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LETTER TO THE EDITORS

Critical illness polyneuropathy after septic peritonitis in a boy with nephrotic syndrome

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Sirs,

Critical illness polyneuropathy (CIP) is a sensorimotor axonal polyneuropathy recognized as a complication of sepsis and multiple organ system failure, such as systemic inflammatory response syndrome (SIRS), in critically ill adults [1]. Clinically, CIP is characterized by a rapidly developing flaccid tetraparesis with reduced deep tendon reflexes and failure to wean from mechanical ventilation [2, 3]. We report here a child who developed CIP after receiving intensive care for peritonitis and acute renal failure that was complicated by nephrotic syndrome.

A 1-year-old boy was admitted to hospital because of new-onset nephrotic syndrome and treated with prednisolone (2 mg/kg/day). On day 6, he developed pneumococcal peritonitis and oliguric acute renal failure. He was treated with antibiotics and intravenous immunoglobulin 200 mg/kg/day, but his general condition deteriorated. He required continuous hemodiafiltration for 4 days and ventilator support for 5 days. In total, he received neuromuscular blocking agents for 5 days and ampicillin for 13 days. His condition and renal function improved within 2 weeks, but he was still in bad humor, and a flaccid paralysis predominating at the distal parts of the lower limbs was observed. The deep tendon reflexes had disappeared, but central nervous system dysfunction was absent. Results of blood tests, brain magnetic resonance imaging, and electroencephalography were also normal. Electrophysiological tests on day 24 revealed reductions in the compound muscle action potential (CMAP) amplitudes, while measurements of the motor and sensory conduction velocities and distal latencies were normal. These findings suggested primary degeneration of the axons supplying the limbs. A physical rehabilitation program was started, with the result that the patient was able to stand at 7 weeks, walk at 9 weeks, and run at 4 months. Electrophysiological tests performed on days 47 and 78 revealed improvement in the CMAP amplitudes (Fig. 1). After a 4-week course of oral prednisolone, partial remission of the nephrotic syndrome had been achieved. A renal biopsy showed focal segmental glomerulosclerosis (FSGS) for which he received cyclosporine in combination with prednisolone. This treatment regimen ultimately resulted in remission.

The current diagnostic criteria for CIP require that both the patient's history and electrophysiological tests show evidence of an axonal motor and sensory polyneuropathy. In CIP, the CMAP and sensory action nerve potential amplitudes are reduced, with minor changes in the conduction velocities and latencies, consistent with axonal degeneration of the peripheral motor and sensory nerve fibers. CIP generally shows spontaneous improvement within weeks to months, but recovery may be limited when the neuropathy is severe. No specific therapies are available. Hypoalbuminemia and the use of neuromuscular blocking agents and steroids are reported to be risk factors for the development of CIP [2, 3]. Our patient's condition was complicated with severe hypoalbuminemia due to FSGS, which may have caused endneural edema and be associated with the development of CIP. Strict glycemic control and intravenous immunoglobulin therapy may significantly reduce the incidence of CIP in adults [3, 4]. Our patient received intravenous immunoglobulin and

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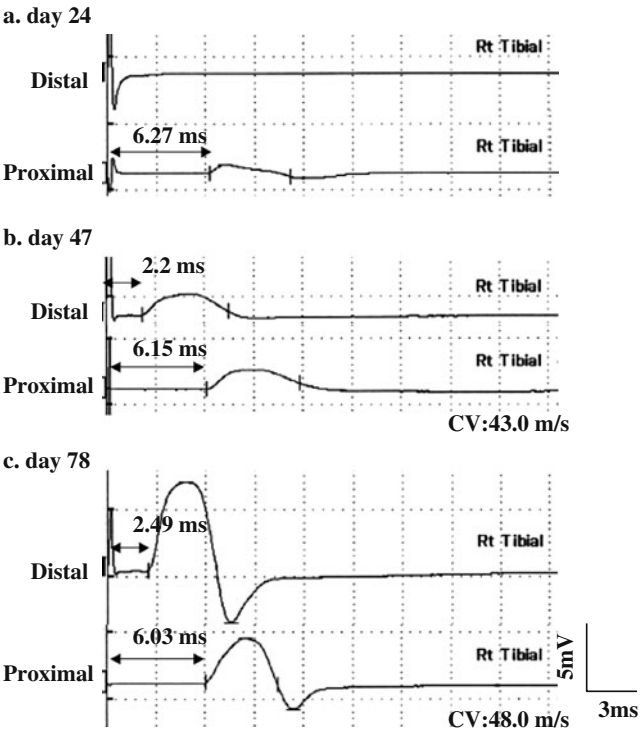

Fig. 1 Nerve conduction studies of the right tibial nerve on days 24, 47, and 78. Marked reductions in the compound muscle action potentials (CMAPs) are evident on days 24 and 47, with continuing improvement on day 78. The distal and proximal latencies (*double arrows*) remain almost unchanged within normal ranges. The nerve conduction velocities (*CV*) on days 47 and 78 are normal

albumin during the early stage, and his serum glucose levels were maintained within the normal range. Although we were unable to prevent the development of CIP, our patient fully recovered once provided early rehabilitation.

In conclusion, a precise diagnosis of CIP can be difficult in children, with the result that the condition may be missed in many patients. Careful neurological examination and early electrophysiological testing are helpful in establishing the proper diagnosis and estimating the neurological prognosis. Early diagnosis allows some patients to initiate early rehabilitation and early treatment, which may prevent disuse muscle atrophy and enable complete recovery.

References

1. Bolton CF, Gilbert JJ, Hahn AF, Sibbald WJ (1984) Polyneuropathy in critically ill patients. J Neurol Neurosurg Psychiatry 47:1223–1231
2. Witt NJ, Zochodne DW, Bolton CF, Grand'Maison F, Wells G, Young GB, Sibbald WJ (1991) Peripheral nerve function in sepsis and multiple organ failure. Chest 99:176–184
3. Van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R (2001) Intensive insulin therapy in critically ill patients. N Engl J Med 345:1359–1367
4. Mohr M, Englisch L, Roth A, Burchardi H, Zielmann S (1997) Effects of early treatment with immunoglobulin on critical illness polyneuropathy following multiple organ failure and gram-negative sepsis. Intensive Care Med 23:1144–1149