Acute Gastrointestinal Tract Bleeding – Diagnosis and Intervention

A. Holden
1Auckland City Hospital, Auckland, New Zealand

The evaluation and treatment of acute GI bleeding is complex and frequently demands a multidisciplinary approach. Radiology has a role to localise, characterise and on occasions treat the bleeding.

Upper GI bleeding, defined as bleeding proximal to the ligament of Treitz and accounts for ~70% of all acute GI bleeding. Common aetiologies include erosion or ulcer, variceal bleeding and Mallory-Weiss tear. The diagnosis of upper GI bleeding is usually made on clinical grounds based on the history of haematemesis and melaena although rapid upper GI bleeding may also present with rectal bleeding (haematochezia). The primary investigation modality for upper GI bleeding is endoscopy that also offers potential to treat the bleeding in many cases.

Lower GI bleeding is defined as bleeding distal to the ligament of Treitz and accounts for ~30% of all acute GI bleeding. The prevalence is higher in older patients and aetiologies include diverticular disease, angiodysplasia, colitis and bleeding neoplasms. Patients typically present with haematochezia and signs of blood loss. Although signs vary with the patient’s age and cardiovascular status, patients typically become tachycardic when acute blood loss exceeds 500ml and become systemically shocked when >15% of the circulating blood volume is lost. Acute GI bleeding causing shock is termed “massive GI bleeding” and is usually seen in active bleeding rates > 1ml/min. The mortality for acute lower GU bleeding ranges from 3-18% but increases up to 40% in patients with massive bleeding. The natural history of acute lower GI bleeding is for the bleeding to be intermittent and up to 80% of cases will cease spontaneously.

The current imaging modalities available for acute lower GI bleeding include multidetector CT Angiography, Colonoscopy, Radionuclide Imaging and Catheter Angiography. Radionuclide imaging using Technetium 99m labelled RBCs has the advantage of detecting low rates of blood loss (0.1-0.5ml/min) but has a limited role in acute GI bleeding due to a lack of availability, time consuming procedure and inaccurate localisation. Colonoscopy suffers from limited visualisation, especially in massive GI bleeding. Enteroscopy and capsule endoscopy utility is largely restricted to obscure GI bleeding.
CT Mesenteric Angiography is now the primary investigative modality for acute GI bleeding. The investigation is non-invasive, widely available, rapid and sensitive. Animal models suggest CTA can detect mesenteric artery bleeding at a rate of 0.3-0.4ml/min, comparing favourably with catheter angiography. Most protocols involve non-contrast, arterial (delay of ~25 seconds in “late capillary phase”) and delayed phases with contrast injected at 4-5ml/s and multiplanar reformations. No oral contrast or water is administered. The primary sign of acute GI bleeding is contrast extravasation into the bowel lumen. The extravasated contrast may be linear, swirled or pooled in appearance and the contrast becomes more extensive (and often less dense) within the lumen on delayed images. Some authors measure the attenuation of bowel fluid and use a threshold of > 90HU to diagnose acute bleeding. Ancillary findings include haematoma on non-contrast scans and focal dilatation of a fluid filled bowel segment. Additional tasks for CT include evaluating the arterial anatomy supplying the bleeding site (facilitates subsequent intervention) and identifying possible aetiologies. Bleeding diverticula, tumours and colitis can usually be accurately diagnosed on CT. Angiodysplasia should be suspected in the absence of any underlying bowel pathology. Although both angiodysplasia and diverticula often cease bleeding spontaneously, angiodysplasia is much more likely to recur.

Most studies comparing CTA to DSA have reported sensitivities for CTA of 91-92% and specificities approaching 100%. False positives with CTA may be seen with retained oral contrast, surgical clips and dense lumen debris (recognised on non-contrast CT) and prominent mucosal enhancement in collapsed bowel. False negatives may be seen when blood is diluted in fluid filled bowel loops.

Catheter angiography has a sensitivity for detecting acute GI bleeding of ~ 50 % (23-72%). The sensitivity increases to 84-87% in patients with massive GI bleeding (haemodynamic instability) so a case can be made for taking these patients straight to catheter angiography, particularly if they are of high operative risk. Other indications for catheter angiography include patients who re-bleed after a negative CTA and patients requiring intervention. The angiographic technique includes SMA and IMA selective angiography – the IMA runs are normally performed first and this, along with a urinary bladder catheter, prevents bladder contrast obscuring pelvic vessel detail. Older angiographic techniques including provocation angiography (heparin, thrombolitics) and vasopressin infusions have declined in popularity.

Catheter directed embolisation is achieved with co-axial super-selective microcatheters in marginal arteries or vasa recta close to the site of bleeding. Embolic agents include micro-coils, particles (eg PVA, embospheres) and gelfoam. Using these techniques, early technical success (immediate haemostasis) of 90-95% can be achieved with clinical success (no re-bleeding in the first 30 days) of ~75%. Mild ischaemic complications (transient abdominal pain, asymptomatic ischaemic
stricture) are reported in ~10% of cases with severe complications (symptomatic ischaemic stricture, frank bowel infarction) reported in 2% of cases. Bleeding diverticula are especially well managed with this technique.

References