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Neutropenia Associated with Rifabutin Therapy for Helicobacter pylori Eradication: A Case Report

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Abstract

Rifabutin is a less often used medicine, typically used for M.avium complex infection and tuberculosis. However there's is an increased usage as salvage therapy for drug-resistant H.pylori infection. Rifabutin is the most sought-after antibiotic among individuals with H.pylori that are resistant to clarithromycin or in individuals with previous treatment failures. Nonetheless, it is associated with dangerous complications such as drug-induced neutropenia. It is defined by a significant reduction in neutrophil count. Patients may experience symptoms or be asymptomatic, depending on the severity and underlying medical issues. However, a thorough initial workup by the physician is required to prevent future issues. For patients using rifabutin, a routine follow-up with a complete blood count may be recommended to detect neutropenia early. Prompt treatment with medication withdrawal and consideration of G-CSF such as filgrastim is a given.

Keywords: Rifabutin; H.pylori; Severe Neutropenia; G-CSF

Introduction

Helicobacter pylori (H. pylori) infection is a common gastrointestinal pathogen affecting millions worldwide. It is linked to a variety of gastric diseases, including peptic ulcers, gastritis, and gastric cancer. Traditionally, proton pump inhibitors (PPIs) were used in conjunction with clarithromycin and either amoxicillin or metronidazole as the foundation of eradication therapy. However, increasing antibiotic resistance, particularly to clarithromycin, has prompted the quest for alternate regimens [1]. Rifabutin has emerged as a promising alternative in H. pylori eradication therapy due to its strong antibacterial activity, especially in individuals with previous failed treatments on standard triple therapy [7]. While generally well tolerated, rifabutin medication is not without side effects. Neutropenia, a rare but potentially deadly illness characterised by a low absolute neutrophil count (ANC) that puts people at risk for infections, is one of the drug's infrequent, lesser-known side effects [2-4].

Herein, we present a case of a patient diagnosed with H. pylori infection who developed neutropenia following treatment with rifabutin-based eradication therapy.

Case History

A 56-year-old African American female with a past medical history of hypertension, hyperlipidemia, non-insulin-dependent diabetes mellitus (NIDDM), and gastroesophageal reflux disease (GERD) presented to the emergency department with a complaint of backache and abdominal pain

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that had persisted for the last 10 days. She described the pain as constantly present, rated at 6 out of 10 in severity, non-radiating, and notably aggravated by movement while alleviated by rest.

Upon further history taking, she mentioned being diagnosed with a Helicobacter pylori infection and had been undergoing a treatment regimen consisting of clarithromycin, rifabutin, and bismuth subsalicylate for the last four weeks. She denied experiencing any associated symptoms such as fevers, chills, cough, cold, chest pain, shortness of breath, unexplained weight loss, or engagement in high-risk sexual behaviors.

In the emergency department, her vital signs were stable. Laboratory tests, however, revealed significant findings, including a white blood cell (WBC) count of 0.7 x 10^3/µL and an absolute neutrophil count (ANC) of 336 cells/µL, indicating severe neutropenia. Imaging studies, including a chest X-ray and a computed tomography (CT) scan of the abdomen and pelvis without contrast, were unremarkable and did not reveal any acute pathology. Infectious work-ups such as HIV, Hepatitis B, and C were negative.

Given the severity of her neutropenia and its likely association with her antibiotic regimen, she was admitted to the hospital for further management of severe drug-induced neutropenia. The regime for H.pylori treatment was held. She was placed on neutropenic precautions to reduce the risk of infection. A hematologist was consulted, and she received a single dose of Filgrastim 300 micrograms, a granulocyte colony-stimulating factor (G-CSF), to stimulate the production of neutrophils.

The following day, her WBC count improved significantly to 5.4 x 10^3/µL. Due to the complexity of her H. pylori treatment and the need to manage her drug regimen carefully, she was advised to follow up with her gastroenterologist on an outpatient basis to determine when and how to safely resume her medications. She was discharged on a pain regime for her chronic back pain subsequently and was advised to continue following up with her primary care physician.

Discussion

The development of neutropenia following rifabutin therapy for Helicobacter pylori eradication raises important clinical considerations. Neutropenia, defined as an absolute neutrophil count (ANC) below 1.5 × 10^9 cells/L, is a rare but potentially serious adverse effect associated with rifabutin use. While the precise mechanism of rifabutin-induced neutropenia remains unknown, it is thought to entail either direct bone marrow suppression or immune-mediated processes [5]. Rifabutin may exert its myelosuppressive effects through direct toxicity on hematopoietic progenitor cells or by inducing immune-mediated destruction of neutrophils. Additionally, the formation of reactive metabolites or idiosyncratic immune responses may contribute to neutropenia in susceptible individuals [6].

Clinicians must maintain a high level of suspicion for hematological side effects, especially in patients with predisposing factors such as underlying hematologic disorders, immunosuppression, or concomitant medications known to cause bone marrow suppression. Although the patient discussed above did not carry any predisposing factors or symptoms from neutropenia, this goes on to prove that regular monitoring of complete blood counts before and during rifabutin therapy is essential to promptly identify and manage neutropenia.

In cases of rifabutin-induced neutropenia, treatment discontinuation is typically warranted, with close monitoring for resolution of neutropenia and any signs of infection. Granulocyte colony-stimulating factors (G-CSF), such as filgrastim or pegfilgrastim, may be considered in severe cases to hasten neutrophil recovery and reduce the risk of infection-related complications [2]. These agents stimulate the proliferation and differentiation of neutrophil progenitor cells in the bone marrow, thereby increasing the production of mature neutrophils. An increase in the neutrophil count can often be observed within 24 hours of the first dose, which is what we witnessed.

Furthermore, the decision to use rifabutin in H. pylori eradication regimens should be carefully weighed against the risk of hematological side effects, especially in patients with limited treatment options due to antibiotic resistance [6]. Alternative therapeutic strategies, such as tailored antimicrobial therapy based on susceptibility testing or novel combination regimens, may be considered in these cases to minimize the risk of adverse events while ensuring successful H. pylori eradication.

Conclusion

The case presented here serves as a poignant reminder of the multifaceted nature of therapeutic interventions. While rifabutin has shown promise in the treatment of Helicobacter pylori, it must be used with caution due to potential side effects. The emergence of neutropenia, though rare, underscores the critical need for vigilant monitoring and swift intervention. In the face of such complications, clinicians must balance the imperative of eradicating microbial threats with the imperative of safeguarding patient well-being. Through this report, we aim to raise awareness among clinicians regarding this rare but significant adverse event associated with rifabutin therapy for H. pylori eradication.

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