A Case of Severe Pleural Effusion and Pulmonary Dysfunction Associated with Occupational Exposure to Asphalt Tar Smoke is Reported

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Abstract: Asphalt and tar transportation personnel are often exposed to the polluted air environment of asphalt fumes, tar and diesel exhaust. This long-term occupational exposure can adversely affect lung function, causing fibrosis, pleural effusion, and inflammation. This paper reports a case of pleural effusion in a 35-year-old male who had been engaged in asphalt paving and transportation for 5 years. There was no occupational exposure protection during the working period. The patient had dyspnea, expectoration, and pleural effusion for more than 1 month. After admission, thoracic drainage and pleural cauterization dissection were performed, and the symptoms were relieved. However, pulmonary fibrosis and visceral pleural thickening are challenging to reverse, and patients still have pulmonary dysfunction and the risk of continuing to develop lung consolidation. Therefore, the personnel engaged in asphalt and tar transportation should be well-protected to reduce occupational exposure.

Keywords: Asphalt Fumes; Pleural Effusion; Pulmonary Function; Occupational Exposure

1. Background

Workers engaged in asphalt transportation and paving are exposed to asphalt smoke through inhalation and skin contact throughout their working life, which is one of the main occupational groups most often exposed to toxic and harmful chemical pollutants[1]. Bitumen is a black, highly viscous organism that is a liquid or semisolid product extracted from petroleum[2]. It plays a role in cementing stones and gravel in road construction. Bitumen contains a variety of chemical and biological pollutants, including volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAH), naphthene, inhalable particulate matter (PM), nitrogen oxides and sulfur compounds[3]. When the asphalt is treated at a high temperature in the electrode roller, the above toxic and harmful substances will volatilize a large amount of asphalt smoke[4]. Because the bitumen contains carcinogenic substances, 3, 4-benzo (a) pyrene, the content of which is as high as 2.5%-3.5%, evaporates with the flue gas. PAH can be inhaled and deposited in the respiratory system, causing lung fibrosis, pleural adhesion, pleural effusion, and even lung cancer[5]. Therefore, this paper reports a case of a patient with severe pleural effusion, pleural adhesion, and pulmonary dysfunction due to long-term exposure to asphalt. It is hoped to reduce misdiagnosis and organ damage caused by such occupational exposure in the future.

2. Case presentation

The patient was a 35-year-old male who had been engaged in asphalt tarmac paving and transportation for 5 years. The chief complaint of the patient was dyspnea and expectoration for more than 1 month, with occasional bloody sputum. In the

local hospital, it was misdiagnosed as malignant pleural effusion (neoplastic). Later, closed thoracic drainage was performed in our hospital, and the daily drainage volume was 500ml for 7 consecutive days, draining red-brown pleural effusion. The exfoliative cytology of pleural effusion showed some inflammatory cells and some exfoliated tracheal epithelial cells. A Blood routine test after admission showed that the white blood cell count was 6.34×10^9 g /L, the absolute value of monocytes was 0.76, and the other indicators were standard. The tumor marker CA125 was 81.2 KU /L, and the tumor abnormal glycan protein TAP was 131.185μm^2. Contrast-enhanced CT of the chest 13 days before surgery showed large area of atelectasis in the right lung, multiple patchy high-density shadows in the right lung, patchy soft tissue shadows beside the mediastinum of the lower lobe of the right lung, which seemed to be mildly enhanced, and a large amount of effusion in the right pleural cavity. There was a flocculent high-density shadow in the lower lobe of the left lung (Fig.1A). Symptomatic and supportive treatment including anti-infection, antitussive and expectorant treatment was continued, and pleural effusion was drained. Contrast-enhanced CT of the chest 2 days before surgery showed large area of atelectasis in the right lung, multiple patchy high-density shadows in the right lung, and patchy soft tissue shadows beside the mediastinum of the lower lobe of the right lung, which seemed to be mildly enhanced. There was a large amount of effusion shadow in the right pleural cavity, and the drainage tube shadow was visible. Multiple cystic low-density shadows were found in the posterior mediastinal esophagus and cardia, and the edge of the lesions was enhanced on a contrast-enhanced scan. There was a flocculent high-density shadow in the lower lobe of the left lung (Fig.1B). Pulmonary function tests one day before surgery showed that TLC-SB% was 58.7%, FRC-SB% was 76.6%, and DLCO-SB% was 31.3%, indicating severe restrictive ventilation dysfunction, a severe reduction in pulmonary diffusion capacity, and reduction in alveolar diffusion capacity.

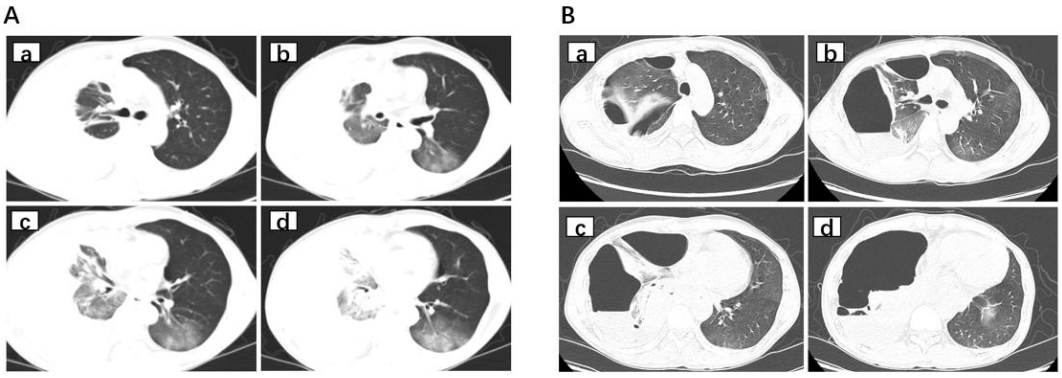

Figure 1. A Massive right pleural effusion with right lung atelectasis; Inflammation of the right lung. Localized emphysema in the apex of the right lung. Inflammation in the lower lobe of the left lung. **B** Massive pleural effusion in the right lung with right atelectasis, right lung inflammation and pulmonary fibrosis, pleural adhesion, and right pneumothorax.

For further treatment, cauterization of pleural adhesion and decortication were performed in the right thoracic cavity under the thoracic cavity. The surgical procedure was as follows: the pleural adhesion was separated under thoracoscopy, the pus was aspirated, the necrotic material was cleaned, and the thickened pleural fibrous plate was stripped. Intraoperative findings: extensive adhesion of the right pleura, consolidation of the right lower lung without expansion, yellow necrosis and fibrinous necrosis on the surface of the right lower lung near the costophrenic angle (Fig.2A). Postoperative pathology showed that the grey-red chest wall and pleural tissue were inflammatory exudation and bleeding tissue. The size of the capsule was about 4.5 × 3.5 × 1.0cm in total, the inner wall of the capsule was grey-red and smooth, and the wall thickness was about 0.1-0.3cm (Fig.2B).

CT examination on the third day after the operation showed that the brightness of both lungs was normal and the texture was disordered. The right lung showed postoperative changes, with large patches of high density shadow and fluid low density shadow in the right thoracic cavity, and local thickening of the pleura. Gas shadow and drainage tube shadow were

found in the right thoracic cavity. In the left lower lobe, there was a small high-density nodule shadow and a cord-like high-density shadow (Fig.2C). The patient's symptoms were relieved after surgery, but his pulmonary fibrosis and consolidation were challenging to reverse, and he still had pulmonary dysfunction. He needed to continue to exercise in the later stage and stay away from asphalt smoke.

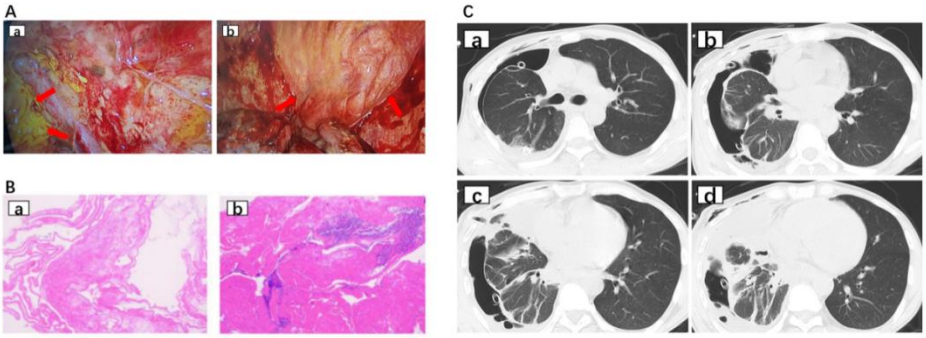

Figure 2. A Intraoperative thoracoscopic findings. **a** Yellow necrosis and fibrinoid necrosis (red arrows) on the lower right lung surface near the costophrenic Angle; **b** Showed pleural thickening, pulmonary fibrosis, and consolidation in the lower right lung layer (red arrows). **B** Chest wall and pleural tissue, all of which showed inflammatory exudation and bleeding tissue. **C** Postoperative changes of the right lung, right lung and left lower lobe inflammation.

A small nodule in the lower lobe of the left lung was recommended for follow-up. Right fluid pneumothorax. There was localized thickening of the right pleura. There was gas in the right chest wall.

3. Discussion

The patient had been engaged in asphalt pavement and transportation for 5 years, and had no daily occupational exposure protection. The patient developed massive pleural effusion, pleural adhesions, and pulmonary dysfunction due to long-term exposure to pitch smoke. There are still pleural thickening and pulmonary consolidation after operation, and long-term pulmonary function exercise is needed in order to obtain better recovery. Regular use of personal protective equipment (PPE) may therefore reduce this health risk. Personal protective equipment shall be strictly used at work, and pollution from construction projects and the emission of toxic substances shall be reduced as far as possible in accordance with regulations. Early assessment of occupational exposure susceptibility biomarkers in workers to reduce adverse health consequences of bitumen related workers**.**

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