Non-lupus Glomerulonephritis in a Patient with Systemic Lupus Erythematosus

Sir,

With reference to the interesting case report by Abdallah *et al*., IgA nephropathy (IgAN)-associated systemic lupus erythematosus (SLE) is increasingly reported worldwide.[1] Abdallah *et al*. did well in addressing various clinical and biochemical spectra of the case in question as well as discussing the relationship between IgAN and SLE.[1] Based on a review of literature on this issue, Abdallah *et al*.[1] mentioned that some studies reported IgAN as a complication of SLE when the two diseases coexist while other studies assumed that IgAN might be a special clinical subtype of SLE. Abdallah *et al*. also stated that the predominant mesangial IgA deposits in renal biopsy might be a special subtype of lupus nephritis (LN).[1] I presume that the following two points might additionally contribute to the association of IgAN with SLE. First, it is worth mentioning that several novel susceptibility genes for SLE and IgAN have been identified in recent genome-wide association studies. Since both LN and IgAN are autoimmune renal diseases, they might share common disease mechanisms that overlap with genetic tendency. This is supported by the demonstration of shared genetics between IgAN and SLE.[2,3] Second, it is obvious that immune complexes (ICs) glomerulonephritis (GN) is a common diagnosis in renal biopsy series of human immunodeficiency virus (HIV)-infected patients. However, there are a variety of glomerulonephritides associated with HIV infection, including IgAN, membranoproliferative GN, membranous nephropathy, lupus-like GN, immunotactoid glomerulopathy, and fibrillary GN. In addition, HIV-related proteins might be implicated in circulating ICs directly related to a response to the infection. In some cases, the relationship of the HIV infection to the GN is unclear. HIV infection is associated with the development of polyclonal hypergammaglobulinemia, which can promote the development of circulating ICs. It is not clear if HIV-associated GN is caused by the passive trapping of these circulating ICs or the *in situ* deposition of antibodies binding to HIV viral antigens.[4] Data on the magnitude of the recent HIV infection in Kuwait are not yet available. However, the data that are available indicated that the adult HIV prevalence rate is 0.1%.[5] Despite such a low prevalence, HIV infection must be considered by Abdallah *et al*. and a suitable diagnostic battery of viral load and CD4 count measurements ought to be contemplated.[1] If that battery reveals HIV infection, the case in question would be considered a novel case report of concomitant HIV infection, SLE, and IGAN as such association has never been reported in the literature.

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Conflicts of Interest

There are no conflicts of interest.

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