Journal of Korean Medical Science
Vol. 9, No. 3, 264-267 June 1994

# A Case of Extra-Hepatic Portal Hypertension Caused by Periportal Tuberculous Lymphadenitis

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***This report describes a case of portal hypertension caused by periportal tuberculous lymphadenitis. There were a few reports of portal hypertension associated with tuberculosis. A 27-year-old man was admitted to the hospital because of recurrent hematemesis for 7 days. There was a history of mediastinal tuberculous lymphadenitis 3 years earlier that was treated with isoniazide, rifampin, ethambutol, and pyrazinamide for 2 years. Clinical evaluation revealed esophageal variceal bleeding and main portal vein obstruction by enlarged periportal lymph nodes. The patient underwent distal splenorenal shunt. Pathologic examination of the excised periportal lymph node revealed chronic granulomatous inflammation with central caseous necrosis. Thereafter the patient took antituberculous medication for 12 months. The patient has not re-bled 3 years since the shunt operation.***

**Key Words** : *Portal hypertension, Tuberculous lymphadenitis.*

## INTRODUCTION

Extra-hepatic portal venous obstruction caused by tuberculous lymphadenitis is quite rare. We report a case of extra-hepatic portal hypertension caused by periportal tuberculous lymphadenitis, which presented with esophageal variceal bleeding.

## CASE REPORT

A 27-year-old man was admitted to the hospital because of massive hematemesis. There was a history of mediastinal tuberculous lymphadenitis 3 years earlier that was confirmed by Ziehl-Neelsen stain and successfully treated with isoniazide, rifampin, ethambutol, and pyrazinamide for 2 years. Thereafter the patient was well until 7 days before admission, when sudden onset of massive hematemesis developed. Evaluation at another hospital revealed esophageal variceal bleeding. The patient was transferred to this hospital due to an uncontrollable variceal bleeding. There was no history of alcohol consumption or prior use of hepatotoxic drugs and no personal or family history of liver disease.

The temperature was 36.5℃, the pulse 80/min, and the respirations 20/min. The blood pressure was 110/70mmHg. The patient was thin with no peripheral lymphadenopathy. Chest examination was negative. A slightly tender spleen was palpated 4cm below the left costal margin, the liver was not felt, and no ascitic fluid was noted. There were no peripheral stigmata of chronic liver disease. Neurologic examination was negative.

The hemoglobin was 12.0g/dl, the white-cell count was 6,000/$mm^3$, the platelet count was

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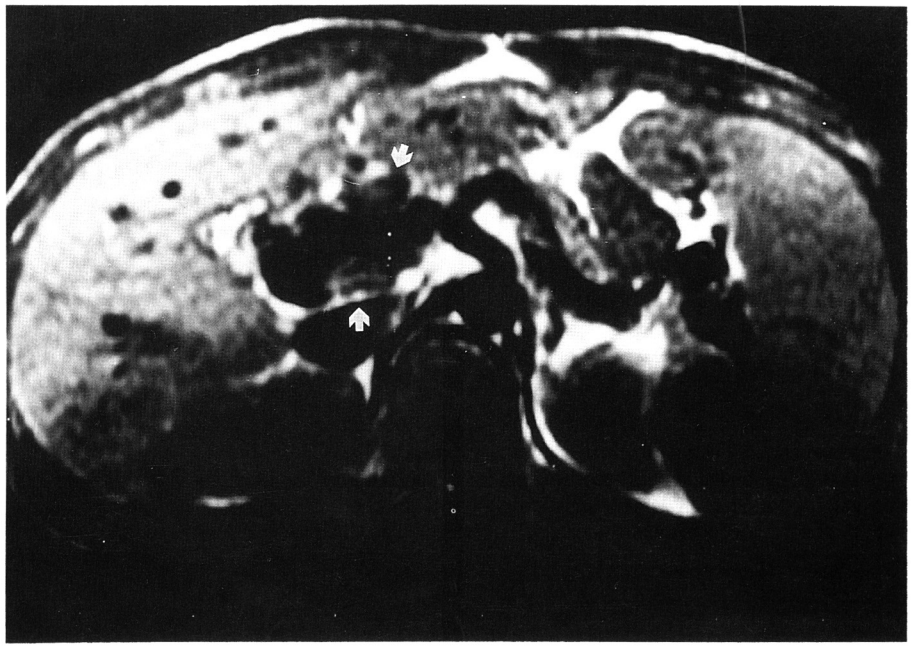

Fig. 1. A T1-weighted MRI scan of the abdomen showing enlarged periportal lymph node(arrow), periportal collateral formation, and splenomegaly.

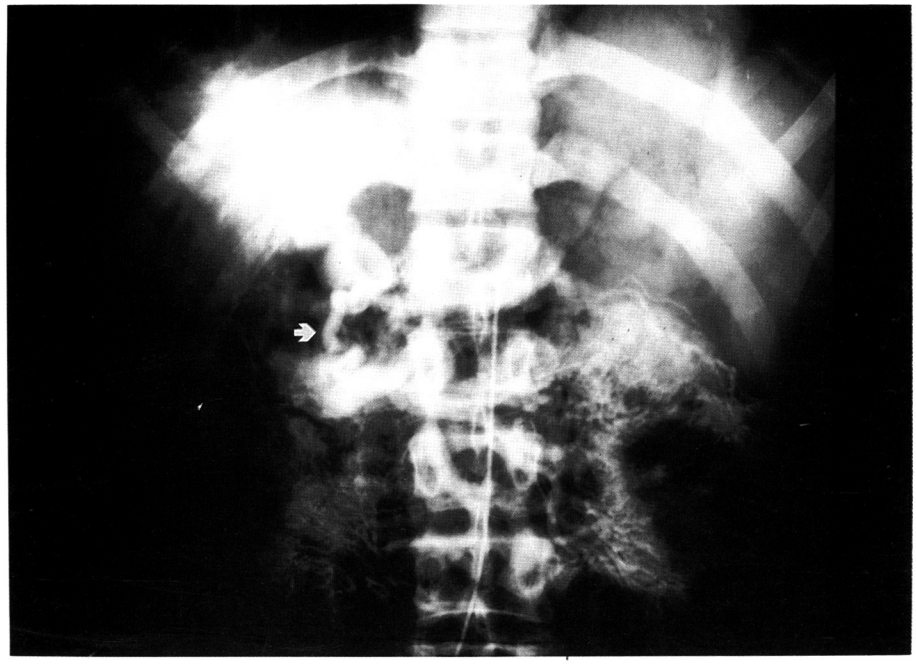

Fig. 2. Venous phase of the selective superior mesenteric artery angiogram showing external compression of the mid-portion of the main portal vein with large periportal collaterals(arrow).

180,000/mm$^3$, the erythrocyte sedimentation rate(ESR) was 25mm/hour. The serum total bilirubin was 0.7mg/dl, the protein 7.4g/dl, the albumin 4.1g/dl, the aspartate aminotransferase 34 IU/L, the alanine aminotransferase 18 IU/L, the alkaline phosphatase 80 IU/L, the urea nitrogen 15 mg/dl, the creatinine 1.0mg/dl, and the glucose 90mg/dl. The prothrombin time was 12.0sec(control 11.1sec); the partial thromboplastin time was 27.5sec(control 30.0sec). The urine was normal. Virologic markers for hepatitis B virus and C virus were all negative. A serologic test for syphilis was negative. Sputum smears and cultures for acid-fast bacilli were negative.

A gastrofiberscopy revealed cobble-stone-like esophago-gastric varices with multiple cherry red spots. A chest x-ray was normal. Computed tomography(CT) and magnetic resonance imaging(MRI) scan of the abdomen disclosed exteranl compression of the mid-portion of the main portal vein by multiple enlarged lymph nodes in the porta hepatis, formation of periportal collateral vessels; marked enlargement of spleen(Fig. 1). Venous phase of selective superior mesenteric artery angiogram showed that contrast material was drained from the superior mesenteric vein to the liver via large collateral channels.(Fig. 2).

The patient underwent distal splenorenal shunt and ligation of coronary vein to reduce variceal venous pressure. The operative findings showed external compression of the main portal vein by multiple enlarged periportal lymph nodes; formation of large periportal collateral vessels, which looked like cavernous appearence; marked enlarged spleen; no abnormalities were detected in the peritoneum, mesentery, liver, gallbladder and pancreas. Microscopic examination of the excised periportal lymph nodes revealed chronic granulomatous inflamation with central caseous necrosis which was consistent with tuberculosis(Fig. 3), but special stains for acid-fast bacilli(AFB) and fungi were negative. The pathologic finding of wedged liver biopsy was normal. Thereafter the patient was treated with cycloserine, ethambutol, ofloxacin, and prothionamide for 12 months. A gastrofiberscopy 1 year later showed small esophageal varices and an abdominal CT scan showed patent splenorenal shunt. Follow-up ESR 1 year later also decreased from 25 to 3mm/hour. The patient has been well 3 years since the shunt operation.

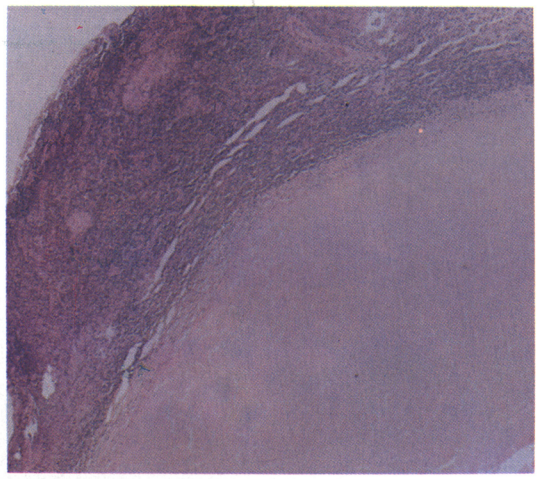

**Fig. 3.** Photomicrographs of the excised lymph node showing chronic granulomatous inflammation with central caseous necrosis.

## DISCUSSION

Extra-hepatic portal venous obstruction is one of the presinusoidal forms of portal hypertension, which is most commonly associated with infection-(Webb et al., 1979). Although hepatic tuberculosis is common in the presence of miliary tuberculosis, there were a few reports of extra-hepatic portal hypertension associated with tuberculosis(Ibrhim et al., 1987; Nogueira et al., 1991; Testart et al., 1978). Furthermore, extra-hepatic portal hypertension caused by periportal tuberculous lymphadenitis in the absence of miliary tuberculosis or tuberculosis of the surrounding structures, such as the liver and gallbladder, is extremely rare. Periportal tuberculous lymphadenitis can occur by virture of contiguous extension from adjacent structures, hematogenous spread, or lymphatic spread. This patient does not have currently active tuberculosis of surrounding structures, or miliary tuberculosis. Thus, it appears to develop by reactivation of latent tuberculous foci in the periportal lymph nodes, established at the time of earlier hematogenous spread from a primary focus, such as the lung. The pathologic examination of the excised lymph node revealed chronic granulomatous inflammation with central caseous necrosis, which is compatible with tuberculosis(Robbins et al., 1984). Tuberculosis is common and of major health problem in Korea. In addition, there was a previous history of mediastinal tubercu-

lous lymphadenitis. Therefore, despite negative AFB stains of the excised lymph node, there is little probability of granulomatous diseases other than tuberculosis in this patient. Extra-hepatic portal venous obstruction most commonly manifests as upper gastro-intestinal hemorrhage. Splenomegaly, anemia, and ascites were also common modes of presentation(Webb et al., 1979). This patient presented with sudden onset of massive hematemesis, but hepatic encephalopathy did not develope despite repeated episodes of massive variceal bleeding, which might be directly influenced by the degree of liver decompensation. The hepatic reserve in this patient was completely normal. Radiologic examinations of the abdomen in this patient suggested portal vein obstruction by enlarged periportal lymph nodes, which was confirmed at surgery.

The management of a patient with extra-hepatic portal hypertension is aimed at correction of underlying disease and prevention fo recurrent bleeding. Definitive surgery for reduction fo portal pressure is usually difficult as there are no suitable veins for a shunt. Therefore postoperative results are usually not satisfactory(Webb et al., 1979). But splenic vein in this patient was normal without thrombosis. Therefore distal splenorenal shunt was successfully performed, which remarkably decompressed the variceal venous pressure and led to control of esophageal variceal bleeding. On the basis of pathologic finding, this patient took antituberculous medication for 12 months. Follow-up evaluation 1 year later showed patent splenorenal shunt and mild esophageal varices with normalization of ESR. The prognosis of this patient considered much better than for those with liver cirrosis. The patient has not re-bled 3 years since the shunt operation.

We believe that periportal tuberculous lymphadenitis should be included in the differential diagnosis of extra-hepatic portal hypertension, especially in the endemic areas of tuberculosis, and suspected, if esophago-gastric varices are present in a patient with previous history of tuberculosis who shows no signs of other liver disease. The condition is very rare but important because of a potential curability with proper management.

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