, paralytic ileus or acute pancreatitis. Although visceral artery aneurysm rupture is a rare clinical presentation, physicians should have a high index of clinical suspicion in patients presenting with severe abdominal pain and haemodynamic instability. As rupture is associated with a high mortality prompt recognition is required to initiate appropriate management to prevent further morbidity.

**REFERENCES**
