

Letters to the Editor

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Beneficial effect of talc pleurodesis in refractory pleural effusion due to Behçet's disease

Sirs,

In patients with Behçet's disease (BD), pleural effusion due to pleural vasculitis or superior vena cava syndrome (SVCS) can be seen in the course of the disease (1, 2). The development of SVCS in BD is mostly related with vascular inflammation and thrombotic tendency (1-4). Treatment options for pleural effusion due to SVCS encompass immunosuppressives and video-assisted thoracic surgery (1-4). To the best of our knowledge, there exists no report in the literature showing the beneficial effect of talc pleurodesis in recurrent pleural effusion due to SVCS of BD.

A 24-year-old man was admitted to our hospital with dyspnoea and swelling in the face and neck in January 2005. He had been diagnosed as having BD associated with Budd-Chiari syndrome (BCS) in 2003. Because of the presence of low-grade oesophageal varicosities and mild impaired hepatic functions, he had been taking warfarin with low intensity and azathioprine 50 mg/day.

On physical examination, he was found to have a venous fullness of the neck, hepatosplenomegaly and decreased respiration voice in the right hemi thorax. Laboratory findings were as follows: haemoglobin 10.4 g/dl, WBC 9000 mm³, erythrocyte sedimentation rate (ESR) 85 mm/h, total protein 5.8 mg/dl, albumin 2.7 mg/dl (N: 0.01-0.3). Chest radiogram showed a pleural effusion in the right hemi thorax. A thorax computed tomography disclosed thrombotic occlusion of the superior vena cava and the brachiocephalic vein. Because of the severity of his dyspnoea, thoracentesis was performed. The white cell count of the pleural fluid was 200/mm³. The pleural fluid was transudate. Thoracentesis had to be repeated seven times within a period of just 10 days to relieve his respiratory distress due to relapse in accumulation of the pleural fluid. The thorascopic imaging showed dilated vessels on both pleura and diaphragm surfaces. Following the pleural biopsy of the area affected, pleurodesis was performed with talc of 5 gr. Pathological evaluation of his biopsy specimen showed a reactive mesothelial hyperplasia characterized by multinuclear mesothelial cells without evidence of vasculitis. The patient has been safe from accumulation of the pleural fluid over the past twelve months with no recurrence.

A number of pulmonary problems associated with BD have been defined, which problems can be classified into three groups (1) pulmonary artery aneurysm (2) pulmonary parenchymal changes, and (3) others including pulmonary artery occlusion, pleural effusion and pulmonary obstructive airway disease (3, 4). Pleural effusion can be transudative, exudative or chylous. It has been reported that the causes of pleural effusion were thrombosis of the superior vena cava or vasculitis of the pleura (3, 4). In our case, no evidence of vasculitis was found in the pleural biopsy. Because our patients also had BCS, pleural effusion may have originated from ascitic fluid. It has been reported that pleural effusion can be seen in the course of hepatocellular failure due to transportation of ascitic fluid into the pleural space (5). Our patient had been having BCS for twenty four months and he had only minimal ascites. Moreover, we ascribe recurrent pleural effusion to superior cava syndrome rather than to minimal ascites. We also rule out the possibility the hypoalbuminemia could have triggered development of the pleural fluid, considering the fact that the patient had already been suffering from hypoalbuminemia over the past two years. The treatment options for pleural effusion seen in BD include immunosuppressives (ISs), together with anticoagulation and video-assisted thoracic surgery. Guralnick *et al.* reported a case with chylothorax due to SVCS as a presenting symptom of BD (6). Chylothorax has been regressed by a combination of ISs and anticoagulation in this case. As to our patient, AZA and warfa-

rin, which may result in hepatotoxicity and haemorrhage, were given in small doses, because he had low-grade oesophageal varicosities and mild impaired hepatic functions. We assumed that pleurodesis could be an alternative approach to our case. Talc pleurodesis has been successfully used in persistent and recurrent pleural effusion due to malign diseases (7). Our case is the first of its kind in that pleurodesis with talc was performed for the treatment of recurrent pleural effusion due to SVCS of BD. In BD, there is an increased inflammatory response to the exogenous stimulating factors. A typical example of this entity is pathergy reaction that may be induced by needle prick and applying urate crystalline to the skin (8). Theoretically, it could be speculated that talc application may result in an inflammatory response in pleural space as a result of the pathergy reaction. However, our case did not develop any inflammatory reaction, nor has he referred to our clinic with recurrent pleural effusion since his discharge. Despite this promising result, we suggest that further clinical experience is needed to be able to consider talc pleurodesis in the treatment of recurrent pleural effusion due to BD.

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