A Case Report on Post-Transplant Erythrocytosis

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Introduction

- Persistently elevated haemoglobin and haematocrits levels that occurs following renal transplantation and persist for more than six months in the absence of thrombocytosis, leukocytosis or any other potential cause of erythrocytosis. [1]
- A haematocrits level greater than 51 percent in renal graft recipient have been generally used as a cut off value. [2]
- Occurs in 10-20 percent of the recipients of renal allografts, most often during the first 2 years following transplantation.

- Male gender, presence of native kidney, smoking, transplant renal artery stenosis, type of immunosuppressant used (more frequently in cyclosporine-treated patient), rejection free course with well-functioning renal graft and adequate erythropoiesis prior to transplantation.[3]
- Considering the relatively high incidence of PTE and its potential fatal outcome, within diagnosis and treatment would play an important role in preventing these complication.

Case report

- A 35-year-old non-smoker and non-diabetic male
- Hypertensive for 7 years and chronic kidney disease for 1½ years.
- He was on maintenance haemodialysis for 6 months in thrice weekly schedule.
- He was undergone a successful live related kidney transplant (LRKT) operation on January, 2018.
- Donor was his wife.

- His pre and post-operative period was uneventful.
- He received Inj. Basiliximab and Inj. Methyl Prednisolone as induction therapy and maintenance therapy with prednisolone, tecrolimus and mycophenolet mofetil.
- His daily urine output around 4-5 liters with normal renal function (serum creatinine level in between 0.8 to 1.21 mg/dl).

- During his routine follow up after 6 months, he was asymptomatic with normal urine output (2.5-3 lt/day) but haemoglobin and haematocrits were raised.
- He had no history of diarrhea, vomiting or polyuria and did not get any diuretic prior to this episode.
- His blood pressure was controlled (120/80 to 110/70 mm Hg) with AntiHTN drugs (Tab. Amlodipine and Tab. Prazosin).

- Last 3 follow up
- He showed persistently elevated haemoglobin level (18.1 to 19.4 gm/dl) with raised haematocrits (54.6 to 58.9%).

Level of haemoglobin, haematocrits and renal function

Date	Hb (gm/dl)	PCV (%)	TC of WBC (/cmm)	Platelet count (/cmm)	Urea (mg/dl)	Creatinine (mg/dl)
14/01/2018 (Pretransplant)	7.0	20.8	5400	100000	73	5.5
21/01/2018 (Post transplant)	8.0	23.6	7580	128000	45	2.7
29/01/2018 (During discharged)	9.7	28.9	8430	302000	32	0.8
02/02/2018 (1st follow-up)	13.3	30.2	14070	394000	32	1.1
04/03/2018 (2nd follow-up)	12.4	29.6	9040	276000	28	1.0
08/07/2018 (5th follow-up)	15.7	52	12370	232000	34	1.2
09/09/2018 (6th follow-up)	18.1	54.6	8600	306000	35	1.1
06/10/2018 (7th follow-up)	19.4	58.9	7950	221000	27	1.0
07/11/2018 (8th follow-up)	18.5	56	9060	211000	21	0.9

Other investigation:

- RBS- 5.4 mmol/lt,
- ALT 26 U/l, Bilirubin- 1.2 mg/dl,
- S. Tecrolimus levels-6.0 ng/ml (Normal value 5-10 ng/ml)
- Urine RME protein trace, pus cell- 0-2/ HPF, Epithelial cell- 0-2/HPF, RBC- 2-4/HPF,
- USG of KUB with prostate (with color Doppler) showed normal transplanted kidney with good vascular flow up to periphery.

Discussion

- Erythrocytosis following renal transplantation has been recognized since 1965 [5,6]
- The published prevalence of PTE in kidney transplant recipient ranges from 6.5 to 38.4%. [7]
- Patients with polycystic kidney disease (PKD) and glomerulonephritis were more likely to develop PTE. [11]
- Renal artery stenosis of the native or transplanted kidney was suggested as a risk factor for PTE in early case reports.[2,5]

- The following hormonal systems and growth factors have been implicated in the pathogenesis of PTE:
  - Erythropoietin
  - Hematopoietic growth factors such as insulin-like growth factor-1 (IGF-1) and its binding proteins and serum-soluble stem cell factor (sSCF)
  - Renin-angiotensin system (RAS)
  - Endogenous androgens.

- In the absence of treatment, PTE spontaneously remits in one-fourth of patients within two years from onset.
- In the remainder of patients, it persists for a number of years, after which it may remit in association with deteriorating renal function due to chronic rejection.[12]
- Approximately 60 percent of patients experience malaise, headache, plethora, lethargy, and dizziness.

- 10 to 30 percent develop thromboembolic events and may involve both veins and arteries and are manifested as thrombosis of digital or branchial arteries, thrombophlebitis, stroke, or pulmonary embolus.[13]
- 1 to 2 percent eventually die of associated complications if untreated and if erythrocytosis does not spontaneously remit. [13]

- The diagnosis of PTE is made by
  - Clinical features and laboratory finding of complete blood count with haematocrits.
  - Exclusion of common causes of
    - nontransplant-associated erythrocytosis, including malignancies
    - chronic obstructive pulmonary disease (COPD).

- Option of PTE treatment are as follows:
  - If hemoglobin concentration <18.5 g/dL- Angiotensin receptor blocker (ARB) or angiotensin-converting enzyme (ACE) inhibitor
  - If hemoglobin concentration >18.5 g/dL- Phlebotomy
  - Others
    - Theophylline
    - Antiproliferative agents such as azathioprine or mammalian (mechanistic) target of rapamycin (mTOR) inhibitors

Conclusion

- PTE should be borne in mind when kidney transplant recipient presented with high haemoglobin and haematocrits level. Early diagnosis and prompt action may reduce the complication and fatal outcome.

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Present Situation at BIRDEM

- 130 live related successful kidney transplantation done since 2004.
- This is the first case of PTE

THANK YOU