Neurology Asia 2026; 31(2) : 833 – 835

Reversible myotonic myopathy induced by colchicine: A rare mimic of myotonic dystrophy

Seyma Aykac, Esra Asıkdogan, Fikret Bademkıran, Ibrahim Aydogdu

Ege University Faculty Of Medicine, Neurology Department, Izmir, Turkey

Abstract

Colchicine is widely used in the treatment of familial Mediterranean fever (FMF) and other inflammatory disorders and is generally well tolerated. However, neuromuscular toxicity may rarely occur. Although colchicine-induced myopathy has been increasingly recognized, the coexistence of clinical myotonia with electrophysiologically documented myotonic discharges remains exceptionally uncommon. We report a 45-year-old woman with FMF who developed diffuse myalgia and progressive gait disturbance over five days following an increase in colchicine dosage to 3 mg/day. Neurological examination revealed proximal muscle weakness and percussion myotonia. Laboratory studies showed elevated creatine kinase and hepatic transaminase levels, with preserved renal function. Muscle magnetic resonance imaging was normal, myositis-specific antibodies were negative, and nerve conduction studies were unremarkable. Needle electromyography demonstrated myogenic motor unit potentials with prominent myotonic discharges, predominantly in proximal lower-limb muscles. Colchicine was promptly discontinued, and intravenous hydration was initiated. Within one week, liver enzyme levels normalized, clinical myotonia resolved, and repeat electromyography showed complete disappearance of myotonic discharges, although mild myopathic features persisted. The absence of multisystem involvement, family history, and the rapid clinical and electrophysiological recovery after drug withdrawal strongly argued against a hereditary myotonic disorder and supported a diagnosis of colchicine-induced myopathy with transient myotonia. This case highlights a rare but reversible manifestation of colchicine-related neuromuscular toxicity and emphasizes the importance of considering a pharmacological etiology in patients presenting with acute or subacute proximal weakness and myotonia. Early recognition and prompt withdrawal of colchicine are essential, as clinical and electrophysiological recovery is typically complete.

Keywords: Colchicine-induced myopathy, clinical myotonia, myotonic discharges, drug-induced myopathy, neuromuscular toxicity

INTRODUCTION

Colchicine is widely used in the treatment of familial Mediterranean fever (FMF) and other inflammatory disorders and is generally well tolerated; however, neuromuscular toxicity may rarely occur. Colchicine-induced myopathy has been increasingly recognized, yet the coexistence of clinical myotonia with electrophysiological myotonic discharges remains exceptionally uncommon. Awareness of this presentation is important, as prompt discontinuation of the drug typically results in full recovery. Here, we report a relatively young FMF patient who developed reversible myopathy accompanied by both clinical and electrophysiological myotonia following colchicine dose escalation, highlighting an underrecognized manifestation of colchicine toxicity.

CASE REPORT

A 45-year-old woman presented with diffuse myalgia and progressive gait disturbance over five days. Her medical history included FMF, hypothyroidism, and ischemic stroke. She was receiving acetylsalicylic acid, clopidogrel, levothyroxine, and colchicine. Although she had taken colchicine for two years without adverse effects, the dose had recently been increased to 3

Address correspondence to: Seyma Aykac; Medical Doctor, Neurologist and Neurophysiologist, Ege University Faculty Of Medicine, Neurology Department, Izmir, Turkey. Email: symaykac@gmail.com

Date of Submission: 14 December 2025; Date of Acceptance: 4 January 2026

https://doi.org/10.54029/2026wni

mg/day, exceeding the commonly recommended therapeutic range of 1–2.5 mg/day for FMF. Shortly thereafter, she developed transient diarrhea followed by progressive proximal muscle weakness. Neurological examination revealed proximal weakness (Medical Research Council grade 3/5 in the lower limbs, 4/5 in the upper limbs, and 3/5 in neck flexion) and percussion myotonia in the thenar muscles. Laboratory studies showed elevated AST (268 U/L), ALT (293 U/L), and creatine kinase (588 U/L), with preserved renal function. Muscle MRI of the shoulders and thighs was normal, and myositis antibodies were negative. Nerve conduction studies and late responses were unremarkable. Needle electromyography demonstrated myogenic motor unit potentials with prominent myotonic discharges, mainly in proximal lower-limb muscles. Following colchicine discontinuation and intravenous hydration, liver enzymes normalized within one week, clinical myotonia resolved, and repeat EMG showed complete disappearance of myotonic discharges, although mild myopathic features persisted. Overall, the clinical, biochemical, imaging, and electrophysiological findings supported the diagnosis of colchicine-induced myopathy with both clinical and electrophysiological myotonia.

DISCUSSION

This case expands the spectrum of colchicine-induced neuromuscular toxicity by documenting the rare coexistence of clinical myotonia and EMG-confirmed myotonic discharges. Colchicine-related myopathy, and less commonly neuropathy, typically occurs in older individuals or in those with renal or hepatic dysfunction or interacting medications.[1] In contrast, our patient was relatively young, had preserved organ function, and no recognized drug interactions, yet developed reversible myopathy and myotonia following a dose increase. This observation underscores that colchicine toxicity may occur even in the absence of traditional risk factors.

From a differential diagnostic perspective, the combination of proximal weakness, myalgia, and myotonia in an adult patient closely resembles myotonic dystrophies, particularly types 1 and 2. These autosomal dominant multisystem disorders are characterized by slowly progressive muscle weakness with myotonia and frequent extramuscular manifestations, including early-onset cataracts, cardiac conduction abnormalities, and endocrine dysfunction.[2] Electromyography in myotonic dystrophies typically demonstrates widespread myotonic discharges with chronic myopathic changes, while serum creatine kinase levels are normal or only mildly elevated.[2] In our patient, the absence of a family history, lack of multisystem involvement, the subacute onset shortly after colchicine dose escalation, and the rapid clinical and electrophysiological recovery after drug withdrawal strongly argue against an underlying hereditary myotonic dystrophy. Instead, these features support a toxic, reversible myopathy with transient muscle membrane instability. Muscle biopsy was not pursued, as the diagnosis was sufficiently supported by the clinical, biochemical, and electrophysiological findings together with prompt reversibility after cessation of colchicine.

Mechanistically, colchicine disrupts microtubule-dependent intracellular transport, leading to myofibrillar disorganization and vacuolar degeneration in skeletal muscle.[1] Clinically, patients typically present with subacute proximal weakness and elevated creatine kinase or hepatic transaminases.[1] Muscle imaging is often normal, and when biopsied, vacuolar myopathy with accumulation of autophagic material is usually observed.[1]

Electrophysiologically, colchicine-induced myopathy is characterized by myogenic motor unit potentials; however, overt clinical myotonia and myotonic discharges have been reported only rarely.[3–5] The complete resolution of both clinical myotonia and EMG abnormalities after colchicine discontinuation in our patient supports a transient, drug-induced disturbance of muscle membrane excitability rather than a primary channelopathy or hereditary myotonic disorder.

In conclusion, colchicine-induced neuromuscular toxicity should be considered in patients presenting with acute or subacute proximal weakness, elevated creatine kinase, and normal muscle imaging, even in younger individuals with preserved organ function. In the presence of clinical myotonia accompanied by myotonic discharges on EMG, especially when reversibility is observed, a drug-related etiology such as colchicine toxicity should be taken into consideration. Early recognition and withdrawal of the offending drug are essential, as recovery is typically complete.

DISCLOSURE

Ethics: Informed consents have been obtained from the patients for this publication.

Financial support: None

Conflict of interest: None

REFERENCES

1. Fernández-Cuadros ME, Goizueta-San-Martin G, Varas-de-Dios B, *et al.* Colchicine-induced rhabdomyolysis: Clinical, biochemical, and neurophysiological features and review of the literature. *Clin Med Insights Arthritis Musculoskelet Disord* 2019;12:1179544119849883. doi:10.1592/phco.24.17.1784.52334.
2. Hahn C, Salajegheh MK. Myotonic disorders: A review article. *Iran J Neurol* 2016;15:46–53.
3. Caglar K, Safali M, Yavuz I, Odabaşi Z, Yenicesu M, Vural A. Colchicine-induced myopathy with normal creatine phosphokinase level in a renal transplant patient. *Nephron* 2002;92:922-4. doi:10.1159/000065439.
4. Rutkove SB, De Girolami U, Preston DC, *et al.* Myotonia in colchicine myoneuropathy. *Muscle Nerve* 1996;19:870-5. doi:10.1002/(SICI)1097-4598(199607)19:7<870::AID-MUS9>3.0.CO;2-6.
5. Duarte J, Cabezas C, Rodríguez F, Clavería LE, Palacin T. Colchicine-induced myopathy with myotonia. *Muscle Nerve* 1998;21:550-1. doi:10.1002/(sici)1097-4598(199804)21:4<550::aid-mus19>3.0.co;2-0.