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CRITICAL ILLNESS POLYNEUROMYOPATHY

Abdukadirova D. T. Abdulhamidov M.A

Andijan State Medical Institute

Department of Neurology

Critical illness polyneuromyopathy (CIPNM) develops during prolonged respiratory support after the onset of respiratory failure in patients with sepsis, multiple organ failure, acute distress syndrome, after cardiac or genitourinary surgeries, as a complication of prolonged bed rest due to metabolic disorders, acid-base imbalance, nutrient deficiency, and the pathological effects of certain medications.

Objective: Optimization of rehabilitation in critical illness polyneuromyopathy.

Materials and Methods: We examined 23 patients with critical illness polyneuromyopathy syndrome.

Results and Discussion: Approximately 30% of patients under our observation requiring respiratory support developed neuromuscular disorders within 7 days of stay in intensive care units (ICU). In 60% of patients with sepsis or systemic inflammatory response syndrome, critical illness polyneuromyopathy was detected, and its incidence reached 100% among patients with multiple organ failure. Risk factors for developing CIPNM included old age, female sex, treatment with corticosteroids and aminoglycosides, hypoxia, hypotension, hyperthermia, hyperglycemia, and hypoalbuminemia.

Preventive measures for CIPNM development include providing adequate nutrition, glucose control, proper restorative treatment, and cautious use of drugs such as corticosteroids, neuromuscular transmission blockers, and certain antibiotics. To prevent CIPNM, adequate treatment and prevention of sepsis, systemic inflammatory response syndrome, and multiple organ failure are essential. Respiratory physiotherapy should be performed to treat and prevent hospital-acquired pneumonia. To prevent pressure ulcers, regular repositioning of the patient and use of an anti-decubitus mattress are advisable.

Strict blood glucose control is necessary because hyperglycemia prolongs CIPNM. Intensive insulin therapy (compared to standard insulin regimens), despite the risk of hypoglycemia, reduces the duration of respiratory support, ICU stay, and 180-day mortality. Maintaining blood glucose levels between 80–110 mg/dL significantly lowers CIPNM incidence compared to patients with glucose levels of 180–200 mg/dL.

Rehabilitation should ideally begin early in CIPNM cases in the ICU. Initially, only low-intensity therapeutic exercises are performed to preserve muscle strength, joint mobility, and prevent joint contractures. The rehabilitation program may be long-term and include mechanotherapy. Therapeutic physical training is necessary for patient adaptation to daily life activities and increasing mobility. Successful CIPNM treatment requires coordinated care by neurologists and rehabilitation physicians. Rehabilitation interventions that reduce neuromuscular weakness play a vital role in improving patients' quality of life and reducing treatment costs.

For maximal effectiveness of restorative treatment, continuity in rehabilitation is necessary, and patients should continue rehabilitation on an outpatient basis. Rehabilitation planning is based on the severity of neurological symptoms, pathogenesis of the primary disease, recovery pace, and the level of social support.

Conclusion: Thus, an interdisciplinary approach is required for effective treatment of patients with CIPNM to optimize social and professional rehabilitation and reduce patient disability.