Primary Cutaneous Nocardiosis in a Patient Taking Adalimumab Therapy for Crohn’s Disease

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INTRODUCTION

The introduction of biologic immunomodulators, in particular antibodies targeted against tumor necrosis factor alpha (TNF-alpha), has revolutionized treatment of Crohn’s disease. However, this comes at the expense of a higher risk for opportunistic infections due to a generalized immunosuppressive effect. Bacterial and opportunistic infections are well-known complications of anti-TNF agents.1,2 Nocardia has been reported rarely among patients on anti-TNF agents.3

TNF plays a role in the clearance of nocardia in animal models.4 Immunosuppression, anti-TNF treatment in particular, may favor growth and dissemination of nocardia. Pulmonary and cutaneous nocardiosis has been reported in Crohn’s disease patients on infliximab.5,6 Nocardiosis has been reported with adalimumab therapy in rheumatoid arthritis patients.7,8 Our search did not reveal any cases of nocardiosis while on adalimumab therapy in Crohn’s disease patients.

Crohn’s disease patients receiving biologic agents, in particular, tumor necrosis factor (TNF)-alpha inhibitors are immunosuppressed and are prone to develop opportunistic infections. We report a rare case of primary cutaneous nocardiosis in an immunocompromised patient on chronic anti-TNF for underlying severe Crohn’s disease.

CASE REPORT

A 36-year-old Caucasian gentleman with a history of Crohn’s disease treated with adalimumab presented with a cellulitis-like rash on his forehead (Figure 1). Five weeks previously, he had struck his forehead on a construction pole, sustaining a laceration which required sutures. On presenta-
DISCUSSION

Human nocardiosis is caused by nocardia species which is an ubiquitous soil inhabiting bacteria and considered an opportunistic pathogen. It can affect the skin and classically disseminate to involve the lungs and brain. It is a difficult infection to treat and carries high mortality if disseminated. Nocardiosis has been well known to affect AIDS patients, transplant recipients, and long term corticosteroid treated individuals. Now, it is being recognized increasingly in immunosuppressed patients on anti-TNF agents. Previous use of corticosteroids has been identified as a risk factor, as noted in our case, and was present in more than 50% of cases in earlier studies.

Early diagnosis is essential and nocardia should be considered in the differential diagnosis of any patient presenting with draining skin lesion or painful rash who is receiving anti-TNF agents or has in the recent past. Anti-TNF therapy doubles the risk of opportunistic infections in inflammatory bowel disease patients. This underscores the importance of adherence to guidelines for their prevention and management. A high level of vigilance and scrutiny in examination and follow up are highly recommended for this group of patients.

CONCLUSION

A high level of suspicion must be enforced when patients receiving TNF-alpha inhibitors present with skin manifestations, in particular, cellulitis-rash or draining lesion. Early identification and treatment of nocardia is pivotal to prevent disseminated disease and mortality.

REFERENCES


Keywords: nocardia infections, adalimumab, Crohn’s disease