Comment on: Malignant hypertension and nephrotic range proteinuria without hematuria: IgA nephropathy

Sir,

I read with great interest the published article by Goswami *et al*., entitled "malignant hypertension and nephrotic range proteinuria without hematuria: IgA nephropathy (IgAN)". They presented a 39-year-old male who was admitted with sudden onset blurring of vision, generalized headache and vomiting. On examination, the patient had a blood pressure (BP) of 210/110 mm of Hg. Kidney biopsy was indicative of IgAN by significant mesangial IgA deposits and negative C1q deposits. According to Oxford classification, the morphologic lesions were as follow: M1, S1, E0 and T2.[1] In this article, I would like to emphasize a few points. Indeed, there was no reasonable etiology for malignant hypertension in this patient. There was no description of interstitial vessels, whereas various studies revealed the presence of thrombotic microangiopathy (TMA) in the background of IgAN, which is a factor for inappropriate high BP in IgAN.[2] Recently a retrospective study of 128 IgAN patients, from Paris revealed a 53% morphologic lesions of TMA, either acute or organized, in arteries and/or arterioles.[3] In a study on 136 kidney biopsies proved for IgAN, we found 2 patients had morphologic lesions of TMA and 10 patients had capillary tuft fibrinoid necrosis.[4,5] In fact, few publications existed regarding the presence of TMA in IgAN. However, it seems to be uncommon in the setting of IgAN and its significance, as a concomitant histologic finding, is unclear.[2,3] This morphologic lesion can be a reason for high BP in these patients. It is possible that IgAN has different presentation between different regions[2,3] and while the vasculopathy in IgAN is ill-understood, therefore its role on the aggravation of IgAN needs further investigation.

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