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Original Article

Histoanatomical changes in pulmonary interstitium in chronic air pollutant exposure- A cross-sectional study.

Amrita Pravi[1*], Neelam Sinha[2], Rashmi Prasad[3]
2nd year PG student, Department of Anatomy, Nalanda Medical College, Patna, India[1]
Professor & HOD, Department of Anatomy, Nalanda Medical College, Patna, India[2]
Professor, Department of Anatomy, Nalanda Medical College, Patna, India[3]

Abstract

Background
Abnormal collagen deposition, interstitial proliferation, inflammatory cell infiltration, and occasionally lung fibrosis are characteristics of a variety of disorders together referred to as interstitial lung disease (ILD).
Objectives- The study's objective is to analyze the histoanatomical changes in the pulmonary interstitium of IPF patients and determine if these changes are related to long-term exposure to occupational and environmental air pollution.

Materials and methods
It was a prospective, observational study. The study was carried out at the Nalanda Medical College and Hospital (NMCH), Patna, Bihar, India. The study was conducted for 12 months. In all, 150 patients were enrolled in the study. Participants had to be at least 40 years old, have been diagnosed with IPF, have had histopathological confirmation of the disease through autopsy, transbronchial lung biopsy, or surgical lung biopsy, have enough lung tissue samples available for microscopic examination, and have given their informed consent for participation and the use of clinical data and tissue specimens for research.

Results
Patients who received a lung transplant did so at an average age of 58.1 ± 7.5 years, whereas the average age at death was 71.4 ± 7.9 years. Out of the 150 patients, 25 (16.7%) had exposure to organic dust. The most frequent cause of these, involving 15 patients (10.0%), was farming-related activities. Four patients (2.7%), two (1.3%), and two (1.3%) were found to have been exposed to wood dust, flour dust, and cotton dust, respectively.

Conclusion
The findings of this study demonstrate that a considerable percentage of individuals with idiopathic pulmonary fibrosis have previously been exposed to organic or inorganic air pollution at work.

Recommendations
As this was a short-term study, further research is needed with a longitudinal study design and a larger sample to achieve more definitive results.

Keywords: *Particulate matter, Interstitial lung disorders, Idiopathic pulmonary fibrosis, Air pollution*
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Corresponding author: *Amrita Pravi**
Email:*dramritapravikmc@gmail.com*
2nd year PG student, Department of Anatomy, Nalanda Medical College, Patna, India

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Introduction

Abnormal collagen deposition, interstitial proliferation, inflammatory cell infiltration, and occasionally lung fibrosis are characteristics of a variety of disorders together referred to as interstitial lung disease (ILD) [1]. Based on their origin and morphologic appearance on biopsy or high-resolution computed tomography (HRCT), ILDs can be divided into several kinds [2].

Environmental exposures such as silicosis, asbestosis, and hypersensitivity pneumonitis (HP) can cause some ILDs. Idiopathic interstitial pneumonia (IIP) is the term used when the cause is unknown. This includes idiopathic pulmonary fibrosis (IPF), idiopathic nonspecific interstitial pneumonia, cryptogenic organizing pneumonia, ILD related to respiratory bronchiolitis, desquamative interstitial pneumonia, lymphoid interstitial pneumonia, acute interstitial pneumonia, and idiopathic pleuroparenchymal fibroelastosis. ILDs can have a variety of causes, but most frequently manifest as progressive pulmonary fibrosis, respiratory failure, and hypoxemia, which can be fatal.

Asthma particles create asbestosis, cotton fiber dust particles generate byssinosis, and silica particles cause silicosis, among other diseases that are induced by inhaling various dusts [3]. Although the exact cause of IPF is uncertain, some environmental risk factors have been identified as predisposing factors, including viral infections, metal or wood dust exposure, and cigarette smoking [4, 5].

One in eight people dies as a result of air pollution worldwide, with outdoor pollution being responsible for half of these deaths [6]. The World Health Organization's (WHO) pollution limit of PM2.5 < 10 µg/m3 is exceeded in the places where almost 92% of the world's population lives [7]. An estimated 69.7 million disability-adjusted life years (DALYs) are lost globally as a result of exposure to outdoor air pollutants [8].

It can be difficult to identify modest interstitial abnormalities, despite their prognostic importance, and in nearly half of the cases, there is a higher chance that they will go unreported [9].

Air pollution has been linked to the onset and aggravation of long-term lung conditions such as asthma and chronic obstructive pulmonary disease (COPD), but little is known about how it affects ILDs. The study's objective is to analyze the histoanatomical changes in the pulmonary interstitium of IPF patients and determine if these changes are related to long-term exposure to occupational and environmental air pollution.

Methodology

Study design

It was a prospective, cross-sectional, observational study.

Study settings

The study was carried out at the Nalanda Medical College and Hospital (NMCH), Patna, Bihar, India. The study was conducted for 12 months.

Study population

In all, 150 patients were enrolled in the study. Participants had to be at least 40 years old, have been diagnosed with IPF, have had histopathological confirmation of the disease through autopsy, transbronchial lung biopsy, or surgical lung biopsy, have enough lung tissue samples available for microscopic examination, and have given their informed consent for participation and the use of clinical data and tissue specimens for research. Patients having a known history of radiation-induced lung injury, patients with current pulmonary infections or cancer at the time of biopsy, and patients with insufficient or non-representative lung biopsy specimens were all excluded.

Data collection

A standardized data sheet was used to gather comprehensive demographic and clinical data, such as gender, age at diagnosis, body mass index (BMI), smoking history, clinical symptoms, lung transplant status, follow-up time, and clinical symptoms. Using a standardized questionnaire and in-depth patient interviews, occupational exposure history was evaluated. Information regarding past or present work settings, exposure duration, and particular interactions with known organic materials was all recorded in the questionnaire.

Study procedure

Depending on the clinical rationale and patient agreement, lung tissue samples were acquired by transbronchial biopsy or surgical lung biopsy. The tissues were sectioned at a thickness of 5 µm, fixed in 10% formalin, embedded in

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paraffin, and stained with Masson's trichrome and hematoxylin and eosin (H&E) stains.
After that, skilled pulmonary pathologists analyzed each sample using light and polarized microscopy to look for histoanatomical alterations like collagen deposition, interstitial thickening, fibrosis, alveolar architectural distortion, and particle infiltration.

Efforts to reduce bias

To reduce interviewer bias and recall, a standardized questionnaire and skilled interviewers were employed. To lessen diagnostic bias, blinded pathologists conducted histopathological evaluations. To prevent selection bias, only verified and well-preserved IPF instances were included. Cross-checking data entry helped to avoid mistakes.

Statistical analysis

Using the single population proportion formula, the sample size was established. The p-value was set at 0.5, and the margin of error was set at 0.05 because no prior comparable research had been conducted in India. The non-response rate was set at 10%. We obtain a sample size of 150 after extrapolating the risk factor assessment studies to a design effect of 1.5.
Data were initially entered in Microsoft Excel. The data has been presented as either the number of participants (n) with percentages (%), or mean with standard deviation (SD).

Ethical considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of NMCH, Patna, Bihar, India.

Informed consent

Written informed consent was obtained from all participants before their enrolment in the study.

Results

182 people in all were first evaluated for possible inclusion based on a clinical suspicion of IPF. 170 of these were evaluated for eligibility by looking over histopathological findings and clinical records. Following the application of inclusion and exclusion criteria, 150 patients were enrolled in the study once their eligibility was verified. All 150 individuals gave their informed consent and had sufficient lung tissue samples on hand for histoanatomic analysis. Following recruitment, no participants were excluded or lost to follow-up.
Lung tissue samples were taken at an average age of 63.9 ± 10.1 years, whereas the mean age at diagnosis for the 150 IPF patients was 63.2 ± 9.8 years. Patients who received a lung transplant did so at an average age of 58.1 ± 7.5 years, whereas the average age at death was 71.4 ± 7.9 years. The follow-up period averaged 61.8 ± 38.6 months. The baseline clinical and exposure profile of patients with idiopathic pulmonary fibrosis is displayed in Table 1.

Table 1. Baseline clinical and exposure profile of patients diagnosed with idiopathic pulmonary fibrosis

Parameter	Value
Average age at diagnosis (years)	63.2 ± 9.8
Age at lung tissue sampling (years)	63.9 ± 10.1
Mean age at death (years)	71.4 ± 7.9
Mean age at transplant (years)	58.1 ± 7.5
Average follow-up duration (months)	61.8 ± 38.6
Male Participants	99 (66.0%)
Female Participants	51 (34.0%)
Active smoker	23 (15.4%)
BMI (kg/m²)	27.6 ± 5.1
Exposed to organic particulate matter	18 (12.0%)
Exposed to industrial or environmental inorganic agents	37 (24.7%)
No identifiable occupational exposure reported	95 (63.3%)

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Out of the 150 patients, 25 (16.7%) had exposure to organic dust. The most frequent cause of these, involving 15 patients (10.0%), was farming-related activities. Four patients (2.7%), two (1.3%), and two (1.3%) were found to have been exposed to wood dust, flour dust, and cotton dust, respectively. Of the patients, 36 (24.0%) had exposure to inorganic, chemical, or diesel-related substances. The most common exposure in this group was asbestos, which affected 13 patients (8.7%), followed by stone dust and metal dust. The distribution of occupational pollutant exposure among IPF patients is shown in Table 2.

Table 2. Distribution of occupational pollutant exposure among IPF patients

Exposure to the Pollutants	Value
Organic Dust Exposure	25 (16.7%)
Farming-related	15 (10.0%)
Cotton dust	4 (2.7%)
Flour dust	2 (1.3%)
Wood dust	2 (1.3%)
Inorganic/Chemical/Diesel Exposure	36 (24.0%)
Asbestos	13 (8.7%)
Metal dust	6 (4.0%)
Stone dust	6 (4.0%)
Glass dust	3 (2.0%)
Cement dust	2 (1.3%)
Diesel exhaust fumes	2 (1.3%)
Sand dust	2 (1.3%)
No Occupational Exposure Identified	89 (59.3%)

Discussion

The primary objective of this study was to analyze histoanatomical changes in the pulmonary interstitium of patients diagnosed with IPF and assess their potential association with long-term occupational and environmental air pollutant exposure. The key findings revealed that a significant proportion (40.7%) of patients had identifiable occupational exposure to organic or inorganic pollutants. Among these, organic dust exposure from farming-related activities was most common, while asbestos constituted the major source of inorganic exposure. Histological examination consistently showed features such as collagen deposition, interstitial thickening, fibrosis, alveolar architectural distortion, and particulate matter (PM) infiltration.

Silica particles with a diameter of less than 1 µm were found in both polarized light microscopy and SEM-EDS analyses of two postmortem samples of individuals who had a history of exposure to ambient dust [10].

These results suggest a potential link between chronic pollutant exposure and the histopathological changes observed in IPF, supporting the hypothesis that occupational and environmental pollutants may contribute to the development or progression of fibrotic interstitial lung diseases. The frequent presence of particulate matter in lung sections, even in patients without classical pneumoconiosis, reinforces the idea that subclinical or low-level exposures may still exert harmful effects on lung parenchyma.

When the samples were screened for PM using polarized light microscopy at a maximum resolution of 0.84 µm and a magnification of 200×, some of the tiny particles were missed. Inorganic dust in polarized light microscopy was linked to histological fibrotic changes in two earlier observational studies of forensic postmortem samples of very young people without known respiratory diseases [11, 12].

A study by Rabeyrin et al. in 2015 reported dust deposits in up to 18% of open lung biopsies from IPF patients, suggesting that such particles are not uncommon in the disease context [13]. Similarly, a study by Lee et al. in 2015 demonstrated a poorer prognosis in IPF patients with a history of dust exposure, especially to wood, metal, and chemical particulates [14].

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Contrary to what was found, a retrospective case-control study of IPF patients who had known exposure to mold or bird dust had a higher survival rate than those who had not, although their survival was lower than that of patients with chronic hypersensitivity pneumonitis [15].

Generalizability

Being a single-centre study, its results may not fully apply to other regions or healthcare settings.

Conclusion

The findings of this study demonstrate that a considerable percentage of individuals with idiopathic pulmonary fibrosis have previously been exposed to organic or inorganic air pollution at work. IPF patients' lungs showed notable histoanatomical alterations after years of exposure to industrial and environmental contaminants, which may indicate a role for these exposures in the development of the disease.

Recommendations

As this was a short-term study, further research is needed with a longitudinal study design and a larger sample to achieve more definitive results.

Limitations

Since this study was conducted in a single urban tertiary care facility, it may not be feasible to extrapolate the findings to the broader population. Additionally, the study's sample size was too small to draw conclusions and extrapolate findings.

List of abbreviations

NMCH- Nalanda Medical College and Hospital
WHO- World Health Organization
IIP- Idiopathic interstitial pneumonia
ILD- Interstitial lung disease
HRCT- High-resolution computed tomography
H&E- Hematoxylin and eosin
SD- Standard Deviation
BMI- Body mass index
COPD- Chronic obstructive pulmonary disease
DALYs- Disability-adjusted life years
HP- Hypersensitivity pneumonitis
PM- Particulate Matter

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Source of funding

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Conflict of interest

The authors declare no conflict of interest.

Author contributions

All authors contributed to the study design, data collection, analysis, and manuscript preparation.

Data availability

The data generated during this study are available from the corresponding author upon reasonable request.

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