A case of acute polyradiculoneuritis and acquired toxoplasmosis

Sir: From the literature it seems that acute acquired toxoplasmosis rarely precedes acute polyradiculoneuritis of the Guillain-Barré type. We found only three cases (reported in German and French), where screening of patients with acute polyradiculoneuritis disclosed antibodies against Toxoplasma gondii in titres compatible with recent infection. None of them had clinical toxoplasmosis. Owing to the possible serious consequences of failure to diagnose toxoplasmosis, we report a patient with this condition developing acute polyradiculoneuritis.

The patient was a 21-year-old unskilled labourer working for an electrician, and had a history of drug-abuse including morphine and heroin for four years until the age of 18. He experienced hay fever when exposed to various species of grass, grain and bruces. During the autumn of 1981 occipital and cervical lymphadenopathy developed. He did not feel ill and had no fever. Treatment with penicillin was given without effect. About 4 December his fingers and feet started to tingle. Further treatment with penicillin had no effect. Next the power of his legs weakened and the legs gave way when he was walking. On admission on 17 December, generalised lymphadenopathy was present. Muscular power, tone and proprioception in the legs were diffusely reduced, so that he could walk only with great difficulty with ataxia. Left-sided superficial abdominal reflexes, ankle jerks and plantar responses were absent. The rest of the nervous system was intact. He looked fit and had no rash. Total white blood cell count was normal though in two samples 78 or 59% of the leucocytes were lymphocytes (some resembling those found in virus infections). Haemoglobin level, serum-aspartate-aminotransferase, test for infectious mononucleosis, Wassermann reaction of blood and CSF, chest radiology and urine examination were all normal. CSF total protein content was 0·81 g/l but without pleocytosis. Suboccipital lymph node biopsy showed diffuse hyperplasia and focal histiocytosis as in toxoplasmosis. Bone marrow examination added nothing further.

On further questioning the patient revealed that he had been plucking ducks recently and that he lived together with two cats which hunted on their own and frequently passed stools on the floor. Antitoxoplasmosis treatment was commenced with sulfadiazine 1 g daily in combination with pyrimethamine 25 mg twice daily for two days followed by 25 mg daily. The Sabin-Feldman dye test for toxoplasma-antibodies (SFDT) titre was 1:6250 and complement fixation test (CFT) titre more than 1:128, which implied recent infection.

Difficulty in walking progressed and the patient had trouble controlling his hands. He was transferred to the Department of Neurology on 25 December, where examination showed that all superficial abdominal reflexes had disappeared, the tendon jerks were sluggish in the arms but no reduction of power. Strength in all muscle groups of the lower extremities was reduced, more proximally than distally. The tendon reflexes in the legs were absent. He could not stand without support. During the next three days there was some reduction of strength of handshake, and flexion and extension of elbow-joints. For the next two days the condition was stationary and in the following three weeks he recovered fully and treatment with antitoxoplasmotic drugs was discontinued. The patient's body temperature had remained normal during his illness and the liver and spleen were never enlarged. On 8 January a further rise of serum toxoplasma antibodies (titres of 1:31250 by SFDT and 1:256 by CFT) proved that an acute toxoplasma infection was present. CSF protein had now risen to 3-0 g/l while cell content was still normal. Ten days later the SFDT titre had fallen to 1:1250 while the CFT titre was unchanged.

At follow-up examination, October 1982, the patient was well and fit for work. A few small lymph nodes were felt in the axillae and groins. Slight wasting of the right leg now was present. Muscular tone was decreased bilaterally and the left plantar response was absent. The rest of the neurological examination was normal. SFDT titre was the same as on 18 January 1982, while the CFT titre had gone down to 1:64.

Toxoplasma antibodies occur in the sera of at least 2% to 36% of adults. Most cases of toxoplasmosis pass unnoticed. A small number cause lymphadenopathy but run a benign course. Toxoplasma gondii may persist in cysts in tissues after infection with a potential for uncontrolled multiplication if the host is subjected to immunosuppressive therapy. In patients receiving corticosteroids and/or cytotoxic drugs toxoplasmosis may be fatal from encephalitis or myocarditis. Isolation or demonstration of the parasites is rarely feasible. Therefore serological tests are the mainstay of the diagnosis. The Sabin-Feldman dye test or the indirect fluorescent antibody test are the most precise methods. In the previously reported cases of acute polyradiculoneuritis associated with high titre of toxoplasma antibodies (initial dye test titre 1:4000), the plasma level was maximal when first measured, although in one case the complement fixation titre was rising while the dye test titre was falling. None of them had clinical toxoplasmosis. A child of 9 years of age progressed to involvement of larynx and pharynx with respiratory insufficiency. This child had an episode of bilateral papilloedema and convulsions but computed tomography of the brain showed no abnormality. The other cases of a girl of 10 and a man of 64 progressed to a severity comparable to our patient. All the cases from the literature were left with some sequelae. Only one of them had pyrimethamine and sulfadiazine, in combination with prednisone for one month. A case with low back pain and bilateral paresis of both legs has been published, where toxoplasma were present in the CSF.10 In this case pleocytosis of the CSF was present disappearing after treatment with sulfapyridine. No attempt to isolate parasites from the CSF was undertaken in our case. A history of preceding infection is common in acute polyradiculoneuritis but no prior large scale study seems to have included screening for toxoplasma antibodies.

In conclusion, toxoplasmosis ought to be excluded in cases of the Guillain-Barré syndrome, as treatment with pyrimethamine and sulfadiazine might improve the prognosis and shorten the recovery phase if toxoplasmosis is found. In any case immunosuppressive therapy should not be given for acute polyradiculoneuritis without ruling out toxoplasmosis.

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References

3. Lavaud J, Cochois B, De Leersnyder H.


Accepted 14 September 1983

Matters arising

Spatial disorientation in right hemisphere infarction

Sir: Personal involvement in the care of a close relative can make reading a related paper especially interesting for a physician. It was in this sense that I read JD Meerwaldt’s article about the prognosis of spatial disorientation due to right hemisphere infarction with particular interest.1 In his study only one of 16 patients did not recover completely within six months after an ischaemic stroke. I have had the unfortunate opportunity to follow my father’s illness with the same syndrome and my comments seem relevant.

He is presently 82 years old, and was in excellent health until three years ago. He had a few brief episodes of dizziness prior to May 1981, when he fell off his bicycle as a result of one such episode. On initial neurological examination he had mild left sided hemiparesis, left homonymous hemianopia in addition to the usual right parietal signs: constructional and dressing apraxia, prosopagnosia and profound spatial disorientation. This was so severe that he could not find his way in and around the house that he had lived in for over 15 years. CT scan and angiograms were not performed, but he had no midline shift on his echoencephalogram and the EEG showed right temporo-parietal slowing only. He was discharged from hospital with the diagnosis of an ischaemic stroke due to probable middle cerebral artery occlusion. I first had the opportunity to examine him about two months after the initial event. By then the hemiparesis had almost completely disappeared, and there was only visual extinction, but no hemianopia, on the left side. He still had difficulties with dressing himself and was unable to “build” a house from matches. He was unable to name directions, could not describe how to get to the houses of his children, how to find the local railway station, market place etc. He recovered from some of his symptoms by July 1983, when last examined. He still complained of stiffness in his left arm, made frequent mistakes with naming directions and was unable to get home from a distance of a few hundred yards. The disorientation was especially noticeable in darkness, and a light had to be left on in his bedroom to help his orientation in the evening and at night.

It would not be prudent to compare the results of a test done on 16 patients with the clinical findings on one patient. The disparity between the quick recovery in Meerwaldt’s 15 cases and my father’s slow and incomplete recovery, however, is quite noticeable. Age difference might have been one of the reasons (my father was 80, the oldest in Meerwaldt’s study 70). Meerwaldt commented on the difference between his and Benton’s2 findings, and explained it on the basis of different aetiologies and presence or absence of cerebral oedema. My father in all probability, although there was no CT scan or angiographic proof, had an ischaemic infarct, and should be comparable to Meerwaldt’s cases. I am inclined to believe that the discrepancy is basically artificial and related to the unnatural examination technique used by Meerwaldt. Neither the rod, nor the line orientation test can be used as substitutes for testing in real life situations and for inquiring from relatives about the patients’ behaviour. This, of course, is difficult to quantify and difficult to use in scientific research. This method alone, however, is unlikely to provide us with the information about prognosis in this syndrome that practising neurologists are looking for. It would be advisable to complement it with corresponding clinical information in similar future studies.

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References


Meerwaldt replies:

In a previous article about spatial disorientation1 26 patients with an infarct in the posterior region of the right hemisphere were tested two weeks and one year after the stroke. Five patients showed only a partial recovery of spatial functions and had still symptoms which could be attributed to disturbances of spatial perception. These five patients had the largest lesions on CT scanning.

The findings in these patients are in accordance with those described by Dr Suranyi concerning his father. As no CT scan was made, exact information about the aetiology, location and the volume of the lesion is lacking, but possibly he had a large lesion as our patients had. I agree with Dr Suranyi that neither the rod orientation test nor the line orientation test can be used as substitutes for testing in real-life situations, but I would state that none of the patients who showed a complete recovery of spatial disorientation as tested with the rod orientation test had any complaints after one year which could be attributed to disturbances of spatial perception.

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Notice

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For all information contact: Prof. E.S. Vizi, c/o Congress Secretariat, Fifth Meeting of ESN, Motesz, POB 32, Budapest, H-1361, Hungary.
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*J Neurol Neurosurg Psychiatry* 1984 47: 219-220
doi: 10.1136/jnnp.47.2.219

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