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Colchicine-Associated Hypoglycemia in Diabetic Dialysis Patients: Observations from a Tanzanian Dialysis Unit: A Case Reports

Huda Akrabi[1,2], **Datius Mutalemwa**[2], **Kajiru Kilonzo**[1,2] **and Abel Mwanga**[2]

[1]Department of Medicine, KCMC University, Moshi, Tanzania

[2]Department of Internal Medicine Department, Kilimanjaro Christian Medical Centre, Moshi, Tanzania

[*]**Corresponding author:** Huda Akrabi, Department of Medicine, KCMC University, Moshi, Tanzania, E-mail: drakrabi@gmail.com

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Abstract

Background: Colchicine is widely used for the treatment of acute gout flares due to its anti-inflammatory properties. Beyond this traditional role, colchicine has been implicated in modulating various physiological systems, including glucose metabolism. Notably, it exhibits a biphasic effect on blood glucose levels causing hypoglycemia at lower doses and hyperglycemia at higher doses. However, its impact on glycemic control in diabetic patients undergoing dialysis remains poorly characterized.

Case report: We report two cases from the Dialysis Unit at Kilimanjaro Christian Medical Centre (KCMC) in northern Tanzania involving two Black African diabetic males aged 66 and 62 years with End-Stage Renal Disease (ESRD) on maintenance hemodialysis. Both patients experienced a notable and reproducible decline in blood glucose levels following colchicine administration for gout flares. The hypoglycemia occurred in the absence of other identifiable precipitating factors and resolved upon withholding colchicine.

Discussion: These observations raise important questions regarding the potential glucose-lowering effects of colchicine in dialysis dependent diabetic patients. The exact mechanism remains unclear but may involve colchicine's influence on insulin sensitivity, hepatic glucose production, or inflammatory pathways that indirectly affect glucose metabolism. Given the already high risk of hypoglycemia in ESRD patients, these findings warrant careful consideration and further investigation.

Conclusion: Colchicine may have an under recognized hypoglycemic effect in diabetic patients undergoing dialysis. Clinicians should be alert to this potential interaction, particularly in patients with poor glycemic reserve. Further research is needed to elucidate the mechanisms involved and assess whether colchicine could be repurposed as an adjunctive agent for glycemic control in selected ESRD populations.

Keywords: Dialysis; Gout; Cholchicine; Hypoglycemia; Diabetes

Introduction

Colchicine, a microtubule inhibitor historically used in the management of acute gout flares, has garnered renewed attention for its diverse pharmacological effects. Beyond its anti-inflammatory role in gout, recent studies have highlighted colchicine's potential benefits in cardiovascular disease, including the prevention of atrial fibrillation post-cardiac surgery and reduction of adverse cardiovascular events following myocardial infarction [1,2,3]. These expanding indications have broadened colchicine's use across multiple patient populations, including those with significant comorbidities.

Of particular interest is colchicine's impact on glucose metabolism, an area that remains underexplored. Existing evidence suggests a biphasic glycemic response: lower doses may be associated with hypoglycemia, while higher doses can induce hyperglycemia [4,5]. The mechanisms underpinning these effects are not fully elucidated but are thought to involve modulation of inflammatory cytokines, oxidative stress and insulin signaling pathways [6]. This is especially relevant for patients with Chronic Kidney Disease (CKD) or End-Stage Renal Disease (ESRD), where glucose regulation is often unstable and small shifts in glucose homeostasis can lead to significant clinical consequences [7].

In dialysis-dependent diabetic patients, the potential for colchicine to influence glycemic control is clinically significant but poorly documented. Patients with ESRD often exhibit altered drug metabolism and are already at high risk for hypoglycemia due to reduced renal gluconeogenesis and frequent comorbidities [8]. Recognizing and understanding the impact of commonly used medications such as colchicine in this context is essential for optimizing care.

In this report, we describe two cases from the Kilimanjaro Christian Medical Centre (KCMC) Dialysis Unit in northern Tanzania, where colchicine administration coincided with a marked reduction in blood glucose levels in diabetic ESRD patients. These observations suggest a possible glycemic effect of

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colchicine in the dialysis population and highlight the need for further investigation into its role as a potential modulator of glucose metabolism in high-risk patients.

Moreover, in the era of expanding colchicine use including its investigation in inflammatory complications of COVID-19 understanding its broader metabolic effects is critical [9]. These cases underscore the importance of pharmacovigilance and personalized therapy, particularly in vulnerable populations such as diabetic patients on dialysis.

Case Series

Case 1

A 66-year-old Black African male with a background of End-Stage Renal Disease (ESRD) on intermittent hemodialysis for the past two years presented with a three-month history of progressively worsening pain in the right first Metatarsophalangeal (MTP) joint. The pain was described as severe, particularly nocturnal and aggravated by movement. There was no associated history of fever, trauma or systemic symptoms.

The patient had a history of type 2 diabetes mellitus, managed with subcutaneous insulin (40 IU in the morning and 30 IU in the evening) and well-controlled hypertension managed with dietary measures and regular hemodialysis. Dialysis was administered via a functioning left brachiocephalic arteriovenous fistula. He denied alcohol use or other known gout precipitants.

On examination, the patient was obese and mildly pale, with no signs of cyanosis, jaundice or lymphadenopathy. Vital signs were within normal limits: temperature 36.4°C, blood pressure 134/78 mmHg, heart rate 87 bpm. Capillary blood glucose was 11 mmol/L. Local examination of the right foot revealed swelling, warmth and tenderness over the first MTP joint, with restricted active and passive movement. No sensory deficits were noted. Other systemic examinations were unremarkable.

Laboratory investigations revealed a serum uric acid level of 472.71 µmol/L, glycated hemoglobin (HbA1c) of 8.51% and normal serum calcium and phosphate levels. Radiographic imaging of the affected joint demonstrated punched-out erosions with overhanging margins, consistent with tophaceous gout.

A diagnosis of gout was established. The patient was initiated on febuxostat 40 mg once daily and colchicine 250 mcg every other day for a two-week course. Notably, during this period, his capillary blood glucose levels remained well-controlled, ranging between 4.5 mmol/L and 6 mmol/L, without any changes in insulin regimen or dietary intake. However, after completion of colchicine therapy, he experienced worsening glycemic control, with blood glucose levels increasing to 12–16 mmol/L. This prompted titration of insulin doses and reinforcement of dietary modifications, leading to gradual normalization of blood glucose.

Case 2

A 62-year-old Black African male with a history of End-Stage Renal Disease (ESRD) on maintenance hemodialysis presented with a two-month history of progressively worsening sharp pain and stiffness in the left first Metatarsophalangeal (MTP) joint. There was no history of trauma, fever or systemic symptoms.

His ESRD had been managed with thrice-weekly hemodialysis for the past 2.5 years. Comorbid conditions included type 2 diabetes mellitus, treated with Sitagliptin 50 mg once daily and hypertension managed with Nifedipine 40 mg twice daily, Hydralazine 50 mg twice daily and Furosemide 80 mg twice daily. The patient denied alcohol consumption and had no previous diagnosis of gout.

On physical examination, he was alert and well-oriented, with no signs of pallor, cyanosis or jaundice. His vital signs were stable: temperature 36.6°C, blood pressure 144/74 mmHg, pulse rate 78 bpm. Capillary blood glucose was 5 mmol/L. Local examination of the left great toe revealed tenderness, warmth to palpation and a limited range of motion on both flexion and extension of the joint. There was no edema or other joint involvement. The remainder of the physical examination was unremarkable.

Laboratory workup showed a serum uric acid level of 272 µmol/L. Serum calcium, phosphate and Parathyroid Hormone (PTH) levels were within normal limits. Radiographic imaging of the affected foot demonstrated classic punched-out lesions at the first MTP joint, consistent with a diagnosis of gout.

The patient was initiated on colchicine 250 mcg every other day for a planned two-weeks. However, during this period, he began experiencing recurrent hypoglycemic episodes, with capillary blood glucose readings ranging between 2.0 and 4.0 mmol/L. There were no changes in his antidiabetic regimen, dietary intake or dialysis prescription. Following the discontinuation of colchicine, his blood glucose levels stabilized within the normal range without additional intervention.

Result and Discussion

Colchicine is a well-established anti-inflammatory agent used for the prevention and treatment of acute gout flares and other autoinflammatory conditions. However, its effects on glucose metabolism, particularly in patients with advanced renal dysfunction, have attracted growing interest due to its potential

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bidirectional glycemic influence and risk of adverse events.

Evidence suggests that low-dose colchicine (≤1.5 mg/day) may be associated with a reduced incidence of diabetes mellitus and hypoglycemia, potentially through anti-inflammatory mechanisms that improve insulin sensitivity [10,11]. In contrast, higher doses such as 2 mg/day over 10 days or a single bolus of 3 mg have been linked to impaired insulin secretion and hyperglycemia, possibly due to beta-cell toxicity or interference with glucose metabolism [12].

In the present report, all three patients with End-Stage Renal Disease (ESRD) undergoing hemodialysis were prescribed low-dose colchicine (250 mcg every other day) as prophylaxis for gout. The first case demonstrated improved glycemic control following initiation of colchicine and febuxostat, despite no changes in insulin regimen, suggesting a potential glycemia-lowering effect.

Conversely, the second and third patients both of whom had previously stable blood glucose developed recurrent hypoglycemic episodes during colchicine therapy, which resolved upon discontinuation of the drug. These divergent outcomes underscore the complex and individualized nature of colchicine's metabolic effects in diabetic patients on dialysis.

The variability in glycemic response may reflect differences in residual renal function, insulin sensitivity, concomitant medications and the inflammatory milieu. Given that colchicine is primarily metabolized by the liver and excreted by both biliary and renal routes, its pharmacokinetics are significantly altered in ESRD, increasing the risk of drug accumulation and toxicity. Reported adverse effects in such patients include gastrointestinal symptoms, neuromuscular toxicity, hypotension and central nervous system disturbances.

Although low-dose colchicine is generally considered safe in ESRD with appropriate dosing adjustments, these cases emphasize the need for heightened clinical vigilance. Close monitoring of blood glucose and individualized risk-benefit assessment are essential, particularly in diabetic patients receiving insulin or oral hypoglycemic agents.

In summary, this case series highlights the potential dual effects of colchicine on glucose metabolism in dialysis-dependent patients with diabetes mellitus. While some patients may benefit from improved glycemic control, others may be at risk of serious hypoglycemia. Further studies are warranted to better define the underlying mechanisms, identify predictors of glycemic response and establish safe prescribing practices for colchicine in this vulnerable population.

Conclusion

These cases underscore the delicate balance required in managing patients with coexisting gout, diabetes mellitus and End-Stage Renal Disease (ESRD). While colchicine remains an effective agent for preventing acute gout flares, its use in patients with advanced renal dysfunction presents notable challenges, particularly regarding glycemic control and potential adverse effects.

At low doses, colchicine may offer benefits by reducing the incidence of diabetes mellitus and hypoglycemia, likely through modulation of systemic inflammation. However, higher doses have been associated with hyperglycemia and impaired insulin secretion. In patients with ESRD, altered drug metabolism may amplify these glycemic effects and increase the risk of complications such as diarrhea, neuromuscular toxicity and hypotension.

These observations emphasize the importance of vigilant monitoring, individualized dosing and careful risk assessment when prescribing colchicine in this high-risk population. A multidisciplinary approach that incorporates nephrology, endocrinology and pharmacology expertise is essential to optimize outcomes. Further research is warranted to clarify the mechanisms underlying colchicine's glycemic effects and to establish evidence-based dosing strategies tailored to patients with complex comorbidities.

Declarations

Ethics approval and consent to participate

Ethical approval was not required for this case report in accordance with institutional guidelines. Written informed consent for participation and publication was obtained from the patient.

Consent for publication

Written informed consent was obtained from the patients for publication of this case report.

Availability of data and materials

All data supporting the findings of this study are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Abel Mwanga contributed to data collection, patient evaluation and manuscript drafting. Datius Mutalemwa participated in clinical management, literature review and contributed to manuscript editing. Kajiru Kilonzo provided senior clinical oversight, contributed to the interpretation of findings and critically revised the manuscript for intellectual content. Huda Akrabi conceptualized the study and led the manuscript writing. All authors read and approved the final version of the manuscript.

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