FIVE CASES OF NEUROCYSTICERCOSIS DIAGNOSED IN SYDNEY

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Abstract. Cysticercosis, once rare in Australia, is now more frequently diagnosed. This change reflects the countries of origin of new immigrants and the destinations of Australians travelling. Five cases of neurocysticercosis diagnosed at Westmead Hospital in Sydney are described. Two involved Australians, a father and son who had visited eastern and southeastern Asia 10 years before presentation. The other three included immigrants from Chile and India and a visitor from Timor. Ages ranged from 5 to 57 years. Three individuals presented after focal seizures involving the upper limb, one had a long standing history of neurological dysfunction and one suffered from persistent headaches. In all cases computed tomographic scanning (CT) or magnetic resonance imaging (MRI) revealed cystic brain lesions and three of the five were seropositive as well. Four were treated with praziquantel and in one the lesions regressed significantly following treatment. However, the lesion in one case had decreased in size prior to treatment and that in the untreated individual also became smaller.

INTRODUCTION

Patterns of imported disease reflect both travel by residents of a country and immigration. Recent changes in the countries of origin of new immigrants to Australia and in the destinations of Australians travelling overseas have led to some parasitic diseases, previously extremely rare, being detected regularly. One of these is cysticercosis, infection with the larval stage of *Taenia solium*. Crimmins *et al* (1990) described eight cases of neurocysticercosis seen at two hospitals in eastern Australia, the patients involved being immigrants from Eastern Europe, Asia or Central America and ranging in age from 14 to 73 years. A further two cases, one in an immigrant from Laos and the other in an Australian who had travelled to Asia, including Southeast Asia nine years previously, were described by McDowell and Harper (1990). Five previously unreported cases diagnosed at Westmead Hospital in Sydney are described in this paper.

CASE REPORTS

Case 1

A 27 year-old Australian male who had visited Thailand, Hong Kong, Southern China and Indonesia with his family in 1978. He presented at hospital in June 1988 following a focal seizure involving the right upper limb; he was not treated at this time. In September 1988 he had a further seizure involving the right upper and lower limbs, accompanied by dysesthesia. This lasted for 5 to 10 minutes, with no loss of consciousness. He had suffered from bifrontal headache for 12 months. Physical examination was normal, as were a full blood count and biochemical profiles. An electroencephalogram revealed sharp delta activity in the left frontal region. Examination of six stool specimens revealed no cells, cysts or eggs. The cerebrospinal fluid (CSF) protein level was 520 mg/l (normal 150–450), the glucose level was 3.4 mmol/l (normal 2.8–5.5) and there were no cells or organisms seen or cultured. CSF cryptococcal antigen was not detected and no acid fast bacilli were seen or grown. Venereal Disease Reference Laboratory, human immunodeficiency virus antibody and Mantoux tests were all negative. A cerebral CT scan revealed six small lesions in both temporal and parietal lobes, associated with surrounding edema. There was no hydrocephalus. Stereotactic biopsies of two of the lesions produced tissue identified as cysticerci. Serum sent to CDC, Atlanta Georgia, was antibody positive by enzyme-linked immunoelectrotransfer blot assay (EITB). The patient

was treated with phenytoin, praziquantel (50 mg/kg/day in 3 divided doses for 15 days) and dexamethasone (16 mg/day). Twenty months after completion of treatment there have been no recurrences of symptoms and the lesions are regressing.

Case 2

A 57 year-old male Australian, father of case 1 and a frequent traveller to Southeast Asia, was tested serologically following diagnosis of his son's condition. The result of the test (EITB) was positive. He gave a history of persistent generalized headache for several years. CT scans and MRI of the brain demonstrated multiple cysts in the cerebral hemispheres and around the 4th ventricle. He was treated with phenytoin, praziquantel (50 mg/kg/day in 3 divided doses for 15 days) and dexamethasone (16 mg/day). The headache persisted and two further doses of dexamethasone were given over 14 days each with partial relief of the headache. At 12 months followup he still has residual headache although on CT scanning there has been marginal improvement.

Case 3

A 49 year old Chilean male from Valparaiso who presented for assessment of long standing epilepsy. He had emigrated to Australia in 1979. In 1974, whilst in Chile, he had experienced a total of eight grand mal seizures. Phenytoin was commenced, but ceased in 1976. In 1980 the seizures recurred and phenytoin was recommenced. An EEG performed was normal. A cerebral CT scan showed paraventricular calcification and small, low density lesions in the left temporal and right parietal lobes. A diagnosis of tuberous sclerosis was made. In 1986 he experienced acute onset of weakness and paresthesia of the left leg. A myelogram and CT scan revealed a uniformly expanded spinal cord at the second thoracic vertebral level. A laminectomy was undertaken at that level. Histological specimens examined were normal. Carbamazepine was added to his treatment regimen. In October 1988, cerebral MRI was performed. This revealed multiple well defined cystic lesions with mural nodules in both cerebral hemispheres and basal ganglia. The ventricular system was not dilated. Subcutaneous nodules were found on the left arm and right thigh. That on the thigh was excised and examined, revealing a degenerate cysticercus of *Taenia solium*. X-rays of soft tissues revealed widespread calcified lesions. Serum and CSF were antibody positive on the EITB (CDC). He was treated with praziquantel (50 mg/kg/day in 3 divided doses for 15 days) and dexamethasone (16 mg/day).

Case 4

This 5 year-old girl arrived in Australia from Timor with her grandmother the day before presentation to hospital with a right-sided focal seizure, which lasted for 15 minutes, primarily involving the arm. There was no loss of consciousness. A biochemical profile and chest X-ray performed were normal. The cerebral CT scan showed a single enhancing lesion in the left parietal region with surrounding edema, which was subsequently confirmed on MRI scanning. The EEG showed slowing from the left posterior quadrant. Cerebrospinal fluid examination was normal. Serology for cryptococcosis, toxoplasmosis and echinococcosis were within normal limits and the Mantoux test non-reactive. Immunodiagnosis for cysticercosis by EITB was also negative.

A diagnosis of cysticercosis was made on the basis of the CT and MRI findings, clinical presentation and country of origin. Although a second CT scan, without enhancement, one week after the first revealed that the lesion had regressed to a small amount of edema in the subcortical white matter, because followup was uncertain, it was decided to offer treatment with praziquantel. This was completed without complications. The patient returned to Timor continuing anticonvulsants and was lost to further follow-up.

Case 5

This 12 year-old girl who had lived in the Punjab region of India until the age of 9 years, returned to India for a holiday early in 1990. During this time she experienced a focal seizure of the right hand while opening a door. There were no other symptoms or loss of consciousness. A CT scan performed in India showed a ring

enhancing lesion in the left parietal cortex. The focal seizures returned shortly after her return to Sydney. An MRI scan showed extensive edema surrounding a cortical lesion which appeared to contain a small inclusion.

Cysticercosis seemed likely because of the exposure history, the clinical presentation and the scan appearances. The cerebrospinal fluid was normal and appropriate serological assays and a Mantoux test were negative. EITB for cysticercosis was also negative. As follow-up was assured and the benefits of praziquantel treatment uncertain, she was reviewed with further scans at two and five months after initial presentation. In subsequent scans there has been progressive reduction in the lesion, the last showing only a pinpoint area of enhancement with contrast. She has had one recurrence of focal seizures, with subsequent adjustment of her anticonvulsant medication, but no other problems.

DISCUSSION

The diagnosis of human cysticercosis has been significantly enhanced by the application of new imaging techniques, particularly MRI (Camargo and Marshall, 1987) coupled with improved immunodiagnostic assays, but a significant degree of clinical acumen is also required in many instances. The spectrum of possible neurological abnormalities associated with neurocysticercosis is varied and non specific (Del Brutto and Sotelo, 1988). The variation relates to the number of lesions, their site in the brain and the nature of the host's immune response. There is no pathognomonic feature or typical syndrome of neurocysticercosis and even the most common clinical sign, epilepsy, occurs in only 52% of cases (Sotelo *et al*, 1985). Fifty percent of cases have mixed forms of the disease and 25% exhibit normal neurological function (Del Brutto and Sotelo, 1998). Case 2 probably represents an instance of an individual with multiple lesions yet normal brain function because his headaches could well have been due to other causes. Cases 1, 4 and 5 had localized partial seizures, probably indicative of the position of the cysticerci in the brain parenchyma. The range of symptoms exhibited by Case 3 over 14 years relate to the wide dissemination of cysticerci throughout his body.

In the first three cases the provisional diagnosis was supported by subsequent testing of serum and CSF by EITB at the Centers for Disease Control in Atlanta, Georgia. This immunodiagnostic test is currently not available in Australia. Initial assessment of the test suggested that it was 98% sensitive and 100% specific (Tsang *et al*, 1989) but the fact that cases 4 and 5 were serologically negative suggests that the sensitivity may not be high in those individuals with only a single lesion. This highlights the issue of the value of screening family members following the diagnosis of cysticercosis in an individual. The father of Case 1 was only diagnosed as having cysticercosis after he was found to be seropositive on EITB. Further investigation revealed multiple brain lesions. Had he had a single lesion the test may well have been negative. Despite this limitation the practice of screening family members would appear to be worthwhile as it should detect those individuals at greater risk with multiple cysticerci.

This series of cases also emphasizes the need for clinicians in countries such as Australia to be aware that cysticercosis may be a cause of a diverse range of neurological presentations, not only in immigrants, but in nationals who have travelled to endemic regions, even for a very brief period.

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