Clinical Section

President F B Gibberd FRCP

Meeting 7 June 1974
(continued from July 'Proceedings', p 422)

Cases

Amœbic Liver Abscess Presenting Thirty-two Years After Acute Amœbic Dysentery

Roy W Jones[1] BSC MB (for B I Hoffbrand DM MRCP)
(Whittington Hospital, London NI9)

G B, man aged 50

History: Admitted February 1974 with a one-week history of rigors, continuing fever (38–39°C), malaise, anorexia and generalized aches and pains. Treated at home with ampicillin.

He was born in Burma and lived in the Far East until 1963 when he settled in England. He smokes up to 60 cigarettes a day, drinks little alcohol and gave a past history of malaria (last attack 1962).

On examination: Pyrexial (39°C) with a few coarse crepitations at both bases. Liver just palpable but not tender.

Investigations: ESR 125 mm in 1 hour (Westergren). Hb 12.0 g/100 ml. No malarial parasites seen. WBC 20 300/mm³ (neutros. 90%); alkaline phosphatase 18.0 K-A units/100 ml (later 25 K-A units); albumin 3.1 g/100 ml (later 3.4). Chest X-ray showed old calcified lesions of right upper zone of lung. Blood cultures, mid-stream urine, extended Widal and antinuclear factor tests all negative.

Treated initially with co-trimoxazole and his temperature settled.

Further questioning revealed that he had had acute amœbic dysentery in 1942, and in view of his raised phosphatase, a liver scan was performed. This showed a single large space-occupying lesion in the middle of the right lobe (Fig 1A).

Serology (Dr A L Jeanes): Fluorescent amœbic antibody test positive (initially at a titre of 1:64, later 1:256). Hydatid CFT, α-fetoprotein, hepatitis B antigen, autoimmune profile and examination of stools all negative.

Present address: Brompton Hospital, London SW3 6HP

Treated with metronidazole 400 mg three times daily for five days and ESR fell to 15 mm in 1 hour with normal liver function tests and improvement of the liver scan (Fig 1B).

8 May 1974: Further rigor, mild abdominal pain and watery diarrhœa. Tender in epigastrium and over right lobe of liver laterally. Sigmoidoscopy revealed mild inflammation only. Fæces showed numerous pus cells and some red blood cells but

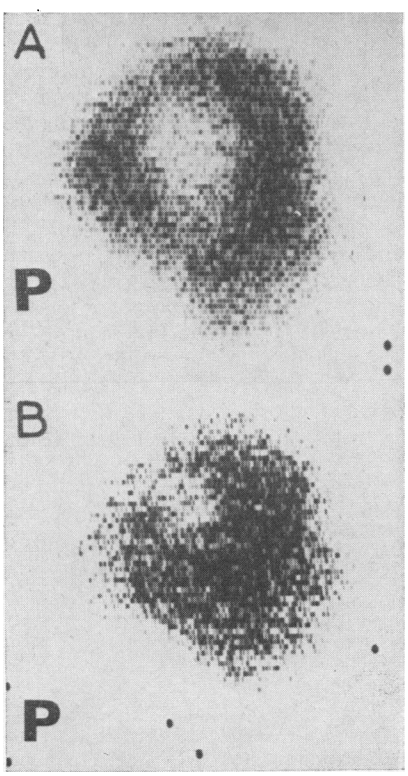

Fig 1 *Liver scans (technetium) right lateral view.* A *(28.2.74), before treatment.* B *(23.4.74), after treatment. P=posterior*

no amœbæ or cysts. ESR 34 mm, WBC 12 500/mm³, phosphatase normal. Barium enema normal.

This illness was considered to be due to incomplete eradication of the amœbæ possibly with superimposed intestinal amœbiasis. He was therefore restarted on metronidazole 800 mg three times daily for 10 days, with clinical improvement.

Discussion

The long latent period which may exist between the intestinal infection and hepatic involvement (up to fifty years in a case described by Paulley 1961) has not been explained. Few cases of hepatic amœbiasis have concurrent amœbic dysentery and only some 25% will give a past history of it (Sepúlveda *et al.* 1959). Examination of the fæces yields cysts and trophozoites in 17–50% of cases but much depends on the skill and patience of the examiner.

Radioactive liver scanning, when available, has largely replaced liver aspiration for confirmation of the diagnosis, and in following treatment.

Serology, including immunofluorescence, hæmagglutination and complement fixation, should be used whenever the diagnosis is suspected. Positive results are obtained in 60–80% of cases of symptomatic intestinal amœbiasis and 95–98% of cases of hepatic amœbiasis (Stamm 1970); serology is thus useful in excluding the diagnosis of amœbic liver abscess. However, antibodies persist for a long time after cure and a positive result may be due to past infection.

Aspiration of an amœbic abscess is unjustifiable for purely diagnostic reasons. It should be reserved for cases with a palpable mass, persistent localized tenderness, a markedly raised right hemidiaphragm or failure to respond to drug therapy (Powell 1971). Delay in diagnosis increases the morbidity and mortality (Wright 1966) and hepatic amœbiasis should be considered as a differential diagnosis in anyone who has ever been in the tropics even without a past history of dysentery.

REFERENCES

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Powell S J (1971) In: Management and Treatment of Tropical Diseases. Ed. N G Maegraith and H M J Gilles. Blackwell, Oxford; p 3

Stamm W P (1970) *Lancet* ii, 1355

Sepúlveda B, Jurich H, Bassols F & Muñoz R (1959) *American Journal of Digestive Diseases* 4, 43

Wright R (1966) *British Medical Journal* i, 957

Dr V W Johnson (*New Cross Hospital, Wolverhampton*) asked if calcification of the liver was a common finding in amœbic liver disease.

Dr Jones replied that it was an extremely uncommon finding.

Relapsing Meningoencephalitis ? Cerebral Sarcoidosis

L J Findley MB MRCP (for F Clifford Rose MB FRCP)
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D B, man aged 36, right-handed. Airduct erector

History: Presented in March 1973 complaining of left-sided headaches for three weeks, photophobia and subjective right-sided weakness. On examination there were no objective physical signs. Symptoms followed mild head trauma to the left parietal region. He was investigated for possible subdural hæmatoma and all investigations, including skull and chest X-rays, skull echo-encephalogram, EEG and left carotid arteriogram, were normal. ESR 15 mm in 1 hour (Westergren). CSF not examined. Discharged well after two weeks.

Relapse 1 (April 1973). Following rapid deterioration he complained of drowsiness, headache and paræsthesia over left side of face. On examination: meningism associated with right-sided hyperreflexia. ESR 70 mm in 1 hour; hæmoglobin, differential white count, serum proteins and immunoglobulins normal. CSF at normal pressure: total protein 60, glucose 60 mg/100 ml; lymphocytes 70/mm³; bacterial cultures sterile. Lange colloidal gold curve and CSF Wassermann reaction negative. Continued to deteriorate after admission and was treated as tuberculous meningitis with streptomycin 1 g daily, isoniazid 150 mg twice daily and para-aminosalicylic acid 6 g twice daily. Steroids were given as dexamethasone 2 mg three times daily. After starting treatment he improved steadily and was symptomatically and clinically quite well within four weeks.

June 1973: CSF normal; steroids discontinued.

July 1973: Cultures for mycobacteria negative; streptomycin discontinued.

Relapse 2 (August 1973). He complained of severe cervical pain, paræsthesiæ in hands and feet and incontinence of urine. On examination there was weakness and wasting of intrinsic muscles of both hands.

Investigations confirmed CSF changes similar to those of first illness (total protein 70, glucose 60 mg/100 ml; lymphocytes 70/mm³, light microscopy and bacterial cultures negative). ESR 13 mm in 1 hour; the following investigations were also normal: hæmoglobin, full count and differential leukocyte count, serum total and differential proteins, serum immunoglobulins, serology for syphilis, toxoplasma dye test and complement-fixation test, brucella agglutination reaction, CSF culture for cryptococcus, viral cultures and antibody studies, leptospira complement-fixation test, urinary and serum calcium