ISSN 1948-5182 (online)

World Journal of *Hepatology*

World J Hepatol 2023 February 27; 15(2): 123-320

Published by Baishideng Publishing Group Inc

World Journal of Hepatology

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The *WJH* is now abstracted and indexed in PubMed, PubMed Central, Emerging Sources Citation Index (Web of Science), Scopus, Reference Citation Analysis, China National Knowledge Infrastructure, China Science and Technology Journal Database, and Superstar Journals Database. The 2022 edition of Journal Citation Reports® cites the 2021 Journal Citation Indicator (JCI) for *WJH* as 0.52. The *WJH*'s CiteScore for 2021 is 3.6 and Scopus CiteScore rank 2021: Hepatology is 42/70.

RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: *Yi-Xuan Cai*; **Production Department Director:** *Xiang Li*; **Editorial Office Director:** *Xiang Li*.

NAME OF JOURNAL
World Journal of Hepatology

ISSN
ISSN 1948-5182 (online)

LAUNCH DATE
October 31, 2009

FREQUENCY
Monthly

EDITORS-IN-CHIEF
Nikolaos Pyrsopoulos, Ke-Qin Hu, Koo Jeong Kang

EDITORIAL BOARD MEMBERS
https://www.wjgnet.com/1948-5182/editorialboard.htm

PUBLICATION DATE
February 27, 2023

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World J Hepatol 2023 February 27; 15(2): 311-317

DOI: 10.4254/wjh.v15.i2.311

ISSN 1948-5182 (online)

CASE REPORT

Autoimmune hepatitis and eosinophilia: A rare case report

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Specialty type: Gastroenterology and hepatology

Provenance and peer review: Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): 0
Grade C (Good): C, C
Grade D (Fair): 0
Grade E (Poor): 0

P-Reviewer: Nguyen TL, Vietnam; Rodrigues AT, Brazil

Received: November 1, 2022
Peer-review started: November 1, 2022
First decision: December 13, 2022
Revised: December 13, 2022
Accepted: January 5, 2023
Article in press: January 5, 2023
Published online: February 27, 2023

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Abstract

BACKGROUND

Autoimmune hepatitis consists of a chronic liver disease whose etiology is unknown. It is comprised of relevant immunological aspects and of immune-mediated liver injury. Eosinophilia may be a considerable feature, particularly happening in male patients.

CASE SUMMARY

We report here a Crohn´s disease patient presenting with de novo hypergammaglobulinemia, circulating autoantibodies and elevated transaminase levels. He also had significant peripheral eosinophilia and elevated immunoglobulin E levels at diagnosis. The pathology findings from liver biopsy were compatible with autoimmune hepatitis with eosinophilic infiltration.

CONCLUSION

This is the first report of autoimmune hepatitis with exuberant eosinophilic infiltration in the liver and bone marrow, described in a patient with Crohn's disease.

Key Words: Autoimmune hepatitis; Eosinophilia; Bone marrow; Crohn's disease; Case report

Core Tip: There are very few reported cases of autoimmune hepatitis presenting with peripheral blood eosinophilia. This is the first report of autoimmune hepatitis with exuberant eosinophilic infiltration in the liver and bone marrow, described in a patient with Crohn's disease.

Citation: Garrido I, Lopes S, Fonseca E, Carneiro F, Macedo G. Autoimmune hepatitis and eosinophilia: A rare case report. *World J Hepatol* 2023; 15(2): 311-317
URL: https://www.wjgnet.com/1948-5182/full/v15/i2/311.htm
DOI: https://dx.doi.org/10.4254/wjh.v15.i2.311

INTRODUCTION

Peripheral blood eosinophilia is considered either a primary or a secondary phenomenon[1]. Primary eosinophilia often takes place within hematologic malignancies where cytogenetic or bone marrow histologic evidence regarding the clonal expansion of these cells can be found. On the other hand, causes of secondary eosinophilia include parasitosis, medications, malignancies and inflammatory or allergic conditions. Idiopathic eosinophilia consists of a diagnosis of exclusion, when no primary or secondary causes are detected.

Peripheral blood eosinophilia may be found in several hepatobiliary and gastrointestinal disorders. Indeed, some gastrointestinal diseases are eosinophil-mediated pathologies, such as eosinophilic gastroenteritis, inflammatory bowel disease, *Helicobacter pylori* infection, gastroesophageal reflux disease, collagenous colitis and celiac disease[2]. In addition, hepatic eosinophilia has been presented associated to primary biliary cirrhosis, sclerosing cholangitis, eosinophilic cholangitis and eosinophilic cholecystitis.

Currently, there are very few reported cases of autoimmune hepatitis presenting with peripheral blood eosinophilia, usually associated with other autoimmune conditions[3]. Hereafter we explore a case of autoimmune hepatitis associated with peripheral blood and tissue eosinophilia. This report aims at making physicians aware of that association in order to consider this diagnosis in a patient who presents elevated transaminases in concert with a high eosinophil count.

CASE PRESENTATION

Chief complaints

A 36-year-old Caucasian male with asthma and Crohn's disease (Montreal classification A2L2B1), was under azathioprine until 4 years ago when it was discontinued due to clinical and endoscopic remission. His asthma was under control since childhood. He was not on medication and had no known drug allergies. The patient was asymptomatic.

History of present illness

In routine analysis, it was noticed a new-onset cytocholestasis (aspartate aminotransferase 86 U/L, alanine aminotransferase 240 U/L, gamma-glutamyl transferase 288 U/L, alkaline phosphatase 794/L) without hyperbilirubinemia or coagulopathy.

History of past illness

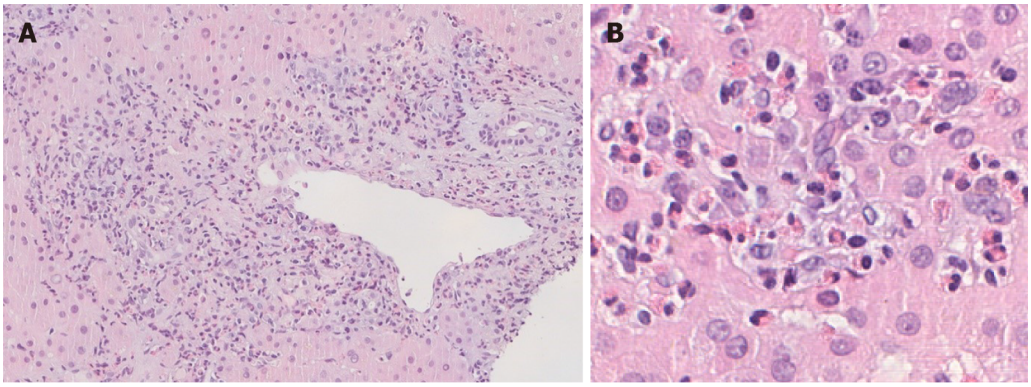
The patient denied any history that can suggest viral prodrome, sick contacts, recent travel, medication ingestion (comprising herbal or over-the-counter), exposure to well water, exposure to recreational drugs, tattoos, alcohol, high-risk sexual behavior or blood transfusions.

Physical examination

Normal.

Laboratory examinations

Antinuclear antibody was positive (1:100, speckled pattern) as well as anti-smooth muscle. All other liver-related autoantibodies were negative (anti-mitochondrial, anti-liver-kidney microsomal, anti-soluble liver antigen and antineutrophil cytoplasmic). Immunoglobulin G (IgG) levels were elevated (3650 mg/dL). Serology for human immunodeficiency virus, hepatitis A virus, Epstein-Barr virus, cytomegalovirus, and herpes simplex virus type 1 and 2 were negative. Polymerase chain reaction considering hepatitis B, C and E viruses were negative, too. Alpha-1 antitrypsin, ceruloplasmin and iron tests were all normal. The same could be perceived for thyroid function.



DOI: 10.4254/wjh.v15.i2.311

Figure 1 Liver biopsy. A: Portal tract inflammation with intense lymphoplasmacytic infiltrate and interface hepatitis (HE ×20); B: Intralobular hepatic parenchyma with numerous eosinophils (HE ×400).

Blood tests presented an absolute white blood cell count of 33.46 × 10^9/L. Differential count indicated 89.5% of eosinophilia, as well as an absolute eosinophil count of 30 × 10^9/L. In addition, immunoglobulin E (IgE) levels were elevated (8803 kU/L). Blood cultures and parasitological examination of the stools were negative. FIP1L1-PDGFRA fusion transcript was not detected.

Imaging examinations

The abdominal ultrasound showed a liver with a normal appearance and no intra-or extrahepatic biliary ductal dilation.

Histologic examination

A liver biopsy was then performed, revealing infiltration of the portal tracts and intralobular hepatic parenchyma by numerous eosinophils (Figure 1). Lymphoplasmacytic portal tract inflammatory infiltrate with interface hepatitis was identified as well as small aggregates of plasma cells. The interlobular bile ducts appeared intact and iron and copper stains were negative. Furthermore, bone marrow biopsy showed marked eosinophilia, with normal maturation and absence of blasts (Figure 2).

FINAL DIAGNOSIS

The score regarding simplified diagnostic criteria of the International Autoimmune Hepatitis Group was 7 (likely diagnosis of autoimmune hepatitis)[4]. The score considering the revised original pretreatment scoring system of the International Autoimmune Hepatitis Group was 17 (definite diagnosis of autoimmune hepatitis)[5]. Due to lack of evidence for parasitic infection, the reactive bone marrow and the absence of other systemic conditions or drugs, the patient was diagnosed with autoimmune hepatitis with peripheral blood eosinophilia.

TREATMENT

He started treatment with prednisone at 40 mg/d. Cytocholestasis (Figure 3A) and eosinophilia (Figure 3B) progressively improved. The corticosteroid dose was gradually titrated and azathioprine 2 mg/Kg was then added.

OUTCOME AND FOLLOW-UP

After a three-month treatment, follow-up tests showed a normal eosinophil count, liver IgG levels and function tests. These data supported the definite diagnosis of autoimmune hepatitis.

DISCUSSION

Autoimmune hepatitis is a chronic liver disease which is responsible for up to 20% of chronic hepatitis in Western countries. It has a mean annual incidence of 1.9 per 100.000 individuals and a prevalence of

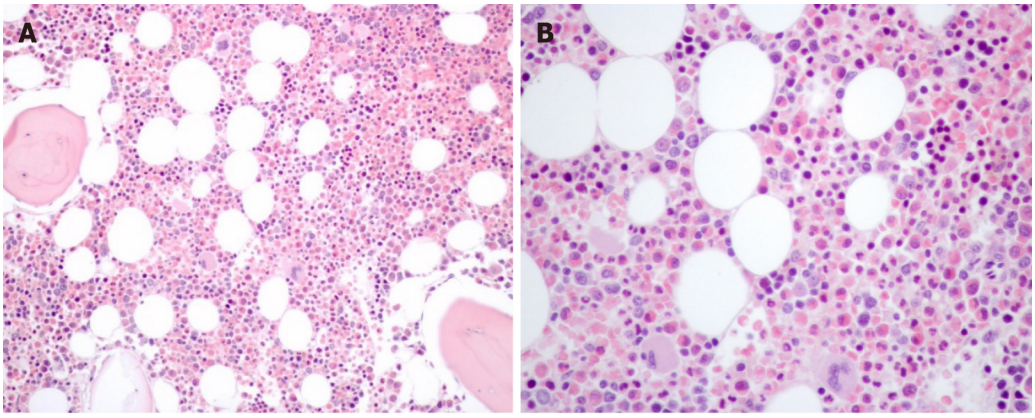


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Figure 2 Bone marrow biopsy. A: Bone marrow slightly hyperplastic, trilinear, with myeloid predominance and eosinophilia (HE ×100); B: Increased number of eosinophils (both precursors and mature forms) (HE, ×400).

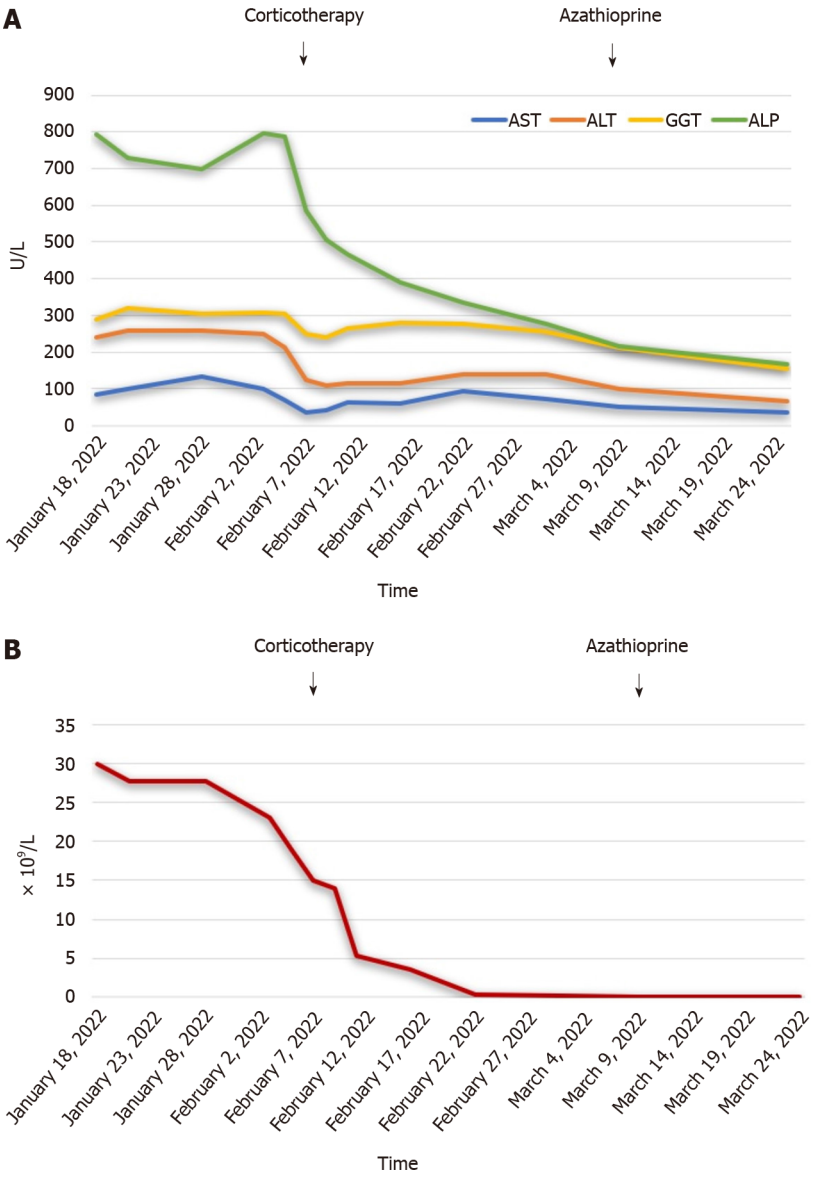

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Figure 3 Follow-up. A: Evolution of liver function tests; B: Evolution of eosinophil count.

Table 1 Reported cases of autoimmune hepatitis associated with peripheral blood eosinophilia

Ref.	Panush *et al* [8]	Kane *et al*[9]	Terrier *et al* [10]	Omata *et al* [13]	Chowdry *et al*[3]	Farani *et al* [11]	Makino *et al*[12]	Present case
Age	14 yr old	41 yr old	16 yr old	49 yr old	18 yr old	41 yr old	7 yr old	36 yr old
Sex	Male	Female	Male	Female	Male	Male	Male	Male
Transaminases	AST 700 U/L; ALT 1560 U/L	AST 200 U/L; ALT --	AST 200 U/L; ALT 320 U/L	AST 1019 U/L; ALT 772 U/L	AST 955 U/L ALT 1194 U/L	AST 70 U/L; ALT 67 U/L	AST 419 U/L; ALT 306 U/L	AST 86 U/L; ALT 240 U/L
Eosinophil count	7.437 × 10^9/L	2.64 × 10^9/L	63.2 × 10^9/L	1.2 × 10^9/L	3.3 × 10^9/L	4.9 × 10^9/L	9 × 10^9/L	30 × 10^9/L
IgG level	2300 mg/dL	2600 mg/dL	--	1930 mg/dL	2760 mg/dL	3180 mg/dL	5234 mg/dL	3650 mg/dL
Positive antibodies	ANA, SMA	SMA	SMA	Negative	ANA, SMA	ANA	ANA	ANA, SMA
Hepatic eosinophilia	No	Yes	No	Yes	Yes	Yes	Yes	Yes
Autoimmune disease associations	Coombs positive hemolytic anemia	Ulcerative colitis, autoimmune thyroid disease	Ulcerative colitis	None	None	Arthritis	None	Crohn's disease

AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; IgG: Immunoglobulin G; ANA: Anti-nuclear antibody; SMA: Anti-smooth muscle antibody.

16.9 in northern European population[6]. It is characterized by hypergammaglobulinemia, circulating autoantibodies and elevated transaminase levels[7]. Peripheral blood eosinophilia is present much less frequently. It has been described in a few cases, usually in association with other autoimmune conditions, such as Coombs positive hemolytic anemia, autoimmune thyroid disease, ulcerative colitis and arthritis[8-11]. As far as we know, this report is the first one concerning autoimmune hepatitis with peripheral blood eosinophilia described in a patient with Crohn's disease. There are also some cases of blood eosinophilia associated with isolated autoimmune hepatitis, in the absence of other autoimmune conditions[3,12,13].

The development of hypereosinophilia when there is also ulcerative colitis and autoimmune hepatitis, which are two autoimmune conditions with a Th2 bias, suggests that a Th2-T-cell population is at the crossroads of the pathophysiology underlying these autoimmune diseases[10]. Other authors suggest that the concurrent existence of these processes mirrors related abnormal immunological events [8]. Another mechanism of eosinophilia can be the result of mast cell activation, which may take place in cholestatic liver disease in which mast cell-derived mediators cause activation and eosinophil chemotaxis[14].

It should be noted that, despite the fact that autoimmune hepatitis is most usually found in women (3:1 ratio), our report presents the case of a male patient[7]. Indeed, most cases described in the literature of autoimmune hepatitis with peripheral blood eosinophilia have also occurred in men (Table 1), which makes us ponder whether eosinophilia in autoimmune hepatitis can be considered a characteristic related to males.

Similar to Omata and colleagues, our patient also has a long history of asthma[13]. Nevertheless, the eosinophilia and elevated IgE levels were not associated with exacerbation of asthma but rather with elevated liver function tests. In fact, our patient had asthma under control for many years. In addition, other causes of eosinophilia, particularly immediate hypersensitivity to common allergens and parasitic infection, were excluded.

Liver biopsy is considered a prerequisite for the diagnosis of autoimmune hepatitis[15]. The classic histologic picture of autoimmune hepatitis includes interface hepatitis with dense plasma cell-rich lymphoplasmacytic infiltrates, emperipolesis, hepatocellular rosette formation, pycnotic necrosis and hepatocyte swelling. The discovery of hepatic eosinophils (even though it is not the predominant inflammatory cell type) enables the diagnosis of autoimmune hepatitis. In a study that describes the use of liver biopsy assessment in the discrimination of idiopathic autoimmune hepatitis *vs* drug-induced liver injury, Suzuki and colleagues discovered that intra-acinar eosinophils could be seen in 32.1% of times and portal tract eosinophils could be found in 60.7% of times regarding autoimmune hepatitis cases. Both of them were more usual than in cases of drug-induced liver injury[16].

In our case, the conjunction of plasma cells with interface hepatitis strongly supported the diagnosis of autoimmune hepatitis. However, the most striking aspect of this patient's disease was the exuberant hepatic eosinophilia. Similarly, the bone marrow aspirate showed a marked increase in normal-appearing cells of the eosinophil series. In this case, tissue eosinophilia was marked and blood eosinophilia was significant. In contrast, the other authors reported that none or only a few eosinophils were

present in the liver biopsy[3].

With regard to treatment, it should be noted that in all cases there was an improvement in liver tests as well as in the eosinophil count. Our patient had a favorable response under treatment with corticosteroids and azathioprine. Other authors also used 6-mercaptopurine and mycophenolate mofetil with an equally favorable response[11,13]. There is a need for long-term cautious management so as to prevent the progression into liver failure or hepatic cirrhosis[17].

CONCLUSION

Pathophysiology of autoimmune disorders is incompletely understood. The coexistence of different diseases could suggest common pathogenic mechanisms. Herein we report a case of autoimmune hepatitis associated with peripheral blood eosinophilia and exuberant liver eosinophilia. It is our goal to emphasize this infrequent presentation of autoimmune hepatitis.

FOOTNOTES

Author contributions: Garrido I did literature review and drafted the manuscript; Garrido I, Lopes S, Fonseca E, Carneiro F, and Macedo G have critically revised and finalized the manuscript; All authors have approved the final version of the manuscript.

Informed consent statement: The patient signed informed consent.

Conflict-of-interest statement: All the authors have no disclosures to report.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

Country/Territory of origin: Portugal

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S-Editor: Liu JH
L-Editor: A
P-Editor: Liu JH

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