Conclusions Serum CIC and IL-6 significantly correlated with clinical SLEDAI, which is higher degree of correlation than anti-dsDNA, C4 and C3 levels. Our study suggested that CIC and IL-6 can be used as alternative biomarkers to determine SLE activity.

Background and aims Patients with lysinuric protein intolerance (LPI) due to inherited defect of cationic amino acid transport in intestine and renal tubules may have aberrant immune responses leading to multiple organ involvement. The renal involvement with immune complex glomerulonephritis in LPI is albeit rare and has not been well established.

Methods We report a 4-year-old boy manifested nephrotic syndrome with renal histological findings showing immune complex glomerulonephritis highly suggested of lupus nephritis, but the initial serology survey excluded the diagnosis of SLE initially. The diagnosis of lysinuric protein intolerance was established and SLE developed 1 year later. Renal manifestations in patients with LPI and the coexistence of LPI with SLE are reviewed.

Results The initial renal involvement in LPI included renal tubular dysfunction, nephritic and nephrotic syndrome. During follow-up, some patients developed renal function impairment and may progress to end stage renal disease. Glomerulus was the major involved lesion with the most common histological finding was immune complex glomerulonephritis. Five patients, including our patient, with LPI coexisted with SLE have been reported during follow-up. These patients characterised female predominant, young onset age, predominant renal involvement, and poor prognosis. Our patient supported the suggested mechanism of macrophage activation. Treatment with steroid and cyclosporine accordingly led to remission of nephritis.

Conclusions LPI was not only a disorder of amino acid wasting but also a complex multisystemic disease with aberrant immune responses. LPI-associated glomerulonephritis shares similar characteristics on renal histology with lupus nephritis. Both macrophage activation and excess arginine accumulation might play roles on the pathogenesis.

Background and aims Lupus and lupus nephritis progression and flares are difficult to predict. Recently, osteoproteregerin, endothelin-1, CXCR3+/ CD4+ T cells and MCP-1 mRNA expression in urine sediments have been described as possible biomarkers of lupus and lupus nephritis. But their relationship with histological activity has not been sufficiently explored. It is desired that biomarkers of a disease should more rapidly reflect disease progression which would allow shorter clinical proof of concept trials and should be able to predict flares, measure current disease activity and severity, predict progression of disease and prognosis.

Methods It is likely that continued inflammatory events seen in lupus could be due to failure of the resolution of inflammation. Thus, the balance between inflammation and resolution is tilted more in favour of pro-inflammatory events and/or failure of production of pro-resolution molecules at the most appropriate time leading to non-resolution of inflammation. One such endogenous pro-resolution and anti-inflammatory molecule is lipoxin A4, whose deficiency could lead to continuation of inflammation in lupus and lupus nephritis.

Results It was noted that low plasma and urinary lipoxin A4 indicated disease activity and progression of disease, while a fall in its levels were noted prior to impending flares and increase in disease activity; and an increase in the levels of lipoxin A4 suggested resolution of inflammation and amelioration of disease process.

Conclusions It is suggested that measurement of plasma and urinary lipoxin A4 will be a good biomarker to predict flares, measure current disease activity and severity, predict progression of disease and prognosis.