Summary

Three cases of intestinal obstruction directly attributable to the use of a hydrophilic colloid laxative have been reported. A survey of the literature has revealed only two similar cases.

The pathological process in this type of obstruction is discussed, and some suggestions are made with regard to its management.

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Medical Memoranda

Hepatic Coma due to Liver Metastases

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Hepatic coma occurs as a complication of almost any serious diffuse liver disease, such as cirrhosis and acute hepatocellular necrosis. Sherlock (1958) in 108 cases of hepatic coma saw no hepatic malignancy, while Willis (1960) in 120 cases of hepatic coma from Singapore saw eight cases of hepatoma and one case of metastasizing carcinoma of the stomach.

CASE HISTORY

A man aged 59 was admitted on 19 August 1963 after seven weeks' upper abdominal pain. Previous investigations revealed no abnormal signs. The results of a barium-meal examination and radiographs of the spine and chest were normal. A diagnosis of peptic ulcer had been made, but despite a gastric diet and antacids pain increased. On admission he complained of anorexia and a loss of 2½ st. (15.9 kg.) in two months. There was no nausea, vomiting, haematemesis, melaena, or any alteration of bowel habits. Examination revealed a slightly distended abdomen with some free fluid. The liver was hard, irregular, slightly tender, and was palpable 5 cm. below the ribs and 11 cm. from the xiphisternum. The sclerae were icteric. A small polypoid mass was found in the rectum. Biopsy and histology revealed a benign polyp. The results of further physical examination were normal.

The results of investigations on admission were as follows: haemoglobin 91% (13.2 g./100 ml.), W.B.C. 11,400/c.mm., E.S.R. 45 mm. in one hour (Westergren), blood urea 20 mg./100 ml., serum sodium 117 mEq/l., potassium 3.7 mEq/l., chlorides 93 mEq/l., serum bilirubin direct positive (3.6 mg./100 ml.), alkaline phosphatase 40 units, flocculation tests were normal, S.G.O.T. 80 units/ml., total serum protein 5 g./100 ml. (albumin 3.9 g., globulin 1.1 g.), stool benzidine was negative, prothrombin concentration 12%. Results of a barium enema were negative.

His condition deteriorated in the first week (26 August) and he developed mild peripheral circulatory failure, but no drop of blood-pressure (140/90–120/80 mm. Hg). The left foot became cold and blue, arterial pulsations were absent below both popliteals, and he became more jaundiced and apathetic. Biochemical investigations showed serum bilirubin 10 mg./100 ml., serum alkaline phosphatase 72 units, normal flocculation tests, S.G.O.T. 29 units/ml., serum cholesterol 130 mg./100 ml., serum proteins were unaltered. Next day serum bilirubin was 23 mg./100 ml., alkaline phosphatase 48 units, S.G.O.T. 420 units/ml., blood urea 115 mg./100 ml.

On 27 August he became disorientated and drowsy, fetor hepaticus was first noticed, and flapping tremors of outstretched hands were demonstrated. Severe hepatic coma developed with intense jaundice, and test results were serum bilirubin 23 mg./100 ml., alkaline phosphatase 48 units, flocculation tests normal, S.G.O.T. 420 units/ml., blood urea 115 mg./100 ml., while serum electrolytes and bicarbonate remained unaltered. Despite treatment with oral neomycin, withdrawal of protein, and maintenance of fluid and electrolyte balance, he became comatose and died 11 days after admission.

At necropsy Dr. Manners (consultant pathologist) demonstrated metastases in the liver. There were massive, multiple carcinomatous nodules with central necrosis, and macroscopically there was little remaining liver tissue. Lungs showed scattered small metastases beneath the pleurae. The body of the pancreas was completely replaced by carcinomatous tissue extending into the tail, but the head was normal. The brain was carefully dissected but showed no metastases. The kidneys were normal.

COMMENT

The patient died of hepatic coma due to massive liver metastases from a primary carcinoma of the pancreas. This pathological sequence is an unusual occurrence in the United Kingdom. Watkinson (personal communication, 1963) has never seen this, since the majority of patients with metastases in the liver die from other causes before complete failure of liver function. Approximately 85% of the liver tissue was destroyed by neoplasm in this patient with little constitutional upset until the last two months. In chronic hepatic disease such an accelerated course of hepatic coma is unusual without severe precipitating factors—such as massive gastro-intestinal bleeding (occult-blood tests on the patient were negative), hypotension (his blood-pressure was maintained above 120/70 mm./Hg), infections (none were found at necropsy), electrolytic disturbance (serum sodium was consistently low—117 mEq/l., and serum potassium was from 3.7 to 5.8 mEq/l.), or shock (a mild degree observed three days before death). Rapid increase in serum alkaline phosphatase and bilirubin suggested acute massive hepatocellular necrosis with obstructive jaundice. Rapid elevation of S.G.O.T. to 420 units/ml. in hepatic coma suggested Budd-Chiari syndrome, but the necropsy disproved this. Pryse-Davies and Wilkinson (1958) stated that about 50% of patients with liver metastases show slightly raised S.G.O.T. (ranging from 35 to 100 units). Levels exceeding 100 units are found only in patients in whom liver necrosis accompanies massive liver metastases. The present findings could indicate that massive liver metastases alone could cause marked elevation of S.G.O.T.

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