

Hepatic Tuberculosis Fistulized into the Thoracic Wall

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Abstract

The hepatic tuberculosis is rare. The interest of this Knowledge consists in the diagnostic problems particularly in its primitive and macronodular forms. It touches all ages with a peak of frequency during 17 - 50 years old, and a female prevalence. The clinical picture is polymorphous but it is still dominated by the prolonged fever and the alteration of the general state.

Keywords: Liver Abscess, Mycobacterium Tuberculosis, Antibacillary.

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1. INTRODUCTION

Hepatic localization of tuberculosis is rare and represents less than 1% of all tuberculosis sites. It can take on different forms. The clinical examination and routine biological tests are not revealing. The positive diagnosis is confirmed by the detection of the Koch bacillus during a direct examination, a culture, and amplification using PCR techniques, and by the detection of granulomas with caseous necrosis in biopsy fragments.

The treatment for primary hepatic tuberculosis is 6 months, including 2 months of triple therapy and 4 months of dual therapy [1].

2. CASE REPORT

A 24-year-old young man, with no particular medical or surgical history, active military personnel, with a known exposure to tuberculosis in the barracks. The onset of his illness dates back to 03 weeks before his hospitalization, marked by the appearance of a right basal-thoracic parietal swelling, associated with pain in the right hypochondrium. Without digestive or respiratory symptoms (no cough, hemoptysis) or night sweats, everything evolves in a context of apyrexia and weight loss amounting to 10 kg in 1 month.

The clinical examination revealed a patient in fairly good general condition, afebrile, normotensive, with conjunctiva of normal color.

The inspection revealed a swelling near the 10th and 12th right ribs, fistulated, which discharges purulent fluid. Palpation revealed a tender hepatomegaly. The lymph node areas were free. The rest of the somatic examination was particular.

The biological evaluation was normal, except for a slightly elevated CRP. The serological tests were negative.

The chest X-ray showed no obvious lesions. An abdominopelvic MRI showed a subcapsular cystic lesion of the right liver in contact with a subcutaneous collection of the right lateral thoracic wall.

The decision for a surgical intervention was made, with the drainage of the subcutaneous collection from the thoracic wall and the collection of a sample for bacteriological analysis.

The result of the GeneXpert on the sample taken came back positive. Thus, the diagnosis of fistulized hepatic tuberculosis in the right thoracic wall was maintained, and anti-bacillary treatment was started for 9 months.

The patient progressed favorably under medical treatment, and the follow-up ultrasound showed a complete resolution of the hepatic collection.



3. DISCUSSION

The average age of patients with hepatic tuberculosis was 30 years, with extremes ranging from 17 to 50 years [2]. No difference in frequency between

the two sexes [3-5], although in a series of cases (14 cases of hepatic tuberculosis collected in the gastroenterology department of Ibn Rochd Hospital, Casablanca between January 1990 and January 2001),

they noted a clear female predominance with 12 women for 2 men, a sex ratio of 0.16 [6]. There is a higher incidence of hepatic tuberculosis in the black race [3-5].

A digestive localization is present in 5 to 30% of cases of tuberculosis occurring in individuals infected with HIV [7]. These are hepatic forms in more than 50% of cases, compared to 15% in the non-infected population. Digestive localizations are very rare when the number of lymphocytes is above 250/mm³. However, when this rate is below 100/mm³, liver damage is more frequent in the form of miliary.

Localized infection is extremely rare in people infected with HIV: 9 cases of liver abscess have been reported in the literature [8].

Unlike secondary involvement, primary hepatic tuberculosis is rare. Its frequency concerning the digestive sites of tuberculosis is 0.5% and 1.2% [9, 10].

The clinical approach to hepatic tuberculosis is always very delicate because hepatic tuberculosis is considered a clinically silent disease or, in symptomatic forms, the image is nonspecific or even misleading [11].

It is most often confused with a pyogenic or amoebic liver abscess and is often diagnosed during an autopsy or after a laparotomy due to its nonspecific presentation [12].

Fever can take on different forms: moderate or very high; it is frequent but not constant, and persists [2] in the absence of specific treatment. Abdominal pain was the most common reason for consultation; it was reported by Alvarez (45%) [13], Essop (66%), Hersh (50%), and Maharaj (46%). In our case, the pain was found; it was localized in the right hypochondrium. Asthma, anorexia, weight loss, and night sweats [14] are signs often found in patients with hepatic tuberculosis; hepatomegaly can be painful or painless.

Portal hypertension and jaundice are rare or even exceptional; jaundice is often related to the compression of extrahepatic bile ducts [15] (Table 1).

The contribution of biological tests to the positive diagnosis of hepatic tuberculosis is very limited. The sedimentation rate is almost constantly elevated, but in a variable manner [16]. C-reactive protein and fibrinogen may also be elevated [17]. Moderate normocytic anemia may be observed. Pancytopenia may be found in the context of hypersplenism or associated medullary involvement. Apart from the elevation of alkaline phosphatase, liver function remains normal.

Abdominal ultrasound generally reveals hypoechoic lesions in most cases of tuberculous liver abscesses [18]. However, a hyperechoic lesion was reported in one case [19]. Computed tomography

generally shows a low attenuation lesion with or without enhancement [20].

Thus, the specificity of ultrasound and computed tomography is low in detecting hepatic tuberculosis, but to define the site, size, and nature of the abscess, their value is indispensable [12].

It is difficult to diagnose hepatic tuberculosis based on clinical, biological, and radiological data, hence the need for histological confirmation.

The liver fragment can be obtained either by a trans-parietal biopsy, a laparoscopic biopsy, or during an exploratory laparotomy. The detection of tuberculous bacilli by culture is only positive in 10% to 35% of cases [21].

Molecular biology techniques for amplifying BK DNA are not part of routine examinations. They allow for obtaining the diagnosis in 48 hours [22, 23]. Recently, Alcantra *et al.*, [22] developed the use of PCR for the identification of BK in the biopsy fragment. He reported a 100% success rate in cases with caseating necrosis compared to 78% in cases with a granuloma or another lesion suspected of tuberculosis.

The caseous necrosis test is positive in 67% of cases for Alvarez *et al.*, in 83% for Essop *et al.*, and in 51% for Maharaj *et al.*, [24, 25]. Even though caseous necrosis does not exclusively represent tuberculosis [26], the result of anti-bacillary treatment indirectly confirms the diagnosis. The absence of a positive tuberculosis culture and PCR results does not influence the final diagnosis.

4. CONCLUSION

Hepatic tuberculosis accounts for less than 1% of all tuberculosis cases. Most of these cases are immunocompromised individuals infected with HIV. It is most often a secondary form to another tuberculous focus, and more rarely a primary form. This location raises a number of difficult diagnostic problems due to its polymorphic clinical, biological, radiological, and histological expression, hence the importance of always considering this diagnosis in the face of any granulomatous or pseudo-tumoral liver disease, particularly in a country with endemic tuberculosis and in an immunocompromised setting.

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