A Case Study of Lupus Nephritis

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Abstract:

Clinical History: A 21 year old female K/c/o SLE since 1 year on steroids and methotrexate came with c/o skin pigmentation following on exposure to sun since 1 month and c/o generalized bodyache, multiple joint pain both small & large joints, having multiple mouth ulcer and generalized swelling all over the body, facial puffiness and edema of eyelids & frothy urine since 1 month.

Clinical Examination: Patient was conscious and oriented, normal temperature, pulse:62/min, BP:146/96mmhg, B/L air entry clear, CVS examination remarkable, pallor+, edema+, no cyanosis, clubbing, icterus.

Management: Patient was diagnosed as lupus nephritis grade 4 and 2 dose of inj cyclophosphamide 500mg iv and patient was kept on oral prednisolone 40mg, tab HCQ 200mg, tab ramipril 5mg and advised for regular follow up for inj. Cyclophosphamide for 6 months and plan to keep on maintainance dose of tab mycophenolate mofetil after 6 months.

Discussion: Lupus nephritis is the result of a type-III hypersensitivity reaction. This occurs when immune complexes are formed. Anti-double- stranded DNA (anti-dsDNA), an autoantibody, binds to DNA, which forms an anti-dsDNA immune complex. These immune complexes deposit on the mesangium, subendothelial, and/or subepithelial space near the glomerular basement membrane of the kidney. This leads to an inflammatory response with the onset of lupus nephritis, in which the complement pathway is activated with a resultant influx of neutrophils and the release of proinflammatory cytokines. The activation of the complement pathway results in low C3 and C4, which indicates active LN.