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TUBERCULOUS LIVER ABSCESS IN AN IMMUNOCOMPETENT PATIENT: A CASE REPORT

AMANDA FIGUEIREDO GARCIA[1], JORDANA SILVA DIAS[2], TATIELE BARBOZA DOS REIS GOMES[2]

1. Resident at the Federal University of Jataí, Jataí/GO, Brazil
2. Undergraduate student at the Federal University of Jataí, Jataí/GO, Brazil.

ABSTRACT

An immunocompetent male patient presented with severe pain in the right upper quadrant and fever ranging from 39 to 40 °C. Initial laboratory findings were nonspecific. Contrast-enhanced abdominal computed tomography revealed a hepatic abscess, for which videolaparoscopic drainage and collection of purulent material were performed. Culture identified Mycobacterium tuberculosis, and the Rapid Molecular Test for Tuberculosis (TRM-TB) was positive, demonstrating rifampicin sensitivity and confirming the diagnosis of hepatic tuberculosis. The patient was started on standard antituberculous therapy, showing progressive clinical improvement and being discharged after 21 days. This case highlights the diagnostic challenge of extrapulmonary tuberculosis in immunocompetent individuals and emphasizes the importance of including this etiology in the differential diagnosis of hepatic abscesses, particularly in endemic regions.

Keywords: Colonoscopy, Hepatic tuberculosis, Liver abscess, Extrapulmonary tuberculosis, Immunocompetence, Mycobacterium tuberculosis.

INTRODUCTION

Mycobacterium tuberculosis (M. tuberculosis) is a gram-positive, aerobic bacillus transmitted via aerosols between humans, with patients with pulmonary tuberculosis being the main source of contagion[1] due to the presence of bacilli in sputum. The establishment and development of the disease after exposure are directly associated with the host's immune system capacity to control bacterial proliferation.[2,3] Primary infection is mostly asymptomatic and may remain in a latent state for variable periods.[4] It is reported that disease activation occurs in only 5–10% of individuals exposed to the bacterium during their lifetime, whereas the remaining 90–95% immunologically control M. tuberculosis, resulting in a subclinical condition and, in some cases, complete bacterial elimination.[3]

M. tuberculosis primarily affects the lungs but may spread to other organs due to its lymphatic and hematogenous dissemination capability, causing disease in multiple sites such as the central nervous system, genitourinary tract, osteoarticular system, pericardium, and gastrointestinal tract. Tuberculosis is therefore classified into two major

clinical groups: pulmonary tuberculosis (PTB) and extrapulmonary tuberculosis (EPTB), based on their different clinical manifestations.[5,6]

Pulmonary tuberculosis (PTB) is the most common form of the disease[7], generally developing after an individual's first contact with the bacillus.[1] It typically presents with insidious symptoms such as low-grade fever, night sweats, and loss of appetite, which makes early diagnosis challenging.[1] Extrapulmonary tuberculosis (EPTB), on the other hand, is a severe and disseminated form of the disease that can affect multiple organs, including the liver. Its clinical presentation varies depending on the site involved and may include nonspecific symptoms such as fever, weight loss, fatigue, abdominal pain, and asthenia.[7]

It has been observed that factors such as immunosuppression and social conditions, including malnutrition, are associated with the manifestation and severity of extrapulmonary forms[1]. In HIV-negative patients, the most common extrapulmonary manifestation is pleural tuberculosis.[1] Hepatic tuberculosis, however, is a rare form of extrapulmonary tuberculosis, with an atypical and nonspecific presentation, especially when it occurs in isolation.[8]

EPIDEMIOLOGY

This Among visceral abscesses, hepatic abscess is the most frequent, as the liver is particularly susceptible to the formation of purulent collections, whether solitary or multiple. Its annual incidence is estimated at approximately 2.3 cases per 100,000 inhabitants and is more prevalent in men than in women. Despite its relative rarity and nonspecific clinical presentation, it is a condition associated with high morbidity and mortality, reaching around 15% in Western countries.[9,10]

Tuberculous liver abscess, in turn, is an uncommon clinical entity, with approximately 100 cases described in the medical literature. Its incidence, based on hospital records, ranges from 0.029% to 1.47%, whereas autopsy studies report a prevalence between 0.3% and 1.4%.[11,12]

Hepatic and biliary involvement by Mycobacterium tuberculosis occurs most frequently in association with miliary pulmonary tuberculosis, although the gastrointestinal tract may also serve as an entry point for the bacillus. Autopsy studies demonstrate simultaneous involvement of the liver and spleen in 80–100% of extrapulmonary tuberculosis cases. The primary form of hepatic tuberculosis, occurring in the absence of systemic dissemination, accounts for less than 1% of all reported cases.[10,11]

Hepatobiliary tuberculosis shows a male predominance of approximately 2:1. The most common symptoms include abdominal pain, hepatomegaly, jaundice, fever, and chills. Hepatomegaly is the most frequent clinical finding, reported in 94–100% of cases, and the liver may present as nodular on palpation in about 55% of patients, mimicking hepatic neoplasia. Splenomegaly is observed in 25–57% of cases, while jaundice occurs in approximately 35% of patients. Alterations in liver enzymes—elevated aspartate aminotransferase (AST) and alanine aminotransferase (ALT)—are common, described in 91–94% of cases, often associated with hypoalbuminemia and hyperglobulinemia.[9,12]

DIAGNOSIS

The diagnosis of tuberculous liver abscess is based on a combination of imaging methods and specific laboratory tests. Among the most commonly used imaging examinations

are abdominal ultrasound (US) and computed tomography (CT) of the upper abdomen, which allow the detection of suspicious hepatic lesions. However, clinical manifestations are nonspecific—including low-grade fever (37.3°C to 37.8°C), right upper quadrant pain, hepatomegaly, weakness, and night sweats—which frequently hinders early diagnosis.[9,13]

Hepatomegaly is a common finding and is usually associated with elevated alkaline phosphatase, while transaminases often remain within normal limits. Relevant differential diagnoses include ruptured amebic liver abscess and pyogenic abscess secondary to colonic diverticulitis or appendicitis, which may present with similar CT characteristics.[13,14]

Jaundice may occur due to tuberculosis invading the biliary system, leading to ductal obstruction and mimicking hilar neoplasms. Other pathological formations, such as hepatic tuberculomas, may also complicate the differential diagnosis, especially when associated with portal hypertension or hemobilia.[14,15]

Both US and CT have limited specificity for hepatic tuberculosis. Therefore, a triphasic liver CT scan—including arterial, portal venous, and delayed phases—is recommended to improve characterization of space-occupying hepatic lesions.[12] These examinations are essential for determining the location, size, and multiseptated nature of the abscess, in addition to identifying the different evolutionary stages of the disease—from granulomas with or without caseous necrosis to fibrosis and calcification in the scarring phases.[13]

Definitive diagnosis is established by isolating acid-fast bacilli (AFB) in aspirated abscess samples, by the growth of Mycobacterium tuberculosis in culture, or through molecular tests—such as the Rapid Molecular Test for Tuberculosis (RMT-TB)—which demonstrate high sensitivity and specificity.[11,12,14]

TREATMENT

The therapeutic approach to tuberculous liver abscess involves both clinical and surgical measures, depending on the extent of the lesion and the patient's response to drug therapy.[15,16]

Clinical management follows the standardized protocol of the Brazilian Ministry of Health for drug-sensitive tuberculosis, based on the combination of isoniazid (INH), rifampicin (RIF), pyrazinamide (PZA), and ethambutol (EMB), known by the acronym RHZE. The regimen consists of a two-month intensive phase with RHZE, administered as a fixed-dose combination (FDC)—each tablet containing 150 mg of rifampicin, 75 mg of isoniazid, 400 mg of pyrazinamide, and 275 mg of ethambutol—followed by a four-month maintenance phase with the RH regimen (isoniazid and rifampicin). This treatment is recommended for all forms of tuberculosis in patients older than 10 years of age, except in cases of neurotuberculosis.[17,18]

Surgical treatment is indicated in cases where clinical management fails, when there are large purulent collections, or when there is a risk of abscess rupture. The main modalities include open, laparoscopic, or percutaneous drainage, the latter being guided by ultrasound or computed tomography. Intraoperative ultrasound can be particularly useful for the precise localization of abscess cavities, optimizing drainage and reducing the risk of complications.[15,17]

Adequate control of the pyogenic cavity is essential for therapeutic success and for preventing recurrence. Postoperative follow-up should include regular clinical evaluation with monitoring of symptoms such as fever and pain, as well as laboratory assessment

Figure 2. (A) Patient positioning for pre-oxygenation. (B) Video laryngoscopy.

(complete blood count, CRP) and serial imaging—preferably ultrasound—to confirm the complete regression of the lesion.[18,19]

CASE REPORT

A 55-year-old male patient, weighing 70 kg, with a history of type 2 diabetes mellitus (T2DM), hypertension, former smoker (40 pack-years, quit 2 years ago), and former chronic alcohol user (40 years of consumption, quit 2 years ago). Current medications included losartan 50 mg every 12 hours, metformin 850 mg once daily, and glibenclamide 5 mg. He presented to the emergency department with a 7-day history of severe right upper quadrant pain and fever ranging from 39–40°C, with no improvement after the use of simple antipyretics or after 72 hours of empiric antibiotic therapy (azithromycin) prescribed at another facility. He denied nausea, vomiting, diarrhea, and urinary tract symptoms.

On initial medical evaluation, the patient was in regular general condition, febrile, alert, conscious, and hemodynamically stable. Physical examination revealed abdominal tenderness on palpation in the right upper quadrant, with no palpable organomegaly. Medications were administered for pain and fever control, and additional diagnostic tests were requested. Contrast-enhanced CT imaging showed a hypodense lesion with irregular borders in hepatic segment VI, without contrast enhancement, measuring 7.7 × 4.8 × 6.4 cm (estimated volume of approximately 120 mL), associated with a minimal amount of free fluid in the pelvis, and no regional lymphadenopathy. Laboratory results: leukocytosis of 18,890 (neutrophils 83%, lymphocytes 7%), platelets 45,000, hemoglobin 12.20 g/dL, ALT 657.0 U/L, AST 687.0 U/L, alkaline phosphatase 369 U/L, GGT 642 U/L, creatinine 2.5 mg/dL, urea 72 mg/dL, sodium 132.0 mEq/L, lactate 2.3 mmol/L, D-dimer > 4.0, glycated hemoglobin 12.1%. Serologies (HBsAg, VDRL, anti-HIV, anti-HCV) were all negative.

A diagnostic hypothesis of liver abscess was established. Azithromycin was discontinued, and empiric antibiotic therapy with ceftriaxone, metronidazole, and piperacillin/tazobactam (Tazocin) was initiated. In addition, a therapeutic and diagnostic surgical intervention—videolaparoscopic drainage of the abscess—was performed, including hepatotomy with drainage of a moderate amount of purulent material and placement of a drain in the abscess cavity. The purulent material collected during surgery was sent to the laboratory. Analysis isolated Escherichia coli (E. coli) (83,000 CFU), and the Rapid Molecular Test for Tuberculosis (RMT-TB) was positive, demonstrating rifampicin sensitivity.

After the procedure, the patient continued to present with persistent fever, with only subtle laboratory improvement (GGT 507 U/L, alkaline phosphatase 228 U/L, AST 23 U/L, ALT 31 U/L, leukocytosis 10,900 with 81% neutrophils). A contrast-enhanced CT scan was performed for postoperative evaluation following abscess drainage, showing a heterogeneous, amorphous surgical cavity with septations, measuring 13 × 9 × 10 cm (average volume 611 cm^3), containing spontaneously hyperdense foci suggestive of blood content and gas bubbles, with no evidence of abnormal contrast enhancement. The drain was in an appropriate position. A reoperation via laparotomy was then performed for repeat drainage of a septated hepatic abscess, with replacement of the Penrose drain and new collection of material for culture, which resulted positive for Enterococcus faecium (E. faecium).

Following the positive RMT-TB in the hepatic abscess sample, tuberculosis screening was

conducted. The patient had no respiratory symptoms and no epidemiological risk factors. He underwent analysis of two sputum samples for AFB and RMT-TB, both negative. Chest imaging showed no abnormalities. Pulmonary tuberculosis (PTB) was therefore excluded.

Because the abscess sample demonstrated mixed flora, a diagnosis of extrapulmonary tuberculosis (EPTB) presenting as a tuberculous and bacterial hepatic abscess was established. The patient was evaluated by the Infectious Diseases service, which recommended treatment with ceftriaxone plus metronidazole, in addition to the RHZE regimen. The patient remained hospitalized for 21 days for inpatient treatment. After progressive clinical improvement, remaining afebrile for more than 48 hours, and showing improvement in laboratory parameters (Hb 10.5 g/dL, leukocytosis 11,520 with 75% neutrophils and 13% lymphocytes, platelets 369,000, CRP 11.7 mg/L, AST 16 U/L, ALT 20 U/L, total bilirubin 0.83 mg/dL, direct bilirubin 0.43 mg/dL, and indirect bilirubin 0.40 mg/dL), and following multidisciplinary discussion, hospital discharge was proposed. Outpatient treatment with ciprofloxacin and metronidazole for 4–6 weeks was recommended, depending on clinical and laboratory reassessment and monitoring of the hepatic lesion. The standardized Brazilian RHZE regimen was continued. The patient was referred for follow-up in the Infectious Diseases and General Surgery outpatient clinics.

DISCUSSION

The case presented involves a rare pathology with nonspecific clinical features described in the literature but associated with high morbidity and mortality, reaching approximately 15% in Western countries. It is also considered a rare—though not exceptional—form of extrapulmonary tuberculosis. Most reported cases are associated with miliary pulmonary tuberculosis and spread primarily through hematogenous dissemination.[10] Bestow published the first scientific description of tuberculous liver abscess (TLA) in 1858. In one of his randomized studies, he demonstrated that 0.34% of patients with hepatic TB ranged in age from 6 months to 72 years, with a mean age of 39.2 years—an age distribution that closely approximates that of the patient in the present case. Another epidemiological aspect consistent with the literature is the higher prevalence in males, as studies show that among 2.3 cases per 100,000 people, the condition is more frequent in men.[10,20]

Tuberculosis is not commonly encountered in routine clinical practice because M. tuberculosis infections are typically controlled by the host's immune system, meaning that clinical manifestations of TB generally occur in high-risk groups with continuous exposure or in patients with some degree of immunodeficiency.[2,0] In extrapulmonary presentations, this becomes even more epidemiologically evident.[17,19] Therefore, the patient developed an unusual infection in an even rarer anatomical site (the liver), considering that the incidence of hepatic abscesses, based on hospital records, ranges from 0.029% to 1.47%, while autopsy studies report a prevalence between 0.3% and 1.4%.[18,20]

Reviewing the clinical history, the patient did not present clear immunosuppression, given the exclusion of HIV co-infection (negative serologies) and the absence of other comorbidities directly affecting immune function. Nevertheless, he did present an important factor that could justify impaired immunomodulation in controlling tuberculosis: decompensated type 2 diabetes mellitus, with an admission glycated hemoglobin level of 12.1 mg/dL. The immune-modulating impact of hyperglycemia is well established in the literature.[21,22]

Type 2 diabetes mellitus, a chronic disease marked by a slow and progressive increase in inflammatory dysregulation, is associated with elevated levels of pro-inflammatory cytokines (IL-6 and IL-1β), which in turn lead to an increase in macrophage activity. This imbalance results in higher levels of pro-apoptotic factors and heightened inflammatory stimulation.[23] This mechanism may help explain the development of the hepatic abscess, considering that the form presented by this patient accounts for less than 1% of all cases of extrapulmonary tuberculosis (EPTB).[17,19] Thus, it can be inferred that the patient did not have full immunocompetence, which facilitated the unusual presentation of tuberculosis as a hepatic abscess in an HIV-negative individual.

The tuberculous liver abscess, as illustrated in this case, results from microbiological contamination of the hepatic parenchyma, which can occur via the biliary ducts, arterial or portal vessels, or directly. The etiology of pyogenic collections—which may originate from agents such as Mycobacterium tuberculosis, Staphylococcus aureus, protozoa, and helminths—highlights the significant potential for mixed infections, as confirmed in this patient by the presence of both E. coli and M. tuberculosis.[10]

Both ultrasound (US) and computed tomography (CT) are accurate for assessing the location, size, and multiseptated nature of a tuberculous liver abscess. The use of US, in addition to CT, not only as a diagnostic tool but also for follow-up, has been highlighted in many case reports of tuberculous liver abscess (TLA). This preference is mainly justified by its speed, cost-effectiveness, easy accessibility, and the absence of radiation exposure for the patient. Therefore, this method could have been better utilized both for diagnosis and especially for follow-up in the case presented.[24,25]

Although sputum samples in this case tested negative for acid-fast bacilli (AFB), studies indicate that detection is more easily achieved in caseous necrotic tissue; however, its absence should not completely exclude the diagnosis, particularly in regions with high TB incidence[11,15]. In the case evaluated, however, the combination of factors (clinical evaluation and imaging showing no signs of pulmonary involvement), along with negative AFB results, allowed pulmonary TB to be excluded as a diagnostic possibility.

The initial empiric antibiotic therapy with azithromycin was inappropriate, even in the absence of a confirmed diagnosis of TLA, as this macrolide is better suited for bacterial infections of the respiratory tract, skin, soft tissues, ears, oropharynx, and certain sexually transmitted infections, limiting its effectiveness in hepatic infectious conditions such as liver abscesses.[25,26]

Some studies argue that the dense fibrous tissue surrounding abscesses and their substantial size may prevent adequate penetration of antibiotics into the lesion. For this reason, a six-month four-drug antituberculosis regimen (RHZE) is considered the most appropriate therapy and was successfully used in our patient, in conjunction with appropriate antibiotic coverage, resulting in clinical improvement and resolution of the TLA. In addition, because of the mixed bacterial infection, antibiotic therapy with ceftriaxone and metronidazole was appropriately used to target E. coli and E. faecium.[22,26]

CONCLUSION

Tuberculous liver abscess represents an extremely rare manifestation of extrapulmonary tuberculosis and is often difficult to diagnose due to its nonspecific clinical and laboratory

presentation. This case report highlights the importance of clinical suspicion in patients with hepatic abscesses refractory to conventional treatment, particularly in individuals with comorbidities that compromise immune response, such as uncontrolled type 2 diabetes mellitus.

Definitive diagnosis through the Rapid Molecular Test for Tuberculosis (RMT-TB) was essential for guiding appropriate therapy, underscoring the role of molecular methods as indispensable tools in current clinical practice. Combined management—including surgical drainage and targeted antimicrobial therapy, particularly the RHZE regimen—proved effective in resolving the condition and preventing complications.

This case reinforces the need for a multidisciplinary approach and broad etiological investigation in hepatic abscesses of indeterminate origin, including tuberculosis as a differential diagnosis even in the absence of pulmonary involvement. Early identification and appropriate treatment are crucial for achieving favorable outcomes and reducing the morbidity and mortality associated with this rare condition.

REFERENCES

1. Barros ERS, Mello IGKA, Franco CJM, Dodó EA, Lima DGL, Lima MFS. Tuberculose pulmonar e extrapulmonar no contexto clínico e terapêutico. Brazilian Journal of Health Review. 2025 Mar;8(2):e78079–e78079.

2. Li Z, Yang L, Li L, Ye Z, Gong W. Mycobacterium tuberculosis: Immune Response, Biomarkers, and Therapeutic Intervention. MedComm. 2024 Jan;5(1):e419.

3. Orgeur M, Sous C, Madacki J, Brosch R. Evolution and emergence of Mycobacterium tuberculosis. FEMS Microbiol Rev. 2024 Mar 1;48(2):fuae006

4. Rezende TCGD, Vitorino VBS, Borges DS, Castro LFG, Braz RF, Camilo RP, Vieira GA. Tuberculose hepática: relato de caso de um quadro raro e de difícil diagnóstico. Sociedade brasileira de clínica médica. 2024 Nov;22(1):45-50.

5. Lima ACC, Galvão ABZ, Barros GE, Rabello JC, Quintiere BBH, Ferreira IMO, Gonçalves ABM, Napoli AER. Tuberculose Extrapulmonar. Guia Prático Clínica Médica. editora pasteur. 2024 Mar;50: 288–296.

6. Figueiredo AVC, Duarte AO, Vieira MRV, Paranhos SVO, Valadão AF, Magalhães FA. Perfil clínico e epidemiológico dos pacientes com tuberculose extrapulmonar no município de Ipatinga-MG. BJSCR. Jun-ago 2021;35(1):2317–4404.

7. Araújo FGA, Saotome L, Pierini JLG, Ruivo GF. Tuberculose extrapulmonar com envolvimento renal e intestinal: um desafio no diagnóstico precoce. Research, Society and Development. 2023 Dec;12(14): e80121444516-e80121444516.

8. Kumar SV, Senthil N, Rajendran V, Logithasan N, Chenguttuvan A. Extra pulmonary tuberculosis manifesting as liver abscess. Cureus. 2024 Oct;16(10): e70988.

9. Brito AS, Bezerra LC, Cruz IS, Roque HP, Aguiar ABVC, Nogueira MTM, Leite LMBF. Management of hepatic abscesses: an integrative review. Amadeus Int Multidiscip J. 2023;7(13):5-23.

10. Kim JY, Kim SH, Lee JY, Lee YJ, Han JK. A population-based study of pyogenic liver abscess in Korea: incidence, mortality and temporal trends during 2007–2017. J Clin Med. 2021;10(16):3558.

11. Karaosmanoglu AD, Onur MR, Sahani DV, Tabari A, Karcaaltincaba M. Hepatobiliary tuberculosis: imaging findings. AJR Am J Roentgenol. 2016 Oct;207(4).

12. Agarwala R, Dhooria S, Khaire NS, Mishra S, Verma S, Shah J, Mandavdhare HS, Kumari S, Dutta U, Sharma V. Xpert MTB/RIF for diagnosis of tubercular liver abscess. A case series. Infez Med. 2020 Sep 1;28(3):420-424.

13. Yang C, Liu X, Ling W, Song B, Liu F. Primary isolated hepatic tuberculosis mimicking small hepatocellular carcinoma: A case report. Medicine (Baltimore). 2020 Oct 9;99(41):e22580.

14. Lobato PRN, Cruz MFS, Macedo BFS, Quaresma TR, Alves GRM, Teixeira JPAO, Valente MA, Hermes MF, Peres AVS, Santos DW. Hepatic abscess due to tuberculosis in an HIV patient: a case report. Braz J Health Rev. 2020;3(4):7296-7300.

15. Rezende TCGD, Vitorino VBS, Borges DS, Castro LFG, Braz RF, Camilo RP, Vieira GA. Tuberculose hepática: relato de caso de um quadro raro e difícil diagnóstico. Rev Soc Bras Clin Med. 2024;22(1):45-50

16. Ha CW, Shin SD, Goh MJ, Song BG, Kang W, Sinn DH, Gwak GY, Paik YH, Choi MS, Lee JH. Miliary Tuberculosis Initially Presenting as an Isolated Hepatic Abscess. Korean J Gastroenterol. 2025 Jan 25;85(1):78-82.

17. Sharma V, Ahuja V. Hepatic tuberculosis. In: Treatment and Management of Tropical Liver Disease. Elsevier. 2025 Jan;13:104-110.

18. Malewadkar S, Shetty V, Vyas S, Doctor N. Undiagnosed Hepatic Tuberculosis Presenting With Intraabdominal Rupture and Hemoperitoneum, Requiring Urgent Hepatic Resection. J Clin Exp Hepatol. 2021 Jul-Aug;11(4):506-510.

19. Rabahi MF, Silva Júnior JLR, Ferreira ACG, Tannus-Silva DGS, Conde MB. Tratamento da tuberculose. J Bras Pneumol. 2017;43(5):472-486.

20. Lardière-Deguelte S, Ragot E, Amroun K, Piardi T, Dokmak S, Bruno O, Appere F, Sibert A, Hoeffel C, Sommacale D, Kianmanesh R. Hepatic abscess: Diagnosis and management. J Visc Surg. 2015 Sep;152(4):231-43.

21. Mikkelsen RR, Hundahl MP, Torp CK, Rodríguez-Carrio J, Kjolby M, Bruun JM, Kragstrup TW. Immunomodulatory and immunosuppressive therapies in cardiovascular disease and type 2 diabetes mellitus: A bedside-to-bench approach. Eur J Pharmacol. 2022 Jun;15;925:174998.

22. Losie JA, Lam JC, Gregson DB, Parkins MD. Epidemiology and risk factors for pyogenic liver abscess in the Calgary Health Zone revisited: a population-based study. BMC Infect Dis. 2021 Sep 10;21(1):939.

23. Kim JY, Kim SH, Lee JY, Lee YJ, Han JK. A population-based study of pyogenic liver abscess in Korea: incidence, mortality and temporal trends during 2007–2017. J Clin Med. 2021 Nov;10(16):3558.

24. Devi S, Mishra P, Sethy M, Thakur GS. Isolated Tubercular Liver Abscess in a Non-immunodeficient Patient: A Rare Case Report. Cureus. 2019 Dec 3;11(12):e6282.

25. Singh D, Bhatt B, Gupta S, Kumar R. Isolated hepatic tuberculosis: a rare entity in an immunocompetent patient – case report and review of literature. Cureus. 2022;14(3):e22937.

26. Ali N, Ahmad S, Alam S, Ansari J, Naeem M, Shahid M. Primary tuberculous liver abscess: an uncommon presentation in an immunocompetent host. Case Rep Infect Dis. 2022;2022:8945731.

MAILING ADDRESS

AMANDA GARCIA
Rua Dona Julia Prado, 387,C1, Serra Azul, Q10,L07, Jataí/GO - Brazil.
E-mail: dra.amandagarcia@gmail.com

EDITORIAL AND REVIEW

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