Systemic Lupus Erythematosus and Cardiovascular Disease

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**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 et al., 2013, p. 212).
- Signaling abnormalities within the cell lead to a deficiency in the interferon-2-γ (IFN-γ) pathway, causing a decrease in the expression of the cytotoxic T-cell and a decrease in apoptosis in antigen-presenting cells (Weinstein et al., 2011, p. 212).
- T cells migrate to inflammatory areas of the body and have a heightened expression of the CD44 adhesion molecules, facilitating attachment to the endothelial lining of the blood vessel (Tsokos, 2011, p. 212).
- Tissue damage responsible for antibody production and are increased during the active stage of SLE (TSokos, 2011, p. 212). An increased production of auto-reactive antibodies correlates to the expression of IFN-γ in SLE.

### 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, Reif, & Luo, 2016, p. 1).
- Once the immune complexes are deposited into the tissues, the immune system is activated with the ultimate instigation of the innate inflammatory response and promotes tissue damage (Liao, Reif, & 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. Disease onset is usually within a strong pattern of development for 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 causative agent associated with endothelial damage (Turano, 2013, p. 56).
- Several different auto-antibodies have been identified in the pathogenesis of SLE, including anti-dsDNA, antiphospholipid antibodies (aPL), anti-cardiolipin antibodies (aCL), and anti-beta-2-glycoprotein I (anti-b2-gp I) (Liao, Reif, & Luo, 2016, p. 42).
- Overexpression of adhesion molecule glycoproteins promotes endothelial damage, leading to atherosclerosis (Tsokos, 2011, p. 212). The extent of atherothrombotic disease is directly correlated to the duration of SLE symptoms (Turano, 2013, p. 49).
- Erythropoietic changes can also alter the functionality of T cell targets and instigate the auto-immune response in SLE (Mouhous & Tsokos, 2015, p. 2253).

- According to Mouhous & Tsokos (2015), hypertrophy of numerous genes, including COIL1, perforin, CD70, and CD40L, have demonstrated significant associations with SLE (Tsokos, 2011, p. 212).
- The focus of treatment is associated with limiting the effect of immunosuppressive therapy to prevent damage without steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, antimicrobial and immunosuppressive agents (Turano, 2013, p. 49).

**Implications for Nursing Care**

Understanding the pathophysiology of autoimmune diseases, such as SLE, aids in the screening, diagnosis, and early treatment to đức the progression of SLE. Using the traditional Framingham risk factors for cardiovascular disease in patients with SLE is insufficient in providing appropriate preventative therapy (McMahon, K., & Angelopoulos, B. G., 2015, p. 2227).

- Healthcare professionals should screen for cardiovascular risk factors, such as SLE with including, (Tsokos, 2013, p. 51):
  - Hypertension
  - Dyslipidemias
  - Diabetes Mellitus
  - Obesity
  - SLE disease activity
  - Smoking
  - Antiphospholipid or lupus anticoagulant antibodies

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 upon modifiable risk factor management to provide better long-term patient outcomes and improve quality of life.

**References**


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**Figure 1**, Malar Radh

**Figure 2** Pathophysiology of SLE (Tsokos, 2011, p. 2122)