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HEMATOCRIT LEVELS IN PATIENTS WHO HAVE RECEIVED RENAL TRANSPLANTS - A RETROSPECTIVE STUDY

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ABSTRACT

Post transplant erythrocytosis is defined as persistently elevated hematocrit level ≥51%, or hemoglobin level ≥17gm%, following kidney transplant. The prevalence of PTE and response to treatment in a tertiary health care centre in North Chennai were assessed in this retrospective study. The medical records of 168 renal transplant recipients at Stanley Hospital, Chennai were reviewed. The age, sex, date of onset, duration of PTE, complications, treatment modalities and renal functions in PTE patients were reviewed. The treatment given was either Phlebotomy or medication with Angiotensin-converting enzyme inhibitor. Of the 28 patients who developed PTE, twenty were males and eight were females. 21 patients had received live related transplants and 7 had received cadaver transplants. Four patients were treated with Phlebotomies and 24 patients were given Angiotensin-converting enzyme inhibitor. Remission was seen in all patients. PTE is a benign condition not affecting graft function and responding to ACE inhibitor or Phlebotomy.

KEY WORDS: **Kidney transplant, Post transplant erythrocytosis, Post transplant polycythemia, Secondary erythrocytosis**

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INTRODUCTION

Post transplant erythrocytosis (PTE) is defined as a persistently elevated hematocrit (Hct) level equal to or greater than 51%, or a hemoglobin (Hb) level equal to or greater than 17 gm%, or both, in the absence of other causes. Post transplant erythrocytosis is a common condition which develops in kidney transplant recipients. PTE is reported to develop in 10-20% of kidney transplant recipients, mostly one to two years after transplant and may persist in patients with an increased risk for thrombosis. We retrospectively reviewed this complication in patients who had received renal transplant at Government Stanley Medical College Hospital, Chennai. The aim of the present study was to assess the prevalence of post-transplant erythrocytosis (PTE) and the response of PTE to treatment in a tertiary health care centre in North Chennai.

MATERIALS AND METHODS

This retrospective study was done in the Department of Nephrology, Government Stanley Medical College Hospital, Chennai. The subjects were 168 renal transplant recipients who had received kidney transplants from either cadaver or live related donors. The medical records of 168 renal transplant recipients attending regular follow-up at Stanley hospital, Chennai were reviewed. Patients with persistently elevated Hematocrit values equal to or greater than 51% or Hemoglobin levels equal to or greater than 17 gm%, or both, in the absence of other causes, on two or more consecutive hospital visits within a week, were confirmed to have developed Post Transplant Erythrocytosis. The age, sex, date of onset, duration of PTE, complications, treatment modalities and renal functions at the onset of PTE and after three months in all PTE patients, were reviewed. Of the 28 patients who developed PTE, four patients were treated with Phlebotomies, and 24 patients were given Angiotensin-converting enzyme inhibitor (T.Enalapril 10mg/day).

RESULTS

Out of the 168 kidney transplant recipients, 28 patients developed post transplant erythrocytosis, the prevalence being 16.6%. The sex distribution of the patients who developed PTE, is shown in Table 1.

Table 1
Sex distribution of patients who developed PTE

No. of patients who developed PTE (n=28)	
Males	Females
20	8

The type of renal transplant that PTE patients had received i.e. number of patients who had received live related transplants / cadaver transplants is shown in Table 2.

Table 2
Type of renal transplant received by PTE patients

No. of patients who received Live related transplant	No. of patients who received Cadaver transplant
21	7

The mean age of the patients who developed Post transplant erythrocytosis was 29.6 yrs.
Figure 1 shows the duration between kidney transplant and development of PTE, and the number of patients who developed PTE during that period.

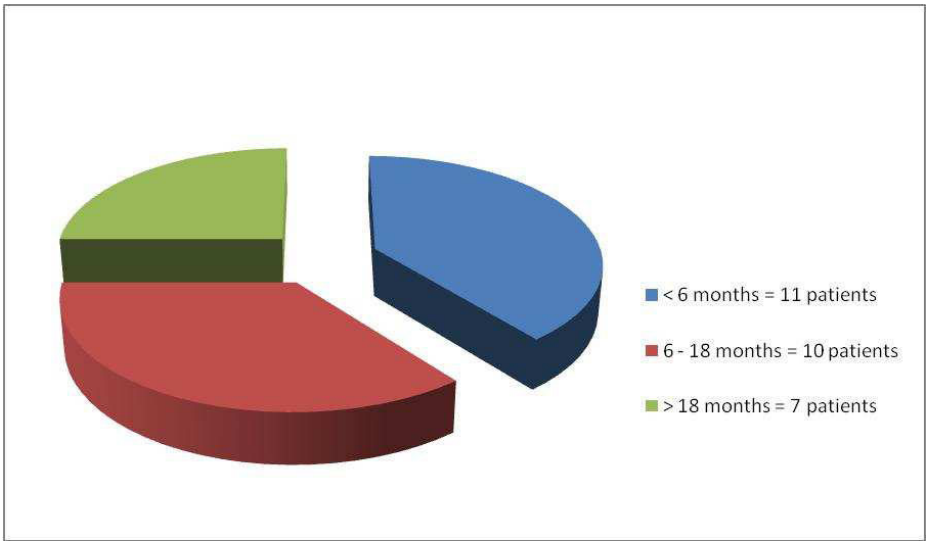

Figure 1
No. of post transplant recipients who developed PTE during various time periods

Average period of onset of PTE was 8.2 months after transplant.

Treatment given
Out of the 28 patients who developed PTE, four patients were treated with Phlebotomies and 24 patients were given Angiotensin-converting enzyme inhibitor (T. Enalapril 10mg/day). Remission of post transplant erythrocytosis was seen in all patients within four weeks of treatment. The mean pre-treatment Hct and mean post-treatment Hct are shown in Table 3.

Table 3
Mean pre-treatment and post-treatment Hct

Mean Pre-treatment Hct	Mean Post-treatment Hct
55.8%	45.3%

No thromboembolic complications occurred. Renal graft functioning was unaffected in study group.

DISCUSSION

A complete blood count can provide vital information about the internal environment of the organism [1]. The hematocrit (Hct) and hemoglobin (Hb) levels particularly assume importance in kidney transplant recipients. The normal Hct levels are 47% in males and 42% in females respectively and the normal Hb levels are 16 gm% in males and 14 gm% in females respectively [2]. PTE is clinically defined as a persistently elevated Hct level equal to or greater than 51%, or a Hb level equal to or greater than 17 gm%, or both, in the absence of other causes [3]. The prevalence of PTE varies between 10% and 20% of all renal transplant recipients [4,5]. In this study, PTE developed in 28 of 168 renal transplant patients, the prevalence being 16.6%, which is similar to the reported prevalence of other studies [6]. Male patients were affected more (97%) in our study, as seen in other studies [7]. PTE usually develops 8 to 24

months after successful renal transplant [3,8]. In our study, PTE appeared at an average of 8.2 ± 5 months after transplant.

Pathogenesis of PTE

Although not fully understood, the pathogenesis of PTE appears to be multifactorial. Considerable evidence points to the participation of at least three hormonal systems: the erythropoietin, the endogenous androgen, and the renin-angiotensin systems [9,10]. Erythropoietin overproduction has been found in renal transplant patients with PTE, which directly stimulates erythropoiesis. Some studies show that erythropoietin overproduction has been found to be ten times higher in renal transplant patients with PTE than in their counterparts with normal hematocrit values [11], but some other studies have shown that erythropoietin levels may be within normal limits[8]. Androgens exert direct dose-dependent stimulation of erythroid progenitors and can promote erythropoiesis indirectly via their stimulatory effect on endogenous erythropoietins or via renin-angiotensin system activation. This may explain the high prevalence of PTE in male patients, as reported previously [7] and as we have shown in our study.

Renin-Angiotensin II causes increased erythropoietin secretion. In vivo administration of renin or angiotensin II causes increased erythropoietin secretion[12]. Decreased concentrations of erythropoietin and Hct after ACE inhibition or angiotensin II receptor blockade have been observed in patients with PTE [13,14]. Ac-SDKP (goralatide) is a tetrapeptide that is a normal inhibitor of the entry of pluripotent stem cells into the S phase, thereby diminishing erythropoiesis. Ac-SDKP is metabolized by angiotensin converting enzyme; it may therefore accumulate in the presence of an ACE inhibitor [15-17]. In our study, 24 patients received ACE I or angiotensin II receptor antagonists, and their Hct levels decreased to within normal limits within 3 months after the onset of treatment. Thromboembolic complications develop in 10% to 30% of patients with PTE [4] but we did not observe any thromboembolic events in our study.

CONCLUSION

Post transplant erythrocytosis is a common benign condition seen in kidney transplant recipients. Male patients are affected more than female patients. The prevalence of PTE seen in our study is similar to the prevalence of similar studies. PTE does not affect graft function. It responds fairly well to Angiotensin converting enzyme inhibitor or Phlebotomy.

CONFLICT OF INTEREST

Conflict of interest declared none

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