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Lupus Nephritis: A Synopsis of Pathophysiology and Implications for Advanced Nursing Practice
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Introduction of Pathophysiology
Systemic Lupus Erythematosus (SLE) can be described as a chronic, complex, autoimmune disorder (McCance & Huether, 2014). As the disease progresses, it disproportionately affects women, and those of Afro-Cuban, Hispanic, Asian, and American Indian descent. SLE has a prevalence of 2.0 to 7.6 cases per 100,000 persons in the United States. Characterized by chaotic autoantibody production, complement alterations, and formation of immune complexes, SLE has the potential to generate devastating damage to multiple organ systems. Formed from the binding of autoantibodies and self-antigens, immune complexes often result in renal damage, a significant complication of the disease. Through careful analysis and synopsis of literature, the writer intends to provide the audience with pathophysiological concepts defining lupus nephritis (LN). Within ten years of SLE diagnosis, between 50% and 60% of adults diagnosed will go on to develop LN (Itchey, 2014). Of those with LN, up to 17% will go on to develop and stage renal disease (Bomback & Bomback, 2016). The devastating course of this condition provides the impetus for new interest for the purpose of this project, as we have sought to gain valuable knowledge regarding the aetiology and implications for advanced nursing practice.

Clinical Manifestations
Many patients experience no symptoms at all. Typically, LN is suspected in SLE patients producing abnormal urinalysis results, possibly with an elevated serum creatinine level. Patients may demonstrate persistent proteinuria greater than 0.5 grams per day, random protein-creatinine ratios greater than 0.5 grams, and the production of urine with active sediment consisting of blood cells and/or casts greater than 5 without urinary tract infection. Serum creatinine, blood urea nitrogen, and antinDNA studies may be elevated, while glomerular filtration rate is usually decreased. An elevated serum creatinine is indicative of renal insufficiency to be elevated with both active SLE and LN. A prominent player in type I hypersensitive reactions, IgE is a significant player in active SLE. The biologic basis for the histopathologic alterations associated with use of immunosuppressants and corticosteroids in SLE is the patient's inability to destroy immune complexes. It is of note to recognize the disproportionate number of non-white women of lower socioeconomic status diagnosed with LN. Furthermore, SLE related and stage renal disease is linked to lower socioeconomic status (Bomback, 2014). The significance of hypertension and level proteinuria is directly linked to the presence of LN and LN patients. Additionally, patients with active SLE and LN will exhibit hypertension, proteinaemia, reduced glomerular filtration rate, hypoalbuminaemia and nephrotic syndrome. The differentiation between class III and IV is made through the determination of the percentage of glomeruli affected. When 50% of glomeruli are involved, class IV is diagnosed, whereas less than 50% involvement is consistent with class III. Class V lupus membranous-nephropathy, is characterized by hyperplasia capillary wall thickening and subepithelial immune complex deposits on light and electron microscopy. Class V patients may present with hypertension and microscopic hematuria without significant serum creatinine elevation. Class V, advanced sclerosing lupus nephritis, is characterized by the scarring of at least 90% of glomeruli. Patients with LN are also noted to be elevated with both active SLE and LN. A prominent player in type I hypersensitive reactions, IgE has historically been identified in autoimmune disease. Dima et al. (2016) examined elevated autoimmune IgE levels to be significant in relationship with active SLE and LN. The advanced practice nurse should suspect LN in SLE patients presenting with selected symptoms. Hahn et al. (2012) identify these symptoms as:

- Malar (Butterfly) rash
- Edema of the upper and lower extremities
- Changes in urine output
- Weight gain

In addition to the above symptoms, patients with active LN typically demonstrate signs of active SLE as described by Hahn et al. (2012). Additional symptoms include:

- Malar (Butterfly) rash
- Light sensitivity
- Fatigue
- Arthritis and arthralgia
- Oral ulcers
- Gastrointestinal upset
- Photosensitivity
- Lesions of the mouth and nose
- Pleurisy
- Rejection of a phenomenon

Implications and Conclusion
Understanding the pathophysiology of LN is paramount to early diagnosis and treatment of lupus nephritis. Bober et al. (2015) state that patients and providers face a three-year mortality rate of roughly 27% (Zoome-Saemi et al., 2015). Given the autoreactivity and inflammation which contribute to renal damage, inducing early intervention in the development of LN is crucial. This is accomplished through the use of potent immunosuppressants and corticosteroids over a course of time in order to allow for tissue healing (Pons et al., 2016). Complete remission occurs in a small number of patients, with the majority of LN patients relapsing. The goal of maintenance therapy is to limit relapses through continued use of drugs, all while minimizing the toxic effects and long-term complications associated with medications. While the most advantageous length of maintenance therapy time remains ambiguous, 12–18 month-long courses have been studied (Parkh et al., 2014). The advanced practice nurse should suspect LN in SLE patients presenting with selected symptoms. Hahn et al. (2012) identify these symptoms as:

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Routine care of the SLE patient should be completed every three to six months, with some studies completed at every office visit to assess for the presence of protein, blood cells, and casts. Serologic work up should include complete blood cell count, coagulation studies, metabolic panel, complement, immunoglobulin, and antiDNA antibody testing. Careful attention should be paid to the physical assessment as well. Aside from the above-mentioned symptoms, visual inspection of the face and extremities for evidence of edema, and the skin for dermatologic signs of renal disease such as thickening of the epidermis and pigmentation alterations, through evaluation of skin biopsy, should also be considered. Information gained from inquiries regarding urinary habits can be crucial for early diagnosis of LN. For patients with suspicion for LN, prompt renal biopsy should be completed to determine the extent of kidney involvement (Hahn et al., 2012). If medication is indicated for LN, patients should be seen every two weeks for medication adjustments. Additionally, it is crucial for the advanced practice nurse to guide the patient to a nephrologist for immunosuppressive medications and utilization of multi-disciplinary team members such as dieticians, endocrinologists, pathologists, and hematologists.

References
Figure 2: The malar rash associated with active SLE.

Figure 3: This table displays renal biopsy findings consistent with the different classifications of lupus nephritis (Zager, 2011).

Table 1: International Society of Nephrology/Pathology classification of lupus nephritis (Sleiman, 2016).

Class I: Mild active lupus
Class II: Moderate single lesion
Class III: Mild active lupus
Class IV: Moderate single lesion
Class V: Severe active lupus
Class VI: Severe active lupus

Figure 4: The differential renal function of systemic lupus erythematosus (SLE).