Letter

Acute polyneuritis with cranial nerve involvement following Campylobacter jejuni infection

Sir: Infective agents have rarely been isolated from the 10–20% of patients with acute idiopathic polyneuritis who have had a preceding gastrointestinal illness. An association between Guillain-Barré syndrome and Campylobacter jejuni enteritis has recently been reported. It has been suggested that patients with evidence of preceding Campylobacter jejuni infection manifest a significantly more severe form of the disease. We report a single case in which an acute, short-lived and benign neurological illness characterised by multiple cranial nerve lesions and areflexia occurred in association with this organism.

A 27-year-old motor mechanic developed a diarrhoeal illness with the passage of watery blood-stained motions. Campylobacter jejuni was isolated from his stools at another hospital and his symptoms settled after 7 days. Seven days after the onset of this illness he noticed slurred speech, nasal regurgitation of liquids and some tingling in his hands. Three days later he developed double vision. His symptoms progressed over a further 10 days and he was transferred to this hospital. The patient had no other symptoms, had been well prior to this illness, ate a normal diet and consumed a small quantity of alcohol. There was no history of drug ingestion or of exposure to any toxin prior to this illness.

Examination showed an incomplete, symmetrical, internal and external ophthalmoplegia. Eye movements were limited to 40° downgaze and 10° abduction and adduction. Pupils were equal but dilated and reacted only sluggishly to light.

Speech was nasal with complete paralysis of the soft palate. Facial muscles were weak bilaterally. There was possibly some inco-ordination of the limbs. All deep tendon reflexes were absent with the exception of the supinator jerks which were just present with reinforcement. The plantar responses were flexor. Examination was otherwise unremarkable.

Investigations which were normal included full blood count, urea and electrolytes, B12 and folate, thyroid function, liver function tests and chest radiography. Hepatitis BsAg, Paul Bunnett test, RPR and TPHA and screening for porphyrins were all negative. CSF examination performed 14 days after the onset of his neurological illness showed less than 5 cells/mm³, a glucose content 3.6 mmol/l and a total protein of 0.26 g/l. Motor nerve conduction studies were normal with maximal velocities. The sural sensory action potential recorded antidromically was absent. Median and ulnar nerve sensory action potentials were attenuated and dispersed but with normal latency.

At the time of admission the patient's condition was beginning to improve. Fourteen days later the paraesthesiae in his hands were much reduced, and the eye movements and palate markedly improved. Thirty eight days after the onset of his neurological illness recovery was objectively complete. The only residual symptom was transient double vision during rapid horizontal pursuit movements. Fourteen days later he was asymptomatic and the reflexes had fully recovered. Sensory action potentials in the median and ulnar nerves were much less dispersed and a sural sensory action potential was now recordable and within normal limits.

It has recently been claimed on retrospective serological grounds that Campylobacter jejuni is the most common single pathogen yet reported in association with the Guillain-Barré syndrome. Patients with serological evidence of a preceding infection with this organism were considered to manifest a significantly more severe form of the disease. A search of the literature has revealed only three case reports in which Campylobacter jejuni was isolated from patients who subsequently developed Guillain Barré syndrome. These cases provide some support for the conclusions of the serological study but may not be representative of the true spectrum of severity of this disease. The patient in whom this association was first reported remained confined to a wheelchair eight months after the onset of his illness. The subject of another report appeared to be less severely affected but six months after the onset of symptoms remained disabled by tremor, slight rigidity and distal weakness and wasting. In the sole report of Miller-Fisher syndrome associated with Campylobacter infection the patient required artificial ventilation for 25 days and underwent plasma exchange before recovery.

We consider that our patient's illness was an unusual example of acute post-infective polyneuritis presenting with predominantly cranial nerve involvement. It thus most closely resembled the Miller-Fisher syndrome. While the role of C jejuni infection in the pathogenesis of Guillain Barré syndrome and its variants remains uncertain, this case demonstrates that its isolation from patients with these illnesses does not necessarily infer a poor prognosis.

We are grateful to Dr Michael Hayward for performing the nerve conduction studies in this case.

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References


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