Late Presentation of Portal Vein Thrombosis as a Complication of Appendicitis

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SUMMARY: A case of appendicitis which resulted in portal pyaemia, hepatic and splenic abscesses, presumed portal vein thrombosis and subsequent portal hypertension is described. It confirms the merit of ultrasonic imaging in monitoring abscess resolution but questions the ability of the technique to recognise acute suppurative portal endophlebitis.

Introduction

Portal pyaemia and thrombosis are recognised complications of appendicitis and other intra-abdominal infections. Abscess formation in hepatic and splenic parenchymata may result. It is commoner in children than adults (1). Diagnosis has been improved by the use of abdominal ultrasound (2). A case is reported here where diagnostic ultrasound was used to monitor the resolution of hepatic and splenic abscesses, but which failed to demonstrate portal vein thrombosis. Two years later the patient developed portal hypertension.

Case Report

G.I., a 19 year old soldier, had a two day history of nausea, vomiting and pyrexia. He developed abdominal pain with guarding and rebound tenderness in the left iliac fossa on the second day and was admitted to hospital. Plain X-ray films showed isolated and distended loops of small bowel and queried appendicitis. Within 24 hours he became jaundiced and was transferred to the infectious diseases unit of another hospital. A hepatitis screen was negative, and ultrasound performed two days after initial admission demonstrated an enlarged spleen but normal liver, gall bladder and common bile duct. Five days after admission he developed signs of intra-peritoneal sepsis and at laparotomy was found to have a perforated gangrenous appendix surrounded by a pelvic abscess which had walled off. Appendicectomy was performed and the abscess was drained. He was treated with broad spectrum antibiotics but remained pyrexial. One week post-operatively, ultrasound demonstrated an enlarged liver with generalised inhomogeneity and an area of reduced echogenicity in the right lobe which was thought to represent a developing abscess. The spleen was still enlarged and also showed a region of reduced echogenicity which it was thought might be an abscess. The progress of these two lesions was followed over a period of four weeks and the liver abscess was seen to grow slightly and then slowly resolve. Although the splenic lesion also resolved, albeit more slowly, splenomegaly persisted. No reference was made at this time to the main portal vein. He was discharged from hospital five weeks after admission and instructed to contact his general practitioner.

He next presented 15 months later seeking to apply for helicopter pilot training. He already held a private pilot's

licence. At examination the smooth edge of an enlarged liver was palpable 4-5 finger-breadths below the costal margin. Mild hepatic dysfunction was confirmed by the finding of a gamma glutamyl transpeptidase (GGT) level of 64 u/l, and a bilirubin of 47 μ mol/l. Viral, autoimmune, iron and copper studies and immunoglobulins were all normal. Ultrasound of the liver revealed well defined, highly echogenic bands transversing the liver, cavernous transformation of the portal vein, a large hepatic artery, and splenomegaly. Subsequently portal venous obstruction surrounded by collaterals was demonstrated by computerised tomography. Histology of a liver biopsy was normal. Barium swallow showed thickening and irregular folds in the distal oesophagus and these were confirmed on oesophagoscopy to be grade I and grade II varices.

As a result of these findings he was medically discharged from the services and warned about the dangers of contact sports and the possibility of bleeding from oesophageal varices.

At this time the original ultrasound scans were reviewed. The first 2 examinations had demonstrated the splenic vein. Subsequent views showed a portal vein containing internal echoes which may have been thrombus. Flow studies had not been performed at the time, in the absence of suitable Doppler equipment.

Discussion

Suppurative endophlebitis of the portal vein is a recognized complication of intra-abdominal sepsis, including appendicitis. Portal vein thrombosis (PVT) accounts for 50% of adults who present with non-cirrhotic extra-hepatic portal venous obstruction (3). The commonest modes of presentation are bleeding from oesophageal varices or splenomegaly. Initial diagnosis may be difficult but it should be suspected whenever there is liver involvement in the presence of infectious disease in the abdomen and pelvis. Hepatomegaly is present in 50% of cases. Splenomegaly is always found and may be the presenting sign. Management of PVT is aimed at the underlying cause and then control of subsequent portal hypertension (3).

This patient probably suffered PVT at the time of his perforated gangrenous appendicitis. Although ultrasound was used extensively to monitor the progress of hepatic and splenic complications, and is the diagnostic

tool of choice (2), no comment was made about the integrity of the portal venous system. It was fortuitous that his hepatomegaly was noted 15 months later.

This case serves to highlight a potential inadequacy in the use of ultrasound to monitor portal pyaemia. If a clear image of the portal vein cannot be obtained, thrombosis should be excluded using Doppler ultrasound or enhanced computerised tomography.

REFERENCES

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