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Short Report

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Hyperimmune anti-CMV globulins and roxadustat for treatment of Parvovirus B19-associated anemia in kidney transplant recipients - A step ahead compared to intravenous immunoglobulins?

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Abstract

Parvovirus B19 (PVB19) may cause refractory and severe anemia in kidney transplant patients. Cellular tropism of PVB19 is associated with suppression of the red blood cell population, targeting erythroid progenitor cells, leading to persistent pure red cell aplasia with preserved leucocytes and platelets. Most PVB19 infection cases occur within three months posttransplant [1]. Although there is no specific treatment for PVB19, reduction of immunosuppression and Intravenous Immunoglobulins (IVIG) are used in clinical practice [2].

Introduction

Hyperimmune anti-CMV globulins (CMV-HIG) contain high titers of antibodies against CMV but also against other pathogens. Compared to the polyspecific IVIg products, CMV-HIG formulations exhibit higher CMV binding activity with 4-fold higher neutralizing activity [3]. Its activity against PVB19 is unknown [4]. There is no data on the treatment of PVB19-associated anemia with CMV-HIG and roxadustat. We have treated two patients with early onset PBV19 with CMV-HIG 2 ml/kg in three doses in combination with roxadustat. Both received mycophenolate, tacrolimus, and steroid maintenance.

Case report

A 50-year-old male who received a renal allograft from a deceased donor in June 2022 developed severe anemia 9 months post-transplant with serum hemoglobin 58 g/L unresponsive to erythropoietin treatment. He was admitted to the hospital and was dependent on erythrocyte transfusions. The evaluation revealed PVB19 infection (3,77x10^{10} IU/mL). Mycophenolate was stopped, and he received CMV-HIG 2 ml/kg in three doses in combination with roxadustat, which resulted in rapid recovery. Three weeks later, his serum hemoglobin was 88 g/L, and two months later, 106 g/L, with a decline in viral load (PVB19 2,99x10^{6} IU/mL) and stable allograft function.

Another patient was a 48-year-old male who developed anemia four months after the transplantation. His serum hemoglobin was 73 g/L and was not responsive to erythropoietin treatment. He was diagnosed with PBV19 infection (9,24x10^{10} IU/mL) and treated with immunosuppression reduction, CMV-HIG 2 ml/kg in three doses in combination with roxadustat. Three weeks later, his serum hemoglobin was 121 g/L. Two months later, PVB19 decreased to 436000 IU/mL with serum hemoglobin 142 g/L without roxadustat. His allograft function remained stable. Mycophenolate was reintroduced in both patients after recovery from severe anemia and continued in half of the initial dose. These cases show that CMV-HIG may exert action against the PVB19. This may be the result of the neutralization of the virus but also of the immunomodulation.

Conclusion

In conclusion, treatment with hyperimmune anti-CMV globulin in combination with roxadustat may represent a novel option for treating resistant PVB19-associated anemia in kidney transplant recipients. The rarity of PVB19 limits the conduction of prospective trials to assess the optimal dose and duration of treatment.

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