Intensive Care Unit–Acquired Weakness

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KEYWORDS

• ICU-acquired weakness • Critical illness neuromyopathy • Critical illness myopathy • Critical illness polyneuropathy • Post-ICU syndrome

KEY POINTS

- ICU-acquired weakness (ICUAW) contributes substantially to the disability experienced by patients in the aftermath of critical illness.
- A combination of critical illness myopathy (CIM) and critical illness polyneuropathy (CIP), both of which are principally caused by the downstream effects of inflammation, and muscle atrophy comprise ICUAW.
- A variety of tests can be used to diagnose ICUAW and its components CIM and CIP, including strength testing, electrophysiology, and muscle ultrasound, although each has important limitations to consider.
- Risk factor modification including aggressive treatment of provoking inflammatory conditions and minimization of sedation and early mobilization can improve outcome.

Video content accompanies this article at http://www.neurologic.theclinics.com.

INTRODUCTION AND DEFINITIONS

Although Sir William Osler reported a "rapid loss of the flesh" in patients with prolonged sepsis as early as 1892, the cause of neuromuscular weakness associated with critical illness was not fully recognized until almost a century later.[1] Technological advances in cardiopulmonary support made during this time increased the number of patients that survive critical illness; however, it was increasingly observed that their survival would come at the unfortunate cost of physical disability and a reduction in quality of life caused by weakness acquired in the intensive care unit (ICU).[2] Ultimately, this syndrome of limb and respiratory weakness that frequently develops in the wake of critical illness was termed ICU-acquired weakness (ICUAW).[3] Nerve and muscle dysfunction in the setting of critical illness, recognized even before the coining of

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the term ICUAW, represents the most prominent cause of ICUAW: critical illness polyneuropathy (CIP) and critical illness myopathy (CIM).[4,5] CIP and CIM remain distinct clinical entities, although they commonly co-occur. The term critical illness neuromyopathy (CINM) has been developed to describe the spectrum of co-occurrence of CIP and CIM in patients with ICUAW.[6]

ICUAW is a clinical undifferentiating definition describing the weak critically ill patient. In contrast, CINM (and its subcomponent entities CIP and CIM) is a pathophysiologic definition describing the weak critically ill patient with evidence of associated neuromuscular dysfunction (**Fig. 1**).

This review discusses the epidemiology and risk factors of ICUAW, reviews the suspected pathophysiology, describes the distinguishing clinical and electrophysiologic features and the associated challenges in diagnosing ICUAW/CINM in the ICU population, examines the evidence behind management options for the disease, and investigates the impact ICUAW/CINM has on prognosis.

EPIDEMIOLOGY AND RISK FACTORS

The incidence of ICUAW can approach a staggering 80% of critically ill patients, although this figure varies according to the presence, number, and severity of risk factors and the time point at which the patient is evaluated. Multiple studies have investigated the risk factors associated with ICUAW and those most consistently implicated relate to the severity of the underlying critical illness and inflammation. Specifically, sepsis, shock, and the presence and degree of multiorgan failure are the risk factors that have most frequently and reliably been associated with ICUAW.[3,7–9] Septic encephalopathy, a disease whose pathophysiology is speculated to be at least in

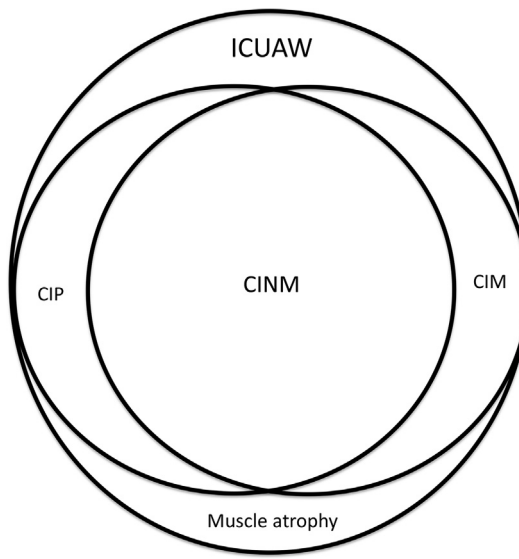

Fig. 1. Association between ICUAW, CINM, CIP, and CIM. Critically ill patients who develop clinical signs of weakness have ICUAW. Most patients with ICUAW have evidence of nerve and/or muscle dysfunction and are further characterized as having CIP and/or CIM, respectively. The term CINM is used to describe the common co-occurrence of CIP and CIM in the individual patient. Muscle atrophy also likely plays a role in the development of ICUAW and may coexist with CINM.