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A rare case of antifibrotic therapy in a patient with fibrotic pulmonary sarcoidosis

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Резюме

Sarcoidosis is a systemic inflammatory disease of unknown origin that can progress to fibrocavernous disease. Published data on antifibrotic therapy for fibrotic sarcoidosis are contradictory, largely due to an insufficient number of studies. In randomized trials, nintedanib has demonstrated efficacy in reducing the progression of interstitial lung disease; however, it has not shown a positive effect specifically in fibrotic sarcoidosis. Some individual studies have reported beneficial effects of antifibrotic therapy in cases of fibrotic sarcoidosis. Due to the limited and conflicting data, each observation of antifibrotic therapy in fibrotic sarcoidosis is valuable. **The aim.** Here, we describe our experience of using antifibrotic therapy in a patient with fibrotic pulmonary sarcoidosis. **Methods.** The patient, a 66-year-old woman with sarcoidosis-associated pulmonary fibrosis, has been receiving antifibrotic therapy (nintedanib 150 mg twice daily) since October 2022. Efficacy and safety were assessed before initiation and after 12, 24 and 32 months of antifibrotic therapy. **Results.** The respiratory deterioration slowed down, evidenced by an increased distance in the 6-minute walk test. Pulmonary function decline was also reduced, with no decrease in FEV_1, FVC, FRC, or RV. Computed tomography scans revealed no progression in the volume of pulmonary fibrosis. **Conclusion.** The use of antifibrotic therapy was associated with stabilization of pulmonary function and no progression of pulmonary fibrosis. Further studies on antifibrotic therapy in fibrotic sarcoidosis are warranted.

Key words: fibrotic pulmonary sarcoidosis, progressive pulmonary fibrosis, interstitial lung disease, antifibrotic therapy.

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Редкий случай антифибротической терапии у пациентки с фиброзирующим саркоидозом легких

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Abstract

Саркоидоз – системное воспалительное заболевание неизвестной этиологии, прогрессирующее вплоть до фиброзно-кавернозной формы. Данные по антифибротической терапии при фиброзирующем саркоидозе противоречивы, а число исследований ограничено. По данным рандомизированных исследований продемонстрирована эффективность препарата нинтеданиб в снижении риска прогрессирования интерстициальных заболеваний легких, однако положительного эффекта при фиброзирующем саркоидозе не показано. Отдельные исследования свидетельствуют о возможной эффективности антифибротической терапии. В связи с ограниченностью и противоречивостью данных представляет интерес каждый случай ее применения. **Целью** работы являлась демонстрация клинического наблюдения применения антифибротической терапии у пациентки с фиброзирующим саркоидозом. **Методы.** Пациентка 66 лет с фиброзирующим вариантом саркоидоза легких с октября 2022 г. получала антифибротическую терапию – нинтеданиб 150 мг 2 раза в день. Эффективность и безопасность оценивались до начала лечения и через 12, 24 и 32 мес. применения антифибротической терапии. **Результаты.** Отмечалось снижение дыхательной недостаточности, которое выражалось в увеличении дистанции при выполнении 6-минутного шагового теста. Также отмечено более медленное снижение легочной функции (отсутствие снижения объема форсированного выдоха за 1-ю секунду и форсированной жизненной емкости легких, функциональной остаточной емкости и остаточного объема). По данным компьютерной томографии отмечено отсутствие прогрессирования фиброзных изменений в легочной ткани. **Заключение.** На фоне антифибротической терапии у пациентки замедлилось снижение легочной функции и не прогрессировал легочный фиброз. Необходимо продолжить изучение эффективности антифибротической терапии при фиброзирующем саркоидозе.

Ключевые слова: фиброзирующий саркоидоз, прогрессирующий легочный фиброз, интерстициальное заболевание легких, антифибротическая терапия.

Конфликт интересов. Каждый из авторов заявляет об отсутствии коммерческих или финансовых взаимоотношений (например, консультирования, владения акциями, долевого участия, патентов или лицензий и др.), которые могли бы привести к конфликту интересов в связи с представленной статьей.
Финансирование. Исследование выполнено без финансовой поддержки.
Этическая экспертиза. Исследование проведено в соответствии с принципами Хельсинкской декларации Всемирной медицинской ассоциации. Пациентка подписала информированное добровольное согласие на участие в исследовании.

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Sarcoidosis is a systemic inflammatory disease of unknown origin that can lead to progressive fibrocavernous disease, affecting about 20% of patients [1]. Persistent or recurrent sarcoidosis activity is observed in one third of patients and is referred to by various terms, including severe, refractory, or progressive pulmonary sarcoidosis [2–5]. Patients with long-term disease are at the highest risk of developing fibrosis.

Important immunological and clinical features of fibrosing sarcoidosis have been identified. An antigen initiates the immune cascade by activating interstitial dendritic cells (DCs), alveolar macrophages, and type 2 alveolar epithelial cells (AEC-II). $CD4^+$ T cell activation leads to the release of proinflammatory cytokines, which promote the organization of macrophages into granulomas. Fibrosis begins at the periphery of sarcoid granulomas, initially serving to inhibit further granuloma formation. Later, collagen deposition leads to fibrosis, loss of parenchymal tissue, and ultimately to end-stage sarcoidosis [1]. Activation of interstitial dendritic cells, alveolar macrophages, AEC-II, and $CD4^+$ cells leads to the release of proinflammatory cytokines, promoting granuloma formation. Multiple proinflammatory cytokines are upregulated, including interferon-gamma (IFN-γ) and tumor necrosis factor-alpha (TNF-α). TNF-α levels in alveolar macrophages are higher, and transforming growth factor-beta (TGF-β) levels are lower in patients with progressive disease compared to those with stable sarcoidosis. These findings have sparked interest in TGF-β as an anti-inflammatory cytokine with a potential role in disease resolution [1, 6, 7]. Radiographic patterns of fibrotic sarcoidosis include bronchial distortion, linear fibrosis, and honeycombing. A recent study examining pulmonary sarcoidosis phenotypes using high-resolution CT (HRCT) scans achieved 97% interobserver agreement regarding the existence of distinct phenotypes, with seven HRCT phenotypes categorized as either non-fibrotic or probably fibrotic [8, 9].

Pulmonary function tests often reveal a restrictive pattern but may also indicate airway obstruction [10]. Patients with fibrotic sarcoidosis are at risk of developing complications such as pulmonary hypertension, recurrent infections, and mycetoma formation within fibrocystic regions. There is currently no proven successful treatment for these patients. Most rely on corticosteroids and immunosuppressive drugs, which may increase susceptibility to infections and potentially stimulate further fibrosis progression [10].

The development of algorithms for managing patients with progressive fibrosis is of particular interest. Results from the NBUILD study, which included various forms of progressive fibrosing interstitial lung diseases (ILDs), showed that antifibrotic therapy yielded no positive response in fibrotic sarcoidosis [10]. A systematic review evaluating nintedanib for progressive pulmonary fibrosis found no significant difference in the annual rate of FVC decline between nintedanib and placebo in patients with fibrotic sarcoidosis, in contrast to other fibrosing ILDs [12].

However, another study reported a positive effect of nintedanib on non-idiopathic pulmonary fibrosis, and fibrotic lung diseases, including fibrotic sarcoidosis [13]. The updated *American Thoracic Society* (ATS), *European Respiratory Society* (ERS), *Japanese Respiratory Society* (JRS), *Latin American Thoracic Society* (ALAT) guidelines for idiopathic pulmonary fibrosis (IPF) and progressive pulmonary fibrosis in adults provide a conditional recommendation for the use of nintedanib in cases of progressive pulmonary fibrosis unresponsive to standard treatments for fibrotic ILDs other than IPF [14]. The 2022 federal guidelines mention fibrosing sarcoidosis as a possible outcome of pulmonary disease and emphasize that antifibrotic therapy is considered only in cases of progressive fibrosis and end-stage disease [15]. Further studies are needed to evaluate the efficacy, effectiveness, and adverse events of nintedanib in patients with progressive pulmonary fibrosis due to specific ILDs.

Publications on the use of antifibrotic therapy in fibrotic pulmonary sarcoidosis are highly contradictory, due mainly to the limited number of observations. More studies are needed with longer follow-up and the identification of more reliable biomarkers to predict fibrotic sarcoidosis [16].

The progression of fibrotic pulmonary sarcoidosis is associated with functional impairment and development of pulmonary hypertension. The treatment is complex and may involve anti-inflammatory therapy if granulomatous activity persists, rehabilitation, and – in carefully selected patients – antifibrotic therapy or lung transplantation [17].

Currently, there are no clear recommendations for the management of patients with fibrotic pulmonary sarcoidosis. Reliable biomarkers for predicting fibrosis risk are lacking, and the mechanisms underlying fibrosis in sarcoidosis are not fully understood. The role and effectiveness of antifibrotic therapy in this condition remain undetermined. Therefore, any clinical experience with antifibrotic therapy in patients with fibrotic pulmonary sarcoidosis is of great interest.

The aim of our study was to evaluate the effectiveness and safety of antifibrotic therapy in progressive pulmonary fibrosis secondary to pulmonary sarcoidosis.

Methods

An observational study was conducted in real clinical practice. The patient is a 66-year-old woman with sarcoidosis-associated pulmonary fibrosis. The diagnosis of sarcoidosis was established clinically and radiologically, with histological verification 14 years ago. Periods of disease activity were treated with courses of systemic corticosteroids and cytostatics. Subsequently, progression of lung tissue damage was observed, necessitating permanent administration of systemic corticosteroids. This treatment was complicated by steroid-induced chronic adrenal insufficiency. Over the past five years, the patient's respiratory failure progressed, requiring continuous oxygen therapy.

Antifibrotic therapy with nintedanib (150 mg twice daily) was initiated in October 2022. Efficacy and safety were assessed before treatment initiation and after 12, 24, and 32 months of therapy. Respiratory function was evaluated using spirometry, body plethysmography, lung diffusion testing, and the 6-Minute Walk Test (6MWT). Chest computed tomography (CT) results were also reviewed.

Results

At baseline (2022), the 6MWT was stopped after 2 minutes due to shortness of breath, tachycardia (120 bpm), and decreased oxygen saturation (70%). After 12 months of antifibrotic therapy (2023), the patient was able to walk 100 meters in 6 minutes, with a heart rate of up to 102 bpm and oxygen saturation between 73% and 79%. At 24 and 32 months after starting therapy (2024 and 2025), further improvements were noted: the patient covered 220 and 230 meters, respectively, with heart rates up to 100 bpm and oxygen saturation ranging from 75% to 80%.

Functional study data – including spirometry, body plethysmography, and lung diffusion capacity – are presented in the Table.

As can be seen from the presented data, after 12 months, the functional indicators were practically no different from the initial ones after 12 months. While a positive trend is observed for FEV_1, FVC, FRC, RV (Figure 1) after 24 and 32 months.

The initial chest CT scan (2022) featured, multiple broncho- and bronchioloectasis of the honeycombing, decreased volume of the upper and middle lobes, mediastinal lymphadenopathy, and fibrous changes, mainly in the upper and middle lobes. The fibrous changes remained unaltered, the prevalence of reticular changes and honeycombing did not improve, and lymphadenopathy decreased after 12, 24, and 32 months of the therapy (Figure 2, 3).

Adverse events were observed in the second week of antifibrotic therapy in the form of nausea, diarrhea, decreased appetite, which required a reduction in dosage to 150 mg once a day. Subsequently, these events decreased, and nintedanib was resumed at a dosage of 150 mg 2 times a day. Currently, these side effects occur rarely, and the patient manages them reducing the dosage to 150 mg per day with subsequent increase to the full dosage. In general, drug withdrawal was not required.

Discussion

The introduction of antifibrotic therapy has improved both the quality and duration of life for patients with pulmonary fibrosis. According to the results of a randomized study,

Table
Functional study data
Таблица
Данные функционального исследования

Indicator	Best (2022)	% predicted	Best (2023)	% predicted	Best (2024)	% (2024)	Best (2025)	% (2025)	Dynamics
FEV_1	0.45	18	0.49	20	0.53	24	0.63	29	↑
FVC	0.45	15	0.48	17	0.63	23	0.72	26	↑
FRC, l	0.79	30	0.8	30	0.84	32	1.05	40	↑
TLC, l	1.47	31	1.48	31	1.58	34	1.68	36	↑
VC max, l	0.77	30	0.79	31	0.81	32	0.78	31	–
ERV, l	0.06	8	0.06	8	0.07	10	0.22	33	↑
IC, l	0.69	38	0.7	38	0.74	41	0.63	35	–
RV, l	0.70	36	0.72	37	0.78	41	0.83	43	↑
RV%TLC, %	47	–	48.6	–	48.97	–	49.43	–	↑
DL_{CO}, mmol/min/kPa	2.39	38	2.41	38	2.44	39	1.98	32	↓
DL_{COc}, mmol/min/kPa	2.49	40	2.5	40	2.53	41	2.06	33	↓
VA, l	1.55	35	1.56	35	1.58	36	1.57	36	–
Hb, g/l	128	–	127	–	123	–	123	–	
DL_{CO}/VA, mmol/min/kPA/l	1.54	108	1.54	108	1.60	112	1.31	92	↓

Note: FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; FRC, functional residual capacity; TLC, total lung capacity; VC, volume capacity; ERV, expiratory reserve volume; IC, inspiratory capacity; RV, residual volume; DL_{CO}, diffusing capacity of the lungs for carbon monoxide; VA, alveolar volume; Hb, hemoglobin.

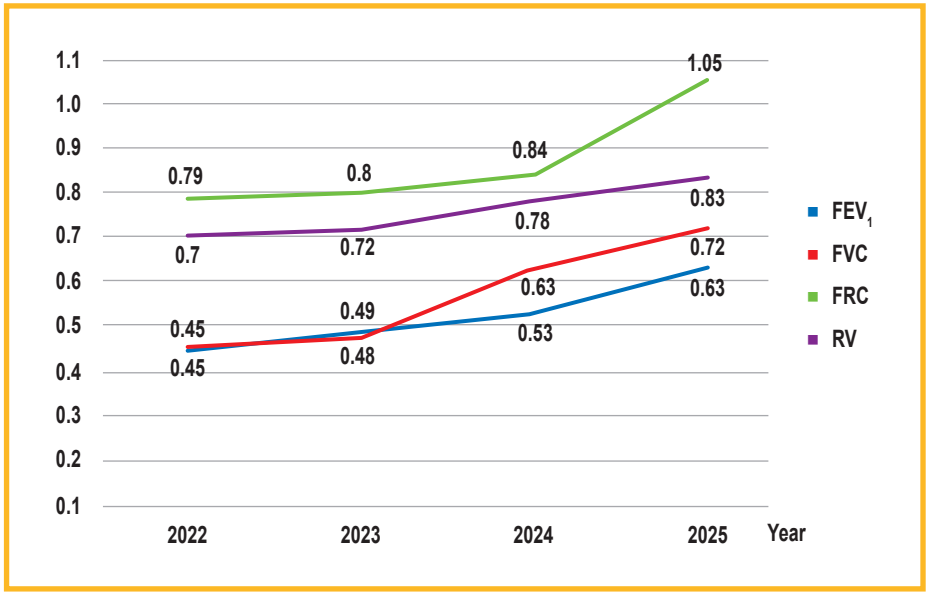

Figure 1. Changes in the functional indicators
Note: FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; FRC, functional residual capacity; RV, residual volume.

Рис. 1. Динамика функциональных показателей

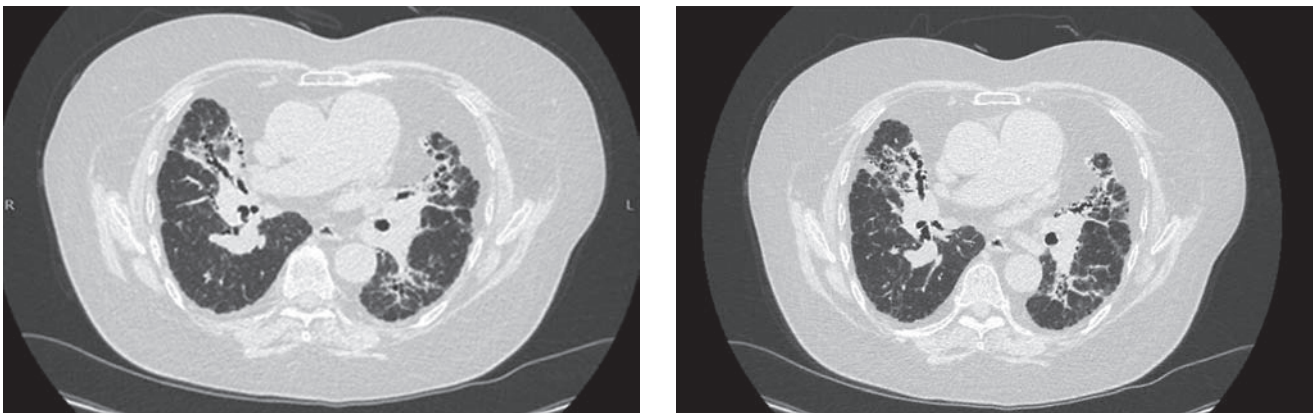

Figure 2. Computer tomography scan of the chest before antifibrotic therapy and after 12 months
Рис. 2. Компьютерная томограмма органов грудной клетки до начала антифибротической терапии и через 12 мес.

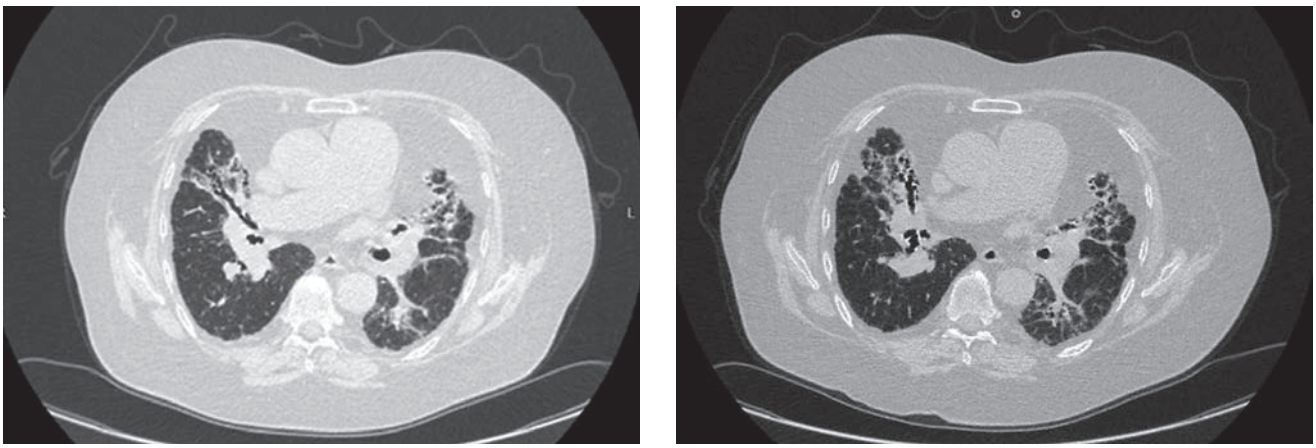

Figure 3. Computer tomography scan of the chest after 24 and 32 months of antifibrotic therapy
Рис. 3. Компьютерная томограмма органов грудной клетки на фоне антифибротической терапии через 24 и 32 мес.

nintedanib demonstrated efficacy in reducing the risk of progression of interstitial lung disease . However, no positive effect was observed with nintedanib in a group of patients with fibrotic sarcoidosis. There are isolated studies indicating a positive effect of antifibrotic therapy in fibrotic pulmonary sarcoidosis.

Due to the lack of data and small sample sizes in these studies, each case of this therapy in patients with fibrotic sarcoidosis is of particular interest.

In our observation, a slowdown in the progression of respiratory disorders was noted, as evidenced by greater in the distance achieved during the 6-Minute Walk Test.

A slower decline in pulmonary function was also observed, confirmed by the absence of decreases in FEV_1, FVC, FRC, and RV. According to CT scans, there was no progression in the volume of pulmonary tissue fibrosis.

However, it is difficult to unambiguously interpret the positive trend in functional pulmonary parameters in the presence of persistent pulmonary fibrosis. Perhaps this is due to improved muscle strength, increased tolerance to physical activity, or easier performance of respiratory maneuvers during functional studies. It should also be noted that the patient experienced side effects during antifibrotic therapy with nintedanib, including nausea, diarrhea, and decreased appetite. These effects decreased with a temporary reduction in the dosage to 150 mg per day and did not require discontinuation of the drug.

Conclusion

This report demonstrates the experience of using antifibrotic therapy with nintedanib in a patient with fibrotic pulmonary sarcoidosis. Antifibrotic therapy with nintedanib was effective and safe in fibrotic sarcoidosis over 32 months of use. During therapy, pulmonary function degradation slowed down and fibrotic changes in the lung tissue did not progress according to computed tomography. Long-term use of antifibrotic therapy was not accompanied by any severe adverse events. Further studies are necessary to determine the feasibility of antifibrotic therapy in fibrotic pulmonary sarcoidosis.

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Serikova A.S. – significant contribution to collection and analysis of the data and interpreting the results, significant contribution to writing the article, editing, approving the final version of the manuscript
Vizel A.A. – idea of the article, significant contribution to the concept of the study, formalizing the task, data analysis, interpreting the results and writing the article, making significant edits, approving the final version of the manuscript

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