Acquired toxoplasmosis presenting as hepatitis

A. VETHANYAGAM
Senior House Officer

A. D. M. BRYCESON
Consultant Physician

Hospital for Tropical Diseases, 4 St. Pancras Way, London NW1 0PE

Toxoplasmosis affects over a quarter of the world’s population (CHRISTIE, 1974). The causative organism, Toxoplasma gondii, is a coccidian parasite which is found in its asexual phase in most mammals and birds. In the cat family, both domestic and wild, it has been shown to exist in the sexual phase, too (FELDMAN, 1968; GRAY et al., 1972: HUTCHISON et al., 1970). The disease in man may be asymptomatic or symptomatic. 90% of clinical cases manifest as generalized lymphadenopathy usually with low grade fever and malaise (HUTCHISON et al., 1969). Jaundice with or without hepatosplenicomegaly is rare. The case reported here illustrates the importance of considering toxoplasmosis in the differential diagnosis in patients presenting with fever and jaundice.

Case report

A 25-year-old British male was admitted on 9th June, 1975. In April he had spent a few weeks in towns in Peru and on 19th May had ventured into the jungle where he drank unboiled stream water. The next day he developed diarrhoea which was controlled by “mexaform”. A week later he noticed loss of appetite, darkening of urine and jaundiced sclerae. A fine maculo-papular rash, which did not itch, appeared over the trunk and sparsely over the limbs; it lasted a week. He rested in bed till 6th June and then flew back to U.K. because of persisting jaundice, malaise and headache. There had been a brief association with a Siamese cat a month before his visit to Peru.

On examination the patient was thin at 59.5 kg and slightly icteric. There was a solitary small soft lymph node palpable in the left axilla. The liver and spleen were palpable at the costal margins. There was no rash or lesion in the mucousae, conjunctivae or optic fundi. Investigation showed: haemoglobin 13.1 g/100 ml, leucocytes 9,100/mm³, serum bilirubin 1.3 mg/100 ml (conjugated bilirubin present), glutamic oxaloacetic transaminase (SGOT) 19 I.U./l, alkaline phosphatase 15 KA.U./100 ml, albumin 3.2 g/l00 ml, globulin 4.2 g/100 ml—with increased alpha-2 and gamma fractions. Hepatitis B antigen was not detected. One cervical node was biopsied: histology showed nonspecific, moderate follicular and sinusoidal hyperplasia.

Further blood cultures were negative. Serological studies for toxoplasmosis are shown in the table. On 23rd June, for the first time, generalized enlargement of cervical, axillary and inguinal lymph nodes was detected. One cervical node was biopsied: histology showed nonspecific, moderate follicular and sinusoidal hyperplasia.

Four weeks after admission, still febrile and lighter by 3.4 kg, treatment was started with sulphadimidine 0.5 g six hourly and pyrimethamine 25 mg daily, with rapid resolution of fever and splenomegaly and gradual resolution of lymphadenopathy.

When seen on 12th August he was well, weighing 61 kg, apyrexic and with no abnormal physical or biochemical findings. In March 1976 he remained well and weighed 68 kg.

Discussion

The diagnosis of active acquired toxoplasmosis was made on serological evidence and confirmed by the rapid response to treatment. A positive Sabin & Feldman dye test alone can signify a previous infection although a titre of 1:16,000 is highly suggestive of an active infection. The rise in titre of the complement fixing antibody, which is known to appear late in infection, is diagnostic of an active acquired infection (CHRISTIE, 1974; HUTCHISON et al., 1969; JEWELL et al., 1972). The histology of the liver was helpful in that it excluded viral hepatitis. Though toxoplasmosis may cause characteristic histological changes best seen in the posterior cervical lymph nodes, the liver morphology is seldom diagnostic (JONES, 1975).

The clinical features of interest in this case were the rash, the jaundice, the prolonged fever, the late appearance of the lymphadenopathy and the extreme malaise. The rash is commoner in congenital toxoplasmosis but may occasionally be seen in acquired cases, where lymphadenopathy is not the predominant feature (JONES et al., 1965). Lymphadenopathy is usually an early feature and seldom arises so late in the disease (HUTCHISON et al., 1969)—four weeks in this instance. Jaundice is seen in a small proportion of cases and is often late in appearing. In their two cases of hepatitis due to toxoplasmosis, VISCHER, BERNHEIM & ENGELBRECHT (1967) recorded prodromal illnesses of two and three months, and considered this characteristic (REMINGTON, 1974).

In this case the features which first aroused suspicion that the diagnosis of infective hepatitis was incorrect, were the prolonged low grade fever and headache which are found in 40% of cases of toxoplasmosis (VISCHER et al., 1967). This suspicion was supported by the failure...
of serum glutamic oxaloacetic transaminase to reach the characteristic high levels expected in infective hepatitis. In clinical practice, because it can be difficult to differentiate between toxoplasmosis and infectious hepatitis, cases presenting with fever and jaundice can be misdiagnosed. Fortunately, both conditions are self limiting; so, in the absence of an immune deficiency which may permit fatal dissemination of toxoplasmosis, no grave harm is done, although treatment shortens the duration of fever and malaise in toxoplasmosis, which may otherwise be as profound as in hepatitis and even more prolonged. The effect of treatment in hastening resolution of lymphadenopathy is less certain (Vischer et al., 1967).

The source of infection in this case is uncertain. Oocysts remain viable in moist soil for a year (Christie, 1974) and water from the jungle stream would be as likely a source as the pet cat.

Table – Toxoplasma antibody titres

<table>
<thead>
<tr>
<th>Date</th>
<th>Sabin Feldman dye test</th>
<th>Complement fixation test</th>
</tr>
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<tbody>
<tr>
<td>17.6.75</td>
<td>Not done</td>
<td>Negative</td>
</tr>
<tr>
<td>27.6.75</td>
<td>1:16,000</td>
<td>Not done</td>
</tr>
<tr>
<td>7.7.75</td>
<td>1:20,000</td>
<td>1:80</td>
</tr>
<tr>
<td>12.8.75</td>
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<td>1:80</td>
</tr>
<tr>
<td>16.3.76</td>
<td>1:2,000</td>
<td>1:80</td>
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References


