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# Pegcetacoplan: Results of the VALIANT Trial and Real-World Experience

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Complement-mediated glomerular diseases, particularly C3 glomerulopathy (C3G) and immune complex–mediated membranoproliferative glomerulonephritis (IC-MPGN), are increasingly recognized as progressive diseases driven by dysregulation of the alternative complement pathway. Historically, management relied largely on supportive care and empirical immunosuppression, but recent advances in complement-targeted therapy are changing the therapeutic landscape. A major challenge in clinical practice is that complement-mediated injury often begins early, even in patients with apparently mild clinical presentation. Preserved kidney function or low-grade proteinuria does not necessarily indicate benign disease, and delayed diagnosis may result in irreversible nephron loss before effective intervention can be initiated. The importance of early recognition was illustrated through several patient cases with diverse clinical presentations. One patient with persistent proteinuria experienced progressive deterioration despite delayed referral and conventional immunosuppressive therapy, highlighting the consequences of late diagnosis and delayed initiation of targeted treatment. Another patient with biopsy-proven C3G demonstrated repeated relapse and treatment-limiting toxicity from corticosteroids and mycophenolate mofetil, emphasizing the limitations of traditional immunosuppression. A pediatric patient identified through school urine screening initially presented with only microscopic hematuria, mild proteinuria, and preserved kidney function, but subsequently developed progressive disease confirmed as C3G on kidney biopsy. This case particularly underscored that apparently “mild” disease may still reflect active complement-mediated injury and progressive nephron loss. Accurate diagnosis requires a high index of suspicion, comprehensive complement and serologic evaluation, and timely kidney biopsy with detailed light microscopic, immunofluorescence, and electron microscopic assessment. Current KDIGO guidelines were developed before the emergence

of robust complement inhibitor trial data and therefore provide limited guidance regarding the use of complement-targeted therapy, treatment duration, or patient selection. Recent clinical trial data from the VALIANT study have significantly advanced the field. VALIANT was a randomized, double-blind, placebo-controlled trial evaluating pegcetacoplan, a C3 inhibitor, in patients with C3G and IC-MPGN, followed by an open-label extension phase. Patients treated with pegcetacoplan demonstrated rapid and substantial reductions in proteinuria compared with placebo, with consistent efficacy across multiple subgroups. Importantly, histologic analyses from follow-up kidney biopsies showed reductions in C3 deposition and features of active glomerular injury, providing tissue-level evidence that complement inhibition directly modifies the underlying disease process. Safety data demonstrated an acceptable infection profile during the randomized period, although long-term monitoring remains important in clinical practice. In summary, complement-mediated injury begins early and may progress silently despite preserved kidney function or mild proteinuria. Early recognition, timely kidney biopsy, and accurate diagnosis are essential to allow patients access to emerging disease-specific therapies. The treatment paradigm in C3G and IC-MPGN is rapidly shifting from nonspecific immunosuppression toward mechanism-based complement inhibition with the goal of preserving long-term kidney function.

**Keywords:** C3G, IC-MPGN, complement inhibitor