

Original Article

A case report of portal hypertension and upper gastrointestinal hemorrhage caused by portal tuberculosis

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Abstract: Abdominal tuberculosis (TB) is a rare type of extra-pulmonary TB that can occur in any organ or tissue of the abdominal cavity. The order of frequency of occurrence reported previously in the literature is as follows: gastrointestinal tract, retroperitoneal tissue, mesenteric lymph nodes, and abdominal organs (< 1%). Cases of invasion to the portal vein, liver, and pancreas and concurrent pancreatic portal hypertension are extremely rare. So far, only 6 related cases have been reported at home and abroad. However, there has been no report of simple abdominal TB causing portal vein stenosis and subsequent gastrointestinal hemorrhage. Our center reported the case of a 30-year-old female with a 3-year history of pulmonary TB, recent hematemesis, melena, anorexia, and pancytopenia. Computed tomography (CT) showed a left hepatic mass, portal vein stenosis, esophageal varices, and splenomegaly. After CT-guided biopsy failed to identify the cause, surgical intervention determined that the portal vein stenosis in this patient was due to caseous necrosis that had invaded the portal vein trunk. These findings suggest that when patients present with portal hypertension and esophageal varices of unknown cause, abdominal TB should be included in the differential diagnosis. This study also describes the effect of lymph node TB on the duodenal ligament of the liver.

Keywords: Abdominal tuberculosis, liver and duodenal ligament, lymph node tuberculosis, portal hypertension, gastrointestinal hemorrhage

Introduction

With the development of anti-tuberculosis drugs, tuberculosis (TB) has gradually been brought under control throughout the world. However, according to statistical data collected over the past decade, the global incidence of TB is showing a gradual upward trend, or an annual increase of about 1.1%. This is largely due to the epidemic of diseases affecting the human immune system, such as acquired immunodeficiency syndrome (AIDS). Today TB occurs not only in Asia and Africa but its incidence in Europe and the America is also increasing year by year [1]. It has been reported that in the past 2 decades, the incidence of TB in the United Kingdom has increased. New strains of drug-resistant TB have also emerged, so that early diagnosis and effective treatment are particularly critical [2]. Abdominal TB is a

rare disease. Data show that the incidence of extra-pulmonary TB accounts for only 10% to 15% of cases, whereas abdominal TB accounts for only about 10% of extra-pulmonary TB [3]. There are no specific symptoms or signs of abdominal TB, which may include splenomegaly, ascites, cough, diarrhea, weight loss, chills, fever, etc. There are also no specific imaging findings, so that the diagnosis of extra-pulmonary TB, distinguishing it from a mix of concomitant medical conditions, remains difficult. Pathological biopsy prompted by a cheese-like granuloma is the gold standard for the diagnosis of TB. Currently recognized methods of TB transmission or extension include (1) the spread of infectious secretions by oral transmission, (2) the spread of *Mycobacterium tuberculosis* through the metastasis of lymphoid tissue, (3) transmission of *M. tuberculosis* through blood transfer.

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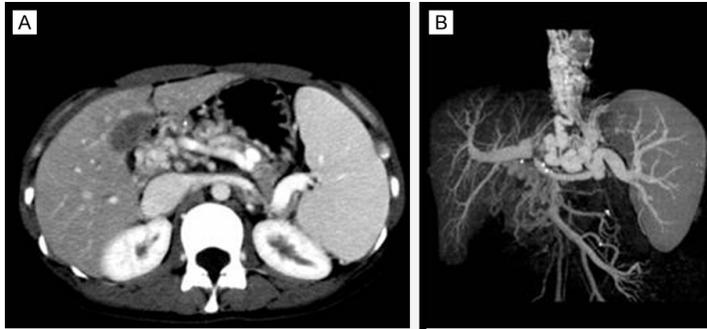


Figure 1. A, B. Seen here is portal vein stenosis, the tortuous expansion of venous blood vessels in front of the pancreas, coronary varicose veins, and inflammatory lesions in the left hepatic lobe.



Figure 2. The trunk of the portal vein is seen to be packed with dense caseous tissue.

Case report

A female patient, 30 years of age, had been diagnosed with pulmonary TB 3 years earlier and treated with a triple regimen of isoniazid, rifampicin, and ethambutol for 1 year. On regular follow-up, no recurrence of pulmonary TB was found. This time she was admitted with a chief complaint of “hematemesis, melena 3 days.” Laboratory tests showed leukocytes at $3.38 \times 10^9/L$; hemoglobin 95 g/L; platelets $92 \times 10^9/L$; normal liver and kidney function; prothrombin time, 11.7 s; hepatitis B negative; purified protein derivative (PPD) test negative; human immunodeficiency virus (HIV) and hepatitis C virus (HCV) negative; CA199: 17 U/mL; No TB lesions on chest CT. Abdominal ultrasound indicated portal vein stenosis, along with a tortuous expansion of the vein in front of the pancreas. Abdominal CT showed spleno-

megaly, portal vein stenosis, esophageal varices, and lesions in the left hepatic lobe (see **Figure 1A, 1B**).

After discussion among experts in our department, this case was preliminarily diagnosed as upper gastrointestinal hemorrhage, portal hypertension, splenomegaly, and esophageal varices and the possibility of cancer was not ruled out. Owing to inability to do a puncture biopsy for a clear diagnosis, it was decided to perform a laparotomy. On intraoperative view, the middle

of the portal vein was wrapped by a lot of soft tissue. The texture of the liver’s duodenal ligament was hard (**Figure 2**), and there were serious adhesions. A 1×1 cm piece of soft tissue was cut intraoperatively and frozen. In view of the pathological findings and the patient’s history, abdominal TB was considered (**Figure 3**).

At this point, the symptoms of portal hypertension and upper gastrointestinal hemorrhage were believed to be caused mainly by pre-hepatic portal hypertension due to stenosis of the portal vein and the compression of inflammatory tuberculous lesions. *M. tuberculosis* apparently invaded the main portion of the portal vein, making its wall fragile. Splenectomy, cholecystectomy, and artificial blood vessel catheterization in the portal vein were applied during the surgery (**Figure 4**). Histopathological examination of postoperative paraffin sample indicated: chronic hepatic inflammation, splenic sinus expansion and congestion, and chronic cholecystitis. There was no suggestion of malignancy in 2 pathological reports, so the possibility of cancer was ruled out. The patient received long-term aspirin anticoagulation therapy after surgery. Isoniazid, rifampicin, ethambutol, and pyrazinamide were applied as a quadruple anti-TB regimen for 9 months. On follow up, multiple endoscopic examinations showed no esophageal or and gastric varices. Currently, 48 months after her operation, the patient reports no recurrence of gastrointestinal hemorrhage.

Discussion

TB laboratory tests rely mainly on bacteriological smears and culture, which are also the main basis for the diagnosis of TB. Because of the

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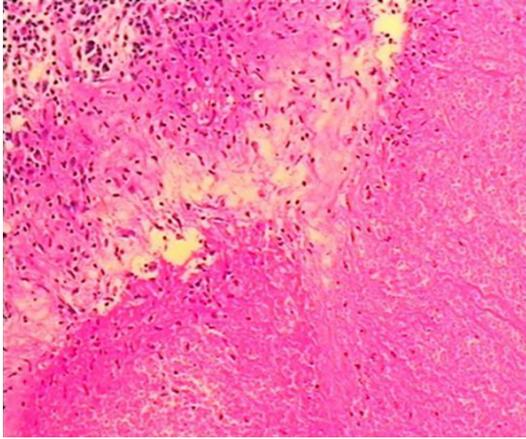


Figure 3. Pathology of hilar tissue shows chronic granulomatous inflammation associated with coagulation necrosis.



Figure 4. Surgical excision of the invaded part of portal vein showing artificial blood vessels.

low positive detection rate of abdominal TB in this manner, along with the absence of specific clinical symptoms or indications on imaging, the diagnosis is more difficult than that of pulmonary TB, posing a great challenge for the treating physician. Especially when there is no obvious mass in the abdomen and one is unable to perform a qualitative puncture biopsy, surgical intervention is particularly critical. In recent years, development of immunological methods for the detection of TB has been of tremendous help in the diagnosis and identification of TB. As an emerging immunological method, T-SPOT.TB technology is conducive to the early diagnosis and treatment of TB owing to its high level of detection even in the diagnosis of recessive TB. However, this method is not widely used because of its high cost. An early,

moderate, combined, regular, full course of treatment is the key to the success or failure of abdominal TB treatment. China's treatment guidelines recommend the use of chemotherapy initially when a positive TB smear is found in uncomplicated abdominal TB and also the implementation of DOTS strategy. Some patients need localized treatment along with surgery, immunotherapy, and the use of steroids to obtain the best therapeutic effect.

As a potential systemic disease, abdominal TB can infect any organ or tissue in the abdominal cavity, causing a variety of clinical symptoms. Veeragandham et al. indicate that intra-abdominal TB in children has been reported. This too appears to be gradually increasing, seriously affecting the normal function of the gastrointestinal tract, retroperitoneal tissue, lymph nodes, and abdominal organs [4]. Because there are no specific early special clinical symptoms, acid-fast staining is only 3% to 20% positive in the microbial examination of ascites [5, 6]. Ultrasound and CT-guided mass puncture biopsy is the gold standard for the diagnosis of abdominal TB, but without substantive occupied lesions, the clinical diagnosis of peritoneal TB remains difficult. A poor prognosis is suggested when there is a high frequency of bleeding, fatigue, abdominal pain, jaundice, weight loss, and other symptoms. Singh et al. have also reported a case of liver failure from primary liver TB, leading to the death [7].

Based on the preceding summary, a special type of abdominal TB is proposed: hepatic duodenal ligament lymph node TB. The hepatic duodenal ligament is located between the transverse groove on the surface of the liver and the duodenal bulb, connecting the liver to stomach ligaments on the left side and forming a pore structure with the peritoneum after dislocation for mesh on the right side. The peritoneum is rich in blood vessels, lymph nodes, lymphatic vessels, nerve plexuses, and so on. The portal vein is an important blood vessel formed by the confluence of the splenic vein and the superior mesenteric vein behind the pancreas and then continues into the liver. There are few vascular networks and lymphatic vessels in this area. The case reported from our center had regional portal hypertension and upper gastrointestinal hemorrhage resulting from the formation of a ring of inflammatory

stenosis near the tubercular portal lymph node. Cases reported by Chiu, Jazet, and Lee, et al. also confirm this type of extra-hepatic portal hypertension, which is caused by intra-abdominal TB and then leads to hemorrhage and other symptoms of portal hypertension [8-10]. Jazet et al., have also confirmed that TB bacilli can invade the bile duct, resulting in obstruction [9]. TB affecting the duodenal ligament lymph nodes can move upward to invade the liver or downward to cause reflux and obstruction of the splenic vein, causing splenic vein thrombosis, hyper-splenism, and gastrointestinal hemorrhage. There can also be an inflammatory response from the pancreas itself. The pancreas is a relatively isolated organ. Pancreatic TB is also extremely rare and must be distinguished from pancreatic cancer, retroperitoneal tumor, and pancreatitis. The retrospective analysis via histological examination by Xia et al. of 16 cases of pancreatic tuberculous granuloma and peri-pancreatic lymph node TB [11] indicates that an unexplained pancreatic mass should prompt consideration of the possibility of pancreatic and peri-pancreatic lymph node TB. Retro-peritoneal TB often manifests as a large number of unexplained ascites. Therefore TB of the hepatic duodenal ligament lymph node occupies a special position in the differential diagnosis owing to the common absence of an obvious abdominal mass. Thus this condition should be included in the differential diagnosis of portal hypertension and esophageal varices of unknown cause.

Conclusion

In summary, for patients with portal hypertension and esophageal varices of unknown cause, intra-abdominal TB should be included in the differential diagnosis. TB of the hepatic duodenal ligament and lymph nodes is extremely rare. It is also very difficult to diagnose, but good clinical results can still be achieved with regular and effective anti-TB treatment. Early diagnosis and treatment can prevent the occurrence of acute complications.

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Disclosure of conflict of interest

None.

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