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(CASE REPORT)

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Pulmonary embolism and pneumomediastinum: Concomitant complications of pulmonary tuberculosis

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Abstract

The possible association between pulmonary embolism and tuberculosis (TB) infection has attracted growing interest among practitioners and researchers in recent years. Despite these efforts, pulmonary embolism is often under-diagnosed in patients with active TB. Although considered an uncommon phenomenon, pulmonary embolism appears to be a potential risk based on recent data. Similarly, although rare, pneumomediastinum can occur in patients with tuberculosis due to a variety of mechanisms. We present here the case of a young man presenting with respiratory symptoms, with no risk factors other than his smoking habit. This case report highlights the importance of considering these two complications in patients with tuberculosis. We hope that in the future, the management of tuberculosis will incorporate greater recognition of the associated risks, notably pulmonary embolism and pneumomediastinum.

Keywords: Tuberculosis; Pneumomediastinum; Pulmonary Embolism; Macklin Effect

1. Introduction

Tuberculosis affects the majority of developing countries every year, and remains one of the world's leading causes of death [1]. The disease is mainly caused by Mycobacterium tuberculosis, and is spread by respiratory droplets when coughing [2]. Pulmonary embolism, a frequent complication, carries a high mortality risk, warranting evaluation and appropriate treatment, including venous thromboembolism (VTE) prophylaxis in TB patients [4]. Pneumomediastinum is a medical condition characterized by the abnormal presence of air in the mediastinal region. Although rare in pulmonary tuberculosis, its occurrence may indicate a severe complication.

2. Case report

A young patient, A. A., 25 years old, chronic smoker with a consumption of 7 packs per year, with no history of pulmonary tuberculosis and no recent contact with the disease, has presented for 15 days with a productive cough accompanied by whitish sputum, an episode of severe hemoptysis, and right laterothoracic pain (VAS 4/10), all in a context of night sweats, fever reaching 39°C, and asthenia, anorexia and weight loss of 5 kilograms in one month.

On general examination, the patient is conscious and well oriented in time and space. His conjunctivae are slightly discolored, and his oxygen saturation is 95% on room air. His respiratory rate is 30 cycles per minute, his heart rate is 85 beats per minute, and his blood pressure is 10/06 cmHg.

Pleuropulmonary examination revealed subcutaneous emphysema in the thoracic and cervical regions on palpation. However, cardiovascular and lower limb examinations revealed no particular abnormalities.

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The chest X-ray (Figure 1) reveals a cavity in the right lobe accompanied by bilateral diffuse nodular and micronodular opacities, as well as linear hyperclarity along the mediastinum and cardiac contours, with continuous diaphragm sign. In addition, subcutaneous emphysema is observed at the cervical level on both sides. The XPERT genetic test performed on sputum showed average detection of Mycobacterium tuberculosis without rifampicin resistance.

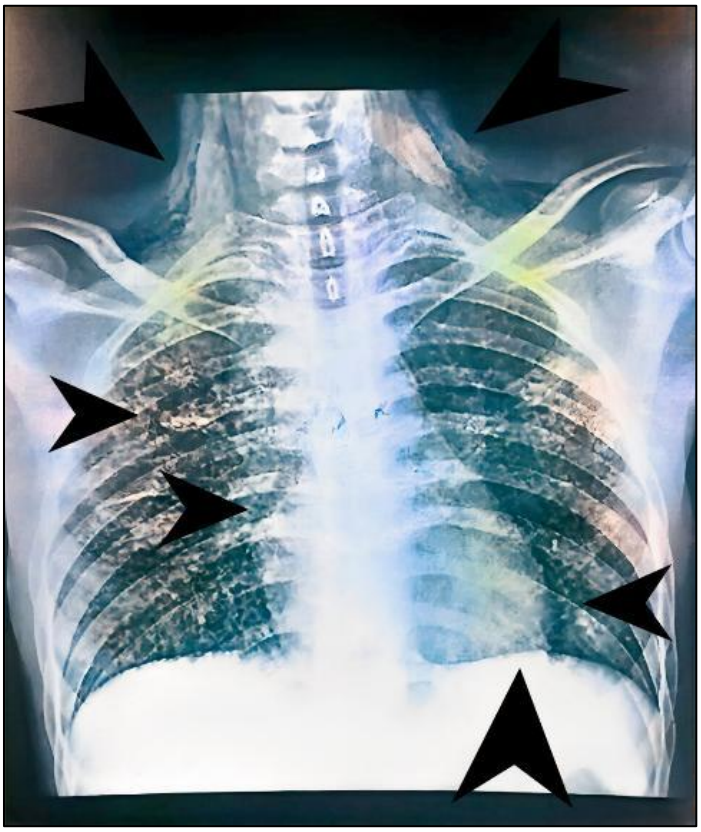
A thoracic CT scan (Figure 2, 3) shows scattered intra-parenchymal micronodules of peribronchial distribution associated with intra-parenchymal cavities and foci of dilatation of superinfected cylindrical and monolifromous bronchi, suggesting in the first instance pulmonary tuberculosis. A focus of condensation in the ventral segment of the culmen, related to pulmonary infarction, and pneumomediastinum of moderate severity, accompanied by subcutaneous emphysema affecting the anterolateral cervical and thoracic soft tissues, are also observed.

A lung perfusion scan (Figure 4, 5, 6, 7) reveals a well-systematized, peripherally-based, triangular perfusion defect in the anterior segment of the culmen, in favor of pulmonary embolism. A patch of poorly systematized right upper lobar hypocaptation, corresponding to intra-parenchymal cavities on the associated scannographic sections, is also observed, probably of tuberculous origin.

The initial laboratory workup revealed a CRP elevation of 157, with no other notable abnormalities.

Therapeutic management consisted in admitting the patient to our unit for observation, strict rest, oxygen therapy, administration of curative dose anticoagulants of low molecular weight heparin, hemostatic treatment in case of hemoptysis, analgesic treatment for pain, and initiation of antibacillary treatment with ERIPK4, 4 tablets per day for an initial weight of 52 kilograms.

The course was favorable, with resolution of cough, hemoptysis and chest pain, as well as reduction in pneumomediastinum and subcutaneous emphysema. 10 days after starting treatment, the patient regained his appetite and gained 5 kilograms in one month.



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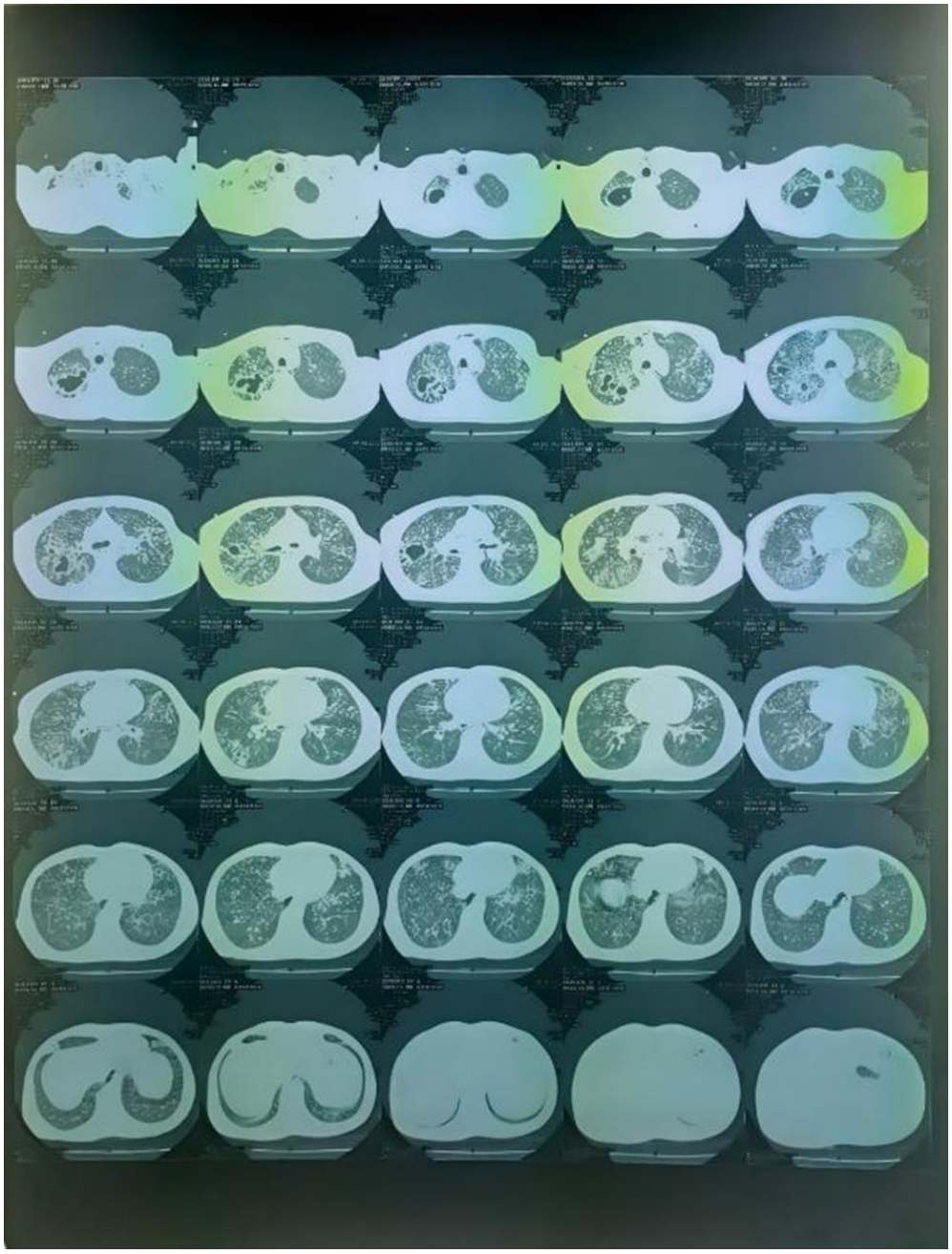

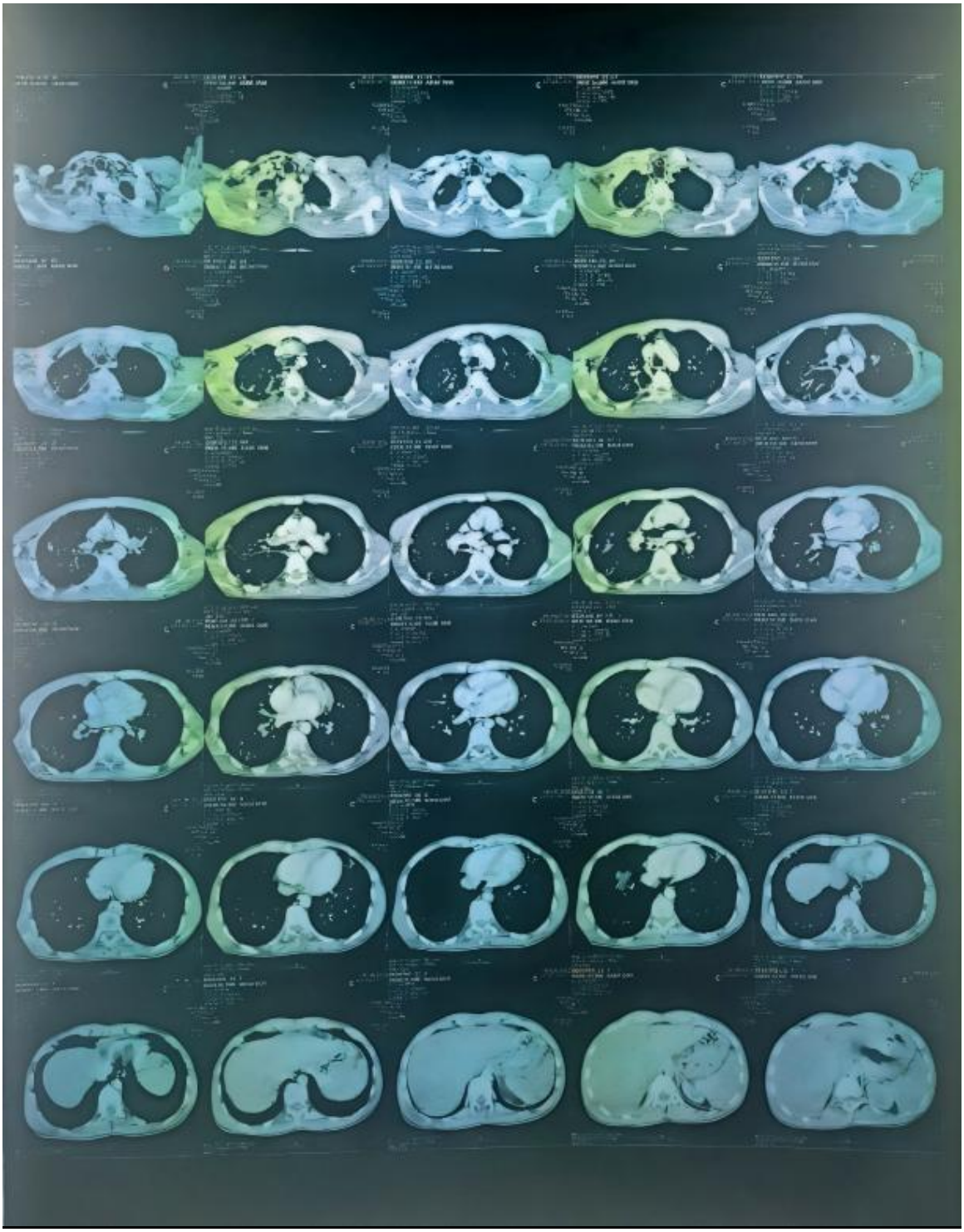

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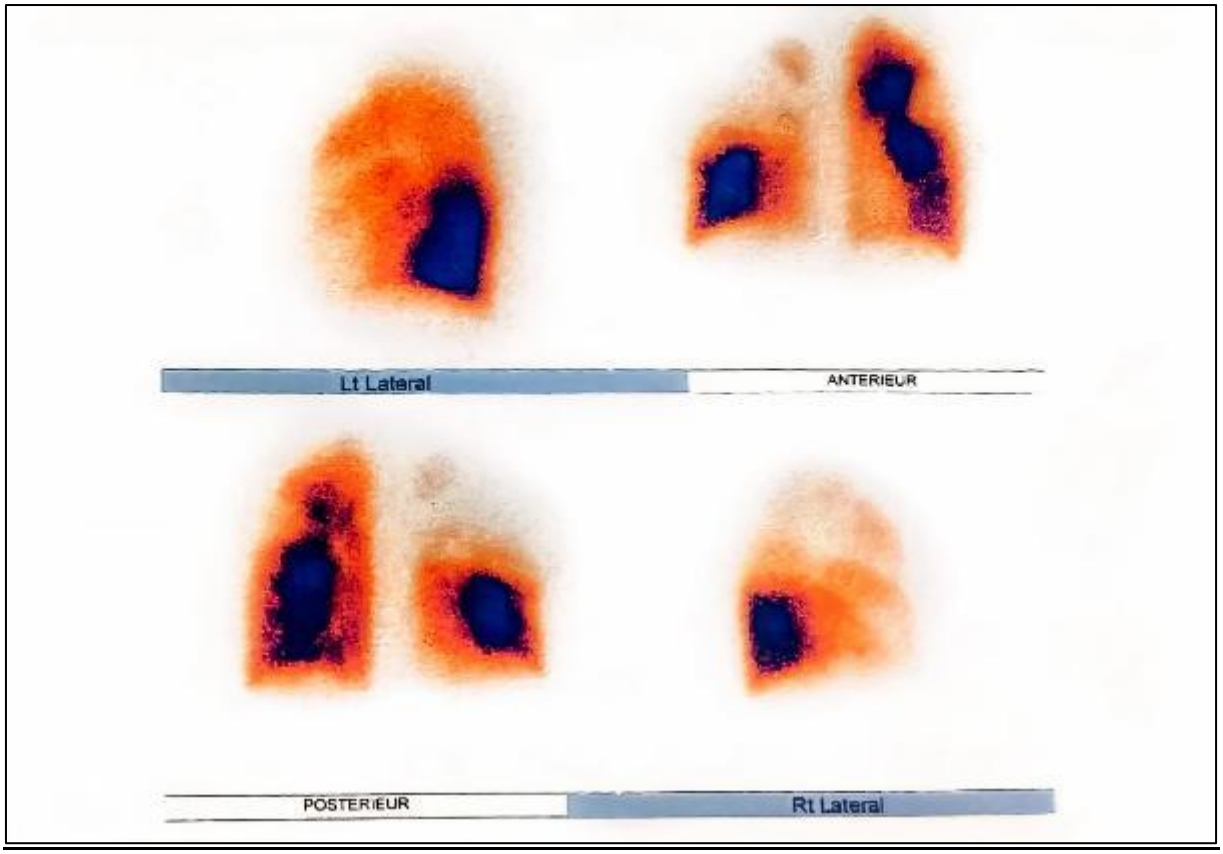
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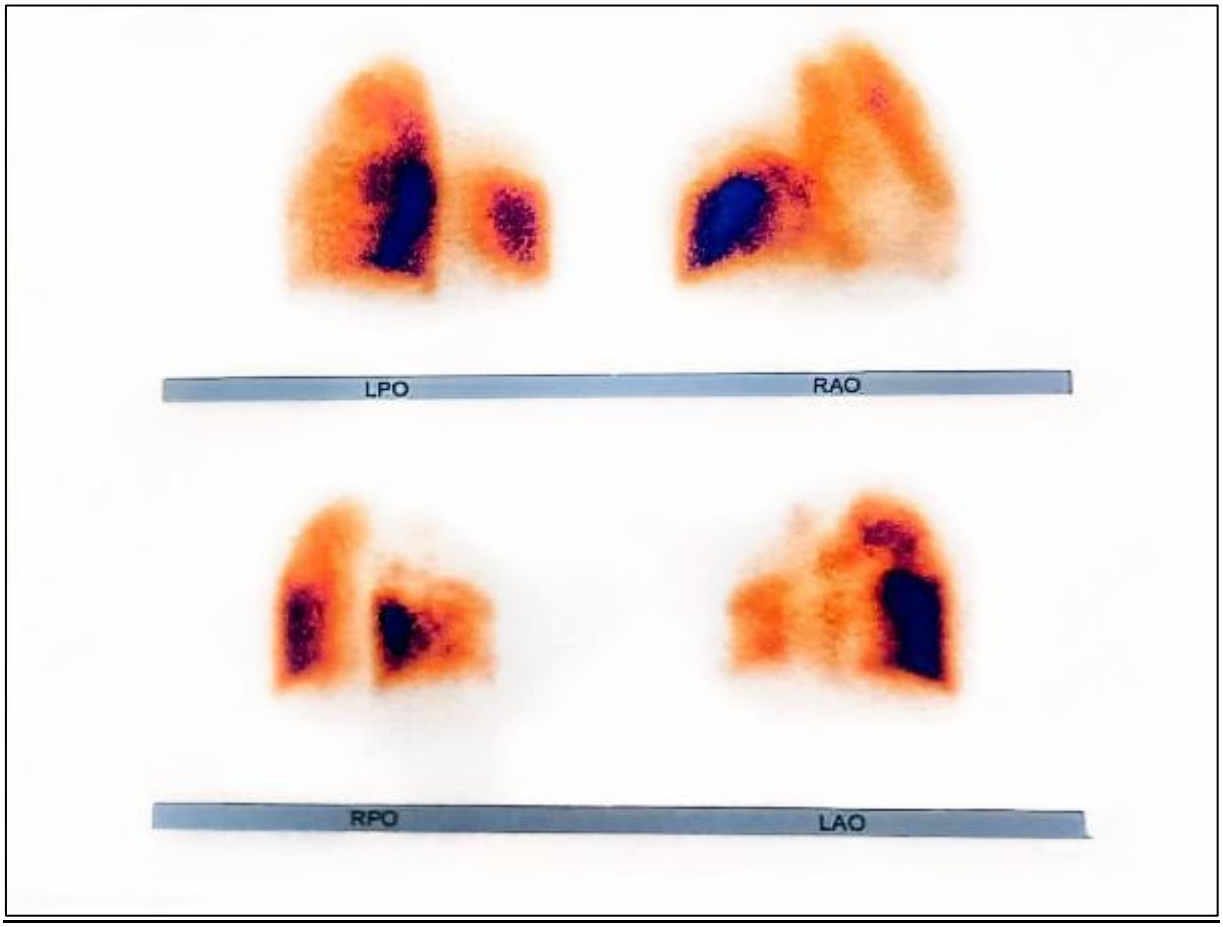
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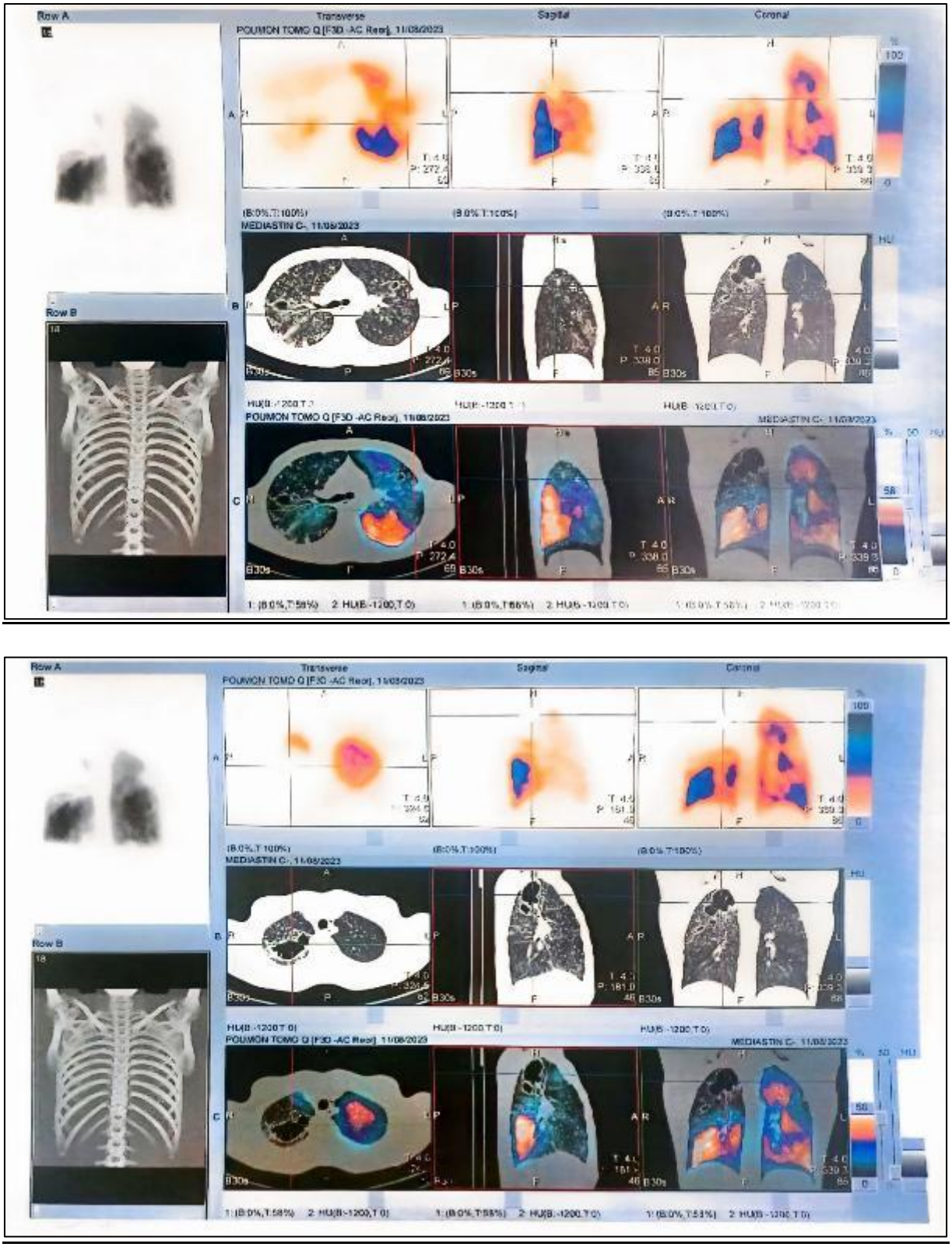

Figure 4 - 7 A lung perfusion scan reveals a well-systematized, peripherally-based, triangular perfusion defect in the anterior segment of the culmen, in favor of pulmonary embolism. A patch of poorly systematized right upper lobar hypocaptation, corresponding to intra-parenchymal cavities on the associated scannographic sections, is also observed, probably of tuberculous origin

3. Discussion

Tuberculosis infection manifests itself in a variety of ways, producing a range of symptoms depending on the system it affects. Pulmonary involvement, which is the predominant manifestation, contributes to pulmonary symptoms, but is not strictly limited to cough with or without hemoptysis, chest pain and breathing difficulties. Hypercoagulability in patients with active tuberculosis has begun to be reported in recent years. According to a study published in 2019, only 49 (0.6%) of 7,905 patients diagnosed with TB were predisposed to the formation of thrombogenic profiles and complications such as deep vein thromboembolism (DVT), pulmonary embolism (PE) or both. A greater proportion of patients had PE than DVT, 42.9% and 26.5% respectively. The remaining 30.6% of patients had DVT and PE simultaneously [5,6].

The very first report to demonstrate a strong correlation between active tuberculosis and pulmonary embolism was published in 1950. A total of 634 autopsies (24.3%) revealed pulmonary embolism in cases of active tuberculosis, whereas the incidence of pulmonary embolism was 23.1% in the series as a whole [7]. People with active TB infection are almost as likely to develop VTE as those with neoplasia [4]. Other publications have focused on hemostatic changes in the context of tuberculosis infection, leading to hypercoagulability.

The study by R.BOUCHENTOUF, published in 2021 in the JFVP [8], has shown that thromboembolic events are often concomitant with the diagnosis of tuberculosis, but can sometimes precede tuberculosis disease or occur during its treatment.

In our case, the patient had no other risk factors for thromboembolism, apart from his recently diagnosed tuberculosis infection and smoking history. This underlines the importance of considering tuberculosis infection as a potential risk factor for thromboembolic complications, even in the absence of other obvious risk factors.

At the same time, pneumomediastinum, characterized by the abnormal presence of air in the mediastinal region, can occur in patients with pulmonary tuberculosis for several reasons:

The pulmonary necrosis associated with tuberculosis can lead to the formation of cavities in the lungs, which, if ruptured, can release air into the mediastinum. In addition, this disease can promote the creation of breaches in the airways, allowing air to diffuse into the mediastinum through these breaches.

The severe, persistent cough often observed in tuberculosis patients can also cause alveolar ruptures (Macklin effect), allowing air to infiltrate the mediastinum. In addition, the inflammation caused by tuberculosis infection can weaken lung structures, increasing the risk of air leakage into the mediastinum.

In our case, the presence of subcutaneous emphysema dissecting the cervical and thoracic soft tissues, associated with pneumomediastinum, could be attributed to the Macklin effect, a phenomenon where increased alveolar pressure during coughing leads to alveolar dissection and air leakage into the mediastinum and along soft tissue interstitial spaces.

4. Conclusion

According to new research data, there appears to be a frequent association between tuberculosis infection and pulmonary embolism. Although the development of pneumomediastinum in patients with tuberculosis is rare, it remains possible due to the different mechanisms described above, and can lead to serious complications for these patients. This case highlights the importance of considering tuberculosis as a potential risk factor for the development of thromboembolic events, as well as a possible contributory factor in the development of pneumomediastinum. All these factors must be taken into account when managing patients with active pulmonary tuberculosis, in order to prevent life-threatening complications such as pulmonary embolism and pneumomediastinum.

Compliance with ethical standards

Disclosure of conflict of interest

The author declares no conflict of interest.

Statement of informed consent

All participants included in the study provided informed consent.

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