A) COMENTARIOS A ARTÍCULOS PUBLICADOS

Comment on "IgM nephropathy in children: clinicopathological analysis"

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Dear Editor,

Arias et al.1 deserve compliments for sharing their experience on 13 children presenting with idiopathic nephrotic syndrome (INS) and diffuse dominant deposits of immunoglobulin M (IgM) on immunoflourescence (IF) study of renal biopsies. This pattern of immunohistochemical findings in combination with a variety of morphological lesions is popularly called IgM nephropathy (IgMN), the most controversial entity in the recent nephrology history.²⁻⁶ The article by Arias et al.1 is helpful in clarifying some of the controversies on the topic, but includes a small number of patients. I take this opportunity to emphasize some points about this disease.

The observed frequency varies among the studies.1-3 We,2 in particular, found a high frequency of 18.5% of IgMN in the largest study on children as compared to 5.17% observed by Arias et al.1 A close scrutiny of both studies will help explain this discrepancy. The denominator used to calculate the frequency of IgMN in our study comprised of biopsied children presenting with INS, whereas, Arias et al. used all biopsies for this purpose. A host of other factors also contribute to this phenomenon in different studies, such as disease definition, minimum threshold of IgM positivity used to define the disease, exclusion or inclusion of lesions of focal segmental glomerulosclerosis and so on.4

The authors used the term of minimal change disease (MCD) in Table 2 for

describing the pattern of minor glomerular alterations in IgMN. I think, it is better to avoid this term, as it denotes a definite disease entity. Instead, the term of, minor glomerular changes, be used to describe the above pattern of lesions in IgMN.

The authors did not give the minimum threshold of IgM positivity used to define the disease and the central measures±dispersion of follow-up period. Only minimum follow-up of six months is stated.

A number of discrepancies in the numbers of morphological patterns in results and figure captions and some other places are found.

In results, it is stated that hematuria was found in four patients, but in Table 1, it is present in seven patients. Similarly, high blood pressure is stated to be present in two patients, while in Table 1, it is found in three patients.

The caption of Table 3 reads as "Histological findings and treatment received". However, there are no histological findings in the table contents.

No units for serum creatinine in Table 1 and for creatinine and proteinuria at one year are given in Table 3.

Another point of ambiguity is the timing of the classification of treatment responses into, for example, corticoresistant or cortico-dependant. Whether it was done before performing the biopsy or at last follow-up? It needs clarification for a better understanding of the disease course.

Finally, it is heartening to note that western investigators have listened to our calls and a group has found in an experimental study in mice that IgM activates the complement system within the glomerulus and leads to

glomerulosclerosis.⁶ This represents a landmark study in the investigation of this disease. We hope that the same group will continue their efforts to elucidate the pathogenesis of the disease in near future.

Conflict of interest

The authors declare that there is no conflict of interest associated with this manuscript.

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