INTRODUCTION

Systemic lupus erythematosus (SLE) is an unpredictable multi-systemic autoimmune life-long disease whose aetiology and pathogenesis are incompletely understood. Clinical features are diverse and episodic with differring immunological manifestations. Both adults and children with SLE have a significant morbidity and mortality, which is closely related to renal involvement ***(Stephen et al., 2008).***

There is renal involvement in the great majority of patients with SLE: 66-90%, at sometime of the evolution ***(Jayne, 2007)***, meanwhile, lupus nephritis (LN) occurs in more than one-third of patients. Production of nephritogenic autoantibodies, glomerular immune complex deposition, and cytokine overproduction have been postulated to contribute to the pathogenesis of lupus nephritis ***(Tucci et al., 2005)***.

The TNF superfamily cytokines weak inducers of apoptosis (TWEAK) induce the mesangial cells, podocytes and endothelial cells to secrete pro-inflammatory chemokines including the monocyte chomoattractant protein-1(MCP-1), interleukin-10 (IL-10) which are crucial in the pathogenesis of lupus nephritis **(Schwartz et al., 2007).**

The experimental data and the study on human renal tissue in patients with glomerulonephropathies indicate that MCP-1 plays a main role in progression of inflammatory processes in kidney diseases ***(Wagrowska-Danilewicz et al., 2005).***

MCP-1 is a chemokine that is thought to be responsible for monocyte and T lymphocytes recruitment in acute inflammatory conditions and may be an important mediator in chronic inflammation ***(Dong Qing, 2005).***

LN is a major cause of morbidity and mortality in SLE. As the course of LN is often unpredictable, it is important to identify reliable, noninvasive methods to repeatedly assess the condition of the kidneys in these patients ***(Schwartz., 2007)***.

The treatment of SLE nephritis, while effective, is associated with significant morbidity and mortality. These side effects could be mitigated if the onset, severity, and response of renal flare could be predicted, and therapy modified accordingly ***(Rovin, 2007)***.

Current disease markers include serum C-reactive protein and complement levels, antibodies to double-stranded DNA and proteinuria., lack both sensitivity and specificity for LN. Renal biopsy remains the gold standard for assessment of LN disease activity. Serial renal biopsies, however, are not appropriate in clinical practice. There is therefore an important unmet need for biomarkers that discriminate disease severity, assess response to therapy and more accurately predict disease relapses. These biomarkers would allow early implementation of appropriate treatments with the hope of preventing disease progression ***(Neeraj and David., 2009).***