


Intensive Care Unit Acquired Weakness Associated With Acute Severe Asthma

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Clinical Medicine Insights: Case Reports

Volume 15: 1–4

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DOI: 10.1177/11795476221106759



ABSTRACT

CONTEXT: Neuromuscular complications in the intensive care unit (ICU) are frequent, multifactorial, and clinically difficult to recognize during their acute phase. The physical examination is the starting point for identification.

CASE REPORT: We present a patient with a history of poorly controlled asthma who was admitted to the ICU with status asthmaticus. After 4 days of being under ventilatory support, he developed muscle weakness. The diagnostic approach made it possible to establish myopathic and neurological compromise through electrophysiology studies.

CONCLUSIONS: ICU-acquired weakness (ICUAW) can bring long-term consequences, early identification, and management, as well as preventive measures, are essential to minimize chronic disability and morbidity.

KEYWORDS: Muscle weakness, risk factors, diagnosis, Intensive care unit, ICU-acquired weakness, critical illness

RECEIVED: March 31, 2022. **ACCEPTED:** May 21, 2022.

TYPE: Case Report

FUNDING: The author(s) received no financial support for the research, authorship, and/or publication of this article.

DECLARATION OF CONFLICTING INTERESTS: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Learning Points

- ICU-acquired weakness (ICUAW) is a multifactorial condition produced by impaired muscle function
- The physical examination is the starting point for identification of ICUAW
- Postural changes, splints, application of electrical neuromuscular stimulation during ICU stay are preventive actions that impact in patient recovery and outcomes.

Background

Various severely ill survivors suffer neuromuscular disorders acquired or aggravated during their stay in the intensive care unit (ICU).¹ ICU-acquired weakness (ICUAW) is characterized by impaired muscle function, and in which no other cause than the acute disease or its treatment can be identified.¹ Risk factors include sepsis and/or shock, multiple organ failure, metabolic variables such as hyperglycemia, and interventions such as the duration of mechanical ventilation and deep sedation.²

ICUAW can occur even several hours after ICU admission and outcomes are heterogeneous; while some people fully recover, others experience persistent weakness that leads to functional disability and diminished quality of life.¹ Currently, there is no gold standard for the early diagnosis of ICUAW.³ The physical examination is the starting point for identification. Once muscle weakness (MD) is evident, neurophysiological studies, electromyography, nerve conduction studies, and

the Medical Research Council (MRC)³ skeletal muscle strength assessment are used to determine the presence of ICUAW. To improve the knowledge of this condition, this paper reports a case of ICUAW and discusses its clinical manifestations, risk factors, diagnostic approach, findings, and relevant literature.

Case Presentation

We present the case of a 43-year-old female with a history of difficult-to-manage bronchial asthma, hospitalized in the last 8 months on 10 occasions for acute exacerbations, asthmatic crisis. She presented with a 24-hour history of dry cough, progressive dyspnea, psychomotor agitation, and audible wheezing that did not improve with outpatient use of salbutamol and ipratropium bromide. She was admitted in critical condition, diaphoretic, tachycardic (heart rate 110 beats/minute), dyspneic with supraclavicular retractions (respiratory rate of 30 breaths/minute, ambient oxygen saturation 87%), auscultation with abolished vesicular murmur in both lung fields, Glasgow coma scale (GCS) 12/15. Arterial blood gases (ABG) analysis revealed respiratory acidosis and hypoxemia (pH: 7.21 PCO₂: 66.5 PO₂: 53.4, HCO₃: 26.5 BE -2.8, SaO₂ 79.1% PaO₂/FiO₂: 178).

Given the imminence of ventilatory failure, invasive ventilatory support was provided, with midazolam (loading dose 0.2 mg/kg, maintenance dose 0.05 mg/kg/h) and propofol



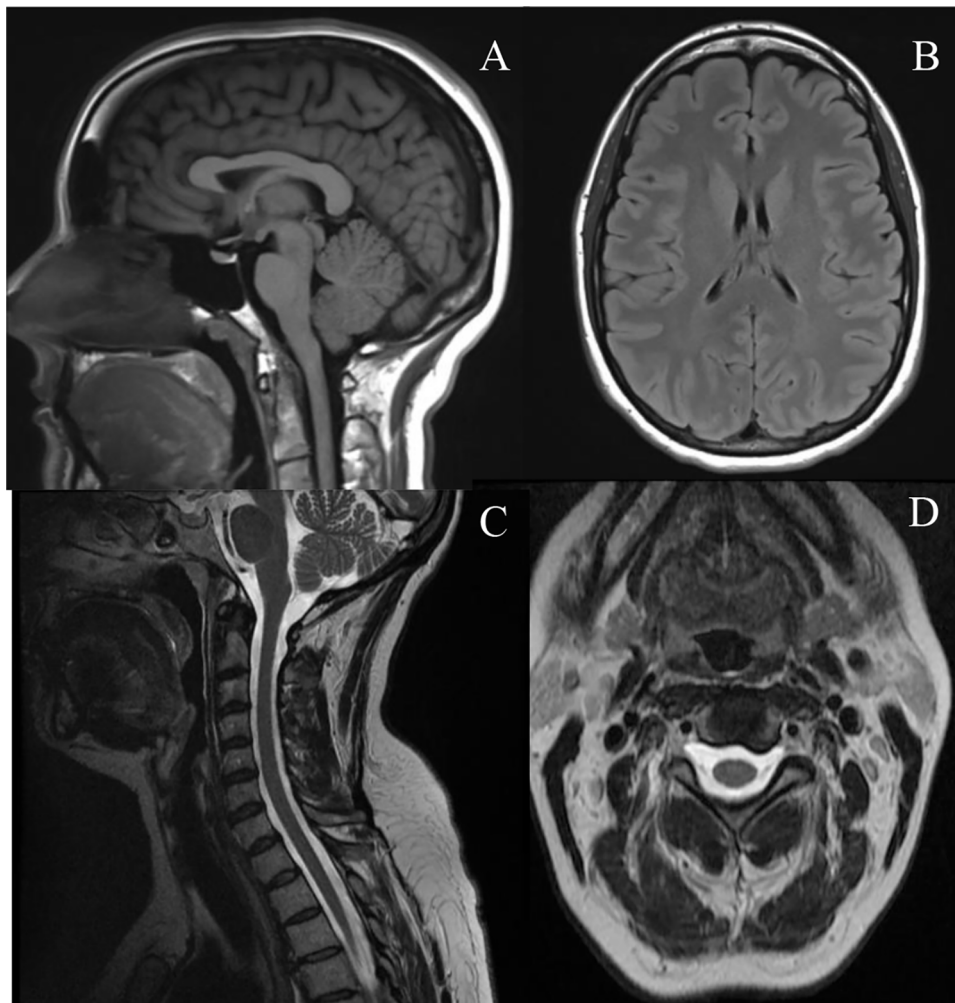


Figure 1. Brain MRI: Sagittal T1 (A) and Axial Flair (B) being normal. Cervical MRI: Sagittal T2 (C) and Axial T2 (D) with normal findings.

(loading dose 1 mg/kg, maintenance dose 1 mg/kg/h) for improved mechanical ventilator-patient synchrony, and she was transferred to the ICU. She was managed with hydrocortisone 100 mg IV every 8 hours, magnesium sulfate, and antibiotics (piperacillin tazobactam and clarithromycin), considering infection as the cause of the asthmatic crisis. At day 2 a short-term (48-hour) infusion of neuromuscular blocking agents (NMB) was associated (cisatracurium initial dose of 0.15 mg/kg and maintenance dose of intermittent 0.03 mg/kg IV bolus. Train of 4 used to monitor the paralysis maintaining a range of 1-2). On the fourth day, she presented improvement in ABG; Ventilatory weaning was indicated, suspending dual sedation and NMB. Light sedation with dexmedetomidine was started. On day 6, her GCS was 15/15, but she had weakness of the neck flexor muscles, facial paresis, she could not move all 4 limbs (muscular strength 1/5 in lower limbs, 2/5 in upper limbs), she had flaccid hyporeflexia, with preserved sensitivity. There was no alteration in cranial nerves.

She was assessed by neurology who requested brain and cervical MRI (Figure 1) and a study of cerebrospinal fluid, which were found to be normal. The MRC score was 37 points.

Electromyography (EMG) was performed, revealing signs of denervation and irritability (myopathic pattern), polyneuropathic compromise with axonal pattern in conduction velocity, Figure 2.

Thus, the diagnosis of ICUAW was confirmed, physiotherapy and comprehensive rehabilitation were started, the ventilator was withdrawn it was achieved on day 10. At hospital discharge, 30 days after admission, the patient had an MRC score of 55 points, normal ABG control with no acidosis neither hypoxemia; symptomatic resolution was achieved.

Discussion and Conclusions

We present the case of an asthmatic patient subjected to mechanical ventilation, due to an exacerbation of her disease treated with corticosteroids and NMB, who, by solving her illness, develops ICUAW. The physical examination was the starting point for her identification.³ She clinically presented typical findings given by quadriplegia, symmetrical, flaccid, more pronounced at the proximal level, with cervical and facial involvement, respecting ocular muscles.¹ When it can be explored, sensitivity is normal and tendon reflexes are diminished.¹

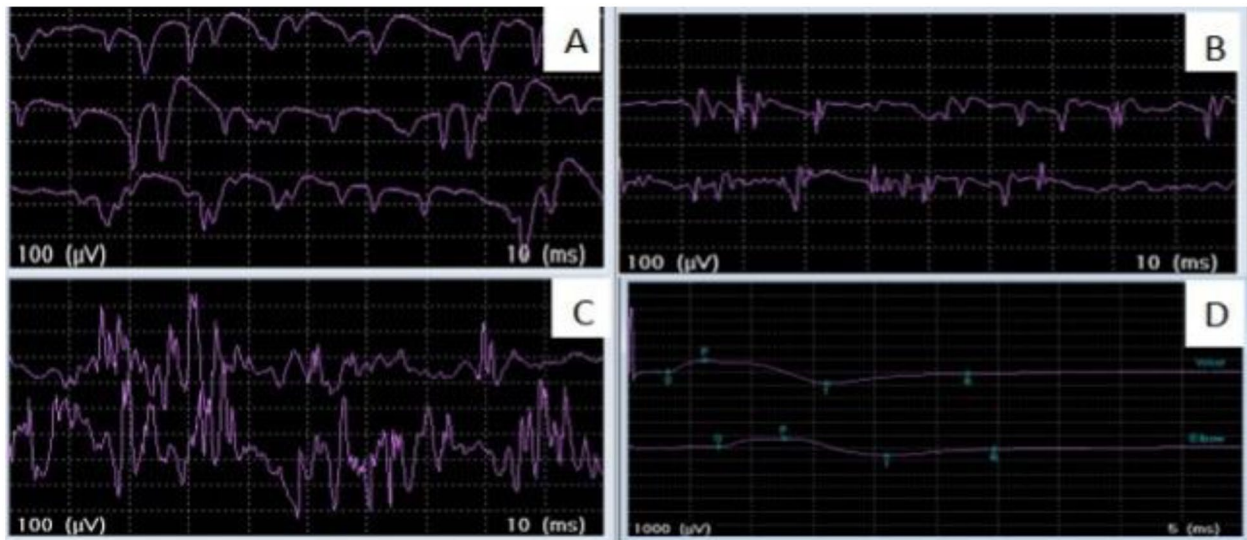


Figure 2. Electromyography of the right peroneus longus (A) and left tibialis anterior (B) muscles show spontaneous pathological activity given by positive waves and fibrillations and left biceps (C) with increased recruitment with potentials of low amplitude and short duration. Nerve conduction velocity of the median motor nerve (D) was found to be normal, with decreased amplitude of potentials bilaterally, no sensory potentials are evoked. Neurophysiological study of 4 limbs with signs of fiber denervation and irritation with a myopathic pattern and polyneuropathic involvement with an axonal pattern.

It is worth mentioning that patients under sedation usually respond to painful stimuli with facial grimaces without achieving limb withdrawal.¹ On the other hand, in awake patients, the MRC scale can be used, whose score ranges from 0 to 60, considering abnormal below 48 points⁴; this patient presented 37 points compatible with mild quadriplegia to severe quadriplegia.

If there's atypical presentation or inconclusive status, it is necessary to investigate differential diagnoses such as stroke, neuroinfection, upper motor neuron syndrome, Guillain-Barré syndrome, myasthenia gravis, rhabdomyolysis, muscular dystrophy, spinal cord compression.⁴ In this case, differential diagnoses were explored. Brain and cervical MRI and lumbar puncture, subsequently proceeding to perform the EMG.

Muscle weakness can originate from a peripheral neurogenic disorder "critical illness polyneuropathy" (CIP), a myogenic muscle fiber disorder "critical illness myopathy" (CIM), or a combination of these called "critical illness neuropathy."⁴ Differentiation between them occurs through electrophysiological studies.³ Typical signs of CIP are reduced amplitude of compound muscle action potentials as well as sensory nerve action potentials. CIM manifests with a motor unit potential of short duration, low amplitude, with early recruitment.⁴ In this case, the EMG showed that it was a critical illness neuropathy.

Among the risk factors associated with ICUAW, the most identified is hyperglycemia; however, female gender, mobility restriction, age-related physical conditioning, the use of parenteral nutrition, myo/neurotoxic drugs, steroids, NMB have also been described.² Muscle weakness due to long-term use of corticosteroids occurred due steroids catabolic effect on skeletal muscles, that caused type II muscle fiber atrophy and proximal muscle weakness.⁵ The effect of corticosteroid

therapy on ICUAW is complex and may also depend on the duration and cumulative dosage of the corticosteroids.⁶ ICUAW caused by excessive steroid use is difficult to diagnose due to insignificant changes in muscle enzymes and lack of specificity and diversity in the electrophysiological results.⁶ Nevertheless, disease related to long-term use of corticosteroids can only be determined with clinical symptoms.⁵ In this case we considered steroid usage as a risk factor which contribute to develop ICUAW.

Prolonged mechanical ventilation (>10 days) is associated with 67% of ICUAWs, however, 1 in 10 patients with at least 24 hours of mechanical ventilation ends up with ICUAW.² In this patient, ICUAW was identified 6 days after being under mechanical ventilation with standard doses of steroids.

Currently, there is no specific treatment.¹ Prevention is essential with postural changes, splints, application of electrical neuromuscular stimulation⁵ and early identification after muscle-neuro-anatomical correlation and risk factors, bearing in mind the implications for morbidity and mortality and quality of life.

Author Contributions

The authors confirm contribution to the paper as follows: Study conception and design: Diana Borré-Naranjo, Amilkar Almanza-Hurtado. Data collection: María Cristina Martínez. Analysis and interpretation of results: Tomás Rodríguez. Draft manuscript preparation: Carmelo Dueñas-Castell. All authors discussed the results and contributed to the final manuscript.

Consent for Publication

No written consent has been obtained from the patients as there is no patient identifiable data included in this case report

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