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Toxoplasma encephalitis in a raw steak eater

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Summary

A case of adult acquired toxoplasma encephalitis is described. The patient was in the habit of eating raw or very lightly cooked steak, and could have obtained her infection from this source.

Introduction

The vast majority of acquired infections with the protozoan parasite, *Toxoplasma gondii*, are symptomless and are recognized by finding serum antibodies (Beverley, 1969). The commonest clinical manifestation of acquired toxoplasmosis is a glandular-fever-like syndrome with a negative Paul Bunnell reaction (Siim, 1956). Other modes of presentation described include uveitis (Duke-Elder, Ashton & Brihaye-van Geertruyden, 1953), atypical pneumonia (Ludlam & Beattie, 1963), myositis (Chandar, Mair & Mair, 1968), myocarditis (Mullan, Henry & Beverley, 1968), and hepatitis (Vischer, Bernheim & Engelbrecht, 1967). Meningo-encephalitis, the most important feature of congenital toxoplasmosis (Nutt & Beverley, 1963) also occurs with the acquired disease; Sabin (1941), Sexton, Eyles & Dillman (1953) and Kayhoe *et al.* (1957) have reported severe cases which reached necropsy. However, since Sabin's original paper 30 years ago there have been few cases of toxoplasma encephalitis diagnosed during life (Beverley, 1969; Fleck, 1971). The following case is of particular interest in that the patient was fond of eating practically raw steak. This fact may be relevant to the as yet uncertain mode of transmission of the organism in man.

Case report

A 23-year-old married theatre sister was admitted to hospital in October 1970 as an emergency having become confused on her way to work. She had been

well until 8 days previously when she developed diarrhoea and vomiting which lasted 4 days. Forty-eight hours prior to admission she became drowsy, developed headache, photophobia and pain about the eyes and vomited. A history was obtained later that she ate extremely undercooked and, on occasions, even raw steak. She kept a budgerigar at home. There was no other history of contact with animals.

On examination: Temperature 99° F. Drowsy, irritable with photophobia and mild neck stiffness. Fundi normal. No neurological signs, lymphadenopathy or splenomegaly. General examination normal.

Investigations: Hb, 16.2 g/100 ml; WCC, 7700/mm³; lymphocytes, 52%; ESR 6 mm/hr. Cultures and microscopy of blood, urine, stool—normal. No viruses isolated from stool. CSF: normal pressure. WCC, 1/mm³; protein, 38 mg/100 ml; sugar, 50 mg/100 ml; WR and Lange, negative. No viruses grown. Glandular fever screening tests, negative. Extending Widal compatible with previous TAB—no rise in titre. Immunoglobulin levels—normal. Mantoux 1 in 10,000—positive. Serum glutamic oxaloacetic transaminase 76 Karmen units/min/ml. ECG—widespread T wave flattening. Serum bilirubin, alkaline phosphatase, chest X-ray, Reiter CFT and Kahn—all normal. Toxoplasma dye test titre—zero.

Progress. The patient's symptoms and low-grade irregular fever subsided spontaneously over 3 weeks and she was discharged to convalesce at home. Ten days later she was re-admitted with further headache, photophobia and fever, and a complaint of dropping things from her right hand. The physical signs were unchanged as were her blood count and CSF. The ECG and SGOT were now normal. An EEG was also normal. The toxoplasma dye test titre had now become positive at 1 in 2000. No toxoplasma were isolated from stool or CSF. A second serum showed no rise in titre or evidence of co-incidental infection with mumps, herpes simplex, lymphocytic choriomeningitis viruses, or leptospirosis.

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Treatment with Sulphatriad 3 g and pyrimethamine 25 mg daily relieved her symptoms and fever after 48 hr. The drugs were discontinued when thrombocytopenia developed 10 days later. Folinic acid was then given intramuscularly with a good haematological response. The toxoplasma dye test titre rose to 1 in 64,000 and the patient's symptoms recurred a third time, 4 months after her initial admission. A month's course of sulphadiazine 6 g, pyrimethamine 25 mg and folinic acid 9 mg i.m. daily was given uneventfully and 6 months later she remains well. At no time was there any lymphadenopathy clinically.

Comment

The diagnosis of acute toxoplasmosis seems well established here with the appearance of high antibody titres in the toxoplasma dye test during the course of a remitting encephalitic illness. A sub-clinical myocarditis was also documented upon serial ECGs in the earlier stages of the disease.

Toxoplasma gondii is widely dispersed in man and many species of animals and birds but the mode of transmission is uncertain. It has been suggested that ingestion of infected meat could be a source of infection in man. The organism is readily killed by only moderate degrees of heat as may be found in grossly undercooked joints of meat (Garnham & Lainson, 1960). However, Desmonts (1962) has shown that introducing uncooked meat into the diet of institutionalized children was followed by the production of toxoplasma antibodies. The dietary habits of the present patient may therefore be relevant to her illness. The diarrhoea and vomiting which preceded the encephalitic illness by about 1 week, could conceivably have been due to massive gastro-intestinal infestation following ingestion of infected raw steak. Toxoplasma was not isolated from the patient's budgerigar's stools and the patient's husband's toxoplasma dye test titre was zero 2 months after the onset of her illness. The latter is a further pointer against a common airborne or

dietary source. Her husband is not a raw steak eater.

Pyrimethamine is an antifolate agent and may produce haematological complications. Folinic acid prevents or reverses these and does not interfere with the toxoplasma effect (Beverley, 1969). It should therefore be given routinely with sulphonamide and pyrimethamine in the treatment of toxoplasmosis.

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