Case report

Idiopathic granulomatous hepatosplenic lesion in a case of chronic liver disease

Abtract

Granulomatous liver disease is common and can indicate underlying systemic issues. Liver biopsy findings may confuse clinicians. Some patients have abnormal liver panels without symptoms, while others show systemic manifestations. Identifying the underlying cause allows proper treatment, but in some cases, even extensive workup fails to provide a diagnosis, leading to frustration. Here, we present a similar case of chronic liver disease with hepatosplenic granulomas where the diagnosis remains elusive despite an extensive workup.

Keywords: [granulomatous liver disease , chronic liver disease , autoimmune hepatitis , cirrhosis , portal hypertension]

1. Introduction

Granulomatous liver disease is a frequently encountered issue, which can serve as an important indicator of an underlying systemic disease [1]. The presence of hepatic granulomata in liver biopsy results can often create confusion for both clinicians and consultants [2]. Such a condition may lead to abnormal liver panel results without any noticeable symptoms in patients. Alternatively, patients might display systemic manifestations of the underlying disease [3]. Identifying the systemic process is crucial as it enables the application of appropriate therapy and can lead to improvements in the natural course of the disease. Thorough evaluation of systemic symptoms and pertinent laboratory tests frequently facilitates a precise diagnosis [4].this article aims to provide insights into addressing consultations concerning the detection of hepatic granulomata on liver biopsy.

2.case presentation

A 42-year-old female presented with a three-week history of abdominal distension and jaundice for the last two weeks. She had no history of fever, vomiting, or arthralgia.

Her past medical history included complaints of generalized pruritus and type two

diabetes mellitus. There was no history of similar complaints or gastrointestinal malignancy in her family. She also denied any history of contact with tuberculosis patients, travel, illicit sexual activity, iv drug abuse, or intake of complementary medicine. She is a mother of two children with no history of abortion. Her medications included metformin. Clinical examination showed a normal build, she was afebrile, hemodynamically stable, with no peripheral lymphadenopathy. Abdominal examination suggested no tenderness, and shifting dullness was present, with hepatosplenomegaly noted.

laboratory studies showed white blood cell count of 8600 cell /cu.mm, hemoglobin of 10.3 gm/dl,normocytic hypochromic and hematocrit of 31.1% with platelets count 3.64lakh/cu.mm ,peripheral blood smear reported normal with no abnormal or immature cell. Serum calcium 8.9mg/dl. Liver profile showed total bilirubin of 2 mg/dl, total protein of 7.2 g/dl, albumin of 2.7 g/dl,globulin of 4.5g/dl , ast of 135 u/l, alt of 66 u/l, and alkaline phosphatase of 1281 u/l with ggt of 775 u/l. Ascitis fluid analysis suggestive of high saag with low protein transudative features. Etiological workup showed negative results for biomarker of hepatitis b virus , hepatitis c virus , hiv and autoimmune marker like ana , anti mitochondrial antibody . Immunoglobulin level showed normal result. Egd showed grade one esophageal varices with portal hypertensive gastropathy.chest x ray showed normal lung parenchyma with no evidence of mediastinal lymhadenopathy and fibrosis.

Ultrasonograph of abdomen showed : multiple ill defined iso to hypoechoic lesion liver and spleen and hepatosplenomegaly with normal billiary anatomy

Computed tomography of the abdomen (figure 1) showed multiple ill defined iso to hypoechoic lesion involving all the segments of liver , with no increased vascularity spleen was enlarged with similar type of diffuse lesion with normal gallbladder and pancreas.no dilatation of portal vein , common bile duct noted or ihbr noted.

Magnetic resonance imaging (figure 2)showed liver; spleen showed multiple hyperintense lesion of varying sizes diffusely scattered .no evidence of extrahepatic billiary obstruction or intrahepatic obstruction.

Liver biopsy (figure 3,4) revealed numerous non caseous granulomas were scattered throughout the hepatic lobules with evidence of fatty infilteration , mallory denk bodies and fibrotic seta suggestive of fibrosis. Bile ducts appeared normal in number and morphology and hepatic vasculature was unremarkable.

Extensive workup done to rule out causes of hepatic granulomas like acid fast bacillus stain , pcr of tissue done showed negative of any microorganism . Tissue further subjected to different stain to rule out storage disorders like wilson`s disease and hemochromatosis showed negative results.

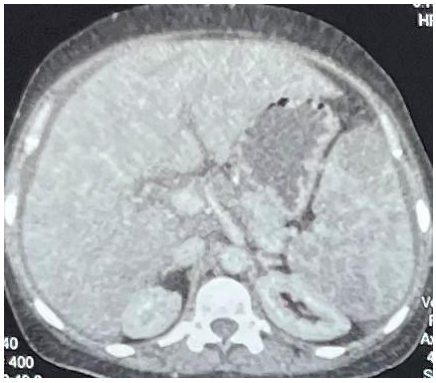

Fig 1 Computed tomography of the abdomen

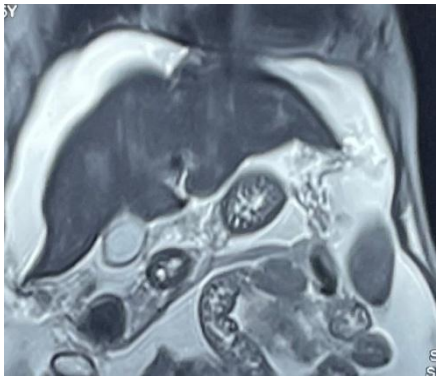

Fig 2 Magnetic resonance imaging

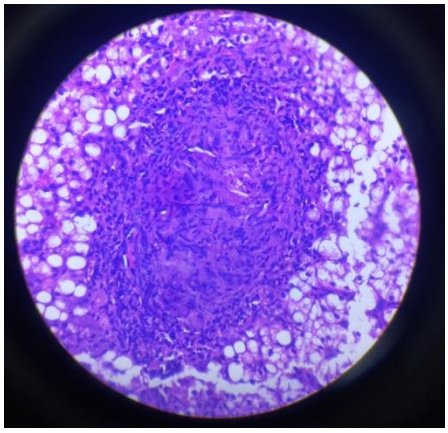

fig 3 Liver biopsy image 1

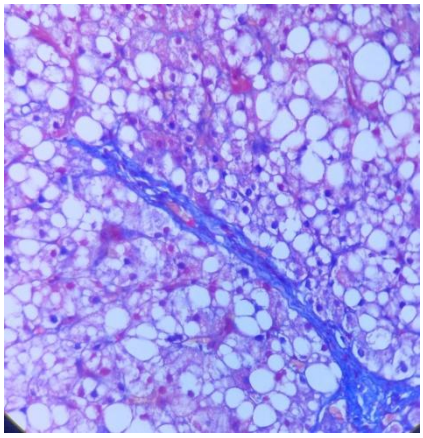

fig 4 Liver biopsy image 2

Outcome : the patient started on weight based dose of ursodeoxycholic acid for raised alkaline phosphate and pruritis and diuretics for ascitis , her symptoms and liver function test showed improvement. The patient will continue to be monitored for further changes or complication.

3.Discussion

Hepatic granulomas, which are localized accumulations of inflammatory cells, can be observed in around 2% to 10% of patients who undergo liver biopsie[5]. Hepatic granulomas are linked to various conditions. In india, the predominant causes include tuberculosis, sarcoidosis, drug-induced reactions, neoplastic diseases, and primary biliary cholangitis[6]. Occasionally, isolated granulomas can be an unexpected discovery in a normal liver biopsy or in individuals with existing liver conditions like viral hepatitis, without any connection to their clinical symptoms or response to treatment [7].

Indeed, a significant proportion, ranging from 10% to 36%, of granulomas are found to have no identifiable cause even after undergoing thorough and comprehensive evaluations[8].

4. Conclusion

In conclusion, while a significant portion of hepatosplenic granulomas may persist as idiopathic and benign entities, the power of effective communication should not be underestimated. By fostering an environment of collaboration and understanding, healthcare providers can embark on a more focused and pragmatic diagnostic journey. This patient-centered approach not only mitigates the burden of exhaustive and fruitless investigations but also instills a sense of empowerment in patients and their families as they navigate the complexities of this enigmatic medical condition.

Competing interests: no

Ethical approval (where ever applicable) : As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

Consent (where ever applicable) : As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

References

1.williams o, fatima s. Granuloma. 2022 sep 19. In: statpearls [internet]. Treasure island (fl): statpearls publishing; 2023 jan–. Pmid: 32119473.

2.culver el, watkins j, westbrook rh. Granulomas of the liver. Clin liver dis (hoboken). 2016 apr 27;7(4):92-96. Doi: 10.1002/cld.544. Pmid: 31041038; pmcid: pmc6490265.

3.coash m, forouhar f, wu ch, wu gy. Granulomatous liver diseases: a review. J formos med assoc. 2012 jan;111(1):3-13. Doi: 10.1016/j.jfma.2011.11.023. Epub 2012 feb 2. Pmid: 22333006.

4.joshi v, jain s, sharma v, khippal n, chaturvedi a. Granulomatous hepatitis with miliary mottling: a rare cause. J assoc physicians india. 2018 sep;66(9):97-98. Pmid: 31321943.

5. Lamps lw. Hepatic granulomas: a review with emphasis on infectious causes. Arch pathol lab med. 2015 jul;139(7):867-75. Doi: 10.5858/arpa.2014-0123-ra. Pmid: 26125427.

6. Lagana sm, moreira rk, lefkowitch jh. Hepatic granulomas: pathogenesis and differential diagnosis. Clin liver dis. 2010 nov;14(4):605-17. Doi: 10.1016/j.cld.2010.07.005. Pmid: 21055685.

7. Tahan v, ozaras r, lacevic n, ozden e, yemisen m, ozdogan o, mert a, tabak f, avsar e, celikel ca, ozbay g, kalayci c, senturk h, tozun n. Prevalence of hepatic granulomas in chronic hepatitis b. Dig dis sci. 2004 oct;49(10):1575-7. Doi: 10.1023/b:ddas.0000043366.18578.15. Pmid: 15573907.

8. Adams dh. Hepatology: a textbook of liver disease, 4th edition: edited by zakim, boyer. Philadelphia: saunders, 2003, £230.00, pp 1765. Isbn 0-7216-9051-3. Gut. 2003 aug;52(8):1230–1. Pmcid: pmc1773747.