This condition has characteristic findings on excretory urography and ascending urography as demonstrated in our case. Graham *et al.* (1965) have used lymphangiography in the diagnosis of this condition but Halverstadt (1968) states that it may not always be conclusive in this disease and an abnormal lymphangiogram is not always an absolute diagnostic criterion for idiopathic retroperitoneal fibrosis.

The definitive treatment favoured is ureterolysis in the early stages but for a patient coming with uraemia or anuria this is combined with nephrostomy or ureterostomy. The ureters can be approached retroperitoneally on either side but this has the disadvantage of not preventing re-investment of the ureter by the fibrotic process. To overcome this certain special procedures such as closing the channel or filling it with fat, or transplanting the ureter away from its former course, or wrapping the ureter with fat, or embedding it in a muscular bed are advisable.

The ureters can be approached transperitoneally and this was advocated by Ormond (1948) and Raper (1955) as the better approach, as both the ureters can be inspected and treated and moreover the chances of re-investment with fibrosis are rare as the posterior peritoneum is stitched beneath the freed ureters. This procedure has however, the drawback that ureteroplasty or nephrostomy, if desirable, is made more difficult.

Shaheen & Johnston (1959) have recorded a case in which the recurrence of ureteral obstruction with symptoms after bilateral ureterolysis was successfully treated with cortisone. Radiotherapy was suggested by Oppenheimer, Narins & Simon (1952) and Bradfield (1953) for the treatment of this condition but its success has not been proved. Harrow & Solan (1962) think that the radiosensitivity of the fibrous mass may be due to its marked vascularity. With all these empirical therapies there is no record of the plaque remaining unchanged or receding although the patient might show clinical improvement.

References

ALBARAN (1905) Quoted by Hache *et al.* (1962).

- BRADFIELD, E.O. (1953) Bilateral ureteral obstruction due to compression by chronic inflammation. *Journal of Urology*, **69**, 769.
- CHARNOCK, D.A., RIDDELL, H.I. & LOMBARDO, L.J. Jr. (1961) Retroperitoneal fibrosis producing ureteral obstruction. *Journal of Urology*, **85**, 251.
- GRAHAM, J.R., SUBY, H.I., KERR, W.S., Jr., & FRALEY, E. (1965) Retroperitoneal fibrosis—a missing link in the chain. *Journal of Urology*, **93**, 144.
- HACHE, L., UTZ, D.L. & WOOLNER, L.B. (1962) Idiopathic fibrosis retroperitonitis. *Surgery, Gynaecology and Obstetrics*, **115**, 737.
- HALVERSTADT, D.B. (1968) Problem in the use of urographic and lymphangiography in the diagnosis of the idiopathic retroperitoneal fibrosis. *Journal of Urology*, **99**, 423.
- HARROW, B.B. & SOLAN, J.A. (1962) Idiopathic retroperitoneal fibrosis. *Journal of the American Medical Association*, **182**, 38.
- HOFFMAN, W.H. & TRIPPLE, O.T. (1961) Retroperitoneal fibrosis—etiologically consideration. *Journal of Urology*, **86**, 222.
- KENDALL, A.R. & LAHEY, W.H. (1961) Sclerosing Hodgkin disease vs. idiopathic retroperitoneal fibrosis. *Journal of Neurology*, **86**, 217.
- MULVANEY, W.P. (1958) Periureteritis obliterans—a retroperitoneal disease. *Journal of Urology*, **79**, 410.
- ORMOND, J.K. (1948) Bilateral ureteral obstruction due to envelopment and compression by inflammatory retroperitoneal process. *Journal of Urology*, **59**, 1072.
- OPPENHEIMER, G.D. & GOLDMAN, H. (1962) Periureteral fibrosis, an unusual complication of renal biopsy. *Journal of Urology*, **88**, 611.
- OPPENHEIMER, S.D., NARINS, L. & SIMON, M. (1952) Radiotherapy in treatment of non-specific inflammatory stricture of ureter. *Journal of Urology*, **67**, 476.
- PARTINGTON, P.F. (1961) Diffuse idiopathic fibrosis. *American Journal of Surgery*, **101**, 239.
- RAPER, F.P. (1955) Idiopathic retroperitoneal fibrosis involving the ureter. *Journal of Urology*, **82**, 436.
- SAMELLA, W. (1961) Ureteral obstruction due to compression by an idiopathic retroperitoneal inflammatory process. *Journal of Urology*, **85**, 828.
- SHAHEEN, D.J. & JOHNSTON, A. (1959) Bilateral ureteral obstruction due to an inflammatory retroperitoneal process. *Journal of Urology*, **82**, 51.
- TREVOR, R.W. (1958) Reticulum cell sarcoma producing retroperitoneal periureteric fibrosis. *New England Journal of Medicine*, **258**, 268.
- UTZ, D.C., ROOK, E.D., SPITTEK, J.A. & BARTHOLEMMEW, L.G. (1965) Methysergide and retroperitoneal fibrosis. *Journal of the American Medical Association*, **191**, 983.
- VEST, S.A. & BARELARE, B. (1953) Periurethritis plastica. *Journal of Urology*.

Amoebic pericarditis

B. C. BANSAL
M.D.

D. S. GUPTA
M.B., B.S.

Medical College Hospital, Rohtak, India

In 1860, Rouis from Algeria published the necropsy findings of a patient with 'tropical abscess' of the liver which had extended into the pericardium. Germillon (1899) is credited with the first clinical

diagnosis of amoebic pericarditis. Lamont & Pooler (1958) reported that the frequency of amoebic pericarditis is about 2.8%. Takara & Bond (1958) reported the frequency of amoebic pericarditis to be

about 7.5% in a series of 293 cases of pleuropulmonary, pericardial and cerebral amoebiasis collected from the world literature. It has been reported that suppurative amoebic pericarditis usually results from rupture of an amoebic abscess of the left lobe of the liver, and is usually fatal. While reviewing the literature, Norris & Beemer (1956) collected forty-seven examples of suppurative amoebic pericarditis with only two survivors. Lamont & Pooler (1958) reported only two survivors out of seven cases of amoebic pericarditis. However, in a recent study by Macleod, Wilmot & Powell (1966) a somewhat higher survival rate has been reported.

Suppurative amoebic pericarditis developing as a complication of right-lobe amoebic liver abscess is very rare. Paulley (1961) reported that in his series of seventeen patients of amoebic pericarditis, only one developed from a right-lobe amoebic liver abscess. Takara & Bond observed that 'rarely does an abscess of the right lobe of liver give rise to amoebic pericarditis'. In a series of twenty cases of amoebic pericarditis, reported by MacLeod *et al.* (1966), none had developed the pericarditis as a complication of amoebic liver abscess of the right lobe only. There were, however, two cases who, in addition to having a left lobe liver abscess, had one in the right lobe also.

Lamont & Pooler (1958) have described three stages of development of amoebic pericarditis: an initial, slightly purulent effusion, then rupture of the hepatic abscess, often resulting in death of the patient by cardiac tamponade and lastly, a stage of constrictive pericarditis developing after several weeks.

Case report

D. B., 19 years, male, presented on 22 March 1966, in a forward Army Hospital, with irregular fever of 7 days' duration, pain over right shoulder and dry cough of 5 days' duration. He had had a short attack of fever, lasting for 5 days, a month prior to this illness.

On examination. Temperature 98.6°F, pulse 70/min regular, BP 110/80 mmHg, no clubbing, no jaundice and no toxemia. Abdomen was soft, liver just palpable, soft with regular margins and non-tender. Upper border of the liver was normal on percussion. No other abnormality in abdomen. Respiratory and cardiovascular systems also did not reveal any abnormality.

Investigations. Hb 14.0 g/100 ml, WCC 10,600/mm³, poly 74%, lympho 18%, mono 2% and eosino 6%. The patient was put on symptomatic treatment and was kept under observation. He improved initially, became afebrile and remained so till 4 April 1966 when he again started running an irregular

fever, the right shoulder pain recurred, and he started looking toxic. There were still no abnormal physical signs except for a just palpable, soft, non-tender liver. The blood count showed polymorphonuclear leucocytosis. Intramuscular penicillin and streptomycin were added to the treatment. On 8 April, 1966 the patient became breathless and developed a pleural rub in the right infra-mammary and infra-axillary region. The chest X-ray revealed an enlarged cardiac silhouette with loss of contours over its borders; the right cardiophrenic angle was clear. There was no abnormality in the lungs and also there was no rise of the right dome of diaphragm (Fig. 1). Screening confirmed the pericardial effusion. The right lobe of the liver was enlarged about 1 cm, and non-tender. A diagnostic pericardial tap revealed a straw-coloured exudate, which was sterile on culture. A presumptive diagnosis of tuberculous pericarditis with dry pleurisy was considered and the patient was put on anti-tuberculous treatment. No improvement occurred and patient's general condition deteriorated. On 18 April 1966, the patient suddenly worsened and developed cardiac tamponade. X-ray revealed a marked increase in cardiac silhouette. An immediate paracentesis produced 40 oz of typical anchovy-sauce pus. The diagnosis of amoebic pericarditis following rupture of hepatic abscess was obvious.

FIG. 1. X-ray taken on 18 April 1966, showing massive pericardial effusion following rupture.

FIG. 2. Follow up X-ray showing pneumopericardium and a cavity in right lung adherent to pericardium.

He was then given emetine by injection, with chloroquin and di-iodohydroxy quinoline. His condition improved, but he developed cardiac tamponade again intermittently, and had to be subjected to paracentesis on three more occasions. Further X-rays showed a very large quantity of air

in the pericardium, which could not have been accidentally introduced during tapping. In one, a very interesting feature was noted. There was a cavity with fluid level in lower segment of right lung, filling the right cardiophrenic angle (Fig. 2). This explained the air in the pericardium. The recurring cardiac tamponade was in part due to this excessive air under pressure, having entered the pericardium from the involved lung segment.

The patient made an uneventful recovery and was followed for a period of 16 months, when a chest X-ray revealed normal cardiac silhouette. There was no clinical, radiological or ECG evidence of constrictive pericarditis.

Acknowledgment

We wish to thank the Director General of the Armed Forces Medical Services, India, for permission to publish this case since it was treated by the authors while in the Army Medical Corps.

References

- GERMILLON, W. (1899) These, Paris. Quoted by Fiegel (1959).
- LAMONT, N.M.E. & POOLER, N.R. (1958) Hepatic amoebiasis—a study of 250 cases. *Quarterly Journal of Medicine*, **27**, 389.
- MACLEOD, I.N., WILMOT, A.J. & POWELL, S.J. (1966) Amoebic pericarditis. *Quarterly Journal of Medicine*, **35**, 293.
- NORRIS, D.L. & BEEMER, A.M. (1956) Amoebic pericarditis: report of a case with brief review of literature. *Journal of Tropical Medicine and Hygiene*, **59**, 188.
- PAULLEY, J.W. (1961) Amoebic liver abscess in Great Britain. *British Medical Journal*, **1**, 462.
- ROUS (1860) Mentioned by Vergoz and Hermenjat Gerin (1932).
- TAKARA, T. & BOND, W.M. (1958) Pleuropulmonary, pericardial and cerebral complication of amoebiasis. *Surgery, Gynecology and Obstetrics*, **107**, 209.

Temperate sprue

M. J. KENDALL

T. J. BAYLEY

Queen Elizabeth Hospital, Birmingham B15 2TH

FOLIC acid deficiency and megaloblastic anaemia may occur in malabsorptive states. Some patients with tropical sprue respond to folic acid therapy even in small doses (O'Brien & England, 1964), and although not yet adequately explained the anaemia, malabsorption and abnormal jejunal mucosa may all return to normal in some cases treated in this way. Thus, it seems that folic acid deficiency may result from, or may play some part in, causing malabsorption. Tropical sprue has a striking geographical and local distribution, occurring all over the Far East but not in Africa, in some Caribbean islands

but not in Jamaica, and it may occur in particular areas of a country and in epidemic form (Stefanini, 1948; Baker, Mathan & Joseph, 1962). Cooke and his colleagues (1963) have described a syndrome called 'temperate sprue', two of their patients having steatorrhoea which improved with folic acid therapy. A further patient with malabsorption which responded to parenteral folic acid has recently been described (Drummond & Montgomery, 1970) in whom steatorrhoea disappeared rapidly with treatment. The patient reported here shared many of the features seen in tropical sprue, particularly as seen