Rehabilitation Practice and Science

Volume 39
Issue 4 *Taiwan Journal of Physical Medicine and Rehabilitation (TJPMR)*

Article 6

12-31-2011

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Recommended Citation

Li, Chia-Chen; Hsieh, Shih-Fu; Lin, Ming-Chuan; Huang, Chung-Wei; and Horng, Yi-Shiung (2011) "Tetraplegia Caused by Critical Illness Polyneuropathy Superimposed on Diabetic Neuropathy: A casereport," *Rehabilitation Practice and Science*: Vol. 39: Iss. 4, Article 6.
DOI: https://doi.org/10.6315/2011.39(4)06
Available at: https://rps.researchcommons.org/journal/vol39/iss4/6

Case Report

Tetraplegia Caused by Critical Illness Polyneuropathy Superimposed on Diabetic Neuropathy: A Case Report

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Objective: To describe a patient with the critical illness polyneuropathy superimposed on diabetic neuropathy and to highlight the importance of early rehabilitation after critical illness. Methods: A 42-year-old man with a previous history of diabetic polyneuropathy developed flaccid tetraplegia with respiratory failure after septic shock due to a liver abscess. This clinical manifestation, combined with the result of an electrodiagnostic study, suggested the acute exacerbation of polyneuropathy. Because a pre-existing polyneuropathy history complicated a differential diagnosis, we performed a comprehensive work-up to rule out other systemic diseases or metabolic disorders that may cause polyneuropathy. Finally, the diagnosis of critical illness polyneuropathy superimposed on diabetic neuropathy was established. Result: After 6 months of rehabilitation, the patient was able to ambulate with a walker under supervision and perform the activities of daily living with partial assistance. However, he still had muscle weakness, sensory impairment, autonomic dysfunction, and chronic disabilities. Conclusion: Critical illness polyneuropathy, resulting in limb weakness and difficulty in weaning from a ventilator, commonly occurs in critically ill patients and may cause severe disability. Early diagnosis and prevention of this condition are important to reduce the incidence and cost of hospital stays. Early rehabilitation can be a safe and feasible intervention and has the potential to improve clinical outcomes of critical illness. (Tw J Phys Med Rehabil 2011; 39(4): 239 - 246)

Key Words: critical illness, polyneuropathy

INTRODUCTION

Critical illness polyneuropathy (CIP) and critical illness myopathy (CIM), alone or in combination, occur commonly in critically ill patients who have been on mechanical ventilation for more than one week.[1] CIP is an axonal polyneuropathy that affects both sensory and motor nerves. CIM describes a spectrum of muscle pathologies seen in critically ill patients. Due to the frequent coexistence of CIP and CIM and the difficulty in discrimination between CIP and CIM, the umbrella descriptive term “critical illness polyneuromyopathy” (CIPNM) is used.[2] It manifests with muscle weakness in the limbs and respiratory muscles and is associated with higher mortality, prolonged duration of mechanical ventilation, and increased length of hospital stay.[3] However, this neuromuscular impairment might be missed during

Submitted date: 22 February 2011 Revised date: 10 June 2011 Accepted date: 28 June 2011

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the acute phase of critical illness and taken simply as the result of prolonged immobilization.[4]

Moreover, it is difficult to diagnose CIP while there is a pre-existing polyneuropathy because of extended differential diagnosis. There are currently few reports that describe how to approach patients in this condition.[5-7] Here, we describe the case of a 42-year-old man with a previous history of diabetic polyneuropathy who developed flaccid tetraplegia with respiratory failure after septic shock due to a liver abscess. On the basis of his clinical course and the results of an electrophysiological study, the diagnosis of CIP superimposed on diabetic neuropathy was established. After 6 months of rehabilitation, the patient was able to achieve significant functional gain in the activities of daily living and ambulation. We discuss the differential diagnosis in this situation to remind physicians of the importance of an early diagnosis. Further, we address the outcomes of patients with CIP. Finally, we review the literature regarding the prevention and treatment and highlight the potential benefits of early rehabilitation.

CASE REPORT

A 42-year-old man had a previous medical history of type 2 diabetes mellitus without regular medical control for many years. In 2001, he had developed progressive dysesthesia in a stocking-glove distribution, which was followed by weakness in the bilateral distal lower limbs in 2004. Symptoms of autonomic dysfunction, such as impotence, postural hypotension, constipation, and diarrhea were also noted. A nerve conduction study (NCS) performed in October 2006 revealed sensori-motor polyneuropathy with axonal denervation and demyelination (Table 1, 2). Based on his clinical features, his long-standing diabetes, and the results of the electrophysiologic study, a diagnosis of diabetic polyneuropathy with autonomic neuropathy was made.

In January 2007, the patient was admitted to the intensive care unit (ICU) with intubation due to liver abscess with septic shock and the presentation of high fever, respiratory distress, and consciousness disturbance. The patient was intubated and received antibiotic therapy. He underwent computed tomography (CT) guided aspiration and drainage of the abscess on the 5th day of hospitalization. The fever subsided and extubation was performed on the next day. His consciousness also returned. However, he was unable to move his four limbs and trunk. Neurological examination revealed the absence of a deep tendon reflex and decreased sensation in his four limbs and middle-lower trunk. One week later, the muscle power of his bilateral upper limbs improved to a medical research council (MRC) score of 3/5, while paralysis of his bilateral lower limbs remained. During this period of time, the use of aminoglycoside for six days was reported, but not the use of neuromuscular blocking agents or corticosteroids. Magnetic resonance imaging (MRI) of the brain and the low cervical and thoracic spine revealed no significant abnormalities. Creatine phosphokinase (CPK) level was within normal range. A NCS demonstrated prolonged distal latency, slowness of motor nerve conduction velocity (NCV), absent or markedly reduced amplitude of compound muscle action potentials (CMAPs), and absent sensory nerve action potentials (SNAPs) in both his upper and lower extremities (Table 1, 2). These findings were consistent with the pattern of CIP.

Because a pre-existing polyneuropathic history complicated a differential diagnosis, we performed a comprehensive work-up to rule out other systemic diseases or metabolic disorders that may cause polyneuropathy. First, we reviewed his history; there was no history of toxin exposure or systemic diseases other than diabetes mellitus. Second, we checked basic screening tests including the complete blood count, C-reactive protein (CRP), fasting blood glucose, renal function, liver function, and urinalysis. The results showed microcystic anemia, hyperglycemia, glucosuria, proteinuria associated with diabetes mellitus, and elevated CRP associated with liver abscess. Negative findings for antinuclear antibody (ANA), anti-Ro, anti-La, antineutrophil cytoplasmic antibody (ANCA), rheumatoid factor, serum protein electrophoresis, and cryoglobulins excluded autoimmune disease. The results of chest X-ray, abdominal CT, and tumor markers (including alpha-fetoprotein (AFP), cancer antigen-125 (CA-125), cancer antigen 19-9 (CA 19-9) showed no evidence of malignant disorders. Hepatitis C virus antibody and human immunodeficiency virus (HIV) tests were negative. After excluding polyneuropathy secondary to other systemic or metabolic diseases, the diagnosis of CIP superimposed on diabetic neuropathy was established. Additionally, urinary retention was noted, and a videourodynamic study re-

Table 1. Summary of three sequential motor nerve conduction studies

Nerve	Test date	Distal latency (ms)	Amplitude (mV) distal	Amplitude (mV) proximal	NCV* (m/s)	F latency† (ms)
		right/left	right/left	right/left	right/left	right/left
Median	Oct 2006	6.5/5.8	3.4/6.0	2.9/5.6	35/39	38.6/37.9
	Feb 2007	6.5/5.8	3.8/5.4	3.4/4.8	41/42	NR‡/37.7
	Apr 2007	6.1/5.4	4.2/7.9	3.7/6.9	39/40	42.2/37.7
Ulnar	Oct 2006	4.5/3.8	5.2/4.6	5.2/3.9	30/40	37.1/39.4
	Feb 2007	4.4/4.6	4.2/3.0	3.8/2.5	38/37	NR/44.1
	Apr 2007	4.9/4.4	3.5/2.6	3.4/2.2	35/36	43.4/42.7
Peroneal	Oct 2006	NR/NR	NR/NR	NR/NR	NR/NR	NR/NR
	Feb 2007	NR/NR	NR/NR	NR/NR	NR/NR	NR/NR
	Apr 2007	NR/NR	NR/NR	NR/NR	NR/NR	NR/NR
Tibial	Oct 2006	5.5/5.2	1.2/2.4	1.0/1.1	31/29	NR/NR
	Feb 2007	9.0/7.8	1.2/2.0	0.6/1.7	28/31	63.2/NR
	Apr 2007	5.1/4.2	0.7/1.7	0.5/1.4	26/32	80.4/NR

*NCV = nerve conduction velocity; †F latency = minimal latency of F waves; ‡NR = no response.

Table 2. Summary of three sequential sensory nerve conduction studies

Nerve	Test date	Amplitude (μV)	NCV* (m/s)
		right/left	right/left
Medial	Oct 2006	NR†/NR	NR/NR
	Feb 2007	NR/NR	NR/NR
	Apr 2007	NR/NR	NR/NR
Ulnar	Oct 2006	6.0/4.0	25/26
	Feb 2007	NR/NR	NR/NR
	Apr 2007	4.0/3.0	38/42
Sural	Oct 2006	NR/5.0	NR/29
	Feb 2007	NR/NR	NR/NR
	Apr 2007	NR/NR	NR/NR

*NCV = nerve conduction velocity; †NR = no response.

vealed underactivity of detrusor function. A sympathetic skin response study revealed no response in either upper or lower extremities, indicating autonomic dysfunction.

At the beginning, the patient presented with severe muscle weakness, predominantly in the lower limbs, and was completely dependent on others for functional mobility and activities of daily living. On the 14th day of hospitalization, he began to participate in a bedside rehabilitation program comprising physical and occupational therapy, starting with active-assisted exercises, bed mobility, and then sitting over the edge of the bed.

Two months later, the muscle strength of his bilateral lower limbs improved to a MRC score of 2-3/5. Although a follow-up NCS showed no obvious improvement compared to the previous study (Table 1, 2), electromyography (EMG) in bilateral vastus medialis, tibialis anterialis, and gastrocnemius muscles revealed positive sharp waves, polyphasic waves with decreased amplitude, and reduced recruitment in motor unit action potentials (MUAPs), indicating early re-innervation. The rehabilitation program proceeded to sitting balance training, using cycling pedals, standing practice, bed-to-chair transfer

and then walking with a walker step by step. After 6 months of rehabilitation, the muscle power of the upper limbs improved to a MRC score of 4-5/5, and the muscle power of the lower limbs improved to a MRC score of 3-4/5, allowing the patient to ambulate with a walker under supervision and achieve partial independence in daily activities. However, the patient continued to experience muscle weakness, sensory impairment, erectile dysfunction, urinary retention, diarrhea and constipation.

DISCUSSION

CIP is the most common neuromuscular alteration seen in the intensive care unit.[1] Its incidence in patients with sepsis and systemic inflammatory response syndrome (SIRS) is 68-100%.[8] The major risk factors include the duration of ICU stay, mechanical ventilator usage, persistent sepsis and SIRS or multiple organ failure, hyperglycemia, and the use of corticosteroids or neuromuscular blockers.[3,5,9] Aminoglycosides have received particular attention as some studies associated their use with CIP.[10-12] However, other authors could not confirm these findings.[13-15] The clinical features of CIP are difficulty in weaning from the ventilator, distally predominant muscle weakness (especially in lower extremities), muscle atrophy, and hypo- or areflexia. Facial weakness or other cranial nerve involvement is rare. Sensory loss can be present but is often difficult to test in sedated or intubated patients in the ICU. This patient, with the risk factors of mechanical ventilation, persistent sepsis and SIRS, hyperglycemia, and the use of aminoglycoside, presented weakness in four limbs after a critical illness. CIP was highly suspected.

To make a diagnosis of CIP, it is necessary to perform a systemic review and rule out other possible etiologies. We developed a practical algorithm for a clinical approach to patients developing acute tetraplegia after ICU admission (Figure 1). The approach that was used to manage our case is as follows: First, due to the presentation of consciousness disturbance and tetraplegia, we arranged brain and spine MRI to exclude central nervous system (CNS) lesion. Next, due to the unremarkable findings of the neuroimaging studies, we performed an electrodiagnostic study to investigate the peripheral nerves and muscles. The NCS revealed a marked reduction of both CMAPs and SNAPs in four limbs, which indicated a progression of sensori-motor axonal polyneuropathy after his critical illness. However, the clinical course could not be simply explained by an acute deterioration of diabetic symmetric distal polyneuropathy, as it usually progresses slowly and irreversibly.[16] The acute exacerbation of symptoms and gradual recovery were suggestive of new-onset, reversible lesions superimposed on diabetic neuropathy that developed during critical illness in this patient. CIP, often characterized by mixed sensori-motor axonal degeneration,[1,6,8,9,17-19] was the most likely etiology.

Further, a comprehensive work-up to exclude other etiologies of axonal polyneuropathy was done. According to his clinical history and laboratory results, this patient had a long-standing history of diabetes mellitus but no evidence of other systemic diseases and metabolic disorders, such as nutrition deficiencies, chronic renal failure, endocrine diseases, connective tissue diseases, or exogenous intoxication. Finally, we established the diagnosis as CIP superimposed on diabetic neuropathy based on his clinical presentation and the results of the electrophysiological study.

Additionally, CIM may develop during critical illness. Needle electromyography studies are helpful for diagnosing CIM. The typical features include short-duration and low-amplitude motor unit action potentials and early recruitment during volitional contraction.[1,6-8,17,18] However, this patient was not submitted to electromyography during the acute weakness stage due to noncooperation. The electromyography study performed two months later did not reveal this characteristic pattern of myopathy. Further, a definitive diagnosis of muscle involvement requires histological confirmation from a muscle biopsy.[1,17-22] We did not perform a muscle biopsy because it was not necessary for clinical decision-making in the presence of symptomatic improvement. Due to this lack of sufficient evidence, we could not conclude whether CIM coexisted with CIP in this patient.

Almost all cases of CIP show improvement over time. The recovery is usually more rapid and complete for the upper limbs and proximal lower limbs, followed by recovery of the respiratory system and finally of the distal lower limbs.[23] However, chronic disability is still a common finding. A systemic review[24] reported that 180

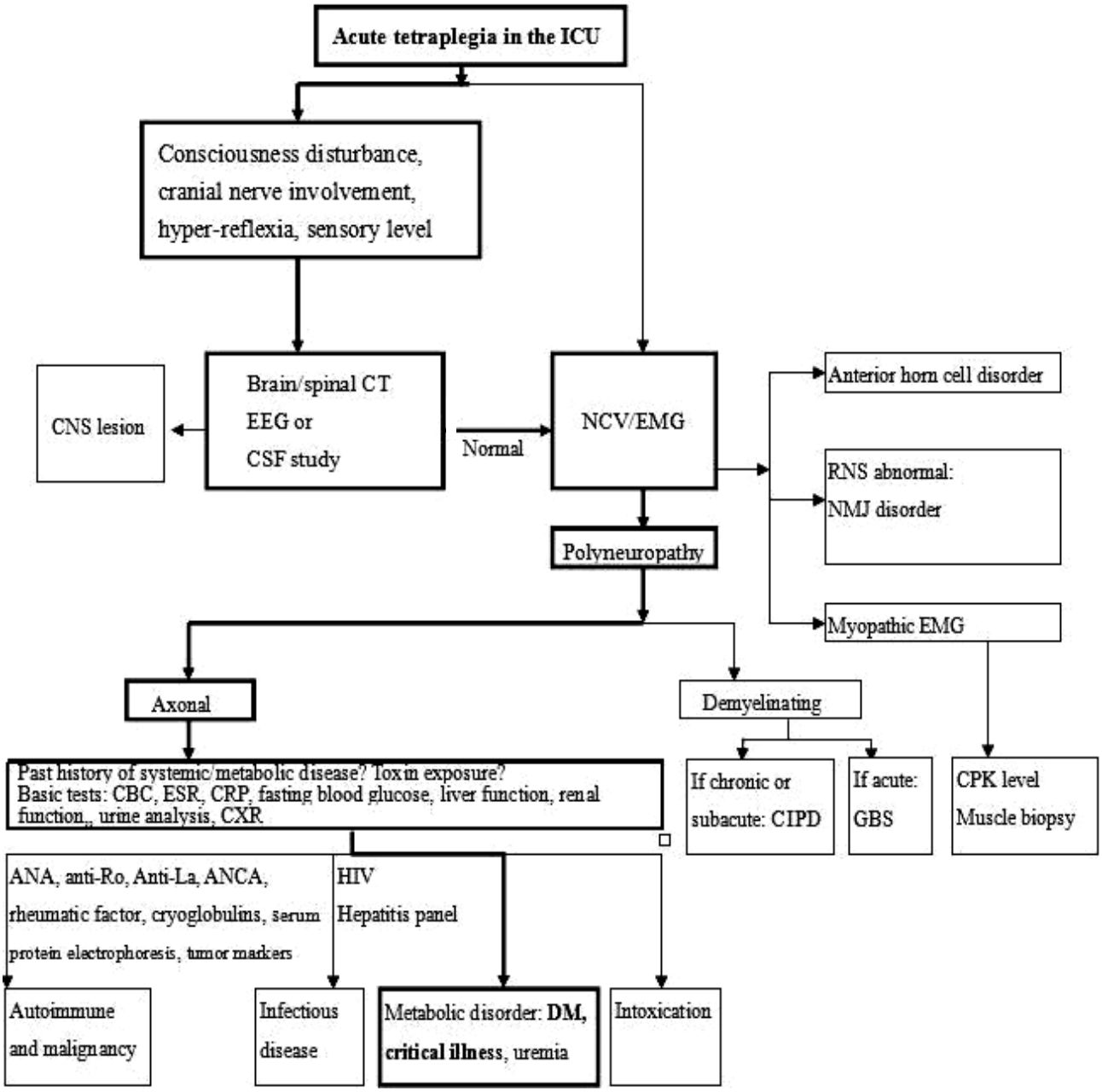

Figure 1. Algorithm for the clinical approach to acute tetraplegia in the ICU. CPK = creatine phosphokinase; CNS = central nerve system; CT = computed tomography; CSF = cerebrospinal fluid; EEG = electroencephalograph; NCV = nerve conduction velocity; EMG = electromyography; RNS = repetitive nerve stimulation; NMJ = neuromuscular junction; CIDP = chronic inflammatory demyelinating polyneuropathy; CIP = critical illness polyneuropathy; GBS = Guillain Barré syndrome; DM = diabetic mellitus; AIDS = acquired immune deficiency syndrome; CBC = complete blood count; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein; ANA = antinuclear antigen; ANCA = antineutrophil cytoplasmic antibody; CXR = chest X-ray.

of 263 patients (68.4%) had complete functional recovery, regaining the ability to breathe spontaneously and walk independently; 74 patients (28.1%) had severe disability with tetraparesis, tetraplegia, or paraplegia. Persistent milder impairments were common even in patients with complete functional recovery, including reduced or absent deep tendon reflex, stocking and glove sensory loss, muscle atrophy, painful paresthesia, and foot drop. ICU patients with CIP treated in a neuro-rehabilitation setting resulted in a good functional outcome.[25] This patient underwent rehabilitation treatment for 6 months had a great functional recovery, which should be much better than that of the nature course. However, the patient still had muscle weakness, sensory impairment, and autonomic dysfunction. The persistent deficits in motor, sensory, and autonomic function were partially attributed to the irreversible neuropathy caused by diabetes mellitus.

At present, there are few options for the prevention and/or treatment of CIP. Thus, the knowledge of risk factors and the implementation of preventative strategies, including strict control of blood glucose, limits on the use of neuromyopathic drugs, mobilization of patients, and prevention of sepsis and SIRS, are crucial in the care of high-risk patients.[3,26] Substantial evidence shows that intensive insulin therapy reduces the incidence of CIP, the duration of mechanical ventilation, the duration of ICU stay, and 180-day mortality.[27] However, intensive insulin therapy is significantly associated with an increase in hypoglycemia. Controversies exist regarding the risks and benefits of strict glycemic control in critically ill patients. However, limited evidence shows no significant effect of corticosteroids on the incidence of CIP.

Recent studies have demonstrated improved clinical outcomes with early mobility in the ICU. An uncontrolled study examined routine, multidisciplinary rehabilitation therapy provided twice daily to 103 patients with mechanical ventilation in the ICU.[28] This research demonstrated that early activities, included sitting and ambulation, are feasible and safe in patients with respiratory failure. Moreover, this program led to 69% of patients ambulating more than 100 ft (30 m) by ICU discharge. A subsequent larger study that compared an early mobility group to a control group receiving typical care showed that early mobility patients were out of bed earlier with a shorter length of stay in the ICU and in the hospital.[29] More recently, a randomized controlled trial assessed the efficacy of combining the daily interruption of sedation with physical and occupational therapy on the functional outcomes in patients receiving mechanical ventilation in the ICU.[30] This intervention resulted in better functional outcomes upon discharge from the hospital, a shorter duration of delirium, and more ventilator-free days compared with standard care. However, the positive results of early rehabilitation require further confirmation by larger multicenter trials. Further evaluation of long-term benefits is needed for the implementation of this strategy in critical care.

CONCLUSION

CIP resulting in limb weakness and difficulty in weaning from the ventilator commonly occurs in critically ill patients and may cause severe disability. Early diagnosis and prevention of this condition are important to reduce the incidence and the cost of a hospital stay. Early rehabilitation can be a safe and feasible intervention, with the potential to improve clinical outcomes of critical illness.

ACKNOWLEDGMENT

This study was supported by a grant from Tzu Chi General Hospital, Taipei Branch (TCRD-TPE-99-25).

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重症多發性神經病變合併糖尿病神經病變導致四肢癱瘓：病例報告

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目的：報告一個重症多發性神經病變(critical illness polyneuropathy)合併糖尿病神經病變(diabetic neuropathy)的病例，並且強調急性急症後早期活動(early mobility)的重要性。方法：一位四十二歲男性有糖尿病多發性神經病變病史，因肝膿瘍引起敗血性休克後導致弛緩性四肢癱瘓合併呼吸衰竭，臨床表現及電學檢查結果疑似多發性神經病變急性惡化。因先前的多發性神經病變病史使得鑑別診斷更加複雜，因此我們安排一系列完整檢查來排除其他可能造成多發性神經病變的系統性或代謝性疾病，最後確診為重症多發性神經病變合併糖尿病神經病變。結果：經過六個月的復健，病人可以在監督下使用助行器行走，並且在部分協助下執行日常生活活動。但是他仍然表現肌肉無力，感覺異常，自主系統失調以及慢性失能。結論：重症的病人常發生重症多發性神經病變造成肢體無力，脫離呼吸器有所困難，並導致嚴重失能。早期診斷及預防對於減少此疾病發生率及住院花費相當重要。早期復健是安全而可行的介入方法，且可能改善急性急症臨床預後。（台灣復健醫誌 2011；39(4)：239 - 246）

關鍵詞：重症(critical illness)，多發性神經病變(polyneuropathy)

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