Biologic Treatment in Glomerular Disease

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The classification of glomerular disease has been based almost entirely on renal histology, but an increasing understanding of the pathogenesis is now introducing change relevant to the development of newer therapies. The introduction of corticosteroid and immunosuppressive drugs undoubtedly improved the outcome of patients with many forms of nephrotic syndrome, lupus nephritis and ANCA-associated vasculitis, but this non-specific treatment has been associated with serious short- and long-term toxicity, especially in patients with frequently relapsing disease. During the last 10 years, new biologic (targeted) treatment has expanded our therapeutic armamentarium, especially in refractory, relapsing or intolerant patients with ANCA-associated vasculitis, lupus nephritis and less commonly for other glomerular diseases.

This special topic section of *Nephron Clinical Practice* provides the reader with a comprehensive overview of the widening area of biologic treatments in an increasing number of glomerular diseases. The first paper [1] returns

to the standard immunosuppressive treatment demonstrating its limitations and the current unmet need of, at least, similarly effective and less toxic treatment. To illustrate the close connection between a better understanding of the pathogenesis of the disease and targeted treatment, the papers on the pathogenesis of ANCA-associated vasculitis [2], lupus nephritis [3], the role of anti-PLA2R antibodies [4] in membranous nephropathy and complement in membranoproliferative glomerulonephritis [5] precede the articles dedicated to the treatment of secondary [6–8] and primary glomerulonephritides [9–11]. The final contribution opens new horizons by reviewing the emerging biologic treatments [12].

We hope you enjoy this introduction to the complex area of the pathogenesis and targeted treatment of glomerular disease and that this information will contribute to greater awareness, earlier diagnosis and referral, better treatment and improved outcome for patients with immune-mediated glomerular diseases.

References

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