Hindawi

Disease Markers

Special Issue on

# Disease Markers for IgA Nephropathy

# CALL FOR PAPERS

IgA nephropathy (IgAN) is the most common form of primary glomerulonephritis worldwide and a frequent cause of end-stage kidney disease (ESKD). Approximately 40% of patients with IgAN progress to ESKD within 20 years from diagnosis, as there are no disease-specific treatment. The diagnosis of IgAN depends on performing a kidney biopsy and the inherent risks of this invasive procedure frequently delay the diagnosis. Thus, new strategies for early diagnosis of IgAN that would enable early and effective medical intervention are urgently needed.

Pathological analysis of renal biopsy tissue is the gold standard for diagnosis of IgAN as well as assessment of disease severity and prognosis. However, the findings may vary, depending on the time point of renal biopsy during the 20-year course of IgAN and previous medications. Thus, renal biopsy, when performed, provides only a snapshot in time and does not allow monitoring of responses to treatments or assessment of efficacy of new therapies in clinical trials. Therefore, simple and safe biomarkers are desirable.

It is now widely accepted that development and progression of IgAN depend on a multihit process, codependent on environmental and genetic factors. This process includes elevated production of aberrantly glycosylated serum IgA1 molecules (1st hit) that are recognized by IgG or IgA autoantibodies (2nd hit), leading to formation of nephritogenic immune complexes (3rd hit), some of which deposit in the kidney and induce glomerular injury (4th hit). Although our knowledge of IgAN pathogenesis expanded significantly since the disease discovery in 1968, we still do not have disease-specific therapy to prevent the progression of IgAN to ESKD. To develop a curative treatment, new strategies for early diagnosis, disease-specific targets, and methods for assessment of clinical responses in clinical trials need to be identified and developed.

In this special issue, clinical and experimental studies that emphasize a diagnostic, prognostic, and/or disease activity marker(s) for IgAN are preferable.

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