
Letters

Cerebellar encephalopathy in cat scratch disease

Sir: Cat scratch disease is a type of subacute regional adenitis first recognised by Forshay1 in 1932 and elaborated by Debre2 in 1950. Until recently cat scratch disease was thought to be due to an unidentified non-bacterial agent, but in 1983 it was reported that a pleomorphic gram negative bacillus was responsible.3,4 In 1985 a highly pleomorphic gram positive bacillus of the Rochia genus was identified and isolated from lymph nodes of a patient with cat scratch disease using the Warthin-Starry silver impregnation stain. The organisms were sensitive in vitro to a variety of antibiotics including penicillin, erythromycin and cotrimoxazole.5

The diverse presentations of cat scratch disease include osteomyelitis, thrombocytopenic purpura and the ocu-lo-lingual syndrome of Parinaud.6 Neurological manifestations are uncommon but encephalopathy, myelitis, radiculitis, optic neuritis with compressive neuropathy7 and arteritis8 have been reported. Encephalopathy is the most common, presenting with confusion, coma, seizures accompanied by diffuse high voltage slow wave changes in the EEG.9 Since the first description of encephalopathy in 1952 there have been 25 reports.9-13 The majority recover completely within a few weeks.

Since there have been no previous reports of cerebellar encephalopathy associated with cat scratch disease we present such a case highlighting the difficulties in making the diagnosis and stressing the benign prognosis without treatment.

A previously fit 14 year old boy presented to his General Practitioner with a febrile illness. Over the course of 3 weeks he became increasingly unsteady and clumsy and developed slurred speech. He was found to have an enlarged left epitrochlear lymph node. On admission to hospital, we discovered that he had recently acquired a kitten which he had scratched his left hand 4 weeks before.

On examination, he was apyrexial and had a single 2 cm rubbery non-tender left epitrochlear lymph node. He had normal cardiovascular, respiratory and abdominal systems. He was conscious, orientated and alert with normal cortical functions. He had dysarthria but other cranial nerves were normal. He had normal strength, tone and reflexes in all limbs with flexor plantar responses and no sensory abnormality. He showed marked intention tremor of both arms (left worse than right), bilateral dysdiadochokinesia and incoordination of heel shin movements. He showed a broad based high stepping gait with truncal ataxia at rest and whilst walking. There were no primitive reflexes.

Findings included haemoglobin 13.4 g/dl, white cell count of 13.5 x 10^9/l (76% neutrophils, 16% lymphocytes, 8% monocytes). A biochemical profile was normal. Chest and skull radiographs and CT brain scan were normal. The CSF showed 4 red cells mm^-3 and no white cells with a total protein of 0-70 g/l (IgG 0-10 g/l) with no organisms and no growth on culture. Visual evoked responses (P = 98-4 ms R = L) were normal; brain stem evoked responses and somatosensory testing were also normal. Histology of a biopsy of the left epitrochlear node showed a granulomatous abscess typical of cat scratch disease. Microscopy of the fluid aspirated from the node showed some leucocytes with occasional gram negative bacilli which did not grow on culture. Culture for tuberculosis and antibiotic titres to viruses were negative. Cat scratch disease skin antigen was not available locally or from the Central Public Health Laboratory, Colindale. The node was stained with the Warthin-Starry technique5 but no bacilli were seen. The patient made a good recovery and was completely normal after 3 months.

The diagnosis of cat scratch disease was presumed on the basis of (1) a recent febrile illness, (2) a history of a cat scratch with a visible inoculation site, (3) characteristic lymph node histology, and (4) negative studies for other pathogens. We were unable to perform the cat scratch disease skin test.8 The interval between the initial illness and the onset of ataxia was similar to the 3 to 30 days described previously in other neurological syndromes associated with cat scratch disease.11-14 The presence of bilateral signs with truncal ataxia suggests a midline vermis lesion. There was no evidence of a tumour or of a demyelinating disease. The raised CSF protein has been reported previously11,14 although it is unusual.11,13 The raised CSF IgG suggests that this cerebellar syndrome may be determined by auto-immune mechanisms though no lymphocytes were present. Patients with cat scratch disease encephalopathy reported have made a complete recovery.

We were unable to reproduce the recent findings of gram positive bacilli using the Warthin-Starry stain. This may be due to difficulties inherent in the stain technique itself, but it has been suggested that failure to identify the agent may be related to the small number of viable organisms present in the late stages of the disease when the biopsy is taken.5 It may be significant that gram negative bacilli were seen on microscopy which did not grow on culture.

There are still doubts as to the aetiological agent of cat scratch disease and the newly isolated organisms do not respond to antibiotics in vitro.13 This single case does not give any further clues as to the aetiology of cat scratch disease, but does emphasise that the diagnosis of cat scratch disease should be considered in obscure cases of cerebellar encephalopathy.

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References

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