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Idiopathic Pulmonary Alveolitis (Clinical Case)

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Abstract: The article presents a clinical case of a rare disease – idiopathic pulmonary alveolitis. A 24-year-old male patient was admitted to the Republican Scientific Center for Emergency Medical Care with a referral diagnosis of "Pneumonia". Recurrent episodes of pneumonia had been occurring frequently over the past 3 years, for which the patient had repeatedly received antibacterial therapy on both outpatient and inpatient settings. He had no history of COVID-19; tuberculosis was ruled out. Upon admission to our clinic, signs of chronic hypoxia were observed (cyanosis, digital clubbing), with SpO_2 at 86% (on oxygen). Spirometry showed a vital lung capacity of 1850 mL. ECG indicated right heart strain. Chest X ray revealed a reticular enhancement of the lung pattern due to diffuse deformation of the interstitial tissue. Based on the above clinical features, the patient was diagnosed with idiopathic pulmonary alveolitis. Steroid therapy and calcium antagonists were added to the treatment regimen. As a result of this treatment, the patient's condition significantly improved, and he was discharged home in a stable condition for outpatient follow up. This clinical case illustrates the characteristics and treatment principles of this extremely rare disease.

Keywords: Hamman–Rich syndrome, fibrosing alveolitis, clinical presentation, diagnosis, treatment, steroid therapy.

Introduction: Idiopathic pulmonary alveolitis was first described in the 1930s–1940s by L. Hamman and A. Rich, who identified the development of characteristic pulmonary fibrosis in this disease [1]. The diagnostic frequency of this disease is increasing. The average age of patients is 50 years, and it is more common in males. In this disease, progressive fibrosis of the pulmonary connective tissues develops, and without appropriate treatment, this process leads to the patient's death within 2–6 months. In the 1960s, J. Scadding made a significant contribution to the study of this problem and introduced the term "fibrosing alveolitis" [2]. In the countries of the former Soviet Union, the term "idiopathic fibrosing alveolitis" is more commonly used, while in America, Europe, and Great Britain, the term "fibrosing alveolitis" began to be used [3,4].

Hamman–Rich syndrome belongs to the group of primary pulmonary fibroses. Its etiology is unknown. Morphological changes are characterized by the development of fibrosis in the interalveolar septa and along the small pulmonary vessels, accompanied by inflammatory processes. This condition leads to impaired gas exchange across the alveolar–capillary membrane, resulting in respiratory failure and dyspnea. Many authors, considering the morphological changes of this disease, call it "diffuse fibrosing alveolitis" and include it among systemic connective tissue diseases such as rheumatoid arthritis and scleroderma [5]. Histological examination may reveal morphological changes including thickening of the

visceral pleura, hyalinosis in the lung tissue, fibrosis due to the proliferation of collagen fibers in the alveolar septa, and decreased pneumatization of the lung tissue. In addition, capillaritis (vasculitis of capillaries), homogeneous thickening of vessels, and edema of the lung parenchyma are noted [6,7].

In making the diagnosis of idiopathic pulmonary alveolitis, the following criteria are taken into account: a previous diagnosis of pulmonary fibrosis in the patient; worsening dyspnea within the last 2 months; a "reticular fibrosis" pattern on X-ray and MSCT; and the absence of infection (tuberculosis and other pathogens) on microbiological examination.

This article presents a clinical case of a very rare disease in clinical practice – Hamman–Rich syndrome – which is therefore not well known to our physicians.

Description of the Clinical Case

A 24-year-old male patient was treated for Hamman–Rich syndrome. He was admitted to our clinic on December 20, 2022, and discharged home on December 28, 2022. Medical history No. 48300/4324.

Complaints upon admission: Dyspnea worsening with physical exertion, difficulty expectorating sputum, chest tightness and pain, palpitations.

Medical history: The patient has considered himself ill for 10 years. At the onset of the disease, he was diagnosed with nonspecific pneumonia. The patient had no history of COVID-19. Tuberculosis was ruled out. The disease worsened over the last 2 months. The patient has no harmful habits. Several courses of strong antibacterial and anti-inflammatory drugs in inpatient settings were ineffective. He consulted a physician due to worsening mild cough and dyspnea.

Objective examination: Cyanosis of the face and digital clubbing (drumstick fingers) were observed. SpO_2 was 86% (on oxygen). Respiratory rate was increased – 28 breaths per minute. Peripheral lymph nodes were not palpable. On auscultation of the lungs, vesicular breathing was diminished, and fine moist rales were heard in the lower lung fields. The right border of the heart was enlarged, and an accentuated second heart sound (P2) was heard over the pulmonary artery.

Electrocardiogram (ECG): Right ventricular strain, decreased interventricular conduction, and repolarization disturbances in leads III and aVF were noted.

Chest X-ray: The lung pattern showed increased deformation, and the interstitial tissue appeared as coarsened reticular nodules.

Spirometry: External respiratory function changes were observed – a decrease in vital capacity (VC) due to restrictive impairment (VC = 37% of predicted).

Peripheral blood analysis: Hemoglobin – 162 g/L, erythrocytes – 5.4×10^{12}/L, color index – 0.9, leukocytes – 4.0×10^{9}/L, band neutrophils – 5%, segmented neutrophils – 60%, lymphocytes – 20%, monocytes – 8%, eosinophils – 3%, ESR – 50 mm/h. Mycobacterium tuberculosis was not found in sputum.

MSCT (Multislice Computed Tomography): Signs of interstitial pneumonia and reticular fibrosis were detected (see figure).

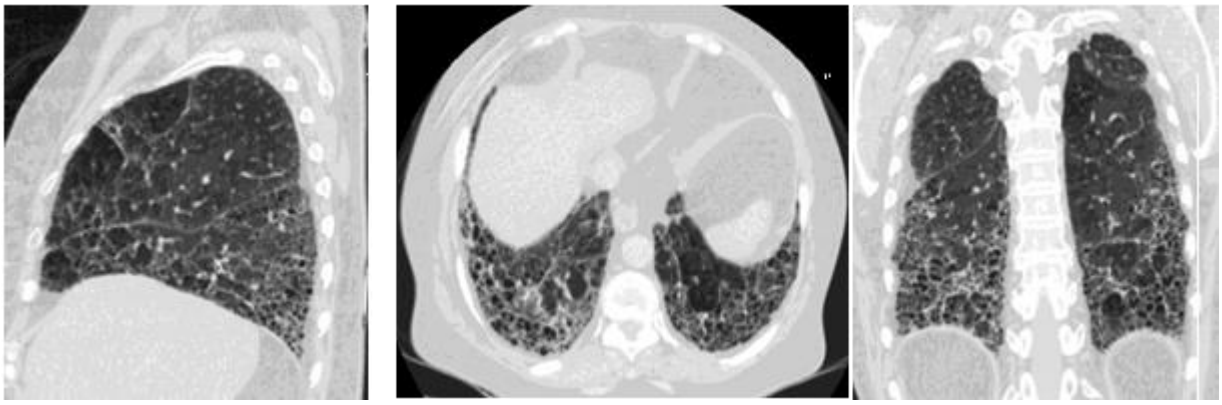

Figure. MSCT: Interstitial pneumonia, reticular fibrotic tissue.

Pharmacological Therapy

The patient received the following therapeutic measures:

1. Oxygen therapy
2. Calcium channel antagonists in small doses
3. Steroid hormones
4. Antibacterial therapy to prevent secondary pulmonary infections
5. Symptomatic therapy

As a result of this therapy, the patient's condition improved significantly, but cyanosis of the lips and dyspnea persisted. Antibacterial therapy (amikacin, ceftriaxone), aminophylline (eufillin), and Pulmicort inhalations were added to the inpatient treatment. Glucocorticosteroids (GCS), calcium antagonists, and

oxygen therapy led to some improvement in pulmonary function but did not produce a satisfactory result.

Long-term treatment with glucocorticoids leads to certain adverse complications. In the treatment of idiopathic pulmonary alveolitis, there is no clear evidence that combination therapy with GCS and immunosuppressants (e.g., azathioprine and cyclophosphamide) prolongs patient survival. However, combination therapy may improve pulmonary function. Currently, warfarin and phosphodiesterase-5 inhibitors (sildenafil group) are not used in idiopathic pulmonary fibrosis due to lack of efficacy.

Thus, the clinical features in the patient described above are consistent with Hamman–Rich syndrome.

Diagnosis of Idiopathic Pulmonary Alveolitis

Diagnosing idiopathic pulmonary alveolitis poses significant difficulties for the clinician. The diagnosis should be based on a thorough medical history, excluding previous nonspecific inflammatory lung diseases, chronic obstructive bronchitis, segmental pneumonia, COVID-19, pulmonary tuberculosis, and lung cancer. The following findings indirectly support the diagnosis of Hamman–Rich syndrome: progressive dyspnea, facial cyanosis, palpitations, difficulty expectorating sputum, digital clubbing ("drumstick fingers"), and lack of response to nonspecific treatment.

Furthermore, a comparative diagnostic workup requires:

- Radiological findings (reticular deformation of lung tissue)
- MSCT findings (interstitial pneumonia, typical reticular fibrotic pattern)
- Spirometry (reduced vital capacity)
- Laboratory findings (leukocytosis and elevated erythrocyte sedimentation rate)

In our opinion, in prolonged forms of the disease with structural lung tissue damage, punch biopsy is recommended.

Of particular importance are the findings on chest X-ray and MSCT, specifically interstitial pneumonia and the typical reticular fibrotic pattern.

CONCLUSION

In the diagnosis of idiopathic pulmonary alveolitis, it is necessary to perform radiography, spirometry, and MSCT, as well as to conduct a differential diagnosis with all other pulmonary diseases in order to reduce diagnostic errors. Therapeutic measures should include calcium channel antagonists, steroid hormones, and oxygen therapy.

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