was found to have hepatosplenomegaly which had first been noted in 1958. A liver biopsy showed the pathognomonic features of PBC with bile-duct destruction, proliferation of the bile ductules, granulomata and an inflammatory cell reaction (Scheuer, 1967) (Figs 1 and 2). At that time the total bilirubin was 9 $\mu\text{mol/l}$, alkaline phosphatase 8 KAu./100 ml (upper normal limit 17 KAu.), alanine aminotransferase 95 u. (normal range, 4–17 u.), aspartate aminotransferase 168 u. (normal range 3–12 u.), IgM 2.8 g/l (normal range 0.3–2.0 g/l), mitochondrial antibodies were present in a titre $> 1/1280$, positive bile canaliculi antibodies, negative smooth muscle antibodies and negative hepatitis-associated antigen. A wedge biopsy performed at laparotomy confirmed the diagnosis of PBC.

She remained well until 1970 when she developed diabetes mellitus and pancytopenia with a hypercellular bone marrow ascribed to hypersplenism. In 1975 she was admitted with ascites. At that time she was deeply pigmented but anicteric and without pruritus. A repeat liver biopsy showed progression of her PBC to a stage 4 cirrhosis (Scheuer, 1967). She responded to diuretics, but in 1976 she was readmitted with a haematemesis and died. Although a barium swallow had shown oesophageal varices, the cause of her haematemesis was not determined and no post-mortem examination was allowed.

Her alkaline phosphatase and 5-nucleotidase levels remained normal throughout her illness. Thus the alkaline phosphatase was 8 KAu. in 1963, 8 in 1967, 14 in 1970, 8 in 1975 and 11 KAu. in 1976. On electrophoresis the alkaline phosphatase was predominantly of hepatic origin with a minor component showing the mobility of the bone isoenzyme.

Discussion

This case has several unusual features. Despite evidence of progressive disease she had a prolonged survival without jaundice or pruritus. Zeegen *et al.*, (1969) found that patients can develop PBC and portal hypertension without the classical symptoms of PBC. However, the alkaline phosphatase was raised in every case.

It has been postulated (Schaffner, 1975) that the raised alkaline phosphatase is due to bile regurgitation into the portal tract tissues through damaged ducts. In their review, Sherlock and Scheuer (1973) reported just two patients, both discovered incidentally, who had an initial alkaline phosphatase of less than 20 KAu., but no follow-up was recorded.

The present patient had a persistently normal alkaline phosphatase until her death and is believed to be the first such case reported. This underlines the lack of specificity of biochemical tests and, while histology is often not diagnostic (Scheuer, 1967), it emphasizes its value in diagnosing the disease.

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Gastro-colic fistula secondary to benign gastric ulceration

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Summary

An 80-year-old woman developed osteoarthritis of the knees as a complication of her obesity. Indomethacin treatment precipitated gastric ulceration, which led to the formation of a gastro-colic fistula.

GASTRO-COLIC fistula is a rare condition and, unlike gastro-jejuno-colic fistula which usually occurs secondary to partial gastrectomy, is most often a complication of invasive gastric or colonic carcinoma (Cody *et al.*, 1975). Other possible causes include

Crohn's disease, intra-abdominal abscess and tuberculosis. The following report describes a case of gastro-colic fistula resulting from a benign gastric ulcer.

Case report

An 80-year-old woman was admitted to hospital following several haematemeses. She gave a 6-month history of intermittent vomiting after meals. The vomitus had initially consisted of unaltered food only, but later contained 'coffee ground' material and occasional streaks of fresh blood. One week before admission small amounts of fresh blood were passed per rectum followed by frequent melaena stools. At this point the vomitus became foul-tasting and her husband complained that her eructations made a foul smell in the room. Over the 6-month period she had lost 20 kg in weight but, apart from general malaise, there were no other associated symptoms. Her only significant past history was that of osteoarthritis affecting both knees for which she had been taking indomethacin (50 mg, b.d.) for 5 years.

Although only 1.35 m tall she was grossly overweight at 85 kg. She was pale and looked generally unwell. There was tenderness on palpation with associated guarding in the left hypochondrium. Examination was otherwise normal.

Hb was 10.8 g/dl, with a microcytic, normochromic picture and ESR (Westergen) of 77 mm in the first hour. Blood urea was slightly elevated at 10.8 mmol/l and serum potassium low at 3.3 mmol/l. Iron deficiency was confirmed with serum iron of 8 µmol/l. and total iron binding capacity of 61 µmol/l. Faecal occult blood was positive. Barium meal was performed because of the high possibility of gastric carcinoma, but this demonstrated a gastro-colic fistula with no evidence of tumour or other abnormality.

After blood transfusion and fluid and electrolyte replacement, laparotomy was carried out. This showed the transverse colon to be adherent to the greater curvature of stomach by an area of dense

fibrous thickening and, on opening the stomach, that an underlying deep ulcer crater was present. The connection was completely excised and stomach and colon separately closed. On histological examination the gastric ulcer was benign with evidence of focal haemorrhage and a granulation tissue-lined fistula led from the base of the ulcer through to normal colonic mucosa.

After a difficult postoperative period she made a complete recovery and was very well at follow-up 6 weeks later.

Discussion

Indomethacin is a well recognized cause of benign gastric ulceration (Boardman and Hart, 1967) and a common site for such ulcers is the greater curvature of stomach (Emmanuel and Montgomery, 1971). It is suggested that in the present case an indomethacin-induced ulcer underwent localized perforation causing a fistulous connection to transverse colon. Such painless perforation is uncommon but not unknown (Haubrick, 1974). Diagnosis was confirmed by barium meal although this is possible in only 25% of cases of gastro-colic fistula, barium enema being the investigation of choice (Thoeny, Hodgson and Scudamore, 1960).

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