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Case Report

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Unusual Spread of Disseminated Tuberculosis in a Pregnant Lady: Rapidly Developing Tubercular Massive Pleural Effusion, Pulmonary Nodule and Hepatic Nodular Tuberculosis Mimicking Hepatic Metastasis

Richmond Ronald Gomes

Professor, Medicine, Ad-din Women's Medical College Hospital, Bangladesh

Corresponding author
Richmond Ronald Gomes, Professor, Medicine, Ad-din Women's Medical College Hospital, Bangladesh.

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ABSTRACT

Tuberculosis (TB) is prevalent in underdeveloped and developing countries, mainly in rural areas, with indistinct clinical manifestations. Lungs are the most affected organs; however, tuberculosis may invade almost all human body systems, including the liver. Isolated liver tuberculosis is still considered a rare condition and its atypical clinical presentation challenges the clinical acumen of the treating physician. There is difficulty in reaching the correct preoperative diagnosis of nodular hepatic tuberculosis that presents as a space occupying lesion. It is usually unsuspected and confused with primary or metastatic carcinoma of the liver. Regarding the fact that Mycobacterium tuberculosis culturing may be challenging in most settings, imaging and tissue diagnosis plays a vital role in diagnosing, treatment guiding, and patient follow-ups. In this report, we describe a rare case of a 22 weeks primi with disseminated tuberculosis presenting as rapidly developing unilateral massive pleural effusion necessitating intercostal tube drainage and hepatic nodular tuberculosis.

Keywords: Tuberculosis, Hepatic Nodule, Disseminated Tuberculosis

Introduction

Tuberculosis infection counts for a significant proportion of worldwide morbidity and mortality, especially in the tropical climate. In the past few years, the incidence has risen in many developing nations. Tuberculosis is an airborne disease and has severe outcomes in which Mycobacterium tuberculosis is the primary pathogen. The pulmonary organ is the main system to be affected; nonetheless, tuberculosis may affect the other parts of the body. Extrapulmonary tuberculosis made up roughly 15%-20% of all cases, with abdominal TB accounting for less than 1% [1].

One of the rarest entities of tuberculosis is hepatic TB, reported in only a few pieces of literature nowadays have been described. Hepatic tuberculosis is usually associated with an active pulmonary or military tuberculosis [2,3]. Internal calcification may lead to the diagnosis; nevertheless, some non-specific diagnostic pictures and pathology confirmation might be requisite [4,5]. However, since these features may sometimes overlap with other diseases, such as benign and malignant masses, they were frequently misinterpreted as uncertain lesions; therefore, it is mandatory to be able to recognize and distinguish between malignant metastatic and TB lesions. Last but not least, the diagnosis of hepatic tuberculosis is based on histopathological and microbiological findings from the liver biopsy [6].

Case Report

Mrs X, 19 years old, 22 weeks primi, hailing from rural Bangladesh presented to us with the complaints of fever for one month and upper abdominal pain for 20 days. According to the statement of the patient, she is amenorrhoeic for 21 weeks 4days & was reasonably well 1 month back. Then she gradually developed high grade, irregular, intermittent fever. Highest recorded temperature was 102 F. Fever was associated with chills & rigors, subsided with paracetamol. There were no H/O night sweats. Initially, fever was not associated with abdominal pain, headache, vomiting, chest pain, cough, breathlessness, haemoptysis, altered level of consciousness or

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any urinary complaints. She had no recent significant travel history or contact with any active TB patients. In last 15 days, she gradually developed progressive right upper abdominal pain, which was diffuse, non-radiating, moderate to severe in intensity and not related to food intake or empty stomach. Pain was not subsided with analgesics. Her bowel habit was normal. On query, she also had multiple joint pain in both small and large joint of both upper and lower limb for last 2 months. It was moderate to severe in intensity. Pain persists throughout the day & night but more marked in the morning. She had no history of photosensitivity, oral ulcer or Raynaud`s phenomenon. She had no previous bad obstetric history including pre-eclampsia, eclampsia, or abortion. On general examination, she is malnourished, ill looking, with pulse 110 beats/min, BP 110/60 mm of Hg, temperature 102o F, respiratory rate 17 breaths/min, anemia (++). Jaundice, cyanosis other skin and nail changes were present. On abdominal examination, abdomen was distended. Tenderness was present in both hypochondriac and epigastric region. Liver was enlarged, 2 cm from right costal margin in right mid clavicular line, tender with sharp margin and smooth consistency. There was no other organomegaly. Fundal height was 20 cm from pubic symphysis. Ascites could not be elicited due to gravid uterus. There was grade 3 tenderness over all small and large joints of both upper and lower limbs with no obvious sign of synovitis. On respiratory system examination there is stony dullness with reduced air entry over left mid to lower chest. Other systemic examination revealed no abnormalities. CBC revealed normocytic normochromic anemia (Hb%-6.9 gm%, MCV 83.8 fl, MCH 28.2 pg). Total WBC and platelet counts were normal. PBF was non-conclusive. S. creatinine, serum ALT, serum lipase, reticulocyte count was normal. CRP -194 mg/dl (normal <5 mg/dl), S. bilirubin 2.50 mg/dl (normal 0.3-1.0 mg/dl), s. albumin 20.3g/l (normal 35-52 g/l), direct coombs test- negative, urine RE- protein (++), pus cell 0-3/HPF, blood CS- no growth, anti CCP- negative, ANA- moderately positive. USG of whole abdomen revealed enlarged liver (17 cm) three large SOL (10.3×8.0 cm, 9.8× 7.1 cm and 10.9×6.8 cm) involving both lobes of lover with moderate left sided pleural effusion and mild ascites (figure 1). CXR showed bilateral pleural effusion(L>R) with pulmonary nodule in right lower zone (figure 2).

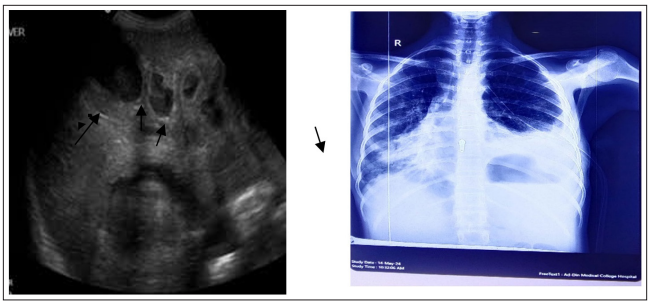

Figure 1: USG of whole abdomen showing three hypodense nodules in both hepatic lobes (black arrows) and **figure 2:** CXR showing bilateral pleural effusion(L>R) with pulmonary nodule in right lower zone (black arrow).

ENA Profile - SS-A/Ro60KD positive, Anti-ds-DNA - 0.500 U/ml (negative), C3, C4 level- normal, Urinary total protein - 0.36 gm/24hrs, Alpha feto protein - 45.73 ng/ml(normal), CEA - <0.200 ng/ml(normal). She was started treatment with meropenem, systemic steroid and hydroxychloroquine. After starting treatment, fever was subsided initially with significant improvement of joint pain but abdominal pain was persisting. On 5th day of starting treatment, patient again developed fever with progressive shortness of breath. Repeated chest examination left sided massive pleural effusion with signs of mediastinal shifting. (figure 3).

USG of chest revealed left sided massive septated pleural effusion with right pulmonary nodule and moderate ascites. As there was a nodule in the lung & the liver, we planned for CT guided FNAC from lung nodule. Patient was pregnant, so gynaecological consultation was taken for possible termination of pregnancy. But Gynae department refused for the termination of pregnancy. So, we would consider for insertion of intercostal tube and patient was on intercostal chest tube drainage for next 10 days. (figure 4)

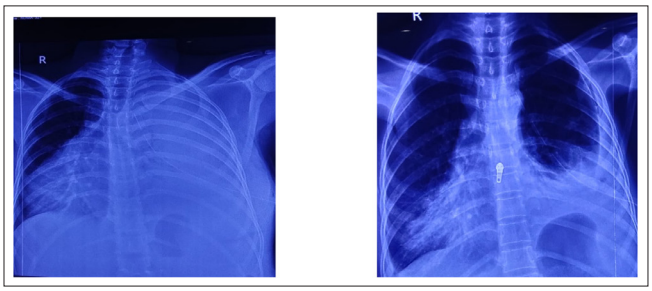

Figure 3: Chest X-ray PA view showing left sided massive pleural effusion with mediastinal shifting and **figure 4:** chest X-ray 10 days after insertion of chest tube.

In the meanwhile, 2 units PRBC was transfused. Patient was treated symptomatically with continuation ofsteroid & Hydroxychlorquine. Fever was subsided & joint pain was completely resolved. But abdominal pain was persisting. As CT guided FNAC could not be possible during her pregnancy period, we advised for USG guided FNAC from hepatic nodule which revealed caseating granuloma favoring tuberculosis (figure 5).

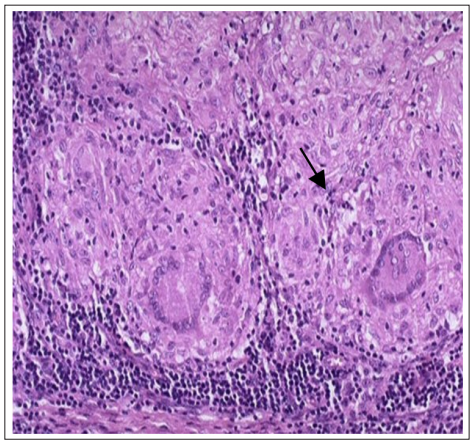

Figure 5: Histopathology of FNAC from hepatic nodule revealed granuloma with central caseating necrosis (Black arrow).

We started anti tubercular medication, according to body weight with continuation of oral steroid & hydroxylcholorquine After 5 days of starting anti Tubercular medication, patient's well-

being was good, no fever or joint pain & abdominal pain was improving. Then we planned for discharge with Anti Tubercular medications, oral steroid and hydroxychloroquine. During discharge her Hb was 9.1 g/dl, CRP 45 mg/l, ALT 30 u/l. Patient was afebrile and there was no abdominal pain. On OPD follow up after 1 month, she was afebrile, can eat well and gained 4 kg weight. Fetus was also doing well. There is a plan to continue anti tubercular medication for 1 year.

Discussion

Tuberculosis (TB) is defined as a disease affecting the lungs and systemic organs entailed by M. tuberculosis infection (MTB), aerobic bacilli bacterium. According to WHO, it remains a primary public health issue, with the estimated prevalence involving around one-third of the global population and new infections roughly 1% of the global population annually. In 2013, it was counted that there were about 9 million cases of novel TB infections and approximately 1.3-1.5 million deaths worldwide [7,8].

Despite the pulmonary system being the primary organ affected by tuberculosis, other systems may be invaded by the pathogen as well, with unclear and vague clinical presentation and radiology features. Hepatic TB is a rare type of extrapulmonary TB, and its incidence has been rising among immunocompromised patients. It occasionally happens in the second to sixth decade of an individual life, with a higher proportion in males [7-10].

Hepatic tuberculosis rarely happens and comprises less than 1% of all tuberculosis infections. Kok et al, reported an overall incidence of 0.3% for isolated hepatic tuberculosis [11]. It is uncommon because of the low oxygen pressure in the hepatic tissue, which hinders the growth of aerobic microorganisms. This problem may occur in almost all age groups but more frequently in young adults [10-12]. Classically, there are three patterns of hepatic involvement in TB i.e. miliary hepatic, nodular hepatic and biliary tract TB [13]. The first two involve the hepatic parenchyma while the later one involves the biliary tree. Hepatic tuberculosis lesions that appear as masses larger than 2mm in diameter are referred to as macronodular and pseudotumoural tuberculosis. On the basis of imaging examinations alone, these lesions are virtually indistinguishable from many other focal lesions of the liver, such as hepatocellular carcinoma) metastases and Hodgkin's disease, so pathological examination is necessary fordiagnosis [14].

The ailment may arise as a primary disease itself or emerge from other TB focal sites as a secondary infection. Miliary disease in the liver develops as the tuberculous bacilli reach the organ via the hepatic artery from pulmonary tuberculosis. In several conditions, the pathogen may invade the liver from the portal vein, especially with the gastrointestinal tract involvement [1-9]. In the localized hepatic TB, the portal vein route is the expected course. The bacteria may also reach the liver through the lymphatic system or due to the rupture of the lymph nodes containing the bacilli along the portal tract. Regardless of the entry port, the liver responds to this invasion by forming a granuloma tissue.

In general, this condition might not have a clinical manifestation; instead, it was usually detected accidentally when the patient was being evaluated for vague symptoms. Among the most prevalent complaints are loss of appetite (64 %), weight loss (64 %), fever (50 %), and jaundice (42.3 %) [15]. Jaundice has been reported for 35% of cases and is usually an obstructive type. During physical exam, hepatomegaly emerges as the most common physical finding, observed in approximately 96 % of cases and splenomegaly occurring in a range of 18 % to 55 % of cases [15,16].

Elevated serum levels of ALP and liver enzymes may be observed particularly in biliary TB 18,19,20. Imaging studies may detect a solitary space occupying lesion in the liver, manifesting as hypoechoic mass on sonography and a hypodense mass with or without hyperdense rim on CT scan [17-21]. As such, hepatic tuberculoma can mimic hepatocellular carcinoma or hepatoma. In the cross-sectional imaging study, hepatic TB may be comprehensively classified as micronodular and macronodular forms. Micronodular configuration refers to miliary tuberculosis where the lesions measure roughly 0.5-2 mm in diameter, while macronodular fashion may appear as 1-3 cm multiple lesions or a large tumor mass. Such a lump is typically pictured with low attenuation or without peripheral enhancement on CT (hypo or non-enhancing center of the lesion represents the caseating necrosis area, whereas the peripheral edges are similar to the outer granuloma tissue). A low attenuated lesion with center enhancement may be visible as the acute phase of the disease takes course. In addition, a mixed-type hepatic TB has also been described, with micronodular and macronodular features coexistence. Specific radiological indicators for hepatic TB are lacking. Nevertheless, certain signs such as the “target sign” (characterized by rim-enhancement of tuberculomas with caseous necrosis) and the “cluster sign” (involving the merging of small tuberculomas to form an abscess) could suggest the presence of hepatic TB.

Relying solely on imaging findings, it's challenging to distinguish these diffuse hepatic lesions resembling metastases from other masses such as hepatocellular carcinoma (HCC), Hodgkin lymphomas, or metastatic growths. Often, these lesions are misidentified as primary or metastatic carcinoma [22]. In our case, the condition was mistakenly diagnosed as bronchial carcinoma with hepatic metastasis. If the diagnosis is still in doubt, laparoscopy is the next investigative method of choice, as it is less invasive than laparotomy.

The unique and nonspecific presentation of hepatic TB makes diagnosis challenging through non-invasive methods, including imaging. This is further complicated by the fact that other localized liver deposits, such as metastases, can present with similar imaging characteristics. Consequently, histopathological examination of a biopsy specimen from the liver lesion remains the gold standard for hepatic TB diagnosis [16]. Histopathological assessments of hepatic TB typically reveal epithelioid granulomas in 80 %-100 % of cases, caseating necrosis in 30%-83 % of cases, and the presence of acid-fast bacilli (AFB) on smear examination in 0 %-59 % of cases [15]. In our case, hepatic deposits displayed epithelioid granulomas and caseous necrosis, while AFB were not detected on smear examination.

However, hepatic TB may be challenging to treat, given the possibility of contralateral lobe reactivation, even though the

disease resides in the ipsilateral lobe [9,10,12]. Therefore, prompt identification and remedy are indispensable in order to preserve the remaining liver function. It is vital to acquire knowledge and awareness about the broad spectrum of manifestation and discovery because early identification of extrapulmonary TB remains a challenge in many instances. High clinical suspicion is required to recognize this entity for medical management; even so, liver biopsy and pathology investigation provide a definitive diagnosis. Delayed treatment may instigate hepatic failure and death. Often, it is mandatory to explore further organ involvement and inspect for additional coexistent problems such as malignancy.

Once the diagnosis of hepatic TB is established, conventional ATT for 1 year will cure the disease nearly in 100% of cases. Typical anti tuberculosis medications (isoniazid, rifampicin, pyrazinamide, and ethambutol) are the recommended protocol. In biliary TB causing obstruction of the biliary tree, there may be a need for ERCP (Endoscopic Retrograde Cholangio-Pancreatography) to relieve the obstruction. Tuberculosis should be kept in the differential diagnosis of space occupying lesions of the liver especially in endemic areas. Our patient has been given ATT and ion follow up; he was found to be afebrile and had recent weight gain of 4 kg in a period of 1 months of therapy.

Conclusion

Isolated hepatic tuberculosis is a rare entity with potentially curable outcome, but presents a diagnostic challenge due to its atypical presentation. Hepatic tuberculosis has unspecified clinical manifestation, and therefore, imaging modality, along with CT/USG-guided fine-needle biopsy, offers an excellent diagnostic value. The existence of hypodense nodule with dilated biliary duct associated with lobar atrophy indicated a consistent imaging feature of hepatic TB, especially with active lung disease. Unless there is a high suspicion of tuberculosis, its diagnosis is often ignored. settings. Timely diagnosis and treatment of TB are vital to avoid surgery, linked with elevated morbidity and mortality risks.

Conflict of interest: None declared

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