

## Case Report

# Colchicine Induced Rhabdomyolysis and Acute Kidney Injury-Case Report

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## Abstract

Rhabdomyolysis, the rapid breakdown of skeletal muscle, is a rare manifestation of colchicine toxicity. We report a case of colchicine induced rhabdomyolysis and acute kidney injury with hepatic impairment. We expect that this case report adds to the existing literature on this subject.

## Keywords

- Acute kidney injury
- Colchicine
- Gut
- Rhabdomyolysis

## INTRODUCTION

Colchicine is an antiinflammatory agent in that it is effective against acute gouty arthritis. Moreover, it has a prophylactic agent for patients with chronic gout. But its therapeutic value is limited by its narrow therapeutic index. Nausea, vomiting, diarrhea and abdominal pain are the most common and earliest untoward effects of colchicine. Colchicine has also been reported to cause alopecia, agranulocytosis, muscular weakness. Furthermore, myopathy and neuropathy also have been noted with colchicine treatment [1].

Limited case reports of colchicine-induced rhabdomyolysis have been published [2-5]. This report discusses a case of colchicine-induced rhabdomyolysis in a patient with acute renal failure and elevated liver function tests.

## CASE REPORT

A 88-year-old man was admitted to the hospital due to persistent diarrhea, vomiting, and diffuse weakness. Past medical history included hypertension, cerebrovascular occlusion and gout. Approximately one month prior to admission, he had been started on colchicine and allopurinol. Physical examination revealed dry and pale skin. His pulse rate were 65 beats/minute and blood pressure were 130/85mm Hg. Laboratory workup showed elevated serum creatinine of 2.74 mg/dL and blood urea nitrogen of 141mg/dL, elevated serum ALT concentration of 131 IU/L, AST concentration of 155 IU/L, and direct bilirubin of 0.16 mg/dL, elevated serum creatine kinase (CK) of 5050 IU/L and CK-MB of 99 IU/L but with normal Troponin-I. Electrolytes including sodium 138 mmol/L, potassium 3.6 mmol/L were within normal limits. His urine was bloody in appearance and urine analysis showed blood reaction with dipstick test, but there were no erythrocytes on microscopic examination. Colchicine was withdrawn and ace inhibitor drug was stopped because of the renal impairment. Ultrasound scan of the kidneys

was normal. Rhabdomyolysis was diagnosed on clinical and biochemical grounds. Neurophysiologic study showed a pattern of severe myopathy with axonal sensorimotor neuropathy. Intravenous infusion with normal saline 2500mL per day was administered. Thereafter, progressive normalization of CK and renal function results were observed. In view of his clinical presentations of elevated CK, impaired renal function and proximal weakness, colchicine was thought to be the causative factor for rhabdomyolysis in conjunction with acute renal failure and elevated liver function tests because of onset of symptom temporal relationship with medication, improvement following colchicine discontinuation and the lack of any alternative explanations for myotoxicity. Fourteen days after colchicine withdrawal, his muscle power had returned to normal and he was mobilising independently. He was discharged home with instructions to continue his clinical follow-up with his primary care physician within two weeks.

## DISCUSSION

Rhabdomyolysis is defined acute and massive muscle fiber necrosis accompanied by the release of muscle-related metabolites into the bloodstream [6]. Investigation confirmed the diagnosis of rhabdomyolysis, and discontinuation of colchicine resulted in resolution of clinical and biochemical features of rhabdomyolysis. Neuromuscular adverse effects of colchicine in the form of myoneuropathy and myotonia are well recognised [7,8]. Colchicine-induced rhabdomyolysis is a rare complication and only few cases reported in literature [2-5].

Patients with impaired renal function appear to be at a higher risk for colchicine induced neuromuscular adverse effects [8]. As in our cases, acute renal failure in conjunction with elevated liver function tests appear to increase the possibility of colchicine-induced toxicity, specifically, rhabdomyolysis.

The dosage and duration of colchicine ingestion does not

seem to be clearly related to the risk of developing muscle toxicity [8]. On an average most patients reported with myopathy consumed 1mg or more of the drug for more than six months [7,8]. Our patient had consumed 1mg of the drug daily for four weeks, similar to a previously described case of rhabdomyolysis.

This case highlights an unusual but important-to-note complication of colchicine use in old-aged patient. Like in our cases, the most common complication is gastroenteritis and this may be predispose to prerenal acute renal failure. Moreover, renal function impairment increases colchicine toxicity because of narrow drug therapeutic index. Also, drug induced liver dysfunction and neuromuscular toxicity can be seen according to drug toxicity. When colchicine is used in conjunction with renal and/or liver impairment, this may cause vicious cycle.

## CONCLUSION

Our report emphasize that a rare but serious and potentially life threatening neuromuscular adverse effect of colchicine. Since renal impairment is a predisposing factor for neuromuscular toxicity, cautious dosing of colchicine is warranted in the presence renal insufficiency. Hence any patient on the drug should be carefully monitored for symptoms of myotoxicity with measurement of creatine kinase when clinically indicated.

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