Cat scratch disease presenting as acute encephalopathy

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An unusual case of primary meningo-encephalitis followed by partial complex seizure in a 9-year-old boy was found to be a symptom of cerebral *Bartonella henselae* infection or cat scratch disease. Despite one clinical relapse at 4 weeks post-presentation, he remained seizure free on carbamazepine for one year. Six months after stopping carbamazepine, however, he developed deja vu phenomena and absence seizures with EEG abnormality. Restarting carbamazepine improved his symptoms.

A nine-year-old previously well boy presented to the emergency department with a 4 h history of acute confusional state, unstable gait, vomiting, right-sided facial numbness and flexion posturing of the arms. He was breathing spontaneously, mildly tachycardic at 110 beats per minute and hypertensive at 150/80 mm Hg. His Glasgow coma score was 9, with small and equal-sized pupils. There was no history of drug ingestion, rash, fever, or lymphadenopathy. Initial investigations revealed a blood sugar of 9 mmol/l and a pH of 6.98, with a mixed respiratory and metabolic acidosis. The C-reactive protein, blood count, urea and electrolytes, liver function test and blood coagulation screen were within the normal ranges. Serum paracetamol, alcohol and urine toxicology screens were negative. Following two right-sided focal fits predominantly affecting the face, he required two doses of intravenous lorazepam and transfer to the intensive care unit for intubation and ventilation, intravenous acyclovir and a loading dose of phenytoin. A cranial computed tomography (CT) scan (without contrast) was normal. Cerebrospinal fluid (CSF) analysis revealed 3 red cells/mm³, with no leukocytes or organisms. The CSF glucose was 4 mg/dl and protein was 170 mg/dl. The EEG with high voltage and slow rhythm suggested underlying cerebral pathology consistent with encephalitis, but no epileptic activity was seen.

On return to the ward, his residual problems included slurred speech, amnesia and ataxia, which improved sufficiently to allow discharge one week later. Blood, CSF and urine cultures were sterile and a PCR testing of the CSF for herpes simplex virus, cytomegalovirus and varicella Zoster virus were negative. Serology and CSF PCR for *Bartonella henselae* were, however, positive. Subsequent questioning revealed contact with cats, but no scratches or bite marks preceding admission.

He was readmitted 4 weeks later with a right-sided focal fit and vacant episodes necessitating seizure control with lorazepam, a loading dose of phenytoin and carbamazepine was commenced. With a diagnosis of *Bartonella* infection, oral rifampicin was started. A cranial magnetic resonance imaging (MRI) scan was normal and another EEG, performed a month from onset, revealed medium voltage spikes compatible with partial complex epilepsy. He was seizure free on carbamazepine for one year. Despite some suggestion that academic progress was slower than before the infection, he appeared to return to normal 18 months later. Six months after stopping carbamazepine he was noted to have deja vu phenomena and absence seizures. Restarting carbamazepine improved his symptoms.

**DISCUSSION**

Cat scratch disease (CSD) is caused by *B henselae*, a fastidious Gram-negative bacillus identified in 1992. Healthy domestic cats are carriers. Some 40% of cats are bacteraemic at any one time. Cat fleas (*Ctenocephalides felis*) play a major role in cat-to-cat transmission. Approximately 90% of patients have some history of contact with cats (although not necessarily a bite or scratch).

Typically, a scratch, bite or lick is followed 5–10 days later by a non-tender papule, then ipsilateral regional lymphadenopathy, often cervical, develops 1–3 weeks later.

Atypical presentations occur in 5–14% of symptomatic cases. Encephalopathy, found in 2% of cases, may occur up to 6 weeks after infection. Seizure was the presenting symptom of CSD in 27 of 34 patients with CSD encephalopathy. CSF changes may be minimal, with a mild pleocytosis or slightly elevated protein. As with herpes simplex encephalitis, the EEG may show early changes of diffuse slowing or focal abnormalities with a normal CT or MRI scan. Of 65 patients with encephalopathy, 13 were abnormal on CT or MRI scanning. Abnormalities include focal changes, infarcts or diffuse grey and white matter lesions. There are reports of persisting neurological sequelae such as a 7-year-old boy who presented with seizures and choreoathetosis and in whom involuntary movements persisted, and long-term weakness and hemiplegia in a child with carotid arteritis.

Serology is the diagnostic method of choice, although confirmatory. PCR can also be used to aid diagnosis. The disease is usually self-limiting resolving in 1–2 months. Management for simple cases with analgesics, antipyretics and topical hot compresses for lymphadenopathy usually suffices. Antimicrobials are usually not needed, although azithromycin, rifampicin, trimethoprim–sulphamethoxazole, gentamicin and ciprofloxacin have been used.

**Case summary**

A 9-year-old boy presented to our emergency department with a 4 h history of acute confusional state, a Glasgow coma score of 9 and right-sided focal fit necessitating lorazepam and phenytoin. Blood gases showed mixed metabolic and respiratory acidosis. He was admitted, intubated and ventilated. The initial CSF examination was normal and the EEG showed a high voltage slow rhythm pattern. Serology and CSF PCR for *B henselae* were positive. Despite contact with cats he had no scratch marks. He was discharged home after a week. Four weeks later, he re-presented with right-sided focal fits and vacant episodes. His cranial MRI was normal; EEG was compatible with partial complex epilepsy. He was discharged home on rifampicin and carbamazepine. Carbamazepine was stopped after a year. Six months later he developed absence seizures. Re-starting carbamazepine improved his symptoms.

CSD is caused by *B henselae*. Encephalopathy is found in 2% of all cases. Serology is the diagnostic test of choice and PCR can confirm the diagnosis. The disease is usually self limiting although antimicrobials can be used. Long-term prognosis is typically excellent. We suggest considering CSD as a differential diagnosis in patients presenting with acute encephalopathy.
In severe lymphadenopathy and systemic illness, doxycycline and rifampicin are effective. Treatment duration ranges between 5 days and 6 weeks in immunocompromised patients or endocarditis. Isolated case reports suggest that corticosteroids may be beneficial in encephalopathy. Overall, the long-term prognosis is typically excellent, with or without antimicrobials. Prevention of infection is best achieved by the control of flea infestation in cats and kittens. Our case could have been missed because of atypical presentation. We suggest considering CSD as a differential diagnosis in patients presenting with acute encephalopathy even in the absence of classic features of the disease.

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REFERENCES

Images in emergency medicine

Pacemaker prohibited

Most European ski resorts no longer use photographic identity as evidence of a valid lift pass. Due to technological advances, the new “hands-free” pass which works by electronic recognition has supervened. Unfortunately these external electromagnetic influences (EMI) may affect conventional pacemakers. Thus, any keen skier with a pacemaker would be disappointed to see this warning sign attached to the ski lift turnstiles.

Interaction of an EMI with a pacemaker is a rare but possible serious experience. The result may range from a minor rhythm disturbance to a terminal event. Fortunately, most modern pacemakers have a “noise” mode into which they switch when confusing signals are encountered. This “noise” mode reverts to a generic heartbeat pacing that is sustained until the confusing signal is removed. The real worry is that the only warning is on the turnstile at the base of the slope and the possibility of pacemaker interference is not widely advertised upon purchase of a lift pass.

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