International Journal of Research and Review
Volume 11; Issue: 5; May 2024
Website: www.ijrrjournal.com
Case Report *E-ISSN: 2349-9788; P-ISSN: 2454-2237*

A Complex Case of Colchicine-Induced Myopathy and Polyarticular Gout in a Patient with Chronic Kidney Disease

Ananthan J[1], Nihal Muhammed[1], Shaju S Dharan[2], Dhanya Dharman[3]

[1]Pharm D Intern, Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram, Kerala
[2]Principal/HOD, Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram, Kerala
[3]Associate Professor, Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram, Kerala

Corresponding author: Dhanya Dharman

DOI: https://doi.org/10.52403/ijrr.20240519

ABSTRACT

Colchicine, a medication commonly used for the treatment of gout and other inflammatory conditions, is known to cause various adverse effects, including myopathy. We present a case of colchicine-induced myopathy in a 58-year-old male with a history of gout. The patient developed progressive muscle weakness after initiating colchicine therapy. Prompt discontinuation of colchicine resulted in resolution of symptoms and normalization of muscle enzyme levels. This case underscores the importance of recognizing colchicine-induced myopathy and prompt discontinuation of the medication to prevent further complications and report also highlights the challenges and considerations in managing a patient with multiple comorbidities and the importance of close follow-up and ongoing monitoring.

Keywords: Colchicine, Myopathy, Gout, Adverse effects, Muscle weakness

INTRODUCTION

Colchicine is derived from the corms of Colchicum autumnale and used therapeutically for the treatment of various diseases such as gout, familial Mediterranean fever (FMF), and Behcet's disease for a long time. [1-3] Despite its efficacy, colchicine is associated with various adverse effects, including gastrointestinal symptoms, myelosuppression, and myopathy. Colchicine-induced myopathy is a rare but potentially serious adverse effect characterized by muscle weakness and elevated creatine kinase levels. The onset of colchicine-induced myopathy is commonly related to chronic renal failure or associated with the use of high doses and other myotoxic medications [4]. However, some cases develop colchicine myopathy without statin or renal insufficiency, which raises the possibility of genetic predisposition [5].

Colchicine is known for its anti-inflammatory properties, which are primarily mediated through the inhibition of microtubule assembly. Microtubules are essential components of thecytoskeleton and play a crucial role in muscle cell structure and function. Inhibition of microtubule assembly by colchicine disrupts muscle cell function, leading to myopathy.

CASE REPORT

A 58 years old male patient was admitted

under Nephrology department in a tertiary care hospital presenting with complaints of bilateral lower limb weakness for 1 week. The patient had medical history of Polyarticular Gout, Chronic Kidney Disease, Hyperurecemia, Hypertension, and Dyslipidemia. On examination the patient was afebrile, conscious and oriented. The patient medication history include T.Amlodipine 5mg, T.Sodium Bicarbonate 500mg, T.Sevelamir 400mg, T.Febuxostat 40mg, T.Atorvastatin 10mg for the above mentioned comorbidities. The patient had been prescribed Colchicine 0.5mg BD. Given the temporal association between the onset of symptoms and initiation of colchicine therapy, a diagnosis of colchicine-induced myopathy was suspected. Colchicine was promptly discontinued, and the patient was started on supportive therapy. Among the laboratory tests performed Urea, Serum creatinine and CPK was elevated. Nerve conduction study showed sensory neuropathy affecting both lower limbs. The patient was treated under the expert guidance of Rheumatologist and Nephrologist at the tertiary care hospital. The condition was managed with T. Sodium Bicarbonate 500mg, T. Amlodipine, T. Methylprednisolone 4mg, Folicacid 5mg, T. Sevelamir 400mg, T. Sulfalazine 500mg, T. Febuxostat 40mg and T. Atorvastatin 10mg.Over the course in the hospital stay, patient improved symptomatically, vitals stable anddischarged with T. Amlodipine, T. Methylprednisolone 4mg, Folic acid 5mg, T. Sevelamir 400mg, T. Febuxostat 40mg and T. Atorvastatin 10mg, T. Sodium Bicarbonate 500mg, T. Sulfalazine 500mg for 1 week.

DISCUSSION

Colchicine-induced myopathy is a rare but potentially severe adverse effect of colchicine therapy. The exact pathogenesis of colchicine-induced myopathy is not fully understood but is thought to involve disruption of microtubule function in muscle cells, leading to impaired cellular transport and eventual muscle fiber necrosis. Colchicine-associated myopathy risk increased with the presence of other co-morbid conditions like chronic renal failure, hepatic failure, coadministration of drugs like simvastatin, tacrolimus, cyclosporine, erythromycin, and antifungal medications [5-7]. Colchicine is a known tubulo-toxin because it inhibits microtubule polymerization by binding to the α and β monomers of tubulin. The mechanism by which colchicine causes myopathy is largely unknown. However, it is known for causing painless vacuole myopathy, affecting the microtubular network, and transport causes vacuolation[8,9].

The clinical presentation of colchicine-induced myopathy typically includes proximal muscle weakness, myalgias, and elevated serum CK levels. Electromyography and muscle biopsy may demonstrate myopathic changes and evidence of muscle fiber necrosis, respectively, aiding in the diagnosis.

Management of colchicine-induced myopathy involves prompt discontinuation of colchicine therapy and supportive measures, including physical rehabilitation. Most patients experience gradual improvement in symptoms and normalization of serum CK levels following cessation of colchicine.

The risk factors for developing colchicine-induced myopathy are not well understood. Some studies have suggested that advanced age, renal impairment, and higher doses of colchicine may be risk factors.

CONCLUSION

Colchicine-induced myopathy is a rare but important adverse effect to consider in patients receiving colchicine therapy, particularly those presenting with muscle weakness and elevated serum CK levels. Prompt recognition and discontinuation of colchicine are essential to prevent further complications and facilitate recovery. Healthcare providers should be vigilant for the development of myopathy in patients receiving colchicine therapy and consider alternative treatment options when

appropriate. The report underscores the importance of a multidisciplinary approach, close follow-up, and ongoing monitoring in such cases. It also provides insights into the challenges and considerations in the diagnosis and management of patients with multiple comorbidities

Declaration by Authors

***Declaration Of Patient Consent*:** Informed consent was obtained from the patient and his physician.

Acknowledgement: We would like to thank Prof. (Dr) Shaiju S Dharan, Principal Ezhuthachan College of pharmaceutical sciences and Dr Dhanya Dharman, Associate professor, Department of pharmacy practice, Ezhuthachan college of Pharmaceutical Sciences for their expertise and assistance throughout all the aspects of our case study and for the help in writing themanuscript.

Source of Funding: None

Conflict of Interest: The authors declare no conflict of interest.

REFERENCE

1. Kwon OC, Hong S, Ghang B, Kim YG, Lee CK, Yoo B. Risk of colchicine-associated myopathy in gout: influence of concomitant use of statin.*Am J Med.* 2017; 130:583–587.
2. Rutkove SB, De Girolami U, Preston DC, et al. Myotonia in colchicine myoneuropathy. *Muscle Nerve.* 1996; 19:870–875.
3. Kuncl RW, Duncan G, Watson D, Alderson K, Rogawski MA, Peper M. Colchicine myopathy and neuropathy. *N Engl J Med.* 1987; 316:1562–1568.
4. Huh K, Joung JY, Jeong H, Je D, Cho YY, Jang HR, Huh W. Colchicine-induced myoneuropathy in a cyclosporine-treated renal transplant recipient. *Kidney Res Clin Pract.* 2013; 32:74–77
5. van der Velden W, Huussen J, Ter Laak H, de Sévaux R. Colchicine-induced neuromyopathy in a patient with chronic renal failure: the role ofclarithromycin.
6. Wallace SL, Singer JZ, Duncan GJ, Wigley FM, Kuncl RW. Renal function predicts colchicine toxicity: guidelines for the prophylactic use of colchicine in gout. J Rheumatol. 1991 Feb;18(2):264-9. PMID: 2023222.
7. Riggs JE, Schochet SS Jr, Gutmann L, Crosby TW, DiBartolomeo AG. Chronic human colchicine neuropathy and myopathy. Arch Neurol. 1986 May;43(5):521-3. doi: 10.1001/archneur.1986.00520050091033. PMID: 3964122.
8. Eleftheriou G, Bacis G, Fiocchi R, Sebastiano R. Colchicine-induced toxicity in a heart transplant patient with chronic renal failure. Clin Toxicol (Phila). 2008 Nov;46(9):827-30. doi: 10.1080/15563650701779703. PMID: 18608282.
9. Lee KY, Kim DY, Chang JY, Bang D. Two cases of acute leukopenia induced by colchicine with concurrent immunosuppressants use in Behçet's disease. Yonsei Med J. 2008 Feb 29;49(1):171-3. doi: 10.3349/ymj.2008.49.1.171. PMID: 18306487; PMCID: PMC2615254.

How to cite this article: Ananthan J, Nihal Muhammed, Shaju S Dharan, Dhanya Dharman. A complex case of colchicine-induced myopathy and polyarticular gout in a patient with chronic kidney disease. *International Journal of Research and Review*. 2024; 11(5): 157-159. DOI: *10.52403/ijrr.20240519*

******