list of possible causes of chronic meningitis.

J DUBOST†
IL VIALLARD†
B SAUVEZIE*

Unit of Clinical Immunology*
(Prof S RAMPON)
Department of Biochemistry†
Centre Hospitalier Universitaire
63000 Clermont Ferrand
France

References


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Acute acquired toxoplasmosis causing neuroptico-meningoencephalitis in an immune compromised boy

Sir: Neurological involvement occurs in only 4–8% of cases of symptomatic acquired toxoplasmosis.† The disorders include encephalopathies, meningoencephalitis, and enlarging cerebral mass lesions but, to date, optic neuritis has been reported only incidentally.‡ § Immunocompromised hosts usually are affected.

A 13-year-old Caucasian boy, with unrecognised rare psychomotor seizures since the age of ten, developed, in mid-December 1981, progressive bilateral amaurosis with slight obtundation, moderate occipital headache and transient macular rash on the left inferior eyelid. On 8 Jan, 1982, inability to count fingers at 30 cm, normal peripheral visual fields on confrontation, bilateral reactive mydriasis, full ocular movements and normal ocular fundi were noted by a consultant ophthalmologist. Methylprednisolone 20 mg per day resulted in minimal improvement in vision, but fever and a generalised tonic-clonic seizure occurred.

On admission, on 19 Jan, 1982, central temperature was 38.3°C, and there was minimal obtundation. The bilateral central scotomas persisted, but ocular movements, pupils and ocular fundi were normal. Clinical examination was otherwise normal. CT scan showed bilateral areas of decreased density in the medial prefrontal regions with slight enhancement after contrast injection and no mass effect. EEG and right carotid angiography were normal. Lumbar puncture (table) showed 70/mm3 mononuclear cells with rare plasmocytes and normal protein, immunoglobulin G and glucose levels. Bacterial cultures were negative. Erythrocyte sedimentation rate was 3 mm/h. Routine blood and urine analysis and chest radiographs were normal. Tuberculin skin test was one plus. Daily infusions with 200 mg hydrocortisone hemisuccinate were given, with an immediate correction of the temperature and a moderate improvement in vision.

Two weeks after admission, the results of serodiagnosis were received. They were positive for Toxoplasma gondii (table) but negative for Rickettsia, Leptospira, Treponema pallidum, Brucella, Salmonella, Mycoplasma pneumoniae, Chlamydia, Myxovirus, Enterovirus, Herpes simplex virus, Cytomegalovirus, Adenovirus. The Paul-Bunel-Davidson reaction was negative. From 5 Feb, spiramycin 3 g and sulfadiazine 3 g per day were given orally for 45 days, with prednisone 30 mg per day for the 1st month, 15 mg per day for the 2nd month and 15 mg any other day for the 3rd month. Vision dramatically improved and the patient was discharged.

Three months later there was no complaint. Visual acuity, visual fields, ocular fundi and CSF analysis were normal. CSF inoculation into mice was negative. In August 1982, results were normal for serum and urinary electrophoresis and immunoelectrophoresis, serum circulating immune complexes, complement components, auto-antibodies to nuclear and organ antigens. Peripheral blood count, T and B cells percentages, T-cell subsets as determined by OKT monoclonal antibodies gave, as the only abnormal result, a slight increase of phenotype T 8+ (F Touraine, Hôpital Neurologique, Lyon). The HLA-typing was positive for the A2, B7, BW1, and CW1 antigens.

The patient was periodically re-examined up to Jan 1984. Fever and optic neuritis never recurred. After Jan 1983, the Sabin-Feldman test positivity decreased to 1:800 in serum. In Jan 1984, the electro-oculogram was normal and pattern-reversal visual evoked responses were normal for the right eye (102 ms) but delayed for the left eye (116 ms). The cryo-preserved original sera were retested in May 1984 (J Andre, Institut Pasteur, Lyon), using the IgM-ELISA kit for T gondii (Labsystems, Helsinki). There was a slightly positive result for the first sample only.

This immunocompetent boy suffered from an acute meningoencephalitis with bilateral optic neuritis. The diagnosis of T gondii infection was considered from the results of initial serodiagnostic tests performed twice at a two-week interval. The Sabin-Feldman test was highly positive in the serum when first performed and a single high titre of 1:32 000 or more is considered as diagnostic.† The possibility of missing the ascending phase of the antibody production is well-known,§ and, in our case, the overt disease had been progressing for at least 5 weeks. The IgM-IFA test was negative, but antitoxoplasomic IgM production can disappear after the first weeks of the infection, especially in

Table 1  CSF abnormalities (Steroids were started in February 1982)

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<td>Cell/mm³</td>
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<tr>
<td>M: Polymorphs</td>
<td>26</td>
<td>26</td>
<td>26</td>
<td>26</td>
<td>14</td>
<td>1</td>
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<td>M: Monocytes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5</td>
<td>1</td>
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<tr>
<td>LY: Lymphocytes</td>
<td>(Ly: 71%)</td>
<td>(Ly: 30%)</td>
<td>(Ly: 16%)</td>
<td>(Ly: 5%)</td>
<td>(Ly: 4%)</td>
<td>(Ly: 2%)</td>
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<tr>
<td>Protein g/l (% Gammaglobulin)</td>
<td>1.23</td>
<td>1.43</td>
<td>1.71</td>
<td>1.02</td>
<td>1.18</td>
<td>(10)</td>
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<tr>
<td>Glucose g/l (Blood Glucose level: g/l)</td>
<td>0.27 (1.15)</td>
<td>0.30 (1.18)</td>
<td>0.50</td>
<td>0.40 (0.90)</td>
<td>0.63</td>
<td>0.58</td>
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*Letters

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patients with a very active synthesis of specific IgG. Negative results in the IgM-IFA test have also been attributed to steric competition from high levels of specific IgG as in our case. This bias may be avoided by IgM-ELISA tests and, when the original sera of our patients were retested with the IgM-ELISA assay, we found slightly positive results for the first sample only (Table). Cultures from CSF were negative for T. gondii, but they were made after the course of antibiotics. Diagnosis was confirmed by the close correlation between acute symptoms and high dye test titres, the clinical recovery obtained with corticoids and specific antibiotic therapy, and the sixfold decrease in dye test titres and the IgM-ELISA assay. A two-year follow-up did not afford an explanation other than toxoplasmosis for the initial symptoms and signs. The acquired nature of toxoplasmosis in this case seemed probable, in view of the clinico-serological correlations, lack of the classical features of congenital toxoplasmosis and positivity of IgM-ELISA test.

Loss of vision in acquired toxoplasmosis is usually due to chorioretinitis or changes in the media of the eye, associated with inflammation. Only two cases of cerebral blindness with involvement of central visual pathways have been reported to date. Optic neuritis is exceedingly rare. Single cases have been reported in association with overt ocular lesions. We are aware of only three reports with normal ocular fundi. In Hogan's case there was concurrent definite multiple sclerosis. In Wood's case, the dye test was positive at 1:256 only, and there were no physical signs other than optic neuritis. In Wood's case, a classical picture of optic neuritis evolved in a 25-year-old patient with acute febrile illness, cervical enlarged glands and mononucleosis. Toxoplasmosis was diagnosed by a dye test positive at 1:1024 and complement fixation plus four. In the case of Rieger, chill is, left acute optic neuritis, right eye upward paresis was observed in a 49-year-old patient. Dye test was positive at 1:64 and, three months later, at 1:1024. There was a canine source of infection. Such a low incidence of optic neuritis in acquired toxoplasmosis is probably due to the obligatory intracellular parasitism of T. gondii, although the presence of the parasite in the optic nerve has been mentioned. It is important to consider acquired toxoplasmosis in the diagnosis of acute meningoencephalitis even in immunocompetent hosts. Acute optic neuritis is not an argument for discarding the diagnosis. Prompt confirmation of T. gondii infection by laboratory investigations is critical for this potentially curable disease.

Table CSF and antitoxoplasmic antibody studies

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<td>Serum</td>
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<tr>
<td>Dye test titre</td>
<td>1:51 200</td>
<td>1:51 200</td>
<td>1:51 200</td>
<td>1:51 200</td>
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<td>IgM-ELISA test</td>
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<td>—</td>
<td>&lt;20</td>
<td>&lt;20</td>
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<td>CSF</td>
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<td>White cells/mm³</td>
<td>27</td>
<td>24</td>
<td>&lt;2</td>
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<td>Total proteins (mg/l)</td>
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<td>N</td>
<td>N</td>
<td>N</td>
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<td>Immunoglobulin G (mg/l)</td>
<td>270</td>
<td>260</td>
<td>300</td>
<td>310</td>
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<td>Dye test titre</td>
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<td>IgM-IFA test</td>
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IFA = Immuno Fluorescent Antibody
PAGE = Poly Acrylamide Gel Electrophoresis
N = Normal

For the IgM-ELISA test, results are expressed in enzymatic units (<20 U = negative; 20-40 U = doubtful; >40 U = positive).

References


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“True” cystic meningioma.

Sir: It has long been known that a meningioma may be associated with cysts which increase its compressive effect. These tumours often have been described as cystic meningiomas, although this is an unsatisfactory term. The case we present...