MEDICAL MEMORANDA

Preoperative Diagnosis of Intrahepatic Mycotic Aneurysm

J. HYWEL JONES, C. B. JONES, D. L. ASBURY

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Aneurysm of the hepatic artery is a rare cause of gastrointestinal bleeding. The diagnosis is usually made at laparotomy but is difficult when the aneurysm is intrahepatic. This paper describes the fourth case in which the diagnosis of intrahepatic aneurysm of the hepatic artery was made before operation.

Case Report

A 30-year-old housewife was transferred to Manchester Royal Infirmary in November 1970 because of recurrent haematemesis. As a child she had an attack of rheumatic fever which left her with mitral incompetence but good exercise tolerance. In 1966 during her first pregnancy she developed subacute bacterial endocarditis. This was complicated by a right hemiplegia attributed to a mycotic aneurysm shown by cerebral angiography. Her baby was born prematurely in January 1967. In April she was admitted under the care of Dr. H. J. Wade because of fever and severe headaches diagnosed as a cerebral abscess. The infection resolved after a two-month course of penicillin.

She remained well until July 1968 when she had an attack of severe epigastric pain radiating between the shoulder-blades followed immediately by haematemesis, which relieved the pain. Over the next two years she had recurrent attacks of the same type which necessitated frequent blood transfusions. She had no dyspepsia but barium-meal examinations showed deformity of the duodenal cap, and in August 1970 a partial gastrectomy was performed. The operation specimen showed scarring of the duodenum but no ulcer crater. A fortnight later she was re-admitted with a further attack of pain and haematemesis. Over the next few months she had recurrent haematemesis of increasing severity, which culminated in her admission to Manchester Royal Infirmary for further investigation.

On admission she was anaemic and had signs of mitral incompetence with evidence of a residual right hemiplegia. There were no other abnormal signs. The haemoglobin was 8.9 g/100 ml, with a normocytic, normochromic blood film. Liver function tests were: serum bilirubin 1.2 mg/100 ml, alkaline phosphatase 21 K.A. units, albumin 4.5 g and globulin 2.6 g/100 ml, and alanine transaminase 62 units and aspartate transaminase 28 units/ml. Liver scan showed reduced uptake of the isotope in the region of the porta hepatis and uneven uptake in the left lobe. Coagulation studies and prothrombin time were all within normal limits. Barium swallow and meal examination showed no evidence of oesophageal varices and no ulceration at the stoma or in the gastric remnant. Gastroscopy on two occasions showed no source of the bleeding. Since the previous episode of bacterial endocarditis suggested the possibility of a visceral mycotic aneurysm as the cause of her symptoms, a coeliac axis angiogram was performed. Injection of the contrast medium reproduced her pain exactly and the films showed an aneurysm of the right hepatic artery (see Illustration).

Twelve hours after the angiogram she was awakened by a further attack of pain and haematemesis. She was shocked and required vigorous transfusion, but did not appear jaundiced. Laparotomy was performed next day, and at operation the aneurysm was found just protruding from the porta hepatis. It was supplied by the right hepatic artery. In order to obtain an adequate view of the aneurysm it was necessary to remove the gall bladder. Altered blood was found in the gall bladder and biliary tree. The right hepatic artery was ligated and divided

Selective coeliac arteriogram showing aneurysm (arrowed) arising from right hepatic artery.

distally. Immediately the aneurysm collapsed and pulsations stopped. There was no back bleeding from the distal end of the artery. She made an uneventful recovery from the operation. A postoperative liver scan showed no change from the preoperative picture except for a possible increase of uptake of the tracer in the region of the porta hepatis. The highest serum enzyme values in the week after the operation were alanine transaminase 140 units and aspartate transaminase 74 units/ml.

She remained asymptomatic until she was readmitted with a subarachnoid haemorrhage in December 1971. Bilateral carotid and vertebral angiograms showed no abnormality. A week after her admission she had sudden loss of consciousness with constricted pupils and died an hour later. Necropsy showed an intrahepatic, intraventricular, and basal subarachnoid haemorrhage with old intracerebral infarcts, but no aneurysm was found in a detailed examination of the circle of Willis and the other intracranial arteries. The heart was enlarged with old rheumatic scarring of the mitral and tricuspid valves. Injection of the common hepatic artery with Micropaque showed no evidence of aneurysm in a radiograph of the excised liver.

Comment

About 190 cases of hepatic artery aneurysm have been reported (Rutten and Sikkenk, 1971), and about 75% have been extrahepatic (Guida and Moore, 1966). Most of the aneurysms eventually rupture into the peritoneal cavity, the biliary system, duodenum, portal vein, or stomach. Without surgical treatment the prognosis is poor.

The combination of upper abdominal pain, gastrointestinal haemorrhage, and jaundice is very suggestive of the diagnosis. Jaundice is the least common of these features and results from pressure of an enlarging aneurysm on the common bile duct or from its rupture into the biliary tree, causing obstruction by blood clot. Though haemobilia was found at

Royal Infirmary, Manchester

J. HYWEL JONES, B.Sc., M.R.C.P., Senior Medical Registrar
C. B. JONES, F.R.C.S., Surgical Registrar
D. L. ASBURY, F.F.R., Senior Registrar in Radiology

operation in the present case jaundice was never severe enough to be clinically detectable. The pain was presumably due to transient distension of the biliary system by blood.

The diagnosis of mycotic aneurysm was suggested by the history of bacterial endocarditis, though an interval of 15 months elapsed between adequate treatment of the infection and onset of the abdominal symptoms. In the three previously reported cases of preoperative diagnosis of intrahepatic aneurysm the lesion was attributed to biliary infection (Cohen *et al.*, 1966), idiopathic medial necrosis (Weaver *et al.*, 1968), and bacterial endocarditis (Stack *et al.*, 1968). In two of these cases blood was found in the biliary tree at a previous operation (Cohen *et al.*, 1966; Stack *et al.*, 1968).

It is well recognized that hepatic artery aneurysms may complicate subacute bacterial endocarditis. Of 217 patients with mycotic aneurysms due to blood-borne infection 187 showed evidence of endocardial disease, and the hepatic artery and its branches were involved 19 times (Stengal and Wolferth, 1923). Local or systemic infection has been regarded as the commonest cause of hepatic artery aneurysm (Guida and Moore, 1966), but some authors believe that since the advent of antibiotics arteriosclerosis has become the most important factors (Rutter and Sikkenk, 1971).

Because of the risk of recurrent haemorrhage after ligation of a hepatic artery bearing an aneurysm, Cohen and his colleagues (1966) treated a patient with an intrahepatic aneurysm by lobectomy. In a case reported by Weaver *et al.*

(1968) the patient was similarly treated and it has recently been stated that lobectomy offers the only chance of cure of intrahepatic aneurysm (Rutter and Sikkenk, 1971). In the present case ligation of the right hepatic artery proved a successful treatment, and this experience has been shared by other workers (Stack *et al.*, 1968).

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Requests for reprints should be sent to Dr. J. Hywel Jones, Singleton Hospital, Sketty, Swansea SA2 8QA.

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Dysphagia, Abdominal Pain, and Sarcoid Granulomata

R. J. DAVIES

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Although granulomata have been found in the stomach and intestine of patients with generalized sarcoidosis (Palmer, 1958), involvement of the gastrointestinal tract rarely gives rise to symptoms. Scadding (1967) described two cases in which sarcoid infiltration of the abdominal lymph nodes appeared to be the cause of pain, but in a further review of the literature only one case could be found in which sarcoidosis involving the oesophagus had been recognized during life (Polachek and Matre, 1964). In the following case sarcoid granulomata were found in both the oesophagus and the abdominal lymph nodes, and symptoms were severe.

Case Report

In 1955 a 46-year-old symptomless Caucasian man was found on radiography to have a shadow in the right upper lung field. Sputum and laryngeal swabs were negative for acid-fast bacilli. He began to complain of huskiness of the voice, and in January 1957 a further chest radiograph showed definite enlargement of both hilar regions and a paratracheal shadow. The possibility of sarcoidosis was considered, though a Mantoux test at 10 tuberculin units was positive. Bronchoscopy was clear and a scalene node biopsy showed no evidence of disease. On direct laryngoscopy the vocal cords were thickened, though no histological abnormality was found on biopsy. The hilar and paratracheal shadows resolved spontaneously.

In 1958 he developed progressive difficulty in swallowing, and barium studies three years later showed severe loss of peristalsis. Oesophagoscopy in 1963 showed a swelling in the upper third of the oesophagus, and exploration of this showed a tumour to be present in the muscle wall. Patchy degeneration of muscle with non-cascating giant cell granulomata were found on histological examination (see illustration). No acid-fast bacilli or fungi were found on special staining. A muscle biopsy specimen from the left deltoid, electromyography, and an edrophonium test were all normal.

In 1969 the patient was admitted to hospital as an emergency with pain in the right lower quadrant of the abdomen. A soft mass was felt, and two weeks later appendicectomy was performed. The appendix was histologically normal, as were biopsy specimens of muscle and vessels from the abdominal wall. The pain returned three months later.

Histological section of oesophageal biopsy specimen.

When reinvestigated in January 1971 his general health was maintained despite great difficulty in swallowing, severe abdominal pain, and a husky voice. His haemoglobin level was 14.2 g/100 ml,