ABSTRACT: Several recent studies have attributed the occurrence of acute myopathy in intensive care unit patients to the combination of corticosteroids and neuromuscular junction blocking agents (NMBAs) used for mechanical ventilation. We present 4 patients who developed acute myopathy after administration of high doses of glucocorticoids during sedation without any NMBAs. All patients had elevated creatine kinase levels. Electrophysiological studies indicated normal motor and sensory nerve conduction velocities but reduced motor nerve response amplitudes. Needle electromyography identified abnormal spontaneous activity; motor unit potentials were polyphasic of low amplitude and short duration, characteristic of a myopathic process. Muscle biopsy demonstrated a prominent acute necrotizing myopathy in all 4 patients with a loss of thick filaments. Our observations support glucocorticoids rather than NMBAs as the main offending drug in acute corticosteroid myopathy. The predisposing factor should be the hypersensitivity of paralyzed muscles to corticosteroids regardless of the drug inducing paralysis: NMBAs or propofol. © 1997 John Wiley & Sons, Inc. Muscle Nerve 20: 1371–1380, 1997

Key words: corticosteroids; myopathy; electromyography; muscle blocking agents; propofol

ACUTE CORTICOSTEROID MYOPATHY IN INTENSIVE CARE PATIENTS

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Prolonged muscle weakness sometimes persists after discontinuation of neuromuscular junction blocking agents (NMBAs) or sedative drugs used for mechanical ventilation in intensive care unit (ICU) patients. "Critical illness polyneuropathy" is often suspected, but several recent reports have identified cases of acute quadriplegic myopathy after high doses of corticosteroids.

Acute corticosteroid myopathy (ACM) in patients being treated for severe asthma has been recognized with increasing frequency (44 cases described in the literature) since its first description in 1977 by MacFarlane and Rosenthal.25 A few patients without asthma history have been described.20,21

Many factors have been hypothesized to explain the development of this acute myopathy but most investigators2–5,7,9–12,19,21–25,27,29–32,35 now agree that large doses of parenteral corticosteroids in association with NMBAs are responsible.

In 1994, Zochodne et al.36 published a series of 7 patients with acute myopathy after receiving NMBAs; 3 of these patients did not receive corticosteroids. Recently, Gooch18 confirmed the long-term use of NMBAs, either alone or in combination with other medications or disorders, to explain prolonged flaccid paralysis in some intensive care patients.

To have a better understanding of the mechanisms underlying the occurrence of acute myopathy, we present 4 cases of acute myopathy diagnosed in the ICU of our hospital between July 1992 and December 1993.

All our patients received high doses of glucocorticoids. In contrast to previous reports, hypnotic doses of sedative agents were used for adaptation to mechanical ventilation without the use of NMBAs.

CLINICAL FEATURES

The main symptom indicating a neuromuscular pathology in these 4 patients was a flaccid quadriplegia
diagnosed after discontinuation of sedative drugs with difficulty in weaning patients from ventilatory support because of respiratory muscle weakness. Clinical examination indicated depressed or absent muscle stretch reflexes, preserved sensibility, and severe amyotrophy; strength was grade 0–1/5 in proximal and distal muscles. Cranial nerve function was intact without ophthalmoplegia.

Creatine kinase (CK) levels and CK-MB fractions were measured daily during the first 4 weeks. All patients had elevated CK levels but normal CK-MB fractions. ECG confirmed the absence of myocardial necrosis. No intramuscular injections were administered during the stay in the ICU.

Patient 1 (S.M., male, 52 years) was admitted for asthma. Patient 2 (P.J., male, 64 years) had a history of stable chronic obstructive bronchopneumopathy and developed acute respiratory failure 5 days after a prostatectomy for adenocarcinoma. Patient 3 (V.A., male, 68 years) underwent a partial left lung resection for an epidermoid carcinoma with respiratory failure; 2 weeks after surgery he complained of progressive dyspnea requiring mechanical ventilatory assistance for a severe hypoxemia due to a nosocomial pneumonia complicated by septic shock. Patient 4 (W.M., female, 82 years) was admitted for cardiopulmonary failure without any respiratory antecedent. None of the patients were known to have a neuromuscular disorder prior to admission to the ICU.

All these patients were treated with high-dose intravenous glucocorticoids (methylprednisolone) for acute respiratory failure. They were all intubated and received hypnotic doses of sedative agents (propofol and benzodiazepines). Only patient 2 was treated with aminoglycoside antibiotics.

The relationship between the use of sedative agents and glucocorticoids and the CK level is demonstrated for each patient in Figures 1–4. The exact timing of EMG and biopsy are indicated in these figures.

Patient 3 died 6 weeks after admission to the ICU of respiratory complications. For the other patients, improvement was slow with recovery of limb motor function in 10–20 days. Walking without crutches was possible after 2 months for patient 1, after 5 months for patient 4, and after 8 months for patient 2.

METHODS

**Electromyography/Nerve Conduction Study.** Electrophysiological studies were conducted in the ICU by the same examiner (P.H.) using standardized methods.

![Graph](image-url) **Patient 1 S.M. 52 years**

**FIGURE 1 to 4.** Relationship between the use of sedative agents and glucocorticoids and the CK level for each patient. The delay between admission to the ICU and the EMG and biopsy is also indicated. Patient 1: S.M., 52-year-old male; EMG on day 18, biopsy on day 21. Patient 2: P.J., 64-year-old male; EMG on day 16, biopsy on day 27. Patient 3: V.A., 68-year-old male; EMG on day 22, biopsy on day 23. Patient 4: W.M., 82-year-old male; EMG on day 21, biopsy on day 26. Hatched bars = methylprednisolone IV (mg/day); cross-hatched bars = creatine kinase (normal < 190 IU/L).
techniques according to Kimura's principles with a Nicolet Viking device (Madison, WI).

Motor and orthodromic sensory nerve conductions were recorded using surface electrodes after surface stimulation; amplitudes were measured peak to peak for motor responses and baseline to negative peak for sensory responses. Motor nerve conduction was recorded from the common peroneal, posterior

**Patient 2  P.J.♂ 64 y**

![Graph of Patient 2's data]

- methylprednisolone IV mg/24 hrs
- propofol IV 70 - 30 ml/hr
- Creatine Kinase (nl < 190 u/l)
- EMG on day 16  BIOPSY on day 27

**Patient 3  V.A.♂ 68 y**

![Graph of Patient 3's data]

- methylprednisolone IV mg/24 hrs
- propofol IV 70 - 30 ml/hr
- Creatine Kinase (nl < 190 u/l)
- EMG on day 22  BIOPSY on day 23
tibial, and ulnar nerves. Sensory nerve conduction was recorded from the sural, median, and ulnar nerves. H-response was obtained from the soleus muscle. H-reflex-wave conduction velocity was estimated by the formula described by Vecchierini-Blineau and Guiheneuc with reference to the subject's height: H-reflex conduction velocity (in m/s) = [height of subject (in cm) × 0.8/time interval H–M − 1 (in ms)] × 10. The normal value is above 50 m/s.

Needle electromyography was performed with concentric electrodes. The first EMG was performed between day 4 and day 12 after discontinuation of sedative drugs; serial electrophysiological studies were performed after 1 and 4 months in patients 1, 2, and 4.

**Muscle and Nerve Biopsy.** Muscle biopsy was performed in all patients between days 9 and 15 after discontinuation of sedative drugs. All muscle biopsies were analyzed by the same examiner (J.M.B.). Biopsy samples of the muscle vastus lateralis (patients 2 and 4) or gastrocnemius (patients 1 and 3) were immediately frozen in liquid nitrogen and cryostat sections were processed according to the following methods: hematoxylin-eosin, modified Gomori trichrome, periodic acid Schiff (PAS), oil red O, NADH-tetrazolium reductase, ATPase (pH 9.6, 4.4, and 4.2), succinic dehydrogenase, immunohistochemistry of desmin, dystrophin, and spectrin. Other muscle samples were fixed and paraffin sections were stained with hematoxylin-eosin, Masson

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**Table 1. Motor nerve conduction.**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Peroneal Amp. (mV)</th>
<th>CV (m/s)</th>
<th>Tibial Amp. (mV)</th>
<th>CV (m/s)</th>
<th>Ulnar Amp. (mV)</th>
<th>CV (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R</td>
<td>L</td>
<td>R</td>
<td>L</td>
<td>R</td>
<td>L</td>
</tr>
<tr>
<td>1</td>
<td>0.6</td>
<td>0.7</td>
<td>45</td>
<td>45</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>2</td>
<td>Abs</td>
<td>0.4</td>
<td>Abs</td>
<td>48</td>
<td>0.9</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Abs</td>
<td>0.7</td>
<td>Abs</td>
<td>43</td>
<td>1.2</td>
<td>0.8</td>
</tr>
<tr>
<td>4</td>
<td>0.5</td>
<td>Abs</td>
<td>43</td>
<td>Abs</td>
<td>0.6</td>
<td>1</td>
</tr>
</tbody>
</table>

Normal values: >2.5, >40, >2.5, >40, >10, >50

*Abs: absent.*
trichrome and immunohistochemistry of myelin basic protein, and neurofilaments. For electron microscopy, muscle specimens were fixed in 2.5% gluteraldehyde and postfixed in 1% osmium tetroxide. Thin sections were stained with uranyl acetate and lead citrate.

In patients 1 and 3, a sural nerve biopsy was also performed and examined with light and electron microscopy including morphometric analysis on semithin sections stained with paraphenylenediamine and viewed with a light microscope and a video camera connected to a computer (CAMON system6). Reference values have been calculated by morphometric analysis of the sural nerve of 5 adult patients in whom a neuropathic process could be excluded. The mean density of myelinated fibers was 14,005 ± 1659.

RESULTS

Electromyography/Nerve Conduction Study. Electrophysiological findings are provided in Tables 1–3. Motor conduction velocities were normal for the four limbs with a decrease in the compound motor action potential (CMAP) amplitudes. Sensory nerve conduction velocities were normal for sural and median nerves with normal distal latencies. Sensory nerve conduction velocities were normal, slightly decreased, or unobtainable for ulnar nerves. Sensory action potential amplitudes were normal or slightly decreased for sural nerves; they were decreased for median and ulnar nerves.

H-reflex wave on the soleus was obtained in 3 patients and absent in patient 1 (a subjacent neuropathy was suspected in this patient and confirmed by nerve biopsy).

Needle EMG revealed sharp waves and fibrillations in almost all muscles at the first EMG. Only a few small polyphasic motor unit potentials were detected in some of the muscles at the first stage of the quadriplegia. Short-duration and low amplitude polyphasic motor unit potentials with a tendency to early recruitment were observed when recovery began.

These electrophysiologic data suggest acute myopathy with associated axonal neuropathy in cases with decreased sensory action potential amplitudes. Decreased CMAP amplitudes are common in myopathy.22 Serial electrophysiological studies performed in patients 1, 2, and 4 showed progressive improvement of the recruitment patterns of voluntary contractions and an increase of the CMAP amplitudes with complete recovery of normal values for patient 1 and partial recovery for patients 2 and 4 after 4 months.

Muscle and Nerve Pathology. The four muscle biopsies varied considerably in fiber diameter, ranging from 7 to 74 µm (rarely 96 µm in patient 4). Typing

### Table 2. Sensory nerve conduction.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sural</th>
<th>Median</th>
<th>Ulnar</th>
<th>H-reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Amp. (µV)</td>
<td>CV (m/s)</td>
<td>Amp. (µV)</td>
<td>CV (m/s)</td>
</tr>
<tr>
<td>R</td>
<td>L</td>
<td>R</td>
<td>L</td>
<td>R</td>
</tr>
<tr>
<td>1</td>
<td>6.8</td>
<td>6.5</td>
<td>53</td>
<td>46</td>
</tr>
<tr>
<td>2</td>
<td>4.1</td>
<td>4.6</td>
<td>43</td>
<td>43</td>
</tr>
<tr>
<td>3</td>
<td>2.6</td>
<td>3.6</td>
<td>45</td>
<td>41</td>
</tr>
<tr>
<td>4</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Abs: absent.

### Table 3. EMG.

<table>
<thead>
<tr>
<th>Patient</th>
<th>First dorsal interosseous</th>
<th>Biceps brachialis</th>
<th>Tibialis anterior</th>
<th>Rectus femoris</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Spont. act.</td>
<td>MUAPs</td>
<td>Spont. act.</td>
<td>MUAPs</td>
</tr>
<tr>
<td>1</td>
<td>—</td>
<td>—</td>
<td>0</td>
<td>P, S</td>
</tr>
<tr>
<td>2</td>
<td>2+</td>
<td>NR</td>
<td>2+</td>
<td>P, S</td>
</tr>
<tr>
<td>3</td>
<td>2+</td>
<td>NR</td>
<td>2+</td>
<td>NR</td>
</tr>
<tr>
<td>4</td>
<td>2+</td>
<td>NR</td>
<td>2+</td>
<td>NR</td>
</tr>
</tbody>
</table>

P = polyphasic motor unit potentials; S = short duration or low amplitude motor unit potentials; NR = no MUAPs recruited; 0 = none; 1+ = persistent trains two areas; 2+ = three or more areas; 3+ = all areas.
of muscle fibers was impossible because of poor differentiation between type 1 and type 2 fibers, except in patient 4 who showed preferential atrophy of type 2 (A and B) fibers and type grouping of types 1, 2A, and 2B. Numerous scattered fibers showed necrotic changes at various stages of evolution (Fig. 5 A & B): loss of myofibrils with cloudy or granular appearance; floccular changes; invading myophages; and basophilic cells. Some small basophilic regenerating muscle cells contained large vesicular nuclei with prominent nucleoli. The necrotic changes might appear limited to the center or a segment of the fiber (axial and segmental necroses). The necrotic fibers had generally lost their desmin and glycogen contents as well as their sarcolemma membrane. In many muscle fibers, the lipid droplets were large and numerous. A few fibers contained one or multiple clear vacuoles, some of which appeared rimmed in patient 4. In the muscles of patients 2 and 3, the necrotic process was more pronounced, and almost all fibers showed prominent degenerative changes. Small lymphocytic infiltrates were infrequently encountered in patient 3. Endomyial sclerosis was minimal or absent. Muscular nerves appeared normal.

On electron microscopy, many muscle fibers showed typical necrotic or regenerative changes. In the less damaged fibers, the M-lines were generally absent and the thick myosin filaments were more or less depleted with loss of A-band density. The thin actin filaments remained well preserved, but the I-bands were frequently shortened with some sinuosities and disruptions of the Z-disks. In some places, the myofibrils appeared disorganized or joined side by side (Fig. 6). These myofibrillar changes and, in particular, the partial depletion of the thick filaments were less pronounced in patient 4. Patient 4 had scattered intermyofibrillar rectangular, not membrane-bound inclusions with lengths of 0.7–1.4 µm and a dense granular substructure, suggesting sarcoplasmic protein crystals (Fig. 7). Streamings, rods, and cytoplasmic bodies were not observed. Cisternae of the sarcoplasmic reticulum were frequently enlarged. Few proliferated triads and tubular aggregates were noted. Glycogen granules and lipid droplets were slightly increased in number. Mitochondria were generally enlarged and they accumulated in some places. The nuclei frequently contained enlarged nucleoli. The undulations of the plasma and basement membranes appeared somewhat increased. The capillaries had well-preserved general morphology. Both sural nerves showed a slightly reduced myelinated fiber density: 10,169/ mm² (73% of mean normal value) for patient 1, and 8690/ mm² (62% of mean normal value) for patient 3. Small and large myelinated fibers were equally affected. Thinned and thickened myelin sheaths, macrophages, and inflammatory lesions were not observed. In longitudinal sections, degenerating nerve fibers were absent suggesting an old or slowly progressive neuropathic process.

**DISCUSSION**

Neuromuscular problems are increasingly being recognized in critically ill patients. They usually present as a diffuse muscle weakness and they are often suspected of contributing to the difficulty in weaning from ventilators. Differential diagnosis of this condition includes the ACM, the "critical illness polyneuropathy," and the prolonged neuromuscular blockade resulting from an accumulation of paralyzing agents due to impaired renal function.

In the literature, patients with ACM typically present with a diffuse muscle weakness affecting both proximal and distal muscles of the four limbs; the
symptoms were often unnoticed until weaning from mechanical ventilation and withdrawal of sedative agents.

Many factors have been proposed to explain the development of acute myopathy: asthma, muscle relaxant drugs, mechanical ventilation, aminoglycoside therapy, β-adrenergic stimulants, theophylline, metabolic disturbances, hypoxia, and so forth.

Williams et al.35 observed that sympathomimetic amines and theophyllines are not primary agents in the development of the acute myopathy. Most investigators2–5,7,9–12,19,21–25,27,29–32,35 concur that large

FIGURE 5B. Histochemistry of ATPase demonstrates poor recognition of fiber types due to weak and uneven staining of muscle fibers, which are more or less atrophic. M. gastrocnemius: patient 1. Cryostat section; ATPase at pH 9.7 (bar = 50 µm).

FIGURE 6. Myofibrils appearing joined side by side, with strongly depleted thick filaments, absent M-lines and preservation of thin filaments and Z-disks. On right side, increased number of glycogen granules and some enlarged mitochondria between disorganized myofibrils showing irregular Z-disks. Muscle gastrocnemius: patient 1 (left) and 3 (right). Electron microscopy; uranyl acetate and lead citrate (bar = 0.5 µm).
doses of parenteral corticosteroids administered during the paralytic state induced by NMBAs or sedative drugs in patients suffering from asthma or any other pathology\textsuperscript{10,21} are responsible for the acute myopathy. It is now well established that the occurrence of acute myopathy is not related exclusively to a particular kind of corticosteroid\textsuperscript{23}; in the literature, we found cases of acute myopathy associated with the following corticosteroid drugs: methylprednisolone \textasciitilde 240 mg/day; hydrocortisone \textasciitilde 1 g/day; prednisone \textasciitilde 50 mg/day; and dexamethasone \textasciitilde 40 mg/day.\textsuperscript{19,21}

In contrast to previous publications, our observations suggest that the risk for occurrence of ACM still exists with doses as low as 60 mg/day of methylprednisolone over 5 days (patient 3).

Zochodne et al.\textsuperscript{36} described acute necrotizing myopathy in 3 patients treated by NMBAs (vecuronium) without receiving any corticosteroids during their ICU stay. They concluded that these observations argue against the suggestion that the myopathy arises from the superimposition of high doses of corticosteroids on paralyzed (functionally denervated) muscle, as suggested by the experiments of Massa et al.\textsuperscript{26}; that is, Zochodne’s group proposed a more direct myotoxic action of vecuronium or a metabolite. It is noteworthy that, among the Zochodne’s patients who did not receive corticosteroids, none underwent a muscle biopsy.

Recently, Gooch\textsuperscript{18} proposed that the long-term use of NMBAs, either alone or in combination with other medications or disorders, explained prolonged flaccid paralysis in 1 patient.

In contrast to the patients of Gooch\textsuperscript{18} and Zochodne et al.,\textsuperscript{36} and in contrast to most of the previous cases of acute myopathy, our 4 patients did not receive any NMBAs. Adaptation to mechanical ventilation was accomplished by hypnotic doses of sedative drugs (propofol). To our knowledge, until now, propofol has not been implicated in the occurrence of acute myopathy in the literature, but this possibility cannot be strictly excluded. Our patients all received high doses of glucocorticoids. Six other cases of ACM without association with NMBAs have already been described in the literature.\textsuperscript{16,21,23,29}

Our observation supports corticosteroids rather than NMBAs as the main offending drug in ACM. Reduced muscle activity seems to be a predisposing factor to corticosteroid toxicity. There are theoretical reasons why reduced muscular activity might predispose to corticosteroid-induced myopathy.\textsuperscript{7,11,30} In denervation or disuse atrophy, the number of cytoplasmic corticosteroid receptors has been shown to increase significantly.\textsuperscript{8,13–15}

In Figures 1–4, we see that the CK elevation occurs about 10 days after administering corticosteroids and sedation for 3 patients and after about 18 days for patient 1. This delay is compatible with the observations of Du Bois and Almon\textsuperscript{13} on immobilized rat muscles: after 8 days of immobilization, the

FIGURE 7. Less pronounced changes: partial depletion of the thick filaments and absence of the M-lines. Lipid droplets and one dense crystalloid inclusion. Muscle vastus lateralis: patient 4. Electron microscopy; uranyl acetate and lead citrate (bar = 0.5 µm).
number of glucocorticoid receptors significantly increased relative to control muscle.

Muscle biopsy is very important in the diagnosis of ACM. One of the strengths of this study is that all patients had muscle biopsies with electron microscopy.

In the muscle of patient 4, the preferential type 2 atrophy could be related to the long-lasting disuse and the type grouping demonstrated a chronic denervation-reinnervation process. The recognition of type 1 and 2 fibers was not possible in the muscle biopsies of the other 3 patients probably because of the loss of myosin and the extended degenerative and necrotic changes; the sural nerve biopsy of two of them (patients 1 and 3) showed signs of slight, prior, or slowly progressive neuropathy. It may be concluded that the neuropathy was preexistent and not related to the acute necrotizing myopathic process demonstrated in the 4 patients. It is unlikely this slight or moderate underlying polyneuropathy made the patients vulnerable to the acute myopathy.

Proliferation of both the T system and sarcoplasmic reticulum (SR) of the triads (so-called duplicated triads) are usually associated with fiber damage.\(^{28}\) Proliferation of the tubular SR (so-called tubular aggregates) may occur in a variety of disorders and could represent an adaptive response of the SR to various insults to the muscle fiber.\(^{17}\)

The present myopathy was characterized by thick filament loss with decreased A-band density and absence of M-lines. This selective depletion of myosin filaments with relative preservation of actin filaments and Z-disks was prominent in patients 1, 2, and 3. In the muscle of patient 4, which showed less pronounced degenerative changes, the A-band density was slightly decreased and the M-lines were absent or attenuated. In addition, this muscle contained intermyofibrillar inclusions resembling sarcoplasmic protein crystals. These crystalloid inclusions could represent deposits of the altered myosin molecules. Selective loss of thick filaments associated with widespread muscle fiber atrophy, with or without muscle fiber necrosis, is a prominent feature of the acute quadriplegic myopathy that affects patients who receive high doses of corticosteroids and, in addition, are also treated by muscle relaxants or have multiple medical problems with anoxia, hypercapnia, or acidosis.\(^{10,21,29,34}\)

Selective loss of thick myofilaments is a rare finding in human muscle; Rouleau et al.\(^ {29}\) found it in denervated skeletal muscles of rats treated with daily injections of dexamethasone in doses comparable with the average relative dose range used in human therapeutics. Perhaps the temporary functional de-

nervation caused by NMBAs also increases the number of glucocorticoid receptors. The same increase in the number of glucocorticoid receptors has been demonstrated by Du Bois and Almon\(^ {13}\) in disuse atrophy; this observation suggests that ACM may occur in immobilized muscles even in the absence of NMBAs.

However, the rise in CK levels in some of our patients were not as high as those of patients in other studies using NMBAs.\(^ {36}\) We may suppose that muscle immobilization is more complete with NMBAs than with propofol.

For an electrophysiologist, differential diagnosis between acute myopathy and ‘‘critical illness neuropathy’’ may be difficult.\(^ {9}\) Normal motor conduction velocities and decreased amplitude of motor action potentials are observed in both conditions. On the contrary, two electrophysiological features are of importance in identifying acute myopathy: the myopathic aspect of motor unit action potentials and the normality of sensory action potentials of distal nerves. Sensory nerve conduction was unobtainable for the ulnar nerve in 2 patients but we know that the ulnar nerve is very sensitive to entrapment neuropathy in bed-ridden patients.

Our neuropathological and electrophysiological results suggest that myopathy is the main factor explaining the severity of muscle weakness in this acute quadriplegia even if a mild axonal neuropathy is common in critically ill patients.\(^ {3-5}\)

The definition of ACM needs further specification: our patients had marked elevation of CK, and muscle biopsies incitated necrosis, but it is noteworthy that some of the 44 cases that we found in the literature indicated normal CK levels\(^ {7,21,23,25}\) or no mention of CK levels\(^ {30}\) and/or no necrosis on biopsy results.\(^ {2}\) From our point of view, CK elevation, myopathic abnormalities of EMG, and muscular necrosis at biopsy are necessary for the diagnosis of ACM. Because CK elevation is sometimes temporary, it might be undetected if samples are not made regularly, at least every 3 days. In terms of outcome, we learn from the literature that recovery is generally good but always long (1–8 months)\(^ {11,16,18,21,24,25,35}\) as in our 3 surviving patients.

In conclusion, if ‘‘critical illness neuropathy’’ is often incriminated in intensive care patients, we must keep in mind that ACM may also give rise to flaccid quadriplegia and difficulty in weaning the patient from ventilatory support, especially when the patient was treated with high doses of corticosteroids. In addition to the differential diagnosis by EMG, it is relevant to search for a peak of the CK in the previous days.
Our observations suggest that ACM may occur in muscles paralyzed by hypnotic doses of sedative agents (propofol) even in absence of NMBAs; in the absence of proven myotoxicity of propofol, our hypothesis is that hypersensitivity of resting muscles to corticosteroids is the predisposing factor.

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