

Clinical Cases of the Development of a Nonspecific Course of Pneumonia in Adolescents

Muminov Otamurod Beknazarovich¹, Umirzakov Zakir Baxriddinovich²,
Mamirov Asamiddin Egamberdiyevich²

¹Assistant of the Department of Internal Diseases №4, Samarkand State Medical University

²Assistant of the Department of Epidemiology, Samarkand State Medical University

ABSTRACT

We conducted a literature review of the materials published in recent years on the prevalence and diagnosis of nonspecific interstitial pneumonia. It is noted that nonspecific interstitial lung pneumonia is no longer a rare disease.

KEYWORDS: *interstitial pneumonia, diagnosis, non-standard interstitial pneumonia*

Introduction

Interstitial lung diseases (ISL) are a heterogeneous group of diseases and pathological conditions of known and unknown nature, characterized by widespread, as a rule, bilateral lesions of the respiratory parts of the lungs (alveoli, respiratory bronchioles) [1]. Until recently, there was no effective treatment for patients with advanced pulmonary fibrosis, partly due to limited knowledge about the pathogenesis of this condition. However, over the past decade, new data have emerged on the etiological, genetic factors and pathogenetic mechanisms of ISL. There is an exponential increase in the number of publications on the pathogenesis of ISL, and this is especially true for idiopathic pulmonary fibrosis (ILF) – the most common (20-30% of all cases of ISL) and severe form of ISL. The current stage of studying various forms of ISL is characterized by the emergence of antifibrotic therapy, which remains in the focus of scientists' attention. In the early stages (preclinical and clinical) studies, new developments are being developed in the treatment of ISL based on obtaining data on the links of the pathogenesis of interstitial lung damage. Currently, in all countries of the world, including the Republic of Uzbekistan, there is a high level of prevalence, disability and mortality in interstitial lung diseases, which determines the need to improve the clinical and organizational forms of specialized pulmonological care for patients with this pathology [7,8].

The aim of our work was to identify the necessary criteria in the laboratory diagnosis of nonspecific interstitial pneumonia for adequate treatment.

We conducted a literature review of the materials published in recent years on the prevalence and diagnosis of nonspecific interstitial pneumonia. It is noted that nonspecific interstitial lung pneumonia is no longer a rare disease.

The isolation of NIP as a separate nosological form helped in the identification of a separate group of patients with interstitial lung injuries, but with a more favorable prognosis than with ELISA [1]. The morphological features of NIP differ in general from those of DIP, ELISA, and COP. However, this

nosology is a particular problem for clinicians, since the characteristic clinical signs of patients with the morphological type of NIP in lung biopsies have not yet been clearly identified. In addition, it is important to note that the determination of the histological type of changes in the NIP biopsy material should strengthen the medical search for a possible cause of their occurrence, in particular, the possibility of the primary manifestation of systemic vasculitis or hypersensitive pneumonitis. The histomorphological type of NIP includes a wide range of histological signs with variations in the severity of inflammation or fibrosis in the walls of the alveoli. Interstitial inflammation or fibrosis may predominate in biopsies, and various quantitative combinations of these may be present [2, 3]. In cases of the so-called "cellular subtype" of NIP, weak or moderate interstitial chronic inflammation is observed, represented mainly by lymphocytes and plasma cells (the latter can be quite numerous [4]). The monotonous nature of the lesion of the pulmonary parenchyma is characteristic, but often the distribution of the lesion areas is focal, and the phenomena of chronic inflammation are especially pronounced in the peribronchiolar interstitium. In the areas of inflammation, there are often changes in the type of alveolocyte hyperplasia (pneumocytes) Type II. A typical sign of NIP is the density of foci of inflammatory cell infiltrates — the highest, in comparison with other variants of IIP [12]. Gross fibrosis is not characteristic or absent at all. In approximately 2/3 of NIP cases, intraalveolar organizing fibrosis (focal organizing pneumonia) may be present, but to a much lesser extent more pronounced than in cases of COP. Lymphoid-cell focal clusters are characteristic. Approximately in 40 % of cases of NIP, the same quantitative ratio of foci of inflammation and fibrosis is found, in foci of fibrosis — often there are intraceptal collagen "balls", lymphocytes, plasmocytes and, occasionally, fibroblasts. In such cases, it is very difficult to differentiate morphologically between ObIP and NIP. The main diagnostic feature is the general uniformity of changes in the pulmonary parenchyma and the absence of large areas of changes in the type of "cellular" lung. Fibroblastic foci are rare, never numerous. In approximately 30% of cases, focal accumulation of macrophages can occur, but macrophages are always combined with the presence of lymphoid cells, which serves as a diagnostic difference between this form of IIP and DAP. In about 25% of cases, focal clusters of lymphocytes with the presence of germinal centers (so-called lymphoid cell hyperplasia) can occur, but such formations are scattered in the parenchyma and are not multiple [4]. In the fibrosing subtype of NIP, dense coarse-fibrous or loose interstitial fibrosis of various degrees of severity is determined in combination with homogeneous connective tissue, without

its obvious dynamic changes, in contrast to the morphology of NIP [2]. Fibroblastic foci, one of the key diagnostic signs of OBIP, are not characteristic of this nosology. The phenomena of fibrosis rarely have a focal character, as a rule, it diffusely affects the pulmonary parenchyma, while the alveolar architecture is relatively preserved, although the interalveolar partitions are expanded due to their moderate fibrosis. Occasionally, it is possible to detect foci of rearrangement by the type of "cellular" lung and a weak degree of proliferation of smooth muscle fibers, but this is not a typical phenomenon for NIP. Histological differential diagnosis. The histological cell subtype of NIP should be differentiated from the variants of hypersensitive pneumonitis, organizing pneumonia, linden, diffuse alveolar damage at the stage of its resolution, and eosinophilic pneumonia. The identification of rare, poorly limited granulomas against the background of structural changes in the parenchyma by the type of histological cell subtype of NIP should, first of all, direct the efforts of clinicians to exclude hypersensitive pneumonitis, infection, systemic vasculitis or drug-induced pneumonitis [4, 5]. It should also be taken into account that histological changes in the type of NIP, and both histological subtypes, predominate in patients with diffuse connective tissue pathology, especially in polymyositis/dermatomyositis. Moreover, it was found that in approximately 25% of patients with collagenoses, pulmonary changes of the NIP type occur before the actual systemic manifestation of the pathology [6]. The histological fibrosing subtype must be differentiated with ObIP, variants of pronounced fibrosis in other forms of IIP, and in addition - with histiocytosis and sarcoidosis [5]. The algorithm for managing a patient with NIP is complex. The prognosis of the disease depends on the time before the diagnosis, as well as on the timeliness of the use of effective medications. Thus, despite the negative or insufficiently significant results of many clinical studies, the search for drugs continues in accordance with the growing understanding of the pathogenetic mechanisms of NIP, and recently there has been an "explosion" at the level of preclinical studies in NIP.

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