DOI: 10.20514/2226-6704-2025-15-6-461-467
УДК [616.24-002.7-06:616-006.44]-039.42-07
EDN: FNRNFA

Е.С. Рванина, Н.А. Кароли

Федеральное государственное бюджетное образовательное учреждение высшего образования «Саратовский государственный медицинский университет имени В.И. Разумовского» Министерства здравоохранения Российской Федерации, Саратов, Россия

ЛЕГОЧНЫЙ ГИСТИОЦИТОЗ ИЗ КЛЕТОК ЛАНГЕРГАНСА: РЕДКАЯ ПАТОЛОГИЯ В ПРАКТИКЕ ПУЛЬМОНОЛОГА

E.S. Rvanina, N.A. Karoli

Saratov State Medical University named after V.I. Razumovsky, Ministry of Health of the Russian Federation, Saratov, Russia

Pulmonary Langerhans Cell Hysiocytosis: A Rare Pathology in The Practice of a Pulmonologist

Резюме

Легочный гистиоцитоз из клеток Лангерганса — редкое заболевание с коварным началом и неспецифическими проявлениями. В статье рассматриваются два клинических случая пациентов с редкой патологией — легочным гистиоцитозом из клеток Лангерганса. Описаны молодые пациенты, курильщики. У обоих пациентов были типичные рентгенологические признаки: кистозные и узловые образования. Диагноз был верифицирован морфологически. В первом случае на фоне прекращения курения отмечается положительная рентгенологическая динамика, во втором — пациентка не прекратила курить, в связи с выраженностью одышки, изменений при компьютерной томографии легких, был назначен преднизолон. На этом фоне значимой положительной динамики получено не было.

Ключевые слова: *гистиоцитоз Х, редкие болезни, диагностика*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

Источники финансирования

Авторы заявляют об отсутствии финансирования при проведении исследования

Соответствие принципам этики

Пациенты дали согласие на опубликование данных лабораторных и инструментальных исследований в статье «ЛЕГОЧНЫЙ ГИСТИОЦИТОЗ ИЗ КЛЕТОК ЛАНГЕРГАНСА: РЕДКАЯ ПАТОЛОГИЯ В ПРАКТИКЕ ПУЛЬМОНОЛОГА» для журнала «Архивъ внутренней медицины», подписав информированное согласие

Статья получена 18.04.2025 г.
Одобрена рецензентом 19.06.2025 г.
Принята к публикации 26.06.2025 г.

Для цитирования: Рванина Е.С., Кароли Н.А. ЛЕГОЧНЫЙ ГИСТИОЦИТОЗ ИЗ КЛЕТОК ЛАНГЕРГАНСА: РЕДКАЯ ПАТОЛОГИЯ В ПРАКТИКЕ ПУЛЬМОНОЛОГА. Архивъ внутренней медицины. 2025; 15(6): 461-467. DOI: 10.20514/2226-6704-2025-15-6-461-467. EDN: FNRNFA

Abstract

Pulmonary Langerhans cell histiocytosis is a rare disease with insidious onset and nonspecific manifestations. The article discusses two clinical cases of patients with a rare pathology — pulmonary histiocytosis from Langerhans cells. Young patients and smokers are described. The diagnosis was verified morphologically. In the first case, positive X–ray dynamics was noted against the background of smoking cessation, in the second case, the patient did not stop smoking, due to the severity of shortness of breath, changes in computed tomography of the lungs, prednisone was prescribed. Against this background, there was no significant positive trend.

Key words: *Pulmonary Langerhans' cell histiocytosis, rare diseases, diagnosis*

Conflict of interests

The authors declare no conflict of interests

Sources of funding

The authors declare no funding for this study

Conformity with the principles of ethics

The patients consented to the publication of laboratory and instrumental research data in the article « Pulmonary Langerhans Cell Hysiocytosis: A Rare Pathology in The Practice of a Pul-monologist» for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

Article received on 18.04.2025
Reviewer approved 19.06.2025
Accepted for publication on 26.06.2025

For citation: Rvanina E.S., Karoli N.A. Pulmonary Langerhans Cell Hysiocytosis: A Rare Pathology in The Practice of a Pul-monologist. The Russian Archives of Internal Medicine. 2025; 15(6): 461-467. DOI: 10.20514/2226-6704-2025-15-6-461-467. EDN: FNRNFA

CS — corticosteroids, CT — computed tomography, LCH — Langerhans cell histiocytosis, FEV_1 — forced expiratory volume during the first second, CRP — C-reactive protein, FVC — forced vital capacity, EchoCG — echocardiography

Langerhans cell histiocytosis (LCH) is a rare disease causing the clonal proliferation of dendritic cells and macrophages that belong to the mononuclear phagocytic system and affecting multiple organs and systems. The concept of histiocytosis was first proposed by Farber in 1941, and this disease got several names, including eosinophilic granuloma, Letterer-Siwe disease, Hand-Schuller-Christian disease, while Liechtenstein renamed it to histiocytosis X in 1952. As lungs are often involved first, the disease was also called pulmonary Langerhans cell histiocytosis. Pulmonary Langerhans cell histiocytosis is a smoking-associated interstitial lung disease characterized by Langerhans cell proliferation and their infiltration in the lung parenchyma [1]. The clinical LCH course in adults is unpredictable, varying from spontaneous regression to progressive respiratory failure even after smoking cessation [2–5]. LCH usually starts insidiously, without specific manifestations (25 %), or with non-specific findings (pneumothorax as the first symptom was detected in approximately 10–15 % patients).

This article demonstrates two cases of pulmonary Langerhans cell histiocytosis with a variable disease course.

Clinical Case Report No. 1. Male patient S., 33 years old.

Patient S., 33 years old, visited the pulmonologist in the polyclinics in September 2023. The patient had no complaints at the time of examination. History: on July 28, 2023, during the routine chest X-ray, first pulmonary lesions were detected (with a relatively satisfactory health and no complaints); the patient was referred to the pulmonologist. Before 2023, he did not undergo chest X-ray for several years. History: the patient had been smoking 1.5–2 packs daily since the age of 13. He had also been involved in welding works for several years, often contacting with the welding aerosol. The family and allergy histories were negative. The patient denied other concomitant diseases and chronic treatment. His household conditions were satisfactory.

No significant alterations of organs and systems were detected during the physical examination. SpO_2 (on room air) 98 %.

On August 2, 2023, the patient underwent computed tomography (CT) of the chest (Figure 1): multiple, predominantly centrilobular nodules sized 0.3–0.7 cm are detected in all pulmonary fields of both lungs. Conclusion: signs of structural pulmonary lesions (differential diagnosis of tuberculosis, sarcoidosis, Pneumocystis pneumonia). Left-sided pleural adhesions. Lymphadenopathy. The TB specialist counseling was recommended.

The patient S. was counseled by the TB specialist, and a PPD test was arranged. The diagnosis of tuberculosis was excluded. Laboratory tests did not reveal any abnormalities; pulmonary function tests (including spirometry, body pletysmography, DLCO) were normal as well.

On September 2, 2023, the patient underwent videothoracoscopy with the left lung biopsy to clarify the diagnosis in the thoracic surgery department of SHI Regional Clinical Hospital (Saratov). Histology: a fibrotic stellate focus with peribronchial cellular infiltration consisting of histiocytes, plasma cells, lymphocytes with an admixture of eosinophils and pigmented macrophages was detected in the pulmonary tissues (with local irregular cysts). Conclusion: interstitial fibrotic focus based on the described histological signs. No signs suggesting active tuberculosis. Accounting for the cellular infiltrate composition, Langerhans cell histiocytosis X cannot be excluded.

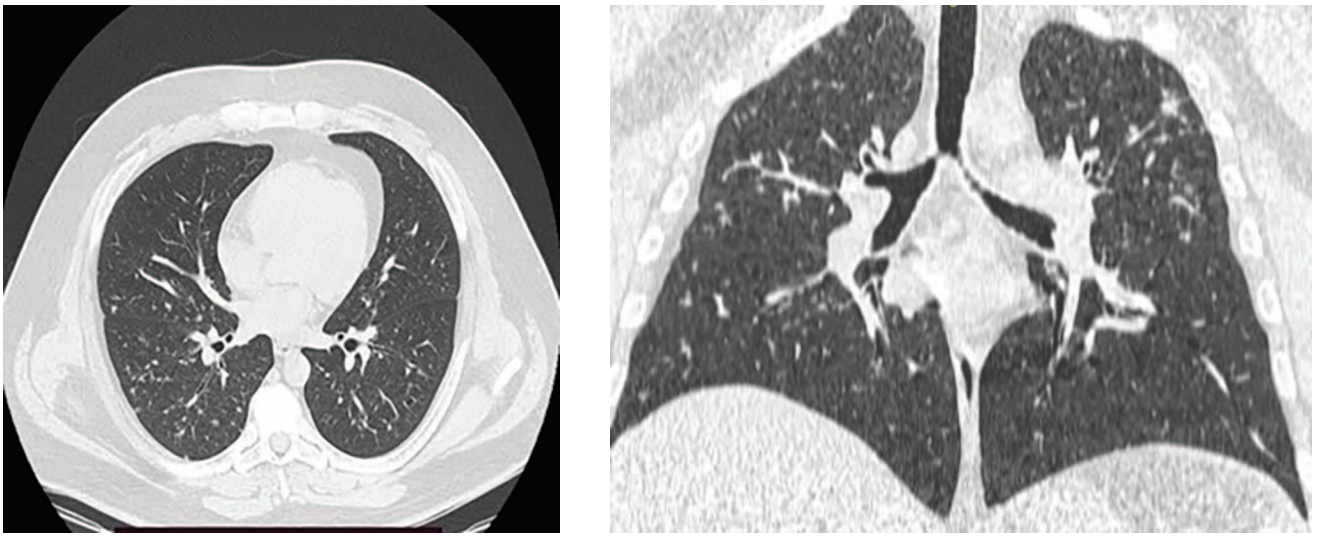
Accounting for the fact that the described CT signs did not correspond to histiocytosis well, it was decided to review the radiological and morphological conclusions. Second radiologist conclusion (acknowledgments to Ya.L. Manakova, SHI Novosibirsk Regional Clinical Hospital): lesions were distributed axially (diffusely, with relatively preserved subpleural areas); craniocaudally (with predominant lesions in upper areas); pulmonary dissemination syndrome: combination of cystic and focal patterns, multiple centrilobular polymorphic foci 0.15–0.65 cm in diameter. Multiple small spherical air-filled cavities with irregularly thick walls. Some cavities were irregularly shaped (clover leaf-shaped, branching).

CT demonstrated signs of pulmonary Langerhans cell histiocytosis.

Pathology blocks were reviewed in the Federal State Budget Institution "Scientific Research Institute of Pulmonology", Federal Medical-Biological Agency of Russia (acknowledgements to M.V. Samsonova, A.L. Chernyaev): pulmonary tissue with thickened interalveolar septa due to lymphoid infiltration, terminal bronchioles with narrowed lumen and significant peribronchiolar infiltration represented by lymphocytes, histiocytes, pigmented brown macrophages, with scarce eosinophils, clear-nucleated cells (Langerhans cells with langerin and CD1a expression); lymphoid aggregates were also observed. Conclusion: histological signs and immunophenotype of Langerhans cell histiocytosis.

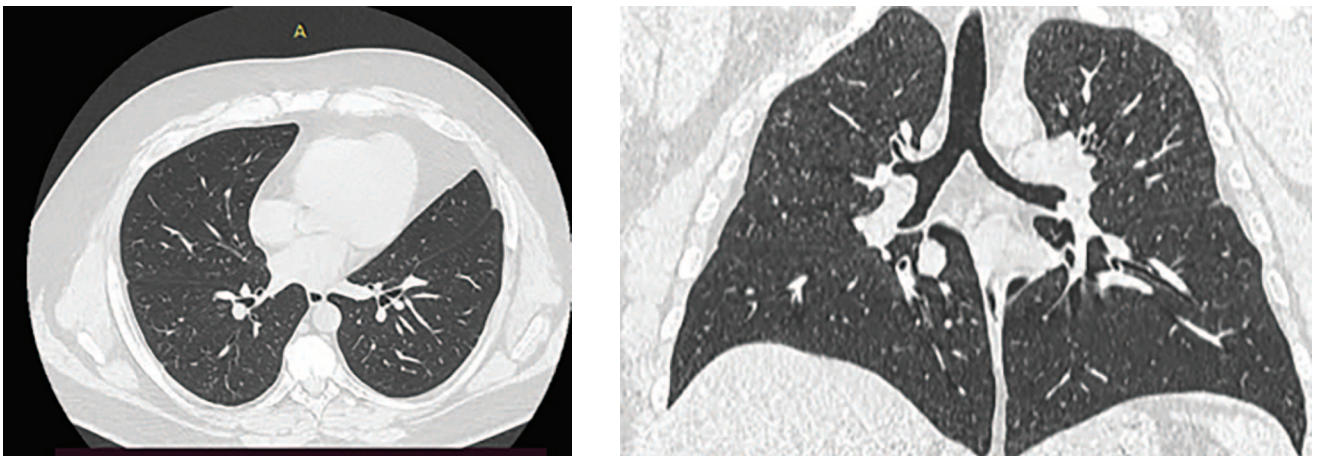
Additional studies (skull X-ray, endocrinologist counseling, EchoCG) did not demonstrate systemic signs of histiocytosis. Thus, the diagnosis of Langerhans cell histiocytosis with pulmonary lesions was established. Regular follow-up and smoking cessation were recommended for the patient. Accounting for the absence of clinical disease manifestations, respiratory dysfunction, no medications were administered.

Follow-up counseling in one year (November 2024): the patient stopped smoking, had no complaints; pulmonary function tests, body pletysmography, DLCO results did not demonstrate negative changes. Follow-up CT of lungs: clearly positive changes — significantly decreased sizes and numbers of focal-cystic pulmonary lesions (Fig. 2).



a) b)

Figure 1. *Computed tomography of the chest (a, b)*



a) b)

Figure 2. *Computed tomography of the chest (a, b)*

Clinical Case Report No. 2. Female patient M., 38 years old.

The patient M. was first admitted to the Pulmonology Department of SHI "Regional Clinical Hospital" (Saratov) on September 2, 2021 complaining of dyspnea of mixed origin with moderate physical exertion, periodic dry cough.

History: she started having the abovementioned complaints since March 2021. She visited the polyclinics at place of residence, and chest X-ray was arranged (pneumofibrosis) (Fig. 3), after which she was referred to the CT of lungs.

Computed tomography demonstrated CT signs of diffuse interstitial changes with ground-glass opacities and multiple air-filled cavities in the pulmonary tissue (Fig. 4).

The patient was referred to the morphological diagnosis verification, i.e. videothoracoscopy with the left lung biopsy. Histology: pulmonary tissue with peribronchial fibrosis, focal emphysema; lumina of several bronchi and their walls contained infiltrates made of large histiocytes and eosinophils; the mediastinal lymph node had a normal histological structure. Conclusion: Pulmonary histiocytosis.

Life history: bad habits: the patients had been smoking 1 pack daily for 13 years; she worked at the household chemical warehouse for 3 years. Prior annual chest X-rays were normal (based on the patient's words).

No significant alterations of organs and systems were detected during the physical examination. SpO_2 (on room air) 97 %. Height 165 cm, body weight 116 kg.

Pulmonary function tests + bronchodilator test (September 3, 2021): restrictive changes cannot be excluded (FEV_1 2.02 L, (66 % of reference values), FVC 2.43 L (69 % of reference values); FEV_1/FVC 83 %). The bronchodilator test was negative. 6-minute test (September 3, 2021): the patient walked 900 m, baseline SpO_2 98 %, after the test — 94 %. Laboratory tests demonstrated elevated CRP levels to 26.8 mg/L (N 0–5 mg/L), elevated glucose levels to 6.9 mmol/L (N 3.9–6.1 mmol/L). Other tests did not reveal any significant alterations. Additional studies (skull X-ray, endocrinologist counseling, EchoCG) did not demonstrate systemic signs of histiocytosis.

Thus, based on CT signs and morphological data, the diagnosis of Langerhans cell histiocytosis with pulmonary lesions was established.

Pathology blocks were reviewed in the Federal State Budget Institution "Scientific Research Institute of Pulmonology", Federal Medical-Biological Agency of Russia (acknowledgements to M.V. Samsonova, A.L. Chernyaev): histological signs and immunophenotype of Langerhans cell histiocytosis.

The patient was consulted online in the GSBSI "Central Scientific Research Institute of Tuberculosis" (Moscow); lung transplantation was to be considered, and systemic corticosteroids (methylprednisolone 8–12 mg daily) were recommended.

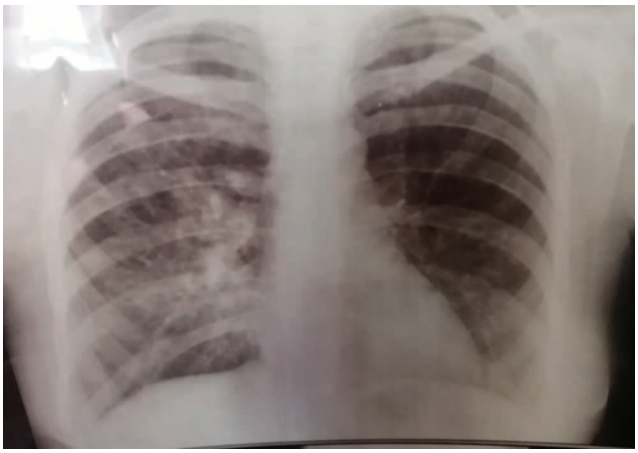
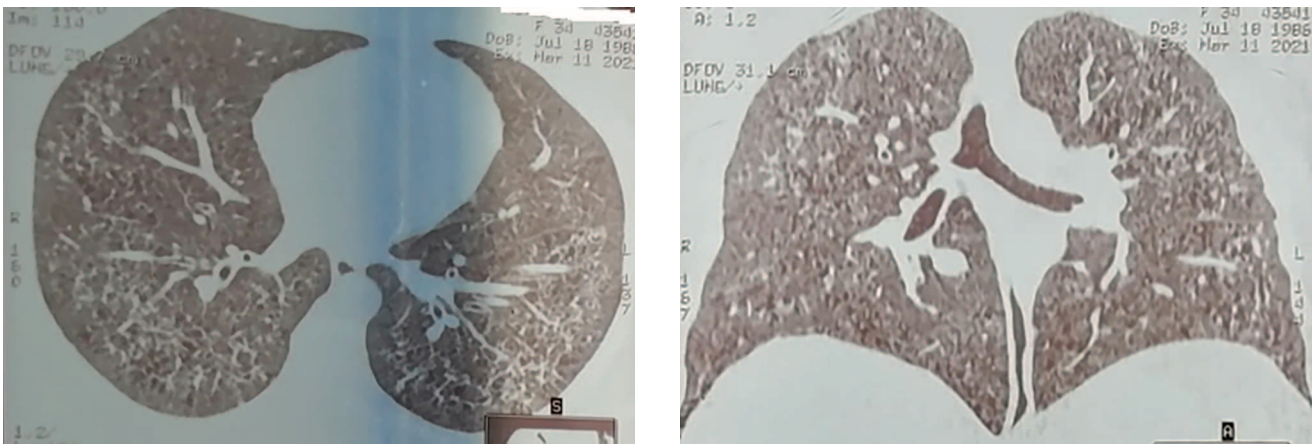
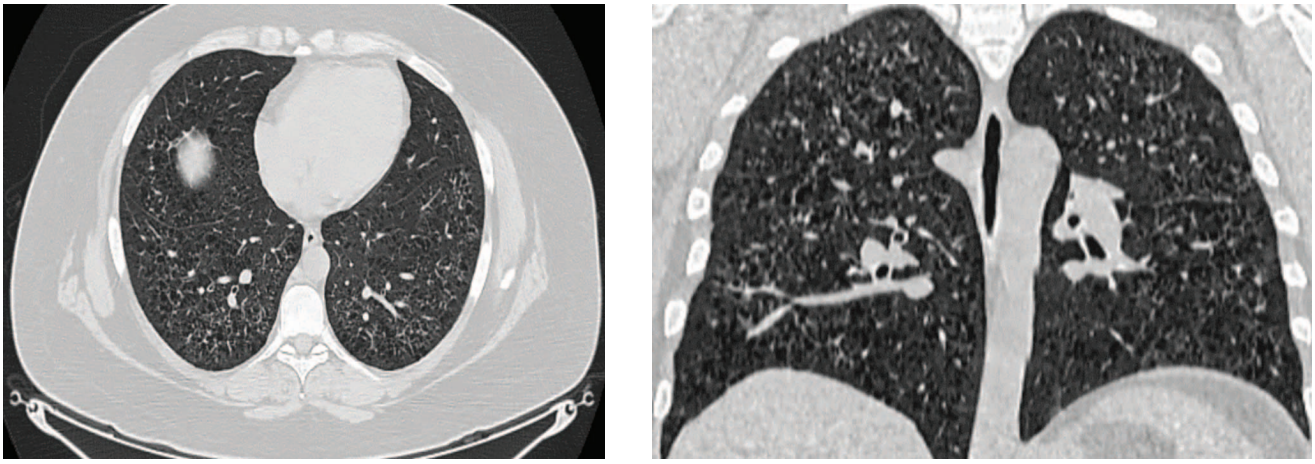


Figure 3. *Chest X-ray*



a) b)

Figure 4. *Computed tomography of the chest (a, b)*



a) b)

Figure 5. *Computed tomography of the chest (a, b)*

Long-term methylprednisolone therapy (8 mg/day) was recommended. The next follow-up was scheduled in 6 months. The patient did not come to the scheduled hospitalization, continuing to take prednisolone 10 mg/day.

In July 2023 she was again hospitalized to the pulmonology department of SHI Regional Clinical Hospital of Saratov. No significant changes concerning the disease severity or signs were reported. Steroid-induced diabetes, Cushing syndrome developed due to long-term prednisolone treatment. CT of lungs (June 19, 2023): significant bilateral emphysema, diffuse reticular lesions, linear streaking without definitive changes (Fig. 5). A consolidated fracture and an osteodestruction focus was detected in the right 8th rib.

Pulmonary function tests: FEV_1 2.16 L, (73 % of reference values), FVC 3.14 L (92 % of reference values); FEV_1/FVC 69 %. The bronchodilator test was negative.

Echocardiography did not demonstrate signs of pulmonary hypertension; no structural or functional alterations were detected.

Laboratory tests: CRP 11.7 mg/L, glucose 6.8 mmol/L, HbA1c 6.01 %.

The patient was recommended to continue prednisolone in the same dose, with the follow-up visit in 6 months. After that, the patient did not come to the follow-up visits.

Discussion

Langerhans cell histiocytoses with isolated pulmonary lesions form a specific group of histiocytoses. LCH occur at any age, mainly in adults (aged 20–40 years), especially in cigarette smokers [5–6]. Currently LCH is considered a smoking-associated excessive immune response in the pulmonary tissue complicated with chronic inflammation, which finally leads to the deposition of Langerhans cells in the interstitial zones of small airways. LCH have been described developing after marijuana smoking, prolonged contact with aroma grass and constant household contacts with fumes [6]. Both active and passive smoking is hazardous — it can also worsen the LCH course [6]. Experimental and clinical studies detected the overexpression of antiapoptotic proteins in dendritic Langerhans cells affected by the tobacco smoke [6], which is possibly one of the main LCH triggers. Both clinical cases presented demonstrate the disease developing in young active smokers.

Priorly LCH was considered a reactive process; however, based on articles from recent years, the clonal disease nature could be proven at least for some patients. Thus, H. Liu et al. detected BRAF V600E expression in LCH cells [6]. A. Roden et al. found the BRAF V600E mutation in 7 (28 %) of 25 examined patients with LCH [7]. Besides, LCH relapses were reported in patients after the lung transplant [6]. Thus, currently LCH is more often considered a myeloid neoplasm with an inflammatory component. On the other hand, in many cases LCH evolution differs from that typical for tumors: the number of Langerhans cells in proliferates gradually decreases, the number of fibroblasts increases, and sclerotic deforming foci develop in lungs.

The incidence of LCH is very small — it constitutes only 4–5 % of all diffuse pulmonary diseases diagnosed using the open lung biopsy [6]. Consequently, LCH can be easily misdiagnosed. It is important that practical physicians are aware of clinical & radiological LCH features.

Patients with LCH usually complain of dyspnea or dry cough (approximately in two-thirds of all such cases) [4]. Constitutional manifestations (asthenia, fever, night sweats, weight loss) may be observed in 10–20 % of these patients. Spontaneous pneumothorax is another common clinical manifestation of the disease (15–20 % cases). It may emerge at any time during the disease and

may be bilateral and/or relapsing; it should be suspected in any patient complaining of worsening dyspnea or chest pain. Hemoptysis is rare and requires the exclusion of other etiologies. Extrapulmonary LCH manifestations usually affect bones (lytic lesions), the pituitary gland (diabetes insipidus), and (less frequently) the skin. Physical examination is usually normal, besides cases of pneumothorax, late-stage LCH, or patients with extrapulmonary lesions. Wheezing is rare, while finger clubbing is very rare [4].

Cystic and nodular shadows in middle or upper lung segments are considered typical radiological signs of LCH: the diagnostic accuracy based on their detection may reach 84 % [4–6], while 10–25 % patients might not have clinical disease manifestations. As the disease progresses, pneumofibrosis worsens, and the cystic remodeling starts affecting all lung segments [4-6].

Pulmonary function disorders are variable and depend on both predominant anatomical lesions and the severity of cystic lesions on computed tomography [4]. Obstructive, restrictive, and mixed patterns have been described. During early stages, pulmonary function tests may be normal in approximately 10 % patients [4]. Decreased DLCO is the most common anomaly, which is observed in 80–90 % cases and primarily reflects the pulmonary vasculature dysfunction. The obstructive pattern of pulmonary disorders is observed in many patients, especially in those with severe cystic lesions. Isolated restrictive lesions are less frequent.

Resting levels of blood gases remain normal for a long time, however physical exertion may caused increased oxygen gradient to alveolar arteries and hypoxemia [4].

Histology and immunohistochemistry of the biopsy specimen is the main diagnostic method of LCH and other histiocytoses [4]. Various methods are used to collect biopsy specimens. The pulmonary tissue for LCH diagnosis can be collected transbronchially, however this method has a low sensitivity. I. Housini et al. demonstrated that the diagnosis was established only 2 (16.6 %) of 12 patients after the transbronchial biopsy [6]. Besides, this method bears the hazard of pneumothorax, which often complicates LCH itself [6]. Bronchoalveolar lavage testing may be helpful, as excessive CD1a-positive cells (>5 %) are often detected in the lavage, although smoking also leads to the increased number of these cells. Due to low sensitivity, bronchoalveolar lavage testing is used only as an auxiliary method [6, 8]. Videothoracoscopy-assisted lung biopsy is the "golden standard" of LCH diagnosis — it can help to obtain sufficient material for the analysis, minimizing the risk of complications [6, 8]. Both patients in our cases underwent the lung biopsy, and the diagnosis of LCH was confirmed morphologically.

The general prognosis for LCH is usually favorable, especially if patients stop smoking early during the disease. Unfavorable prognostic factors include multisystemic lesions, large cysts, significantly decreased DLCO, low FEV_1/FVC ratio, high residual volume to total lung capacity ratio. The first clinical case demonstrates the benign disease course, early diagnosis of pulmonary histiocytosis with positive changes after smoking cessation. The second clinical case represents a less favorable LCH variant: large volume of pulmonary lesions, impaired respiratory function, adverse treatment effects with the preserved risk factor (smoking). This defines a rather unfavorable prognosis for this patient.

Various methods are used in LCH treatment. However, the first recommendation is to stop smoking [4–6, 9]. For example, the study of A. Delobbe et al. demonstrated that the risk of severe respiratory failure in smokers increased more than 10-fold [10]. Just stopping the tobacco smoke inhalation without the drug exposure may lead to improved pulmonary patterns based on computed tomography and ventilation parameters [5]. Stopping contacts with the smoke, smoking (including smoking mixtures and inhalation drugs) is a mandatory prerequisite for efficient LCH treatment.

Besides cigarette smoking cessation, corticosteroids (CSs) are also considered possible treatment methods, especially in patients with significant symptoms and severely impaired respiratory function [5]. However, no standard criteria and approaches exist for CS administration. Cytostatic agents (including chlorodeoxyadenosine, cyclophosphamide, methotrexate) may be considered a possible treatment option for patients with no corticosteroid therapy effects, especially with the involvement of several organs. In our second clinical case systemic CSs were used due to a significant volume of pulmonary lesions, PFT disorders, and respiratory failure (desaturation during physical exertion). One should note adverse effect of prolonged CS treatment (diabetes mellitus, Cushing syndrome) in this patient.

Unilateral pneumothorax is the most common complication of LCH [5]. Based on various data, its rate may range from 16 to 32 % [6]. It was reported that pneumothorax emerged in 16 of 100 patients with LCH, with 10 of those having at least 1 episode of pneumothorax; patients with a complication of pneumothorax usually started LCH in a young age, while the rate of pneumothorax recurrence was approximately 58 % [6]. The rate of pneumothorax recurrences was significantly higher in cigarette smokers than in those who stopped smoking. Pleurodesis is efficient in the setting of recurrent pneumothorax [6]. Lung transplant may be considered for patients with the late-stage disease [4–6]. In this prognostically unfavorable group transplant enhances survival and improves the patient's quality of life. A retrospective study enrolling 39 patients demonstrated the 1- and 10-year survival rates of 76 % and 54 % after the lung transplant, respectively, although recurrences

were especially common in those with other organs affected. In our cases none of the patients developed pneumothorax.

Conclusion

The clinical cases presented demonstrate specific features of this disease: young patient age, smoking as a risk factor, minimum clinical signs (or their absence) with rather significant pulmonary lesions. Chest CT most often demonstrates cystic and focal patterns, multiple centrilobular polymorphic foci of variable diameter. Despite typical radiological symptoms, both cases incorporated the morphological verification of the diagnosis. Pulmonary lesions regressed in the first case with smoking cessation, while the second patient demonstrated progressive pulmonary lesions despite corticosteroid treatment while she continued smoking.

Вклад авторов:

Все авторы внесли существенный вклад в подготовку работы, прочли и одобрили финальную версию статьи перед публикацией

Рванина Е.С.: сбор, анализ данных, интерпретация результатов.

Кароли Н.А.: концепция статьи, анализ, интерпретация данных, написание рукописи, проверка интеллектуального содержания, утверждение рукописи для публикации.

Author Contribution:

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

Rvanina E.S.: data collection, analysis, interpretation of results.

Karoli N.A.: article concept, analysis, data interpretation, manuscript writing, intellectual content verification, manuscript approval for publication.

Список литературы/References:

1. Travis WD, Borok Z, Roum JH, et al. Pulmonary Langerhans cell granulomatosis (histiocytosis X). A clinicopathologic study of 48 cases. Am J Surg Pathol 1993; 17: 971–986
2. Mogulkoc N, Veral A, Bishop PW et al. Pulmonary Langerhans' cell histiocytosis: radiologic resolution following smoking cessation. Chest. 1999; 115: 1452–1455
3. Vassallo R, Ryu JH, Schroeder DR et al. Clinical outcomes of pulmonary Langerhans'-cell histiocytosis in adults. N Engl J Med. 2002; 346: 484–490
4. Lorillon G, Tazi A. How I manage pulmonary Langerhans cell histiocytosis. Eur Respir Rev. 2017; 26: 170070 https://doi.org/10.1183/16000617.0070-2017.
5. Wei P., Lu H.W., Jiang S. et al. Pulmonary Langerhans cell histiocytosis: case series and literature review. Medicine (Baltimore). 2014; 93(23):e141. DOI: 10.1097/MD.0000000000000141
6. Потапенко В.Г., Байков В.В., Зинченко А.В. и др. Гистиоцитоз из клеток лангерганса у взрослых: обзор литературы. Онкогематология. 2022;17(4):16–32. DOI: 10.17650/1818-8346-2022-17-4-16-32
   Potapenko v. G., Baykov v. v., Zinchenko A. et al. Langerhans cell histiocytosis in adults: literature review. Onkogematologiya = Oncohematology 2022;17(4):16–32. [In Russian]. DOI: 10.17650/1818-8346-2022-17-4-16-32
7. Roden A.C., Hu X., Kip S. et al. BRAF V600E expression in Langerhans cell histiocytosis: clinical and immunohistochemical study on 25 pulmonary and 54 extrapulmonary cases. Am J Surg Pathol 2014;38(4):548–51. DOI: 10.1097/PAS.0000000000000129
8. Girschikofsky M., Arico M., Castillo D. et al. Management of adult patients with Langerhans cell histiocytosis: recommendations from an expert panel on behalf of EuroHistioNet. Orphanet J Rare Dis 2013;(8):72. DOI: 10.1186/17501172872
9. Sawalha L., Kumar A., Arshad A. et al. Pulmonary Langerhans cell histiocytosis: radiologic resolution following cessation of second-hand smoking. Clin Respir J. 2017;11(6):1063–7. DOI: 10.1111/ crj.12445
10. Delobbe A., Durieu J., Duhamel A. et al. Determinants of survival in pulmonary Langerhans' cell granulomatosis (histiocytosis X). Groupe d'Etude en Pathologie Interstitielle de la Société de Pathologie Thoracique du Nord. Eur Respir J. 1996;9(10):2002–6. DOI: 10.1183/09031936.96.09102002

Информация об авторах:

Кароли Нина Анатольевна — д.м.н., профессор кафедры госпитальной терапии лечебного факультета ФГБОУ ВО «Саратовский ГМУ им. В.И. Разумовского» Минздрава России, Саратов, e-mail: nina.karoli.73@gmail.com, ORCID ID: https://orcid.ord/0000-0002-7464-826X

Рванина Екатерина Сергеевна — ординатор 1-го обучения, кафедра госпитальной терапии лечебного факультета ФГБОУ ВО «Саратовский ГМУ им. В.И. Разумовского» Минздрава России, Саратов, e-mail: puzanova.ekaterina2016@yandex.ru

Information about the authors

Nina A. Karoli — professor of hospital therapy department of general medicine Department, V.I. Razumovsky Saratov State Medical University of the Ministry of Healthcare of the Russia, Saratov, e-mail: nina.karoli.73@gmail.com, ORCID ID: https://orcid.ord/0000-0002-7464-826X

Ekaterina S. Rvanina — resident of the 1st training, Department of Hospital Therapy, Faculty of Medicine, Federal State Budgetary Educational Institution of Higher Education «Saratov State Medical University named after V.I. Razumovsky» of the Ministry of Health of the Russian Federation, Saratov, e-mail: puzanova.ekaterina2016@yandex.ru

Автор, ответственный за переписку / Corresponding author