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(Acccepted 27 May 1986)

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Acute encephalopathy associated with campylobacter enteritis

We report a case of acute encephalopathy accompanied by increased intracranial pressure in a child with campylobacter enteritis. This complication has not to our knowledge been previously reported.

Case report

A previously healthy 6 year old boy was admitted to the paediatric intensive care unit, confused after a history of being unable to speak for two days. Five days before admission he suffered from abdominal pain and diarrhoea followed by a headache and temperature of 39°C. On admission he was afebrile and well nourished. His pulse was 50 beats/minute, sinus rhythm, blood pressure 100/60 mm Hg, and respiratory rate 20 breaths/minute without distress. There were no signs of meningeal irritation. On neurological examination the child looked drowsy but was arousable with bursts of irritability. He could follow only simple orders with oriented motor responses. He cried on painful stimuli but was unable to speak. His cranial nerves were intact, and sensory and cerebellar examination findings were normal. His deep tendon reflexes were hyperactive, and bilateral Babinski's sign was elicited. Funduscopic examination showed no papilloedema.

Complete blood count showed 9000 white blood cells/mm^3 with normal differential count, haemoglobin concentration 110 g/l, and 200 000 platelets/mm^3. Biochemical values were all within the normal range. Tests on blood gases, coagulation studies, and urine analysis yielded normal results. The cerebrospinal fluid was initially clear with a pressure of 260 mm Hg, but a brisk movement of the child during lumbar puncture caused it to become macroscopically bloody; it contained 70 white blood cells/mm^3, of which 80% were monocytic and 20% polymorphonuclear. Glucose concentration was 5·0 mmol/l (90 mg/100 ml); concomitant blood glucose concentration was 6·1 mmol/l (110 mg/100 ml). Results of all tests and cultures for bacteria and viruses were negative, except for repeated stool cultures, which grew *Campylobacter fetus*, confirmed by serotyping as *C fetus* subspecies *jejuni* serotype 1,18.

An electroencephalogram showed no α rhythms but instead high voltage diffuse δ activity 2-3 H admixed with a few θ waves, consistent with acute encephalopathy. Computed tomography of the brain showed oedema with small compressed ventricles.

He was treated with dexamethasone and mannitol but no antibiotics. After three days in hospital his condition had greatly improved and he could utter a few words. After eight days he resumed his normal behaviour and speech, and neurological examination was normal. Repeated computed tomography of the brain on the fifth day after admission showed normal ventricle size and no oedema. The electroencephalogram returned to normal three weeks after his illness began, and stool cultures contained no campylobacters.

Comment

Over the past two decades *Campylobacter* has been recognised as a major cause of diarrhoeal disease in children.[1 2] Extraintestinal manifestations of campylobacter enteritis, most commonly presenting as bacteraemia without localised infection, occur mainly in early infancy and compromised adults with underlying systemic diseases.[3] In a review of 247 cases Schmidt *et al* found that the central nervous system was affected in 12% of cases of extraintestinal disease.[3]

Besides seizures, a well documented neurological complication of campylobacter enteritis,[4] effects on the central nervous system include meningitis, meningoencephalitis, stroke, subarachnoid haemorrhage, subdural empyema, and the Guillain-Barré syndrome,[3] all of which are associated with invasion of the central nervous system by the bacteria themselves or with localised neurological disease. The pathogenesis of systemic or extraintestinal campylobacteriosis is unclear. Neurological expression has been attributed to a neurotoxin produced by the bacteria, a process similar to that which causes shigella gastroenteritis. This theory is still controversial and has to be proved.[4]

Most extraintestinal neurological manifestations of campylobacter infection are associated with the subspecies *intestinalis*, whereas in our case *C fetus* subspecies *jejuni* was isolated. It has been recently reported that this subspecies can also affect the brain, and our report supports this observation.[5] Infection with *Campylobacter* should be considered when neurological symptoms such as seizures or increased intracranial pressure complicate gastrointestinal disease.

We express our appreciation to Dr P Lerman for interpretation and follow up of the electroencephalogram, and to the staff of the Campylobacter Reference Laboratory, Israeli Ministry of Health, for confirmation of serotype.

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(Accepted 2 June 1986)

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Lack of antibody to HTLV-I and HIV in patients with multiple sclerosis from France and French West Indies

Recent data have suggested that human retroviruses have an aetiological role in some acute and chronic neurological diseases. Human immunodeficiency virus (HIV; formerly known as lymphadenopathy associated virus or human T cell lymphotropic virus type III) was detected in brain tissue and isolated from cerebrospinal fluid of patients with encephalopathy related to the acquired immune deficiency syndrome.[1] We observed a high prevalence (60%) of antibodies to human T cell lymphotropic virus type I (HTLV-I) in serum from patients with tropical spastic paraparesis, a neuromyelopathy common in tropical areas where HTLV-I is endemic.[2] These findings were confirmed in both serum and cerebrospinal fluid from patients with tropical spastic paraparesis from Jamaica and Columbia.[3] Recently, Koprowski *et al* reported that American and Swedish patients with multiple sclerosis had antibodies that cross reacted with HTLV-I or HIV polypeptides and that cells from the cerebrospinal fluid of four out of eight patients contained sequences related to HTLV-I.[4] We report the results obtained when we tested serum from 55 patients with multiple sclerosis from two other geographical areas.

Patients, methods, and results

We studied 96 patients with neurological disease from Paris hospitals (48 with multiple sclerosis, 10 with amyotrophic lateral sclerosis, and 38 with other neurological diseases) and seven black patients with multiple sclerosis from Martinique (French West Indies). All serum samples were tested by enzyme linked immunosorbent assay for HTLV-I (Biotech, using disrupted virions as antigen) and for HIV (Elavia-Pasteur). None of the samples was positive for antibodies to HTLV-I (estimated by the ratio in the assay and the baseline value) or to HIV. Furthermore, the mean reactivity levels of HTLV-I in multiple sclerosis, amyotrophic lateral sclerosis, and the other neurological diseases did not differ significantly when tested by one way analysis of variance.