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 (McCanne & Hauer, 2014). 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 pathophysiologic concepts defining lupus nephritis (LN). Within ten years of SLE diagnosis, between 50% and 60% of all patients diagnosed will go on to develop LN (Ritchie, 2014). Of those with LN, upwards of 17% will go on to develop and stage renal disease (Bose & Silverman, 2015). The devastating course of this condition prompted the writer’s choice of LN as a topic of interest for the purpose of this project, as she wished to gain valuable knowledge regarding the ailment 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 antiDNA studies may be elevated, while glomerular filtration rates (GFR) are decreased. These findings must be correlated with elevated active SLE and LN. A prominent player in type II hypersensitivity reactions, IgG has historically been identified in autoimmune diseases. Dema et al. (2014) examined elevated autoimmune IgG levels to be in significant 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 persistent fatigue as a primary symptom, 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 antiDNA studies may be elevated, while glomerular filtration rates (GFR) are decreased. These findings must be correlated with elevated active SLE and LN. A prominent player in type II hypersensitivity reactions, IgG has historically been identified in autoimmune diseases. Dema et al. (2014) examined elevated autoimmune IgG levels to be in significant relationship with active SLE and LN.

Definitive diagnosis and classification of LN is made by determining the extent of glomerular injury via renal biopsy, urine, and blood studies. The classification scheme used to stage and type the severity of LN is comprised of six classifications. The most frequent form of renal involvement, class I minimal mesangial lupus nephritis, is rarely diagnosed as the urinalysis remains normal, showing minimal if any protein. Biopsy is not warranted at this stage as serum creatinine also remains normal. Class II mesangial proliferative lupus nephritis is characterized by mesangial hypercellularity and proteinuria without renal insufficiency. Subendothelial deposits or glomerular scarring noted on light microscopy, and findings of class III focal lupus nephritis and class IV diffuse lupus nephritis. Hematuria and proteinuria, decreased glomerular filtration rate and hypertension are often seen with class II, while the class III patient will exhibit hematuria, proteinuria, and glomerulosclerosis. Hypertension and nephrotic syndrome. The differentiation between class III and class IV is made through the determination of the percentage of glomeruli involved. Less than 50% of class III LN is diagnosed, while less than 50% involvement is consistent with class III. Class V lupus membranous nephropathy, is characterized by glomerular capillary wall thickening and subendothelial immune complex deposits on light and electron microscopy. Class V patients present with hypertension and microscopic hematuria without significant serum creatinine elevation. Class VI, advanced sclerosing lupus nephritis, is characterized by the scarring of multiple glomeruli, often seen with class IV lupus nephritis. Class VI LN is diagnosed to be most common in all patients. Peripheral edema will be present. Active SLE will also be present with hypertension and microscopic hematuria without significant serum creatinine elevation. Class VI, advanced sclerosing lupus nephritis, is characterized by the scarring of multiple glomeruli, often seen with class IV lupus nephritis. Class VI LN is diagnosed to be the most common form of renal involvement. Class I minimal mesangial lupus nephritis is rarely diagnosed as the urinalysis remains normal, showing minimal if any protein.

Diagnosis

Implications and Conclusion

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 (B. H. Hahn, personal communication, July 12, 2016). This highlights the importance of patient education and health literacy. Discussions regarding disease progression and renal replacement associated with both non-compliance and treatment failure should be part of education once a LN diagnosis is suspected. Additional considerations as identified by Rovin (personal communication, July 12, 2016) include:

- Management of hypertension and hyperglycemia, with goal low density lipoprotein (LDL) < 100 and goal hemoglobin A1c < 6.5
- Administration of angiotensin-converting enzyme (ACE) inhibitors, and angiotensin receptor blocker (ARBs) for those with glomerular disease and protein > 3 months
- Use of prophylactic antibiotics for pneumocystis pneumonia (PCP) prevention in the immunocompromised patient
- Prophylactic pravastatin and aspirin administration for ulcer prevention in those with long term corticosteroid therapy
- Prophylaxis of appropriate medication education, including side effects and risks associated with use of immunosuppressants and corticosteroids
- The importance of the UK patient avoiding nephrotic substances such as non-steroidal anti-inflammatory drugs (NSAIDs) and other salicylates
- Dietary modifications for renal protection, including the need to choose foods low in sodium, potassium, protein, and phosphorus
- Lipoprotein (LDL) < 100 and goal hemoglobin A1c < 6.5
- Screening for malignancy and infection related to immunosuppression

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, complement levels, marker for malnutrition, vitamin D and D, and laboratory test for malignancy. C3 and C4, as well as antiDNA antibodies, should be checked. Careful attention should be paid to the physical examination as well. Aside from the obvious 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 skin, and pigmentation alterations, through evaluation of respiratory status should also be assessed. Information gained from inquiries regarding urinary habits can be a useful tool for the provider as well as the patient.

References


