CASE REPORT AND LITERATURE REVIEW

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EXTREMELY HIGH-DOSE COLCHICINE INTOXICATION WITH NEUROLOGICAL COMPLICATIONS: A SURVIVAL CASE REPORT

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Abstract

Familial Mediterranean fever (FMF) is an autoinflammatory disease with autosomal recessive inheritance, associated with *mediterranean fever* gene mutation, characterized by recurrent episodes of fever, serositis, and arthritis. Colchicine is the gold standard treatment but has a narrow therapeutic index. A 19-year-old male with FMF presented with abdominal pain, weakness, nausea, and vomiting after accidental ingestion of approximately 100 colchicine tablets in a suicide attempt. He developed multi-organ dysfunction, including acute kidney injury (creatinine: 2.33 mg/dL), hepatotoxicity (aspartate aminotransferase: 678 U/L), rhabdomyolysis (creatine kinase: 9996 U/L), and severe pancytopenia (white blood cell count: 710/µL, platelet count: 15,000/µL). Neurological complications included decreased consciousness, apathetic speech, and ataxia. The patient was managed in the intensive care unit with aggressive supportive therapy including intravenous hydration, broad-spectrum antibiotics, and granulocyte colony-stimulating factor (filgrastim) for bone marrow suppression. After 4 days, pancytopenia resolved, and organ functions gradually improved. The patient made a complete recovery. This case demonstrates that survival is possible even after extremely high-dose colchicine ingestion with appropriate supportive care. Close monitoring and patient education are crucial in FMF management to prevent toxicity.

Keywords: Familial Mediterranean fever, colchicine intoxication, creatine kinase

Introduction

Familial Mediterranean fever (FMF) is an autoinflammatory disease with autosomal recessive inheritance that is prevalent among populations originating from the Mediterranean basin. It is associated with mutations in the *mediterranean fever (MEFV)* gene located on chromosome 16. The disease is characterized by recurrent episodes of fever, peritonitis, pleuritis, arthritis, and rarely pericarditis lasting 1-3 days (1). In the pathogenesis of FMF, dysfunction of the pyrin protein encoded by the *MEFV* gene results in abnormal activation of inflammasomes and increased production of interleukin-1 beta (2).

Colchicine is the gold standard in the treatment of FMF, effective in reducing the frequency and severity of attacks and preventing complications such as amyloidosis. However, colchicine has a narrow therapeutic index, and overdose can lead to gastrointestinal symptoms, hepatorenal dysfunction, bone marrow suppression, and neurological disorders (3,4).

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Case Report

A 19-year-old male patient with a known diagnosis of FMF presented to the emergency department with complaints of abdominal pain, generalized weakness, nausea, and vomiting persisting for three days. His medical history revealed that due to insufficient response to the standard colchicine dosage, he had been prescribed an imported colchicine preparation at a dosage of 2×1 tablets daily. However, the patient had been taking it at a dosage of 4×1 daily.

Physical examination revealed tenderness in all quadrants of the abdomen with guarding and rebound. Diagnostic imaging, including upright abdominal radiography and posterior-anterior chest radiography, showed no pathology. Abdominal computed tomography (CT) demonstrated jejunal loop dilatation up to 3.7 cm with air-fluid levels, but no signs of perforation were detected. Laboratory tests revealed C-reactive protein: 175 mg/L; creatinine: 2.33 mg/dL; aspartate aminotransferase: 678 U/L; alanine aminotransferase: 189 U/L; alkaline phosphatase: 401 U/L; gamma-glutamyl transferase: 30 U/L; amylase: 338 U/L; lipase: 183 U/L; creatine kinase: 9996 U/L; white blood cell count: 710/μL (0.71×10^3/μL); neutrophil count: 410/μL (0.41×10^3/μL); and platelet count: 15,000/μL (15×10^3/μL). The patient was admitted to the rheumatology service due to acute kidney injury, FMF attack, and elevated acute phase reactants.

During his hospital course, the patient's general condition deteriorated with the development of agitation, and he subsequently was admitted to having ingested approximately 100 tablets of imported colchicine in a suicidal attempt. He developed a decrease in the Glasgow Coma Scale score, apathetic speech, and ataxia. Cranial CT and diffusion-weighted magnetic resonance imaging showed no pathology. Central nervous system infection was excluded via lumbar puncture.

Due to clinical deterioration, the patient was transferred to the intensive care unit. Muscle strength was evaluated as 2/5 bilaterally in the upper and lower extremities. He developed pancytopenia secondary to bone marrow suppression, and filgrastim therapy was initiated. The pancytopenic state resolved after 4 days. With intravenous hydration, a broad-spectrum antibiotic therapy, and supportive care, renal and hepatic function improved, hematological parameters recovered, and the patient was transferred back to the rheumatology service.

Discussion

The clinical course of colchicine intoxication can be examined in three phases as described in the literature: gastrointestinal symptoms in the first 24 hours, multi-organ failure between 24 to 72 hours, and rebound leukocytosis, or bone marrow aplasia after 72 hours (5). This classic triphasic course was observed in our case.

Bismuth et al. (6) described a case of a 23-year-old FMF patient who developed hepatorenal failure and myelosuppression following ingestion of 60 colchicine tablets. Similar to the symptoms observed in our patient, gastrointestinal symptoms were predominant in the early phase, followed by the development of multi-organ dysfunction.

In a series reported by Finkelstein et al. (7), among 12 FMF patients with colchicine intoxication, pancytopenia developed in 83%, hepatic dysfunction in 75%, and acute kidney injury in 58%. Rhabdomyolysis is reported to be common in cases of colchicine toxicity. This study supports the view that the clinical presentation observed in our case is consistent with the typical course of colchicine intoxication.

Myelosuppression is one of the most serious complications of colchicine toxicity and has been closely associated with mortality in the literature. Critchley et al. (8) demonstrated that granulocyte colony-stimulating factor (G-CSF) therapy in patients with colchicine intoxication shortened the neutrophil recovery time by an average of 4 days and reduced infectious complications. The successful management of bone marrow suppression with filgrastim (G-CSF) therapy in our case is consistent with these findings.

In a case presented by Altiparmak et al. (9), neurological complications (mental status changes, ataxia) were described in a patient who ingested colchicine exceeding 0.5 mg/kg. Similarly, in our case, neurological findings such as decreased Glasgow Coma Scale, apathetic speech, and ataxia, were observed, although no central nervous system pathology was detected on radiological imaging.

In a study by Zhong et al. (10), colchicine toxicity was reported to have a more severe course in patients with pre-existing renal dysfunction, with a mortality rate reaching 16.7%. In our case, although acute kidney injury developed, renal function improved with early and aggressive hydration therapy.

Key Messages

- The consumption of more than 100 colchicine tablets results in serious neurological and hematological complications.
- G-CSF shows promise as a treatment for bone marrow suppression caused by colchicine.
- The patient needs proper education and biochemical monitoring before increasing the dose in FMF.

Conclusion

The case highlights the need for continuous patient monitoring and proper education about colchicine use for FMF treatment. Patient education about proper dosage and regular clinical check-ups helps decrease the chance of toxicity. The management of colchicine overdose requires a multidisciplinary approach together with early diagnosis and aggressive supportive therapy to minimize both mortality and morbidity.

Ethics

Informed Consent: Written informed consent was obtained from the patient.

Footnotes

Authorship Contributions

Surgical and Medical Practices: O.Y., F.A., O.Z., Concept: O.Y., F.A., O.Z., Design: O.Y., F.A., O.Z., Data Collection or Processing: O.Y., F.A., O.Z., Analysis or Interpretation: O.Y., F.A., O.Z., Literature Search: O.Y., F.A., O.Z., Writing: O.Y., F.A., O.Z.

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