Collapsing glomerulopathy in renal allograft biopsies: A study of nine cases

Sir,

I have read with interest the article by Gupta *et al*. published in your esteemed journal.[1] It is an important contribution to the burgeoning literature on this subject, both in native kidneys and in renal allografts, from this region of the world. Although we have not systematically analyzed our data, our experience on collapsing glomerulopathy (CG) in renal allograft biopsies is, more or less similar, to the subject study. Moreover, we have, earlier, published our experience of 10 cases of CG in native renal biopsies.[2] Although, in the later study, we studied the clinicopatholgical features of CG in native kidneys, most of our findings concur with the findings of the study by Gupta *et al*.[1] These studies will definitely contribute new dimensions to the growing etiopathogenetic spectra of CG. But, a note of caution is in order. One needs to be very scrupulous, accurate and consistent in presenting the findings on such rare lesions.

The authors aptly point out that the lesion of CG is distinct from focal segmental glomerulosclerosis (FSGS). Although, at present, CG is classified as a variant of FSGS, growing evidence suggests that this nosologic relationship of CG with FSGS may not last long and, sooner or later, the lesion will be classified separately from FSGS.[3,4] We are also curious as to whether the authors noted global capillary collapse in any of the biopsies. Autoimmune diseases are among the well-documented causes/associations of CG, and it is important to exclude such etiologies.[5]

There are some discrepancies in numbers at different places. The range of posttransplant duration (given in the abstract and results is 12-98 months, while in the accompanying table, the upper limit is 96 months), In the abstract, it is stated that all cases presented with graft dysfunction and severe proteinuria, which is different with regard to the severity of proteinuria in Table 1. The lowest percentage of glomeruli showing segmental collapse is 11.1%, as opposed to 12.5% given in the results. In the results, the range of follow-up duration is given as 3-12 months, as opposed to 2-12 months in Table 1. In the last sentence of the results, the serum creatinine given as 1.5-3.3 mg/dl is incorrect, as opposed to 1.4-2.1 mg/dl in Table 1.

The authors suggest that the favorable outcome of CG in their study might be due to lead time diagnostic bias. A shorter follow-up period may also be an additional factor. Moreover, three patients and not two had serum creatinine >3.4 mg/dl at the time of presentation.

Finally, the authors have not elaborated upon the etiologic factors underlying CG in their patients. In my view, vascular lesions may be contributory, as many of these patients had marked arteriolar hyalinosis. The vascular lesions with consequent ischemia are increasingly being implicated in the etiopathogenesis of CG in renal allografts.[3]

I hope the clarification of the above points will help in better understanding the increasingly recognized CG in both native and transplanted kidneys in the region.

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