J Nephrol Ther 2018, Volume 8
DOI: 10.4172/2161-0959-C2-055

Dissecting the relationships of IgG subclasses and complements in membranous lupus nephritis and idiopathic membranous nephropathy

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Membranous lupus nephritis (MLN) and idiopathic membranous nephropathy (IMN) are kidney diseases with similar morphology, but distinct etiologies, both producing glomeruli with immune deposits. Immunoglobulins and complements, the main components of the deposits, can be detected by immunofluorescence (IF) microscopy semi-quantitatively. Previous researches characterized the immune deposits only individually, but not the interactions between them. Experiments to identify the interactions are too complex, time-consuming and costly. Computational approaches using data visualization, pattern recognition, and statistical inference can be good alternatives to physical experiments. To study these interactions, we analyzed an IF profile of IgG subclasses and complements (IgG1, IgG2, IgG3, IgG4, C3, C1q, and C4) in 53 and 95 cases of biopsy-confirmed MLNs and IMNs, respectively, mainly using information theory and Bayesian networks. We identified significant entropy differences between MLN and IMN for all markers except C3 and IgG1, but mutual information (a measure of mutual dependence) were not significantly different for all the pairs of markers. The entropy differences between MLN and IMN, therefore, were not attributable to the mutual information. These findings suggest that disease type directly and/or indirectly influences the glomerular deposits of most of IgG subclasses and complements, and that the interactions between any pair of the markers were similar between the two diseases. A Markov chain of IgG subclasses was derived from the mutual information about each pair of IgG subclass. Finally we developed an integrated disease model, consistent with the previous findings, describing the glomerular immune deposits of the IgG subclasses and complements based on a Bayesian network using the Markov chain of IgG subclasses as seed. The relationships between the markers were effectively explored by information theory and Bayesian network. Although deposits of IgG subclasses and complements dependent on both disease type and the other markers, the interaction between the markers appears conserved, independent from the disease type. The disease model provided an integrated and intuitive representation of the relationships of the IgG subclasses and complements in MLN and IMN.

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