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Case Report

Parvovirus B19-induced aplastic crisis in a kidney transplant recipient

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Abstract

Introduction: Parvovirus B19 infection is a rare, but important cause of severe anemia in kidney transplant recipients.

Case presentation: We present the case of a 32-year-old male with end-stage kidney disease undergoing living-donor kidney transplantation and subsequently developing profound anemia. Our case emphasizes the heightened risks of developing infectious complications among kidney transplant recipients due to their intense immunosuppressed state and the diagnostic challenges parvovirus B19 presents in this population. In this context, maintaining a high index of clinical suspicion in recipients presenting with severe, erythropoietin-resistant anemia and reticulocytopenia is essential in ensuring a timely diagnosis.

Conclusions: Adopting a tailored, multidisciplinary approach that balances the risks of acute rejection with the need to reduce immunosuppression to promote viral clearance, is paramount to improving hematological and allograft outcomes.

Key words: kidney transplantation; parvovirus B19; severe anemia; Case Report.

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Introduction

The etiology of post-transplant anemia is heterogenous, depending on the time following transplantation [1–9]. Early post-transplant anemia, defined as occurring within the first three months post-transplantation, is mainly associated with surgical complications, recurrent phlebotomy, overall lower hemoglobin targets in the end-stage kidney disease population, inadequate post-transplant erythropoiesis and iron deficiency, specific donor characteristics and allograft dysfunction, as well as recipient characteristics including age and gender [3–8]. Anemia in the immediate post-transplantation period resolves within two to three months due to the endogenous allograft erythropoietin production [3–8]. Persisting anemia, later than three months, is either iatrogenic related to immunosuppression and other medications; infectious complications including parvovirus B19, Ebstein-Barr Virus (EBV), Cytomegalovirus (CMV), BK Polyomavirus, Varicella-Zoster Virus (VZV), and bacterial infections such as tuberculosis and staphylococcal infections; allograft dysfunction; and other comorbidities [1–9].

Parvovirus B19 is a single-stranded DNA virus of the Parvoviridae family that primarily targets erythroid precursors, causing apoptosis and premature cellular death [2]. The infection is usually self-limiting in immunocompetent hosts, manifesting as erythema infectiosum (fifth disease) in children, or a flu-like syndrome, occasionally associated with arthropathy in adults [1]. Maternal infection during pregnancy has been associated with hydrops fetalis, intrauterine fetal death, and miscarriage [1]. It is an important causative agent of transient aplastic crisis, among patients with chronic hemolytic disorders [8].

However, it can lead to severe anemia in immunocompromised patients due to pure red cell aplasia (PRCA) [2,9]. Since the virus affects erythroid progenitor cells, it can lead to severe aplastic anemia, characterized by marked reticulocytopenia and resistance to erythropoietin [2,9]. The prevalence of parvovirus B19 infection among renal transplant recipients ranges between 1% to 10%, with most cases arising within the first year post-transplantation, when immunosuppression is most intense [10,11]. Management includes reduction of immunosuppression, administration of intravenous immunoglobulin (IVIG), and supportive care with blood transfusions [10,12–14].

We present a case of parvovirus B19 infection in a kidney transplant recipient, highlighting the diagnostic and treatment challenges faced in this population.

Case presentation

A 32-year-old male patient with a history of end-stage kidney disease (ESKD) secondary to IgA

nephropathy underwent living donor kidney transplantation. The immediate post-transplant period was notable for a hemoglobin level of approximately 9 g/dL, which was monitored but not aggressively treated at the time. The patient received anti-thymocyte globulin (ATG) as induction therapy and was maintained on standard triple immunosuppressive regimen consisting of tacrolimus, mycophenolate mofetil, and prednisone.

During a routine follow-up approximately two months post-transplantation, laboratory tests revealed severe anemia with a hemoglobin level of 3.9 g/dL, unresponsive to erythropoietin therapy. The patient was admitted to our department for further evaluation and management. At presentation, renal function was normal with a serum creatinine of 1.13 mg/dL and blood urea nitrogen of 33 mg/dL. Urinalysis showed no abnormalities.

A comprehensive workup for anemia was performed, including hemolysis parameters, nutritional studies, hemoglobin electrophoresis, and bleeding studies including gastroscopy and colonoscopy, all of which were negative. Complete blood count showed severe anemia with markedly reduced reticulocyte count (0–1%), suggesting bone marrow failure specifically affecting the erythroid lineage. Bone marrow aspiration confirmed the presence of aplastic anemia with a paucity of erythroid precursors, consistent with PRCA.

Bone marrow aspiration (Figures 1 and 2) revealed a myeloid/erythroid (M/E) ratio of 36/1 and complete suppression of the erythroid lineage. The erythroid precursors were noted to be giant proerythroblasts with visible, large nuclei and a basophilic cytoplasm with presence of pseudopods, highly suggestive of a viral etiology.

In light of these findings, and the clinical presentation of PRCA in an immunocompromised host, parvovirus B19 infection was suspected. Serological testing revealed positive parvovirus B19 IgM antibodies (1.6 U/mL), confirming acute viral infection. Polymerase chain reaction (PCR) for viral DNA was not available at our institution at the time of diagnosis.

Treatment was initiated with reduction of immunosuppressive therapy, targeting trough levels between 3–5 mg and a 50% reduction of the mycophenolate mofetil dose, secession of valganciclovir` and trimethoprim/sulfamethoxazole, administration of intravenous immunoglobulin (IVIG) at a dose of 500 mg/kg/day for 5 consecutive days, and supportive care with blood transfusions. The patient received multiple units of packed red blood cells, resulting in a modest improvement in hemoglobin to 7 g/dL.

During hospitalization, a mild but progressive deterioration in renal function was observed, with

Figure 1. Bone marrow aspirate showing pronormoblasts with visible, large nuclei and basophilic cytoplasm with presence of pseudopods, as well as absence of late-stage erythroid precursors.

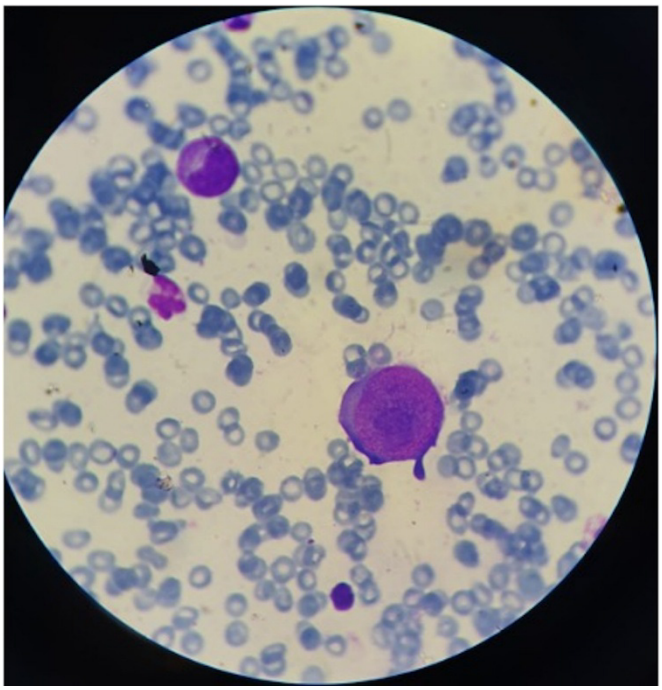
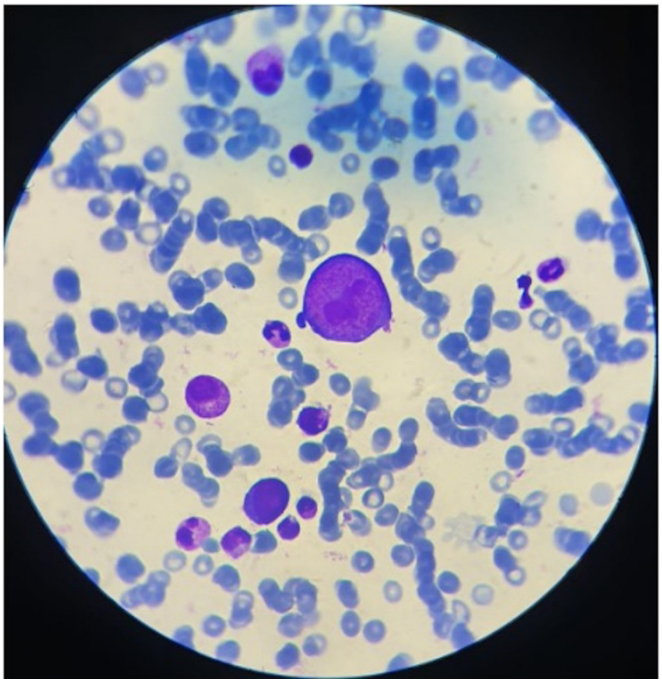


Figure 2. Bone marrow aspirate showing pronormoblasts with visible, large nuclei and basophilic cytoplasm with presence of pseudopods, as well as absence of late-stage erythroid precursors.



serum creatinine increasing from 1.13 to 1.39 and subsequently to 1.7 mg/dL, accompanied by mild proteinuria (75 mg/dL in spot urine). Following the reduction of immunosuppression, IVIG infusions and supportive care, both hemoglobin and renal function gradually improved with creatinine levels dropping to 1.07 mg/dL and hemoglobin levels increasing to 9.7 g/dL. The patient was discharged with recommendations of a close follow-up. A few weeks later he presented with recurrent anemia and underwent another round of IVIG treatment. His current hemoglobin levels remain stable.

Discussion

Parvovirus B19 can have several deleterious effects on kidney allografts through two primary mechanisms: direct viral invasion and immune complex–mediated injury [15–17]. The virus is hypothesized to exhibit a direct tropism for glomerular epithelial cells, particularly podocytes, leading to cellular damage, hypertrophy, and apoptosis [15–19]. This injury contributes to a spectrum of glomerulopathies, including collapsing glomerulopathy, focal segmental glomerulosclerosis (FSGS), and endocapillary proliferative glomerulonephritis [18,19]. Parvovirus B19 also targets glomerular and vascular endothelial cells, likely due to the expression of the P antigen on these cells [15,17]. The P antigen is the B19 receptor on erythrocytes, mediating the introduction of the virus into red blood cells and it is widely expressed in endothelial cells as well, facilitating their invasion [15,17]. This may potentially lead to acute glomerulonephritis and vascular damage [15,17]. Two mechanisms have been put forward to explain the endothelial injury and development of thrombotic microangiopathy (TMA), that is occasionally observed among patients infected with parvovirus B19: direct endothelial invasion and injury or immune complex deposition, which activates inflammatory cascades and results in endothelial damage and thrombosis [15,20]. The direct viral invasion can also affect tubular epithelial cells, causing acute tubular injury and acute kidney injury (AKI) [10]. This can be further exacerbated by interstitial edema, volume depletion associated with viral illness, and calcineurin inhibitor nephrotoxicity [10,11].

Finally, persistent viral replication due to impaired cellular and humoral immunity can lead to recurrent glomerular injury and progressive tubulointerstitial atrophy and fibrosis in immunosuppressed transplant recipients [10,11]. Additionally, several studies have postulated that as vascular endothelium acts as a crucial viral target, it may adopt antigen-presenting functions, upregulating MHC class II expression and promoting activation of adaptive humoral responses, contributing to the development of DSA-s and antibody-mediated rejection (ABMR) [21].

All these events may ultimately contribute to chronic allograft dysfunction.

Our case illustrates the significant challenge of diagnosing parvovirus B19 infection in kidney transplant recipients. The timing of infection approximately 2 months' post-transplantation aligns with current literature, highlighting the increased risk of infectious complications during the first 6 months following transplantation, owing to the intense immunosuppression during this period [10,11]. Symptomatic parvovirus B19 infection can occur as a de-novo infection, or reactivation of a latent infection due to profound immunosuppression [11,22]. Several studies have suggested that the graft could act as a source of viral transmission, leading to primary infections in susceptible recipients with serological mismatches or those receiving potent immunosuppression [11,22].

Indeed, this consideration was raised in our patient as mild anemia was noted immediately post-transplant with hemoglobin around 9 g/dL, suggesting either a latent or donor-derived infection that gradually progressed to severe PRCA as viral replication intensified under immunosuppression.

Diagnosis of parvovirus B19 infection should always be suspected in transplant recipients presenting with severe normochromic normocytic anemia refractory to erythropoietin therapy and marked reticulocytopenia (0–1%) [8,9]. Bone marrow findings of pure red cell aplasia and "giant pronormoblasts" with viral inclusions are characteristic and they were both evident in our case [8].

Diagnostic evaluation includes serological testing with elevated IgM levels indicating presence of infection, as was the case in our patient, as well as PCR detection of viral DNA [10,14]. Notably, immunosuppressed patients may be unable to mount an optimal humoral response to the infection, leading to false-negative serological results, in up to 29% of patients [10,14]. Consequently, PCR testing is now recommended as the gold standard for diagnosis in transplant recipients. Unfortunately, this was not available at our institution at the time of diagnosis [10,14].

Our treatment strategy consisted of a multipronged approach: cessation of medications potentially related to anemia, reduction of immunosuppression including a

50% reduction of mycophenolate mofetil, and lower target levels of tacrolimus ranging 3–5 mg. Nevertheless, reducing immunosuppression especially during the early post-transplant period is associated with higher rejection risks; therefore, immunological risk should be evaluated, and treatment approaches should be tailored accordingly. Additionally, we administered a 5-day course of IVIG (500 mg/kg/day) and supportive care with packed red cell transfusions. This approach reflects current best practices described in the literature [10,12–14]. Nevertheless, the optimal IVIG regimen both in terms of dosage and duration remains controversial. Several regimens have been proposed ranging from 400 mg up to 2 g/kg for 2–10 days, with varying success rates [13,14].

Despite therapy, up to 30% of patients may present with relapses of viremia with or without clinical evidence of anemia, and recurrent courses of IVIG may be necessary to control the infection [10,14]. In cases of persistent viremia and severe anemia despite optimal treatment and following several courses of IVIG regimens, a switch from tacrolimus to cyclosporin or an mTOR inhibitor have been proposed, with several studies confirming the success of this approach in controlling parvovirus B19 viremia and promoting viral clearance; however, this approach is not included in the official recommendations. [12,14,20–22] Several studies have suggested that there may be a causality between systemic infection and the use of tacrolimus, supporting the switch to a different immunosuppressor [2,12,22]. The mechanisms behind this success have not been fully elucidated, however it has been proposed that mTOR inhibitors, such as everolimus, may aid in neutralizing viremia due to their antiviral properties, ability to inhibit viral cell growth and improve memory T cell's functionality [2,12,22].

On the other hand, it is important to note that reduction of immunosuppression, a switch to an mTOR inhibitor-based regimen, and recurrent IVIG therapy may increase the risk of developing anti-HLA antibodies and acute rejection episodes [2,12].

In this context, a personalized approach tailored to the recipient's specific characteristics and immunological risk is essential for achieving effective viremia control and clearance, while minimizing the risk of allograft rejection and dysfunction.

Conclusions

Parvovirus B19 infection in kidney transplant recipients remains a rare but significant diagnostic and therapeutic challenge. Severe anemia associated with reticulocytopenia, unresponsive to erythropoietin therapy, should prompt further investigation to avoid delays in diagnosis. Management is complicated by the need to reduce immunosuppression to control viral replication, which must be carefully balanced against the heightened risk of allograft rejection. This case underscores the importance of considering parvovirus B19 as a potential etiology in transplant recipients presenting with unexplained, persistent anemia, and highlights the necessity of a tailored treatment approach that weighs both viral control and preservation of graft function.

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Conflict of interest

No conflict of interest is declared.

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