MASSIVE GASTROINTESTINAL HAEMORRHAGE: THE RADIOLOGIST'S ROLE IN DIAGNOSIS AND MANAGEMENT

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SYNOPSIS

Massive gastrointestinal haemorrhage is a life-threatening condition. Angiography can play an important role in determining the source and aetiology of the bleeding. Application of therapeutic embolisation may be life-saving. Four cases of massive gastrointestinal bleeding from rather unusual causes are presented to illustrate the role of the radiologist.

INTRODUCTION

Gastrointestinal bleeding may arise from the upper or the lower gastrointestinal tract. Upper GI bleeding presents with hemetemis and/or malaena and lower GI bleeding with bleeding per rectum. However when bleeding is massive it may be difficult to differentiate betweeen the two because fresh blood may be vomited or passed per rectum regardless of the site of bleeding.

The common causes of massive upper GI bleeding are benign ulcers, erosions, varices and Mallory-Weiss tears. Tumours rarely cause massive bleeding.

Massive lower GI bleeding is less common and is usually due to diverticular disease. Polyps and other tumours seldom bleed massively. Angiodysplasia of the colon is being increasingly identified as a cause (1, 2). An incidence of 5% has been reported for acute bleeding from the small bowel (3).

Endoscopy is now well-established as the most accurate examination in the detection of bleeding upper GI lesions (4). The advent of double contrast technique has improved the detection rate in barium meal examinations but not to the level of endoscopy. Besides, this technique requires some patient co-operation which is often unobtainable from an acutely ill patient.

In recent years, angiography has been popularised in the examination of acute GI bleeding. Not only does this examination allow the bleeding site to be localised (2, 5, 6, 7), it holds the potential of allowing measures to be taken to arrest the bleeding. This can be done either by selective arterial perfusion of vaso-constrictor agents (7, 8) or by therapeutic embolisation of the bleeding vessel (1, 9, 10, 11, 12).

In this paper, we present 4 patients who suffered massive gastrointestinal haemorrhage from rather uncommon causes. Angiography played an essential role in the diagnostic work-up and in two patients therapeutic embolisation helped considerably in the management.

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Case Reports

1. HCC, (13) a 69 year old man, was seen because he was passing large amounts of malaena for two days. He had a low B.P. of 100/60 and a slight tachycardia. Nasogastric aspiration ws clear while the rectum was full of stale blood during sigmoidoscopy. 11 litres of blood were needed over the next 18 days because he continued to pass out large amounts of malaena and blood-stained stools.

A barium enema examination showed an extrinsic indentation at the splenic flexure. At coeliac angiography, the mass was seen to be in the tail of the pancreas with tumour vessels being supplied by the transverse pancreatic artery and branches of the splenic artery. There was no extravasation of contrast medium.

Subsequent laparotomy and histopathological examination confirmed a 7-8 cm undifferentiated carcinoma of the tail of the pancreas. The tumour was haemorrhagic and necrotic and was invading into the colon.

2. LKS was found to have leiomyosarcoma of the duodenum with secondaries in the liver. He suffered episodes of massive malaena requiring many units of blood transfusion.

Barium meal examination showed widening of the duodenal loop with an ulcer on the surface of the tumour. Angiography (Fig. 1 & 2) demonstrated tumour vessels supplied by the duodeno-pancreatic arcades. Numerous gelfoam pellets were injected via the superior mesenteric artery to attempt to stop the bleeding (Fig. 3).

The patient developed some abdominal pain and vomiting on the subsequent two days. Malaena stopped but a small episode recurred a week later.

Two months later he began to have massive malaena again. Repeat angiography (Fig. 4) showed that the tumour vessels have re-opened being mainly supplied by the gastroduodenal artery. Re-embolisation was carried out but the effect was very transitory. He continued to pass malaena in large amounts on and off and required many units of blood till he died after about eight months of illness.

3. LWC, a 15 year old girl, was suffering from pulmonary and abdominal tuberculosis. She was emaciated, anaemic and had ascites. Before she could be started on anti-tuberculous chemotherapy, she began to pass massive amounts of malaena. This became fresh blood some hours later. Blood coagulation studies were abnormal. Blood and fresh plasma transfusions were ineffective.

As she was considered unfit for surgery, emergency angiography was carried out to locate the bleeding site for possible therapeutic embolisation. Superior mesenteric angiography (Fig. 5) revealed extravasation of contrast medium from a distal jejunal artery. This was selectively catheterised and occluded with several small pieces of gelfoam (Fig. 6).

Over the next twenty-four hours she passed small amounts of malaena but did not require any more blood transfusions. Subsequently she made a satis-

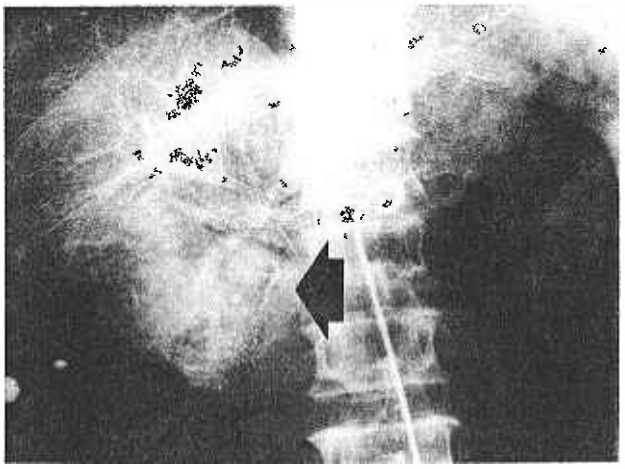

Fig. 1(a)

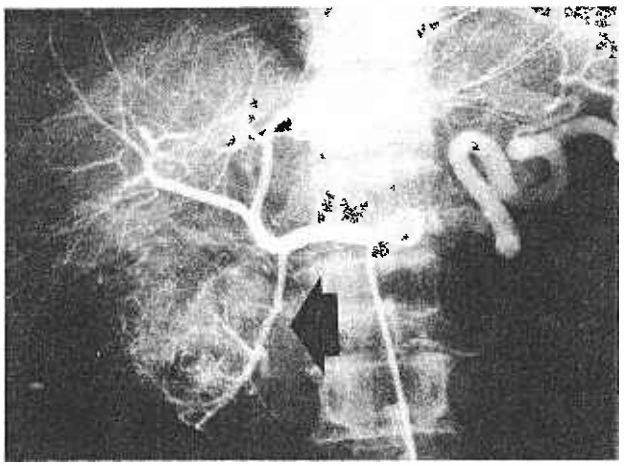

Fig. 1(b)

Fig. 1 : Leiomyosarcoma of duodenum. Hepatic arteriogram a) arterial b) capillary phase. There is neovascularity and tumour staining supplied by the gastroduodenal artery.

Fig. 2 : Leiomyosarcoma of duodenum. Superior mesenteric arteriogram, venous phase. Similar tumour staining as in the hepatic arteriogram because of a large inferior pancreaticoduodenal artery. A metastatic lesion in the liver is revealed.

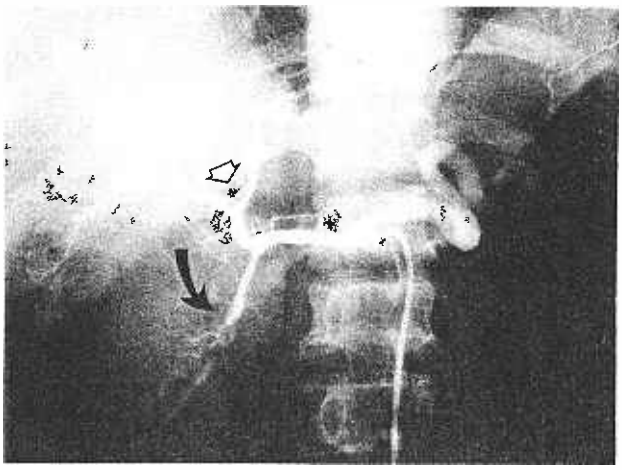

Fig. 3 : Leiomyosarcoma of duodenum. Post-embolisation angiograms showing complete occlusion of tumour vessels. Proximal gastroduodenal artery is still patent. Accidental embolus is lodged at the bifurcation of the hepatic artery.

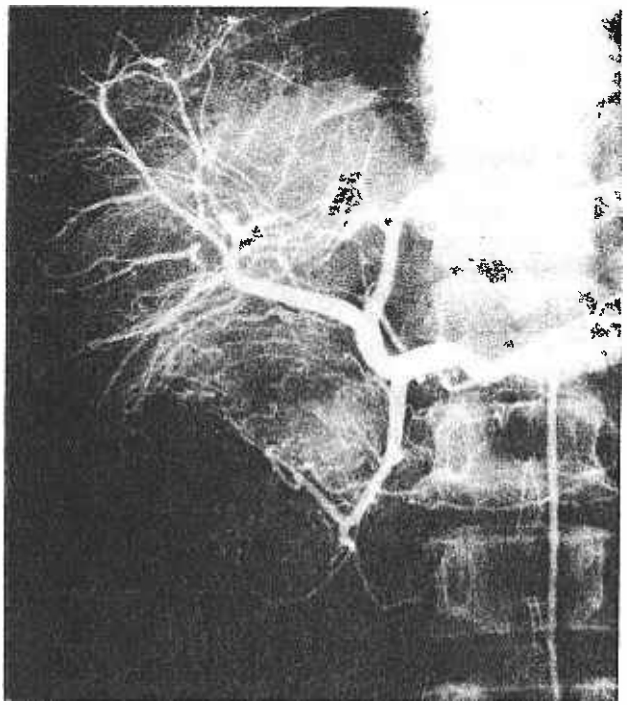

Fig. 4(a)

factory recovery on anti-tuberculous treatment without any recurrence of bleeding or other complications.

4. A.S. was a 48 year old man suffering from advanced alcoholic cirrhosis, hyperuricaemia and chronic renal failure. He was admitted because of one episode of haemetemesis and was severely anaemic. A few hours later he began to pass out large amounts of blood clots per rectum. Endoscopy of the stomach, duodenum and sigmoid colon did not show a bleeding source. He required many units of blood in the following two weeks as he continued to bleed per rectum.

At superior mesenteric angiography (Fig. 7), the source of bleeding was pin-pointed to an arteriovenous malformation in the ascending colon. This was resected surgically but the patient did not survive the operation.

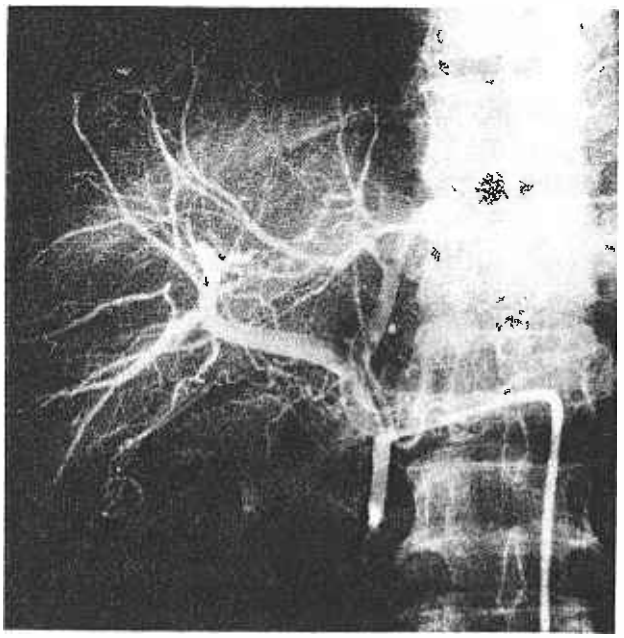

Fig. 4(b)

Fig. 4 : Leiomyosarcoma of duodenum. Follow-up angiograms a) at two months showing recanalisation of tumour vessels b) after repeat embolisation. Gastroduodenal artery is occluded in the distal half. Only a rim of tumour vessels is still being supplied from hepatic branches.

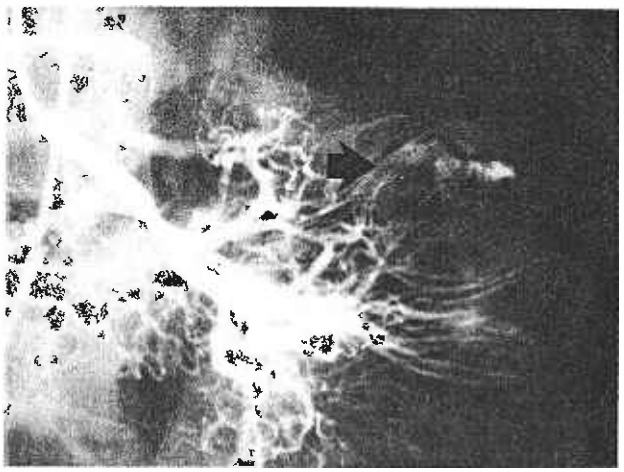

Fig. 5 : Tuberculous ulcer of distal jejunum. Selective jejunal arteriogram. This confirms catheterisation of the bleeding artery. The arterial arcades are well shown. Arrow points to extravasted blood.

DISCUSSION

Massive gastrointestinal haemorrhage can become a life-threatening condition if it does not respond to blood transfusions and bed rest. Endoscopy is the diagnostic procedure of choice to locate an acute upper gastrointestinal bleeding lesion. If this fails to detect the source of bleeding, then angiography should be employed. Barium studies of the gastrointestinal tract should not be performed if the bleeding is massive because it would make angiography impossible for several days.

It has been established in the dog, that active bleeding at a rate of 0.5 cc per minute into the gastrointestinal tract can be detected angiographically. (5)

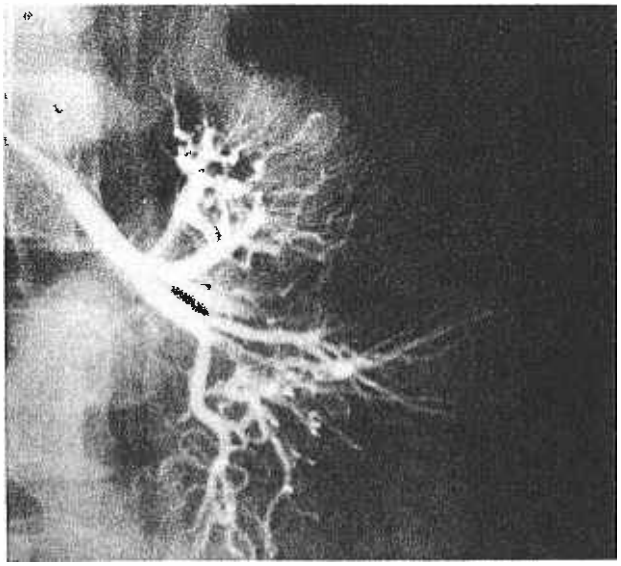

Fig. 6 : Tuberculous ulcer of distal jejunum. Post-embolisation arteriogram. Bleeding has been completely arrested.

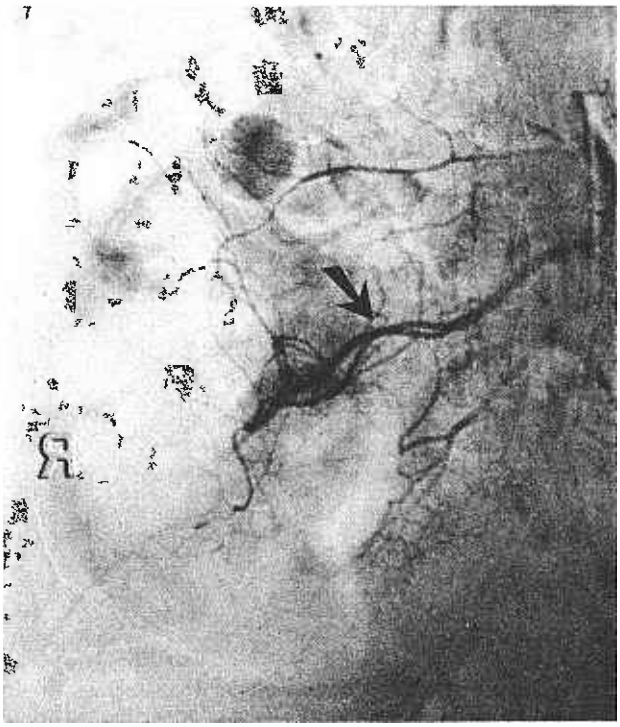

Fig. 7(a)

This is shown by contrast medium extravasation. Once such a bleeding point is demonstrated, vasopressin infusion or therapeutic embolisation can be instituted in an attempt to stop the bleeding. This can save the patient from undergoing a surgical operation or allow him to recover to a more fit state for surgery.

While intra-arterial vasopressin infusion has been used with good success in acute gastrointestinal bleeding, it nevertheless has some drawbacks. These include the long duration in which the catheter is left lying within the artery, the need for a rotary infusion pump, side effects of arrythmias especially bradycardia, fluid retention and some failures and rebleeds. (1, 2) However, diffuse mucosal bleeding is best treated by intra-arterial vasopressin infusion. (7).

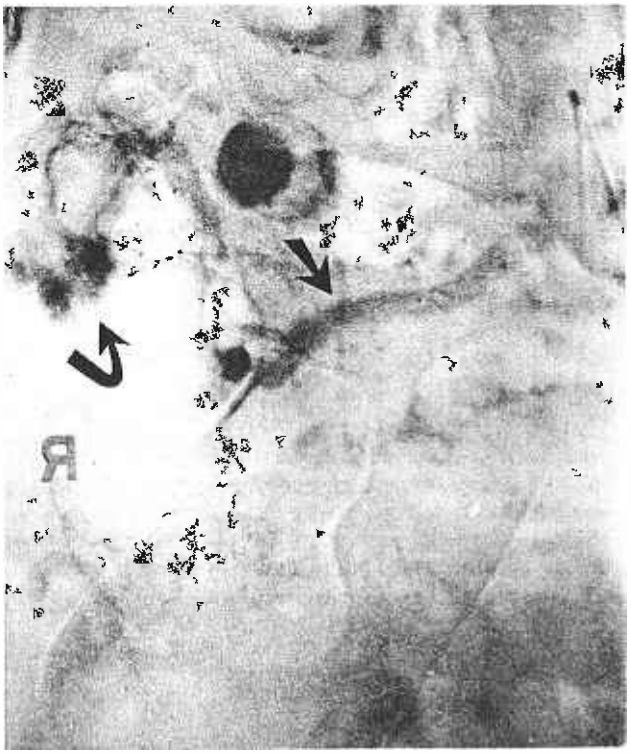

Fig. 7(b)

Fig. 7 : Arteriovenous malformation of colon. Subtraction prints of superior mesenteric arteriogram. a) arterial phase b) capillary phase. Note the large ileocolic artery and the large early draining vein. There is extravasation of contrast medium. (curved arrow)

Enough experience on therapeutic embolisation has accumulated since Rosh et al's report in 1972 (11) to establish the efficacy and safety of this procedure. (1, 9, 10, 12) Rebleeding from the lesion is less common than after vasopressor therapy. Complications are usually due to poor control of the emboli because of inadequate selectivity of catheterisation. Infarction and necrosis of bowel is unlikely because of the rich collateral circulation. However, pre- or post-embolisation gastric surgery increases the risk of infarction because of the attendant surgical devascularisation. (9, 12).

Materials used as therapeutic emboli are numerous. They can be classified as short-acting, long-acting or permanent. The most easy to use are homologous blood clot, gelfoam and polyvinyl alcohol (Ivalon). Homologous blood clot is short-acting and rebleeding is likely. Polyvinyl alcohol causes permanent occlusion (14) and is useful for bleeding tumours and angiodysplasia. On the whole, gelfoam is the most suitable material for gastrointestinal bleeding for benign and self-limiting lesions as it gives a long-acting occlusion of 3 to 4 weeks. (12)

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