Olgu Sunumu
Case Report

Pneumoconiosis accompanied by pulmonary thromboembolism: Case Report

PULMONER TROMBOEMBOLİNİN EŞLİK ETTİĞİ PNÖMOKONYOZ OLGUSU

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ABSTRACT

Pneumoconiosis are parenchymal lung diseases caused by dust accumulation in the lungs and the resulting tissue reaction. A 75-year-old male patient presented with the complaints of fatigue and shortness ofbreath for 6 months. According to his professional history when he was 18 years old, he worked in a lead mine blasting with dynamite for 4 months, and then worked in tunnel and road construction, blasting, sand screening and transportation for 3 months. According to the International Labor Organization (ILO) International Classification of pneumoconiosis radiographs, the chest radiograph was r/q 2/2 + A2. In his history, it was learned that he applied to our hospital with the complaint of bloody sputum in 1982, open lung biopsy was performed for tuberculosis and malignancy, and the biopsy was reported as fibrotic lung tissue. We presented a case of pneumoconiosis presenting with bilateral pleural effusion accompanied by pulmonary thromboembolism.

Keywords: Embolism, pneumoconiosis, tuberculosis

ÖZ

Pnömokonyozlar, akciğerlerde toz birikimi ve bunun sonucu oluşan doku reaksiyonu ile meydana gelen parankimal akciğer hastalıklarıdır. Yetmiş beş yaşında erkek hasta 6 aydır devam eden çabuk yorulma ve nefes darlığı şikayeti ile başvurdu. Meslek öyküsünde, 18 yaşındayken 4 ay kurşun madeninde dinamit ile patlatma işi yaptığı, sonrasında 3 ay boyunca tünel ve yol yapımında patlatma, kum eleme ve taşıma işinde çalıştığı öğrenildi. Pnömokonyoz radyografilerinin International Labour Organization (ILO) Uluslararası Sınıflandırması'na göre akciğer radyografisi r/q 2/2 + A2 idi. Özgeçmişinde 1982 yılında hastanemize kanlı balgam şikayetiyle başvurduğu, tüberküloz ve malignite açısından açık akciğer biyopsisi yapıldığı ve biyopsinin fibrotik akciğer dokusu olarak raporlandığı öğrenildi. Bilateral plevral efüzyon ile prezente olan, pulmoner tromboembolinin eşlik ettiği bir pnömokonyoz olgusunu sunduk.

Anahtar kelimeler: Emboli, pnömokonyoz, tüberküloz

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Pneumoconiosis is the accumulation of inorganic dust, fumes and fibers mostly originating from the industrial environment, causing a fibrotic tissue reaction in the lung (1). Silicosis, coal worker's pneumoconiosis (CIP), asbestosis, berylliosis, hard metal lung disease, mixed dust

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pneumoconiosis and talcosis are the most well-known pneumoconiosis (2). Pneumoconiosis are examined in 2 main groups according to their radiological appearance as simple and complicated. Simple pneumoconiosis is the presence of round or linear opacities less than 1 cm in the chest radiograph. Progressive massive fibrosis (PMF), also known as complicated pneumoconiosis, is the appearance of opacities larger than 1 cm with small opacities on chest X-ray (3,4). Silicosis develops with inhalation of the crystalline form of silica (silicon dioxide). Silica is an essential component of rock and sand. Many different lines of business carry the risk of silicosis (5). Pulmonary thromboembolism (PTE) is defined as pulmonary artery occlusion of thrombotic material mostly originating from the lower extremities. It has a wide variety of presentations ranging from an asymptomatic coincidental finding to sudden death (6). There are studies reporting that the risk of developing PTE increases in conditions with chronic inflammation such as pneumoconiosis. Here, we present a 75-year-old man who presented with bilateral pleural effusion associated with PTE.

CASE REPORT

A 75-year-old male patient presented with the complaints of fatigue and shortness of breath for 6 months. His general condition was evaluated as good, cooperative and oriented. Oxygen saturation was 96%. Respiratory system examination was normal. In the new chest X-ray compared to the chest X-ray in 2016 showed bilaterally sinuses blunt, increase in nodular lesions bilaterally in the upper-middle zone and newly developed a mass lesion of approximately 1.5 cm in diameter in the upper zone of the right lung (Figure 1a, 1b).

Figure 1a.

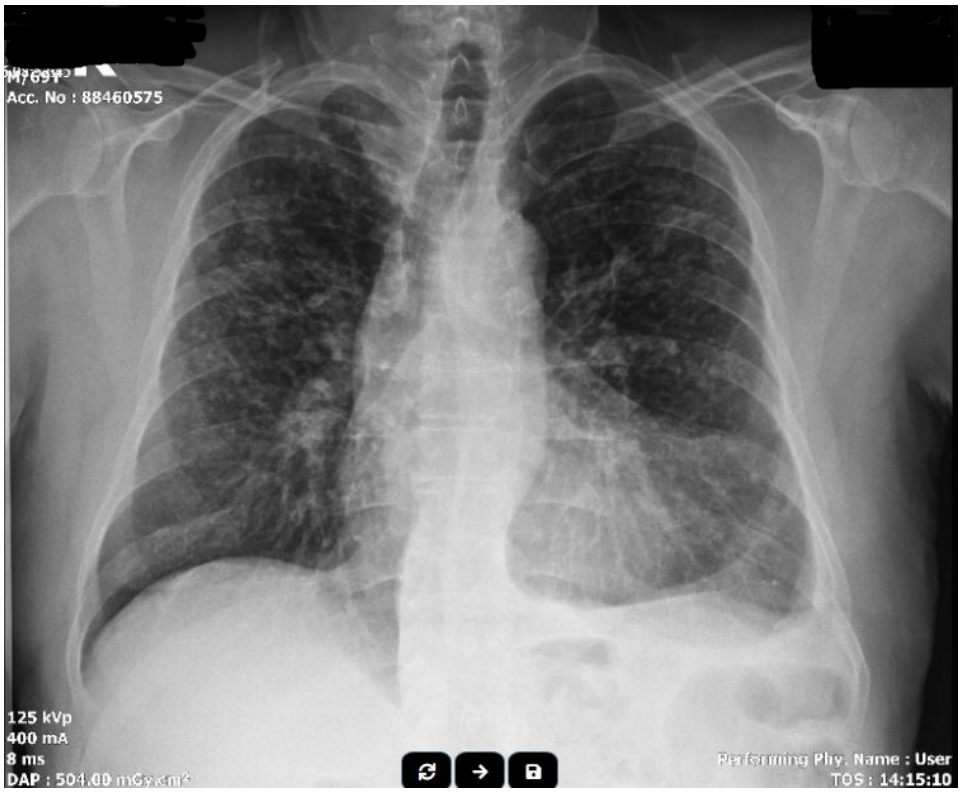
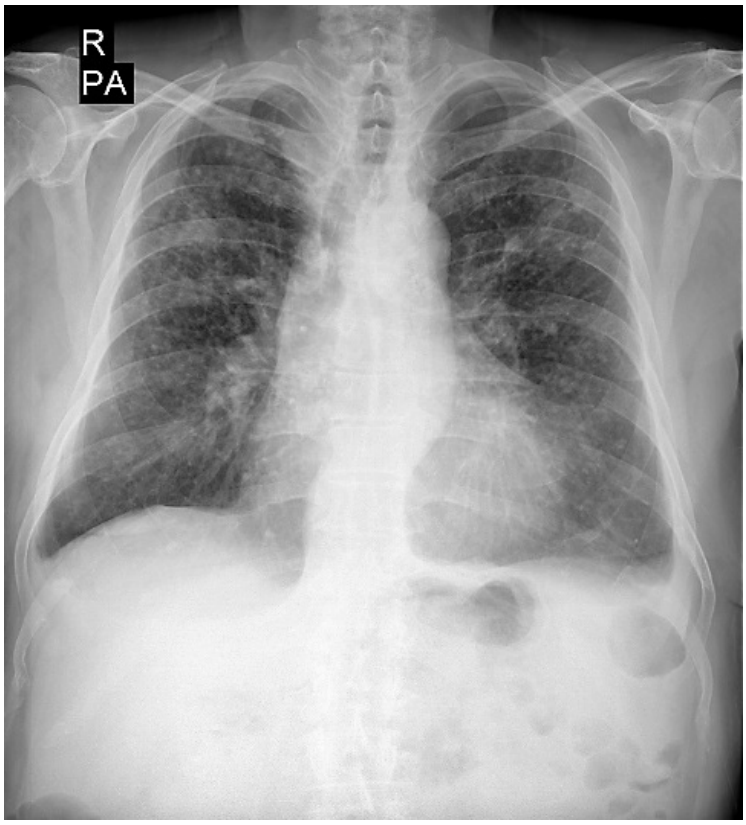


Figure-1 a,b: In the new chest X-ray compared to the chest X-ray in 2016 showed bilaterally sinuses blunt, increase in nodular lesions bilaterally in the upper-middle zone and newly developed a mass lesion of approximately 1.5 cm in diameter in the upper zone of the right lung

Figure 1b



Thoracentesis was performed and as a result of pleural effusion exudate, adenosine deaminase level 8 (IU/L), acido-resistant bacilli (ARB) staining was negative. There was no growth in the pleural fluid culture. PTE accompanying pneumoconiosis was suspected due to ongoing chest pain and negative t on ECG in the patient who presented with D-dimer 1770 (ng/ml). In the thorax CT angiography, filling defects compatible with PTE were observed in several bilateral perihilar and intraparenchymal branches (Figure 2).

Figure 2

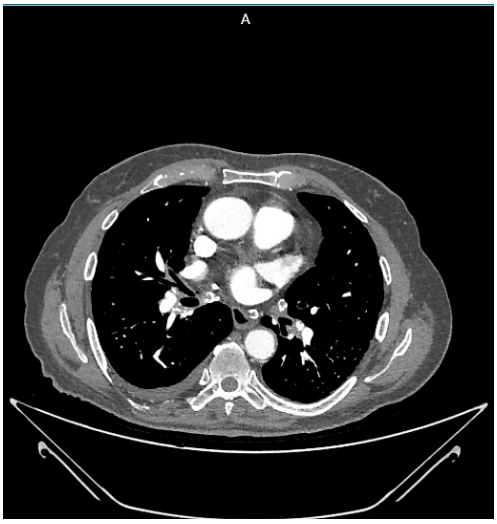


Figure-2: On thoracic computed tomographyfilling defects compatible with PTE in several bilateral perihilar and intraparenchymal branches

Simultaneous lung high-resolution computed tomography showed nodular lesions consistent with pneumoconiosis and progressive massive fibrosis in bilateral upper-middle zones of the lung (Figure 3a,3b).

Figure-3 a,b

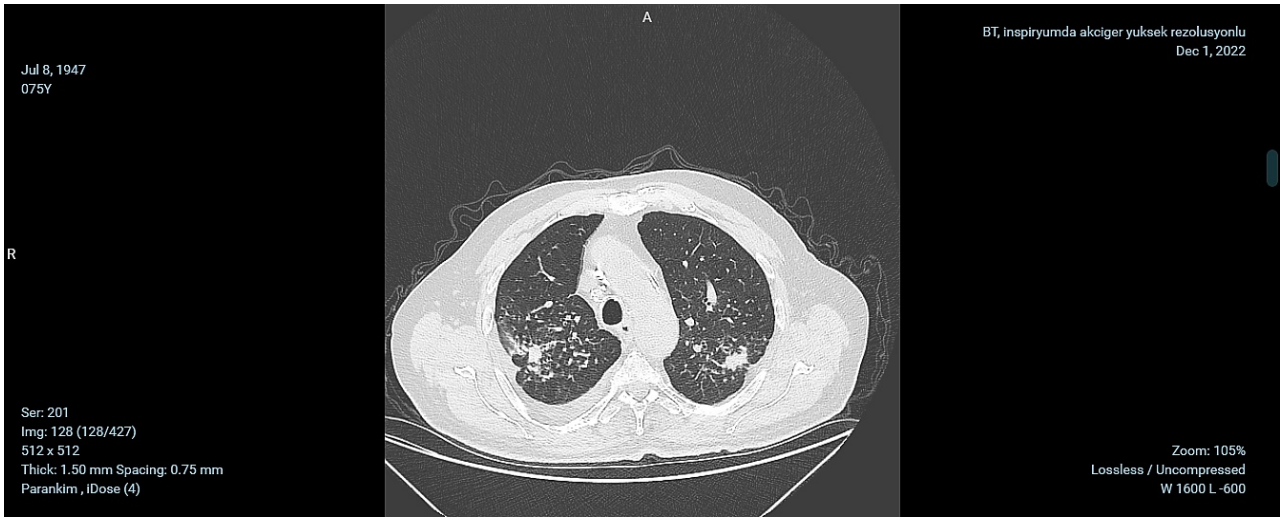


Figure-3 a,b: On thoracic computed tomography nodular lesions consistent with pneumoconiosis and progressive massive fibrosis in bilateral upper-middle zones of the lung

In his professional history, it was learned that when he was 18 years old, he worked in a lead mine blasting with dynamite for 4 months, and then worked in tunnel and road construction, blasting, sand screening and transportation for 3 months. The patient, who later served as a police officer for 22 years, retired in 1997 and did not engage in any additional work. In his history, he applied to our hospital with the complaint of bloody sputum in 1982, open lung biopsy was performed for tuberculosis and malignancy, and the biopsy was reported as fibrotic lung tissue. He had a 20 pack-year smoking history but had not smoked for 30 years. Pulmonary function test was within normal limits. In the follow-up of the patient who was added anticoagulant to the treatment, the pleural effusion regressed and the diagnosis of pneumoconiosis was made and staging and reporting was done. According to the International Labor Organization (ILO) International Classification of pneumoconiosis radiographs, the chest radiograph was r/q 2/2 + A2.

DISCUSSION

Exposure of the respiratory system to inhaled inorganic dusts depends on many factors. The density of the exposed dust, the particle diameter, the shape and the exposure time are important. Personal factors are the person's respiratory rate and type, the distribution and concentration of inhaled particles in the lungs, and the ability of the lungs to clear them. Although the properties and amount of the dust are the same, the type and severity of the reaction in people may differ. This is due to the genetic susceptibility of the people, the difference in the power of the respiratory tract to clear dust, and the presence of cigarette exposure. The respiratory system responds to inhaled substances according to the level, duration and frequency of exposure intensity. If the dust accumulated in the lungs is completely or almost completely healed after the exposure ends and does not cause fibrosis, it is called benign pneumoconiosis. If the dust accumulated in the lungs causes fibrosis with tissue reaction, it is called collagenous pneumoconiosis. This may progress to PMF (2). Although our patient was exposed to

dust for as little as 7 months, fibrotic tissue reaction in his lungs after years shows that dust exposure has multifactorial effects.

According to the Virchow triad, there are three factors that predispose a person to the development of thrombi: venous stasis, vessel wall damage, increased blood coagulation (7). Chronic inflammation is responsible for the pathophysiology of pneumoconiosis and possibly contributes to the formation of venous thromboembolism by causing vessel wall damage (8). In addition, when pulmonary pathologies taken from patients with silicosis and CIP were examined, it was thought that perivascular fibrosis and stasis were observed in the terminal branches of the pulmonary vascular bed, which may lead to the development of venous thromboembolism (9). In the ILO classification of pneumoconiosis radiography, density categories of 0, 1, 2 and 3 were created according to the density of small opacities in the lungs. In a study by Song et al. investigating the clinical features of pulmonary thromboembolism and pneumoconiosis, they found that PTE generally develops in elderly patients with pneumoconiosis in category 2 and above (10). The ILO density category of our 75-year-old patient was 2.

CONCLUSION

Chronic complicated silicosis is generally thought to develop with inorganic dust inhalation for more than 10 years. Occupational dust exposure for 7 months in our case suggests that the physical properties of the dust, cumulative dust exposure and personal characteristics are also important in the development of pneumoconiosis in addition to the exposure time. There are studies reporting an increased risk of developing PTE due to ongoing chronic inflammation and vascular stasis in pneumoconiosis. The thromboembolic state is associated with particularly increased plasma levels of C-reactive protein, IL-6, IL-8, monocyte chemotactic protein-1 and TNF-α due to chronic inflammation. Therefore, the possibility of PTE development should be considered in the follow-up of patients with pneumoconiosis.

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