

Diagnosis and treatment: A biopsy of the masseter was subsequently performed. It revealed infiltration of the muscle by eosinophilic polymorphonuclear cells. The diagnosis of focal eosinophilic myositis was confirmed, and oral corticosteroid treatment upon tapering the dose was initiated.

Results: At the three-week follow-up, the patient exhibited a significant reduction in jugal swelling and a slowly favorable decrease in eosinophil count.

Discussion: This case highlights the rare occurrence of focal eosinophilic myositis affecting the masticatory muscles in the context of hypereosinophilic syndrome (HES). Eosinophilic infiltration of skeletal muscles is a known feature of HES, but involvement of the masticatory muscles has not been previously documented.

Conclusion: Focal eosinophilic myositis involving the masticatory muscles is a novel manifestation of HES. Awareness of this rare presentation is crucial for accurate diagnosis and appropriate management. Corticosteroid treatment appears to be effective in achieving symptomatic relief.

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Pulmonary miliary in anti-TNF treated patient: not always tuberculosis

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Abstract

Histoplasmosis is a prevalent fungal infection in the Americas, associated with exposure during renovation activities, cleaning chicken coops and even adventure sports, such as cave exploration, leading to the inhalation of aerosolized pathogens. HIV individuals and patients receiving immunosuppressive therapy are at increased risk of disseminated histoplasmosis.

We reported here an exceptional case involving a 23-year-old woman admitted to the infectious diseases ward under suspicion of tuberculosis (TB). The patient had a medical history of Crohn's disease, which had been diagnosed a year earlier and treated with anti-TNF therapy (Infliximab) and Azathioprine. Notably, her chest X-ray and quantiferon were normal before the initiation of this treatment.

Over the course of three weeks, she experienced fever, dry cough, night sweats, fatigue, and noticed a left anterior cervical adenopathy, accompanied by a mild, non-pruritic rash on her trunk. Chest X-ray and CT scan revealed a pattern compatible with pulmonary miliary and multiple mediastinal and hilar adenopathies. The onset of symptoms occurred a few weeks after she returned from a five-month laboratory academic internship in Montreal, Canada, during which she also traveled to Costa Rica for two weeks.

Clinical examination showed a fever of up to 39°C, a left anterior cervical adenopathy, and a maculopapular rash on her trunk. Blood analysis indicated a mild inflammatory syndrome (C-reactive protein level of 32 mg/L), while other parameters remained normal including quantiferon. A skin biopsy of the rash yielded non-specific findings, and cultures were negative. Sputum samples were collected, along with a broncho-alveolar lavage which returned negative results for tuberculosis (direct examination and geneXpert for TB). A biopsy of the cervical adenopathy revealed histiocytic epithelioid granulomas with fibrinoid necrosis. Direct examination and geneXpert for TB were negative. Finally, 16S PCR testing on blood, broncho-alveolar lavage and adenopathy samples were positive for Histoplasmosis. The patient received a two-week treatment with Lipid amphotericin B at 3 mg/kg.

This case highlights the occurrence of severe miliary pulmonary histoplasmosis, without apparent exposure, particularly following anti-TNF therapy. It emphasizes the importance of considering alternative diagnoses when presented with clinical symptoms resembling miliary tuberculosis especially in patients under immunosuppressive therapy.

