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Commentary

Anemia Following Kidney Transplantation Caused by Parvovirus Infection

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DESCRIPTION

Anemia following kidney transplantation is a significant problem that has been linked to more hospitalizations and a higher fatality rate. Depending on the parameters used to diagnose anaemia, it is anticipated that 20–50% of patients may experience post-transplant anaemia. Anemia normally goes away three to six months after transplant in patients with healthy renal allografts. Early post-transplant anaemia is probably the result of a complicated process, and determining the underlying reason is essential to selecting the best course of treatment. Three categories may be used to group the most frequent causes of early post-transplant anaemia: insufficient erythropoietin, a lack of iron, or reduced erythropoiesis.

The latter is typically related to immune-suppressive therapy-induced bone marrow suppression or infections, particularly viral ones. Aplastic anaemia can be brought on by a number of viruses, including parvovirus B19, CMV, BK virus, Epstein Barr Virus (EBV), hepatitis B, hepatitis C, and HIV. Following renal transplantation, acute anaemia is brought on by parvovirus B19 infection. Coinfections with other viruses have the potential to exacerbate anaemia, even though parvovirus B19 is most likely the primary culprit in the aetiology of anaemia.

There have been case reports of parvovirus B19-related anaemia in solid-organ transplantation, but none of these dual viral infections with parvovirus B19 and BK causing severe anaemia. One of the rare occurrences of concurrent parvovirus B19 and BK viremia-related acute early post kidney transplant anaemia has been documented in this case. One month after receiving a kidney transplant, the patient's hemoglobin levels were normal. Three months later, however, they suddenly dropped sharply. Early laboratory tests showed reticulocytosis, which was caused by low-grade hemolysis, which contradicted the hypothesis of an anaemia caused by an infection. A quantitative PCR for serum parvovirus B19 DNA was submitted since the patient did not respond to packed cell transfusion or poetic alfa, and the results were extremely positive at more than 100 million copies. The lack of an explanation for the cause of the low-grade hemolysis may have delayed the identification of parvo viremia. Yet, with Intravenous Immunoglobulin (IVIG) therapy and a decrease in immunosuppression, the patient's anaemia totally disappeared. It is still unknown which virus the polyomavirus or the parvovirus caused the anaemia. The exact process through which BK infection results in anaemia is not understood. However, in a study describing the mechanism of polyomavirus-induced myeloproliferative disease in BKV infected mice; the authors proposed that anaemia was caused by the production of cytokines and growth factors by BKV-infected cells, which led to polyclonal proliferation of one or more hematopoietic cell types. Given that the plasma polyomavirus PCR shows only 6,234 copies of the BK virus, which is unlikely to result in kidney failure or other urological issues, it can be assumed that the parvovirus infection was the primary cause of the anaemia. The methods through which viruses induce haematological abnormalities are poorly understood. Nevertheless, a number of explanations have been put out, including direct infection of hematopoietic progenitor cells and the creation of inhibitory cytokines, which affect cell function and reduce the synthesis of hematopoietic components. Parvovirus B19 infection can be acquired by kidney transplant patients either through the donor graft or by the reactivation of an endogenous latent or chronic infection after immunosuppression. Anemia and parvovirus B19 infection were statistically correlated, according to a retrospective assessment by Capenko et al. After a donation, anaemia often doesn't appear for seven weeks. By causing RBC precursor apoptosis, parvovirus B19 multiplies in erythroid precursors and produces RBC aplasia. The virus encodes a nonstructural protein in addition to the two structural proteins Viral Protein (VP1) and Viral Protein (VP2) (NS1). It has been well established that the blood group P-antigen, which is highly expressed on erythroblasts, is the site of the virus's binding. The virus kills erythrocyte progenitors by inducing apoptosis through the (nonstructural) NS1 protein-caspase pathway after attaching to P-antigen. About 90% of positive cases were recorded during the first year following kidney transplantation. Immunosuppression is a key risk factor for parvovirus B19 infection, as seen by the resolution of anaemia when immunosuppression is reduced. Moreover, research has revealed that, as compared to basiliximab, induction treatment with antithymocyte globulin carries a higher risk of parvovirus B19 infection. Immunosuppression reduction should be the first line of treatment for parvo viremia since it will enable the immune

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system to develop particular protection against parvovirus B19. The IVIG therapy now used to treat parvovirus B19 is a result of this observation.

CONCLUSION

IVIG includes antigen-specific antibodies to a variety of possible pathogens, which may offer a degree of passive immunity and help cure viral infections acquired after organ donation, such as parvovirus B19, BK virus, and Epstein Barr virus (EBV).

Parvovirus-related anaemia in patients with renal transplants might be worse by concurrent viral infections.

This highlights the significance of early detection and treatment of this potentially fatal condition.