SPONTANEOUS INTRAPERITONEAL RUPTURE OF AN AMOEBIC LIVER ABSCESS

Report of a Case

KASTURI SARMA, M.B.B.S (Madras)

K. C. NAMBIAR, F.R.C.S.(Eng.), F.R.C.S.(Edin.)

Department of Surgery, Stanley Medical College, Madras

Introduction

Progression of an amoebic abscess of the liver is essentially histiolytic, with minimal inflammatory response and fibrosis. These factors should encourage the development of peritonitis. However, this complication is not as common as is generally believed. Craig⁴ has analysed the literature on the subject, and finds that out of 740 cases of amoebic liver abscess, rupture occurred in 197 cases (26.6%). Most of the abscesses opened into the pleura (70 cases), lung (54 cases), or pericardium (38 cases). The rest ruptured into the colon (6 cases), stomach (8 cases), bile ducts (3 cases), vena cava (6 cases), kidney (3 cases), duodenum (1 case) and lumbar region (8 cases). None of the abscesses ruptured into the peritoneal cavity.

Less than 100 cases of intraperitoneal rupture are on record. In most of the cases injury or an unsuccessful attempt at blind drainage have precipitated the disaster. Spontaneous rupture is very rare and in the majority of cases it would appear to have ended fatally.

The mortality of amoebic liver abscess varies in different series and appears to be proportional to the number of abscesses, even reaching 100% when the abscesses are more than three in number.⁹

Intestinal amoebiasis may also cause peritonitis. This may take the form of a localised patch of peritonitis due to a chronic ulcer eroding through the muscular coat of the intestinal wall. Occasionally a fulminating lesion may produce progressive bowel necrosis, or an accidental injury may perforate a segment of intestine already weakened by ulceration; in either case the resulting acute general peritonitis may be severe enough to prove fatal.^{4, 7, 11}

Case Report

M.P., a 60-year-old man, a destitute, was admitted for vague abdominal pain, constipation, and abdominal distension for the last ten days. The pain was not localized to any part of the abdomen, and it was unaccompanied by vomiting. He had no fever or cough.

Two months before he had had an attack of dysentery and passed about ten motions a day, containing mucus but no blood. The condition improved spontaneously without treatment, and he felt well until a week before he attended the outpatient department with vague dyspepsia and pain in the upper abdomen. His complaints were not considered serious enough to merit special investigation and he was treated symptomatically. After that he began to feel worse. He had not sustained any injury. The abdominal pain became more severe, constant and diffuse. It could not be localized, was not relieved by posture, and did not radiate. The abdominal distension had increased over the previous few days.

He was very anaemic (Hb., 40%; R.B.C. 2.2 million; W.B.C., 11,600, 74% polys.) and had slight oedema of the feet. His tongue was furred. He was ill. (Pulse rate, 104/min.; temperature, 100°F.) His blood pressure was normal.

The abdomen was uniformly distended. There was slight rigidity, which was not, however, localized to any particular quadrant. The liver could not be felt, and there was no liver tenderness. There was no upward enlargement of this organ that could be detected by percussion. No ascites could be made out clinically. Bowel sounds were inaudible. On rectal examination he had a prolapsed pile mass. There was no tenderness in the rectum and no faecal matter could be felt.

There was no oedema of the chest wall and the lungs were clinically normal; there was no evidence whatever of congestion of the right base.

A plain X-ray of the abdomen was taken with the patient in the sitting position. It was normal. He was given two enemata in succession; but no flatus or faeces were passed.

His condition deteriorated over the next six hours and the distension increased. Peritonitis was suspected, and it was decided to perform a laparotomy.

The abdomen was opened by a right para-median incision. The peritoneal cavity contained about two pints of thin, straw-coloured fluid. Flakes of fibrinous

exudate were adherent all over the serous coat of the small intestine. The caecum was hyperaemic and thickened. On pulling out a loop of small intestine from the right hypochondrium a small gangrenous area was found on the inferior surface of the right lobe of the liver, posterolateral to the gall bladder, through which thick pale-pink pus was pouring into the hepatorenal pouch. It was coming from an abscess which was estimated to be about 2 in. by 3 in. in size, in the lower part of the right lobe of the liver. The liver was not enlarged.

The pus was aspirated; Morrison's pouch was drained by a Malecot's catheter brought out through a separate stab incision made in the flank. The area was walled off with greater omentum, and the abdomen was closed in layers.

About two pints of thick pink pus drained into the bedside bottle over the first four post-operative days. Trophozoites of *E. Histolytica* were identified in the pus. The sinus healed in about four weeks' time.

The patient was treated with Chloroquine and received a course of Emetine injections. He made a satisfactory recovery and the bowels moved on the third day. He was discharged from the hospital at the end of six weeks.

Discussion

In all the recorded cases of spontaneous rupture of an amoebic liver abscess into the peritoneal cavity the difficulty of making a correct diagnosis is emphasised. Both Labry⁵ and Benedetti Valentini¹ made a provisional diagnosis of acute appendicitis. The cases reported by Biggam and Ragab² and Chang and Robertson³ were already under treatment for amoebiasis and the condition was therefore suspected. Self's case¹⁰ gave a long history of proved amoebic dysentery, for which he had undergone treatment in the Armed Forces. Yet the diagnosis was by no means easy.

In the present case the clinical manifestations suggested acute intestinal obstruction. This was perhaps due to paralytic ileus of the loops lying in the right hypochondrium. An unusual feature was the absence of vomiting. There was no liver enlargement, and liver tenderness, oedema of the chest wall, and rigidity of the upper abdomen were absent. The patient had no general symp-

toms like fever or rigors. A precise diagnosis was impossible. Peritonitis was suspected but it was not possible to determine, before the abdomen was opened, that it was due to intraperitoneal rupture of an amoebic abscess of the liver.

Another unusual feature of the case was the absence of enlargement of the liver. This may have been due to the rupture of the abscess causing a sudden release of tension, or to the absence of any accompanying hepatitis. Rogers⁴ insists on the usual healthy state of the liver unless there is bacterial infection with secondary abscesses, cloudy swelling, or other lesions like amyloid change due to a chronic abscess. Rolleston⁸ believes that the liver is usually enlarged and after evacuation of the pus, weighs more than normal.

Summary

A case of spontaneous intraperitoneal rupture of an amoebic abscess of the liver is presented. The case showed certain interesting features which are briefly discussed.

Acknowledgements

We are grateful to the Dean, Government Stanley Medical College, Madras, for permitting us to report this case.

REFERENCES

1. BENEDETTI VALENTINI, F. (1929), *Policlinico*, **36**, 280.
2. BIGGAM, A. G., and RAGAB, A. (1931), *J. trop. Med. Hyg.*, **35**, 285.
3. CHANG, C., and ROBERTSON, D. S. (1934), *Chin. med.*, **48**, 375.
4. CRAIG, C. F. (1949), 'Oxford Medicine', Vol. V, p. 81, Oxford.
5. LABRY (1925), *Lyon med.*, **35**, 324.
6. ROGERS, L. (1921), 'Bowel Diseases in the Tropics', p. 3, London: Oxford University Press.
7. ROGERS, L., and MEGAW, J. W. D. (1944), 'Tropical Diseases', p. 230, London: J. & A. Churchill.
8. ROLLESTON, H. (1949), 'Oxford Medicine', Vol. II, p. 356, Oxford.
9. SAMBUC, E. (1913), *Ann. d'hyg. et de med. colon.*, **16**, 48.
10. SELF, J. B. (1955), *Postgrad. med. J.*, **31**, 35.
11. SIMON, S. K. (1925), 'Tice's Practice of Medicine', Vol. p. 270, London: Butterworth.