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**ANSWER: TUMOUR NECROSIS FACTOR ALPHA INHIBITOR (GOLIMUMAB) – INDUCED LEUCOCYTOCLASTIC VASCULITIS OF THE LOWER LIMB**

Figure 1: (a) Non-blanchable purplish lesions in the skin of lower extremity, (b) Histopathological examination of the skin lesion showing leucocytoclastic vasculitis as evidenced by dense perivascular neutrophil infiltration (Blue arrows) and fibrinoid necrosis (Yellow arrow). (H & E stain: 200x)

This will be the first reported case in Asia to date of golimumab-induced leucocytoclastic vasculitis. Spain had reported similar case in the past.<sup>1</sup> It has been postulated that the contributing mechanisms for such leucocytoclastic vasculitis include the Th2-driven antibody reaction on vascular endothelium favoured by tumour necrosis factor alpha blockade as well as the direct vascular wall insult by this drug.<sup>2</sup> In a recent golimumab efficacy and safety randomised controlled study (GO-RAISE) involving 355 patients with ankylosing spondylitis, none of the adverse reactions were due to cutaneous leucocytoclastic vasculitis like ours.<sup>3</sup>

Two months after discontinuing golimumab, patient developed overt albuminuria (3 gm/d) with preserved kidney function. Renal biopsy corresponded to crescentic IgA nephropathy in which he responded well to six monthly intravenous pulse of cyclophosphamide. The skin rashes never recurred after stopping golimumab and the patient's proteinuria has improved and remained stable till date.

Unique feature here is the rare association of spondyloarthritis with cutaneous vasculitis and IgA nephropathy. Golimumab here could have contributed to the development of cutaneous vasculitis. The IgA

nephropathy could represent a direct pathogenic role of IgA on renal system from the patient's underlying axial spondyloarthritis which is also predominantly IgA response.<sup>4</sup> Prompt recognition of vasculitic rash and routine monitoring of urinalysis are pivotal in this case for early treatment initiation and better outcome.

**REFERENCES**

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