

World Journal of *Hepatology*

World J Hepatol 2023 December 27; 15(12): 1253-1343



EDITORIAL

- 1253 From metabolic dysfunction-associated fatty liver disease to metabolic dysfunction-associated steatotic liver disease: Controversy and consensus
Chen L

REVIEW

- 1258 Multifunctional roles of inflammation and its causative factors in primary liver cancer: A literature review
Chen HJ, Huang TX, Jiang YX, Chen X, Wang AF
- 1272 Cell-type specific role of autophagy in the liver and its implications in non-alcoholic fatty liver disease
Raza S, Rajak S, Singh R, Zhou J, Sinha RA, Goel A

MINIREVIEWS

- 1284 Any role for transarterial radioembolization in unresectable intrahepatic cholangiocarcinoma in the era of advanced systemic therapies?
Elvevi A, Laffusa A, Elisei F, Morzenti S, Guerra L, Rovere A, Invernizzi P, Massironi S

ORIGINAL ARTICLE

Clinical and Translational Research

- 1294 Study of liver cirrhosis over twenty consecutive years in adults in Southern China
Wang X, Luo JN, Wu XY, Zhang QX, Wu B

Retrospective Study

- 1307 Peri-operative score for elderly patients with resectable hepatocellular carcinoma
Conticchio M, Inchingolo R, Delvecchio A, Ratti F, Gelli M, Anelli MF, Laurent A, Vitali GC, Magistri P, Assirati G, Felli E, Wakabayashi T, Pessaux P, Piardi T, di Benedetto F, de'Angelis N, Briceño J, Rampoldi A, Adam R, Cherqui D, Aldrighetti LA, Memeo R

Observational Study

- 1315 Serum omentin-1 is correlated with the severity of liver disease in patients with chronic hepatitis C
Peschel G, Weigand K, Grimm J, Müller M, Buechler C

Prospective Study

- 1325 High incidence of periodontitis in patients with ascitic decompensated cirrhosis
Pischke S, Ashouri MM, Peters U, Shiprov A, Schulze Zur Wiesch J, Sterneck M, Fischer F, Huebener P, Mader M, Fischer L, Fründt T, Aarabi G, Beikler T

CASE REPORT

- 1333** Drug induced autoimmune hepatitis: An unfortunate case of herbal toxicity from Skullcap supplement: A case report

Thakral N, Konjeti VR, Salama FW

- 1338** Hepatic pseudotumor associated with *Strongyloides* infection: A case report

Gialanella JP, Steidl T, Korpela K, Grandhi MS, Langan RC, Alexander HR, Hudacko RM, Ecker BL

ABOUT COVER

Editorial Board Member of *World Journal of Hepatology*, Yasuo Tanaka, MD, PhD, Chief Doctor, Department of Gastroenterology, National Center for Global Health and Medicine, Tokyo 162-8655, Japan.
ytanaka@hosp.ncgm.go.jp

AIMS AND SCOPE

The primary aim of *World Journal of Hepatology* (*WJH*, *World J Hepatol*) is to provide scholars and readers from various fields of hepatology with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WJH mainly publishes articles reporting research results and findings obtained in the field of hepatology and covering a wide range of topics including chronic cholestatic liver diseases, cirrhosis and its complications, clinical alcoholic liver disease, drug induced liver disease autoimmune, fatty liver disease, genetic and pediatric liver diseases, hepatocellular carcinoma, hepatic stellate cells and fibrosis, liver immunology, liver regeneration, hepatic surgery, liver transplantation, biliary tract pathophysiology, non-invasive markers of liver fibrosis, viral hepatitis.

INDEXING/ABSTRACTING

The *WJH* is now abstracted and indexed in PubMed, PubMed Central, Emerging Sources Citation Index (ESCI), Scopus, Reference Citation Analysis, China Science and Technology Journal Database, and Superstar Journals Database. The 2023 Edition of Journal Citation Reports® cites the 2022 impact factor (IF) for *WJH* as 2.4.

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

EXECUTIVE ASSOCIATE EDITORS-IN-CHIEF

Shuang-Suo Dang

EDITORIAL BOARD MEMBERS

<https://www.wjgnet.com/1948-5182/editorialboard.htm>

PUBLICATION DATE

December 27, 2023

COPYRIGHT

© 2023 Baishideng Publishing Group Inc

PUBLISHING PARTNER

Department of Infectious Diseases, the Second Affiliated Hospital of Xi'an Jiaotong University

INSTRUCTIONS TO AUTHORS

<https://www.wjgnet.com/bpg/gerinfo/204>

GUIDELINES FOR ETHICS DOCUMENTS

<https://www.wjgnet.com/bpg/GerInfo/287>

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH

<https://www.wjgnet.com/bpg/gerinfo/240>

PUBLICATION ETHICS

<https://www.wjgnet.com/bpg/GerInfo/288>

PUBLICATION MISCONDUCT

<https://www.wjgnet.com/bpg/gerinfo/208>

POLICY OF CO-AUTHORS

<WebSite><https://www.wjgnet.com/bpg/GerInfo/310/</WebSite>>

ARTICLE PROCESSING CHARGE

<https://www.wjgnet.com/bpg/gerinfo/240>

STEPS FOR SUBMITTING MANUSCRIPTS

<https://www.wjgnet.com/bpg/GerInfo/239>

ONLINE SUBMISSION

<https://www.f6publishing.com>

PUBLISHING PARTNER's OFFICIAL WEBSITE

http://2yuan.xjtu.edu.cn/Html/Departments/Main/Index_21148.html

Hepatic pseudotumor associated with *Strongyloides* infection: A case report

Jessica P Gialanella, Tyler Steidl, Kokkola Korpela, Miral S Grandhi, Russell C Langan, H Richard Alexander, Rachel M Hudacko, Brett L Ecker

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): B

Grade C (Good): 0

Grade D (Fair): 0

Grade E (Poor): 0

P-Reviewer: Leowattana W, Thailand

Received: September 13, 2023

Peer-review started: September 13, 2023

First decision: October 16, 2023

Revised: October 21, 2023

Accepted: November 29, 2023

Article in press: November 29, 2023

Published online: December 27, 2023



Jessica P Gialanella, Miral S Grandhi, Russell C Langan, H Richard Alexander, Brett L Ecker, Department of Surgical Oncology, Rutgers Cancer Institute of New Jersey, New Brunswick, NJ 08901, United States

Tyler Steidl, Rutgers New Jersey Medical School, Newark, NJ 07103, United States

Kokkola Korpela, Department of Infectious Disease, Cooperman Barnabas Medical Center, Livingston, NJ 07039, United States

Rachel M Hudacko, Department of Pathology and Laboratory Medicine, Rutgers Robert Wood Johnson Medical School, New Brunswick, NJ 08901, United States

Corresponding author: Brett L Ecker, MD, Assistant Professor, Surgical Oncologist, Department of Surgical Oncology, Rutgers Cancer Institute of New Jersey, 195 Little Albany St, New Brunswick, NJ 08901, United States. brett.ecker@rutgers.edu

Abstract

BACKGROUND

Strongyloides stercoralis is a parasitic infection caused by a roundworm that is transmitted through soil contaminated with larvae. It can infrequently cause hepatic abscesses in immunocompromised patients and is rarely reported to form hepatic lesions in immunocompetent hosts.

CASE SUMMARY

We present a case study of a 45-year-old female who presented with right upper quadrant abdominal pain and constitutional symptoms for several weeks. Cross-sectional imaging identified several malignant-appearing liver masses. Further investigation, including serological testing and histopathologic examination, revealed the presence of serum *Strongyloides* antibodies and hepatic granulomas with extensive necrosis. Following treatment with ivermectin for 2 wk, there was complete resolution of the liver lesions and associated symptoms.

CONCLUSION

This case highlights the importance of considering parasitic infections, such as *Strongyloides*, in the differential diagnosis of hepatic masses. Early recognition and appropriate treatment can lead to a favorable outcome and prevent unnecessary invasive procedures. Increased awareness among clinicians is crucial to ensure the timely diagnosis and management of such cases.

Key Words: Hepatic tumor; Liver mass; *Strongyloides stercoralis*; Parasitology; Case report

©The Author(s) 2023. Published by Baishideng Publishing Group Inc. All rights reserved.

Core Tip: Hepatic pseudotumor is a clinical entity that can mimic malignant tumors of the liver. We report a rare case of hepatic pseudotumor caused by *Strongyloides stercoralis*. Combined with a review of cases indexed in PubMed, we summarize the infectious causes of hepatic pseudotumor. Recognition of hepatic mass-forming parasitic infections may expedite prompt medical management.

Citation: Gialanella JP, Steidl T, Korpela K, Grandhi MS, Langan RC, Alexander HR, Hudacko RM, Ecker BL. Hepatic pseudotumor associated with *Strongyloides* infection: A case report. *World J Hepatol* 2023; 15(12): 1338-1343

URL: <https://www.wjgnet.com/1948-5182/full/v15/i12/1338.htm>

DOI: <https://dx.doi.org/10.4254/wjh.v15.i12.1338>

INTRODUCTION

Inflammatory pseudotumors are mass-like lesions that have been described in various solid organs that mimic the appearance of a neoplasm[1]. Inflammatory pseudotumors represent a heterogeneous group of lesions of both infectious and inflammatory etiology, characterized histologically by the presence of fibroblasts or myofibroblasts and inflammatory cells and the lack of neoplastic cells. When occurring in the liver, hepatic pseudotumor often presents as a solitary, well-defined mass[1]. Due to its radiographic similarity to malignant liver tumors, potential misdiagnosis and unnecessary invasive interventions can occur. Given the variety of clinical presentations, including the absence of symptoms, the incidence of hepatic pseudotumor is not well characterized. It may account for 1% of all resected hepatic tumors[2], highlighting the importance of accurate diagnosis to prevent unneeded surgical resection. The pathogenesis of hepatic pseudotumor is poorly characterized but may be secondary to an exaggerated inflammatory response within the liver[1,3], as they often arise in the setting of infection, autoimmune disease, or recent trauma or surgery[1,3].

Strongyloidiasis is a parasitic infection known to infect the liver and biliary tree[3]. It is caused by the nematode *Strongyloides stercoralis*, which is endemic in regions of West Africa, the Caribbean, and Southeast Asia – where the prevalence may be as high as 40%[4]. The parasitic larvae penetrate the skin and most commonly distribute to the gastrointestinal and pulmonary systems[5]. Less commonly, disseminated strongyloidiasis occurs when additional organs, including the liver, are involved[5,6]. Disseminated strongyloidiasis with hepatic infection has been primarily described in immunocompromised patients[7] but has rarely been observed in immunocompetent hosts[8].

Herein, we present a rare case of strongyloidiasis with hepatic involvement in an immunocompetent patient leading to the formation of a pseudotumor. By review of this case and the published literature, we summarize the incidence, diagnosis and treatment of *Strongyloides* pseudotumor.

CASE PRESENTATION

Chief complaints

A 45-year-old female was referred from the emergency room to hepatopancreatobiliary clinic for evaluation of liver lesions. She described several weeks of weight loss, decreased appetite, early satiety, fatigue, and right upper quadrant abdominal pain.

History of present illness

The patient was initially evaluated in the emergency room, where she reported a 15 lb weight loss secondary to abdominal pain and anorexia. The patient denied any change in bowel habits, sick contacts, or recent travel. The patient reported that her partner had immigrated from West Africa and that she had traveled there with him approximately 5 years earlier. She had also lived for several years in South Carolina, where *Strongyloides* may be endemic.

History of past illness

The patient had a history of hepatic steatosis, uterine fibroids, and morbid obesity. Upper and lower endoscopy had been performed 3 years earlier and were both found to be within normal limits.

Personal and family history

The patient denied any family history of malignancy.

Physical examination

The vital signs were as follows: Body temperature, 36.5°C; blood pressure, 136/76 mmHg; heart rate, 82 beats per min; and respiratory rate, 12 breaths per min. Abdominal exam was notable for tenderness in the right upper quadrant. There was no hepatosplenomegaly. There were no skin lesions present.

Laboratory examinations

Laboratory examination was notable for marked eosinophilia [$0.9 \times 10^3/\text{mL}$ (range 0.0-0.40)] and an elevated sedimentation rate (Westergren; 94 mm/h). Tumor markers were normal (alpha-fetoprotein < 1.8 ng/mL; carbohydrate antigen 19-9 16 U/mL; carcinoembryonic antigen 2.1 ng/mL). Liver function tests were normal, and hepatitis serologies were consistent with previous hepatitis B immunization. Human immunodeficiency virus (HIV) antibody (Ab)/p24 antigen screen, rapid plasma reagin, coccidioides Ab panel, cryptococcus antigen, and quantiFERON-TB Gold Plus were negative. Autoimmune markers were negative (anti-myeloperoxidase Ab < 0.2 units; anti-proteinase 3 Ab < 0.2 units; cytoplasmic antineutrophil cytoplasmic Ab (ANCA) < 1:2 titer; perinuclear ANCA (p-ANCA) < 1:20 titer; atypical p-ANCA < 1:20 titer; angiotensin converting enzyme, serum 42 U/L; anti-cyclic citrullinated peptide Ab, immunoglobulin G (IgG)/IgA 2 U; rheumatoid factor < 10.0 IU/mL; antinuclear Ab direct-negative).

Imaging examinations

An abdominal ultrasound performed in the emergency room noted a focal hypoechoic area within the right hepatic lobe centrally measuring 2.5 cm \times 1.6 cm \times 1.3 cm. Subsequently, a gadolinium-enhanced magnetic resonance image demonstrated a non-cirrhotic liver with a peripherally T2 hyperintense and centrally T2 hypointense 3.7 cm \times 3.2 cm \times 3.7 cm lesion (Figure 1). Signal characteristics raised suspicion for a malignancy, but the rapid interval growth (from ultrasound-estimated size) suggested possible infection. A computerized tomography scan through the abdomen (Figure 2) demonstrated a low-density mass within the right hepatic lobe measuring up to 4.5 cm \times 4.9 cm in the largest dimensions. There was peripheral enhancement with centripetal filling atypical for a hemangioma.

FINAL DIAGNOSIS

The final diagnosis was hepatic pseudotumor caused by *S. stercoralis*.

TREATMENT

The treatment provided to the patient was ivermectin 24 mg by mouth daily for 2 wk.

OUTCOME AND FOLLOW-UP

Liver lesions and systemic symptoms both resolved shortly after treatment (Figure 3).

DISCUSSION

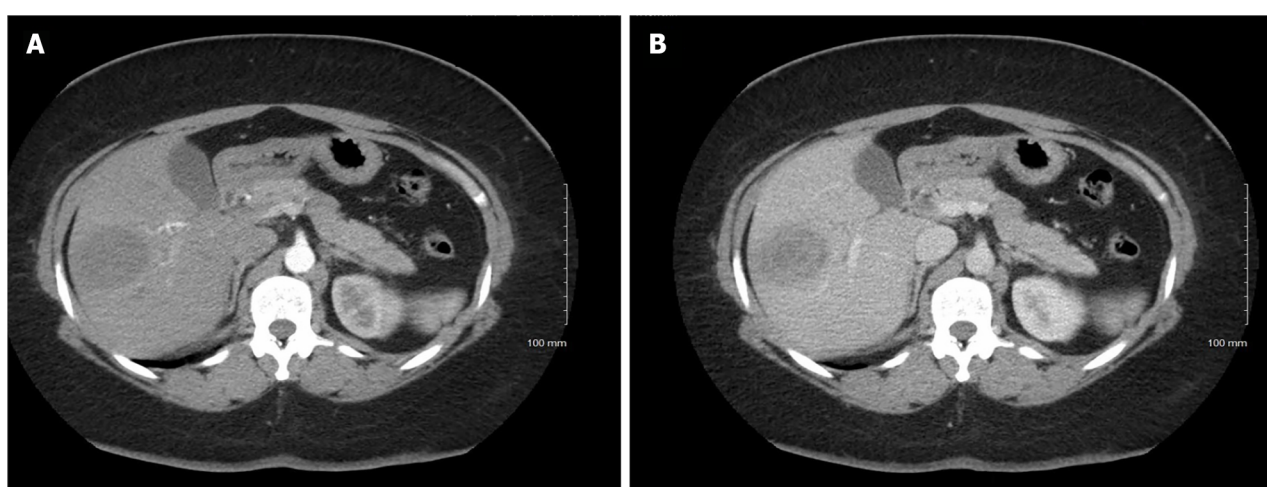
Inflammatory pseudotumor can be idiopathic, infectious, or inflammatory in nature; yet, there are several clinical features of hepatic pseudotumors that suggest an infectious etiology. We present a case of infectious hepatic pseudotumor caused by *S. stercoralis*. The systemic symptoms of anorexia and malaise, coupled with marked eosinophilia, positive *Strongyloides* serum antibodies, and the presence of granulomas with necrosis on pathologic evaluation, along with clinical improvement with antihelminth therapy, support this rare diagnosis and highlight the importance of considering such infectious etiologies in the management of hepatic masses.

To date, the medical literature includes only two published reports of *Strongyloides* hepatic pseudotumor[7,8], which were diagnosed in patients with immunocompromise secondary to HIV or steroid use, respectively. While expected to be rare in the United States, the true prevalence of *Strongyloides* is not known. In the southeastern United States, particularly Appalachia, *Strongyloides* seroprevalence may approach 2%, and may be as high as 10% in South Carolina[9] - where the patient had previously resided. More commonly, *Strongyloides* is endemic to the soil of tropical and subtropical countries, where the nematode larvae migrate through the skin into human hosts and mature to the adult stage[10-12]. *Strongyloides* can perpetually reinfect a patient as the rhabditiform larvae develop into infective filariform larvae and pass into the feces. Strongyloidiasis may remain asymptomatic for many years in immunocompetent patients[10], whereas hyperinfection syndrome and disseminated strongyloidiasis are more common among immunosuppressed hosts[11,12]. In healthy individuals, nematodes have been shown to stay in the body for over 50 years without causing symptoms. It is plausible that this particular case represents a delayed presentation of a clinical exposure years prior with a long period of asymptomatic infection.



DOI: 10.4254/wjh.v15.i12.1338 Copyright ©The Author(s) 2023.

Figure 1 Representative magnetic resonance imaging of hepatic pseudotumor. Gadolinium-enhanced magnetic resonance imaging demonstrated a peripherally T2 hyperintense and centrally T2 hypointense lesion.

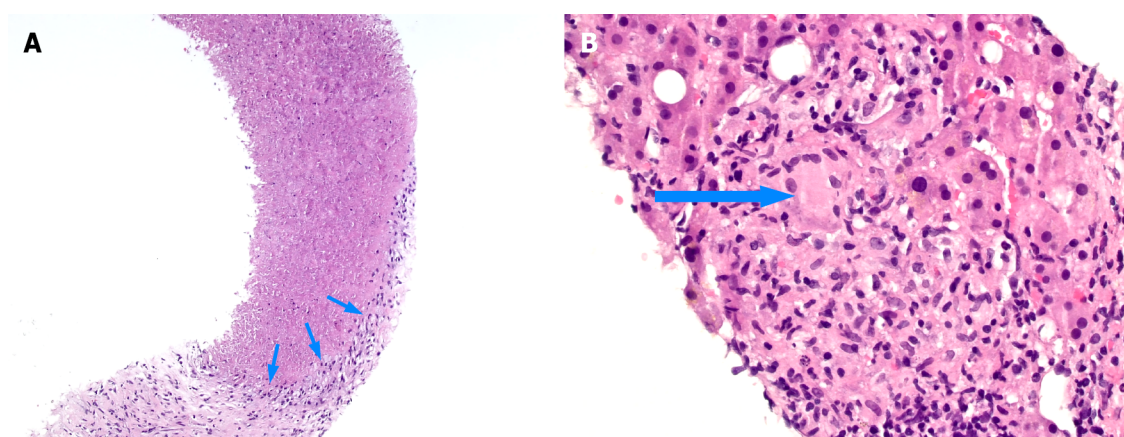


DOI: 10.4254/wjh.v15.i12.1338 Copyright ©The Author(s) 2023.

Figure 2 Representative computerized tomography imaging of hepatic pseudotumor. A-B: Computerized tomography scan through the abdomen with arterial (A) and venous (B) phases demonstrated a low-density mass within the right hepatic lobe measuring up to 4.5 cm × 4.9 cm in largest dimensions. There was peripheral enhancement with centripetal filling; however, the enhancement was contiguous without significant nodularity and thus atypical for a hemangioma.

Other etiologies of infectious hepatic pseudotumor include *Mycobacterium tuberculosis*, *Brucella*, *Bartonella*, *syphilis*, *Candida*, and *Actinomyces*. *Tuberculosis* can cause a localized hepatic lesion, either as a tuberculoma with confluent granulomas and few organisms in immune competent individuals, or a tuberculous abscess, which is often suppurative and contains numerous organisms in immunocompromised hosts[13]. A *Brucelloma* is typically seen in the context of reactivation of remote infection, usually originating from animals or animal products from high-risk regions, typically surrounding the Mediterranean[14]. The clinical presentation of hepatic *Brucelloma* overlaps with many of the symptoms of brucellosis, typically chronic, undulating fevers, malaise, and anorexia. There may be right upper quadrant pain. Diagnosis of hepatic *Brucelloma* is validated by serological positivity and positive cultures on blood or tissue samples. Hepatic cat scratch disease, caused by *B. henselae*, may present with abdominal lymphadenopathy and skin papules. It can form irregular stellate hepatic abscesses, histologically characterized by palisading histiocytes, lymphocytes, and a rim of fibrosis[13]. Hepatic syphilis can occur in both immunocompetent and immunocompromised patients[15-17]. Immunohistochemical stain for *Treponema pallidum* highlights the organisms in these pseudotumors; additionally, there is often periductal edema and neutrophilic pericholangitis in portal tracts outside the mass lesion. Hepatic *candidiasis* is most often observed in immunocompromised patients[18] and on histologic examination, may show granulomas or abscesses containing pseudohyphae and/or yeast forms[13]. Lastly, *Actinomyces* can cause hepatic fibrotic masses containing characteristic sulfur granules and colonies of filamentous organisms[19,20]. In summary, patient history and exposures are important considerations when considering these infectious causes of hepatic pseudotumor in order to guide further testing for diagnostic confirmation.

While most hepatic pseudotumors are presumed to be a response to infection, the causative agent is not always identified. Additionally, there are several malignancies and inflammatory diseases that must be considered. Beyond primary (*i.e.* cholangiocarcinoma and hepatocellular carcinoma) and metastatic liver tumors, there are several neoplasms that particularly resemble hepatic pseudotumor. These include inflammatory myofibroblastic tumor (IMT), follicular



DOI: 10.4254/wjh.v15.i12.1338 Copyright ©The Author(s) 2023.

Figure 3 Histologic examination of the liver lesion. A: The biopsy shows loose fibrous tissue (bottom) with large areas of necrosis rimmed by histiocytes (arrows), hematoxylin and eosin stain (100 ×); B: The adjacent viable hepatic parenchyma contains few non-necrotic granulomas. The arrow denotes a multinucleated giant cell within the granuloma, hematoxylin and eosin stain (400 ×).

dendritic cell tumor, and less commonly, Hodgkin lymphoma. When in question, further testing on tissue samples can help distinguish these diagnoses (IMT with *ALK* or *ROS1* expression; follicular dendritic cell tumor with *in situ* hybridization for Epstein-Barr virus; Hodgkin lymphoma with immunohistochemical analysis). Non-infectious inflammatory hepatic pseudotumor can be observed in IgG4-related disease. While most commonly manifested as a form of sclerosing cholangitis and thus a mimic of cholangiocarcinoma, IgG4-related hepatic pseudotumors can form adjacent to involved ducts[21,22]. Histologically, IgG4-related hepatic pseudotumors are characterized by numerous IgG4-positive plasma cells on immunohistochemical staining.

Several limitations warrant emphasis. For this case, the *Strongyloides* IgG Ab titer was not available, and was only reported by the laboratory as a binary (*i.e.* positive/negative); thus, we cannot comment on the degree of elevation that may be observed with this subset of hepatic disease. Second, for completeness of the presentation of the history and evaluation of this case, we hoped to include a representative image of the *Strongyloides* larvae from the stool examination, but this could not be provided by the commercial laboratory involved.

CONCLUSION

Hepatic masses are a heterogeneous pathology of malignant, inflammatory and infectious etiologies. This rare case study of hepatic pseudotumor caused by strongyloidiasis infection highlights the broad diversity of diagnoses that need be considered, even in geographic regions and patient cohorts not typical of opportunistic infections. Moreover, the clinical presentation – namely, eosinophilia, positive serum antibodies and necrotic granulomas – review the typical features of *Strongyloides*-induced pseudotumor. Further research is warranted to elucidate the underlying biology of hepatic pseudotumor formation and improve diagnostic accuracy. Recognizing the pivotal role of infections in the differential diagnosis of hepatic pseudotumor is paramount for safeguarding patients from undergoing unnecessary invasive procedures. This approach not only facilitates accurate diagnosis and timely initiation of appropriate treatments but also prevents unwarranted interventions, underscoring the significance of a comprehensive consideration of infections in the diagnostic algorithm of hepatic pseudotumor cases.

FOOTNOTES

Author contributions: Grandhi MS, Alexander HR, and Ecker BL contributed to the conceptualization and supervision of the research; Gialanella JP, Alexander RH, Korpela K, and Ecker BL contributed to data collection and data analysis; Gialanella JP and Ecker BL contributed to the manuscript writing; All authors have read and approved the final manuscript.

Informed consent statement: Informed written consent was obtained from the patient for publication of this report and any accompanying images.

Conflict-of-interest statement: The authors have no conflicts of interest to declare.

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

Open-Access: This article is an open-access article that was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution NonCommercial (CC BY-NC 4.0) license, which permits others to

distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <https://creativecommons.org/licenses/by-nc/4.0/>

Country/Territory of origin: United States

ORCID number: Jessica P Gialanella 0009-0005-1383-4260; Brett L Ecker 0000-0003-2536-9261.

S-Editor: Lin C

L-Editor: Filipodia

P-Editor: Lin C

REFERENCES

- 1 Ntinis A, Kardassis D, Miliaras D, Tsinoglou K, Dimitriades A, Vrochides D. Inflammatory pseudotumor of the liver: a case report and review of the literature. *J Med Case Rep* 2011; **5**: 196 [PMID: 21600001 DOI: 10.1186/1752-1947-5-196]
- 2 Torbenson M, Yasir S, Anders R, Guy CD, Lee HE, Venkatesh SK, Wu TT, Chen ZE. Regenerative hepatic pseudotumor: a new pseudotumor of the liver. *Hum Pathol* 2020; **99**: 43-52 [PMID: 32222461 DOI: 10.1016/j.humpath.2020.03.010]
- 3 Pascariu AD, Neagu AI, Neagu AV, Băjenaru A, Bețianu CI. Hepatic inflammatory pseudotumor-like follicular dendritic cell tumor: a case report. *J Med Case Rep* 2021; **15**: 410 [PMID: 34321082 DOI: 10.1186/s13256-021-02957-5]
- 4 Mahmoud AA. Strongyloidiasis. *Clin Infect Dis* 1996; **23**: 949-52; quiz 953 [PMID: 8922784 DOI: 10.1093/clinids/23.5.949]
- 5 Centers of Disease Control and Prevention: Resources for Health Professionals. Oct 5, 2023. [cited 3 August 2023]. Available from: https://www.cdc.gov/parasites/strongyloides/health_professionals/index.html
- 6 Rodriguez-Hernandez MJ, Ruiz-Perez-Pipaon M, Cañas E, Bernal C, Gavilan F. Strongyloides stercoralis hyperinfection transmitted by liver allograft in a transplant recipient. *Am J Transplant* 2009; **9**: 2637-2640 [PMID: 19843038 DOI: 10.1111/j.1600-6143.2009.02828.x]
- 7 Gulbas Z, Kebapci M, Pasaoglu O, Vardareli E. Successful ivermectin treatment of hepatic strongyloidiasis presenting with severe eosinophilia. *South Med J* 2004; **97**: 907-910 [PMID: 15455986 DOI: 10.1097/01.SMJ.0000139936.20116.A7]
- 8 Centers for Disease Control and Prevention. Strongyloidiasis Infection FAQs. Jul 3, 2023. [cited 29 July 2023]. Available from: https://www.cdc.gov/parasites/strongyloides/gen_info/faqs.html
- 9 Cleveland Clinic. Strongyloidiasis. [cited 29 July 2023]. Available from: <https://my.clevelandclinic.org/health/articles/14074-strongyloidiasis>
- 10 Carpio ALM, Mesecha M. Strongyloides stercoralis Infection. Treasure Island (FL): StatPearls Publishing, 2023
- 11 Mora Carpio AL, Mesecha M. Strongyloides stercoralis Infection. 2023 Feb 7. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan- [PMID: 28613795]
- 12 Schär F, Trostorf U, Giardina F, Khieu V, Muth S, Marti H, Vounatsou P, Odermatt P. Strongyloides stercoralis: Global Distribution and Risk Factors. *PLoS Negl Trop Dis* 2013; **7**: e2288 [PMID: 23875033 DOI: 10.1371/journal.pntd.0002288]
- 13 Masia R, Misdraji J. Liver and Bile Duct Infections. *Diagnostic Pathology of Infectious Disease* 2018; 272-322 [DOI: 10.1016/B978-0-323-44585-6.00011-4]
- 14 Barutta L, Ferrigno D, Melchio R, Borretta V, Bracco C, Brignone C, Giraudo A, Serraino C, Baralis E, Grosso M, Fenoglio LM. Hepatic brucellosis. *Lancet Infect Dis* 2013; **13**: 987-993 [PMID: 24156899 DOI: 10.1016/S1473-3099(13)70197-X]
- 15 Malvar G, Cardona D, Pezhouh MK, Adeyi OA, Chatterjee D, Deisch JK, Lamps LW, Misdraji J, Stueck AE, Voltaggio L, Gonzalez RS. Hepatic Secondary Syphilis Can Cause a Variety of Histologic Patterns and May Be Negative for Treponeme Immunohistochemistry. *Am J Surg Pathol* 2022; **46**: 567-575 [PMID: 34864775 DOI: 10.1097/PAS.0000000000001848]
- 16 DeRoche TC, Huber AR. The Great Imitator: Syphilis Presenting as an Inflammatory Pseudotumor of Liver. *Int J Surg Pathol* 2018; **26**: 528-529 [PMID: 29198144 DOI: 10.1177/1066896917745665]
- 17 Hagen CE, Kamionek M, McKinsey DS, Misdraji J. Syphilis presenting as inflammatory tumors of the liver in HIV-positive homosexual men. *Am J Surg Pathol* 2014; **38**: 1636-1643 [PMID: 24921640 DOI: 10.1097/PAS.0000000000000264]
- 18 Moazzam Z, Yousaf A, Iqbal Z, Tayyab A, Hayat MH. Hepatic Candidiasis in an Immunocompetent Patient: A Diagnostic Challenge. *Cureus* 2021; **13**: e13935 [PMID: 33884224 DOI: 10.7759/cureus.13935]
- 19 Kaplan K, Sarıcı KB, Usta S, Özdemir F, Işık B, Yılmaz S. Primary Hepatic Actinomycosis Mimicking Neuroendocrine Tumor. *J Gastrointest Cancer* 2023; **54**: 294-296 [PMID: 35184235 DOI: 10.1007/s12029-022-00806-2]
- 20 Kothadia JP, Samant H, Olivera-Martinez M. Actinomycotic Hepatic Abscess Mimicking Liver Tumor. *Clin Gastroenterol Hepatol* 2018; **16**: e86 [PMID: 29627425 DOI: 10.1016/j.cgh.2017.09.035]
- 21 Zen Y, Harada K, Sasaki M, Sato Y, Tsuneyama K, Haratake J, Kurumaya H, Katayanagi K, Masuda S, Niwa H, Morimoto H, Miwa A, Uchiyama A, Portmann BC, Nakanuma Y. IgG4-related sclerosing cholangitis with and without hepatic inflammatory pseudotumor, and sclerosing pancreatitis-associated sclerosing cholangitis: do they belong to a spectrum of sclerosing pancreatitis? *Am J Surg Pathol* 2004; **28**: 1193-1203 [PMID: 15316319 DOI: 10.1097/01.pas.0000136449.37936.6c]
- 22 Oseini AM, Chaiteerakij R, Shire AM, Ghazale A, Kaiya J, Moser CD, Aderca I, Mettler TA, Therneau TM, Zhang L, Takahashi N, Chari ST, Roberts LR. Utility of serum immunoglobulin G4 in distinguishing immunoglobulin G4-associated cholangitis from cholangiocarcinoma. *Hepatology* 2011; **54**: 940-948 [PMID: 21674559 DOI: 10.1002/hep.24487]



Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA

Telephone: +1-925-3991568

E-mail: bpgoffice@wjgnet.com

Help Desk: <https://www.f6publishing.com/helpdesk>

<https://www.wjgnet.com>

