

Case Report

Hepatic non-caseating granulomas mimicking malignancy: a diagnostic dilemma of mixed infection

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ABSTRACT

Hepatic granulomas present a diagnostic challenge due to their diverse etiologies, including infections, autoimmune disorders, and malignancies. In endemic regions, lesions with lymphadenopathy and metabolic activity on imaging often raise suspicion of malignancy or tuberculosis. We report a case of a 54-year-old female presenting with significant weight loss and multiple hepatic lesions with mesenteric lymphadenopathy, initially suggestive of malignancy. Histopathology revealed non-caseating granulomas, while microbiological investigations were negative. A multidisciplinary panel evaluation highlighted peripheral eosinophilia as a key clue, raising suspicion of a parasitic or mixed infectious etiology. Empirical antiparasitic therapy resulted in rapid resolution of hepatic lesions and normalization of eosinophil counts. This case emphasizes the importance of considering infections as malignancy mimics and highlights the role of clinical acumen and multidisciplinary evaluation in endemic settings.

Keywords: Hepatic granuloma, Malignancy mimic, Eosinophilia, Mixed infection, Parasitic infection, Multidisciplinary evaluation

INTRODUCTION

Hepatic granulomas are identified in approximately 2-10% of liver biopsies and represent a diagnostic challenge due to their broad etiological spectrum.^{1,2} Common causes include infections such as tuberculosis, autoimmune disorders like sarcoidosis, drug-induced reactions, malignancies, and parasitic infestations.

In tuberculosis endemic regions, granulomatous lesions associated with lymphadenopathy and metabolically active imaging findings often raise suspicion of tuberculosis. Malignancy is another differential diagnosis to be considered. However, non-caseating granulomas require careful clinicopathological correlation, as they may also be seen in infections and inflammatory conditions.⁵

Parasitic infections such as toxocariasis, fascioliasis, and related zoonoses are important but under-recognized causes of hepatic granulomas.^{6,7-9} These conditions are frequently underdiagnosed due to limitations in conventional diagnostic methods.

CASE REPORT

A 54-year-old female presented with an eight-month history of unintentional weight loss (from 54 kg to 47 kg), along with night sweats and generalized malaise. There was no history of fever, abdominal pain, altered bowel habits, tuberculosis exposure, or recent travel.

On examination, the patient was afebrile and thin-built, with no lymphadenopathy or hepatosplenomegaly.

Investigations

Liver function tests were within normal limits. Ultrasonography (abdomen) was multiple hepatic lesions with mesenteric lymphadenopathy. Contrast-enhanced imaging and PET-CT was multiple metabolically active hepatic lesions (largest 3.4×2.8 cm; SUV max 7.12), suggestive of malignancy (Figure 1)

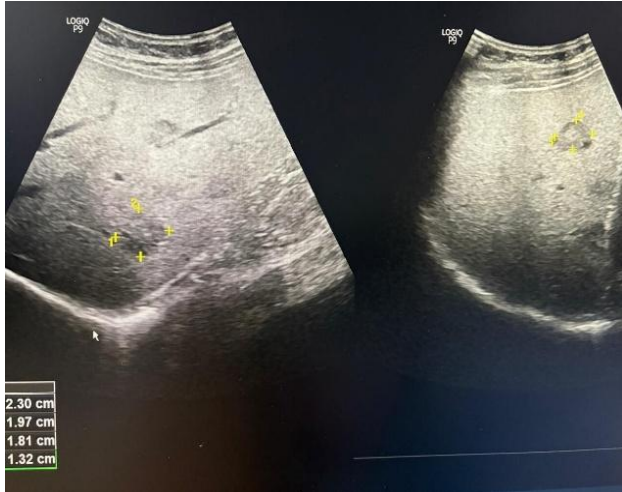


Figure 1: USG abdomen-liver lesions in left and right lobes.

Histopathology

Ultrasound-guided liver biopsy showed well-formed non-caseating epithelioid granulomas and Langhans-type giant cells (Figure 2).

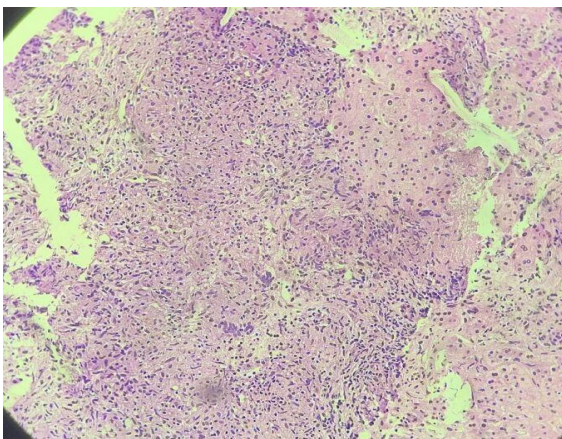


Figure 2: Liver histopathology-granuloma with Langhan cell.

Laboratory workup

GeneXpert for tuberculosis was negative. Acid-fast bacilli culture was negative. Serum angiotensin-converting enzyme was normal. Tumor markers (AFP, CEA, CA 19-9) were within normal limits. Complete blood count

revealed-Eosinophilia: 14%. Stool examination for ova and parasites was negative.

Multidisciplinary panel evaluation

A multidisciplinary panel involving physicians, oncologists, radiologists, and pathologists discussed discordance between imaging and laboratory findings.

Differential diagnoses considered included: Malignancy, tuberculosis, sarcoidosis and parasitic infection.^{2,8-10}

In view of persistent eosinophilia and negative investigations, a probable mixed infectious etiology was considered.

Management and follow-up

The patient was initiated on oral Albendazole 400 mg twice daily and Metronidazole 400 mg twice daily. After one week ultrasonography abdomen showed complete resolution of hepatic lesions (Figure 3). The eosinophil count reduced to 8%. Albendazole was continued at 400 mg once daily for an additional seven days.

At two-week follow-up, eosinophil count normalized (2%), the hepatic lesions remained resolved but the mesenteric lymphadenopathy persisted. An empirical short course of acyclovir 400 thrice daily for 5 days was administered. Subsequent imaging showed resolution of lymphadenopathy. At three-month follow-up, the patient was asymptomatic and had weight gain.



Figure 3: Follow up USG abdomen-liver lesions disappeared- post anti-parasitic treatment.

DISCUSSION

This case exemplifies a diagnostic challenge where hepatic granulomas with lymphadenopathy and increased metabolic activity on imaging closely mimic malignancy.^{11,12} In tuberculosis-endemic regions, such findings often prompt empirical antitubercular therapy or extensive malignancy workup. Several reports have

demonstrated that eosinophilic and parasitic liver lesions can closely mimic malignancy on imaging, including PET-CT, often leading to diagnostic dilemmas and even unnecessary interventions.¹¹⁻¹³

In the current era of advanced diagnostics, where multiple modalities are available, clinical reasoning may at times resemble a jigsaw puzzle in which the pieces do not seamlessly align. Despite extensive investigations, a unifying diagnosis may remain elusive.

In endemic settings such as India, infections are diverse, and many may remain subclinical or undetected by conventional laboratory methods.³ The inability of current diagnostics to reliably detect such infections may lead to under-recognition of their clinical impact. Granulomatous lesions may represent immune responses to occult or mixed infections rather than a single identifiable pathogen.¹⁴ Peripheral eosinophilia is a key diagnostic clue and may indicate underlying parasitic or eosinophilic disorders involving the liver.^{3,4} In this case, the rapid resolution of hepatic lesions following antiparasitic therapy supports a probable infectious etiology. Such a response is atypical for malignancy, tuberculosis, or sarcoidosis.^{5,11}

This case highlights that in endemic regions, infections should be considered early, even when imaging suggests malignancy. Subclinical infections may not be recalled by patients, and over-reliance on investigations without integrating clinical acumen may lead to diagnostic delays.

The persistence of lymphadenopathy despite hepatic resolution suggests a delayed immune response or secondary process. Its eventual resolution cannot be conclusively attributed to antiviral therapy.

Clinical implications

Hepatic granulomas with lymphadenopathy can mimic malignancy. In endemic regions, infections should be considered early in the course of the disease. Subclinical and mixed infections may not be detected by routine diagnostics. Peripheral eosinophilia is a key diagnostic clue. Multidisciplinary evaluation and clinical acumen are essential.

CONCLUSION

Hepatic granulomas with lymphadenopathy can closely mimic malignancy, creating a diagnostic dilemma. In endemic settings, infections-particularly parasitic and mixed infections-should be strongly considered, especially in the presence of eosinophilia and negative conventional investigations. A multidisciplinary approach combined with clinical judgment is crucial. Empirical therapy may serve as both a diagnostic and therapeutic tool in selected cases.

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