Gastric Dieulafoy lesion: a rare cause of massive haematemesis in an elderly woman

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DESCRIPTION
A 78-year-old woman presented to her local accident and emergency department by ambulance, having collapsed following several episodes of voluminous fresh haematemesis with melaena. The patient had extensive medical comorbidities, suffering from type 2 diabetes mellitus, ischaemic heart disease, bronchiectasis and severe pulmonary hypertension. Three weeks previously she had undergone an uncomplicated total hip replacement for osteoarthritis and had been using non-steroidal anti-inflammatory drugs (NSAIDs) for analgesia. She had no history of gastroenterological disease.

The patient responded to initial resuscitative measures sufficiently to undergo oesophagogastroduodenoscopy. The gastroenterologist struggled to achieve any useful view of either stomach or duodenum due to the volume of haemorrhage, and aborted the procedure. The patient rapidly displayed signs of haemodynamic instability and deteriorated into a state of refractory hypovolaemic shock. She was intubated and ventilated.

The patient underwent laparotomy and antrectomy with duodenotomy, which showed no focal gastric or duodenal abnormality. Attempted preservation of the gastric fundus did not arrest the arterial haemorrhage and control was only achieved through total gastrectomy and oesophageojunostomy. The gastric specimen is displayed, with bleeding source highlighted by arrow and inset (figure 1).

The list of differential diagnoses in this clinical situation is extensive (table 1). The most common cause of upper gastrointestinal bleeds (UGIBs) in this population is peptic ulceration, particularly posterior-wall duodenal ulcers, which can generate brisk haemorrhage from the gastroduodenal artery (a branch of the hepatic artery). This diagnosis may have been corroborated by the history of NSAID use; however, failure of endoscopy and angiography to identify a source of bleeding is typical of a more occult source.

The diagnosis is that of a Dieulafoy lesion or ‘persistent calibre artery’ of the stomach. A histomicrograph of the lesion is shown in figure 2, depicting classical features of normal surface gastric mucosa, aside from a haemorrhagic ulcer (arrow) penetrating a thick-walled arterial structure located within the superficial submucosa, representing the source of bleeding.

Dieulafoy lesion is a rare but well-recognised cause of UGIB. Lesions are most commonly located in the proximal stomach but have been reported throughout the gastrointestinal tract. Bleeding may be self-limiting and intermittent, or severe, the latter necessitating urgent intervention. Endoscopic detection is the diagnostic modality of choice, although endoscopic identification of the bleeding source may be extremely difficult, as bleeding is typically intermittent and surrounding mucosa is typically normal or demonstrates only a tiny ulcer.

In the acute setting, profuse bleeding may obscure the endoscopic view. CT angiography may be a diagnostic strategy in the setting of acute bleeding, which may facilitate interventional radiology and arterial embolisation. Definitive
management of catastrophic bleeding may require subtotal or total gastrectomy, and fatal cases are recognised. The patient recovered well in the initial postoperative period but succumbed some months later from refractory pulmonary hypertension.

**Contributors** JC: corresponding author and primary author; organised correspondence between all parties and prepared the final draft for submission; led the concept and design of the article and assimilated the various clinical images of the pathological appearances of Dieulafoy lesions for submission. BC: co-senior author; the consultant surgeon who performed the gastric resection and information from the respective consultants. ML: co-senior author; consultant pathologist; interpreted pathological specimens for submission and described a short clinical vignette on the pathological appearances of Dieulafoy lesions for submission. BC: co-senior author; the consultant surgeon who performed the gastric resection and interpreted/prepared the surgical images for submission, in addition to supplying a short clinical vignette.

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**REFERENCES**


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**Table 1** Differential diagnoses of upper gastrointestinal bleeding

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<thead>
<tr>
<th>Diagnosis</th>
<th>Microscopic features</th>
<th>Macroscopic features</th>
<th>Clinical features</th>
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<tbody>
<tr>
<td><strong>Dieulafoy lesion</strong></td>
<td>Normal surface gastric mucosa aside from defect Histologically normal, tortuous submucosal artery with an abnormally large arterial diameter (1–3 mm) Small mucosal defect varying from 2 to 5 mm, with a fibrinoid necrotic base</td>
<td>Mucosal erosion ~1–5 mm; 75% within 6 cm of the GOJ Attached thrombus may be seen Absence of inflammation at the edge of the mucosal defect</td>
<td>&lt;1% of UGIBs Intermittent painless melena and haematemesis M:F 2:1</td>
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<td><strong>Duodenal ulcer</strong></td>
<td>Mucosal ulceration that can penetrate into submucosa and muscularis propria, or perforate onto the serosal surface; degree of surrounding fibrosis relative to chronicity Background mucosal changes depend on aetiology, for example, Helicobacter pylori gastritis or NSAID-related reactive gastropathy (see below); most other causes, for example, peptic, have non-specific microscopic features</td>
<td>Mucosal defect ~2–4 cm Diffuse, erythematous mucosal borders Bleeding vessel or adherent clot sometimes visible in ulcer base Mucosal defect ~2–4 cm Smooth base with perpendicular borders Bleeding vessel or adherent clot sometimes visible in ulcer base</td>
<td>~50% of UGIBs Symptoms: epigastric abdominal pain, belching, anorexia, haematemesis Risk factors: <em>H. pylori</em> infection, smoking, NSAIDs, steroids, vagal tone (Cushing’s ulcer), burns (Curling’s ulcer), Zollinger-Ellison syndrome (rare)</td>
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<td><strong>Gastric ulcer</strong></td>
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<td><strong>Oesophageal varices</strong></td>
<td>Large dilated submucosal veins Expanded submucosa with elevation of mucosa above normal tissue Haemosiderin-laden macrophages Fresh blood</td>
<td>Dilated submucosal vessels in ‘columns’ Commonly in distal oesophagus Small (1–2 mm) or large (1–2 cm)</td>
<td>5%–10% of UGIBs Haemorrhage risk: Size of varices Anticoagulants Active alcohol use Systemic infection Volume resuscitation can precipitate further haemorrhage—aim for stability and Hb &gt;80 g/L</td>
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<td><strong>Gastritis</strong></td>
<td>Microscopy depends on aetiology, for example, <em>H. pylori</em> gastritis (usually antral but may be pan-gastric; bacteria highlighted by histochemistry); NSAID-related reactive gastropathy (foveolar hyperplasia, fibromuscular lamina propria expansion, paucity of inflammation); acute gastritis (mucosal oedema, haemorrhage and superficial erosions)</td>
<td>Mucosal erythema and oedema, typically associated with friability and superficial mucosal ‘breaks’ (erosions)</td>
<td>10%–20% of UGIBs Heterogeneous phenomenon; the Sydney system or the Operative Link for Gastritis Assessment staging system may be useful in diagnostics/prognostics. Common aetiology: <em>H. pylori</em> colonisation, NSAIDs, alcohol, critical illness Treatment should comprise <em>H. pylori</em> eradication, antacid therapy and cytoprotective agents.</td>
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<td><strong>Mallory-Weiss tear</strong></td>
<td>Longitudinal mucosal lacerations of the distal oesophagus/proximal stomach, with surrounding haemorrhage and acute inflammatory reaction</td>
<td>Longitudinal mucosal lacerations with or without active bleeding and adherent clot Occasionally healing tears may appear as superficial ‘blood blisters’</td>
<td>Up to 10% of UGIBs More common in the young Predisposing factors include alcoholism and hiatus hernia Haematemesis initiated by severe coughing or retching Non-bleeding tears may be managed conservatively, with acid suppression and antiemetics.</td>
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GOJ: gastro-oesophageal junction; Hb: haemoglobin; NSAID, non-steroidal anti-inflammatory drug; UGIBs, upper gastrointestinal bleeds.