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Pseudotumoral Form of Primary Hepatic Tuberculosis: A Rare Case Report with Review of the Literature

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1. Abstract

The isolated location of tuberculosis is very rare constituting less than 1% of all locations. We report through this observation the case of a 23-year-old patient, who complains of chronic pain in the right hypochondrium in a context of deterioration of the general condition. The biological assessment showed a biological inflammatory syndrome. A thoracic-abdominal-pelvic computed tomography (CT) showed an enlarged liver, with regular contours and multiple hypodense nodular formations. An exploratory laparoscopy was performed. The anatomy pathological study of the liver biopsies showed necrotizing epithelioid cell granuloma lesions without histological signs of malignancy. The patient was put on anti-bacillary drugs. with good improvement. Diagnosing hepatic tuberculosis is a challenge for the clinician even in highly endemic countries, given the rarity of this localization.

2. Introduction

Isolated hepatic localization of tuberculosis is very rare representing less than 1% of all localizations [1]. The pseudotumoral form constitutes a particular presentation, characterized by the presence of radiological features resembling a primary or secondary hepatic tumor, and only the histological study of liver biopsies will allow to differentiate between these two entities. We report a case of a pseudotumoral form of isolated hepatic tuberculosis in a 23-year-old patient, whose imaging findings suggested a secondary hepatic tumor localization. Only the histological study of liver biopsies allowed to retain the diagnosis of tuberculosis.

3. Observation

3.1. Patient Information

A 23-year-old patient, vaccinated with BCG (Bacillus Calmette – Guérin vaccine), with tuberculosis contagion (Father treated for pulmonary tuberculosis cured one week before his admission, brother also treated for pulmonary tuberculosis cured a year and a half ago and a sister treated for peritoneal tuberculosis cured 3 months ago), complained for a year of pain in the right hypochondrium, of moderate intensity, intermittent, without other associated digestive manifestations, in particular no abdominal distension, no digestive bleeding externalized, no jaundice, no vomiting, without any associated extra digestive manifestations in particular no respiratory symptoms, all developing in a context of febrile sensations and deterioration of the general state made of weight loss estimated at 10 kg, asthenia and anorexia.

3.2. Clinical Findings

The clinical examination found a patient in poor general condition (weight: 58 kg, height: 1.75 m and body mass index (BMI): 18.93 kg/

m2), a fever (temperature: 38.3°C) and homogeneous, firm and painless hepatomegaly (liver span: 18 cm), without palpable splenomegaly or flank dullness. The pleuropulmonary and lymph node examinations were without abnormalities.

3.3. Diagnostic Assessment

The biological assessment showed a biological inflammatory syndrome (C-reactive protein (CRP) at 85 mg/L and an erythrocyte sedimentation rate (ESR) of 65 at the first hour). The rest of the assessment did not reveal any other abnormalities, notably no leukopenia (8120/mm3) or lymphopenia (1640/mm3), the hepatic enzymes and renal function were normal, and the prothrombin level was 63%. Hydatid, hepatitis B, C and HIV serologies as well as tumor markers (alpha feto protein, CA 19-9 and carcinoembryonic antigen) were negative. An abdominal ultrasound showed a homogeneous liver increased in size measuring 19 cm in regular contours, with the presence of several well-limited rounded hypoechoic nodular formations that did not light up on Doppler, measuring the largest 1.4 x 0.9 cm at segment IV, associated with a homogeneous splenomegaly measuring 14.7 x5.7 cm, and lymphadenopathy of the hepatic pedicle measuring 15 mm for the largest (Figure 1).

A complementary thoracic-abdominal-pelvic computed tomography (CT) showed an enlarged liver (21.8 cm), with regular contours and multiple nodular, hypodense formations, enhancing at the periphery after injection of contrast product measuring for the larger 2.5 x 1.3 cm visible at segment IV, associated with a hilar lymphadenopathy measuring 2 x 1.6 cm, and an enlarged spleen, measuring 11 x 13 cm homogeneous. Without other thoracic and abdomino-pelvic abnormalities (Figure 2).

The tuberculosis tests performed included the search for Koch's bacilli in the sputum, which was negative, and a genomic amplification (Gene-Xpert) of the sputum, which did not show the presence of Acid-Alcohol-Fast Bacilli (AAFB). However, the QuantiFERON-TB Gold test was strongly positive. An exploratory laparoscopy was performed after inconclusive results of an echo-guided liver biopsy. The exploration showed a dysmorphic liver without macroscopically detectable nodular lesions, a slightly enlarged spleen, and hepatic hilum adenopathy measuring 1.5 x 1 cm. The peritoneum, greater omentum, small intestine, and mesentery were normal in appearance (Figure 3). Liver, omentum and peritoneal biopsies were done. The anatomopathological study of the biopsies showed necrotizing epithelioid cell granuloma lesions without histological signs of malignancy. Peritoneal and epiploic biopsies showed chronic inflammatory remodeling without signs of specificity on no visible granulomas or signs of malignancy.

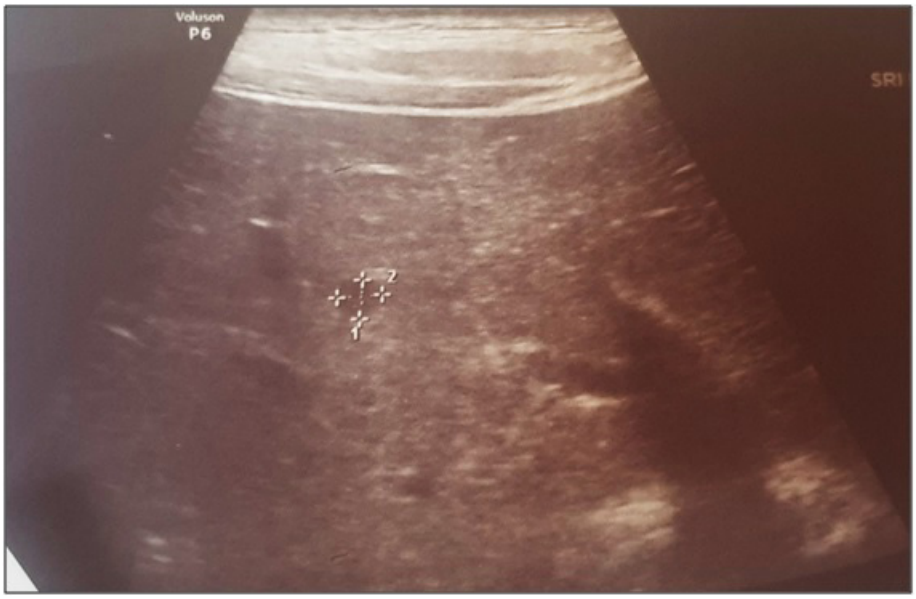

Figure 1: Abdominal ultrasound showing well-defined hypoechoic liver nodules

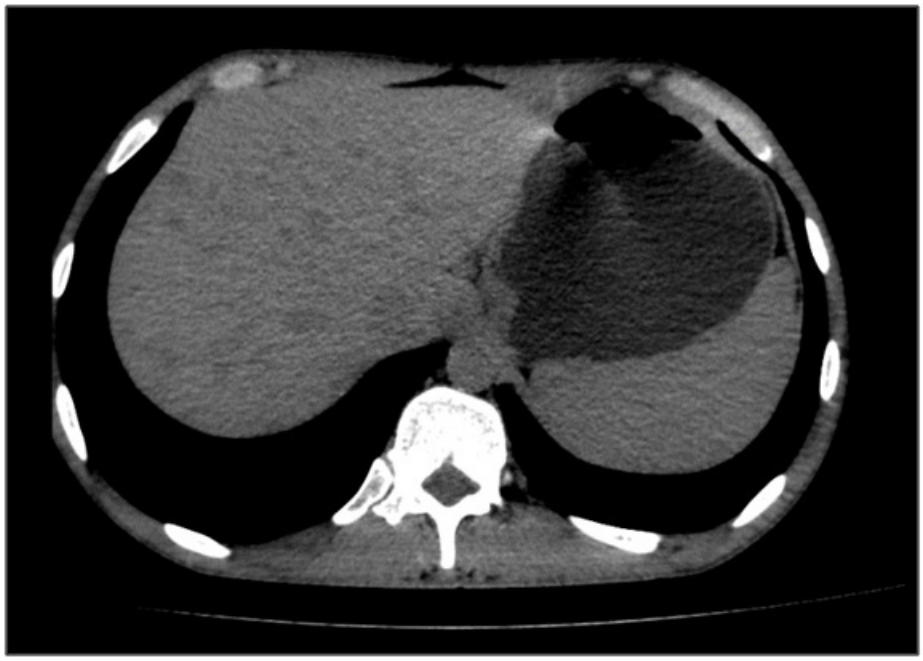

Figure 2: Multiple nodular, hypodense hepatic formations

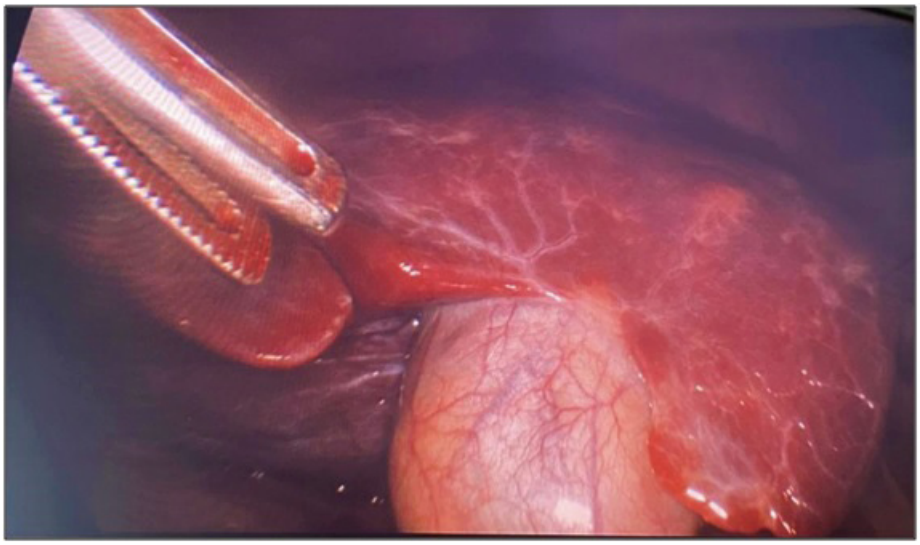

Figure 3: Intraoperative view during exploratory laparoscopy

3.4. Therapeutic Intervention

The patient was put on anti-bacillary drugs combining Isoniazid 5mg/kg, Rifampicin 10 mg/kg, Ethambutol 25 mg/kg and Pyrazinamide 30 mg/kg for two months and then the combination of Isoniazid with Rifampicin for 4 months.

3.5. Follow-Up and Outcomes

After 4 months of treatment, the clinical and biological evolution was favorable with apyrexia, weight gain and resolution of the biological inflammatory syndrome. A control abdominal ultrasound was performed and showed the disappearance of the previously described hepatic nodular lesions.

4. Discussion

Liver involvement in tuberculosis is usually associated with primary active lung infection or miliary tuberculosis, usually by hematogenous dissemination through the hepatic artery [2, 3]. It occurs in up to 80% of cases of disseminated tuberculosis [4]. Hepatic tuberculosis may present as miliary tuberculosis, pulmonary tuberculosis with liver involvement, focal or abscessed tuberculomas, or tuberculous cholangitis [5], [6]. It can also be classified according to the size of the liver lesions: The most common form is micro nodular involvement, which consists of small nodules 0.5 mm to 2 mm in diameter, which appear in 80 % to 100% of patients with miliary tuberculosis. Less frequent is the presentation in macro nodular or pseudotumoral form, with lesions greater than 2 mm in diameter [6]. The diagnosis of a hepatic localization of tuberculosis is most often unrecognized and confused with a primary or secondary liver tumor [4].

Bristowe was the first to describe the hepatic location of tuberculosis in 1858 [7]. Zipser suggested the term "pseudotumor" to describe the macronodular form of hepatic tuberculosis because of

its resemblance to liver tumors. [8]. Isolated hepatic tuberculosis in the absence of immunosuppression is an extremely rare form of the disease, even in highly endemic countries [9]. Inoculation may occur through the pulmonary or gastrointestinal tract, and enteric dissemination may be a consequence of oral ingestion or part of lymphatic or hematic dissemination. Once the Koch's bacillus lodges in the intestine, it reaches the liver through the portal system. The rarity of the local form may be due to a low oxygen content in the liver tissue, probably inhibiting the growth of the tubercle bacillus [6, 10].

In a series of 85 patients with digestive tuberculosis in our department, hepatic involvement represented only 2.3% of all cases [11]. The rarity of the hepatic form of tuberculosis is confirmed by Chong et al who reported only 14 cases of hepatobiliary tuberculosis among 1888 cases of tuberculosis (0.74%), over a period of ten years [12]. Isolated hepatic localization of tuberculosis is extremely rare, representing in a study of 296 cases only 1.34% of all hepatic forms of tuberculosis [13].

The mean age of patients with hepatic tuberculosis is 30 years with extremes ranging from 17 to 50 years [13]. There is no gender predominance noted, [14] with a higher incidence in the black race [15,16]. The clinical manifestations of hepatic tuberculosis take several forms ranging from the absence of symptoms to a severe hepatitis with jaundice and hepatocellular insufficiency [17]. The most reported symptoms consisting of pain in the right hypochondrium, fever, night sweats, anorexia and weight loss. The clinical examination may be normal. However, he can objectify abdominal tenderness with or without a palpable mass, hepatomegaly or cutaneous-mucous jaundice [18].

The value of biological examinations in the positive diagnosis of hepatic tuberculosis is very limited. A biological inflammatory syndrome is usually noted (the sedimentation rate is almost constantly accelerated. C-reactive protein may also be elevated) [19]. Alkaline phosphatase elevation up to 1.5 to 5 times the upper limit of normal may be found in 50 to 75% of cases [20]. Pancytopenia may be observed in the context of hypersplenism or associated bone marrow involvement. Other less specific abnormalities may be seen, including hypoalbuminemia and hyponatremia [4]. Tuberculin reactions are usually positive, [14] but negative results do not rule out the diagnosis.

Radiological descriptions of hepatic tuberculosis are few and non-specific; they may mimic a primary or secondary tumor. On ultrasonography, liver involvement is most often manifested by one or more nodular formations that are usually hypoechoic and sometimes contain calcifications [4, 9]. A hyperechoic character of the nodules has been described in some cases [21,22]. On CT scan, the lesions are often hypo dense, rounded, without or with weak peripheral enhancement after injection of contrast agent, as was the case in our patient, calcification zones can also be found [21,23]. On MRI, the most frequent appearance is that of lesions appearing hyposignal on T1 sequence and hyposignal, isosignal or hypersignal with a peripheral contour in hyposignal on T2 sequence depending on the stage of the disease. Slight peripheral enhancement after injection of Gadolinium may be seen in the portal and later phases [4, 21].

Even in the presence of clinical, biological and radiological arguments in favor of hepatic tuberculosis, histological confirmation of liver biopsies (by transparietal biopsy or during exploratory laparotomy or laparoscopy) remains indispensable to make a positive diagnosis, even though granulomatous lesions are not specific to tuberculosis and can be found in other pathologies. The histological results of liver biopsies in tuberculosis usually show a necrotizing epithelioid cell granuloma lesions. The search for acid-fast bacilli, Ziehl's staining and culture of tissue samples are rarely positive [22, 24]. The use of genomic amplification techniques (PCR) allows the detection of Mycobacterium tuberculosis on liver biopsy fragments with a sensitivity of between 53 and 92% and a specificity of approximately 70% [4, 25]. Anti-bacillary drugs are the base of treatment for hepatic tuberculosis, and a 6 to 9-month regimen (2 months of isoniazid, rifampicin, pyrazinamide and ethambutol followed by 4 to 7 months of isoniazid and rifampicin) is the most recommended [4, [26].

5. Conclusion

The diagnosis of hepatic tuberculosis is a challenge to the clinician even in highly endemic countries, given the rarity of this location. The clinical and radiological presentation may mimic a primary or secondary hepatic tumor. Histological confirmation remains the key to a positive diagnosis.

References

1. Mouhafid El, Elhjouji F, Fahssi A, Yaka M, Njoumi N. A Rare Localization of Tuberculosis: A Case Report and a Review of the Literature. Surg. Sci. 2021; 12(3): 95-101.
2. Rab SM, Beg MZ. Tuberculous liver abscess. Br J Clin. Pract. 1977; 31(10); 157-158.
3. Biswas BK, Pal S, Moulik DJ. Isolated Hepatic Tuberculoma - A Case Report. Iran J Pathol. 2016; 11(5): 427-430.
4. Chtourou L. Medically Managed Pseudo-Tumoral Form of Hepatic Tuberculosis Hepatic Tuberculosis In Its Pseudotumor Form Treated Medically. Pg No: 1-4.
5. Hindi C. Isolated hepatic tuberculosis: pseudotumoral form in one case. Pg No: 1-4.
6. Kalil AN, Coelho FA, Olm GS. Pseudotumoral hepatic tuberculosis. Rev. Colégio Bras. Cir. 1999; 26: 197-199.
7. Bristowe JS. On the connection between abscess of the liver and gastrointestinal ulceration. 2021.
8. Zipser RD, Rau JE, Ricketts RR, Bevans LC. Tuberculous pseudotumors of the liver. Am J Med. 1976; 61(6): 946-951.
9. Zinebi Ai, Rkiouak A, Akhouad Y, Reggad A, Kasmy Z. Lumpy hepatic tuberculosis: an unusual complication in Wilson's disease. Pan Afr Med J. 2014; 17:22.

10. Achem SR, Kolts BE, Grisnik J, MacMath T, Monteiro CB. Pseudotumoral hepatic tuberculosis. Atypical presentation and comprehensive review of the literature. J Clin Gastroenterol 1992; 14(1): 72-77.
11. Qlilat R, Guennoun N, Krati Et K. Digestive tuberculosis at the Mohammed VI University Hospital in Marrakech in relation to 85 cases", Cadi Ayyad - Marrakech, Faculty of Medicine and Pharmacy - Marrakech, 2008.
12. Chong VH. Hepatobiliary tuberculosis: a review of presentations and outcomes. South Med J. 2008; 101(4): 356-361.
13. Essop AR, Moosa MR, Segal I, Posen J. Primary tuberculosis of the liver-a case report. Tubercle. 1983; 64(4): 291-293.
14. Nassar I, Errabih I, Krami L, Hammani H. DIG-WS-36 Primary hepatic tuberculosis: report of 10 cases. J Radiol. 2007; 88(10): 1538.
15. Hickey N, McNulty JG, Osborne H, Finucane J. Acute hepatobiliary tuberculosis: a report of two cases and a review of the literature. Eur. Radiol. 1999; 9(5): 886-889.
16. Roge F, Berthet B, Nikolajevic Z, Assadourian R. Large tuberculous lymphadenopathy of the hepatic pedicle. Ann Shir. 2000; 125(3): 292-295.
17. Huang WT, Wang CC, Chen WJ, Cheng YF, Eng HL. The nodular form of hepatic tuberculosis: a review with five additional new cases. J Clin Pathol. 2003; 56(11) : 835-839.
18. Hassani KIM. Isolated liver tuberculosis abscess in a patient without immunodeficiency: A case report. World J Hepatol. 2010; 2(9): 354-357.
19. Ai J-W, Li Yi, Cheng Qi, Cui P, Wu H-L. Diagnosis of local hepatic tuberculosis through next-generation sequencing: Smarter, faster and better. Clin Res Hepatol Gastroenterol. 2018; 42(3): 178-181.
20. Wong S, Yuen H, Ahuja A. Hepatic tuberculosis: a rare cause of fluorodeoxyglucose hepatic superscan with background suppression on positron emission tomography. Singapore Med J. 2014; 55(7): e101-e103.
21. Mortele KJ, Segatto E, Ros PR. The Infected Liver: Radiologic-Pathologic Correlation. Radio Graphics. 2004; 24(4): 937-955.
22. Tan TC, Cheung AY, Wan WY, Chen TC. Tuberculoma of the liver presenting as a hyperechoic mass on ultrasound. Br J Radiol. 1997; 70(840): 1293-1295.
23. Yu R-S, Zhang S-Z, Wu J-J, Li R-F. Imaging diagnosis of 12 patients with hepatic tuberculosis. World J Gastroenterol. 2004; 10(11): 1639-1642.
24. McMullan GS, Lewis JH. Tuberculosis of the Liver, Biliary Tract, and Pancreas. Microbiol Spectr. 2017; 5(1).
25. Fan ZM, Zeng QY, Huo JW, Bai L, Liu ZS. Macronodular multi-organs tuberculoma: CT and MR appearances. J Gastroenterol. 1998; 33(2): 285-288.
26. Adnani A, Dafiri R. Hepato-splenic tuberculosis in children. J Radiol. 2005; 86(11): 1710-1711.