World Journal of Pharmaceutical and Life Sciences

Impact Factor: 8.21 www.wjpls.org Coden USA: WJPLA7

DIFFUSE INTERSTITIAL LUNG DISEASE (ILD) AT THE FIBROTIC STAGE: ETIOLOGICAL PROFILE AND EXPERIENCE IN THE RADIOLOGY DEPARTMENT OF THE HUICK HOSPITAL IN CASABLANCA: A STUDY OF 60 CASES

Mahdi Daher*[1], Nasma Morsad[2], Nawal Bouknani[3], Najwa Benslima[4], Amal Rami[5]

Radiology Department of the Cheikh Khalifa International University Hospital – Casablanca.

***Corresponding Author: Mahdi Daher**

Radiology Department of the Cheikh Khalifa International University Hospital – Casablanca.

DOI: https://doi.org/10.5281/zenodo.20444578

How to cite this Article: Mahdi Daher*[1], Nasma Morsad[2], Nawal Bouknani[3], Najwa Benslima[4], Amal Rami[5] (2026). Diffuse Interstitial Lung Disease (Ild) At The Fibrotic Stage: Etiological Profile And Experience In The Radiology Department Of The Huick Hospital In Casablanca: A Study Of 60 Cases. World Journal of Pharmaceutical and Life Sciences, 12(6), 210–216.

Article Received on 05/05/2026 Article Revised on 25/05/2026 Article Published on 01/06/2026

ABSTRACT

Objectives: To describe the CT scan findings of diffuse interstitial lung disease (ILD) at the fibrotic stage observed in our radiology department (HUICK). **Methods:** This was a retrospective study conducted over a 3-year period from January 1, 2021, to December 31, 2023. The diagnosis was made following a multidisciplinary decision-making process (MDD) based on clinical, biological, and radiological findings. **Results:** The mean age of the 40 men and 20 women was 74.7 years (range 51–99 years). The clinical signs were dominated by dyspnea (71.6%) and cough (53.3%). A chest CT scan was performed on all patients and showed honeycombing (97.5%), traction bronchiectasis (95.4%), and interlobular or intralobular septal thickening (91.6%). The clinical and radiological etiological investigation of fibrosing pneumonias revealed an idiopathic form, the most frequent etiology in our series, and a form with a known cause. Pulmonary superinfection was the most frequent complication (68.3%). **Conclusion:** In our study, idiopathic pulmonary fibrosis is the most frequent form of fibrosing interstitial lung disease (ILD). Secondary forms remain a diagnosis of exclusion, hence the importance of CT imaging combined with a multidisciplinary etiological investigation.

KEYWORDS:

INTRODUCTION

Fibrosing involvement in diffuse interstitial lung diseases (ILDs) is rare. Its reference prevalence is currently unknown. In France, the prevalence is estimated at 30.1/100,000 people in the Haute-Garonne department, with an estimated annual incidence of 10.9/100,000 inhabitants.[1] For prognostic and therapeutic purposes, ILDs are classified as progressive or non-progressive fibrosing ILDs.[2] The main ILDs that manifest as progressive fibrosing ILDs are ILDs associated with connective tissue diseases, fibrosing hypersensitivity pneumonitis (FHP), sarcoidosis, and idiopathic pulmonary fibrosis (IPF).[3] The prognosis of fibrosing ILD depends on the extent of fibrosing involvement and the progression of the lesions. The diagnosis is therefore based on a combination of clinical and radiological findings. Chest CT scans play a key role in the diagnosis, classification, and monitoring of diffuse interstitial lung diseases.[4] Histopathology strongly supports the diagnosis. The aim of our study is to identify the main fibrosing interstitial lung diseases diagnosed at the Cheikh Kalifa International University Hospital (HUICK).

Patients and Methods

We conducted a retrospective study in the radiology department of the Cheikh Khalifa International University Hospital (HUICK) in Casablanca over a 3-year period from January 1, 2021 to December 31, 2023. During this period, we identified 60 patients with ILD at the fibrotic stage, collected from the pulmonology department, the internal medicine department, and followed radiologically in the radiology department.

All our patients met the inclusion and exclusion criteria. Patients followed in the pulmonology department who underwent a chest CT scan in the radiology department were included in this study. Patients whose chest CT

scan was performed outside of HUICK and patients followed externally were excluded. We studied the following parameters: age, sex, symptoms, CT scan findings according to etiology, and clinical and radiological evolution. Data were collected from radiology software (PACS and RIS) and DxCare (a DPI software).

The diagnosis was made after a multidisciplinary decision (MDD) based on clinical and radiological findings. A chest CT scan was performed on all our patients, with bone and parenchymal windows, acquiring axial slices with coronal reconstructions. The acquisition was reconstructed using a soft filter and a hard filter. The CT scans were performed on a General Electric (GE) multi-detector scanner with a "high resolution" protocol. The acquisition parameters were as follows: 120 kV, 150 mAs, collimation 1.5 mm, field of view limited to the thorax (16 cm).

RESULTS

During the study period, we identified 60 usable records, comprising 40 men (66.7%) and 20 women (33.3%). Males predominated in our study, with a male-to-female ratio of 2. In our series, the patients' ages ranged from 51 to 99 years, with a mean age of 74.7 years. The most affected age group was 71 to 80 years, representing 41.6% (Fig. 1). Interstitial lung disease manifested with two main clinical signs: dyspnea and cough. These two signs were present in 85% of cases. 43 cases (71.6%) presented with exertional dyspnea, and 32 cases (53.3%) had a dry cough. It should be noted that 9 cases (15%) reported other symptoms such as chest pain.

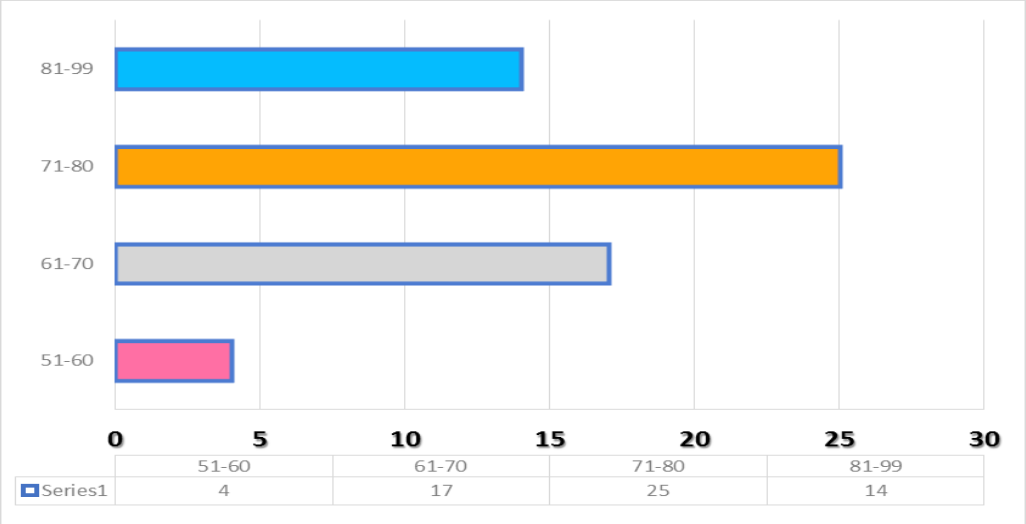

Figure 1: Distribution of patients according to age group.

High-resolution computed tomography (HRCT) of the chest, performed in all patients, demonstrated interstitial lung disease. CT scan findings indicative of interstitial involvement were noted in all cases (Table 1). These included honeycombing in 59 cases, bronchiectasis in 57 cases, intralobular reticulation in 55 cases, and septal and non-septal thickening in 46 and 31 cases, respectively. The distribution of the individual lesions was as follows: peripheral involvement in 23 cases, basal involvement in 19 cases, superior involvement in 5 cases, and middle involvement in 4 cases. Several etiological factors were identified in our series (Fig. 2). Idiopathic pulmonary fibrosis (IPF) was the leading cause of fibrosing lung disease, accounting for 55% of cases. Sarcoidosis was the second most common cause, with a frequency of 16.6%.

Table 1: CT scan appearance of fibrosing ILD.

Signes radiologiques	Nombre de cas	Pourcentage
Rayon de miel	59	98,3%
Bronchectasie de traction	57	95%
Réticulations intra lobulaires	55	91,7%
Épaississements septaux	46	76,7%
Épaississements non septaux	31	51,7%
Micronodule	18	30%
Nodule	13	21,7%
Condensation	9	15%
Verre dépoli	4	6,7%

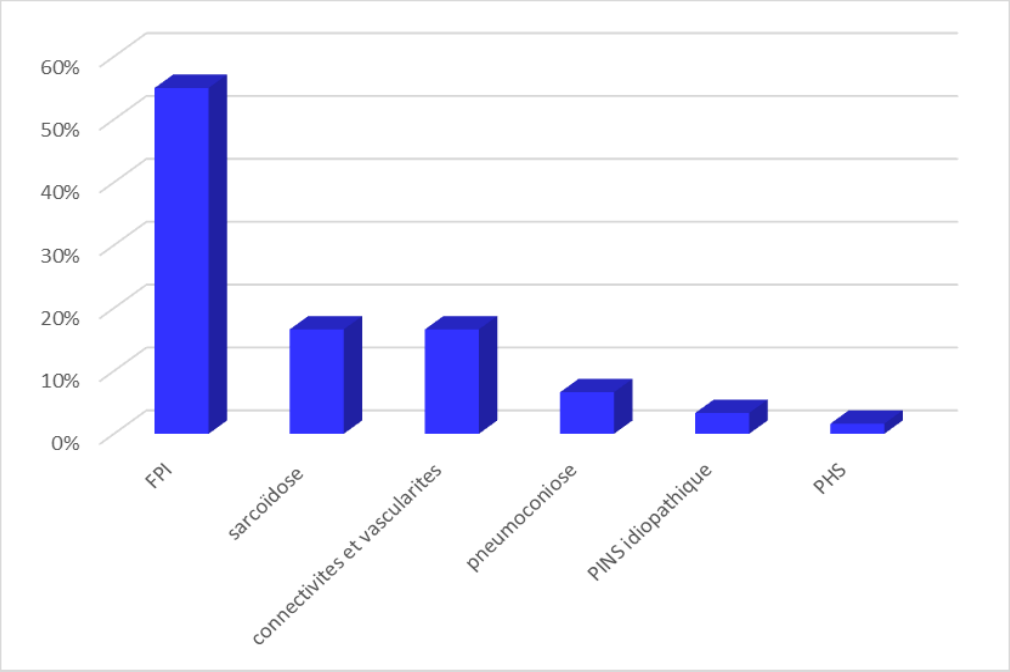

Figure 2: Distribution of patients according to etiological aspects.

The outcome was favorable in all cases except for one death. Analysis of our patients' records revealed that 59 patients experienced at least one complication. Among these complications, we noted 41 cases of exacerbation (68.3%), 11 cases of respiratory failure (18.3%), 5 cases of pulmonary arterial hypertension (PAH) (8.3%), and 2 cases of heart failure (3.3%).

DISCUSSION

Some diffuse interstitial lung diseases, other than IPF, can have a progressive fibrosing course and represent approximately 18 to 32% of cases (Fig. 3).[5] This is a newly described entity that presents a progressive fibrosing phenotype and poses considerable diagnostic challenges in imaging.[6] The most frequent fibrosing forms are ILD associated with sarcoidosis or connective tissue disease and with IPF, with respective prevalences estimated at 30.2, 12.1, and 8.2 cases per 100,000 people.[7]

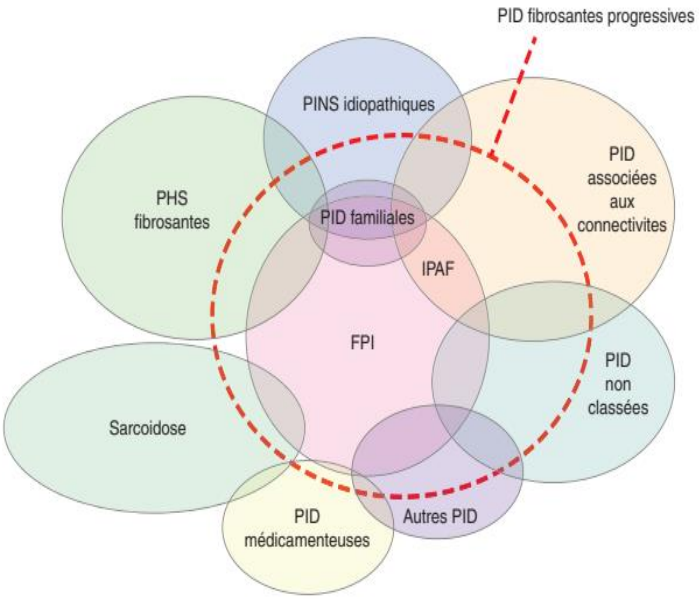

Figure 3. The spectrum of progressive fibrosing ILD.

IPF: idiopathic pulmonary fibrosis; NSIP: nonspecific interstitial pneumonia; IPAF: interstitial pneumonia with autoimmune features; HSP: hypersensitivity pneumonitis

The course of fibrosing interstitial lung disease (ILD) is often difficult to predict, hence the need to assess progression. There is no consensus on the definition of progression. However, progression criteria based on clinical, functional, and computed tomography (CT) findings have been developed (Table 2).[3] Patients with progressive fibrosing ILD have a poorer prognosis than those without progression.[8]

Table 3: Criteria for progressive pulmonary fibrosis

Aggravation clinique	Aggravation fonctionnelle	Aggravation de la fibrose sur la TDM thoracique
Dyspnée Et/ou toux	Diminution sur un an de la capacité vitale forcée (CVF) ≥ 5 % ou de la capacité de diffusion du monoxyde d'azote (DLCO) ≥ 10 %	Majoration de l'extension ou de la sévérité bronchectasies et bronchiolectasies de traction
		Apparition d'opacités en verre dépoli associées à des bronchectasies de traction
		Apparition de fines réticulations intra lobulaires Majoration de l'extension ou de la visibilité des réticulations
		Apparition ou majoration d'images en rayon de miel Majoration de la perte de volume pulmonaire

The diagnosis of fibrosing interstitial lung disease (ILD) is made following a multidisciplinary discussion (MDD) integrating clinical, biological, and radiological data, and confirmed by histological examination of the lung biopsy. Data available in the literature[9] have reported a male predominance, which was also observed in our series. It is also a disease of the elderly, and our series is consistent with this finding, with a mean age of 74.7 years. Clinically, fibrosing ILD manifests as exertional dyspnea and a chronic cough. Thus, the majority of our patients presented with dyspnea and cough, similar to those reported by J.M. Sagne et al. in their series of 61 cases.[10] Imaging, based on chest CT, is a key element in the diagnostic and follow-up assessment of fibrosing lung disease. The semiology of this fibrosing condition essentially involves honeycombing and traction bronchiectasis.[11, 12, 13] For our series, the CT scan data are consistent with those in the literature, with a clear predominance of honeycombing (98.3% of cases) and traction bronchiectasis (95% of cases).

The etiology of fibrosing interstitial lung diseases (ILDs) is numerous and varied, depending on whether it is primary or secondary to an autoimmune disease, occupational exposure, or environmental factors. The main causes of fibrosing ILDs are represented by idiopathic ILDs (IIDs).[1] ILDs are classified as major idiopathic ILDs, rare ILDs, and unclassifiable ILDs.[14] Major idiopathic ILDs include: idiopathic pulmonary fibrositis (IPF) and idiopathic nonspecific interstitial pneumonia (INSP), cryptogenic organizing pneumonia (COP), desquamative interstitial pneumonia (DIP), acute interstitial pneumonia (AIP), and bronchiolitis with diffuse interstitial lung disease (BR-ILD).[15] Rare ILDs, on the other hand, include lymphoid interstitial pneumonia (LIP) and pleuroparenchymal fibroelastosis (PFE). In our study, more than half of the patients presented with ILD, including 33 cases of IPF (Fig. 4) and 2 cases of idiopathic NIPS (Fig. 5). Indeed, IPF is the most frequent form of ILD, as seen in our study.[14, 16, 17] The median age at diagnosis is around 65 to 70 years, with a male predominance, which was also observed in our series. Thus, age and sex constitute two risk factors, as reported by I. Taouil.[18] Moreover, idiopathic NIPS was the second idiopathic form found in our series. Initially and histologically classified as unclassifiable idiopathic ILD, idiopathic NIPS is now considered a distinct clinicopathological entity. This entity remains less frequent than IPF. However, its prognosis with proper treatment is better than that of IPF.[19, 20] The other PII entities were not included in our series due to a lack of available data.

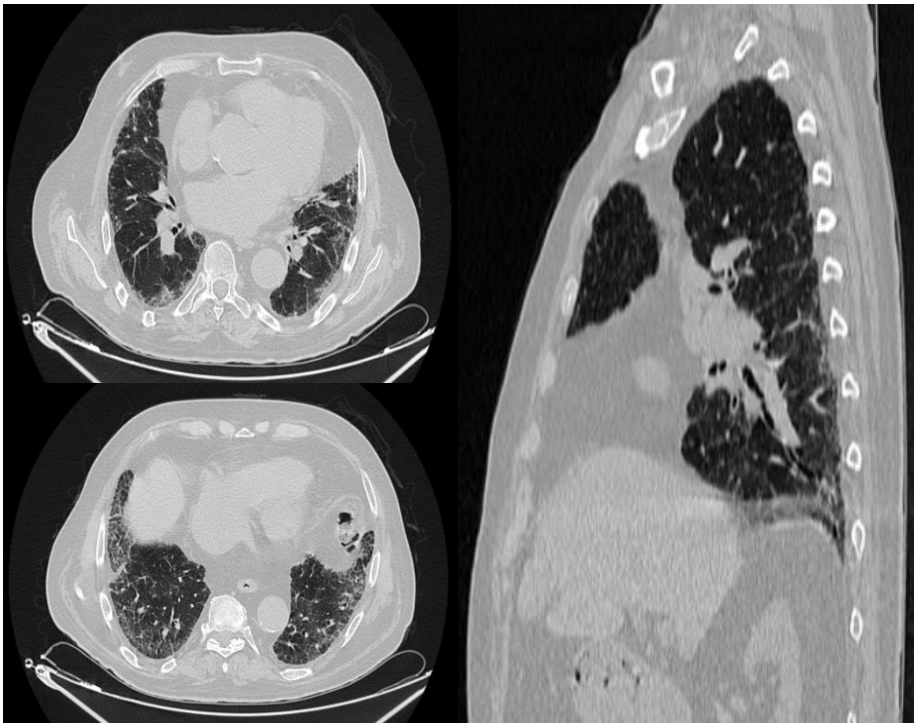

Figure 4: A 74-year-old patient was admitted for respiratory distress. Chest CT scan without contrast, axial and coronal slices; parenchymal window: multiple bilateral septal and non-septal reticulations, predominantly subpleural, associated with a few layers of honeycombing at the lung bases. Peripheral cystic lesions. Diagnosis: IPF.

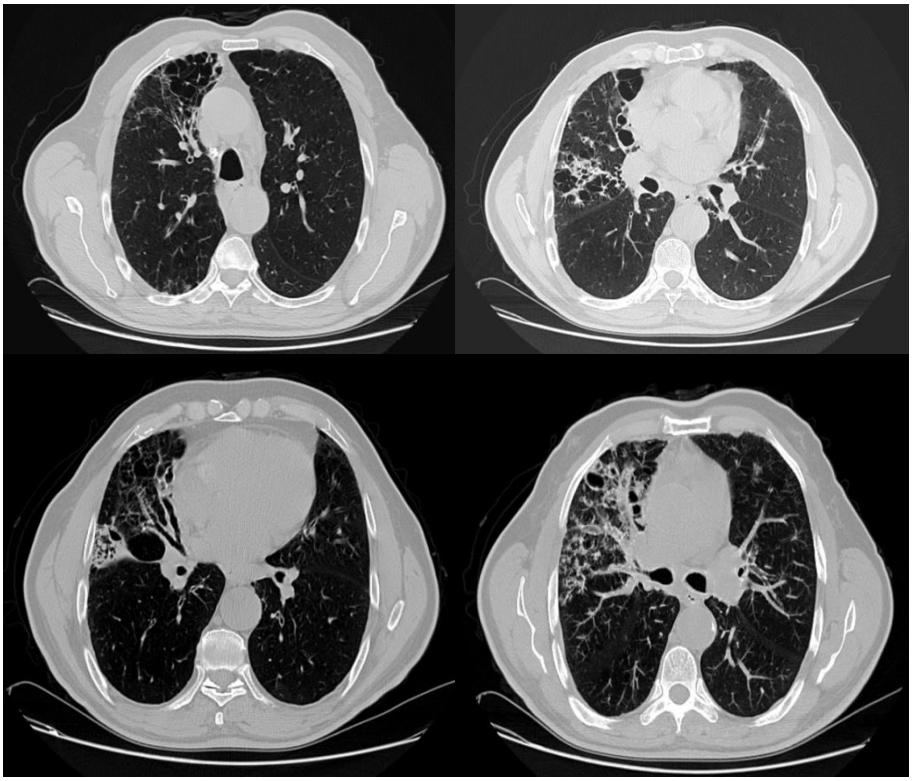

Figure 5. A 56-year-old patient presented with exertional dyspnea. A non-contrast chest CT scan, axial slices; parenchymal window: bilateral apical ground-glass opacities. Septal and non-septal thickening with bronchial distortion, some honeycombing, and traction bronchiectasis predominantly affecting the right middle and lower lobes. Bronchial wall thickening predominantly in the basal lobes with branching micronodules confluent in some areas. Diagnosis: Idiopathic non-insulin-dependent pneumothorax (NIP).

Regarding fibrosing interstitial lung diseases (ILDs) of known cause, the following are included: fibrosing hypersensitivity pneumonitis (FSP), ILDs associated with connective tissue diseases, ILDs associated with granulomatous diseases, and ILDs secondary to occupational exposure. In our series, sarcoidosis was the second most frequent (Fig. 6), after idiopathic pulmonary fibrosis (IPF), and was involved in 16.6% of cases, or ten patients. However, in the literature, sarcoidosis ranks first among diffuse interstitial lung diseases, ahead of connective tissue diseases and idiopathic pulmonary fibrosis.[21] Indeed, it is a systemic granulomatous disease of unknown cause, probably multifactorial, with frequent thoracic involvement (more than 90% of cases). Progression to pulmonary fibrosis accounts for 15 to 20% of cases of sarcoidosis.[22] Finally, the other etiologies found in our series of cases, which could complicate fibrosing involvement[23, 24, 25], were distributed as follows: nine patients with associated connectivity (five cases of rheumatoid arthritis, two cases of Sjögren's syndrome, one case of systemic scleroderma and one case of anti-synthetase syndrome), four cases of pneumoconiosis, one case of Wegener's granulomatosis, one case of fibrosing PHS (Fig. 7, 8, 9).

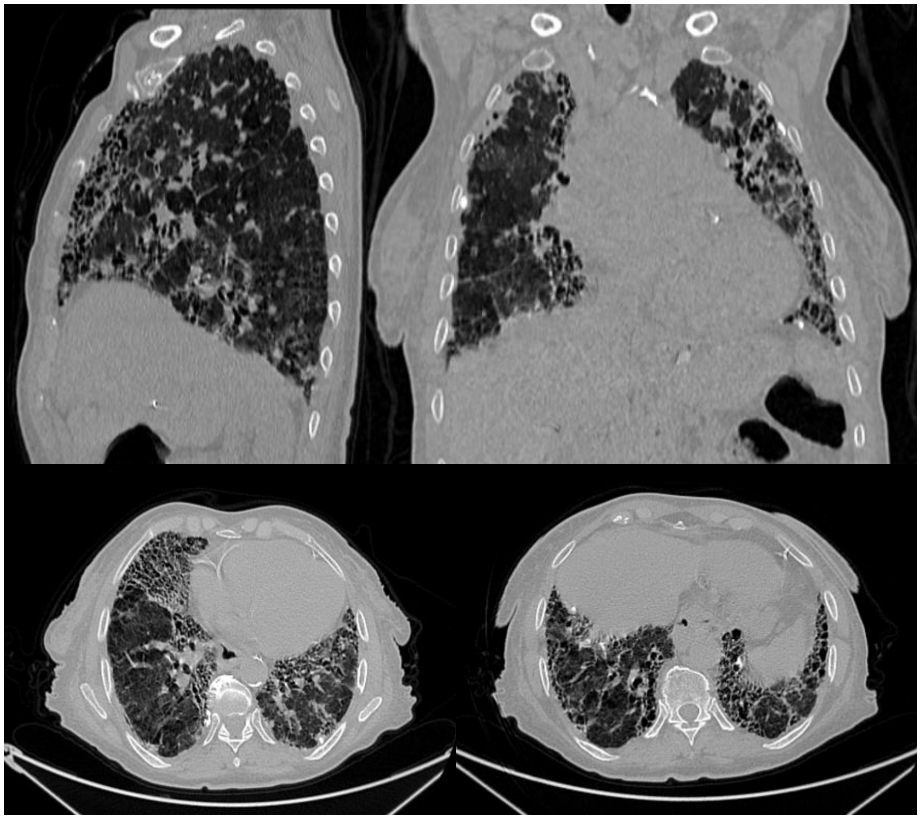

Figure 6: 69-year-old female patient presenting with dyspnea for several weeks. Chest CT scan without contrast, axial, coronal, and sagittal slices; parenchymal window: Diffuse bilateral subpleural septal and non-septal thickening creating a honeycomb pattern. Cylindrical bronchiectasis foci with peribronchovascular thickening. Bilateral ground-glass opacities without a specific distribution.

Regarding complications: an acute exacerbation can occur in patients with progressive pulmonary fibrosis.[26, 27] In our study, this complication affected more than half of the patients, as reported by H. Harraz.[28] The diagnostic criteria for acute exacerbation are the onset of dyspnea in less than one month and the appearance on CT scan of ground-glass opacities and/or superimposed consolidations not explained by another cause (e.g., heart failure).[4]

CONCLUSION

At the end of this study, we observed that IPF is the dominant pathology of ILD at the fibrotic stage. However, secondary forms are not uncommon, hence the importance of CT imaging with a rigorous etiological investigation. Exacerbations of fibrosing ILD represent a poor prognostic factor, particularly in IPF. Regular clinical and radiological follow-up allows for the assessment of the impact of pulmonary fibrosis.

REFERENCES

1. T. Villeneuvea, G. Prévota, F. Lintzb, et al. Epidemiology of fibrosing interstitial lung diseases (ILDs) in Haute-Garonne. Revue des maladies respiratoires, July 2021 ; 38: No. SP. A972–979.
2. V. Cottin, M. Wijsenbeek, M. Kreuter, et al. Non-IPF progressive fibrosing interstitial lung disease (FILD): the patient's journey. Revue des Maladies Respiratoires, Vol. 36, Supplement, January 2019; Pages A136.

3. Carolin Müller, Nicole Mattig, Thomas K. Geisera, Sabina A. Gulera. Progressive fibrosing interstitial lung diseases. Swiss Medical Forum – Forum Médical Suisse 2021;21(11–12):179–183. 4. M. Brauner, H. Ben Romdhane, P.-Y. Brillet, et al. Diagnostic approach to diffuse lung diseases in adults. EMC - Radiology and Medical Imaging - Cardiovascular - Thoracic – Cervical, 2019; 32-362-B-10.
4. M. Hachem, V. Cottin, C. Fournier, and G. Chassagnon. Current status in diffuse interstitial lung diseases. Revue des Maladies Respiratoires Actualités, 2021; 13 : No. 1S2 - pp. 1104-1107.
5. L. Ruez Lantuejoula, E. Reymonda, C. Venota, and G. Ferrettia. 15 minutes to understand progressive pulmonary fibrosis. Journal of Diagnostic and Interventional Imaging, 2023; 6: 289–293.
6. Vincent Cottin, Jean-Christophe Lega, Fabienne Coury and Mouhamad Nasser. Need to improve our knowledge of diffuse interstitial lung diseases associated with connective tissue diseases. Revue du rhumatisme, 2022; 89: 321–325.
7. D. Bouvry. Diffuse fibrosing interstitial lung diseases: diagnostic and therapeutic perspectives. Revue des Maladies Respiratoires Actualités, September 2023; 15(1S2): 1S69-1S73.
8. M. Ijim, L. Aazri, S. Aitbatahar, L. Amro. Fibrosing lung diseases: the epidemiological, clinical, paraclinical, and etiological profile of hospitalized patients in the pulmonology department of Marrakech. Revue des Maladies Respiratoires Actualités, January 2021; 13(1): 188.
9. J.M. Sagne, N. Diatta, Y. Dia. Epidemiological and etiological profile of diffuse fibrosing interstitial lung diseases at the Dalal Jamm Hospital Center in Dakar from November 2021 to August 2023: a report of 61 cases. Revue des Maladies Respiratoires Actualités, January 2024; 16(1): 158.
10. P.-Y. Brillet. New developments in imaging of diffuse fibrosing interstitial lung diseases. Revue des Maladies Respiratoires Actualités, December 2022; 14(2S2): 2S433-2S440.
11. M. Turpin, M. Rémy-Jardin. Diffuse infiltrative lung disease: analysis of the different radiological compartments. Revue des Maladies Respiratoires Actualités, June 2016; 8(2): 80-82.
12. P.-Y. Brillet. Idiopathic pulmonary fibrosis: an update on computed tomography imaging. Revue des Maladies Respiratoires Actualités, September 2015; 7(3): 265-269.
13. R. Diesler, S. Si-Mohamed, F. Thivolet-Bejui, V. Cottin. Classification of idiopathic diffuse interstitial lung diseases. EMC – Pneumologie, 2021, 6-039-K-70.
14. H. Nunes, Y. Uzunhan. Diffuse interstitial lung diseases. Revue des Maladies Respiratoires Actualités, 2014 ; 6(4S1): 80-92.
15. J. Traclet, K. Ahmad, M. Nasser, V. Cottin. Idiopathic pulmonary fibrosis. EMC – Pneumology, 2021; 6-039-K-62.
16. D. Israël-Biet, K. Juvin, K. Dang Tran et al. Idiopathic pulmonary fibrosis: diagnostic and therapeutic management in 2013. Revue de Pneumologie clinique, 2014; 70: 108–117.
17. I. Touila, N. Keskes, S. Bouchareba et al. Prognostic factors in idiopathic pulmonary fibrosis: a study of a Tunisian cohort. Revue des Maladies Respiratoires, 2021; 38: 681–688.
18. M. Brauner, P.-Y. Brillet, N. Naggara et al. Idiopathic diffuse interstitial lung diseases. EMC-Radiology and Medical Imaging - Cardiovascular - Thoracic – Cervical, 2012; 32-464-A-16.
19. Thierry Rochat, Jean-Claude Pache. Idiopathic diffuse interstitial lung diseases: classification and diagnostic approach. Rev Med Suisse, 2010; 6: 2222-7.
20. Florence Jenya, Morgane Didier, Diane Bouvry et al. The lung in sarcoidosis. Revue du rhumatisme monographies, 85: 200–209.
21. M. Cumin, F. Jeny, P. Khagagy. Functional evolutionary profiles of fibrosing pulmonary sarcoidosis. Revue des Maladies Respiratoires Actualités, January 2022; 14(1): 1-284.
22. PY Brillet (1), N Mama (1), H Nunes et al. CT scan semiology of pulmonary involvement in connective tissue diseases. J Radiol, 2009; 90: 1854-68.
23. P. Chauvin, M. Kerjouan, M. Browarski et al. Hypersensitivity pneumonitis. EMC- Pneumologie, 2024; 6-039-E-30.
24. W. Ketata, S. Msaad, H. Gharsalli et al. Pulmonary fibrosis revealing Wegener's granulomatosis. Rev Mal Respir, 2009; 26: 981-4.
25. Lederlin M. Unclassifiable ILD and progressive fibrosing ILD: Dupont P, Martin J, eds. Imaging of diffuse interstitial lung diseases (ILD). Paris: Elsevier Masson, 2023; 117-130.
26. H. Zaibi, K. Chaabi, N. Khalfallah et al. Functional evolutionary profiles of fibrosing pulmonary sarcoidosis. Revue des Maladies Respiratoires Actualités, January 2023; 15(1): 1-288.
27. H. Harraz, Z. Nahid, H. Benllejelloune, N. Yassine. The etiological profile of exacerbations of diffuse infiltrative lung diseases. Revue des Maladies Respiratoires Actualités, January 2021; 13(1): 191.