Neuromuscular Complications of Critical Illness

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KEYWORDS

• Critical illness polyneuropathy • Critical illness myopathy
• Acute quadriplegic myopathy • Sepsis • Neuromuscular disorders

KEY POINTS

- Critical illness neuropathy and myopathy are neuromuscular complications of sepsis or are iatrogenic complications of treatments required in the intensive care setting.
- A combination of neuromuscular complications can occur in one patient.
- The severity of CIP/CIM depends on the length of ICU stay, severity of illness, and definitive diagnosis.
- Recovery depends on the degree of peripheral nerve axonal involvement.

BACKGROUND

Generalized weakness of limb and respiratory muscles in intensive care unit (ICU) patients is a long recognized phenomenon. Neuromuscular complications of critical illness are usually discovered in the patient that has difficulty in weaning from mechanical ventilation that cannot be explained by pulmonary or cardiac compromise.[1] Critical illness polyneuropathy (CIP) and critical illness myopathy (CIM) are the most common entities identified as the cause of neuromuscular weakness in the ICU.[1–3] In 1984, Bolton and colleagues[4] described a polyneuropathy that developed after sepsis and multiorgan failure. Later a myopathy initially linked to steroids and neuromuscular blocking agents (NMBA) was identified.

The association between sepsis and neuromuscular weakness has been known for more than 50 years. In 1955, Erbsloh[5] observed a polyneuropathy that developed in a patient after a prolonged coma. Mertens,[6] in 1961, described "coma-polyneuropathies" in patients who developed hypovolemic shock. Similarly, Bischoff and

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colleagues[7] in 1977 observed four patients develop a severe polyneuropathy after sepsis that they originally attributed to the use of gentamicin.

Charles Bolton, however, provided the most thorough analysis of this phenomenon in the early 1980s. In 1981, Bolton and colleagues[8] described five ICU patients who had difficulty weaning from the ventilator and severe limb weakness. Electrophysiologic studies revealed a primary axonal degeneration of motor and sensory fibers. At the time, possible etiologies including nutritional deficiencies, heavy metal toxicity, antibiotic use, collagen vascular disease, or spinal cord ischemia were ruled out. Therefore, it was suggested that sepsis itself was the underlying cause of the documented neuromuscular weakness.[1]

By 1983, a total of 19 cases of polyneuropathies were prospectively identified and studied through electrophysiologic testing by Bolton.[1] The term "critical illness polyneuropathy" was used to identify this neuropathy because it was clearly associated with sepsis and multiorgan failure. The neuropathy was similarly associated with the encephalopathy being described in patients with sepsis. In their study, 70% of patients with sepsis and multiorgan failure were affected by this polyneuropathy based on electrodiagnostic studies.[4]

APPROACH TO ACUTE NEUROMUSCULAR WEAKNESS IN THE ICU

The approach to a patient who is found to have weakness in the ICU should include a thorough history and general physical and neurologic assessment. The clinical examination may be limited by sedation and/or an encephalopathy. Despite this difficulty, the history, timing, and pattern of weakness must be assessed to differentiate the cause. For example, a distal bilateral motor involvement after a recent infectious illness suggests Guillain-Barré syndrome (GBS). Hyporeflexic quadriparesis after awakening from trauma suggests an acute spinal cord injury. Unilateral limb involvement could be explained by a plexopathy, whereas weakness confined to a single nerve distribution strongly points toward a compression neuropathy.

GBS and myasthenia gravis are neuromuscular disorders directly admitted to the ICU primarily for progressive respiratory weakness, yet critical illness can exacerbate these diseases. Identifying the neuropathy or myopathy associated with critical illness as the source of neuromuscular weakness is important because it may help to avoid performing unnecessary studies, such as imaging of the brain.

Many patients with neuromuscular weakness in the ICU are identified because of failure to wean from mechanical ventilation. Up to 30% of ICU patients may experience difficulty in weaning off the ventilator.[9] Common causes for inability to wean from mechanical ventilation should be ruled out first and include cardiac, pulmonary, and chest wall dysfunction. Neurologic causes account for the other etiologies of failure to wean.[2] In one study, central neurologic causes for failure to wean including stroke and depressed mental status accounted for 26% of all cases. Peripheral neurologic sources accounted for another 17%.[9] CIP was usually the source of peripheral nervous system failure, whereas other peripheral nerve sources (ie, unilateral phrenic neuropathy, defects in neuromuscular transmission, and myopathy) accounted for the remainder. The number of cases identified as peripheral nerve sources of ventilator failure increased with increased use of electrophysiologic studies.

Electrodiagnostic studies are important in the evaluation of patients with neuromuscular weakness and allow for localization of the injury to the motor nerve, neuromuscular junction, or muscle. They may, however, be difficult to perform and interpret in the ICU setting and usually require more than 3 weeks of symptoms to arrive at a diagnosis.[10]