SUPPURATIVE PYLEPHLEBITIS OF THE PORTAL VEIN FOLLOWING A MUCOCELE OF THE APPENDIX

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Suppurative pylephlebitis of the portal vein is a comparatively rare condition. The commonest preceding lesion is acute appendicitis. In 44 cases reported by Eliason,[1] acute appendicitis was found in 40, and in 3 of 4 cases described by Koster and Kasman.[2] It is, however, a rare complication of appendicitis, occurring in only 0·5% of cases.[1] Cases have also been reported in association with diseases of the stomach, rectum, pancreas, spleen, gall-bladder and mesenteric lymph nodes.

Rolleston and McNee[3] regarded the following features as being those of major clinical significance: (1) Evidence of primary cause, (2) picture of sepsis (especially rigors and sweats) and (3) evidence of involvement of the liver.

Although a fair number of cases have been reported in Europeans[4,5,6,7] we have not been able to find any reference to a case in an African. At this hospital over a period of 7 years there have been over 180,000 admissions, included in which were 740 cases of appendicitis. No case of suppurative pylephlebitis has however occurred. We therefore wish to record this case in an African patient.

CASE REPORT

An adult African male (Sotho) aged about 68 years, a factory hand, was admitted to this hospital on 11 July 1955, complaining of cough, hiccough and pain in the right chest for 2 weeks.

He had been born in the vicinity of Johannesburg and had never left the environs of the city. Several months before admission he had an attack of pain in the right iliac fossa, but this disappeared after about one week. He also reported an episode of swelling of the left foot in 1951 and some 'chest trouble' in 1953.

Positive findings on admission included elevated temperature (102°F); an injected pharynx; a blood pressure of 100/50 mm. Hg; an enlarged, tender liver, palpable 3½ inches below the right costal margin, with a smooth, firm edge; a firm non-tender enlarged spleen, palpable 1 inch below the left costal margin. There was no tenderness in the right iliac fossa, nor was any mass felt.

There was no clinical jaundice on admission. Laboratory findings showed serum bilirubin 2·7 mg. per 100 c.c. with a positive direct v. d. Bergh reaction; blood proteins 6 g. per 100 c.c. (albumin 2·2, globulin 3·9); alkaline phosphatase 12·6 King-Armstrong units; blood urea 52 mg. per 100 c.c.; haemoglobin 11·6 g. per 100 c.c., with a leucocytosis of 16,500 per c. mm., of which 89% were neutrophils and 11% lymphocytes; malaria smears were negative. The urine showed 1+ bilirubin and 2+ urobilin. Urine and stool examinations for parasites were negative. X-ray studies showed increased striation at both hilar regions and at the right base, with peaking of the right diaphragm, probably due to old pleural thickening. In addition the liver appeared enlarged. Despite the absence of *E. histolytica* in the stools it was felt that the possibility of amoebiasis could not be excluded and the patient was therefore given 1 gr. of emetine daily for 10 days, and penicillin 500,000 units 6-hourly. At the end of 2 weeks there was no improvement in his condition.

Blood chemistry studies on 27 July revealed a slight fall in the serum bilirubin to 0·6 g.% but no other significant change. Serologic tests for typhoid, melitensis and rickettsiae were negative. Rigors became pronounced (temperature ranging from 101° to 103°F), and his condition deteriorated, with loss of appetite and listlessness. Blood cultures were repeatedly sterile. Intravenous therapy (dextrose and saline) was commenced, with the addition of hydro-cortisone as a supportive measure. He was also given 1 g. of intra-muscular streptomycin daily and 200 mg. of isoniazid orally *tds.*

On 8 August he became clinically jaundiced and markedly disorientated (serum bilirubin 3·4 mg.%). Examination of the cerebrospinal fluid was essentially negative and a repeat haemogram still showed a leucocytosis. In a search for pus the liver was needled, but with negative results and a pneumo-peritoneum showed the liver to be completely free of the diaphragm.

The patient continued to deteriorate and died in a semi-comatose condition 5 weeks after admission.

Post-mortem Findings

Peritoneal cavity contained 2 pints of dark-brown fluid.

Bowel. There was marked dilatation of the appendix, which measured 3 inches in length and ½ inch in diameter and on section contained a large quantity of greenish-white mucoid material which compressed and thinned the wall. The features were those of a mucocele. The remainder of the bowel was normal.

Liver. This organ was slightly enlarged (weight 1,700 g.) and had a smooth surface. On section numerous acute abscesses were seen throughout the parenchyma, varying in size from 1 to 5 mm. in diameter and containing greenish-white muco-pus. These abscesses were surrounded by a narrow zone of congested and compressed liver tissue; the surrounding liver-parenchyma appeared normal. The portal vein contained a large septic thrombus loosely adherent to the wall of the vein about 1½ inches from the porta hepatis, and also large quantities of light-green pus. Septic thrombi were present in the superior mesenteric vein, but not in the splenic vein.

Lungs. Both showed severe congestion and oedema.

Microscopic Examination

Appendix. The distended appendix was filled with homogeneous mucinous material. At one point the muscularis was ruptured.

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There was compression and thinning of the wall, with areas of complete loss of epithelium and atrophy of the lymphoid follicles. The subepithelial tissues showed fibrosis, with foci of fatty metaplasia, and were diffusely infiltrated by chronic inflammatory cells in which chains of Gram-positive cocci were observed. The features were those of a chronic obliterative appendicitis with the formation of a mucocele (Fig. 1).

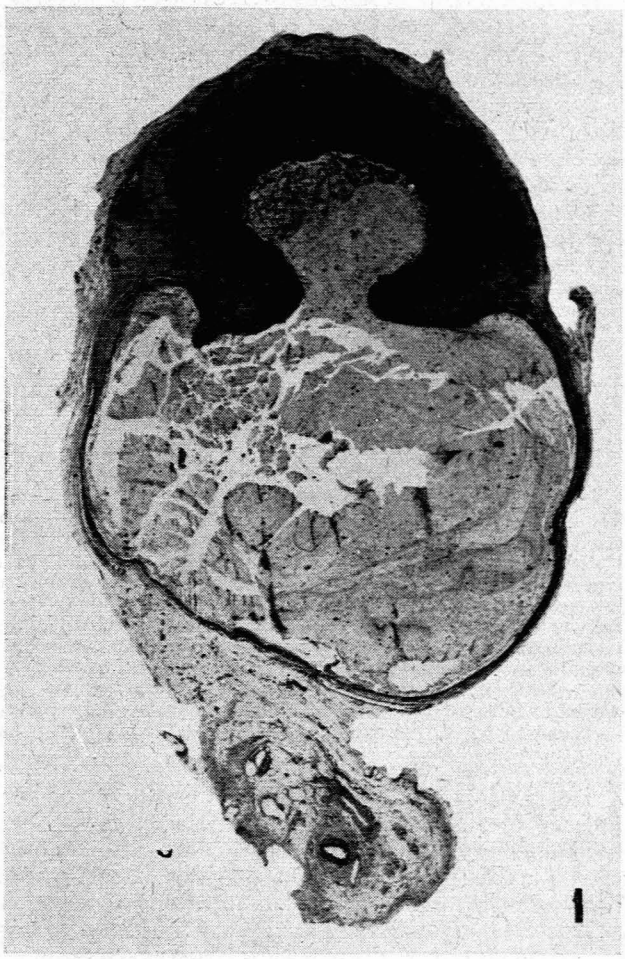

Fig. 1. Section of the appendix, showing the lumen lined by mucus-secreting cells and containing homogeneous mucinous material. The muscularis is ruptured with the formation of a large mucocele lined by a thin layer of fibrous tissue. Haematoxylin and eosin × 7.

Liver. Section of the liver showed the presence of several large acute abscesses lined by degenerating and compressed liver cells and containing necrotic debris densely infiltrated by polymorphs. In some instances it was clear that the abscesses were related to the branches of the portal vein. Chains of Gram-positive cocci were observed in the abscesses. The surrounding liver parenchyma showed marked peri portal haemosiderosis with slight associated fibrosis and chronic inflammatory-cell infiltration (Fig. 2).

Lungs. Section showed the presence of bronchopneumonia, congestion and oedema.

Summary of Post-mortem Findings

1. Chronic obliterative appendicitis with the formation of a mucocele.
2. Suppurative pylephlebitis of the portal vein.
3. Multiple abscesses of the liver.

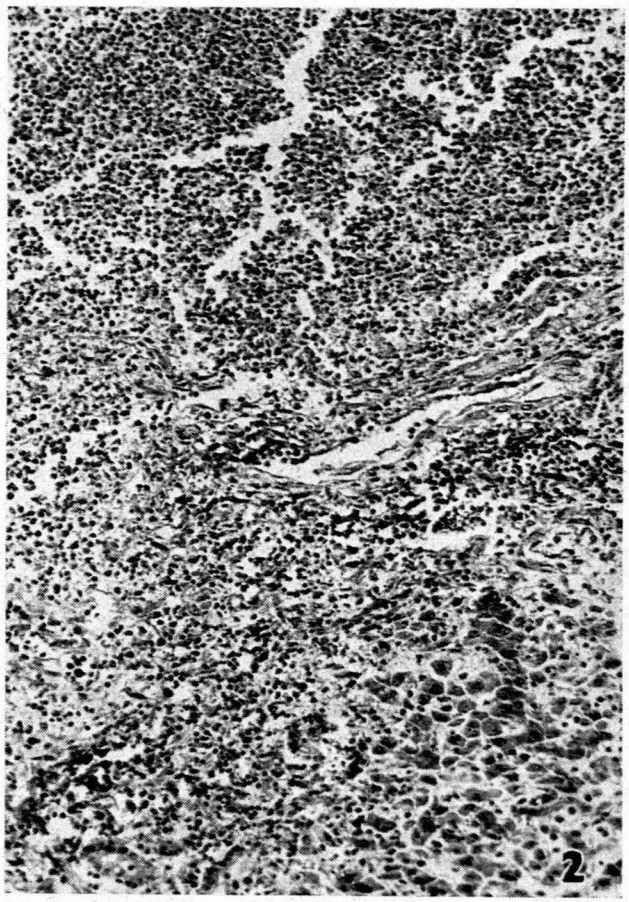

Fig. 2. Section of the liver showing the presence of a large acute abscess lined by degenerating and compressed liver-cells and containing necrotic debris densely infiltrated by polymorphs. Haematoxylin and eosin × 120.

SUMMARY

A case of suppurative pylephlebitis of the portal vein following a mucocele of the appendix is described in an African patient admitted to the Baragwanath Non-European Hospital.

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