УДК: 616.24-002.5-06:616.24-018-002.17]-039.3

Features of tuberculosis' course in fibrosing occupational interstitial lung disease

O.M. Raznatovska, O.S. Shalmin, R.M. Yasinskyi, A.V. Fedorec, A.O. Svitlytskyi, O.A. Svitlytska
Zaporizhzhia State Medical and Pharmaceutical University, Zaporizhzhia, Ukraine

Conflict of interest: none

ABSTRACT. Our own observation of the course of tuberculosis in fibrosing occupational interstitial lung disease – ILD (idiopathic fibrosing alveolitis – IFA) in a patient who was being treated at the Zaporizhzhia Regional Phthisiopulmonology Clinical Treatment and Diagnostic Center is presented. The patient had a long work experience (about 7 years) at a dangerous enterprise related to the filling and repair service of powder fire extinguishers. It is possible to determine the following features of the course of tuberculosis in fibrosing professional ILD (IFA) in the presented case: tuberculosis was diagnosed in patient with untreated IFA and progressively increasing respiratory and heart failure, which led to the progression of the tuberculosis process; tuberculosis was initially chemoresistant (RifTB) and was accompanied by fibrinous endobronchitis; dissemination on the X-ray in the lungs had the following character: small multiple foci that merge and cover the pulmonary pattern mainly next to the lungs' roots and basal parts of the lungs. Considering the presence of a long professional route at a dangerous enterprise, increasing shortness of breath, the patient was not given a timely computed tomography of the lungs, which is the most important component of the IFA diagnosis. As a result, IFA was not diagnosed in a time, which led to the lack of necessary IFA treatment (hormonal therapy and cytostatics). A sharp progression of IFA began after the addition of chemoresistant disseminated tuberculosis. Due to this, respiratory and heart failure progressively increased, which became the direct cause of death.

KEY WORDS: tuberculosis, interstitial lung disease.

Особливості перебігу туберкульозу на тлі фіброзивного професійного інтерстиційного захворювання легень

О.М. Разнатовська, О.С. Шальмін, Р.М. Ясінський, А.В. Федорець, А.О. Світлицький, О.А. Світлицька
Запорізький державний медико-фармацевтичний університет, м. Запоріжжя, Україна

Конфлікт інтересів: відсутній

РЕЗЮМЕ. Представлено власне спостереження перебігу туберкульозу на тлі фіброзивного професійного інтерстиційного захворювання легень – ІЗЛ (ідіопатичний фіброзивний альвеоліт – ІФА) в пацієнта, який перебував на лікуванні в Запорізькому регіональному фтизіопульмонологічному клінічному лікувально-діагностичному центрі. Пацієнт мав тривалий стаж роботи (близько 7 років) на небезпечному підприємстві, пов'язаному із заправкою та ремонтним обслуговуванням порошкових вогнегасників. Серед особливостей перебігу туберкульозу на тлі фіброзивного професійного ІЗЛ (ІФА) в представленому випадку можна виділити такі: туберкульоз було діагностовано на тлі задавненого нелікованого ІФА та прогресивно наростальної легенево-серцевої недостатності, що призвело до прогресування самого туберкульозного процесу; туберкульоз був первісно хіміорезистентним (РифТБ) і супроводжувався фібринозним ендобронхітом; дисемінація на рентгенограмі в легенях мала такий характер: переважно в прикореневих ділянках і базальних відділах легень, густа зливного характеру дрібно-вогнищева, перекривала легеневий малюнок. Ураховуючи тривалий професійний маршрут на небезпечному підприємстві, наростання задишки, пацієнту не було своєчасно проведено комп'ютерну томографію легень, що є найважливішим компонентом діагностики ІФА. Як наслідок, ІФА не було своєчасно діагностовано, що призвело до непризначення відповідного лікування ІФА (гормональної терапії та цитостатиків). Різке прогресування ІФА почалося після приєднання хіміорезистентного дисемінованого туберкульозу. На цьому тлі прогресивно наростала легенево-серцева недостатність, яка стала безпосередньою причиною смерті.

КЛЮЧОВІ СЛОВА: туберкульоз, інтерстиційне захворювання легень.

DOI: 10.32902/2663-0338-2024-1-39-43

Interstitial lung diseases (ILD) are a group of diseases that are mostly unrecognized and/or misdiagnosed, leading to misdiagnosis and incorrect treatment [3, 5-7]. Most ILD are characterized by fibrosis of the interstitial component with the development of gas exchange disorder, which leads to shortness of breath, reduced tolerance to physical exertion and, as a result, reduced quality of life [15].

Idiopathic fibrosing alveolitis (IFA) is one of the most widespread with an extremely unfavorable prognosis among ILD, since the average life expectancy of patients from the moment of diagnosis is up to 3.5 years [2]. The authors indicate that IFA is observed mainly in the elderly and senile: for example, if in the age group from 18 to 34 the incidence rate of IFA is 0.4 per 100,000, then among people aged 75 years and older it is 27.1 per 100,000.

At the same time, patients with IFA often have multiple concomitant diseases that can affect survival, but are not diagnosed in a time [8]. Also, the relationship between the number of concomitant diseases and a decrease in survival was also observed in patients who received antifibrotic treatment.

Many researchers indicate that potential risk factors for the IFA development are the influence of environmental factors [2, 4, 10, 14]. According to the literature [1], fibrosing occupational ILD depends on the nature of the occupational lung disease and is a fibrosing lung disease that can lead to the development of progressive pulmonary fibrosis. Respiratory failure and death are a reality for many patients with progressive pulmonary fibrosis [15]. The diagnosis of occupational ILD requires a high level of vigilance and careful anamnesis collection, since the clinical and radiological manifestations of occupational ILD can be unclear, which leads to late diagnosis and inadequate treatment [13].

IFA is manifested by gradually increasing shortness of breath and non-productive cough, and the sharp progression of the disease is associated with viral infection, the development of pneumonia or diffuse alveolar lesion [2].

N. Akhter & N.A. Rizvi [3] proved in their study that ILD is most often misdiagnosed as tuberculosis. In particular, 73 patients were included in the study, among whom 28 (38.35 %) patients with ILD were treated for tuberculosis before reexamination. Therefore, the researchers strongly recommend that primary care physicians, especially in countries with a high burden of tuberculosis, provide in-depth knowledge about ILD in order to limit the use of antimycobacterial therapy (AMBT) when it is not needed and to diagnose ILD in a time.

The data of the research of M.D. Isah et al. show that IFA is often mistakenly diagnosed and treated as pulmonary tuberculosis with an unfavorable outcome [7]. The authors presented a clinical case of their own observation in a 55-year-old textile trader patient who was initially diagnosed with pulmonary tuberculosis with negative sputum test. The patient came with shortness of breath and cough lasting up to 3 years. He started taking AMBT, which did not improve the clinical condition 2 months before that. After additional examination (computed tomography of the thoracic cavity (CT of the chest cavity), lung biopsy and histological examination), IFA was diagnosed in the patient.

V.L. Dias & K.M. Storrer [6] studied the prevalence of latent tuberculosis infection (LTBI) among patients with ILD, requiring immunosuppression. It is known today, this category of patients, due to treatment with immunosuppressants, has a weakened immune system, which may have a higher risk of LTBI transitioning into an active form of tuberculosis. Thus, the authors found that 9.1 % of patients had LTBI, despite the fact that patients with ILD, receiving immunosuppressants are usually not screened for LTBI. Therefore, the authors strongly recommend not to underestimate this factor and to screen for LTBI in these patients.

S.W. Park et al. [11] found that tuberculosis was more frequent in patients with IFA who received immunosuppressants compared to those who did not receive them. The researchers also found that patients with IFA who had mycobacterial infections and received immunosuppressant drugs tuberculosis or mycobacteriosis developed within 1 year of treatment completion, whereas in patients who did not receive immunosuppressant drugs, they occurred later than 2 years after the diagnosis of IFA. At the same time, 18 % of patients with IFA who suffered mycobacterial infections died during follow-up, 3 patients died due to the progression of pulmonary tuberculosis.

X.T. Qi et al. [12] in an experimental study found that the simultaneous use of the drug Pirfenidone with AMBT (isoniazid + rifampicin + pyrazinamide) reduces lung lesion and the development of secondary fibrosis in pulmonary tuberculosis in mice.

M.J. Chung et al. [5] indicate that patients with IFA have an increased risk of pulmonary tuberculosis, as the detection of a specific process can be difficult due to the fibrosis that underlies it. And the incidence of tuberculosis in patients with IFA was 5 times higher than in the general population. At the same time, atypical manifestations of pulmonary tuberculosis on the CT of the chest in patients with IFA can imitate lung cancer or bacterial pneumonia.

Y.H. Lee et al. also indicate the predominance of atypical manifestations of pulmonary tuberculosis on CT of the chest in patients with IFA [9]. At the same time, the authors found that worse treatment results are observed in patients with such manifestations of tuberculosis in IFA.

Thus, a review of the literature shows that there are difficulties in timely diagnosis not only when detecting IFA, but also tuberculosis on the background of IFA, since atypical manifestations of tuberculosis on the background of IFA can imitate other diseases, which leads to late diagnosis and inadequate treatment. The outcome of this can be both the development of progressive pulmonary fibrosis and the progression of the tubercular process, which can lead to death. In the available literature, we did not find a description of the course of tuberculosis against the background of fibrosing professional ILD, which makes this work relevant.

Therefore, **the aim of the work** was to consider the features of the course of tuberculosis in fibrosing professional ILD (IFA) on the example of our own clinical observation in a patient who was being treated at the Zaporizhia Regional Phthisiopulmonology Clinical Treatment and Diagnostic Center (ZRPCTDC).

DOI: 10.32902/2663-0338-2024-1-39-43

Clinical case

Patient C., 41 years old. It is known from the anamnesis that the patient had a long work experience (about 7 years) at a dangerous enterprise related to the filling and repair service of powder fire extinguishers. Since 2015, he has been receiving treatment for chronic laryngitis (hoarseness of the voice) prescribed by an otolaryngologist. The patient did not suffer from tuberculosis before. The last X-ray examination of the organs of the chest cavity (X-ray) in August 2016, but there are no data from this examination. In March 2017, he consulted to a doctor at his place of residence regarding progressive shortness of breath and weight loss. The patient was prescribed symptomatic treatment without X-ray examination, but the general condition only worsened. On 30.05.2017 dissemination in the lungs was detected on the X-ray (fig.), then the patient was referred to a phthisiologist at ZRPCTDC for further examination.

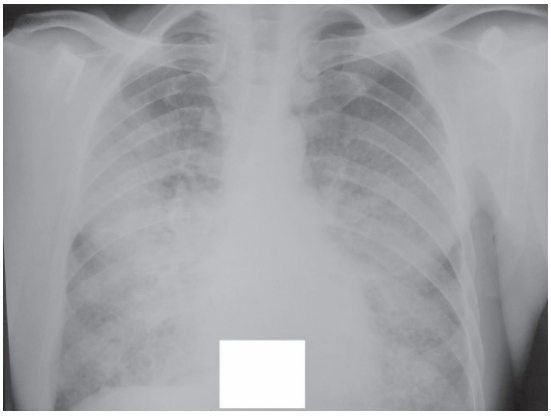

Fig. Chest X-ray from 30.05.2017. There are small multiple foci that merge and cover the pulmonary pattern mainly next to the lungs' roots and basal parts of the lungs on the background of the interstitial component according to the type of "ground glass opacification". The roots are infiltrated

In ZRPCTDC the patient underwent to additional examinations.

Fibrinous endobronchitis was detected during fibrobronchoscopy, and mycobacteria tuberculosis (MBT) resistant to rifampicin (Rif) were detected in bronchoalveolar lavage by molecular genetic method (MG). At the same time, MBT were not detected in the sputum both microscopically (M) and later culturally (K).

Spirography: ventilation failure of the III degree.

Blood test for HIV (rapid test): negative.

Electrocardiogram: the voltage is sufficient, sinus tachycardia, heart rate 105/min, heart electrical axis is not deviated, PQ interval shortening syndrome, signs of right atrial myocardial hypertrophy, diffuse dystrophic changes of the myocardium.

Blood analysis: hemoglobin – 156 g/l, erythrocytes – 4.88×10^{12}/l, leukocytes – 8.2×10^{9}/l, eosinophils – 0 %, band neutrophils – 8 %, segmented neutrophils – 70 %, lymphocytes – 18 %, monocytes – 4 %, erythrocyte sedimentation rate – 18 mm/h.

Urine analysis: specific gravity – 1015, pH – acidic, protein – 0.033 g/l, erythrocytes – 1-2 in the field of vision (v/f), leukocytes – 1-3 in v/f, squamous epithelium – 3-4 in v/f, granular cylinders – 0-1 in v/f.

Biochemical analysis of blood from 12.08.2020: bilirubin – hemolysis, thymol test – 2.8 units, alanine aminotransferase – 0.13 mmol/l/h, aspartate aminotransferase – 1.04 mmol/l/h, total protein – 71.2 g/l, glucose – 4.42 mmol/l, creatinine – 115.7 μmol/l, urea – 2 mmol/l, residual urea nitrogen – 0.93 mmol/l.

Conclusion of the therapist: respiratory failure (RF) of the III degree. Chronic pulmonary heart, decompensation stage. Heart failure (HF) of IIB stage. Cachexia.

Considering the data of the additional examination, the next diagnosed was made: rifampicin-resistant tuberculosis (RifTB) (02.06.2017) disseminated of lungs, destruction-, MBT^{+}, MG^{+}, Rif^{+}, genotypic drug sensitivity test (gDST) 0, M-, K (not ready), phenotypic DST (phDST) 0. Extrapulmonary tuberculosis (ETB) of the intrathoracic lymph nodes (ITLN). Histology 0 (new case of tuberculosis – NTB). ILD: IFA. RF of III degree. Chronic pulmonary heart, decompensation stage. HF of IIB stage. Cachexia.

The patient was hospitalized to ZRPCTDC in serious condition due to severe pulmonary and heart failure. AMBT was prescribed according to DST data, hormonal therapy and symptomatic treatment according to the therapist's recommendations.

Despite the comprehensive therapy, the general condition of the patient worsened due to the development of respiratory and heart failure and on 31.07.2017 the biological death of the patient was determined.

Morphological diagnosis: NTB (02.06.2017). Disseminated pulmonary tuberculosis (phase of progression): multiple bilateral, sometimes confluent, acinous-lobular caseous foci with areas of destructions, $histology^{+}$. ETB of ITLN: large foci of necrosis occupying the entire medullary layer with a single-cell shaft, lymphoid tissue is depleted, with multiple epithelioid-cell granulomas and the presence of Pirogov-Langhans giant multinucleated cells.

Background disease:

1. *IFA:* the walls of the interalveolar septa are thickened with chronic pronounced inflammatory cell infiltration represented by histiolymphocytic elements.
2. Chronic pulmonary heart (thickness of the wall of the right ventricle is 0.7 cm). Bilateral fibrinous pleurisy. *Endogenous intoxication:* focal tubular necrosis of the kidneys, focal centrilobular necrosis of the liver. Parenchymatous dystrophy and venous congestion of internal organs. Cachexia.
3. Chronic erosive-ulcerative gastroduodenitis in the stage of exacerbation. Chronic pancreatitis beyond the stage of exacerbation. Chronic calculous cholecystitis beyond the stage of exacerbation.

Clinical-morphological epicrisis: when comparing clinical and morphological data, it was found: there was a mycobacterial infection with lesion of both lungs and ITLN which was on the background of IFA. In these conditions, respiratory

DOI: 10.32902/2663-0338-2024-1-39-43

and heart failure progressively increased, which became the direct cause of death.

Conclusions

It is possible to determine the following features of the course of tuberculosis in fibrosing professional ILD (IFA) in the presented case:

- tuberculosis was diagnosed in patient with untreated IFA and progressively increasing respiratory and heart failure, which led to the progression of the tuberculosis process;
- tuberculosis was initially chemoresistant (RifTB) and was accompanied by fibrinous endobronchitis;
- dissemination on the X-ray in the lungs had the following character: small multiple foci that merge and cover the pulmonary pattern mainly next to the lungs' roots and basal parts of the lungs.

Considering the presence of a long professional route (about 7 years old) at a dangerous enterprise related to the filling and repair service of powder fire extinguishers, increasing shortness of breath, the patient was not given a timely computed tomography of the lungs, which is the most important component of the IFA diagnosis [1, 2]. As a result, IFA was not diagnosed in a time, which led to the lack of necessary IFA treatment (hormonal therapy and cytostatics). A sharp progression of IFA began after the addition of chemoresistant disseminated tuberculosis. Due to this, respiratory and heart failure progressively increased, which became the direct cause of death.

Література

1. Гаврисюк В.К., Дзюблик Я.О., Меренкова Є.О. та ін. Прогресуючий легеневий фіброз у світлі положень клінічної настанови ATS/ERS/JRS/ALAT 2022 року. *Укр. пульмонол. журнал.* 2022; 30 (4): 51-57. doi: 10.31215/2306-4927-2022-30-4-51-57.
2. Фещенко Ю.І., Гаврисюк В.К., Лещенко С.І. та ін. Ідіопатичний легеневий фіброз: клініка, діагностика, лікування (методичний посібник). – Київ, 2012. – С. 19.
3. Akhter N., Rizvi N.A. Interstitial lung diseases misdiagnosed as tuberculosis. *Pak. J. Med. Sci.* 2018 Mar-Apr; 34 (2): 338-341. doi: 10.12669/pjms.342.
4. Bălă G.P., Râjnoveanu R.M., Tudorache E., Motișan R., Oancea C. Air pollution exposure-the (in)visible risk factor for respiratory diseases. *Environ. Sci. Pollut. Res. Int.* 2021 Apr; 28 (16): 19615-19628. doi: 10.1007/s11356-021-13208-x.
5. Chung M.J., Goo J.M., Im J.G. Pulmonary tuberculosis in patients with idiopathic pulmonary fibrosis. *Eur. J. Radiol.* 2004 Nov; 52 (2): 175-9. doi: 10.1016/j.ejrad.2003.11.017.
6. Dias V.L., Storrer K.M. Prevalence of latent tuberculosis infection among patients with interstitial lung disease requiring immunosuppression. *J. Bras. Pneumol.* 2022 Mar 28; 48 (2): e20210382. doi: 10.36416/1806-3756/e20210382.
7. Isah M.D., Abbas A., Abba A.A., Umar M. Idiopathic pulmonary fibrosis misdiagnosed as sputum-negative pulmonary tuberculosis. *Ann. Afr. Med.* 2016 Oct-Dec; 15 (4): 204-206. doi: 10.4103/1596-3519.194282.
8. Jovanovic D.M., Šterclová M., Mogulkoc N., Lewandowska K., Müller V., Hájková M., et al. Comorbidity burden and survival in patients with idiopathic pulmonary fibrosis: the EMPIRE registry study. *Respir. Res.* 2022 May 27; 23 (1): 135. doi: 10.1186/s12931-022-02033-6.
9. Lee Y.H., Cha S.I., Lim J.K., Yoo S.S., Lee S.Y., Lee J., et al. Clinical and radiological features of pulmonary tuberculosis in patients with idiopathic pulmonary fibrosis. *Respir. Investig.* 2019 Nov; 57 (6): 544-551. doi: 10.1016/j.resinv.2019.08.001.
10. Moss B.J., Ryter S.W., Rosas I.O. Pathogenic mechanisms underlying idiopathic pulmonary fibrosis. *Annu. Rev. Pathol.* 2022 Jan 24; 17: 515-546. doi: 10.1146/annurev-pathol-042320-030240.
11. Park S.W., Song J.W., Shim T.S., Park M.S., Lee H.L., Uh S.T., et al. Mycobacterial pulmonary infections in patients with idiopathic pulmonary fibrosis. *J. Korean Med. Sci.* 2012 Aug; 27 (8): 896-900. doi: 10.3346/jkms.2012.27.8.896.
12. Qi X.T., Zheng L.Y., Fu L., Zhang W.Y., Wang N., Chen X.Y., et al. Protective effect of anti-idiopathic pulmonary fibrosis drug Pirfenidone and Sufenidone (SC1011) on pulmonary injury induced by tuberculosis in a mouse tuberculosis model. *Zhonghua Jie He He Hu Xi Za Zhi.* 2023 Apr 12; 46 (4): 388-395. In Chinese. doi: 10.3760/cma.j.cn112147-20220914-00758.

References

1. Havrysiuk V.K., Dziublyk Ya.O., Merenkova Ye.O., Strafun O.V., Bychenko O.V. Progressive pulmonary fibrosis in light of the ATS/ERS/ JRS/ALAT 2022 clinical guidelines. *Ukrainian Pulmonology Journal.* 2022; 30 (4): 51-57. doi: 10.31215/2306-4927-2022-30-4-51-57.
2. Feshchenko Yu.I., Havrysiuk V.K., Leshchenko S.I., Yachnyk A.I., Lytvynenko G.V., Liskina I.V., et al. Idiopathic pulmonary fibrosis: clinic, diagnosis, treatment (methodical guide). Kyiv, 2012. P. 19.
3. Akhter N., Rizvi N.A. Interstitial lung diseases misdiagnosed as tuberculosis. *Pak. J. Med. Sci.* 2018 Mar-Apr; 34 (2): 338-341. doi: 10.12669/pjms.342.
4. Bălă G.P., Râjnoveanu R.M., Tudorache E., Motișan R., Oancea C. Air pollution exposure-the (in)visible risk factor for respiratory diseases. *Environ. Sci. Pollut. Res. Int.* 2021 Apr; 28 (16): 19615-19628. doi: 10.1007/s11356-021-13208-x.
5. Chung M.J., Goo J.M., Im J.G. Pulmonary tuberculosis in patients with idiopathic pulmonary fibrosis. *Eur. J. Radiol.* 2004 Nov; 52 (2): 175-9. doi: 10.1016/j.ejrad.2003.11.017.
6. Dias V.L., Storrer K.M. Prevalence of latent tuberculosis infection among patients with interstitial lung disease requiring immunosuppression. *J. Bras. Pneumol.* 2022 Mar 28; 48 (2): e20210382. doi: 10.36416/1806-3756/e20210382.
7. Isah M.D., Abbas A., Abba A.A., Umar M. Idiopathic pulmonary fibrosis misdiagnosed as sputum-negative pulmonary tuberculosis. *Ann. Afr. Med.* 2016 Oct-Dec; 15 (4): 204-206. doi: 10.4103/1596-3519.194282.
8. Jovanovic D.M., Šterclová M., Mogulkoc N., Lewandowska K., Müller V., Hájková M., et al. Comorbidity burden and survival in patients with idiopathic pulmonary fibrosis: the EMPIRE registry study. *Respir. Res.* 2022 May 27; 23 (1): 135. doi: 10.1186/s12931-022-02033-6.
9. Lee Y.H., Cha S.I., Lim J.K., Yoo S.S., Lee S.Y., Lee J., et al. Clinical and radiological features of pulmonary tuberculosis in patients with idiopathic pulmonary fibrosis. *Respir. Investig.* 2019 Nov; 57 (6): 544-551. doi: 10.1016/j.resinv.2019.08.001.
10. Moss B.J., Ryter S.W., Rosas I.O. Pathogenic mechanisms underlying idiopathic pulmonary fibrosis. *Annu. Rev. Pathol.* 2022 Jan 24; 17: 515-546. doi: 10.1146/annurev-pathol-042320-030240.
11. Park S.W., Song J.W., Shim T.S., Park M.S., Lee H.L., Uh S.T., et al. Mycobacterial pulmonary infections in patients with idiopathic pulmonary fibrosis. *J. Korean Med. Sci.* 2012 Aug; 27 (8): 896-900. doi: 10.3346/jkms.2012.27.8.896.
12. Qi X.T., Zheng L.Y., Fu L., Zhang W.Y., Wang N., Chen X.Y., et al. Protective effect of anti-idiopathic pulmonary fibrosis drug Pirfenidone and Sufenidone (SC1011) on pulmonary injury induced by tuberculosis in a mouse tuberculosis model. *Zhonghua Jie He He Hu Xi Za Zhi.* 2023 Apr 12; 46 (4): 388-395. In Chinese. doi: 10.3760/cma.j.cn112147-20220914-00758.

DOI: 10.32902/2663-0338-2024-1-39-43

13. Spagnolo P., Ryerson C.J., Guler S., Feary J., Churg A., Fontenot A. P., et al. Occupational interstitial lung diseases. *J. Intern. Med.* 2023 Dec; 294 (6): 798-815. doi: 10.1111/joim.13707.
14. Trethewey S.P., Walters G.I. The role of occupational and environmental exposures in the pathogenesis of idiopathic pulmonary fibrosis: a narrative literature review. *Medicina (Kaunas).* 2018 Dec 10; 54 (6): 108. doi: 10.3390/medicina54060108.
15. Wijsenbeek M., Suzuki A., Maher T.M. Interstitial lung diseases. *Lancet.* 2022 Sep 3; 400 (10354): 769-786. doi: 10.1016/S0140-6736(22)01052-2.

13. Spagnolo P., Ryerson C.J., Guler S., Feary J., Churg A., Fontenot A. P., et al. Occupational interstitial lung diseases. *J. Intern. Med.* 2023 Dec; 294 (6): 798-815. doi: 10.1111/joim.13707.
14. Trethewey S.P., Walters G.I. The role of occupational and environmental exposures in the pathogenesis of idiopathic pulmonary fibrosis: a narrative literature review. *Medicina (Kaunas).* 2018 Dec 10; 54 (6): 108. doi: 10.3390/medicina54060108.
15. Wijsenbeek M., Suzuki A., Maher T.M. Interstitial lung diseases. *Lancet.* 2022 Sep 3; 400 (10354): 769-786. doi: 10.1016/S0140-6736(22)01052-2.

ВІДОМОСТІ ПРО АВТОРІВ / INFORMATION ABOUT AUTHORS

Разнатовська Олена Миколаївна
Завідувачка кафедри фтизіатрії та пульмонології Запорізького державного медико-фармацевтичного університету.
Д-р мед. наук, професор.
26, просп. Маяковського, м. Запоріжжя, 69035, Україна.
ORCID iD: orcid.org/0000-0003-2252-9063

Шальмін Олександр Самуїлович
Професор кафедри фтизіатрії та пульмонології Запорізького державного медико-фармацевтичного університету.
Д-р мед. наук, професор.
26, просп. Маяковського, м. Запоріжжя, 69035, Україна.
ORCID iD: orcid.org/0000-0002-1727-0408

Ясінський Роман Миколайович
Доцент кафедри фтизіатрії та пульмонології Запорізького державного медико-фармацевтичного університету.
Канд. мед. наук.
26, просп. Маяковського, м. Запоріжжя, 69035, Україна.
ORCID iD: orcid.org/0000-0003-4132-731X

Федорець Андрій Васильович
Асистент кафедри фтизіатрії та пульмонології Запорізького державного медико-фармацевтичного університету.
26, просп. Маяковського, м. Запоріжжя, 69035, Україна.
ORCID iD: orcid.org/0000-0003-0994-5265

Світлицький Андрій Олександрович
Доцент кафедри анатомії, гістології, ембріології та топографічної анатомії Запорізького державного медико-фармацевтичного університету.
Канд. мед. наук.
26, просп. Маяковського, м. Запоріжжя, 69035, Україна.
ORCID iD: orcid.org/0000-0001-9603-4501

Світлицька Оксана Анатоліївна
Завідувачка відділення мультимодальної патології Університетської клініки Запорізького державного медико-фармацевтичного університету.
Канд. мед. наук.
26, просп. Маяковського, м. Запоріжжя, 69035, Україна.
ORCID iD: orcid.org/0000-0003-4987-8458

Raznatovska Olena Mykolaivna
Head of the department of phthisiology and pulmonology, Zaporizhzhia state medical and pharmaceutical university.
MD, professor.
26, Mayakovskogo ave., Zaporizhzhia, 69035, Ukraine.
ORCID iD: orcid.org/0000-0003-2252-9063

Shalmin Oleksandr Samuilovych
Professor of the department of phthisiology and pulmonology, Zaporizhzhia state medical and pharmaceutical university.
MD, professor.
26, Mayakovskogo ave., Zaporizhzhia, 69035, Ukraine.
ORCID iD: orcid.org/0000-0002-1727-0408

Yasinskyi Roman Mykolaiovych
Associate professor of the department of phthisiology and pulmonology, Zaporizhzhia state medical and pharmaceutical university.
PhD.
26, Mayakovskogo ave., Zaporizhzhia, 69035, Ukraine.
ORCID iD: orcid.org/0000-0003-4132-731X

Fedorec Andrii Vasyliovych
Assistant of the department of phthisiology and pulmonology, Zaporizhzhia state medical and pharmaceutical university.
26, Mayakovskogo ave., Zaporizhzhia, 69035, Ukraine.
ORCID iD: orcid.org/0000-0003-0994-5265

Svitlytskyi Andrii Oleksandrovych
Associate professor of the department of anatomists, histologists, embryologists and topographic anatomists, Zaporizhzhia state medical and pharmaceutical university.
PhD.
26, Mayakovskogo ave., Zaporizhzhia, 69035, Ukraine.
ORCID iD: orcid.org/0000-0001-9603-4501

Svitlytska Oksana Anatoliivna
Head of the department of multimodal pathology, University Clinic of the Zaporizhzhia State Medical and Pharmaceutical University.
PhD.
26, Mayakovskogo ave., Zaporizhzhia, 69035, Ukraine.
ORCID iD: orcid.org/0000-0003-4987-8458

КОНТАКТНА ІНФОРМАЦІЯ / CORRESPONDENCE TO
Разнатовська Олена Миколаївна
26, просп. Маяковського, м. Запоріжжя, 69035, Україна.
E-mail: raxnatovskaya@gmail.com

DOI: 10.32902/2663-0338-2024-1-39-43