# LETTERS TO THE EDITOR

## COLCHICINE-INDUCED MYOPATHY WITH MYOTONIA

We have read with interest the article by Rutkove and colleagues[4] on myotonia in colchicine myoneuropathy. Recently we have seen a patient who developed a myopathy with myotonic discharges on electromyography (EMG) while taking colchicine.

A 48-year-old man with a history of renal insufficiency (serum creatinine of 3.1 md/dL) and arterial hypertension had been started on colchicine 1 mg per day 2 years earlier. He was referred to outpatient clinic complaining of asthenia, myalgias, and progressive predominantly proximal weakness of all extremities. He had no sensory symptoms, bowel or bladder difficulties, stiffness, or slow relaxation of hand grip. There was no family history of neurologic disease.

General physical examination was normal. Neurological examination demostrated an alert patient with normal cranial nerves except for slight weakness of neck flexors. Muscle power was 4−/5 proximally and 4/5 distally in both arms. Although iliopsoas showed 4−/5 weakness bilaterally, the rest of the muscle groups were normal in the legs. Neither percussion nor grip myotonia was present. Reflexes in all extremities were diminished. All modalities of sensation and cerebellar functions were normal.

Serum creatine kinase (CK) was elevated to 1.136 IU/L (3% MB fraction). Serum creatinine was 3.1 mg.dL, and lactate dehydrogenase was 360 IU/L (normal values: 160–230).

Electromyography revealed abundant, widespread myotonic discharges in all muscles examined. Sporadic fibrillation potentials were observed in the tibialis anterior. Motor unit potentials were generally of small amplitude and short duration, showing increased polyphasia and early recruitment. Nerve conduction studies revealed a slight reduction of motor and sensory action potentials in both lower extremities.

Muscle biopsy revealed a moderate increase of internal nuclei and neurogenic group atrophy and fiber grouping on histochemical reactions. Nerve biopsy demonstrated reduced density of large myelinated fibers.

With the diagnosis of probable myoneuropathy secondary to colchicine, the drug was discontinued. Two weeks later the patient had improved muscle power, and lower serum CK levels as well as the rest of the muscle enzymes. At 3-month follow-up, the patient was asymptomatic, and neurological examination was normal, and so were the serum CK and the rest of the muscle enzymes. EMG performed at this stage detected no myotonic discharges, and the myopathic changes were less marked than in the first study.

It is well known that colchicine can produce features of a myopathy[2] or neuropathy[3] or both in patients with renal insufficiency[5]. Myotonia is a hallmark of several neuromuscular system diseases and is also seen as a reaction to several drugs and chemicals[1]. To date, colchicine has been implicated to cause myotonia in only 4 patients. We present another case in whom colchicine myopathy was accompanied by myotonic discharges on EMG. We stress the importance of recognizing myotonia as a feature of toxic myopathy induced by colchicine.

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