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Idiopathic Pulmonary Fibrosis

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Introduction

Idiopathic pulmonary fibrosis is a lung condition that is persistent and progressive. This disorder causes scar tissue (fibrosis) to form in the lungs, preventing the lungs from adequately transporting oxygen into the bloodstream. People between the ages of 50 and 70 are most commonly affected. Idiopathic pulmonary fibrosis is a type of interstitial lung disease (commonly known as ILD), a group of lung disorders characterised by inflammation or scarring in the lungs. These symptoms are not always associated with idiopathic pulmonary fibrosis; not everyone with these symptoms has the disease. Similar signs and symptoms might be caused by other respiratory disorders, some of which are less serious.

Shortness of breath and a persistent dry, hacking cough are the most prevalent indications and symptoms of idiopathic pulmonary fibrosis. Many people who are impacted also lose their appetite and lose weight gradually. Scarring of the lungs worsens over time in persons with idiopathic pulmonary fibrosis, until the lungs can no longer give enough oxygen to the body's organs and tissues. Some persons with idiopathic pulmonary fibrosis develop more serious lung illnesses, such as lung cancer, pulmonary emboli (blood clots in the lungs), pneumonia, or excessive blood pressure in the lungs' blood vessels (pulmonary hypertension). The majority of those who are affected live for 3 to 5 years after being diagnosed. The disease's progress, however, is exceedingly diverse; some people get seriously ill in a matter of months, while others may live with it for a decade or longer. Idiopathic pulmonary fibrosis usually affects only one person in a family [1].

Description

The global prevalence of idiopathic pulmonary fibrosis is estimated to be 13 to 20 per 100,000 people. In the United States, about 100,000 people are affected, with 30,000 to 40,000 new cases identified each year [2]. The random form of pulmonary fibrosis is less common than familial pulmonary fibrosis. Idiopathic pulmonary fibrosis appears to run in families in only a small percentage of instances. Idiopathic pulmonary fibrosis has no known cause. The fibrosis that develops in the lungs is thought to be caused by aberrant tissue repair after tissue damage. A combination of genetic and environmental variables is most likely to blame for this aberrant repair response. Genetic alterations are likely to enhance a person's likelihood of developing idiopathic pulmonary fibrosis and then being exposed to asbestos [3].

Changes in multiple genes have been proposed as risk factors for idiopathic pulmonary fibrosis, although more genetic influences are likely to remain unknown. Only a small percentage of cases of idiopathic pulmonary fibrosis are caused by recognised genetic abnormalities. However, mutations in the TERC and TERT genes have been detected in roughly 15% of all familial pulmonary fibrosis cases and a lower number of sporadic idiopathic pulmonary fibrosis cases. The TERC and TERT genes code for components of the telomerase enzyme, which keeps telomeres, or structures at the ends of chromosomes, in good shape. Telomeres grow abnormally short as cells divide when telomerase action is reduced. Lung cells with shorter telomeres are more likely to cease dividing or die early [4]. Shorter telomeres are linked to a more severe disease and a faster loss in lung function in persons with idiopathic pulmonary fibrosis. To determine how shorter telomeres contribute to the gradual scarring and lung damage seen in idiopathic pulmonary fibrosis, more research is needed.

Environmental risk factors for idiopathic pulmonary fibrosis have also been investigated by researchers. Exposure to wood or metal dust, viral infections, certain drugs, and cigarette smoking are among these causes. According to some study, gastroesophageal reflux disorder (GERD) may be a risk factor for idiopathic pulmonary fibrosis because affected people may breathe in (aspirate) stomach contents, which can harm the lungs over time.

Idiopathic pulmonary fibrosis is a sporadic disease that affects persons who have no family history of the disease. The inheritance pattern for familial pulmonary fibrosis appears to be autosomal dominant. One copy of a mutated gene in each cell is enough to induce the condition in autosomal dominant inheritance. Some persons who inherit the mutated gene, on the other hand, never develop symptoms of familial pulmonary fibrosis. (This is referred to as diminished penetrance.) It is unknown why some persons with a mutant gene develop the disease while others do not [5].

Age: Although pulmonary fibrosis has been detected in youngsters and infants, it is considerably more common in middle-aged and older people.

Sex: Men are more prone than women to develop idiopathic pulmonary fibrosis.

Smoking: Smokers and former smokers are far more likely than non-smokers to acquire pulmonary fibrosis. Emphysema patients can develop pulmonary fibrosis.

Certain professions: If you work in mining, farming, or construction, or if you're exposed to toxins that can harm your lungs, you're more likely to develop pulmonary fibrosis.

Treatments for cancer: The use of some chemotherapy medications or chest radiation treatments can raise your risk of pulmonary fibrosis.

Genetic influences: Some varieties of pulmonary fibrosis are passed down through generations, and genetic factors may play a role

Pulmonary fibrosis complications can include:

In your lungs, you have high blood pressure (pulmonary hypertension). This illness affects only the arteries in your lungs, unlike systemic high blood pressure. It starts when scar tissue compresses the tiniest arteries and capillaries in your lungs, producing greater resistance to blood flow [6].

As a result, the pulmonary arteries and the lower right heart chamber experience increased pressure (right ventricle). Some types of pulmonary hypertension are life-threatening conditions that worsen over time and can be deadly.

Heart failure on the right side (cor pulmonale). When your heart's lower right chamber (ventricle) has to pump harder than usual to transfer blood through partially clogged pulmonary arteries, this dangerous disease occurs.

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Conclusion

Significant progress has been achieved in our knowledge and management of IPF in recent years. However, improving our understanding of the natural history and pathophysiology of IPF is crucial in order to advance our expertise and make meaningful advances in the care of these patients. Furthermore, we must investigate novel imaging and diagnostic tools to improve earlier diagnosis, as well as educate primary care physicians and pulmonologists to refer patients to an interstitial lung disease expert or a lung transplant facility as soon as possible. During the natural history of pulmonary fibrosis pathogenesis, therapeutic methods must target specific aberrant pathways. Only by addressing these difficulties will we be able to improve the prognosis of illnesses linked to progressive lung disease.

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