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Original Research Article

The outcomes of parvovirus B19 infection in kidney transplant recipients

Chilaka Rajesh[1*], Utkarash Mishra[1], Athul Thomas[1], Jeethu Joseph Eapen[1], Anna T Valson[1], Vinoi George David[1], Karthikeyan Jaganathan[2], Santosh Varughese[1]

[1]Dept. of Nephrology, Christian Medical College, Vellore, Tamil Nadu, India
[2]Dept. of Virology, Christian Medical College, Vellore, Tamil Nadu, India

Abstract

Background: Parvovirus B19 (PVB19) stands as a non-enveloped DNA virus known for its ubiquitous presence as a human pathogen, primarily transmitted via respiratory droplets. It affects immunosuppressed individuals like kidney transplant recipients who present with anaemia which is treatable. A high index of suspicion should be maintained in patients with Anaemia in the post-transplant phase, especially those who have received higher doses of immunosuppressants. Early diagnosis and appropriate intervention can minimize the negative impacts of the infection.
Aim and Objective: To study the clinical outcomes of Parvovirus B19 infection in kidney transplant recipients.
Materials and Methods: This is a single center, retrospective study of clinical outcomes of the Parvovirus B19 infection in kidney transplant recipients over a ten-year period (01 January 2002 to 31 Dec 2022) from a tertiary care hospital in southern India. The clinical data were obtained from electronic medical records of the department of Virology and outpatient department of kidney transplant clinic. The data was entered and results were analysed using SPSS software Ver 22.
Results: A total of 1802 patients underwent renal transplants at the study centre during 01 January 2002 to 31 December 2022. During this period, twelve patients (11 male and 1 female) were diagnosed to have Parvovirus B19 infection. The mean age of these patients was 36.8±12 years. Nine of the 12 patients developed the infection in the first year after renal transplantation with the mean duration of 6.5 ± 3.2 months. Two patients developed infection six years after transplantation and one patient 13 years after transplantation. All these patients presented with refractory anaemia in the post-transplant period with mean haemoglobin concentration of 5.37 ± 0.69 gm/dl. All these patients were similarly managed initially with a reduction of their immunosuppressive drugs but none of them showed response. They were then given 400 mg/kg/day intravenous immunoglobulin (IVIg) which contains protective immunoglobulins from many donors for five consecutive days. Two-thirds of the patients (eight of the twelve) responded to the IVIg while the remaining did not show any response even to a repeat dose of IVIg, they were treated with Inj. Rituximab at the dose of 375 mg/m^2 based on assumption that parvovirus infection triggered auto immune hemolytic anaemia in these patients.
Conclusion: PVB19 stands as a significant and treatable cause of post-transplant anaemia in renal allograft recipients. Vigilance for PVB19 infection in anaemic post-transplant patients, particularly those with heightened immunosuppression, is crucial for early intervention and minimizing adverse outcomes. While reduction of immunosuppression and IVIg therapy may suffice for many cases, refractory cases may necessitate innovative approaches like Rituximab, warranting further exploration through randomized controlled trials.

Keywords: Parvovirus B19, Kidney transplant Recipient, Refractory anaemia, Intravenous Immunoglobulin, Auto immune haemolytic anaemia, Rituximab.

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1. Introduction

Parvovirus B19 (PVB19) stands as a non-enveloped DNA virus known for its ubiquitous presence as a human pathogen, primarily transmitted via respiratory droplets. Its symptomatic or asymptomatic presentation varies based on host age and immunological status. Among immunosuppressed individuals like kidney transplant recipients, the course of infection manifests diversely.[1] While anaemia remains the prevailing symptom,[2] pancytopenia, hepatitis, myocarditis, and neurological complications may also arise.[3,4] PVB19 is an important treatable cause of anaemia in kidney transplant recipients. A high index of suspicion should be maintained in patients with anaemia in the post-transplant phase, especially those who have received higher doses of immunosuppressants. Early diagnosis and appropriate intervention can minimize the negative impacts of the infection.

*Corresponding author: Chilaka Rajesh
Email: dr_rajeshchilaka@yahoo.co.in

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2. Materials and Methods

This is a single center, retrospective study of clinical outcomes of the Parvovirus B19 infection in kidney transplant recipients over a ten-year period (01 January 2002 to 31 Dec 2022) from a tertiary care center in southern India. The clinical data were obtained from electronic medical records of the department of Virology and outpatient department of kidney transplant clinic. A case was defined as kidney transplant patient with refractory anaemia with positive Parvovirus serology or detection of Parvovirus by qualitative PCR technique. The data was entered and results were analysed using SPSS software Ver 22.

3. Results

A total of 1802 patients underwent renal transplants in tertiary care hospital, Southern India during 01 January 2002 to 31 December 2022. During this period, twelve patients (11 male and 1 female) were diagnosed to have Parvovirus B19 infection. The mean age of these patients was 36.8±12 years. In this study, all patients underwent live related kidney transplant. Ten patients received basiliximab as induction agent and two patients received Anti Thymocyte Glbulin (ATG) and Grafalon respectively. 11 out of 12 patients received maintenance immunosuppression with Prednisolone, Tacrolimus and Mycophenolate. One patient received maintenance immunosuppression with Prednisolone, Cyclosporine and Azathioprine. Two out of twelve patients (2/12) had history of Acute Antibody mediated rejection within three months of parvovirus infection. They received anti-rejection therapy in the form of Methylprednisolone pulse, six sessions of plasmapheresis and Rituximab. Nine of the 12 patients developed the infection in the first year after renal transplantation with the median duration of 6.5 ± 3.2 months. Two patients developed infection six years after transplantation and one patient 13 years after transplantation. All these patients presented with refractory anaemia in the post-transplant period with mean haemoglobin concentration of 5.37 ± 0.69 gm/dl. All the patients underwent detailed work of anaemia including bone marrow aspiration and trephine biopsy as a part of the evaluation of refractory anaemia. Nine patients showed features of pure red cell aplasia (**Figure 1**), whereas two patients had normal bone marrow and one had hypocellular marrow. All these patients were tested for Parvovirus infection. Until 2018, Enzyme-linked immunosorbent assay (ELISA) and chemiluminescence immunoassay (CLIA) techniques were in vogue for serological testing for parvovirus serology in our institute. From 2019, the Parvovirus infection was diagnosed by qualitative polymerase chain reaction (PCR) technique. All these patients were similarly managed initially with a reduction of their immunosuppressive drugs (fifty percent reduction of anti-metabolite drugs like Mycophenlate and Azathioprine). None of these patients showed response to reduction of immunosuppression alone. They were then given 400 mg/kg/day intravenous immunoglobulin (IVIg) which is made up of immunoglobulins (IgGs) from the plasma of many donors, for five consecutive days. Two-thirds of the patients (eight of the twelve) responded to the IVIg (**Figure 2**) while the remaining did not show any response even to a repeat dose of IVIg, they were treated with Inj. Rituximab at the dose of 375 mg/m^2 based on assumption that parvovirus infection triggered auto immune hemolytic anaemia in these patients. The auto immune hemolytic anaemia in these patients was diagnosed on the basis of positive Direct Coombs test and increased LDH. These patients had reticulocytopenia which is the characterstic feature of parvovirus infection. Two patients were responded to four doses of Inj. Rituximab 375mg/m2 given once weekly. (**Figure 3**) and one patient required only two doses Inj. Rituximab 375 mg/m2 given a week apart. One patient remained unresponsive to multiple schedules of IVIg and Rituximab and he is passed away after 8 months of parvovirus infection. All twelve patients had stable graft function with mean serum creatinine of 1.69±1.1 mg/dl. There was evidence of concurrent infections in some of these patients, viz. BK viraemia in two patients and treated with reduction of immunosuppression, cytomegalovirus (CMV) infection in two patients which was treated with oral Valganciclovir, and one patient had non-tuberculosis mycobacterial infection (Mycobacterium fortuitum) as anterior abdominal wall abscess. One patient in this cohort expired 5 years later with diagnosis of urosepsis with refractory septic shock. One patient had *E.coli* sepsis with refractory septic shock and expired 7 years of after kidney transplantation. The baseline characteristics were detailed in **Table 1** and **Table 2**.

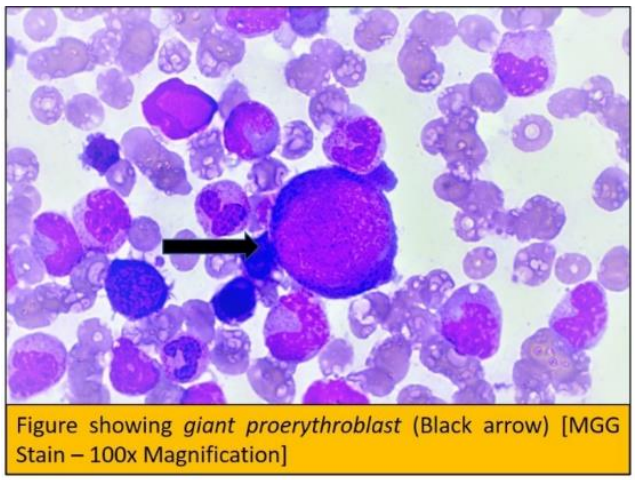

Figure 1: Bone marrow aspiration in Parvovirus infection

Table 1: Demographic, clinical characteristics, outcome of patients with parovirus B19 infection

Variable	**Patient**											
	1	2	3	4	5	6	7	8	9	10	11	12
Age, Years	55	60	26	38	32	27	24	37	48	40	31	24
Sex	F	M	M	M	M	M	M	M	M	M	M	M
Year of KT	2002	2009	2011	2014	2015	2016	2017	2017	2019	2019	2020	2022
Type of KT	Live	Live	Live	Live	Live	Live	Live	Live	Live	Live	Live	Live
Induction IS	Basiliximab	Basiliximab	Basiliximab	Basiliximab	Basiliximab	Basiliximab	Basiliximab	Basiliximab	ATG	Basiliximab	Grafalon	Basiliximab
Maintenance IS	Cyclo/Pred/ AZA	Pred/Tac/ MPA	Pred/Tac/ MPA	Pred/Tac/ MPA/Leflu	Pred/Tac/ MPA	Pred/Tac /MPA	Pred/Tac MPA-Stopped Luecopenia	Pred/Tac/ MPA	Pred/Tac/ MPA	Pred/Tac/ MPA	Pred/Tac/ MPA	Pred/Tac/ MPA
TAC trough (ng/ml)	-	7	4.4	6.4	6.3	6.1	7.1	5.2	3.0	5.8	10.8	6.8
MPA AUC (mg.h/L)	-	60	52.5	-	55.2	52.8	-	33	-	-	56.5	42.8
Onset of infection after KT	13 years	6 years	4 months	6 years	5 months	3 months	7 months H/o ART	4 months	11 months H/o ART	8 months	2 months	6 months
Clinical presentation	Anaemia	Anaemia & Flu symptoms	Anaemia & Flu symptoms	Pancytopenia	Anaemia	Anaemia	Pancytopenia	Anaemia	Anaemia & flu symptoms	Anaemia	Anaemia	Anaemia
Lowest Hb level (gm/dl)	6.0	5.0	5.2	5.8	6.0	5.0	6.0	5.0	6.2	5.8	4.2	3.8

Table 2: Demographic, clinical characteristics, outcome of paro virus B19 infection

Variable	**Patient**											
	1	2	3	4	5	6	7	8	9	10	11	12
Leucopenia	No	No	No	Yes	No	Yes	Yes	Yes	yes	No	No	No
Lowest WBC count	-	-	-	2400	-	4000	2300	3700	3700	-	-	-
Graft dysfunction	No	No	No	Yes (BKVN)	No	Yes (Sepsis induced)	No	No	No	No	No	No
PVB19 IgM	+	+	+	+	+	+	+	+	NP	NP	NP	NP
PVB19 PCR	NP	NP	NP	NP	NP	NP	NP	NP	Detected	Detected	Detected	Detected
Bone marrow findings	HBM	Normal	PRCA	Normal	PRCA	PRCA	PRCA	MEC	PRCA	PRCA	PRCA	PRCA
Treatment	IVIg +RIS	IVIg+RIS	IVIg+RIS	IVIg+RIS	IVIg+RIS	IVIg+RIS	IVIg+RIS	IVIg+RIS	IVIg+RIS	IVIg+RIS	IVIg+RIS	IVIg+RIS
IVIg total dose (gm)	2	2	2	2	2	2	4	2	2	4	8	8
IVIg dose mg/kg/day	400	400	400	400	400	400	400	400	400	400	400	400
Rituximab	No	No	No		No	No	**Yes**	No	No	**Yes**	**Yes**	**Yes**
Months after Improvement in haemoglobin	1	2	1	Passed away due to sepsis	1	-	4	1	1	4	3	Persistent anaemia and required multiple transfusions
Recurrence of PVB19 infection	No	No	No		No	Yes (at 5 years)	No	No	No	No	No	No
Years of follow up	5	5	7		No	Passed away (Sepsis)	5	5	3	3	3	Succumbed to illness

M- Male, F- Female, KT- Kidney transplantation, LD- Living donor, DD- Deceased donor, IS- Immunosuppression, ATG- Rabbit Anti thymocyte globulin, BKVN- BK Virus Nephropathy, Pred- Prednisolone, Cyclo- Cyclosporine, AZA- Azathioprine, MP- Methyl prednisolone, Tac- Tacrolimus, MPA – Mycophenolate, + or -, Positive or Negative, NP- Not performed, PRCA- Pure red cell aplasia, HBM- Hypocellular bone marrow, MEC- Megaloblastic Erythroid Cells, Hb- Hemoglobin, WBC- White blood cell, IVIg- Intravenous Immunoglobulin, PVB19- Parvovirus B19, RIS- Reduction of Immunosuppression.

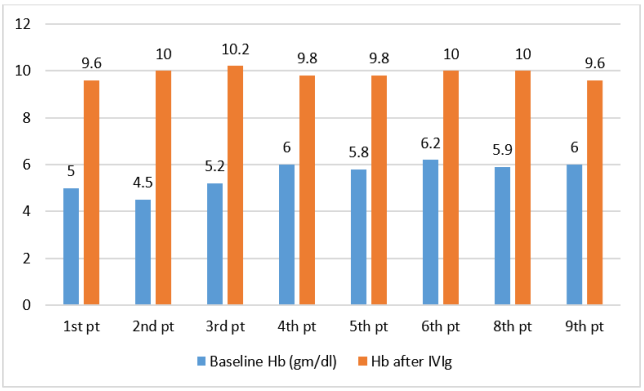

Figure 2: Hemoglobin trend in patients treated with IVIg

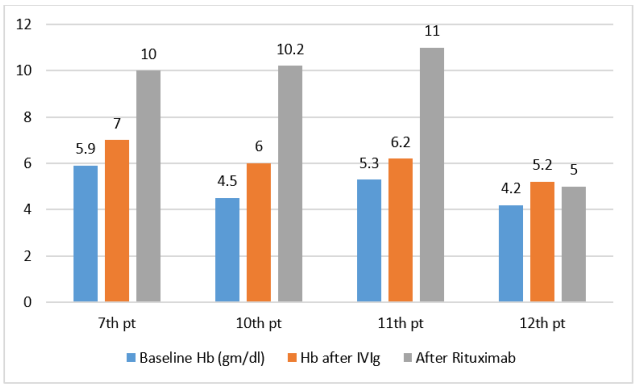

Figure 3: Hemoglobin trend in patients treated with Rituximab

4. Discussion

Although allograft dysfunction due to PVB19 is rare, it has been documented, possibly arising from donor-derived infection, as the virus can persist in seropositive individuals for years.[5] Managing PVB19 infection in kidney transplant recipients is challenging. It requires balancing the reduction of immunosuppression to control the infection and the risk of transplant rejection, especially in the early transplant phase.

Kidney transplant recipients are especially vulnerable to infections like PVB19,[7] due to the use of induction agents such as anti-thymocyte globulin (ATG) and alemtuzumab, as well as anti-rejection drugs. Nearly 40% of these recipients suffer from chronic anaemia, with some developing erythropoietin-resistant anaemia.[8] PVB19 targets CD36 erythroid progenitor cells in the bone marrow, disrupting red blood cell production. The virus binds to the P blood group antigen, found on red blood cells, their precursors, and other cells like endothelial cells, cardiomyocytes, and placental trophoblasts.[9]

Upon entering cells, the virus replicates, transcribes RNA, translates proteins, and assembles new virus particles in the nucleus. Electron microscopy can reveal virus particles in high concentrations within the nucleus. The cytopathic effect of PVB19 infection is evident in giant pronormoblasts in the bone marrow, with large nuclear inclusions, cytoplasmic vacuolization, and marginated chromatin (**Figure 1**). In immunocompetent individuals, the infection typically resolves with an antibody response. However, kidney transplant recipients often experience persistent pure red cell aplasia (PRCA), with normal white blood cell and platelet counts. PCR testing shows PVB19 positivity in about 23% of recipients with persistent anaemia.[9]

Immunosuppression is a major risk factor for infections in transplant recipients, as shown by improved anaemia when immunosuppression is reduced.[10] Induction therapy with ATG increases infection risk compared to non-depleting agents like basiliximab.[11] In our study, two patients received ATG for induction, and two had early acute antibody-mediated rejection within six months, requiring anti-rejection treatment as per protocol. Eleven out of twelve patients were on maintenance immunosuppression with Prednisolone, Tacrolimus, and Mycophenolate, while one patient was on Prednisolone, Cyclosporine, and Azathioprine. The mean Mycophenolic Acid Area under the Curve (MPA AUC) during PVB19 infection was 50.4 + 15.5mg.h/L. Four patients stopped MPA due to other infections and leucopenia, and one substituted Azathioprine for Mycophenolate.

PVB19 detection involves molecular techniques or antibody testing. IgM assays indicate recent infection[13] but may be unreliable due to delayed or inadequate antibody responses. PCR detects viral DNA in blood, bone marrow, and biopsy samples, even in asymptomatic individuals,[14] suggesting active infection. Histopathological features of PVB19 infection in kidney allografts include thrombotic microangiopathy and collapsing glomerulopathy.[15-18] Our patients were initially diagnosed with antibody detection by ELISA, transitioning to PCR-based diagnosis from 2019.

During PVB19 infection, no graft dysfunction was observed initially (mean serum creatinine 1.69±1.1 mg/dl), but some patients later developed dysfunction due to BK virus nephropathy or urosepsis, leading to fatalities in later stages.

There are no specific antiviral treatments for PVB19 infection. The American Society of Transplantation recommends Intravenous Immunoglobulin (IVIg) therapy which containts protective antibodies, along with reduced immunosuppression, if feasible.[19] Anaemia often responds to additional IVIg courses, and spontaneous resolution can occur with improved immunodeficiency. For refractory cases, Foscarnet may be considered, with positive outcomes reported in kidney transplant recipients.[20]

In our study, the initial treatment approach involved reducing immunosuppression, particularly anti-metabolite drugs. Three patients required cessation of Mycophenolate due to refractory infection. IVIg therapy was effective in 8 out of 12 patients (66.6%). However, four patients developed

autoimmune haemolytic anaemia (AIHA), marked by reticulocytopenia and erythroid hypoplasia in the bone marrow. PVB19 can trigger AIHA, as shown by elevated LDH levels and positive direct Coombs test results. Since Rituximab was used off-label for refractory autoimmune haemolytic anaemia and Epstein - Barr virus (EBV) infections,[21] it was administered at 375mg/m² to four patients, based on the assumption that PVB19 caused AIHA in these patients. Rituximab led to favourable outcomes in some patients, although one case remained refractory to IVIg and Rituximab, indicating persistent viremia and anaemia, which led to patient death. The anaemia improvement with IVIg and Rituximab is shown in **Figure 2** and **Figure 3**. Foscarnet could not be administered to non-responders due to financial constraints.

Parvovirus B19 infection is a key factor in autoimmune hemolytic anaemia (AIHA). Various infections, such as M. pneumoniae, EBV, measles, varicella, adenovirus, mumps, and rubella, can trigger AIHA. Most of these lead to the formation of cold agglutinins, characterized by IgM autoantibodies targeting the I/i polysaccharide antigen on red blood cells. However, some cases show reactivity with the P polysaccharide antigen.[22-25]

In addition to causing transient aplastic crisis in individuals with reduced red blood cell survival, PVB19 can also trigger AIHA. Bertrand *et al*. documented five cases of AIHA caused by acute PVB19 infection in otherwise healthy children.[26] Several cases of PVB19-induced AIHA associated with hemophagocytic syndrome have also been reported.[27-29]

PRCA linked to AIHA involves both humoral and cytotoxic immunity. One hypothesis suggests the presence of two autoantibodies targeting the erythroid series, with the dominant antibody determining the clinical manifestation. Both peripheral blood and bone marrow cells mediate a cytotoxic effect. Taniguchi *et al.* described two immunological mechanisms contributing to PRCA pathogenesis: one mediated by T-lymphocytes and the other by complement-dependent IgG suppression of erythroid progenitors. Both T-lymphocyte activity and autoantibody-mediated inhibition of erythropoiesis affect CFU-E and BFU-E levels.[30]

5. Conclusion

PVB19 stands as a significant and treatable cause of post-transplant anaemia in renal allograft recipients. Vigilance for PVB19 infection in anaemic post-transplant patients, particularly those with heightened immunosuppression, is crucial for early intervention and minimizing adverse outcomes. While reduction of immunosuppression and IVIg therapy may suffice for many cases, refractory cases may necessitate innovative approaches like Rituximab, warranting further exploration through randomized controlled trials.

6. Ethical Approval

This study had approval of institutional ethical committee vide minute number vide minute number 13641 dated 02.12.2020).

7. Source of Funding

None.

8. Conflict of Interest

None.

9. Acknowledgement

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