www.nephropathol.com DOI:10.12860/JNP.2013.41 J Nephropathology. 2013; 2(4): 258-259

Journal of Nephropathology

Comment on: IgM nephropathy: Can we still ignore it

Muhammed Mubarak [1,*]

[1] Histopathology Department, Sindh Institute of Urology and Transplantation, Karachi, Pakistan

ARTICLE INFO

Article type:
Letter to Editor

Article history:
Received: 8 May 2013
Accepted: 20 May 2013
Published online: 1 September 2013

Keywords:
IgM nephropathy
Nephrotic syndrome
Renal failure

Implication for health policy/practice/research/medical education:
IgM nephropathy (IgMN) is still a largely controversial entity in the domain of primary glomerulopathies. It was initially identified and characterized by Western researchers in late 1970s. More recently, the disease is being increasingly reported from the developing countries. The etiology and pathogenesis of the condition are still largely unknown. More research is needed to address these unresolved issues of the disease.

Please cite this paper as: Mubarak M. Comment on: IgM nephropathy: Can we still ignore it. J Nephropathology. 2013; 2(4): 258-259, DOI: 10.12860/JNP.2013.41

Letter to editor

Sir,

The recently published review on IgM nephropathy by Prof. Vanikar in your esteemed journal is a timely contribution on this subject (1). The author's group has previously published its experience on this disease in India and seems to be in a good position to share their views on this disease (2, 3). The author has succeeded in bringing forth some of the important aspects of the disease. She has covered the epidemiology, pathology, clinical presentation and prognosis of the disease in sufficient detail (1). However, the description of pathogenesis of the disease, which is still largely speculative, suffers from some discrepancies. There is no dearth of studies on the epidemiopathological aspects of the disease, especially from developing countries, in near past. What are lacking are the mechanistic studies deciphering the pathogenetic pathways involved in the disease process. This is partly due to the relative apathy of investigators in the developed countries toward this disease entity. One notable exception is the group from Finland, which is continuously working on this subject and has published the longest follow-up study on IgMN in the world (4).

There are also some minor discrepancies in the article as following:

In epidemiology, reference 14 is stated to be related to the occurrence of IgMN in transplant biopsies, which is not correct (5). It actually represents the first study describing IgM deposits in patients with hematuria, well before the widely acclaimed studies by Cohen et al and Bhasin et al,

*Corresponding author: Prof. Muhammed Mubarak, Histopathology Department, Sindh Institute of Urology and Transplantation, Karachi, Pakistan. Tel: +9221 99215752, Fax: +9221 32726165, E-mail: drmubaraksiut@yahoo.com

which described this phenomenon in proteinuric conditions (6, 7).

There is discrepancy in the stated mechanisms of the disease as regards suppressor T- cells. In the abstract, it is stated that hyperfunctioning suppressor T-cells are responsible for this disease; whereas in the pathogenesis section, down-regulation of these cells is described as an integral factor in causing this disease.

It is worthwhile to point out here that there are several issues relating to this disease that need more attention of the nephrology and nephropathology community worldwide (8). The definition of the disease and the diagnostic criteria, especially the threshold of immunofluorescence (IF) intensity, need to be defined objectively and precisely, so that inter-observer variation in the diagnosis of the condition is minimized. For example, the author of the subject article has advocated IgM positivity of ≥2+ on IF. Many authors, however, have included cases with trace positivity of IgM in this category (9). The pathogenesis of the disease needs more attention, especially from the researchers in the developed countries. In this context, researchers from developing countries can collaborate with their counterparts in the developed nations (9). A morphological classification with special focus on its prognostic significance is also badly needed. The Oxford classification of IgAN can serve a useful model to emulate (10). Last, but not the least, randomized controlled trials are needed to determine the optimal therapeutic approach for the disease. The review by Prof. Vanikar is a timely call for action by the concerned health care professionals and she needs to be complimented on this endeavor.

References

1. Vanikar A. IgM nephropathy: Can we still ignore it. J Nephropathology 2013; 2 (2): 98-103.
2. Singhai AM, Vanikar AV, Goplani KR, Kanodia KV, Patel RD, Suthar KS, et al. Immunoglobulin M nephropathy in adults and adolescents in India: a single center study of natural history. Indian J Pathol Microbiol 2011;54 (1):3-6.
3. Vanikar AV, Kanodia KV, Patel RS, Suthar KS, Patel HV, Gumber MR, et al. IgM nephropathy in India: a single centre experience. Indian J Pediatr 2012; 79(8):1025-7.
4. Myllymaki J, Saha H, Mustonen J, Helin H, Pasternack A. IgM nephropathy: clinical picture and long term prognosis. Am J Kidney Dis 2003;41 (2):343-50.
5. van de Putte LB, DeLaRiviere GB, Van Breda Vriesman PJ. Recurrent or persistent hematuria, sign of mesangial immune-complex deposition. N Engl J Med 1974; 290:1165-70.
6. Cohen AH, Border WA, Glassock RJ. Nephrotic syndrome with glomerular mesangial IgM deposits. Lab Invest 1978;38(5):610-9.
7. Bhasin HK, Abeulo JG, Nayak R, Esparza AR. Mesangial proliferative glomerulonephritis. Lab Invest 1978;39(1):21-9.
8. Mubarak M, Naqvi R, Kazi JI, Shakeel S. IgM nephropathy in adults: A
clinicopathological study. IJKD 2013; 7(3):214-9.
9. Mubarak M, Kazi JI. IgM nephropathy revisited. Nephro-Urol Mon 2012;4(4):603-8.
10. Mubarak M. Oxford classification of IgA nephropathy: broadening the scope of
the classification. J Nephropathology. 2012;1(1):13-6.