



Rituximab-induced hypogammaglobulinemia in nephrotic syndrome: what is the true burden?

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To the editor,

I read with great interest the article by Choi et al. entitled “Efficacy and safety of long-term repeated use of rituximab in pediatric patients with nephrotic syndrome” [1]. In this single-centre retrospective study, 50 children with nephrotic syndrome treated with repeated courses of rituximab were evaluated. The authors concluded that this treatment strategy was efficacious and safe.

Of note, only 8% patients in this study were reported to develop hypogammaglobulinemia. Indeed, hypogammaglobulinemia is a frequent complication following rituximab and can occur in up to 14–58% of children with nephrotic syndrome [2, 3]. While rituximab is used in various paediatric glomerular diseases, the risk of hypogammaglobulinemia appears to be higher in nephrotic syndrome owing to heavy proteinuria and immunosuppression use [3]. In addition, monitoring and definition of hypogammaglobulinemia were variable among previous trials. Consequently, it is difficult to accurately assess the incidence and significance of rituximab-induced hypogammaglobulinemia.

In the current study, the low rates of hypogammaglobulinemia may be accounted for by the fact that immunoglobulins were monitored only in 31% of patients. It is also unclear how frequently these patients were monitored,

whether concomitant immunosuppression was used, and if there was pre-existing hypogammaglobulinemia. Although the levels of immunoglobulin do not correlate closely with infection [2], these values would be of interest to the readers to understand the severity of hypogammaglobulinemia. In conclusion, immunoglobulin G, A and M should be monitored regularly before and after rituximab, and intravenous immunoglobulin replacement may be considered in selected symptomatic patients with hypogammaglobulinemia.

References

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