Introduction

Systemic lupus erythematosus (SLE) is an autoimmune multisystem disease with a spectrum of clinical manifestations and a variable course characterized by exacerbations and remissions. Antinuclear antibodies (ANAs), especially those to native DNA, are common immunologic abnormalities found in the disease. The broad clinical spectrum of SLE challenges the diagnostic and therapeutic acumen of the physician**.(Salmon et al., 2006).**

There is an increased risk of atherosclerotic phenomenon in systemic lupus erythematosus patients**(Padilla et al.,2009)**.

A growing body of evidences reinforces the close link between systemic lupus erythematosus (SLE) and atherosclerosis which is due to traditional and nontraditional risk factors for cardiovascular diseases**(de Carvalho et al., 2008).**

It is now recognized that SLE has a particular pattern of dyslipoproteinemia characterized by low HDL levels and increased triglycerides, which is aggravated by flares. Multiple mechanisms can induce an altered lipoprotein metabolism in SLE such as cytokines produced during systemic inflammation, autoantibodies and therapy **(de Carvalho et al., 2008).**

Within 3 years of diagnosis, up to 75% of patients with SLE will have at least 1 serum total cholesterol reading _5.2 mmoles/liter (_200 mg/dl) **(Bruce et al., 1999)**, due to numerous factors, including active disease **(Reichlin et al., 2002),** renal injury **(Leong et al ., 1994),** and corticosteroid use **(Svenungsson et al.,2003).**

Systemic lupus erythematosus (SLE) is strongly associated with premature atherosclerotic CAD **(Nikpour et al., 2009).**

One in 10 patients with SLE is diagnosed with clinical CAD, making this complication one of the leading causes of morbidity and mortality in SLE**(Urowitz et al., 2007).**

It has been reported that the dyslipideamia in SLE patients is related to disease activity as well as presence of renal and cardiovascular disease **(Svenungsson et al., 2003).**

Ongoing inflammatory SLE disease activity is associated with CV risk **(Roman et al., 2003).** A six-point increase in the Systemic lupus Erythematosus Disease Activity Index (SLEDAI) score over 1 year correlated with a 5% increase in a 2-year CV risk **(Karp et al.,2008).** This same increase in SLEDAI score was associated with increases of 3.4 mmHg in systolic blood pressure, 1 mg/dl in glucose and 11.6 mg/dl in TG as well as a 2.3 mg/dl decrease in HDL cholesterol.

Renal involvement, which, if present, is usually seen within 5 years of the diagnosis of SLE **(Abu- Shakra et al.,1995),** ranges from mild asymptomatic proteinuria to rapidly progressive glomerulonephritis leading to end-stage renal disease(ESRD). In patients with SLE, the estimated prevalence of nephritis is 40–75% **(Golbus et al.,1994).**

Renal disease is one of the most common internal organ manifestations of SLE. Both hypertension and dyslipideamia are well prescribed with lupus nephritis and renal disease **.** Lupus renal disease is also associated with increased atherosclerosis **[(Selzer et al., 2004)-(Maksimowicz-McKinnon et al 2006)].**In fact, nearly 50% of deaths in lupus patients with renal disease are attributed to CV or cerebrovascular disease **(Appel et al.,1994).**

Although it is widely accepted that renal impairment can disturb lipid profiles, there is now mounting evidence that dyslipidemia can, in turn, accelerate, if not incite, renal damage. In addition to hyperfiltration injury due to endothelial dysfunction and impaired vasomotor response of the afferent arteriole consequent to atherosclerosis ***(Vazquez-Perez et al., 2001),*** dyslipidemia may lead to glomerulosclerosis and tubulointerstitial injury, via mechanisms akin to those involved in atherosclerosis ***(Crook et al.,2003).***

This reciprocal interaction between plasma lipids and renal function warrants attention, especially in patients with the multisystem inflammatory disease systemic lupus erythematosus (SLE), in which both kidney dysfunction

and dyslipidemia are commonly seen and contribute to significant morbidity and mortality **(Manger et al.,2002).**

Results of a meta-analysis of trials of lipid-lowering drugs in renal diseases of various etiologies further suggest that treatment of hyperlipidemia may slow the rate of decline of the glomerular filtration rate **(Fried et al.,2001).**