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OPTIMIZATION OF REHABILITATION OF PARESIS IN CRITICAL CONDITIONS

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Abstract: Guillain-Barre (gee-YAH-buh-RAY) syndrome is a condition in which the body's immune system attacks the nerves. It can cause weakness, numbness or paralysis. Weakness and tingling in the hands and feet are usually the first symptoms. These sensations can quickly spread and may lead to paralysis. In its most serious form, Guillain-Barre syndrome is a medical emergency. Most people with the condition need treatment in a hospital. Guillain-Barre syndrome is rare, and the exact cause is not known. But two-thirds of people have symptoms of an infection in the six weeks before Guillain-Barre symptoms begin. Infections can include a respiratory or a gastrointestinal infection, including COVID-19. Guillain-Barre also can be caused by the Zika virus. There's no known cure for Guillain-Barre syndrome. Several treatment options can ease symptoms and help speed recovery. Most people recover completely from Guillain-Barre syndrome, but some serious illnesses can be fatal. While recovery may take up to several years, most people are able to walk again six months after symptoms first began. Some people may have lasting effects, such as weakness, numbness or fatigue.

Key words: immune attack, paralysis, polyneuromyopathy, sepsis, neuromuscular blockade, rehabilitation

Critical illness polyneuromyopathy (CIPNM) is a syndrome of neuromuscular disorders in patients admitted to intensive care units (ICU), manifested by myopathy and/or progressive sensorimotor neuropathy. It is associated with an increased risk of early mortality. CIPNM typically develops during prolonged respiratory support following respiratory failure in patients with sepsis, multiple organ failure, acute respiratory distress syndrome (ARDS), after cardiac or genitourinary surgeries, and as a complication of prolonged bedrest. It arises due to metabolic disorders, acid-base imbalances, nutrient deficiencies, and the pathological effects of certain medications.

CIM (critical illness myopathy) and CIP (critical illness polyneuropathy) often co-occur in ICU patients. The condition has been defined in various ways: ICU-acquired weakness (ICUAW), ICU-acquired paresis (ICUAP), critical illness myoneuropathy (CRIMYNE), or simply critical illness myopathy/polyneuropathy (CIP/CIM).

The classification of neuromuscular disorders encountered in ICUs includes CIP, prolonged neuromuscular blockade, and CIM (such as acute necrotizing myopathy, cachectic myopathy, rhabdomyolysis).

Approximately 30% of patients requiring respiratory support develop neuromuscular disorders within 7 days of ICU stay. In 60% of patients with sepsis or systemic inflammatory response syndrome (SIRS), CIPNM is detected, and this rate reaches up to 100% in patients with multiple organ failure. CIPNM prolongs ICU stays, increases treatment costs, negatively impacts clinical outcomes—including incomplete recovery due to persistent muscle weakness—and contributes to increased mortality.

Risk factors for CIPNM include old age, female sex, treatment with corticosteroids and aminoglycosides, hypoxia, hypotension, hyperthermia, hyperglycemia, and hypoalbuminemia. Sepsis and SIRS disrupt microcirculation, including within peripheral nerve axons. The release of inflammatory mediators increases capillary permeability, allowing harmful substances (e.g., neuromuscular blockers, corticosteroids, certain antibiotics) to enter and damage neurons.

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The exact pathogenesis of CIPNM in multiple organ failure remains unclear. Myopathy involves accelerated apoptosis, microvascular damage leading to leukocyte activation and cytokine release, disrupted calcium homeostasis, increased proteolysis, and impaired antioxidant defense mechanisms.

Clinical manifestations include weakness of limb and respiratory muscles, decreased deep tendon reflexes, sensory deficits, and pelvic organ dysfunction. Examination includes assessment of speech and swallowing due to bulbar and facial muscle weakness, which can contribute to dysarthria or dysphagia. Despite preserved central respiratory drive, patients often develop hypoxia upon weaning from mechanical ventilation due to peripheral respiratory neuropathy, leading to prolonged ventilator dependency.

Bilateral diaphragmatic paralysis and facial nerve involvement are rare but reported. CIPNM significantly increases ICU mortality due to difficulty in achieving spontaneous respiration. In pediatrics, CIPNM is rare (1.7%), due to lower rates of SIRS in children, and is multifactorial in origin. Swallowing disorders, often transient and resolving within 4 weeks, occur mostly in elderly patients with pre-existing pulmonary disease or tracheostomy.

There is no specific pharmacological treatment for CIPNM. Preventive strategies include adequate nutrition, glycemic control, cautious use of corticosteroids, neuromuscular blockers, and certain antibiotics. Management of sepsis, SIRS, and multiple organ failure is essential. Monitoring of the patient's condition is crucial. Blood pressure control and deep vein thrombosis prophylaxis should be prioritized in immobilized patients. Respiratory physiotherapy is necessary to prevent nosocomial pneumonia. To prevent pressure ulcers, regular repositioning and the use of anti-bedsore mattresses are recommended. Early correction of nutrient deficiencies and initiation of enteral nutrition are key to preventing intestinal dysfunction. Fluid and electrolyte balance, along with the use of diuretics, may prevent renal failure.

Tight glucose control is necessary, as hyperglycemia prolongs the course of CIPNM. Intensive insulin therapy (versus standard therapy) reduces ventilator dependency, ICU stay, and 180-day mortality, although it carries a risk of hypoglycemia. Maintaining blood glucose at 80–110 mg/dL significantly lowers CIPNM incidence compared to levels of 180–200 mg/dL. Insulin exerts anti-inflammatory effects by reducing integrins (cell surface molecules), E-selectins, and nitric oxide levels, protecting endothelial function.

Use of testosterone derivatives and growth hormone has shown no benefit. Experimental studies suggest lacosamide may be effective in treating CIPNM due to its anti-inflammatory and antioxidant properties.

Immunological therapies—including monoclonal/polyclonal antibodies, TNF-alpha inhibitors, IL-1 receptor antagonists, N-acetylcysteine, and plasma exchange—have shown efficacy in patients with sepsis and SIRS. Immunoglobulin G may prevent or reduce CIPNM incidence, especially in gram-negative sepsis, while immunoglobulin M has shown no benefit.

In sepsis-related thrombosis, recombinant activated protein C can reduce CIPNM incidence and ICU mortality. There are no preferred formulations for parenteral nutrition, and studies have not shown a clear link between enteral/parenteral feeding regimens and CIPNM development.

CIPNM adversely affects activities of daily living and quality of life, highlighting the need for early rehabilitation.

Rehabilitation should begin early in ICU with low-intensity therapeutic exercises to preserve muscle strength and joint mobility, and prevent contractures. As patients improve, progressive

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strengthening exercises for upper and lower limbs are introduced. Rehabilitation may be long-term and include mechanotherapy. Physical therapy is essential for improving mobility and adapting to daily physical demands.

A multidisciplinary approach involving neurologists, neuropsychologists, and rehabilitation physicians is critical. Rehabilitation improves quality of life and reduces treatment costs by addressing neuromuscular weakness.

Continuity of care is essential—rehabilitation should continue in outpatient settings. Rehabilitation planning depends on neurological symptoms, disease pathogenesis, rate of functional recovery, and level of social support.

In conclusion, an interdisciplinary approach is necessary for effective treatment of CIPNM to optimize social and professional reintegration and reduce disability.

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