Treatment of Idiopathic Membranous Glomerulonephritis

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Dear Sir,

We have read with interest the editorial by Garatini et al. [1] in the January 1987 issue of your journal.

It is interesting to note that after many years of dealing with idiopathic membranous glomerulonephritis we do not know how to treat it, and what is more frustrating, we do not understand the disease. At the recent meeting of the American Society of Nephrology held in Washington, D.C., there was a debate on how to treat idiopathic membranous glomerulonephritis. No treatment and steroids, with or without cytotoxic drugs, were discussed, and again frustration was the result of the meeting because no answer was arrived at.

The treatment of idiopathic membranous glomerulonephritis with these drugs is based on the good results obtained with them in the treatment of minimal change disease. However, the rationale of this is unclear due to the fact that these two diseases have probably very little in common. Membranous glomerulonephritis is thought to be caused by deposition of immune complexes (IC) in the basement membrane of the capillary wall of the glomerulus (in situ formation and/or deposition of circulating IC). Several studies have shown that removal of the antigen in the so called secondary membranous glomerulonephritis produces its resolution [2]. Idiopathic membranous glomerulonephritis is caused by the IC but the antigen is unknown. It may be that in some cases of idiopathic membranous glomerulonephritis the body is unable to clear the antigen. Though it is not clear why, it could be due to a deficiency in the antibody production (antigen in excess of antibody), production of low-affinity antibodies ('bad antibodies') and/or decreased activity of the reticuloendothelial system, in a word, a failure of the immune system to clear the antigen from the body. If this is so, then we should try to correct the deficiencies of the immune system to improve the rate of IC elimination instead of suppressing it even more. Obviously we would not like to increase the production of low-affinity antibodies (bad antibodies) because, if so, we would probably make the situation worse. However, if some cases of idiopathic membranous glomerulonephritis are due to a low production of antibodies and/or low activity of the reticuloendothelial system, the stimulation of either or both may cure the disease.

Several substances can stimulate the immune system, e.g., Bacillus Calmette-Guérin (BCG), levamisole, or thymic hormones [2] and we do not know which one is safer. However, we feel that some efforts should be directed to investigate what role the immune stimulation can play in the treatment of idiopathic membranous glomerulonephritis.

It may be risky but now may be the time to look for new answers to old questions.

References

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