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LETTERS TO THE EDITOR

Idiopathic thrombocytopenic purpura associated with *Helicobacter pylori* infection in a patient with liver cirrhosis accompanying hepatitis B

Thrombocytopenia is a major hematological disorder observed in patients with liver cirrhosis (LC). The condition is primarily attributed to hypersplenism, which is accompanied by an increased sequestration and accelerated destruction of platelets in the pathologically enlarged and congested spleen.[1—3] Patients with LC are also frequently subject to various disorders of the gastric mucosa, and peptic lesions in the gastroduodenal mucosa have been observed more often in cirrhotic patients than in controls. *Helicobacter pylori* infection is reportedly an important factor in the pathogenesis of such cases.[4—7] Recently, *H. pylori* eradication has been reported as effective for managing *H. pylori*-positive idiopathic thrombocytopenic purpura (ITP). Eradication therapy is drawing attention in ITP, particularly because this approach readily increases platelet counts.[8,9] We describe herein a case of *H. pylori*-related ITP in a patient with LC accompanying hepatitis B. No previous case reports have described associations between *H. pylori*-related ITP and cirrhotic patients.

A 65-year-old man was admitted to our hospital in December 2005 for further evaluation of thrombocytopenia during his monthly follow-up for LC accompanying hepatitis B, hypertension and diabetes. Although his platelet count had been around $5.0 \times 10^4/\mu l$ during his regular follow-up for liver cirrhosis, his platelet count was extremely decreased on admission ($0.4 \times 10^4/\mu l$). Physical examination was unremarkable, without any lymphadenopathy. Computed tomography (CT) and ultrasonography showed splenomegaly (spleen index: 14.3 cm × 8.7 cm). Testing for human immunodeficiency virus yielded negative results, but *H. pylori* infection was diagnosed based on a positive urea breath test. Bone marrow aspiration and biopsy revealed increased megakaryocytes and normal morphological appearance. Cytogenetic analysis of marrow cells also yielded normal results. With these findings, a diagnosis of ITP accompanying thrombocytopenia due to LC was suggested. The patient was subsequently started on prednisone (60 mg/day) along with lamivudine (200 mg/day). His insulin dose was increased appropriately to control the diabetes. Eradication therapy against *H. pylori* (a standard 1-week regimen containing amoxicillin, clarithromycin and lansoprazole) was also started. His platelet count rose to $4.3 \times 10^4/\mu l$ on day 7, but fell to $0.8 \times 10^4/\mu l$ on day 15. The dosage of oral prednisone was reduced by half on day-21 because the steroid did not seem to increase the effect. By one month following initiation of treatment, his platelet count had risen to around $3.0 \times 10^4/\mu L$, and the patient was discharged on 10 mg/day of prednisone in February 2006. On discharge, the 13C-urea breath test yielded negative results. His platelet count three months later ranged from $4.0 \times 10^4/\mu L$ to $5.0 \times 10^4/\mu L$, although oral prednisone and lamivudine had been reduced to 5 mg/day and 100 mg/day, respectively. At the eight-month follow-up, his platelet count remained around $4.0 \times 10^4/\mu L$ to $5.0 \times 10^4/\mu L$ without the use of prednisone. Lamivudine was administered at 100 mg every three days and as of the time of writing, no reactivation of chronic hepatitis B virus infection has been identified.

Many studies have reported the presence of *H. pylori* infection with autoimmune diseases such as chronic thyroiditis, Sjögren's disease, rheumatoid arthritis, and ITP, where *H. pylori* infection is indicated as a possible cause.[10] Among these, ITP is drawing particular attention because the clinical benefit and efficacy of eradication therapy against *H. pylori* has been demonstrated.[8,9] The present case highlights an interesting manifestation in cirrhotic patients, and the true frequency of *H. pylori*-related ITP cases in cirrhotic patients might be significantly higher than the low prevalence suggested by the paucity of reported cases. This association would also greatly facilitate understanding of the mechanisms underlying *H. pylori*-related ITP.

In conclusion, the possible association between thrombocytopenia with LC and *H. pylori*-related ITP should be kept in mind, particularly when thrombocytopenia steadily develops during the clinical course.

Conflict of interest: No conflict of interest to declare.

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Tuberculosis and tracheal bronchus

Anomalies of the lung and bronchial tree are diagnosed with increasing frequency as a result of refinements in modern imaging.[1] Most of these are rare and almost always asymptomatic congenital malformations. Some of these anomalies are found during investigations in patients with respiratory diseases.

Tuberculosis is one of the ten main causes of death in the developing world; pulmonary tuberculosis is the most common clinical form.[2] Even with completion of appropriate treatment, this disease can produce progressive destruction of lung tissue, and cavitation is a common sequela. Following cavitation, many patients can go on to develop fungal ball, recurrent pneumonia and hemoptysis across variable time intervals; some may remain asymptomatic for life.[3] We report below, the case of a patient who had tuberculosis 15 years ago and received appropriate chemotherapy. After 15 asymptomatic years, she presented with acute hemoptysis and further investigation led to a diagnosis of bronchial anomaly.

A 41-year-old female patient presented after an episode of hemoptysis in July 2006. The patient had had pulmonary tuberculosis at 12 years of age and again at 26 years of age; both episodes had been adequately treated and she had experienced no other illness or pulmonary symptoms since then. She presented with a bronchial sound in the right upper quadrant. Plain chest radiography showed a right-apical cavitation. Three specimens of sputum were smear- and culture-negative. Computed tomography imaging revealed an extensive right upper lobe cavitation without signs of active disease and a tracheal bronchus arising distal to the origin of the upper lobe bronchus, so-called posteparterial (Figure 1).

Congenital anomalies most often confused with tuberculosis are unilateral lung hypoplasia, bronchogenic cysts and tracheal bronchus with an anomalous lobe.[4] Although congenital anomalies are often confused with tuberculosis, our case describes a tracheal bronchus in a patient with a large persisting tuberculosis cavity. To-date only three cases of this association have been reported in the literature.[5,6]

Tracheal bronchus is not such a rare event and its association with tuberculosis is probably underestimated. Tuberculosis and other infections can occur as a result of insufficient drainage of the involved bronchi.[6] The identification of this anomaly is very important in order for the physician to perform an adequate bronchoscopy, to avoid difficulties in placement or malpositioning of an endotracheal tube, and for surgical planning when a resection is required.

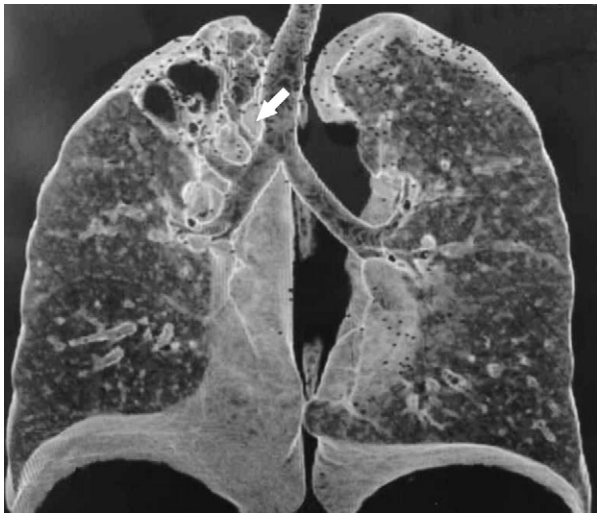

Figure 1 Tracheal bronchus in a patient with sequelae of tuberculosis (3D reconstruction).