Small bowel capillary dilatation in portal hypertension

Charles Gallagher¹, Fiona Bonar¹, James Dempsey² and John Crowe ¹

The Departments of¹ Medicine and ²Pathology, University College Dublin, Mater Hospital, Dublin, Ireland.

Summary: A patient with small bowel capillary dilatation and cirrhosis is reported. This patient had persistent, unexplained gastrointestinal bleeding. Small bowel capillary dilatation appears to be unique to patients with portal hypertension. The possible role of small bowel capillary dilatation in causing gastrointestinal bleeding is discussed.

Introduction

There is little information in the literature on small bowel histology in patients with portal hypertension but most authors feel that it is normal (Rubin & Dobbins, 1965). This report concerns a patient with portal hypertension and small bowel capillary dilatation, a finding which may be unique to patients with portal hypertension.

Case report

In April 1978 a 42 year old woman presented with a 6 month history of ankle and abdominal swelling, diarrhoea and weight loss of 9 kg. She denied any alcohol consumption. Examination showed wasting, ascites and marked peripheral oedema.

Laboratory investigation showed a haemoglobin level of 14.2 g/dl and a mean corpuscular volume of 100 fl. A blood smear showed Howell-Jolly bodies. White cell count was \(6.7 \times 10^9\) l with a normal differential, and platelet count was \(170 \times 10^9\) l. Serum folate was 1.4 ng/ml (normal 6–21) and red cell folate was 60 ng/ml (normal 160–600). Serum iron was 22 μmol/l (normal 11–29) with a total iron binding capacity of 56 μmol/l (normal 48–70) and 2.2 g of a 25 g oral load of D-xylose was excreted over 5 h. Serum albumin and globulin were 21 and 32 g/l, respectively. Alkaline phosphatase was 546 U/l (normal 25–90). Isoenzyme analysis showed that this was all hepatic. Serum glutamic-oxaloacetic transaminase level was 63 U/l (normal 40) and total bilirubin level was 0.6 mg/dl. Smooth muscle antibody was found in blood but mitochondrial antibody, antinuclear antibody and hepatitis B surface antigen were not. Average daily faecal fat excretion was 22 g.

Upper gastrointestinal endoscopy showed small oesophageal varices with diameter up to 3 mm. They were not tortuous. Endoscopy was otherwise normal. Biopsy of the descending duodenum showed partial villous atrophy and liver biopsy showed macronodular cirrhosis. Barium swallow and meal showed only small oesophageal varices. Barium follow through was normal. A diagnosis of cirrhosis possibly secondary to chronic active hepatitis and probable coeliac disease was made. Treatment was commenced with spironolactone, folic acid, salt restriction and a gluten free diet. Her family confirmed that she adhered to the diet.

Her clinical condition continued to deteriorate and 2 months later prednisone was started. In July 1978 she developed persistent melaena. Her haemoglobin was 5.5 g/dl with a hypochromic microcytic blood film. Endoscopy while melaena persisted again showed small oesophageal varices which were not bleeding. The stomach and duodenum were normal and there was no blood in the stomach. Barium swallow and meal again showed only small oesophageal varices. Colonoscopy, barium enema and barium follow through were all performed while she had melaena and were normal. Angiography was performed by selective catheterization of the inferior mesenteric, superior mesenteric and coeliac arteries while she had melaena. Oesophageal varices were seen but there was no evidence of bleeding. No other lesion was seen. In particular, there was no evidence of extra-oesophageal varices during the venous phase.

Her melaena persisted over the next 18 months and she required multiple blood transfusions. Repeated endoscopy while she had melaena showed small oesophageal varices with no evidence of bleeding.

Correspondence: J. Crowe, Ph.D., F.R.C.P.I., Mater Hospital, Dublin, Ireland.

*Present address: Respiratory Investigation Unit, University of Manitoba, Winnipeg, Canada

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Barium swallow, meal and follow through, colonoscopy and barium enema were unhelpful. Intubation enterography showed no evidence of a bleeding site. Duodenal biopsy in February 1979 showed partial villous atrophy. Repeat angiography while melaena persisted showed only oesophageal varices and no bleeding site was seen. Laparotomy with gastroscopy and colonoscopy during surgery was unhelpful. This was also done while she had melaena. Her melaena continued and she required blood transfusions approximately once every 2 months.

At endoscopy in January 1980, multiple areas of hyperaemic mucosa were noted in the duodenum. Histology revealed grossly dilated capillaries containing debris of erythrocytes in the villi of the duodenum and jejunum (Figure 1). In retrospect the previous sections of February 1979 showed a milder form of this (Figure 2). Her gastrointestinal bleeding continued and a portacaval shunt was performed. She died postoperatively. Autopsy permission was not given.

Figure 1  Jejunal biopsy in January 1980 showing stunted villi, abnormal surface enterocytes and dilated capillaries containing debris of erythrocytes. The capillaries are separated from the bowel lumen only by surface enterocytes. Magnification × 1200.

Figure 2  Duodenal biopsy in February 1979 showing severe partial villous atrophy and lamina propria largely replaced by dilated capillaries containing debris of erythrocytes. Magnification × 500.

Discussion

This case report prompts discussion under two headings: firstly, the small bowel capillary dilatation seen in this patient and its possible association with portal hypertension; secondly, the cause of gastrointestinal bleeding in this patient.

Capillary dilatation was seen in both duodenal and jejunal mucosa in our patient and in biopsies 11 months apart. There is relatively little information in the literature on small bowel histology in liver disease but most authors state that it is normal (Rubin & Dobbins, 1965; Summerskill & Moertel, 1962). Norman et al. (1980) found abnormal dilatation of intercellular spaces in jejunal mucosa of patients with portal hypertension but no mention was made of capillary abnormalities. However, Astaldi & Strosseli (1960) suggest that small bowel biopsies of patients with hepatic cirrhosis may show vascular stasis. Their figure 7 demonstrates capillary dilatation similar to that seen in our patient with the additional feature of haemorrhagic extravasation into the stroma. Bank et al. (1964) noted ‘a striking increase in vascularity of the villi’ in the small bowel biopsy of a patient with cirrhosis and oesophageal varices. Unfortunately they did not show histology of the small bowel. We could not find any other reports of small bowel capillary dilatation as seen in our patient in other disease states or in normal subjects. We therefore suggest that this biopsy appearance is found only with portal hypertension but further studies are needed to confirm this. The capillary dilatation is probably caused by increased pressure in the portal system. It is interesting that none of the four patients with cirrhosis and normal small bowel biopsies described by Summerskill & Moertel...
had oesophagogastric varices. This supports our view that capillary dilatation is associated with portal hypertension rather than with abnormal hepatic function. Our patient’s first small bowel biopsy in April 1978 did not show dilated capillaries. It could be speculated that the capillary changes became more marked as growth of villi occurred in response to a gluten free diet but normal villi were never seen. The initial presumptive diagnosis of coeliac disease was therefore never proven but it seems likely that this patient had gluten enteropathy. The possible association in this patient between chronic active hepatitis and coeliac disease is in keeping with previous reports (Pollock, 1977; Swarbrick et al., 1980).

Gastrointestinal bleeding is the commonest presentation of portal hypertension and is often recurrent. Small oesophageal varices were present in this case. The absence of blood in the stomach and, less importantly, the absence of variceal bleeding at angiography and endoscopy suggest that they were not the cause of bleeding. Repeated endoscopies showed none of the other common causes of bleeding. There was no evidence of varices of the small or large bowel (Moncure et al., 1976; Gray & Grollman, 1974) at the venous phase of angiography on two occasions. Angiography showed no evidence of angiodysplasia or other vascular lesions in this case. There is no good information on the false negative rate of the arteriographic diagnosis of angiodysplasia and therefore we cannot completely rule out this possibility.

Our patient’s dilated capillaries were never seen to be actively bleeding. However, gastrointestinal capillary bleeding is very difficult to demonstrate and may go undiagnosed for many years (Feller et al., 1971). In the absence of other bleeding sites, we therefore suggest that the capillary dilatation may have been the cause of bleeding in this patient. It must be emphasized that this hypothesis is speculative. It is interesting that, despite emergency endoscopy, no cause of bleeding is found in up to 27% of patients with portal hypertension and gastrointestinal bleeding (Franco et al., 1977; Waldram et al., 1974). It is possible that some of these patients are bleeding from dilated small bowel capillaries. We therefore suggest that patients with portal hypertension and persistent unexplained gastrointestinal bleeding should have small bowel biopsies.

In conclusion, small bowel capillary dilatation appears to be unique to patients with portal hypertension. Further studies are needed to confirm this and to investigate the possible role of this lesion in causing gastrointestinal bleeding.

References


NORMAN, D.A., ATKINS, J.M., SEELIG, L.L., GOMEZ-SAN-}


