Fatality after hepatic angiography in Zieve's syndrome

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Summary

A patient with Zieve's syndrome is described. Following hepatic angiography, the patient became shocked and eventually died. The hazards of hepatic angiography are discussed and its use in ill patients questioned.

Introduction

The syndrome of jaundice, hyperlipaemia and haemolytic anaemia associated with alcoholic liver disease was first described by Zieve in 1958. A case of Zieve's syndrome is described in which, following hepatic angiography, the patient rapidly deteriorated and eventually died.

Case report

A 66-year-old female was referred to a haematological out-patient clinic with a 10-month history of tiredness, breathlessness, anorexia and weight loss. Apart from occasional attacks of cystitis, her past medical history was uneventful and her family history unhelpful. She admitted to a generous regular alcohol intake, approximately half a bottle of whisky daily.

On examination, she was jaundiced, clinically anaemic and there were signs of recent weight loss. Jugular venous pressure was not elevated but there was mild ankle oedema. The pulse was 100/min and regular, BP 110/80 mmHg and cardiac auscultation was normal. The abdomen was distended and ascites was demonstrated. The liver was hard, non-tender and 4 cm enlarged. A systolic bruit was heard over the liver. The spleen was not palpable.

Investigations

Haemoglobin, 5.7 g/dl; haematocrit 0.200; MCV 118/l; MCHC, 28.5 g/dl; platelets, $140 \times 10^9/l$; WBC, $30.1 \times 10^9/l$; differential, neutrophils, 92%; lymphocytes, 6%; monocytes, 2%; ESR 41 mm in the first hour; reticulocytes, 11.5%. In the peripheral blood film, the red blood cells were macrocytic, normochromic with moderate polychromasia. Prothrombin time 1.5 : 1. Serum B₁₂, 479 ng/l; serum folate, 2.1 µg/l. Coombs' test was negative.

Urea, 5.2 mmol/l; Na, 134 mmol/l; K, 3.3 mmol/l; total CO₂, 24 mmol/l. Serum bilirubin, 275 µmol/l; alanine aminotransferase, < 10 u./l; alkaline phosphatase, 348 u./l. Total protein, 69 g/l; albumin, 33 g/l; α-fetoprotein negative. Serum cholesterol, 9.3 mmol/l; triglycerides, 1.9 mmol/l. A technetium liver scan suggested either multiple small metastases or hepatic cirrhosis. Shortly after admission, the patient developed a low grade fever.

She was transfused with 6 units of packed cells and received vitamin K, neomycin, spironolactone, and a low sodium and protein diet. Four days after admission, the reticulocyte count had fallen to 2.3% and it remained around this level. Hepatic angiography demonstrated an increased arterial supply to an enlarged liver and the appearances were in keeping with an early stage of cirrhosis. Unfortunately, following this procedure, her blood pressure fell to a systolic pressure of 80 mmHg and the pulse rate increased to 120/min. She received 300 ml salt-poor albumin intravenously and because of the deepening jaundice, the possibility of septicaemia was considered. She was started on gentamicin and lincomycin intravenously and was transfused a further unit of packed cells. Her bilirubin during this period rose to 450 mmol/l and 5 days later there was a transient rise in the serum alanine aminotransferase to 75 u./l. Her serum sodium was persistently low and her blood urea had risen to 11 mmol/l. Her electrolyte imbalance proved resistant to all measures tried to correct it and her condition gradually deteriorated until she died 7 weeks after admission.

At post-mortem, the peritoneal cavity contained 3.5 litres of clear bile-stained fluid. The liver weighed 1.3 kg, had a finely nodular surface and was greenish in colour. On cut section, the parenchyma was deeply bile-stained and a micro-nodular pattern of cirrhosis was detectable. There was no evidence of hepatoma. Microscopic examination showed extensive fibrosis of the portal triads without evidence of regeneration, and extensive ante-mortem necrosis with polymorphonuclear infiltration. The surviving hepatocytes contained large amounts of Mallory's hyaline. The appearances were consistent with severe

and prolonged liver damage due to alcohol, and not cirrhosis. No abnormalities were demonstrated in the biliary system or spleen.

Discussion

Selective hepatic angiography was first described by Bierman *et al.* in 1951. Since that time, it has been used in the diagnosis of a wide variety of liver diseases (Alfidi *et al.*, 1968). The hazards of angiography include infection, embolization, thrombosis, extravasation of dye, toxic effects of the contrast medium and damage to other blood supplies to vital organs. In 1974, Goldstein and Bookstein monitored the liver function tests in 26 patients having hepatic angiography and they reported only 2 transient elevations of the serum transaminase levels. Ritchie (1975) using a canine model, suggested that contrast media, in the presence of induced endothelial damage, could produce more extensive damage and severe inflammatory changes, together with red cell aggregation and fibrin formation. He also noted the formation of thrombus.

Although the findings in the present patient were consistent with a diagnosis of Zieve's syndrome, the possibility of hepatoma or hepatic metastases had been raised. The technetium liver scan was unhelpful, as the changes in alcoholic hepatitis can resemble neoplastic deposits. Hepatic angiography was therefore carried out to exclude neoplasia before embarking on prolonged intensive medical therapy in an ill patient. Unfortunately the patient was shocked following her hepatic angiography and over the subsequent 48 hr she became more jaundiced, the serum bilirubin level rising to 450 mmol/l. In addition, 4 days after the procedure, the serum

alanine aminotransferase level rose transiently to 75 i.u. There was no evidence of further haemolysis to account for these changes. At that time, it was considered that her symptoms could be the result of a bacteraemia but no organism was isolated and no clinical improvement occurred following antibiotic treatment. Her condition had been improving until the time of the hepatic angiography but from that time, her condition rapidly deteriorated. Whether this was the result of a single complication of angiography overwhelming a compromised liver, or a combination of the hazards is impossible to say. Complications of diagnostic arteriography are considered to be uncommon but perhaps the use of hepatic angiography in the ill patient should be reconsidered in the light of this experience.

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