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Polyneuropathy in critically ill patients

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Record: 77 of 100
POLYNEUROPATHY IN CRITICALLY ILL PATIENTS¹

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A common cause of muscle weakness in the intensive care unit

More than 30 years ago, during a 4-year period, we observed five patients who presented in the ICU with unexplained difficulty in weaning from mechanical ventilation, and with limb weakness. At that time weaning difficulties had been attributed to diaphragmatic fatigue, and limb weakness to a catabolic myopathy. Clinical signs indicated a motor and sensory polyneuropathy, and electrophysiological tests, a primary axonal degeneration of motor and sensory fibres. Autopsy studies of both the central and peripheral nervous systems in three patients confirmed the electrophysiological findings. The polyneuropathy improved substantially in the two remaining patients.

The cause was uncertain. The onset after ICU admission, the unremarkable CSF examination and the presence of an axonal rather than demyelinating neuropathy, made Guillain-Barré syndrome unlikely. Blood tests were negative for standard causes of axonal neuropathy. We speculated critical illness itself might be the cause. At that time critical illness had been further defined as sepsis and multiple organ failure. Disturbance of the microcirculation, as occurs in severe sepsis, could cause nerve hypoxia and distal axonal degeneration.

Comprehensive retrospective and prospective studies in ours and other centres indicated the polyneuropathy

occurred in up to 70% of critically ill patients.² Phrenic nerve conduction and needle electromyography of the diaphragm and autopsy studies revealed that the cause of the difficulty in weaning was axonal degeneration of the phrenic and chest wall nerves.

At the same time we were observing the polyneuropathy, others were observing a myopathy, most commonly designated acute quadriplegic myopathy. Like the neuropathy, it was potentially reversible. Our electrophysiological and morphological studies had suggested muscle was involved, as well as nerve. We called it 'septic myopathy'. Subsequent studies have shown muscle is commonly involved in critically ill patients, at times involving muscle only, with varying morphological changes of myosin deficiency and necrosis. The entity of critical illness myopathy has been defined by Lacomis and colleagues.³

In addition to the polyneuropathy, all of our patients had early evidence of septic encephalopathy. It consisted of varying degrees of depressed consciousness, generalised EEG abnormalities but unremarkable CT head scans and CSF examinations.²

While the encephalopathy, polyneuropathy and myopathy tend to improve after the critical illness has been brought under control, long term follow-up has shown residual effects causing varying degrees of mental and neuromuscular disability.

Intensivists are now instituting a 'least sedation method'.⁴ The mental status and muscle strength can be tested at the time of periodic withdrawal of sedative drugs. This allows better monitoring of sedation

and, if weakness is demonstrated, electrophysiological and muscle biopsy studies. The nature of neuromuscular disorder having been identified, effective rehabilitation and attempts at long term prognosis are then possible.

There have been interesting investigations of the pathophysiology.² The nerve membranes demonstrate abnormalities of excitability. Muscle membranes are inexcitable on direct stimulation. The prolonged duration of the compound muscle action potential, now known to be typical of critical illness myopathy, is due to decreased muscle membrane conduction velocity. Dysfunction of sodium channels, as originally suggested by Rich *et al*, may explain these phenomena.⁵

Further basic studies are needed, as are efforts to utilise clinical, electrophysiological and muscle biopsy methods in the ICU to detect these nervous system effects and institute mental and physical rehabilitation. The greatest hope is for the discovery of a 'magic bullet' to interrupt the septic cascade and prevent these devastating nervous system effects.

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