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Lupus: Open Access

Short Commentary

A Brief Note on Lupus Nephritis

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COMMENTARY

Lupus as a diagnosis requires the combination of characteristics clinical and laboratory findings. Systemic lupus erythematosus is best regarded as a syndrome, in which a number of varying immunological events may be lead to a similar clinical picture. The presence of antibodies directed against components of the cellular nucleus is detected in the serum of>95% of patients. However, lupus patients typically express a plethora of autoantibodies, including rheumatoid factor, by up to 50%, antilymphocyte antibodies, and APL in 30% of patients to specify a few [1]

There are various clinical factors in the genesis of lupus like genetics, environmental, immunodeficiency, incidence and prevalence, sex and age etc..

Genetics: Patients with lupus have defects in all arms of the immune system. Multiple gene involvement in the etiology of lupus is suggested by the increased risk of SLE or other autoimmune disorder in certain families. Major histocompatibility complex (MHC) associations have not had a strong impact in human lupus nephritis. Low TNF production is associated with greater susceptibility to lupus, including the interlukin (IL)-1 receptor antagonist, IgGm allotypes, T-cell receptor genes and drug hydroxylation – but none have yet been described independent of linkage disequilibrium with known associations. Ironically lupus appears to be rare in West Africa, whereas the incidence and prevalence in descendants of West Africans is increased in the Caribbean, North America and Europe. This pattern may reflect genetic admixture, as well as possible environmental factors [2]

Immunodeficiency: A proportion of patients with lupus have inherited immune-deficiencies. Immunoglobulin (Ig)A deficiency is associated with lupus nephritis more often than would be expected by chance. These individuals are prone to a variety of sinopulmonary and gastrointestinal infections, implicating greater antigenic stimulation of a susceptible subject in etiopathogenesis. Defective Fc receptor function has also been implicated, and is MHC-linked [4].

Environmental Factors: The earliest hypotheses for the pathogenesis of SLE suggested an inciting infection of tubercular, viral, or bacterial origin. Infections, including retrovirus, have been evaluated as candidates for including the lupus syndrome. In recent study, country of birth was shown to affect the risk of rheumatic disease. First generation immigrants from Iraq and Africa had a higher risk of lupus than did native-born Swedes; these increased risks were also seen in the second generation. These findings support the concept that both genetic and environmental factors are involved in the etiology of SLE [5].

Incidence and prevalence : A number of studies have examined the incidence of SLE, giving figures varying from 1.8 to 7.6 new cases per 100,000 per year. The incidence of SLE is much lower in children. The incidence and prevalence of SLE nephritis differs among patients of different racial/ethnic backgrounds. Despite continued investigation, these differences remain poorly understood.

Sex and age in the presentation of lupus: Female sex is a major risk factor for the development of lupus. The female:male ratio rises 3:1 in pre-pubertal children up to 4.5:1 throughout older childhood and adolescence to the 8-12:1 reported in series of adult-onset patients, falling back to 2:1 in those patients over 60 years of age. In past researchers described elderly onset lupus as "milder" lupus, with lower frequency of nephritis. However race confounds the relationship between age of SLE onset and severity of disease.

It seems likely that all forms of lupus nephritis must evolve through a symptomless phase before becoming overt, but the prognosis of "silent" nephritis has been little studied; occasionally such patients have evolved into renal failure.

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Received: March 16, 2020; **Accepted:** April 9, 2020; **Published:** April 16, 2020

Citation: Acharya D (2020) A Brief Note on Lupus Nephritis. Lupus: Open Access. 5:154. DOI: 10.35248/2165-8056.20.10.154.

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