Hypokalaemia mimicking Guillain-Barré syndrome

The rapid onset of areflexic weakness is usually due to Guillain-Barré syndrome, but other important causes include metabolic derangements, the periodic paralyses, botulism and poliomyelitis. We report a case mimicking Guillain-Barré syndrome clinically, and initially electrophysiologically, due to the hypokalaemia of renal tubular acidosis, and we document serial nerve conduction studies during recovery.

A 48 year old woman was fit until 1983 when she embarked upon a severe diet involving laxative abuse, losing 50% of her body mass in three months. Subsequently she developed mild generalised weakness and was found to be hypokalaemic. The weakness improved with potassium replacement.

After physiotherapy for six weeks, and recovered completely within three months.

The findings of leucocytosis, eosinophilia, the presence of numerous multinucleated phagocytic macrophages in skin biopsy specimens, raised ESR, and serum IgE levels were also confirmed during the second and third attacks of acute fascitis.

During the third admission the following investigations showed no abnormalities in the blood: red cell count, haematocrit, urea, creatinine, electrolytes, glucose, liver and kidney function tests, Venereal Disease Research Laboratory test, rheumatoid factor, antinuclear antibodies and LE cell tests, serum complement and immune complex levels, serological tests for hepatitis B surface antigen and antibody, and for human immunodeficiency viral antibodies.

In the CSF, microscopic examination of stained specimen (Gram) and cultures for acid-fast bacilli, bacteria, and fungi were normal.

The table shows the results of nerve conduction studies during the acute attack and following recovery. During the attack, no F wave could be elicited although normal or near normal F wave latencies were obtained after recovery. EMG showed no evidence of denervation at any stage, in all three episodes.

Meningitis, encephalitis, encephalomyelitis, cerebral vasculitis, seizures, intracranial hypertension, behaviour disturbances, movement disorders, or spinal cord compression are the known neurological manifestations of fascitis. Extravascular cerebral and meningeal spread, immune allergic reactions, and direct compression of the spinal cord are the mechanisms of nervous insult. Our patient had recurrent Guillain-Barré syndrome, developing severe neurological deficit requiring, on one occasion, mechanical ventilation. All episodes were preceded by a severe attack of acute fascitis. Despite the severity of the nervous system dysfunction with supportive treatment there was always almost complete recovery. The diagnosis of fascitis was strongly supported by the history of typical inflammatory episodes, the presence of tender inguinal lymphadenopathy and lymphoedema of the right leg, microfilariae and eosinophilia in the blood smear, and a raised serum IgE level.

It is possible that recurrent Guillain-Barré syndrome reflects a delayed manifestation of acute fascitis, although it is difficult to establish a causal relationship between the two conditions.

B BHATIA S MISRA Department of Neurology, Laboratory of Clinical Neurophysiology, Institute of Medical Sciences, Banaras Hindu University, Varanasi - 220 005, India

Initial investigations revealed raised plasma levels of sodium (153 mmol/l) and decreased levels of potassium (1.9 mmol/l), with abnormal renal function (urea 22 mmol/l, creatinine 333 mmol/l). Glucose, calcium, magnesium, and creatinine phosphokinase were normal. A full blood count showed a raised white count of 22 000/ml (90% neutrophils). Plasma osmolality was 300 mosm/kg and urine 289 mosm/kg consistent with an element of renal androgenic diabetes insipidus. Arterial blood gases revealed a metabolic acidosis (pH 7.1, bicarbonate 6 mmol/l, base excess -24 mmol/l, pO2 24 and pCO2 31 kPa on inspired 30% oxygen). Urine analysis showed a pH persistently below 6.5 with a sodium of 105 mmol/l and potassium of 24 mmol/l. CSF analysis was normal.

The table shows the results of nerve
conduction studies performed in the intensive care unit on days 2, 3, and 7. The first study showed a marked reduction in the amplitude of surface recorded compound muscle action potentials (CMAPs) to delayed conduction of the median, ulnar, and lateral popliteal nerves, with slight prolongation of distal motor latencies and delay and paucity of F waves. Motor conduction velocities were normal but sensory action potentials were of normal amplitude. Electromyography with a concentric needle electrode in the tibialis anterior revealed no spontaneous activity, but no motor units were seen under voluntary control and there was only a small visible muscle twitch to nerve stimulation (the patient was mildly sedated for ventilation, but neuromuscular blocking drugs were not administered). An electrocardiogram on admission was normal.

Initial treatment consisted of intravenous potassium and fluids with parenteral antibiotics, as serum potassium rose, her strength improved and leg reflexes returned. Between days 2 and 3, proximal muscle power increased from grade 2/5 to 4/5, and distal power increased from grade 3/5 to 4+/5 on the MRC scale. This was associated with a compensatory increase in the size of CMAPs, and a decrease in distal and F wave latencies to normal as serum potassium levels rose from 1.9 to 3.2 mmol/l (table 1). A repeat EMG of upper and lower limb muscles on day 3 showed a severely reduced interference pattern with motor units firing irregularly at 10–20 Hz at 1–2 mV. Occasional units were of spiky configuration but most were of normal form and amplitude.

The metabolic acidosis and polypnea persisted and potassium remained at 3–5 mmol/l, so parenteral bicarbonate was given, with 5% dextrose and spironolactone for secondary hyperaldosteronism. After three days she was given oral bicarbonate (3·6 g NaHCO₃ and 17·g KHO₃). This therapy was associated with a progressive improvement in her condition and seven days after admission her muscle power and respiratory function had fully recovered. Nerve conduction studies at this stage had also returned to normal. Her electrolytes and polypnea gradually improved and by discharge her potassium was 4·2 mmol/l, creatinine 176 μmol/l, and she was passing three litres of urine a day.

The most striking initial electrophysiological abnormality was a severe and generalised reduction in CMAP amplitudes; this, in association with mainly distal slowing of motor and sensory nerve conduction and some F wave latency prolongation, was compatible with, although not specific for, the acute phase of predominantly distal Guillain–Barre syndrome. The rapid recovery of CMAP amplitudes in association with the resolution of hypokalaemia and improved muscle power, however, suggests that the low potassium was responsible for the clinical and electrophysiological abnormalities. The recognised electrophysiological features of hypokalaemia, as seen in periodic paralysis, are related to muscle membrane inexcitability5 rather than nerve involvement.

Nevertheless, the exact pathophysiological mechanism of muscle weakness in these conditions is not well established; serum potassium concentration is not consistently related to the occurrence or degree of weakness, and electromyography late in the course of hypokalaemic periodic paralysis may show both neurogenic and myopathic features.1 In the present case, the abnormalities of motor conduction and electromyography could be explained by inexcitability of muscle fibres, especially those supplied by large, fast conducting myelinated nerve fibres. Although the hyperkalaemic effects may have contributed to the prolonged distal latencies in the initial intensive care unit study, cooling causes an increase in CMAP amplitude and therefore could not be responsible for the most prominent neurophysiological abnormality.1 Furthermore, conduction block in distal motor nerve fibres as part of Guillain–Barre syndrome would tend to decrease with cooling, rather than the reverse.

A further interesting feature of this case is the apparent differential response of skeletal and cardiac muscle to hypokalaemia, as the ECG hypokoncordsia in the horizontal position was normal. Hypokoncordsia in the horizontal and vertical planes were recognised in our patient as a surprising finding for the syndrome, were present without involvement, when present, is usually manifest as external ophthalmoplegia with slowed saccades.5

There have been a number of published reports of hypokalaemic weakness resembling Guillain–Barre syndrome. Causes have included the periodic paralyses, barium toxicity, renal tubular acidosis, and even ingestion,16 but none have documented serial electrophysiological studies. This case reinforces the need for awareness of the effects of electrolytes, in particular potassium, calcium and magnesium, in both the clinical and electrophysiological assessment of weak patients.

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References


Polynuropathy following parathion poisoning

Delayed neurotoxicity is a sequela of poisoning with certain organophosphorus compounds, and correlates with the irreversible inhibition of neuropathy target esterase (previously known as butyrylcholinesterase). This enzyme is widely distributed in the nervous system, but its physiological role is not yet known. In humans, the delayed axonal polyneuropathy occurs one to three weeks after intoxication with cholinergic signs are often present. The likelihood of organophosphorus compounds producing a delayed polyneuropathy is predicted by their ability to induce a similar syndrome in adult hens, and these compounds are called ‘neurotoxic’.1 Recent clinical reports, however, have shown that compounds not usually effective in producing delayed neurotoxicity in adult hens may do so in humans following massive exposure.13 Because the pathophysiology in these intoxications is not yet understood, we present the clinical features of an additional case of ethylparathion induced delayed polyneuropathy.

A 23 year old man suffered from depression since the age of 19. He had attempted suicide on three occasions. His admission followed the ingestion of 15 g of ethylparathion (E605 forte) and the infliction of a gunshot injury to his neck, penetrating the esophagus. Surgical treatment of this injury prevented gastric lavage. Ethylparathion serum levels were initially 400 ng/ml, increased to 550 ng/ml at day five and did not begin to decrease until day eight despite 10 haemoperfusions during this time.
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T T Warner, S Mossman and N M Murray

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