Indeed anticonvulsants are often not mentioned as part of the medical armamentarium in treating vertigo. In contrast to the situation with 8th nerve dysfunction, patients with 5th nerve dysfunction (tic douloureux) are usually offered intensive therapy with anticonvulsive medication prior to surgical considerations.

The response to phenytoin in this patient, while clinically gratifying, does not provide insight as to the pathogenesis of his vertigo. The situation is akin to that in trigeminal neuralgia where both peripheral and central mechanisms have been proposed. Indeed this disorder shares with trigeminal neuralgia features of a trigger, paroxysmal response, cranial nerve involvement, and response to phenytoin. This case demonstrates that similar trials of anticonvulsant therapy are indicated in patients with other irritative 5th nerve dysfunctions.

ROBERT SLATER
(University of Pennsylvania School of Medicine)
The Neuro-Otology Group,
Delaware County Medical Center,
Broomall, PA 19008, USA.

References


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Normal sural nerve morphometry in acute uraemia

Sir: We have reviewed the literature, and so far as we are aware, in only seven reported cases of acute uraemia were the sural nerves histologically studied. Of the five cases reported by Dinn and Crane, four had no clinical evidence of peripheral neuropathy, of whom two revealed segmental demyelination and remyelination in their sural nerves. The cases without histological evidence of neuropathy had uraemia of sudden onset and short duration. In a separate study published in the same year, myelin abnormalities were also observed in three cases, including one without clinical neuropathy. The latter case underwent biopsy 4 days after onset of uraemia and recovered completely in 2 weeks.

Recently, we had the opportunity to examine two acute uraemic cases, initially seen by nephrologists, as part of a longitudinal study for screening neuropathy in uraemia.

Case 1 was a 20 year old male who developed acute uraemia due to volume depletion after 4 days of high fever and several bouts of vomiting. The patient recovered completely after 10 days, and did not require dialysis. Sural nerve biopsy, after obtaining informed consent, was done on the second day of the uraemia state (fig). Case 2 was a 33 year old female who developed acute uraemia due to cardiogenic shock after suffering from acute myocardial infarction. She succumbed after 4 days from progressive increase in creatinine levels and anuria. The sural nerve was obtained during necropsy, 3 hours after death (fig). Both cases did not have clinical neuropathy, but, after systematic evaluation and grading, we found evidence of demyelination and remyelination in the sural nerve fibres.

Furthermore, nerve conduction studies of at least four nerves in the extremities did not reveal peripheral neuropathy. The sural nerves were fixed in 3% glutaraldehyde and osmicated. Morphometric studies included transverse fibre preparations of 100 myelinated fibre samples, internodal length and fibre diameter plotting, transverse electron microscopic studies of myelinated fibre populations. In all the methods employed no definite evidence of axonal degeneration or myelin abnormalities were obtained in either case.

It is of particular importance to make

Fig The clinical course of the two cases of acute uraemia.
special reference to some published histopathologic studies in chronic uraemia. The prevailing morphological basis of chronic uraemic neuropathy is known to be axonal degeneration with secondary segmental demyelination and myelin remodelling. A few studies have also shown that in late and severe cases of chronic uraemia, marked pathological changes can be mostly observed in the axon accompanied by some definite changes in the myelin, compared with the early and mild cases. Furthermore, even when some cases had no clinical neuropathy, mild histological evidence of neuropathy, (mostly myelin changes) can be found.

From these findings, we have formulated some important observations. In acute uraemia, especially in the very early stage, morphological changes in the sural nerves may not occur, while mild myelin abnormalities may be noted in the later stages. This proves to parallel the situation in chronic uraemia where duration and severity are found to be independent factors. It is believed, and with this we agree, that in acute uraemia, there may not be enough time for a specific “uraemic toxin” or the presence of a uraemic environment to cause measurable alterations in the clinical, pathological and nerve conduction studies.

While research is extensively pursued regarding the mechanisms involved in uraemic neuropathy, we think our data provide further proof of the need for recognition of duration and severity in the evolution of peripheral neuropathy.

RAYMOND I. ROSALES
JOSE C. NAVARRO
SHUJI IZUMO
MITSUHIRO OSAME
AKIHITO IGATA
OSCAR NAIDAS
LIBERTAD NAZARENO-ROSAS

Third Department of Internal Medicine
Kagoshima University, Japan

Department of Neurology and Psychiatry
and Department of Medicine
University of Sto. Tomas, Philippines


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Anterior tibial compartment syndrome secondary to systemic capillary leak syndrome

SIR: Systemic capillary leak syndrome is an uncommon disease characterised by recurrent bouts of generalised oedema and hypovolaemic shock without eosinophilia. The cause is unknown and the pathophysiology involves the rapid transfer of plasma to the extra-vascular space due to an increase in capillary permeability. We report here a case of this syndrome who developed a bilateral anterior tibial compartment syndrome and skeletal muscle necrosis.

A 37 year old man was admitted to the hospital because of hypovolaemic shock. Two days previously he began to complain of malaise, thirst and myalgia involving both legs, without previous strenuous exercise. On admission physical examination revealed generalised oedema and signs of shock. The haematocrit was 78% and the white blood cell count was 38 000/mm3 with 1% eosinophils. There was metabolic acidosis and hypoaalbuminaemia. Central venous pressure was –5 cm of water. Large volumes of parenteral fluid were administered and the shock improved within 24 hours. Thirty-six hours after admission, he experienced excruciating pain in both legs. On examination, both shins were swollen and tender with motor and sensory loss in the area of the peroneal nerves. The pulses were present. Creatine kinase was 4 859 IU/L (normal up to 85 IU/L), serum glutamic oxaloacetic transaminase was 166 IU/L (normal 7–20 IU/L) and lactic dehydrogenase 772 IU/L (normal 80–240 IU/L). Concentric needle electromyography of the tibialis anterior, peroneus longus and extensor digitorum brevis muscles of both legs showed abundant fibrillation and positive wave potentials. The voluntary pattern on maximal effort revealed a severe loss of motor units in all these muscles. Sensory action potentials of the peroneal nerves were unelicitable. Further examination some months later showed bilateral drop foot with atrophy of the tibialis anterior muscles, sensory loss in both peroneal nerves and the electromyogram detected signs of chronic denervation. The following studies were normal or negative: chest and abdominal radiography, abdominal ultrasonography, blood and urine cultures, echocardiography, Waaler-Rose, antinuclear antibodies, immunoglobulin and complement factors levels, T-3, T-4, TSH, ACTH, cortisol, catecholamines, urinary vanilmandelic acid and 5-hydroxy-indol acetic acid. After this episode, the patient suffered two more similar bouts but without skeletal muscle necrosis. A monoclonal IgG-lambda gammapathy was discovered during the recovery period of the third episode.

Our patient had the typical clinical picture of systemic capillary leak syndrome. Other