re Steroids in the Treatment of Idiopathic Membranous Nephropathy

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Dear Sirs,

I regret that I found the Review by Garrattini and colleagues Nephron 45:1-6 [1987] depressingly unhelpful, because their distant view of the renal glomerulus only emphasises their statement that 'nephrologists and pharmacologists do not meet'. In particular I disagree with their statement that the immunological effects of glucocorticoids are unenlightening. If we move away from the boring and, in this context, useless subject of pharmacokinetics to consideration of the biochemical pharmacology of the corticosteroids, at least the scene is set for rational appraisal.

I discern the following relevant actions of steroids:

(1) In high dosage they suppress the phagocytic function of reticuloendothelial cells and thus the clearance of immune complexes.

They are said to reduce GBM permeability in inflammatory states ...this can now be checked.

They stop complement activation.

They stop the release of lysosomal enzymes from phagocytic cells.

They stop activation of phospholipase A2 [2], and thus they prevent the production of leukotrienes [3], thromboxanes, prostaglandins and platelet-activating factor [4]. In short, they have a very fundamental anti-inflammatory effect in acute situations.

They stop production of the control lymphokine interleukin I by mesangial cells. IL-1 is known to activate

local collagenase [5] and also to stimulate fibroblast proliferation [6]. Thus they have an important chronic anti-inflammatory effect.

I believe that some enlightened personalities will discern in my comments what should be the way forwards.

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