The Pathophysiology of Alpha-1 Antitrypsin Deficiency and COPD

Melissa M. Miller

Otterbein University, melissa.miller@otterbein.edu

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

Part of the Nursing Commons

Recommended Citation
Miller, Melissa M., "The Pathophysiology of Alpha-1 Antitrypsin Deficiency and COPD" (2016). Nursing Student Class Projects (Formerly MSN). 181.
https://digitalcommons.otterbein.edu/stu_msn/181

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.
The Patioplogy of Alpha-1 Antitrypsin Disease and COPD

Melissa M. Miller, BSN, RN
Otterbein University, Westerville, Ohio

The Genetic Variants of Alpha-1 Antitrypsin Deficiency

Alpha-1 antitrypsin (AAT) deficiency is a disorder present in 1 in 2000 people and it is the most common monogenic disorder in the population. AAT is a protease inhibitor encoded by the SERPINA1 gene on chromosome 14. Alpha-1 antitrypsin is synthesized in the liver and secreted into the blood. It travels to the lungs where it diffuses into the interstitium and the alveolar fluid living. Here it inactivates neutrophil elastase, helping to protect the lungs from protease-mediated damage (Brode, Ling, & Chapman, 2012). Signs and Symptoms of Alpha-1 Antitrypsin Deficiency with COPD

The symptoms associated with AAT deficiency include: obstructive pulmonary disease, respiratory infections, and liver disease (Koepke et al., 2013). The most damaging of these mutations is the Z-1 antitrypsin deficiency (Koepke et al., 2013). There is a need for education of healthcare providers regarding the necessity of detecting alpha-AAT deficiency in patients with COPD. AATD patients, asthma, and bronchiectasis have been reported. The reports of asthma thought to be misdiagnosed. Possibly because of the age of the patient or a non-smoking history. The symptoms associated with this disease process can be a problem in patients without the AATD component. Patients present with a cough, wheezing, dyspnea, and excessive sputum production. They may also have a lower oxygen saturation. Another sign is frequent exacerbations and lung infections. The diagnosis of AATD is usually identified after the patient has COPD. Generally, the Pulmonologist will order a blood test to see if the patient has the genetic disorder, especially if they are exhibiting symptoms at a younger age than what is typical for a COPD patient (Strange, 2013). Treatment

Pneumonia and many primary care providers seem to lack the basic education to providers, nurses, and respiratory therapists regarding the importance of education of healthcare providers in identifying this disease. The role of the Pulmonary Advanced Practice Nurse (APN) at the VA is to educate staff. There is a need for education of healthcare providers regarding the necessity of detecting alpha-AAT deficiency in patients with COPD. The most damaging of these mutations is the Z-1 antitrypsin deficiency. There is a need for education of healthcare providers regarding the necessity of detecting alpha-AAT deficiency in patients with COPD. COPD: Respiratory Therapists (RRTs) who perform Pulmonary Function Testing should be to educate staff. Where it diffuses into the interstitium and the alveolar fluid living. Here it inactivates neutrophil elastase, helping to protect the lungs from protease-mediated damage (Brode, Ling, & Chapman, 2012). References


Additional Sources


