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Caring for Cardiovascular Disease in Patients with SLE

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Introduction

In the U.S., approximately 5-20 people per 100,000 are affected with the complex autoimmune disorder called Systemic Lupus Erythematosus (SLE). SLE affects predominately more females than males (9:1female to male), with a higher incidence in African American women. (Barbehaiya, Costenbader, Parks, & Santos, 2017). SLE is a debilitating condition that has multisystem involvement which affects the skin, renal, musculoskeletal, and cardiac systems. "SLE is caused by interactions between susceptible genes and environmental factors, which can include ultraviolet light, infections, and viruses, resulting in an irreversible loss of immunologic self-tolerance" (Garyfallos, Goulielmos, Niewold, & ...Zervou, 2018, p. 59).

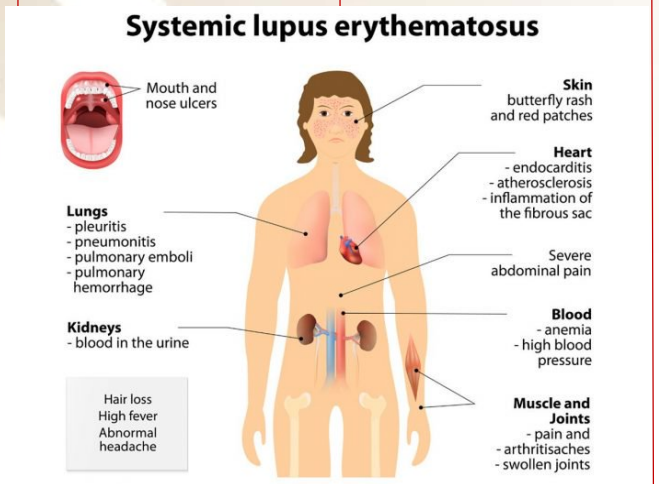
Cardiovascular disease is a leading cause of mortality in the world, and atherosclerosis is a hallmark in the disease. People with SLE have a 6-fold higher risk of developing atherosclerotic lesions that cause cardiovascular disease (Leng, Li, & ...Ye, 2015). "Increasing evidence shows that the cardiovascular morbidity and mortality are significantly higher in SLE than in the general population" (p.23).

Just a couple of years back my mother had a Non ST Elevated Myocardial Infarction which lead to her diagnosis of SLE. Our family was not knowledgeable regarding this condition because no one in our family has SLE. Now every chance I get I try to learn as much about SLE to help better care for my mother and myself. Through research I've learned that I have a few of the risk factors that would put me at a higher incidence of developing the condition. Firstly, I am an African American woman of childbearing age which increases my prevalence of developing the condition. This is an eye opener for because in SLE young women have a 50 times higher risk for myocardial infarction compared to healthy women of similar age distribution (Giannelou & Mavragani, 2017). Secondly, I have a higher incidence due to the fact that SLE is hereditary and my mom has the condition. It is for these reasons that I chose to research SLE.

Signs & Symptoms

- Fatigue
- Fever
- Joint pain, stiffness and swelling
- Butterfly-shaped rash on face
- Photosensitivity
- Chest pain when taking a deep breath
- Hair loss
- Weight loss
- Mouth sores
- Raynaud's phenomenom
- Swollen lymph nodes
- Headaches
- Memory loss
- Seizures

(Paz MD, 2017)



Diagnosis

Lupus is difficult to diagnose because its signs and symptoms mimic other conditions. This is a condition that progresses slowly over years, and it has episodic periods of exacerbation and remission. (Paz MD, 2017). You have to have at least four of the eleven clinical findings to be diagnosed with SLE:

1.) Malar Rash
2.) Discoid Rash
3.) Mouth Ulcers
4.) Photosensitiviety
5.) Arthritis
6.) Lung or Heart Inflammation
7.) Renal Problems
8.) Neurologic Problems
9.) Hematologic Problems
10.) Immunologic Problems
11.) Positive Antinuclear Antibodies

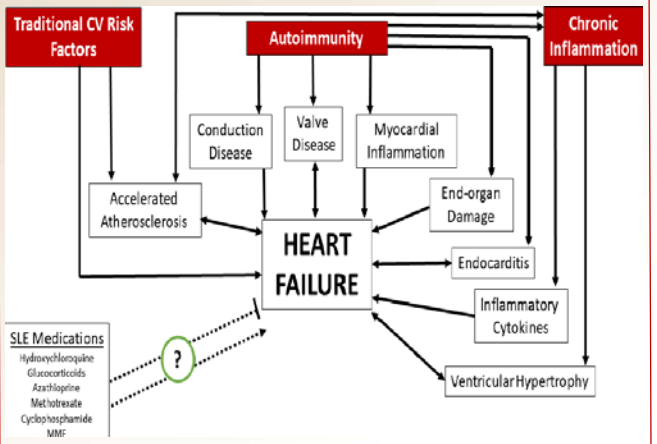
(Paz MD, 2017)

Pathophysiology on Atherogenesis in SLE

The exact mechanisms that lead to increased risk for heart failure in SLE are not known, however data shows that aberrant host-immune response and chronic inflammation lead to accelerated atherosclerosis (Al-Kindi, Dhakal, Kim, & Oliveira, 2018). "In addition, SLE has direct cardiac, vascular, and other systemic manifestations that lead to increased risk of cardiovascular disease and heart failure" (p.188).

Endothelial Dysfunction

Endothelial dysfunction is one of the earliest signs of atherosclerosis, resulting in increased expression of adhesion molecules and impaired vasodilation. An imbalance between circulating apoptotic endothelial cells (ECs) indicate vascular damage and contribute to endothelial dysfunction. Also patients with SLE have increased concentrations of reactive oxygen species and decreased antioxidant defense mechanisms which provide a conducive environment for oxidation of lipoproteins and atherosclerosis (Bortoluzzi, et al., 2018, pp. 2-3).



Monocytes and T-Cell Recruitment and Activation

Monocytes can migrate into the intima and differentiate into macrophages, which leads to a further transformation into foam cells that secrete proinflammatory cytokines (Bortoluzzi, et al., 2018).

T-cells, predominately CD4 cells, have an increased affinity to infiltrate newly formed atherosclerotic plaques (Bortoluzzi, et al., 2018).

Toll-Like Receptors

Toll-like receptors (TLRs) are a class of pattern recognition receptors expressed on multiple cells involved in innate immunity. In SLE, people have a dysregulated activation of TLRs, resulting in upregulated production of autoantibodies and cytokines (Bortoluzzi, et al., 2018). This leads to the recruitment of activated inflammatory cells, self-perpetuating the process of inflammation and plaque formation (p.3).

Cytokines

Cytokines such as interferons alpha and gamma (INF) are involved in atherosclerosis and SLE. "IFN alpha serves as a proatherogenic mediator through repression of endothelial nitric oxide synthase-dependent pathways promoting the development of endothelial dysfunction and cardiovascular disease in SLE" (Bortoluzzi, et al., 2018, p. 3).

IFN gamma participates in atherogenesis by stimulating ECs and macrophage activation, proinflammatory mediator production, adhesion-molecule expression, and by inhibiting smooth muscle cell proliferation and collagen production (Bortoluzzi, et al., 2018, p. 3).

Other cytokines participate in the initiation and perpetuation of the atherosclerotic process by stimulating the activation of macrophages, inducing the secretion of matrix metalloproteinases, upregulating the expression of adhesion molecules on the ECs, increasing the concentration of chemotactic messengers, and affecting the proliferation of smooth muscle cells (Bortoluzzi, et al., 2018, p. 3).

Significance of Pathophysiology

SLE Manifestations increase the risk factors for Heart Failure

Myocardial Dysfunction: Left Ventricular Systolic Dysfunction or Diastolic Dysfunction

Left Ventricular Hypertrophy: Secondary to arterial HTN and correlation between SLE duration and left ventricular mass

Valvular Disease: is common in SLE however over heart failure due to valvular involvement is rare

Conduction System Disease: SLE may manifest as arrhythmias such as atrial fibrillation and complete heart block

Endocarditis: SLE predisposes people to infective and non-infective endocarditis

Myocarditis: can lead to conduction problems, dilated cardiomyopathy, and heart failure

Pericarditis: manifests as pleuritic substernal pain and the most frequent cause of symptomatic cardiac disease in SLE

Anaphospholipid Syndrome: predisposes people to valvular disease, arterial and venous thrombosis, and pulmonary hypertension

(Al-Kindi, Dhakal, Kim, & Oliveira, 2018)

Conclusions

Systemic Lupus Erythematosus is a complex autoimmune inflammatory condition that increases the risk for atherosclerosis. Cardiovascular disease is the leading cause of death in the U.S. and atherosclerosis is the most common risk factor. SLE is non curable however advances in research has allowed people with the condition to live longer. More research is needed on the complex pathophysiology of the condition to better understand the disease process.

Nursing Implications

Active SLE is no longer the major cause of death in the condition. Now mortality arises from conditions such as cardiovascular disease or renal failure complications. Obesity and hypertension are some of the known contributors of atherosclerosis. It is recommended that SLE patients with these conditions do things to try to reverse or control the condition. Bichele and Petri recommend a 500 calorie deficit from their diet as a well as two and a half hours of moderate intensity aerobic exercise weekly to decrease obesity (2014). Also, "guidelines recommend a target blood pressure of less than 130/80 mmHg" (Bichile & Petri, 2014) to manage hypertension.

Advances in disease management have improved survival of SLE patients and shifted the focus to other outcome parameters such as measuring quality of life (Carnarius, Chehab, & Schneider, 2014). Caregivers should be sensitive to the mental and physical attributes that come along with SLE. Fatigue, depression, and pain are common symptoms in SLE that lead to a decreased quality of life.

Depression and anxiety in SLE increases the incidence of co-morbidities such as cardiovascular disease (Carnarius, Chehab, & Schneider, 2014).

The pain and discomfort of SLE can be physically and mentally debilitating. Encourage SLE patients to maintain activity when the symptoms are mild or in remission (Carnarius, Chehab, & Schneider, 2014).

Caregivers should be educated on the medications to help treat SLE, and be ready to advocate on their behalf. Hydroxychloroquine is an antimalarial with known benefits in treating SLE, however the percentage of people taking the medication remains low (Chalumeau, Dunogue, Guern, Imber, & Morel, 2014). Hydroxychloroquine helps "reduce the high cardiovascular risk of SLE patients" (p. 168).

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