



Muscle Weakness Acquired in the Intensive Care Unit: Case Report

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Abstract

Introduction: Intensive Care Unit-Acquired Weakness (ICUAW) is a growing problem, associated with a longer duration of mechanical ventilation, greater permanence and in-hospital mortality. Preventing its risk factors, therefore, becomes fundamental in the approach to the critical patient.

Case Presentation: A 45 year-old male, with no known previous comorbidities, but acromegalic phenotype, presented pale, dehydrated, hypotense and tachycardic with consciousness fluctuations to the emergency service. Investigation pointed to Diabetic Ketoacidosis (DKA) and hypokalemia. The patient underwent neuroimaging studies and Magnetic Resonance (MRI) showed a pituitary macroadenoma with suprasellar invasion. Growth Hormone (GH) and IGF-1 (Insulin Growth Factor-1) levels were increased and diagnosis of acromegaly was then established. In the fifth day there was a peak of Creatine Kinase (CK) with progressive decrease. After 12 days of hospitalization, with resistant hyperglycemia, proximal flaccid tetra paresis and global hyporeflexia (MRC score of 40 points) was noted. Clinical diagnosis of Critical Illness Myopathy (CIM) was established. After 30 days of hospitalization, he was discharged with MRC score of 50 points and improves in CK levels.

Discussion: The presented case shows a CIM, a type of ICUAW. Some important risk factors are endocrine logical (thyroid diseases, diabetes, adrenal and pituitary dysfunctions). The patient presented hyperglycemia and underwent a prolonged period of hospitalization and use of glucocorticoid medications, increasing the risk of the disease. With clinical management and physiotherapy the patient regained autonomy.

Conclusion: ICUAW can bring long-term consequences, so that early identification and management as well as preventive measures are primordial to minimize chronic disability and morbidity.

Introduction

Intensive Care Unit-Acquired Weakness (ICUAW) is a growing problem in critically ill patients and is associated with a longer duration of mechanical ventilation, greater permanence and in-hospital mortality, in addition to higher costs to the health system. Preventing its risk factors, therefore, becomes fundamental in the approach to the critical patient [1].

Case Presentation

Male, 45 years old, with no known comorbidities or use of medications, entered the Emergency Service of Santa Casa de São Paulo, with a drop in general condition, fluctuation in consciousness and dehydration. Upon admission, Glasgow 14, severe dehydration and pale. Ectoscopy showing acromegalic facies, enlarged extremities, acrochordons in the cervical region, macroglossia and skin sweating. Vital signs: Blood pressure 100 mmHg × 60 mmHg, HR 100 bpm, SatO₂ 97% in room air, capillary blood glucose above 500 mg/dL. Laboratory tests were performed, of which alterations are reported: plasma glucose 503 mg/dL (RV 70-99), glycated hemoglobin (HbA1c) 9.8% (RV <5.7%), creatinine 1.9 mg/dL (RV 0.6-1.1), plasma sodium 175 mEq/L (RV 135 to 145), potassium 3.4 mEq/L (RV 3.5 to 5.1), CPK 8023 U/L (RV 38 to 174) and arterial blood gases: pH 7.2, pCO₂ 19, HCO₃ 6.3, BE -3.2 and lactate 3.9. Type 1 urine with 3 ketonuria crosses.

The patient was diagnosed with Diabetes and Diabetic Ketoacidosis (DKA), starting treatment for DKA, prioritizing intravenous hydration, with care not to reduce acute sodium levels, in addition

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to an intravenous insulin pump, after correction of hypokalemia. Due to the fluctuation in consciousness, the patient underwent a cranial tomography, showing a lesion in the pituitary topography, progressing the investigation with Magnetic Resonance (MRI), which showed a Pituitary Macro adenoma with suprasellar invasion, compressing optical chiasma, measuring in its largest axis 6.1 cm.

Pituitary hormone measurements were performed: GH >200 mcg/L, IGF-1 476 ng/mL (RV 112 to 282), Prolactin 21 mcg/L (RV up to 24), ACTH 28p g/mL, Basal cortisol 23 mg/dL (RV >15), TSH 1.2 mUI/L (RV 0.45 to 4.5) and free T4 0.5 ng/dL (RV 0.6 to 1.3).

Diagnosis of Acromegaly due to hormonal changes, lesion in adenohypophysis and suggestive phenotype. Treatment with a somatostatin analogue (Octreotide) 20 mg monthly was started, in addition to a dopaminergic agonist (Cabergoline) 1.5 mg/week.

One day after admission, the patient evolved with a decrease in the level of consciousness requiring Orotracheal Intubation (OTI). He maintained high sodium levels, but already improving DKA. He was kept under OTI for 4 days, being extubated after normalization in sodium levels.

The patient refused the surgical procedure for the treatment of acromegaly. He remained with difficult glycemic control during the period of hospitalization, with improvement in intra hospital glucose levels after about 15 days of admission, using high doses of insulin (2 ui/kg/day). He also received treatment with corticosteroids (hydrocortisone 300 mg/day for 8 days, followed by prednisone 40 mg/day for 18 days) due to the airway infection associated with the condition (sinusitis).

After 12 days of hospitalization, a picture of flaccid tetra paresis of proximal predominance (total MRC score of 40 points), associated with global hyporeflexia, without changes in cranial nerves, sensitivity or in other modalities on neurological examination, was noted. Cerebrospinal fluid was normal. On the fourth day of hospitalization, he presented a Creatine Kinase (CK) of 12665, with a peak on the fifth day of 12888, followed by a gradual decrease during hospitalization. In the two weeks following extubation, the patient showed progressive clinical improvement in the peripheral nervous system, especially after better glycemic control. Due to the presence of generalized muscle weakness after severe, diffuse, symmetrical, flaccid pattern and without involvement of cranial nerves, associated with a sum of MRC less than 48 points, a diagnosis of myopathy of the critically ill patient was then established.

At the time of hospital discharge, 30 days after admission, the patient had a sum of the MRC score of 50 points, good glycemic control, using insulin, improvement in CPK, creatinine and sodium levels. Maintained GH 195 mcg/L (RV <1) and with refusal of surgical procedure. Referred to the endocrinology clinic to follow up on the diagnoses of acromegaly and diabetes; and the neurology outpatient clinic for follow-up of the diagnosis of myopathy.

Given the current epidemiological context, we emphasize that throughout the hospitalization period, the patient did not show any signs or symptoms suggestive of COVID-19 infection.

Discussion

In the case in question, the patient had multiple risk factors for the development of ICU-Acquired Weakness (ICUAW) and had a course of Critical Illness Myopathy (CIM). Although the pathophysiology of CIM is still being studied, the likely mechanisms include

inhibition of protein synthesis, mitochondrial dysfunction, changes in the ubiquitin-proteasome system, oxidative stress and changes in intramuscular calcium homeostasis [2]. Hyperglycemia associated or not with the use of steroids, use of neuromuscular blockers and sepsis are the main risk factors [3].

Myopathy is worsened by multiple endocrine logical and metabolic factors. Among them, thyroid diseases, diabetes, adrenal dysfunctions and acromegaly stand out [4]. The patient in question had hyperglycemia, with DKA, and was diagnosed with acromegaly during hospitalization. Acromegaly, characterized by high plasma levels of GH and IGF-1, has pituitary adenomas as its main etiology. It usually causes slowly progressive proximal myopathy and normal CPK levels [5]. Myopathy presented by the patient probably was not due to the acromegaly, once there was clinical neurological improvement despite maintaining activity of this disease, documented by the presence of high GH levels, even after therapy with somatostatin analog.

Diabetes is associated with multiple presentations of neuropathy and muscle involvement. Pathophysiology may be associated with microvascular changes in muscle and nerve regions, but neuropathies are not common in the first years of the disease [5]. They tend to appear after 10 years of diagnosis and are commonly associated with other micro vascular lesions typical of this disease, such as retinopathy and nephropathy [6]. Regarding the clinical muscle condition, it has been documented in some cases that the cause of the injury is probably due to focal myonecrosis, followed by micro vascular injury [7].

Low carbohydrate diet and hydration should be instituted, in addition to the measurement of glycated Hemoglobin (HbA1c), aiming at the previous diagnosis of diabetes, in those patients with in-hospital hyperglycemia in addition to assisting in the management of the disease at hospital discharge [6]. The insulin prescription should ideally be performed according to the protocol of each institution or with the help of a specialist. Glycemic control aims at an intra-hospital goal between 140 and 180 mg/dl [6-8]. These measures reduce overall mortality and the risk of developing polyneuropathy in critically ill patients. Values below 100 mg/dL are correlated with increased mortality, due to the risk of severe hypoglycemia and, therefore, should be avoided [8,9].

Myopathy mainly affects the proximal and axial muscles and makes weaning from mechanical ventilation and recovery of normal respiratory function difficult. It can be associated with elevation of muscle enzymes, such as increased Creatine Kinase (CK), which can assist in diagnosis, as occurred in the case reported. However, CK values within normal levels can be found in mild cases or at the beginning of muscle involvement and, therefore, should not exclude the diagnosis in the face of a strong clinical suspicion [10].

In the Electroneuromyography exam, changes such as the reduction of the amplitude and duration of the action potentials of the motor unit with early recruitment stand out, in which there may be, in some cases, fibrillations and positive acute waves. In nerve conduction studies, the result is usually normal, but there may be a reduction in the amplitude of the Compound Muscle Action Potential (CMAP) with prolonged duration, suggesting MDC [10].

Muscle biopsy shows signs of myopathy with loss of myosin, severe atrophy of type 2 fast-twitch fibers and loss of the myosin heavy chain. Findings such as accumulation of inflammatory cells, glycogen or lipids are not typical of MDC and indicate the need for

investigation of differential diagnosis, such as immune-mediated inflammatory myopathies, dystrophic or metabolic myopathy [11].

The patient persistently refused to perform invasive exams, as he progressed with evident clinical improvement. Such complementary exams were not performed and a diagnosis was established based on clinical criteria: “Generalized weakness developing after the onset of a serious illness; diffuse weakness (affecting the proximal and distal muscles), symmetrical, flabby and sparing cranial nerves; the sum of the MRC-SS score <48 points, more than once with an interval >24 h; and other causes of weakness unrelated to serious illnesses have been ruled out.”

Conclusion

ICUAW can bring long-term consequences. It is important to pay attention to identify it, in the ICU environment, and provide early rehabilitation in order to minimize chronic dysfunctions, morbidity and disability.

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