Eosinophilic Liver Infiltration

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Abstract

Eosinophilic liver infiltration is a commonly encountered focal eosinophil-related inflammation with or without necrosis, which can be seen on computed tomography (CT) in the presence of peripheral eosinophilia. Although this entity has a relatively benign course, it is related to numerous conditions for which diagnosis may be challenging and requires substantial diagnostic work-up for proper management and care of the underlying disease. We report a case of a 60-year-old man who presented with a 1-week history of right upper quadrant abdominal pain with multiple ill-defined liver hypodensities associated with significant eosinophilia.

Introduction

Eosinophilic organ infiltration has been described secondary to identifiable causes such as drug hypersensitivity, allergic diseases, malignancies, hypereosinophilic syndrome, collagen vascular diseases, and, most commonly, to parasitic infections.¹ Hepatic eosinophilia, or eosinophilic granuloma, has been more frequently encountered since the rise in popularity of computed tomography (CT) scans.¹,² Nonetheless, in clinical practice, underlying causes are often difficult to identify. Radiographic findings may mimic hepatic metastasis, leading to substantial diagnostic work-up. Clinical diagnosis is usually based on a combination of clinical symptoms, medical history, laboratory abnormalities, and imaging findings after exclusion of common identifiable causes.³

Case Report

We report a 60-year-old man who presented after 1 week of persistent right upper quadrant and epigastric abdominal pain that started after eating cooked fish. He complained of subjective fever. He denied sick contacts, contact with domestic animals, or recent travel. His past medical history included schizophrenia, hyperlipidemia, and a shellfish allergy. Medications included simvastatin, quetiapine, bupropion, sulindac, and clonazepam. He denied use of herbs or dietary supplements.

On physical evaluation, the patient was afebrile with an abdomen tender to palpation in the right upper quadrant and epigastric region. Initial laboratories showed leukocytosis associated with marked eosinophilia, with a white blood cell count of 19,200/µL, and 60% eosinophils corresponding to a total eosinophil count of 11,520/µL. Liver enzymes and alkaline phosphatase were mildly elevated. Chest x-ray revealed clear and well-expanded lungs, with no consolidates or effusions. Abdominal/pelvic CT revealed heterogeneous attenuation of the liver with multiple, ill-defined hypodensities, mural thickening of the gastric wall, and multiple, sub-centimeter mesenteric and retroperitoneal adenopathies. A 1.4 x 2-cm portocaval adenopathy was also identified (Figure 1).
The patient was admitted with suspected hypereosinophilic syndrome. Tests for ova, parasites, *Schistosoma, Strongyloides, Toxocara canis, Fasciola*, and acid fast bacilli were negative. An upper endoscopy and colonoscopy were normal except for mild diverticulosis. Liver biopsy demonstrated mild chronic inflammation with eosinophilic infiltration (Figure 2). Testing for chronic eosinophilic leukemia (CEL), a rare but possible cause of eosinophilia, was also negative. Given the patient’s report of past exposure to schistosomiasis, praziquantel was started empirically, pending serology test. *Helicobacter pylori* titers were positive, and treatment was provided accordingly. The symptoms slowly improved despite persistent eosinophilia, and the patient was discharged with close follow-up.

One month later both the eosinophilia and liver tests were normal. A follow-up abdominal MRI showed significant improvement of the multiple, small hepatic lesions and resolution of lymphadenopathy (Figure 3). Follow-up MRI 2 months later demonstrated complete resolution of the multiple scattered hepatic lesions on the basis of known aseptic eosinophilic infiltration (Figure 3).

**Discussion**

Focal eosinophilic infiltration is typically characterized by multiple, small, (<2 cm) hypoattenuate, oval lesions with fuzzy margins, mostly discernable during the portal phase on CT scan. It has a benign course and is mostly detected as an incidental finding in asymptomatic individuals with peripheral eosinophilia (>500/µL). A recent retrospective study correlated abdominal CT findings with peripheral eosinophilia in patients diagnosed with eosinophilic liver disease. The reported peripheral eosinophil count ranged from 500 to 11,689/µL. Although not rare, there is limited data describing the clinical characteristics, pathophysiology, and mechanisms of organ infiltration. The etiology of focal eosinophilic infiltration of the liver is mostly attributed to hepatic migration of parasitic larva; however, medications, allergic disorders, connective tissue disease, and neoplasms have also been implicated. The most commonly described parasites associated with this condition are *Toxocara canis, Fasciola hepatica, Clonorchis sinensis, Spirometra mansonoides, and Taenia solium*. Lymphomas, as well as stomach, colon, and liver cancers have also been associated.

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**Figure 1.** Heterogeneous liver parenchyma with multiples hypoattenuating ill-defined lesions on the portal venous phase.

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**Figure 2.** H&E stain of liver biopsy showing mild mononuclear inflammation with scattered eosinophils infiltrating the periportal and lobular area at (A) 10x magnification and (B) 40x magnification.
The disease course is followed by imaging studies until resolution, which usually occurs at a median of 6 months. Some authors have reported that empiric anti-parasitic medications, such as praziquantel, may shorten the course of disease.\(^1\) Our patient has remained disease-free and asymptomatic 1 year after the initial presentation. The use of empiric treatment with an anti-parasitic drug may have played a role in the early resolution of the patient’s symptoms, peripheral eosinophilia, and liver imaging findings, as has been previously reported.

**Disclosures**

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**Figure 3.** T1 MRI post-contrast portal venous phase (A) 1 month after discharge showing significant improvement of multiple hypointense lesions, and (B) 2 months after discharge showing complete resolution of the hepatic lesions.