

Fibrosing Alveoliti with Systemic Sclerodermy

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Abstract:

pulmonary involvement proceeds in SSD is caused by arterial damage and / or fibrosing process in the pulmonary parenchyma and in the form of two main syndromes rarely combined in one patient - pulmonary arterial hypertension (PAH) and interstitial lung lesions (IPL). [5]. The incidence of pulmonary pathology is high with SSD. Changes are found on chest radiographs in 45-56% of patients. We analyzed the frequency of lung injury in patients with chronic cardiovascular disease in the rheumatological department for five years. IPL identified only at 11.2%. The case of Fibrosing alveolitis was presented in the onset of the disease with its late diagnosis as a demonstration.

Keywords: *fibrosing alveolitis, systemic scleroderma.*

The immune disorders are the basis of pathogenesis of systemic scleroderma (SSD), also a vasculopathy with severe microcirculation disorders and generalized fibrosis [2]. Lung damage affects adversely on the prognosis and ranks first among the causes of death with SSD. Pulmonary disorders are most often detected with the greatest frequency, with multispiral computed tomography (MSCT) of the chest - in 80% of cases [3]. Data from the literature indicate that the first years of the SSD are determinative in terms of the origin and development of viscerites in general and IPL in particular. It is known that the beginning of the fibrous process in the lungs in most patients falls on the first 4-6 years of the disease [4]

The aim of the work was to analyze the incidence of IPL in patients with SSD and the features of their course.

Material and methods. We analyzed 39 case histories of patients with chronic obstructive pulmonary disease who were on treatment at the rheumatological department of the AOKB from 2012 to 2017. Among them, IPL were detected in five patients, which was 12.8%. In four cases IPL diagnosed an average of 5 years in the course of the disease, and in one case it was the debut of the disease. As an example, we present a clinical case of lung injury, in the form of fibrosing alveolitis, which outstripped other manifestations of systemic scleroderma for several years. Patient K., 26 years old, cook, was transferred to the rheumatological department in February 2017 from the pulmonology department, where she had been observed for two years before. She complained of dyspnoea at rest, aggravated with insignificant physical load, dry cough, chilliness of the hands,

discoloration of the distal sections of the hands to red and cyanotic color during excitement and low temperatures, rare choking when taking rough food, general weakness, decreased ability to work. From an anamnesis it is known that since 2012 she began to celebrate periodically a cough with the departure of scant sputum of light color, in 2013, during the fluorograph-ic examination, signs of bilateral pneumofibrosis were revealed, blood leukocytosis was diagnosed, and a chronic bronchitis was diagnosed. In 2014, with repeated fluorographic examination, the previous changes were preserved, a pulmonologist at the place of residence diagnosed with cystic hypoplasia. In 2015, with a gestation period of 25- 26 weeks, shortness of breath appeared during exercise. Deterioration from 37 weeks, when on the background of acute respiratory viral infection noted increased dyspnoea, cough, delivery was performed by caesarean section, postpartum MSCT of the thorax: CT scan can correspond to ELISA in the stage of “cellular lung” formation. To clarify the diagnosis and treatment transferred to the pulmonology department. Objectively: BH 22 in 1 min. When auscultation in the lower lateral thoracic parts of the right and left revealed bilateral crepitus. In blood tests revealed: acceleration of ESR up to 112 mm / h, leukocytosis-13,9x10⁹, iron deficiency anemia of mild severity (Hb-92 g / l, Ayr-3,53x10¹², serum iron-4.78 μmol / l), CRP-65.98 mg / l, cholesterol -6,79 mg / l. Bronchoscopy: bilateral diffuse atrophic endobronchitis, IV. Bronchoalveolar lavage: bronchial epithelium-18%, macrophages-34%; segments-42%, lymphocytes-6%. ECHO-KG: episodes of tachycardia. Increased stress on the right gastric check. Minor regurgitation of the TC. Additional trabeculae in the LV cavity. LV systolic function is preserved. The emission fraction is 65.6%.

Diastolic dysfunction of the prostate is impaired by the first type. There are no signs of LH. Spirogram: severe disturbances of VFL by mixed type (ZHEL-27%, FVC-33%, FEV1-29%). The diagnosis is made: idiopathic pulmonary fibrosis, a chronic course with the formation of a “cellular lung”. DN II. Prednisolone 50 mg / day was prescribed, taken for 6 months, with a decrease in the dose of prednisolone began to note the deterioration of the state of health: increased dyspnea, cough with sputum smeary, chilliness, discoloration of the distal parts of the hands to red and cyanotic color roughness and low temperatures, a rare puffing with the reception of rough food, general weakness, disability. In February 2017, examined by a rheumatologist, due to the presence of fibrosing alveolitis, Raynaud’s syndrome, sclerodactyly, esophagitis, systemic scleroderma was diagnosed and the patient was transferred to the rheumatology department. Pathogenetic pulse-therapy is prescribed: glucocorticosteroids and cyclophosphamide.

Conclusion: We detected a small percentage of IPL in patients with chronic obstructive pulmonary disease, which is different from the literature data and is probably associated with a low diagnosis of this lesion, due to the fact that not all patients undergo MSCT of the thorax. Conducting MSCT in patients with SSD allows not only to reveal the characteristic symptoms of IPL, but also to estimate the extent of the lesion and the stage of development of the pathological process in the lungs, for the timely treatment of IPL [1]. In this clinical observation, fibrosing alveolitis was the debut of systemic scleroderma, having outstripped the other clinical symptoms of the disease for several years, the course of the alveolitis had a progressive nature, there was a significant decrease in pulmonary volumes and a rise in fibrosis.

LITERATURE

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