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Case Presentation

Unusual Electromyographic Findings Associated With Colchicine Neuromyopathy: A Case Report

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Abstract

An 83-year-old man with multiple medical problems, including gout, pseudogout, and renal insufficiency, presented with more than a year of proximal weakness. He had an extensive previous medical workup, including a normal creatinine kinase. His weakness persisted despite endurance and strength training. Electrodiagnostic findings were consistent with a myopathy, although without abnormal spontaneous activity and a length-dependent neuropathy. On the basis of these findings, colchicine was discontinued. The patient experienced marked symptomatic improvement within a week. Myopathies with neuropathies may be found with the use of colchicine. This case was unusual because of the absence of abnormal spontaneous activity and increased creatinine kinase, as typically reported with colchicine myopathy.

Introduction

Colchicine is a commonly used drug for the treatment of gout. Its gastrointestinal toxicity is well known; however, its effects on the neuromuscular system are recognized less frequently. Myopathy is a reported complication with its use and may present in conjunction with a mild length-dependent neuropathy [1]. Although the myopathy generally is associated with increased levels of creatinine kinase (CK) and abnormal spontaneous activity on electromyographic testing, we report a case of colchicine neuromyopathy with atypical laboratory and electrodiagnostic findings.

Case Presentation

An 83-year-old man with a complicated medical history, including gout, pseudogout, renal insufficiency, polymyalgia rheumatica, asthma, deep-vein thrombosis, nephrolithiasis, Waldenstrom macroglobulinemia, bacterial overgrowth syndrome, vitamin B-12 deficiency, and temporal arteritis, presented to our outpatient musculoskeletal clinic with complaints of fatigue and difficulty rising from a chair. He had a 4-year history of weakness, which initially was attributed to polymyalgia rheumatica on the basis of symptoms and an increased sedimentation rate (ESR). He was treated with oral steroids and responded with an improvement in ESR. The following year, he developed weakness and severe hypotension, first thought to be attributable to adrenal insufficiency, then subsequently diagnosed as biopsy-proven temporal arteritis, again requiring treatment with steroids. Two years before his electrodiagnostic study, he was diagnosed with gout and pseudogout and was started on colchicine 0.6 mg daily. Because of recurrent knee effusions, colchicine was increased to twice daily approximately 1 year before presentation.

He then developed progressive weakness and was evaluated by a neurologist, who diagnosed him with probable steroid myopathy and discontinued his steroids. ESR was stable, CK was 65, and creatinine was 1.6 at that time. His weakness persisted despite steroid discontinuation, and he subsequently was referred for physical therapy. Although he participated in resistance and endurance training, he showed no improvement in strength or subjective symptoms. He required push off with his arms to transition from sit to stand and began ambulating with a cane.

At presentation to our outpatient clinic, the patient reported that his movements were slow and he was

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easily fatigued. Functional deficits at presentation included difficulty with opening a water bottle, buttoning shirts, and rising from a chair. He denied numbness or tingling in the limbs. Medications at that time included lansoprazole, levothyroxine, allopurinol, amiloride, furosemide, and colchicine at a dose of 0.6 mg twice daily for gout/pseudogout.

Findings of the physical examination were notable for periscapular, biceps, triceps, and gluteal muscle atrophy and difficulty rising from a chair or low surfaces. Muscle stretch reflexes were 1+ at the bilateral biceps, triceps, and patellae. Sensory examination demonstrated no reported abnormalities with light touch or pinprick testing in dermatomes C5-T1 or L2-S1 bilaterally. The patient was referred for electrodiagnostic testing, with formal manual muscle testing performed by physical therapy shortly before electrodiagnostic examination demonstrating proximal weakness: left hip extension/abduction/external rotation was 3+/5 whereas on the right, these muscle groups were 4/5. Left and right hip adduction/flexion/internal rotation, knee flexion/extension, and ankle dorsiflexion and plantarflexion were 5/5. Shoulder abduction could only be performed to 90°.

Electrodiagnostic studies showed evidence of a chronic length-dependent sensory-motor polyneuropathy with axonal greater than demyelinative features. The right sural and superficial peroneal sensory responses were absent, peroneal motor amplitudes were decreased, and tibial motor nerve distal latency was prolonged with slowed conduction through the leg. Right tibial nerve F wave minimal latencies were prolonged. Abnormal temporal dispersion was not noted. In the upper limb, recording of the radial nerve at the proximal thumb was present; however, no response was obtained with a more distal sensory recording at the digital nerves at the thumb. Median and ulnar sensory distal latencies were prolonged. Needle electromyography showed distal lower limb findings of large-amplitude, long-duration motor unit potentials with reduced recruitment, consistent with a neuropathic axonal process with reinnervation. Conversely, low-amplitude, polyphasic motor unit potentials with early recruitment were noted only in the proximal limb muscles. No resting positive waves or fibrillation potentials were noted (Figure 1 and Tables 1-3).

On the basis of the findings of neuropathy with a myopathy, the diagnosis of colchicine neuromyopathy was considered. Colchicine was discontinued after electrodiagnostics, after discussion with the patient's rheumatologist. The patient reported striking improvement in symptoms within week of medication discontinuation. By 2 weeks, he reported he was not using his cane continuously, and by 3 weeks he had discontinued using his cane indoors. Follow-up at 3 months revealed marked improvement in strength, endurance, and mobility.

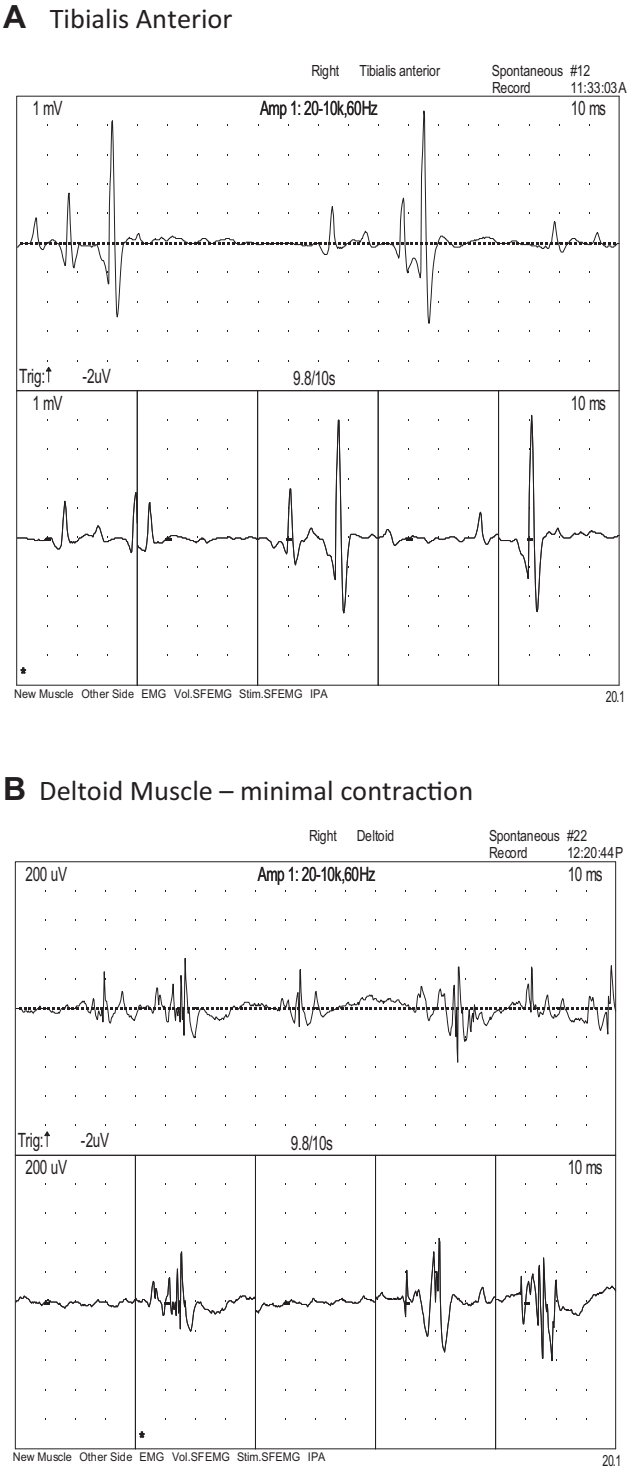

Figure 1. Voluntary motor unit potentials in the tibialis anterior (A) and deltoid (B) muscles. Demonstrated are large-duration voluntary motor unit potentials in the tibialis anterior muscle, and lower amplitude, polyphasic motor unit potentials in the deltoid, the latter recorded with minimal contraction. Note that recordings were captured with different sensitivity settings — 1 millivolt per division in the tibialis anterior, and 200 microvolts per division in the deltoid muscle.