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Case Series

Post-Transplant Erythrocytosis in Kidney Transplant Recipients: Experience from a Single Tertiary Center

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ABSTRACT

Background: Post-transplant erythrocytosis (PTE), defined as haemoglobin >17 g/dL or haematocrit >51%, is an uncommon complication of kidney transplantation. Although frequently asymptomatic, untreated PTE may increase the risk of hypertension and thromboembolic events. RAAS blockade remains the mainstay of therapy.

Case Description: We report nine kidney transplant recipients who developed PTE within the first year post-transplant. Most were middle-aged, had stable graft function, received grafts from living donors. Clinical presentation ranged from asymptomatic erythrocytosis to mild hypertension or headache. Seven patients received ACE inhibitors or ARBs,two required phlebotomy. One patient remained stable without specific treatment. RAAS blockade consistently reduced haemoglobin, and all patients maintained preserved renal function. No thrombotic or neurological complications occurred during follow-up extending up to nine years.

Conclusion: PTE remains a manageable complication when recognised early. RAAS-based therapy provides reliable control;conservative management is adequate for stable, asymptomatic patients.

Keywords*: Post-transplant erythrocytosis; Kidney transplantation; RAAS inhibitors; Phlebotomy.*

INTRODUCTION

Post-transplant erythrocytosis (PTE) is a rare but clinically significant complication of kidney transplantation characterised by persistent elevation of haemoglobin or haematocrit after the correction of postoperative anaemia. In 2009 the Kidney Disease Improving Global Outcomes (KDIGO) group defined PTE as haemoglobin > 17 g/dL or haematocrit > 51 % [1]. Earlier definitions varied by threshold, sex and duration, which explains why prevalence estimates range from 2.2 % to 22 % [2,3]. PTE was first described in 1965 in a kidney transplant recipient [4]. Contemporary studies estimate that 8–15 % of kidney transplant recipients develop PTE and that incidence has fallen with greater use of angiotensin-converting-enzyme inhibitors (ACE-Is) and angiotensin-receptor blockers (ARBs) [5]. RAAS inhibitors became the cornerstone of therapy in the late 1990s because trials showed that captopril, enalapril and related drugs significantly lower haemoglobin and haematocrit in PTE. PTE usually develops within eight months to two years of transplantation [6].

Risk factors include male sex, retention of native kidneys, renal artery stenosis and an uncomplicated transplant course with good graft function. Recipients with robust pre-transplant erythropoiesis or polycystic kidney disease also show increased risk [7]. Pathophysiologically, PTE appears to result from persistent erythropoietin secretion by the allograft and native kidneys; renin–angiotensin system activation, endogenous androgens and insulin-like growth factor-1 further enhance erythropoiesis. Clinical manifestations range from mild malaise, headache and hypertension to serious

thromboembolic events such as stroke or pulmonary embolism. Untreated cases carry a mortality risk of 1–2 %, yet spontaneous resolution occurs in only about one-quarter of patients . Because ACE-Is/ARBs provide effective control, phlebotomy is reserved for refractory disease [8,9].

Although PTE is uncommon in the modern era, timely recognition and treatment are important. Further, this case series reports nine kidney transplant recipients who developed PTE at our institution and analyses their presentation and management to add to the limited literature on this complication.

MATERIAL AND METHODS

This retrospective case series included nine renal transplant recipients (of total 94 patients) who developed post-transplant erythrocytosis, and were followed at our tertiary care center. Clinical details were extracted from patient records using a standardized proforma capturing demographic characteristics, history of smoking, native kidney disease, dialysis duration, donor type, HLA mismatch, induction therapy, calcineurin inhibitor used, comorbidities, and post-transplant complications. PTE was defined as a sustained hemoglobin >17 g/dL or hematocrit >51% occurring beyond three months post-transplant, in the absence of dehydration, hypoxia, or other secondary causes. For each patient, serial hemoglobin, hematocrit, blood pressure, creatinine, symptoms, peak values, time to onset of PTE, transplant renal artery doppler and management strategies (ACE inhibitors, ARBs, phlebotomy, or immunosuppression modification) were documented, along with longitudinal follow-up values up to the latest available visit. Outcomes were analyzed descriptively to compare clinical presentation, treatment response, and temporal trends across cases.

Ethical Considerations

This study was a retrospective review of anonymized medical records of kidney transplant recipients. As per institutional policy, formal approval from the Institutional Ethics Committee was not required for retrospective observational studies using de-identified data. Patient confidentiality was strictly maintained in accordance with the Declaration of Helsinki

CASE DESCRIPTIONS

Case 1

A 29-year-old male with CGN underwent a live-related renal transplant from his 62-year-old father after 6 months of dialysis. He received rATG induction and tacrolimus-based immunosuppression with one HLA mismatch. PTE was first detected 3 months post-transplant, with Hb rising from 11.3 g/dL pre-transplant to a peak of 20.2 g/dL at 4 months. He remained asymptomatic and normotensive, with stable graft function (creatinine 1.3 mg/dL). Management included telmisartan 40 mg daily and two phlebotomies performed in the 4th and 5th months. Hematocrit gradually stabilized over subsequent months without escalation of antihypertensives.

Case 2

A 26-year-old male with CGN underwent live renal transplantation from his father after 12 months of dialysis. Basiliximab induction and tacrolimus were used, with 3/6 HLA mismatch and one treated rejection episode. PTE was detected around 10 months post-transplant, with peak Hb/Hct of 17.8 g/dL/52.1%. The patient remained asymptomatic, normotensive, and maintained acceptable graft function (creatinine 1.67 mg/dL). Enalapril 2.5 mg daily was initiated, resulting in gradual hematological normalization over a year, with no complications or need for phlebotomy. Hemoglobin stabilized to 14–15 g/dL during follow-up.

Case 3

A 24-year-old female, transplanted for suspected CGN after 3 years of dialysis, received a cadaveric graft with ATG induction and tacrolimus maintenance. PTE was detected approximately one year post-transplant, with Hb rising from 9.9 g/dL pre-transplant to 17.3 g/dL (Hct 53%). She remained asymptomatic, normotensive, and had good graft function (creatinine 1.2 mg/dL). Ramipril 2.5 mg daily was initiated for 1.5 months, resulting in a gradual decline of Hb/Hct over subsequent years. Long-term follow-up (2025) showed Hb 15.7 g/dL with stable blood pressure and no PTE-related complications.

Case 4

A 23-year-old female with CGN underwent a live-related transplant from her mother after 9 months of dialysis, receiving ATG induction and tacrolimus therapy. She developed PTE early post-transplant, with peak Hb/Hct of 16.9 g/dL/50.7% and associated hypertension. Her course required escalation of antihypertensives including nifedipine, telmisartan, and ACE inhibitors. Despite initial high values, hemoglobin progressively normalized over several years, remaining within 11–13 g/dL, with stable graft function (creatinine ~1.2 mg/dL). The patient experienced no thrombotic or neurological complications.

Case 5

A 42-year-old male with hypertensive nephropathy received a live transplant from his wife after 3 years of dialysis. He received rATG induction and tacrolimus, with 7/12 HLA mismatch. PTE developed with peak Hb of 17.9 g/dL, though he remained asymptomatic and normotensive (BP 120/80) with excellent graft function (creatinine 0.9 mg/dL). Management included losartan and later metoprolol due to rising hematocrit and hypertension. Serial monitoring over nine years showed fluctuating Hb (15–17 g/dL), requiring intermittent medication adjustments, but no complications occurred.

Case 6

A 38-year-old male with diabetic nephropathy underwent live renal transplantation using basiliximab induction and cyclosporine therapy. PTE was detected within the first year, with Hb 17.4 g/dL and Hct 52.5%. He was asymptomatic except for elevated blood pressure. The primary management was cyclosporine dose reduction, followed by telmisartan in later years. Hb/Hct gradually declined over follow-up, reaching 10–14 g/dL range by 2024–2025, with stable BP and no complications. Graft function remained acceptable (creatinine 1.9 mg/dL).

Case 7

A 21-year-old male with FSGS underwent cadaveric transplantation after 4 months of dialysis, using basiliximab and cyclosporine. He developed PTE within six months, with Hb peaking at 20 g/dL and Hct 61%. He had hypertension requiring amlodipine and later telmisartan. Two phlebotomies were performed due to markedly elevated hematocrit. Over the following year, Hb stabilized around 17 g/dL with controlled BP. No thrombotic or neurological complications were noted, and graft function remained stable (creatinine 1.8 mg/dL).

Case 8

A 26-year-old male with CGN underwent live transplant from his mother after 14 months of dialysis. Basiliximab induction and cyclosporine were used. PTE developed by four months post-transplant, with Hb 18.2 g/dL. The patient remained asymptomatic but required multiple antihypertensives pre- and post-transplant. Enalapril 2.5 mg daily for one month resulted in gradual reduction of Hb/Hct. Hematological values stabilized around 17 g/dL with good graft function (creatinine 1.18 mg/dL). No complications were reported.

Case 9

A 19-year-old female with FSGS underwent a live-related renal transplant from her 55-year-old mother after eight months of dialysis. She received no induction therapy and was maintained on tacrolimus, with 3/6 HLA mismatch and stable graft function post-transplant. PTE was identified at nine months, with haemoglobin rising from 10.5 g/dL pre-transplant to a peak of 16–19 g/dL (Hct up to 57%) over the first post-transplant year. She remained asymptomatic, with no history of headache, hyperviscosity symptoms, or thrombotic complications. Blood pressure ranged between 130–150/80–90 mmHg, and serum creatinine remained stable (0.8–1.3 mg/dL). As she was clinically stable, she did not receive ACE inhibitors, ARBs, or phlebotomy; management was conservative without escalation of antihypertensives. Follow-up over 12 months showed intermittent rises in Hb/Hct but preserved graft function, and PTE remained uncomplicated.

Table 1 : Post transplant erythrocytosis patients details, treatment and follow up

Case	Age/Sex	Native Disease	Donor Type	Induction	CNI	Peak Hb/ Hct	Symptoms	Complications	Treatment	Response
1	29/M	CGN	Live	rATG	Tac	20.2 / 60.3	Headache/ HTN	None	Telmisartan + Phlebotomy	Stabilised
2	26/M	CGN	Live	Basiliximab	Tac	17.8 / 52.1	None	None	Enalapril	Normalised
3	24/F	CGN	Cadaver	ATG	Tac	17.3 / 53	None	None	Ramipril	Sustained control
4	23/F	CGN	Live	ATG	Tac	16.9 / 50.7	Headache/ HTN	None	Nifedipine/Telma/ACEi	Long-term control
5	42/M	HTN nephropathy	Live	rATG	Tac	17.9 / —	None	None	Losartan ± Metoprolol	Mild fluctuations
6	38/M	DN	Live	Basiliximab	CyA	17.4 / 52.5	None	None	↓CyA + Telma	Gradual decline
7	21/M	FSGS	Cadaver	Basiliximab	CyA	20 / 61	HTN	None	Phlebotomy + Amlodipine	Stabilised
8	26/M	CGN	Live	Basilixi	Cy	18.2 /	None	None	Enalapril	Stable

				mab	A	—				
9	19/F	FSGS	Live	None	Tac	19 / 57	None	None	Conservative	Uncomplicated

DISCUSSION

Post-transplant erythrocytosis (PTE) remains an infrequent but clinically important complication of kidney transplantation. In this series, we retrospectively analyzed nine kidney transplant recipients who developed PTE, examining their presentations, management and outcomes to understand whether their course mirrored patterns reported in the literature. PTE has historically been defined as a sustained hemoglobin > 17 g dL^{-1} or hematocrit > 51% and affects roughly 8–15% of kidney transplant recipients [10]. Since use of renin–angiotensin–aldosterone system (RAAS) blockers became widespread, the incidence has declined from ~19% in patients transplanted between 1993–1996 to ~8% in those transplanted between 1997–2005 [2]. Our series of nine cases therefore represents a rare but contemporary snapshot of PTE in the RAAS-inhibitor era.

Clinically, the cases presented here largely conform to recognized risk patterns. PTE typically develops 8 months to 2 years post-transplant, and all but one of our patients developed erythrocytosis within 3–12 months after transplantation. Six of nine patients were male and six received kidneys from living related donors; both male sex and receipt of a well-functioning allograft from a living donor are established risk factors [11]. All patients had good graft function (serum creatinine around 1–2 mg dL^{-1}) and no acute rejection, consistent with reports that PTE is more common in recipients with excellent renal function and retention of native kidneys. None of our patients had renal artery stenosis, and no patient smoked or used diuretics, factors sometimes implicated in the literature [12]. Clinical manifestations in our cohort were mild: two patients presented with headaches or hypertension, while others were asymptomatic. Such variability mirrors prior observations that symptoms range from malaise and plethora to rare thromboembolic events [13].

Therapeutic response also aligned closely with published evidence. International guidelines recommend ACE inhibitors or angiotensin-receptor blockers as first-line therapy, because early clinical trials demonstrated that agents like captopril and enalapril significantly reduce hemoglobin and hematocrit [14]. All nine patients received RAAS blockade (telmisartan, enalapril, ramipril or losartan) and showed gradual declines in hemoglobin, supporting this recommendation. Only two patients required therapeutic phlebotomy, and none required theophylline or the removal of native kidneys. This limited need for phlebotomy is consistent with the British Society of Hematology guidance, which reserves phlebotomy for persistent symptoms due to a lack of evidence for a mortality benefit. Response to RAAS inhibitors in our series was durable; hemoglobin values stabilized between 14–17 g dL^{-1} within months, reflecting the sustained efficacy seen in older trials and more recent observational studies [15].

Outcomes in our cohort were uniformly favorable. None of the patients experienced thromboembolic events, neurologic sequelae or graft loss during follow-up periods up to nine years. Contemporary cohort studies similarly report that, with early recognition and treatment, PTE rarely leads to thrombosis or mortality. A multicenter study of deceased-donor recipients using updated World Health Organization criteria found no association between PTE and graft failure or death, and our experience supports this benign course. Nonetheless, earlier reports noted that thromboembolic complications occur in 10–40% of untreated patients, underscoring the importance of surveillance and prompt management [16,17].

This case series has limitations. The sample size is small and derived from a single center, limiting generalizability. Our retrospective design cannot determine incidence or independent risk factors. We also lacked detailed data on native kidney removal or erythropoietin levels, factors linked to PTE pathophysiology. However, strengths include standardized data collection, detailed longitudinal follow-up, and inclusion of diverse immunosuppressive regimens (tacrolimus versus cyclosporine). These cases therefore offer insight into current clinical practice where RAAS blockade is nearly universal.

CONCLUSION

PTE remains uncommon but clinically relevant after kidney transplantation. Early detection and RAAS-directed therapy ensure effective, sustained control. Our nine-patient series highlights its generally benign course, the safety of conservative management in select cases, and the importance of structured long-term monitoring.

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