Pulmonary Fibrosis

Coreena Wells
Otterbein University, coreena.wells@otterbein.edu

Follow this and additional works at: https://digitalcommons.otterbein.edu/stu_msn

Recommended Citation
Wells, Coreena, "Pulmonary Fibrosis" (2016). Nursing Student Class Projects (Formerly MSN). 174.
https://digitalcommons.otterbein.edu/stu_msn/174

This Project is brought to you for free and open access by the Student Research & Creative Work at Digital Commons @ Otterbein. It has been accepted for inclusion in Nursing Student Class Projects (Formerly MSN) by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact digitalcommons07@otterbein.edu.
Pathophysiology of Fibrosis

According to current research, it is clear that there is not just one factor that determines the likelihood of developing an altered healing pathway leading to fibrosis of the lung. Altered responses during the reconstructive phase of healing lead to increased recruitment of inflammatory and fibrotic mediators such as cytokines, chemokines, and growth factors. Much of the literature confirms that these mediators are increased in patients with pulmonary fibrosis (PF). Full-blown injury, lung epithelial cells are turned on to signal TGFB1-induced expression of contractive tissue growth factor (CTGF). CTGF is a pro-fibrotic cytokine that is induced in fibroblasts when an injury occurs, which acts to contribute to the fibrotic matrix proteins and induce further expression of pro-fibrotic cytokines. TGFB1 is part of the extracellular matrix (ECM) (Otterbein & Wynn, 2009). Chronic inflammation exposes one to develop fibrosis and the progressive fibrotic accumulation and recruitment of platelets, degranulation, cells, and leukocytes along with the ECM, contribute to the breakdown of the damaged endothelium from resolution.

Signs & Symptoms

The signs and symptoms of PF are due to the over production of inflammatory mediators and profibrotic scar tissue. These signs and symptoms include, dryness of mouth, dry cough, fatigue, fever, chest pain, weight loss, and aching in muscles and joints (Wilson & Wynn, 2009).

Causes of PF

The injurious agents leading to the chronic inflammation that procures PF may be an environmental chemical, or biological irritant. Sometimes the cause is unknown such as idiopathic pulmonary fibrosis (IPF). IPF is the most common type of PF.

According to Wilson & Wynn (2009) PF 20% lacks MMP-2 and MMP-9, peptides which are required for successful resolution of inflammatory cells. MMP's help determine the amount of collagen deposited during healing. Some research has suggested that the timing of inflammation may be very important in the causation of lung fibrosis as well. On one hand, research has suggested that inflammation may not be a factor in PF as fibrosis can improve without it. What's more, a more-stage inflammation may actually improve the risk of scarring by slowing cell debris and controlling excessive proliferation. This evidence just goes to show how little is known about PF (Wilson & Wynn, 2009). On the other hand, more evidence shows that fibrosis is initiated by the inflammatory response at first but that it is the extracellular matrix effect that continues and determines the final form of the fibrosis. (Wynn & Ramalanan, 2012). This view is more recent and medicates the fact that inflammatory mediators are found to have higher concentrations in PF patients. Also, a known risk factor of PF is chronic infections which stimulate inflammation. Not only do PF patients have excessive inflammatory responses but certain cells in PF patients have actually shown a heightened sensitivity to some very potent pro fibrotic cytokines such as IL-1α and TGF-α. Research to a study on the responsiveness of fibroblasts, these pro-fibrotic cytokines were shown to interact with each other to stimulate a fibrotic response more so than one stimulator by themselves. That is why the limitation of the PF patients is not just one stimulating factor but requires activation of specific pathways to continue collagen production and extracellular matrix formation. Much more widely accepted due to the fact that inflammatory mediators are found in much damage is present, the tissue components in PF patients have actually shown a heightened sensitivity to some very potent pro fibrotic cytokines such as IL-1α and TGF-α. Much more recently as in idiopathic pulmonary fibrosis (IPF), PF, is the more prevalent disease which is the most common type of PF. Healthcare providers must be aware of current research to aid in accurate diagnoses and development the most effective treatment plans.

Nursing Implications

• Nurses must know the pathophysiology in order to educate PF patients regarding their treatments, medications, and changes in lifestyle.
• A more thorough understanding of the possible contributing factors can allow for an early detection of a specific pathway to continue collagen production and extracellular matrix formation. The development of a new TGFβ receptor pathway has led to the development of new therapies and may reduce the progression of lung fibrosis.
• Initial injury can be caused by, environmental, or biological irritants. Depending on the injury and how acute the injury was, determines the type of outcome. For example, this is a return to normal function. If the damage was more severe, resolution is an acceptable outcome, this is a return to normal function. During repair, scar tissue replaces destroyed tissue. The result of this happens with extensive damage and results in tissue that is much less than it was before the injury. This replacement of lung tissue with less tissue causes lung stiffness and ventilation difficulty. This, in turn, results in decreased gas exchange and hypoxemia.

References

Coreena Wells RN, BSN
Otterbein University, Westerville, Ohio

Pulmonary Fibrosis

The purpose of this poster is to discuss the pathophysiology of pulmonary fibrosis. More specifically the role of the inflammatory and immune systems thought to be responsible for stimulating fibrosis in the lungs will be discussed. Pulmonary fibrosis (PF) is a chronic disease of the lungs involving an altered inflammatory response to injury or infection which results in scarring of the lungs. The lungs are then unable to ventilate or oxygenate the blood. This is largely due to the scar tissue. PF has not been thoroughly studied compared with many of the more prevalent diseases today. PF affects men more often than women but has seen a rise in both genders since 2000. According to Ley & Collard (2013) the lack of research in PF is partly due to the existence of similar conditions such as COPD, other interstitial lung diseases, viral infections, etc. and the unclear definition of PF in the past.

Pathophysiology

Normal lung healing takes place in phases, following the initial injury a period of healing that begins during the acute inflammation and continues until tissue is completely repaired. Depending on the injury and how much damage is present, the tissue heals with or without some degree of scarring to normal function. If the damage was more severe, resolution is an acceptable outcome, this is a return to normal function. During repair, scar tissue replaces destroyed tissue. The result of this happens with extensive damage and results in tissue that is much less than it was before the injury. This replacement of lung tissue with less tissue causes lung stiffness and ventilation difficulty. This, in turn, results in decreased gas exchange and hypoxemia.


determining the treatment plan for patients with PF. Healthcare providers must be aware of current research to aid in accurate diagnoses and development of the most effective treatment plans.