Systemic Lupus Erythematosus and Cardiovascular Disease

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Introduction
Systemic lupus erythematosus (SLE) is a challenging autoimmune disorder that occurs specific flare, remissions, and relapses (Robinson & Currie, 2011, p. 629). This autoimmune process is caused by the lack of tolerance to self-antigens or by the ability of the immune system to recognize self-antigens, including the brain, heart, and kidneys (Chen, Wu, & Li, 2015, p. 1). SLE is also referred to as lupus, a disease that affects women more often than men, especially during their childbearing years (Robinson & Currie, 2011, p. 651). 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 (Wensten et al., 2014, p. 130). Cardiovascular disease, however, continues to be a main cause of death for those diagnosed with SLE (Gilbert & Ryan, 2014, p. 1902). The cardiovascular system is known to be a potential target for patients with lupus, causing 7- to 10-fold increase in cardiovascular disease (CVD) (Weinstein et al., 2014, p. 130). Complications of CVD include pericarditis, valvar, and atherosclerotic changes. Women with SLE, however, are more susceptible to the auto-antibodies and syndromes of other cardiovascular diseases, such as obesity and hypertension. Patients with lupus exhibit antiphospholipid antibody syndrome which can lead to a decrease in the production of anti-CD44 adhesion molecules, facilitating the endothelial lining of the blood vessel (Toskis, 2011, p. 2112). Immune complexes are composed of multiple auto-antibodies and self-antigens that are circulating within the blood stream (Liao, Reil, & Luo, 2011, p. 1). Once the immune complexes are deposited into the tissues, the immune system is activated in which ultimately incites the innate inflammatory response and promotes tissue damage (Liao, Reil, & Luo, 2011, p. 1).

Cellular level
- Abnormal activation of T and B cells, along with altered signaling events within the cell are characteristic in those with SLE (Toskis, 2011, p. 2112).
- Signaling abnormalities within the cell lead to a deficiency of the interferon-2-β (IFN-β) cytokine, causing a decrease in the ability to fight the cytotoxic T cell and a decrease in suppression of the disease process (Toskis, 2011, p. 2112).
- T cells migrate to inflammatory areas of the body and have a heightened expression of the COX-2 adhesion molecule, facilitating the endothelial lining of the blood vessel (Toskis, 2011, p. 2112).

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 blood stream (Liao, Reil, & Luo, 2011, p. 1).
- Once the immune complexes are deposited into the tissues, the immune system is activated in which ultimately incites the innate inflammatory response and promotes tissue damage (Liao, Reil, & Luo, 2011, p. 1).

SLE can be difficult to diagnose as many symptoms are vague and non-specific, such as fatigue, joint pain, fever, and recurrent infections. Females are at a higher risk of developing SLE than males by a 9:1 ratio (Wensten, 2012, p. 39). Currently, there is no formal diagnostic test for SLE, however, the American College of Rheumatology have developed a list of criteria to help diagnosis patients with lupus.

Four of the following symptoms must be present for the formal diagnosis of Lupus (Wensten, 2012, p. 39):
- Malar Rash
- Oral ulcers
- Photosensitivity
- Arthritis
- Serositis
- Pericarditis
- Pericardial or peritoneal involvement
- Blood disorders: anemia, leucopenia, lymphopenia, thrombocytopenia
- Immunologic disorders: positive anti-double-stranded DNA (anti-dsDNA), anti-Sm, antiphospholipid antibodies (APL)
- Positive antinuclear antibody (ANA) test

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 occurs at the tissue level by the formation of immune complexes that accumulate and deposit in organ tissues.

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. Furthermore, women are at a strong prevalence for development of lupus with more than 20 different genetic abnormalities identified (Weinstein, 2012, p. 40).
- Auto-antibodies in patients with SLE are thought to be the initial trigger associated with endothelial damage (Turano, 2013, p. 3).
- Fifty different auto-antibodies have been identified in the pathogenesis of SLE, including anti-dsDNA, antiphospholipid(p-Ab), anti-Sm, and ANA (Weinstein, 2012, p. 42).
- Overproduction of auto-antibodies molecules promote endothelial damage, leading to atherosclerosis (Toskis, 2011, p. 2112). The extent of atherosclerosis is directly correlated to the duration of SLE symptoms (Toskis, 2013, p. 49).
- Erythrocyte changes can alter the functionality of T cells and instigate the autoimmune response (Moulton & Toskis, 2015, p. 2255).

Figure 1. Malar Rash

Figure 2. Pathophysiology of SLE (Toskis, 2011, p. 2112)

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 ineffective in providing successful treatment therapy (McKenna et al., 2015, doi:10.1001/jama.2015.6269157).

Healthcare professionals should screen for cardiovascular risk factors in patients with SLE, including (Turano, 2013, p. 51):
- Hypertension
- Dyslipidemia
- Smoking
- Obesity
- SLE disease activity

- Antiphospholipid or lupus anticoagulant levels

Preventive screenings provide better identification of high risk patients to enable early preventative care. In addition, education should be provided to all patients regarding the risk of cardiovascular disease. Focus should be placed on modifiable 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 be the gold standard treatment in SLE. Long-term use of corticosteroids, however, increases the risk of impaired glucose metabolism, obesity, and renal disease (Weinstein et al., 2011, p. 2112). The incidence of diabetes mellitus has increased in patients with SLE due to decreased renal function, resultant insulin resistance and obesity (Tsokos, 2013, p. 5).

The cardiovascular system is known to be a potential target for patients with lupus, causing 7- to 10-fold increase in cardiovascular disease (CVD) (Weinstein et al., 2014, p. 130). Complications of CVD include pericarditis, valvar, and atherosclerotic changes. Women with SLE, however, are more susceptible to the auto-antibodies and syndromes of other cardiovascular diseases, such as obesity and hypertension. Patients with lupus exhibit antiphospholipid antibody syndrome which can lead to a decrease in the production of anti-CD44 adhesion molecules, facilitating the endothelial lining of the blood vessel (Toskis, 2011, p. 2112).

Once the immune complexes are deposited into the tissues, the immune system is activated in which ultimately incites the innate inflammatory response and promotes tissue damage (Liao, Reil, & Luo, 2011, p. 1).

Conclusion
Systemic Lupus Erythematosus is difficult to diagnose as many symptoms are non-specific, including fatigue, joint pain and fever (Wensten, 2012, p. 39). A comprehensive history and physical assessment should be performed by a healthcare practitioner to determine the possible risk for SLE (Wensten, 2012, p. 39). Early screening and diagnosis, as well as, appropriate risk factor management strategies have improved long-term outcome in patients with SLE who have been treated with early intervention treatment to each patient with SLE, improving the efficacy of treatment to decrease the progression of SLE. Using solely the treatment to each patient with SLE, improving the efficacy of treatment to decrease the progression of SLE. Using solely the treatment to decrease the progression of SLE. Using solely the treatment to decrease the progression of SLE. Using solely the treatment to decrease the progression of SLE. Using solely the treatment to decrease the progression of SLE. Using solely the treatment to decrease the progression of SLE. Using solely the treatment to decrease the progression of SLE.