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Original article

Impact of Anemia on Kidney Transplant Outcome

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Abstract

Introduction. Post-transplant anemia (PTA) is a frequent complication of kidney transplantation (KTx), but it remains unclear whether anemia diagnosed three months after KTx independently predicts deterioration of renal graft function one year after transplantation.

Methods. This retrospective-prospective longitudinal observational single-center study included 95 kidney transplant recipients with complete one-year follow-up. PTA was defined as a hemoglobin (Hb) concentration <110 g/L three months after transplantation, and re–cipients were divided into two groups according to the presence of PTA. Deterioration of graft function was defined as a serum creatinine increase ≥25% or an estimated glomerular filtration rate (eGFR) decline ≥30% at the 12-month compared with the 3-month post-transplant time point.

Results. PTA was present in 27.37% of recipients and was significantly more frequent in women than in men (42.86% vs. 20.90%; p=0.029). Recipients with PTA had significantly lower Hb, hematocrit, and red blood cell counts at all post-transplant time points and significantly lower eGFR at one-year post-transplant compared with recipients without PTA. A serum creati–nine increase ≥25% (23.08% vs. 4.35%; p=0.005) and an eGFR decline ≥30% (19.23% vs. 1.45%; p=0.001) were significantly more frequent in the PTA group. In multivariable regression analyses, PTA independently predicted serum creatinine increase ≥25% (OR 8.485, 95% CI 1.111-64.83) and an eGFR decline ≥30% (OR 27.54, 95% CI 2.28-340.4). Furthermore, deceased-donor transplantation independently predicted a serum creatinine increase ≥25%.

Conclusions. PTA independently predicts deterioration of renal graft function one year after transplantation, and can be helpful in identifying recipients who wa–rrant closer surveillance of the graft function.

Keywords: anemia, graft function, hemoglobin, kidney transplantation, outcome, risk factors

Introduction

Chronic kidney disease (CKD) is estimated to affect 843 million people worldwide and represents a major and growing public health problem, responsible for approximately 1.5 million deaths annually, corresponding to one death every 20 seconds [1]. Kidney transplan–tation (KTx) is the treatment of choice for end-stage kidney disease (ESKD) and, compared with dialysis, is associated with a substantially lower long-term risk of death [2].

Despite continuous advances in surgical technique and immunosuppressive therapy, long-term preservation of graft function remains a major challenge [3], and post-transplant complications contribute substantially to morbidity and mortality in this population [4]. Among hematological complications, post-transplant anemia (PTA) has attracted increasing attention because of its frequency and clinical relevance [4].

PTA has frequently been assessed in the early post-transplant period [5-7], and a hemoglobin (Hb) concen–tration <110 g/L three months after transplantation is an established definition adopted to identify early PTA with potential impact on graft function [5,6]. In a re–cent meta-analysis of studies using various definitions, the median prevalence of PTA was 59.8% within the first three months after transplantation, decreasing to 27.2% between the second and third post-transplant year [8]. PTA typically follows a biphasic pattern: early PTA develops within the first three months after transplantation and is favoured by pre-existing anemia, perioperative blood loss, iron deficiency, and delayed graft function (DGF), whereas late PTA develops thereafter and largely reflects declining graft function and impaired erythropoietin production, with chronic inflammation, infections, and the myelosuppressive

effects of immunosuppressive and other drugs contributing in both periods [4,8].

Pathophysiologically, reduced Hb concentration leads to chronic hypoxia of the renal graft, which may promote interstitial fibrosis, tubular atrophy, and progressive loss of graft function [9,10]. Accordingly, PTA has been associated with deterioration of graft function, graft loss, cardiovascular complications, and increased mortality, although the results of individual studies are not fully consistent [11-13]. Furthermore, the reported prevalence of PTA varies substantially with the definition and timing of assessment [5,8,14], and it has not been fully clarified whether anemia diagnosed three months after transplantation independently predicts deterioration of graft function one year after transplantation. This question is clinically relevant because, unlike most established risk factors for adverse graft outcomes, PTA is readily identifiable and amenable to treatment.

The aim of this study was therefore to evaluate whether anemia diagnosed three months after transplantation independently predicts deterioration of renal graft function one year after KTx.

Material and methods

This retrospective-prospective longitudinal observational study was conducted at the Nephrology Clinic, Clinical Center University of Sarajevo. A total of 116 kidney transplant recipients regularly followed at the outpatient transplant clinic were initially screened, and 17 were excluded because of incomplete follow-up data. Of the 99 recipients included in the study, four died within the first six months after transplantation; the remaining 95 recipients with complete one-year follow-up were included in the analyses of PTA-related outcomes. Baseline characteristics are reported for the entire cohort (99 recipients), whereas all PTA-related comparisons and outcome analyses were performed in these 95 recipients. Inclusion criteria were age over 18 years and performed KTx. Recipients younger than 18 years and those with a diagnosed malignancy were excluded.

For each recipient, sociodemographic data (age, sex) and the following clinical data were collected: pretransplant dialysis modality or pre-emptive transplantation, pretransplant dialysis duration, comorbidities (diabetes mellitus (DM), new-onset diabetes after transplantation (NODAT), hypertension), donor type (living related, living unrelated, or deceased), presence of DGF, and maintenance immunosuppression (tacrolimus or cyclosporine). Laboratory parameters included Hb, hematocrit (Hct), red blood cell count (RBC), serum creatinine, and 24-hour proteinuria, and the eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) 2021 equation. Complete blood count was evaluated at four time points (before transplantation, immediately after transplantation, and at 3 and 12 months after transplantation), whereas serum creatinine, proteinuria, and eGFR were assessed immediately after transplantation and at 3-months and 12-month time point. Data were obtained from the electronic health information system and hospital records.

PTA was defined as an Hb concentration <110 g/L three months after transplantation, and recipients were divided into two groups according to the presence of PTA. Deterioration of graft function was defined as a serum creatinine increase ≥25% or an eGFR decline ≥30% at the 12-month compared with the 3-month post-transplant time point. A composite outcome comprising death and/or the need for hemodialysis one year after transplantation was also evaluated.

Statistical analysis was performed using RStudio. Categorical variables are presented as absolute and relative frequencies, and continuous variables as mean ±standard deviation (SD) or as median and interquartile range (IQR). Categorical variables were compared using the chi-square test, with calculation of odds ratios (OR) and 95% confidence intervals (CI), and continuous variables were compared using the Student t-test. Univariable and multivariable binary logistic regression analyses were used to identify predictors of deterioration of renal graft function and to assess the association between PTA and these outcomes. The multivariable models were adjusted for age, sex, DM, hypertension, and donor type. A two-sided p value <0.05 was considered statistically significant.

Results

Baseline characteristics of the initially included cohort (n=99) are summarized in Table 1. Most recipients were male (71.7%), with mean age of 43.55±11.83 years, hemodialysis was the predominant pretransplant dialysis modality (78.6%), most recipients received a graft from a living donor (71.7%), and tacrolimus-based maintenance immunosuppression was used in 91.9% of recipients. All subsequent analyses included the 95 recipients with complete one-year follow-up, among whom PTA was present in 26 (27.37%).

Comparison of clinical characteristics between recipients with and without PTA is presented in Table 2. PTA was significantly more frequent in women than in men (42.86% vs. 20.90%; p=0.029), and male sex was associated with lower odds of PTA (OR 0.352, 95% CI 0.136-0.913). Hypertension was associated with a significantly lower frequency of PTA (p=0.026; OR 0.164, 95% CI 0.028-0.959). No other variables were significantly associated with PTA.

Recipients with PTA had significantly lower Hb, Hct, and RBC at all post-transplant time points (all p<0.001), whereas pretransplant values did not differ significantly between the groups. Serum creatinine was significantly higher in the PTA group immediately after transplantation (162.8±77.86 vs. 134.9±52.08 μmol/L;

p=0.046), and eGFR was significantly lower immediately after transplantation (p=0.012) and at the 12-month time point (50.46±17.26 vs. 63.80±18.82 mL/min/1.73 m²; p=0.002). Proteinuria was significantly higher in

Table 1. Baseline characteristics of kidney transplant recipients[a]

Characteristic	Value
Age (years), mean ± SD	43.55±11.83
Sex	
Male (n, %)	71(71.7)
Female (n, %)	28(28.3)
Pretransplant dialysis modality	
Hemodialysis (n, %)	77(78.6)
Peritoneal dialysis (n, %)	8(8.2)
Both modalities (n, %)	6(6.1)
No dialysis, preemptive transplantation (n, %)	7(7.1)
Pretransplant dialysis duration (months), median (IQR)	
Hemodialysis	36.0(12.0-64.5)
Peritoneal dialysis	8.5(3.0-32.5)
Both modalities	33.5(31.0-36.0)
Hypertension	
Yes (n, %)	93(93.9)
No (n, %)	6(6.1)
DM	
Yes (n, %)	9(9.1)
No (n, %)	90(90.9)
Donor type	
Living related (n, %)	52(52.5)
Living unrelated (n, %)	19(19.2)
Deceased (n, %)	28(28.3)
DGF	
Yes (n, %)	10(10.1)
No (n, %)	89(89.9)
Maintenance immunosuppression	
Tacrolimus (n, %)	91(91.9)
Cyclosporine (n, %)	8(8.1)
PTA[b]	
Yes (n, %)	26(27.37)
No (n, %)	69(72.63)

Abbreviations: IQR, interquartile range; SD, standard deviation; DM, diabetes mellitus; DGF, delayed graft function; PTA, post-transplant anemia. [a]n=99; dialysis modality was unavailable for one recipient, and the corresponding percentages are calculated among the 98 recipients with documented modality. [b]Assessed in 95 recipients with complete follow-up data; defined as hemoglobin <110 g/L.

Table 2. Comparison of clinical characteristics between recipients with and without post-transplant anemia

Characteristic	PTA (n=26)	Without PTA (n=69)	Test statistic	*p*	OR (95% CI)
Sex			*χ^2=4.791*	*0.029**	
Male (n, %)	14 20.90)	53(79.10)			0.352(0.136-0.913)
Female (n, %)	12(42.86)	16(57.14)			
Age (years), mean ±SD	45.42±13.50	42.33±11.28	t=1.127	0.263	
Pretransplant dialysis modality			χ^2=2.216	0.330	
Hemodialysis (n, %)	18(24.66)	55(75.34)			
Peritoneal dialysis (n, %)	3(37.5)	5(62.5)			
Both modalities (n, %)	3(50.0)	3(50.0)			
Donor type			*χ^2=2.586*	*0.108*	
Living (n, %)	22(31.88)	47(68.12)			2.574(0.791-8.375)
Deceased (n, %)	4(15.38)	22(84.62)			
Glucose metabolism			*χ^2=2.251*	*0.324*	
DM (n, %)	4(50.0)	4(50.0)			2.950(0.674-12.91)
No DM (n, %)	20(25.32)	59(74.68)			
NODAT (n, %)	2(25.0)	6(75.0)			
Hypertension			*χ^2=4.976*	*0.026**	
Yes (n, %)	22(24.72)	67(75.28)			0.164(0.028-0.959)
No (n, %)	4(66.67)	2(33.33)			
DGF			*χ^2=0.451*	*0.502*	
Yes (n, %)	3(37.5)	5(62.5)			1.670(0.369-7.547)
No (n, %)	23(26.44)	64(73.56)			

Abbreviations: CI, confidence interval; DM, diabetes mellitus; NODAT, new-onset diabetes after transplantation; DGF, delayed graft function; OR, odds ratio; PTA, post-transplant anemia; SD, standard deviation. Categorical variables were compared using the chi-square test and continuous variables using the Student t-test. Dialysis modality was compared among the 87 recipients with a documented modality; seven recipients underwent

preemptive transplantation and data were unavailable for one. *p<0.05.

Table 3. Hematological and renal function parameters in recipients with and without post-transplant anemia

Parameter	PTA (n=26)	Without PTA (n=69)	*t*	*p*
Hb (g/L)				
Before transplantation	105.2±21.74	116.0±17.30	-1.884	0.066
After transplantation	91.37±12.38	119.9±19.50	-6.929	<0.001*
3 months	100.8±5.912	129.7±15.02	-9.510	<0.001*
12 months	121.5±17.29	145.9±18.31	-5.889	<0.001*
HCT (%)				
Before transplantation	32.70±6.47	35.15±5.833	-1.342	0.186
After transplantation	28.05±3.962	36.94±6.53	-6.488	<0.001*
3 months	34.41±2.913	42.95±5.571	-7.423	<0.001*
12 months	38.10±4.828	44.57±6.119	-4.847	<0.001*
RBC (×10¹²/L)				
Before transplantation	3.632±0.658	3.797±0.681	-0.821	0.416
After transplantation	3.062±0.445	3.933±0.741	-5.615	<0.001*
3 months	3.755±0.445	4.660±0.697	-6.151	<0.001*
12 months	4.365±0.658	5.063±0.676	-4.520	<0.001*
Serum creatinine (µmol/L)				
After transplantation	162.8±77.86	134.9±52.08	2.019	0.046*
3 months	131.8±35.51	129.8±40.73	0.224	0.823
12 months	147.7±50.36	127.8±49.27	1.746	0.084
eGFR (mL/min/1.73 m²)				
After transplantation	49.08±21.04	61.94±21.96	-2.575	0.012*
3 months	55.27±16.04	61.81±18.07	-1.620	0.109
12 months	50.46±17.26	63.80±18.82	-3.147	0.002*
Proteinuria (g/24 h)				
After transplantation	0.721±0.581	0.418±0.515	2.313	0.023*
3 months	0.476±0.750	0.344±0.382	1.107	0.271
12 months	0.325±0.320	0.238±0.269	1.300	0.197

Abbreviations: Hb, hemoglobin; Hct, hematocrit; RBC, red blood cell count; eGFR, estimated glomerular filtration rate; PTA, post-transplant anemia. Data are presented as mean ± standard deviation. Variables were compared using the Student t-test. Pretransplant hematological parameters and proteinuria were not available for all recipients, and these comparisons were therefore performed in the recipients with available measurements. *p<0.05.

the PTA group immediately after transplantation (p=0.023), without significant differences at later time points (Table 3).

Table 4 shows that at one year post-transplant, a serum creatinine increase ≥25% occurred in 23.08% of recipients with PTA compared with 4.35% of those without PTA (OR 6.60, 95% CI 1.51-28.80; p=0.005), and an eGFR decline ≥30% occurred in 19.23% of recipients with PTA compared with 1.45% of those without PTA (OR 16.19, 95% CI 1.79-146.42; p=0.001).

Table 4. Univariable analysis of one-year outcomes in recipients with and without post-transplant anemia

Outcome	PTA (n=26)	Without PTA (n=69)	χ^2	*p*	OR (95% CI)
Serum creatinine increase ≥25% (n, %)	6(23.08)	3(4.35)	7.724	0.005*	6.60 (1.51-28.80)
eGFR decline ≥30% (n, %)	5(19.23)	1(1.45)	10.09	0.001*	16.19 (1.79-146.42)
Hemodialysis at one year (n, %)	0(0.0)	1(1.45)	0.381	0.537	0.862 (0.03-21.82)
Death at one year (n, %)	1(3.85)	1(1.45)	0.526	0.468	2.72 (0.16-45.15)

Abbreviations: CI-confidence interval, eGFR-estimated glomerular filtration rate, OR-odds ratio, PTA-post-transplant anemia. Categorical variables were compared using the chi-square test. *p<0.05.

In univariable logistic regression, PTA was significantly associated with both a serum creatinine increase ≥25% (OR 6.600, 95% CI 1.513-28.80; p=0.012) and an eGFR decline ≥30% (OR 16.19, 95% CI 1.790-146.4; p=0.013). After adjustment for age, sex, DM, hypertension, and donor type, multivariable logistic regression analysis showed that PTA remained an independent predictor of both outcomes, serum creatinine increase ≥25% and an eGFR decline ≥30% (OR 8.485, 95% CI 1.111-64.83; p=0.039, and OR 27.54, 95% CI 2.280-340.4; p=0.010). Deceased-donor transplantation was also an independent predictor of a

serum creatinine increase ≥25% (OR 17.54, 95% CI 1.335-230.38; p=0.029), while hypertension was inversely associated with this outcome (OR 0.003, 95% CI 0.000-0.164; p=0.004) (Table 5).

Table 5. Univariable and multivariable logistic regression analysis of the impact of post-transplant anemia on deterioration of renal graft function one year after transplantation

			Serum creatinine increase ≥25%		
Predictor	***B***	**SE**	**OR**	**95% CI**	***p***
Univariable logistic regression					
PTA	1.887	0.752	6.600	1.513-28.80	0.012*
Multivariable logistic regression					
PTA	2.138	1.038	8.485	1.111-64.83	0.039*
Age (years)	0.024	0.046	1.024	0.936-1.121	0.601
Female sex	-1.892	1.384	0.151	0.010-2.271	0.172
DM	1.966	1.276	7.143	0.586-87.067	0.123
Hypertension	-5.677	1.975	0.003	0.000-0.164	0.004*
Deceased-donor transplantation	2.864	1.314	17.54	1.335-230.38	0.029*
eGFR decline ≥30%					
Univariable logistic regression					
PTA	2.784	1.124	16.19	1.790-146.4	0.013*
Multivariable logistic regression					
PTA	3.316	1.283	27.54	2.28-340.4	0.010*
Age (years)	-0.002	0.048	0.998	0.908-1.096	0.959
Female sex	-2.907	2.123	0.055	0.001-3.504	0.171
DM	0.670	1.268	1.954	0.164-23.45	0.597
Hypertension	-3.011	2.276	0.049	0.001-4.262	0.186
Deceased-donor transplantation	1.236	1.229	3.442	0.310-38.30	0.314

Abbreviations: B-regression coefficient, CI-confidence interval, PTA-post-transplant anemia, DM-diabetes mellitus, eGFR-estimated glomerular filtration rate, OR-odds ratio, SE-standard error.

Discussion

In this single-center cohort of kidney transplant recipients, the prevalence of PTA three months after transplantation was 27.37%. PTA was significantly more frequent in women and less frequent in recipients with hypertension, whereas age, pretransplant dialysis modality, donor type, glucose metabolism disorders, and DGF were not significantly associated with its occurrence. Recipients with PTA had significantly lower Hb, Hct, and RBC at all post-transplant time points, together with significantly lower eGFR immediately after transplantation and at one year. A serum creatinine increase ≥25% and an eGFR decline ≥30% at one year were significantly more frequent in the PTA group, and after adjustment for age, sex, DM, hypertension, and donor type, PTA remained an independent predictor of both outcomes of deterioration of renal graft function, while deceased-donor transplantation independently predicted a serum creatinine increase ≥25%.

The observed prevalence of PTA in our study closely matches the 28.85% reported by Banjeglav and Zibar, who applied the same Hb threshold at three months in a Croatian cohort [5]. On the contrary, a study using the same threshold at a comparable time point reported a considerably lower rate of 13% [6]. Higher figures emerge where the World Health Organization (WHO) criteria are applied: 76.7% and 87% at three months in two prospective cohorts [15,16], and 51.3% six months after transplantation in a retrospective cohort [17]. The influence of the definition is best illustrated by the study of Einollahi *et al.*, in whose cohort the prevalence was 52.7% according to WHO criteria at six months but only 24.4% when PTA was defined as Hb ≤110 g/L [14]. These differences confirm that the choice of the diagnostic threshold and time assessment directly determine the reported prevalence and support the use of a lower Hb cut-off to identify clinically relevant anemia in transplant recipients.

The higher frequency of PTA among women in the present study is consistent with the findings of Banjeglav and Zibar (p=0.025) [5] and with a large cohort in which female sex was significantly associated with PTA [14]. In women whose menses had ceased before transplantation, periods resume in the majority of cases, at a median of three months [18], the interval at which PTA was assessed in the present study. Menstrual blood loss and the resulting iron deficiency have accordingly been proposed as contributors to the higher frequency of PTA reported in female recipients [19].

The inverse association between hypertension and PTA was unexpected and is difficult to explain pathophysiologically. In our cohort, however, only six recipients did not have hypertension, so this finding is most likely a consequence of the small subgroup size and should be interpreted with caution.

The significantly lower Hb, Hct, and RBC observed in recipients with PTA at every post-transplant time point

were an expected finding, as these parameters are basic indicators of erythropoietic status and directly reflect the presence and severity of PTA. It should be noted that pre-transplant Hb already tended to be lower in recipients who subsequently developed PTA, although this difference was of borderline significance. Preexisting anemia is a recognized contributor to early PTA [4,7], and the lower pre-transplant Hb in this group most likely reflects its persistence into the early post-transplant period rather than a separate phenomenon. Notably, Hb in the PTA group improved gradually over the first post-transplant year, yet deterioration of graft function was more frequent in this group. This observation suggests that early exposure to anemia may have a lasting effect on the graft, which is in line with the finding that early PTA carries the highest risk of adverse outcomes [12]. Serum creatinine was significantly higher in the PTA group only immediately after transplantation; thereafter the difference attenuated and retained as only a strend towards higher values at one year. In contrast, eGFR was significantly lower in recipients with PTA both immediately after transplantation and at 12 months, indicating a more pronounced progressive decline in graft function in this group. Biologically, this may be explained by the mechanisms through which anemia contributes to graft injury: chronic hypoxia resulting from a reduced Hb concentration activates profibrotic mediators that promote tubulointerstitial fibrosis and progressive loss of graft function, while the hemodynamic adaptations accompanying anemia, including compensatory tachycardia and altered renal perfusion, may further contribute to ischemic injury and accelerate chronic graft dysfunction [9,10]. Similarly to our findings, another group of authors found no difference in serum creatinine between recipients with and without PTA at 12 or 36 months after transplantation [20].

Proteinuria was significantly higher in recipients with PTA immediately after transplantation, whereas this difference did not persist at later time points. A cross-sectional study likewise identified proteinuria as a significant risk factor for PTA, although in that cohort the association was observed at the 6- and 12-month post-transplant time points [21]. The proposed mechanism linking proteinuria to PTA may be urinary loss of erythropoietin, iron and other factors relevant for erythropoiesis, although its relevance for sub-nephrotic proteinuria and the transplant population remains uncertain [21].

The central finding of this study is that PTA independently predicted deterioration of graft function at one year after transplantation. The adjusted odds were approximately 8.5-fold higher for a serum creatinine increase ≥25% and 27.5-fold higher for an eGFR decline ≥30%. Larger cohorts have reported associations of PTA with acute rejection, graft loss, and mortality [6,7,11]. Our findings are consistent with the cohort of Kolonko *et al.*, in which recipients with PTA likewise had a significantly lower eGFR at one year after transplantation (43.9 vs. 55.3 mL/min/1.73 m²) and a higher long-term risk of graft loss [22]. A meta-analysis of 17 studies including 16,463 recipients confirmed the association of PTA with graft loss and overall mortality, with early PTA carrying the highest risk [12].

The evidence is nevertheless not uniform. In several cohorts, PTA was associated with graft loss but not with patient survival or cardiovascular events [13,22,23]. One study found no difference in graft function between recipients with and without PTA at 6 and 12 months [24].

Deceased-donor transplantation independently predicted a serum creatinine increase ≥25% in our cohort. This is in line with a systematic review and meta-analysis of 35 observational studies in which deceased-donor grafts were independently associated with one-year death-censored graft loss [25]. Another retrospective cohort study reported a shorter graft and patient survival after deceased-donor transplantation, although in that cohort the difference became apparent only after the first post-transplant year [26]. The functional deterioration of renal graft function observed at the 12-month time point in our cohort may therefore represent an earlier manifestation of the same process, preceding overt graft loss.

In the present study, the frequency of death and return to hemodialysis one year after transplantation did not differ between recipients with and without PTA. With only two deaths and one return to dialysis, the number of events was too small for a meaningful analysis of the composite outcome, and this finding should not be interpreted as evidence against an effect of PTA on recipient survival, that has been demonstrated in substantially larger cohorts and pooled analyses [11,12].

Our study has limitations. It was conducted in a single center with a relatively small sample size, and the low number of adverse events resulted in wide confidence intervals around the effect estimates, so the magnitude of the adjusted OR should be interpreted with caution. In addition, the follow-up was limited to one year, so the longer-term impact of PTA on graft function could not be assessed. In addition and unfortunately, the iron status, including ferritin levels, was not systematically assessed, so the contribution of iron deficiency to PTA and its evolution in the two groups could not be evaluated. The partly retrospective design carries a risk of information bias, four recipients who died within the first six months could not be included in the outcome analyses, and residual confounding cannot be excluded.

Conclusion

Post-transplant anemia, present in more than one quarter of recipients, independently predicted deterioration of

renal graft function one year after kidney transplan–tation. Unlike donor age, donor type, or the degree of human leukocyte antigen (HLA) matching, the PTA is modifiable, and it is detected by a laboratory test al–ready performed at every routine post-transplant visit. The Hb value obtained three months after transplan–tation identifies recipients who warrant closer sur–veillance of graft function, at negligible additional cost. Whether correction of PTA alters that trajectory, and at which Hb threshold, remains to be established in larger prospective studies with standardized defi–nitions of PTA.

Conflict of interest statement. None declared.

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