Summer 2016

Systemic Lupus Erythematosus and Cardiovascular Disease

Jayme York
Otterbein University, jayme.york@otterbein.edu

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

Part of the Nursing Commons

Recommended Citation
York, Jayme, "Systemic Lupus Erythematosus and Cardiovascular Disease" (2016). Master of Science in Nursing (MSN) Student Scholarship. 162.
https://digitalcommons.otterbein.edu/stu_msn/162

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 Master of Science in Nursing (MSN) Student Scholarship by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact shickey@otterbein.edu.
Introduction
Systemic lupus erythematosus (SLE) is a challenging auto-immune disorder that impacts specific organs, membranes, and tissues (Robinson & Carr, 2011, p. 629). This auto-immune process is caused by the lack of tolerance to self-antigens and leads to the activation of the immune system against self-antigens, including the brain, heart and kidneys (Chen, Wu, & Li, 2015, p. 5). SLE is also referred to as lupus, a disease that affects women more often than men, especially during their childbearing years (Robinson & Carr, 2011, p. 631). Over the past 3 decades, the five-year survival rate for patients suffering from lupus has increased from 50% to 95% with decreasing deaths from infection and renal disease (Weston et al., 2014, p. 130). Cardiovascular disease, however, has been a key cause of death for those diagnosed with SLE (Gilbert & Ryan, 2014, p. 1902). The cardiovascular system is known to be a potential target for patient with lupus, causing 7-10 fold increase in cardiovascular disease (CVD) (Weston et al., 2014, p. 130). Complications of CVD include pericarditis, valvular and atherosclerotic changes. Women with SLE are more prone to ischemic heart disease than men (Davies, 2011, p. 2112). SLE can be associated with the signs and symptoms of other superficial disease processes, while the underlying etiology is auto-immune (Weston, 2012, p. 38).

Pathophysiology
The pathophysiology of SLE occurs at both the cellular and tissue level. The cellular level refers to the production and tolerance of auto-antibodies. The clinical manifestations of SLE occur at the tissue level by the formation of immune complexes that accumulate and deposit in organ tissues.

Cellular level:
- Abnormal activation of T and B cells, along with altered signaling events within the cell are characteristic in those with SLE (Tsokos, 2011, p. 2112).
- Signaling abnormalities within the cell lead to a deficiency of the interleukin-10 (IL-10) cytokine, causing a decrease in efficacy of the cytotoxic T-cell and a decrease in symptomatic flare-ups. Presence of the virus T cells (Tsokos, 2011, p. 2112).
- Cells migrate to inflammatory areas of the body and have a heightened expression of the COX4 adhesion molecule facilitating the endothelial lining of the blood vessel (Tsokos, 2011, p. 2112).
- Inflammation of cells are responsible for antibody production and are increased during the active stage of SLE (Tsokos, 2011, p. 2112). An increased production of auto-reactive antibodies correlates to the expression/flares of SLE. The body is not able to control the expression of Treg cells. Patients with SLE have decreased and/or dysfunctional Treg cells, which contribute to the pathogenesis of SLE (Moslen & Tsokos, 2015, p. 2244).

Tissue level:
- SLE is referred to as a type III hypersensitivity with the production of immune complexes that are deposited into peripheral tissues. Immune complexes are composed of multiple auto-antibodies and self-antigens that are circulating within the bloodstream (Liao, Rehl, & Luo, 2014, p. 1).
- Once the immune complexes are deposited into the tissues, the immune system is activated with an inflammatory response ultimately instigates the innate inflammatory response and promotes tissue damage (Luo, Rehl, & Luo, 2016, p. 1).

Significance of Pathophysiology
- According to the CDC (2015), prevalence and incidence rates for SLE are difficult to predict due to the non-specific nature of presenting symptoms. SLE is a strong predictor for development of lupus with more than 20 different genetic abnormalities identified (Weinstein, 2012, p. 38). A comprehensive history and physical assessment should be performed by a healthcare practitioner to identify any possible risk for SLE (Weinstein, 2012, p. 39).
- Early screening and diagnosis, as well as appropriate risk factor management strategies have improved treatment and patient outcomes. Patients with SLE that has improved life expectancy and quality of life (Turano, 2013, p. 51). Cardiovascular disease is becoming the leading cause of death amongst patients with SLE and have been attributed to the new successful treatment options and improved life expectancy (Gilbert & Ryan, 2014, p. 1902). Healthcare providers should focus on patient education regarding the risks of cardiovascular disease associated with SLE and risk factor management (Winston et al., 2007, p. 45).

Implications for Nursing Care
Understanding the pathophysiology of auto-immune disorders such as SLE aids in the screening, diagnosis and early treatment to decrease the progression of SLE. Using the traditional Framingham risk factors for cardiovascular disease in patients with SLE is insufficient in providing prophylactic therapy (McMahon et al., 2014). Healthcare providers should screen for cardiovascular risk factors and mitigate SLE, including (Tsokos, 2013, p. 51):
- Hypertension
- Dyslipidemia
- Diabetes Mellitus
- Obesity
- SLE disease activity
- Renal disease
- Antiphospholipid or lupus anticoagulant levels

Preventative screenings provide better identification of high risk patients and enable early preventative care. In addition, education should be provided to all patients regarding the risk of cardiovascular disease. Focus should be placed on modifying risk factor management of blood pressure, weight, smoking status, and activity level.
- Treatment using aggressive blood pressure management should be maintained in patients with SLE. Use of a multi-drug regimen, including angiotensin-converting enzyme (ACE) inhibitors, and thiazide diuretics, may be necessary to control hypertension (Turano, 2013, p. 52). The use of beta-blockers can precipitate Raynaud’s phenomenon in patients with SLE and should be utilized only as a second-line treatment (Turano, 2013, p. 51). Corticosteroids are considered to be the gold standard treatment in SLE. Long-term use of corticosteroids, however, increases the risk of developing obesity and diabetes, thereby, increasing the risk of cardiovascular disease (Turano, 2013, p. 51).
- Limiting individualized plans of care can be helpful to treat each patient with SLE, improving the efficacy of treatment and prolonging the progression of SLE.

Conclusion
Systemic Lupus Erythematosus is difficult to diagnose as many symptoms are vague and non-specific, including fatigue, joint pain, fever and recurrent infections. Female is at a higher risk of developing SLE than males by a 9:1 ratio (Westenst, 2012, p. 39). Currently, there is no definitive diagnostic test for SLE; however, the American College of Rheumatology have developed a list of criteria to help diagnose patients with lupus (Weinstein et al., 2014, p. 130).

References
Turano, L. K., & Hahn, B. H. (2014). A panel of biomarkers is associated with increased risk of the presence and progression of atherosclerosis in women with systemic lupus erythematosus. Arthritis & Rheumatology, 66(1), 130-139. doi:10.1002/art.38204
Jayme York RN, BSN
Otterbein University, Westerville, Ohio