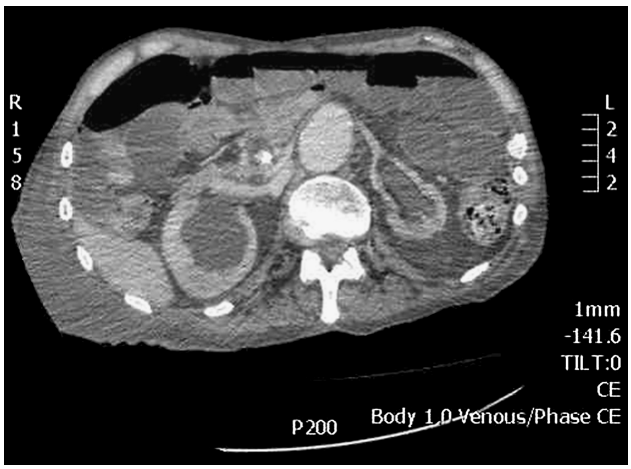

Fig. 1. Bilateral hydronephrotic kidneys.

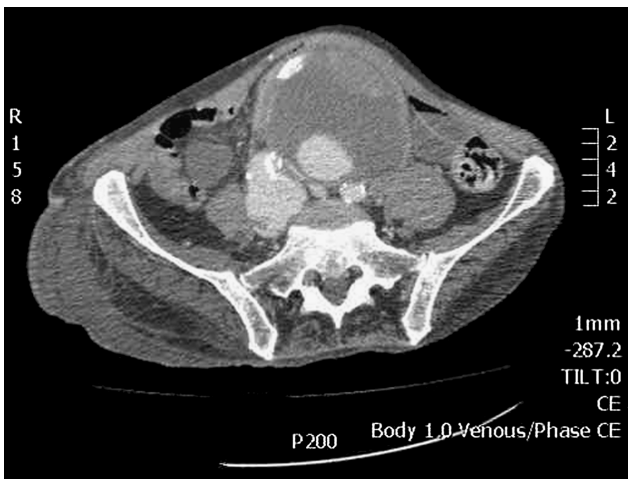

Fig. 2. A large infrarenal abdominal aortic aneurysm with extensive fibrosis.

Early hydronephrosis developing within the first year occurs in 10–20% of patients following surgical graft repair of AAA. The most probable cause for this is mechanical due to the compression of the ureter against the native iliac artery from the anteriorly placed graft [2].

In a prospective study of 101 patients who underwent aortofemoral and aortoiliac reconstructive surgery, 12% of patients developed mild to moderate hydronephrosis. All patients were asymptomatic, and the obstruction resolved spontaneously in 10 of 11 patients within 3 months of onset.

The incidence of delayed hydronephrosis occurring 1 year or more post surgical repair is unknown. This complication may be relatively common after reconstructive vascular surgery as in our patient, especially in association with infected grafts [3]. The mechanism for the development is not fully understood. The most widely held view is that this complication develops secondary to an inflammatory process causing fibrosis [4].

The long time interval is unusual and suggests perhaps a different mode of inflammatory pathways compared to what is commonly seen in early obstructive uropathy associated with surgical graft repair of AAA. More research is needed to elucidate the mechanisms underlying chronic periaortitis [5].

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Validity of haemoglobin A1c and glycoalbumin for an appropriate evaluation of glycaemic control in Japanese diabetic patients with chronic renal failure

Sir,

Although the validity of glycoalbumin (GA) instead of haemoglobin A1c (HbA1c) measurement in patients on haemodialysis (HD) has recently been discussed by some investigators [1,2], an appropriate indicator for glycaemic control in patients with pre-dialysis chronic renal failure (CRF) has only rarely been reported [1]. The application of erythropoietin (EPO) for the treatment of renal anaemia increases the proportion of young erythrocytes over old erythrocytes in peripheral blood [3], and HD procedure per se causes the mechanical destruction of red blood cells (RBC). These conditions may reduce the half-life of HbA1c. On the other hand, GA is affected by an accelerated turnover of albumin in the case of nephrotic syndrome frequently observed in pre-dialysis patients due to a massive loss of protein into the urine [2]. The aim of the present study is to evaluate the validity of both indicators in Japanese patients with diabetes separately according to their CRF stage, either undergoing HD or not and either being treated with EPO or not.

Methods

Four hundred and seventy-five patients with diabetes (279 males, 33 type 1 diabetes, 63 ± 13 years old, mean ± SD) were enrolled from November 2007 to June 2009 at Kurume University Hospital and Ito Clinic, in which 97 were treated with maintenance HD (Group HD, no haemodialysis filtration), 112 had impaired renal function with their serum creatinine (S-Cr) levels >1.2 mg/dL in male and 0.9 mg/dL in female subjects (Group RD), and 266 had normal renal function (Group N). Estimated GFR (eGFR) was calculated according to the formula modified by the Japanese Society of Nephrology in 2008 [eGFR for male = $194 \times \text{S-Cr}^{-1.094} \times$

Table 1. Characteristics of diabetic patients

	Group N (n = 266)	Group RD (n = 112)	Group HD (n = 97)	P-value[a]
Age (years)	61.1 ± 13.8	63.6 ± 14.3	64.2 ± 12.0	NS
Gender (male/female)	147/119	63/49	69/28	<0.05
Duration of diabetes (years)	8.9 ± 7.5	13.7 ± 9.6*	18.4 ± 8.4*,**	<0.01
Type 1/type 2 diabetes	26/240	4/108	3/94	NS
Diet only/OHA/insulin	58/145/86	20/44/72	18/4/77	<0.01
BMI	24.0 ± 3.9	24.6 ± 4.5	21.8 ± 3.5*,**	<0.01
FPG (mg/dL)	140.7 ± 38.4	125.7 ± 42.6*	147.3 ± 46.9**	<0.01
PPG (mg/dL)	184.7 ± 63.8	172.8 ± 65.0	212.4 ± 56.7*,**	<0.01
Mean PG (mg/dL)	161.8 ± 44.6	149.2 ± 45.5*	178.9 ± 43.2*,**	<0.01
HbA1c (%)	7.2 ± 1.4	6.6 ± 1.4*	6.0 ± 1.0*,**	<0.01
GA (%)	21.5 ± 5.3	19.7 ± 5.5*	24.1 ± 5.6*,**	<0.01
Serum albumin (g/dL)	4.16 ± 0.37	3.61 ± 0.60*	3.63 ± 0.32*	<0.01
Serum creatinine (mg/dL)	0.72 ± 0.17 (female: 0.3~0.86; male: 0.44~1.17)	2.89 ± 1.99* (0.9~10.78)		<0.01
eGFR (mL/min/1.73 m^2)	80.8 ± 22.3 (41.4~168.6)	24.4 ± 13.7* (3.5~57.8)		
Hb (g/dL)	13.9 ± 1.6	11.5 ± 2.1*	10.3 ± 1.3*,**	<0.01
EPO (+)	0	33	93	<0.01

Descriptions for each abbreviation are given in text except for OHA as 'oral hypoglycaemic agent'. Data are presented as means ± SD or number of subjects. Ranges of serum creatinine and eGFR are given in parenthesis. One-way ANOVA with Scheffe-type multiple comparison test or chi-square test was used for statistical analysis.NS, not significant.
[a]ANOVA or chi-square test.
*P < 0.05 vs. Group N.
**P < 0.05 vs. Group RD after multiple comparison test.

Table 2. Univariate analysis on mean PG as an outcome variable in RD patients stratified by CKD stages

Group of patients	Predictive variable	r	P-value
CKD stage 3 (n = 44)	HbA1c	0.760	<0.0001
	GA	0.529	0.0003
CKD stage 4 (n = 31)	HbA1c	0.822	<0.0001
	GA	0.374	0.0384
CKD stage 5 (n = 37)	HbA1c	0.573	0.0002
	GA	0.267	0.1106

EPO was used in 3 (7%) of CKD stage 3, 7 (23%) of CKD stage 4 and 23 (62%) of CKD stage 5 patients. r, Pearson's correlation coefficient.

$age^{-0.287}$; eGFR for female = 0.739 × (eGFR for male) mL/min/1.73 m^2]. Patients were restricted to those whose HbA1c differences had been within 0.3% and whose diabetes treatment had not been altered during the preceding 3 months before the determination of GA and HbA1c. Both fasting plasma glucose (FPG) and post-prandial PG (PPG), 2 h after they had eaten their usual breakfast, were measured to calculate mean PG (the average of FPG and PPG had been demonstrated to show an excellent correlation with HbA1c by Ozmen *et al*. [4]). HbA1c was measured by routine HPLC, which was standardized according to the Japan Diabetes Society. GA was measured by an enzymatic method using the Lucica GA-L kit (Asahi Kasei Pharma Corp., Tokyo, Japan).

Results

Serum creatinine level was higher, and albumin (Alb) and haemoglobin (Hb) levels were lower in both CRF groups than in Group N (Table 1). Furthermore, Alb level was lower in RD with EPO treatment (3.28 ± 0.58 g/dL). In RD patients, mean PG correlated much better with HbA1c (r = 0.727) than with GA (r = 0.448), with the equivalent r-value to that of Group N (r = 0.753) (Figure 1A and B). However, a portion of the RD patients treated by EPO showed a weaker correlation between mean PG and HbA1c (r = 0.473) or GA (r = 0.249). After the stratification of RD patients according to CKD stages based on eGFR, mean PG correlated better with HbA1c than GA in each stratified group. In CKD stage 5, however, these correlations were considerably weaker than those in other groups, which is associated to the higher proportion of EPO treatment (Table 2). In contrast, mean PG correlated much better with GA (r = 0.753) than with HbA1c (r = 0.592) in HD patients, while the r-value was significantly higher (P = 0.02 by Fisher's Z-transformation) than that in Group N (r = 0.608) (Figure 1D). In HD patients, the regression line between mean PG and HbA1c (r = 0.592) was significantly (P = 0.02) shallower than that in Group N (Figure 1C). In multiple regression analysis, coefficient of determination (R^2) to explain mean PG was 52.9% by HbA1c alone and 55.6% by four variables (HbA1c, GA, Hb and Alb) in RD patients, implying that 95% (52.9%/55.6%) of the variation of mean PG could be explained by HbA1c alone in this group of patients. In contrast, GA alone explained 98% (56.7%/58.2%) of R^2 in HD patients.

Discussion

Our study had three major findings. First, HbA1c is a robust and accurate index for glycaemic control in diabetic patients with pre-dialysis renal dysfunction and without treatment by EPO. Second, in HD patients, GA is a rigorous marker for the evaluation of blood glucose level in line with a previous report [5]. Third, no surrogate index exists for pre-dialysis RD patients treated with EPO. It is possible that the failure of GA to correlate mean PG in RD is attributable to its scattered distribution, probably caused by the fluctuation of serum albumin concentration in this group of patients. In contrast, lower HbA1c level in HD patients is attributable to EPO treatment because most patients in this group received this medication.

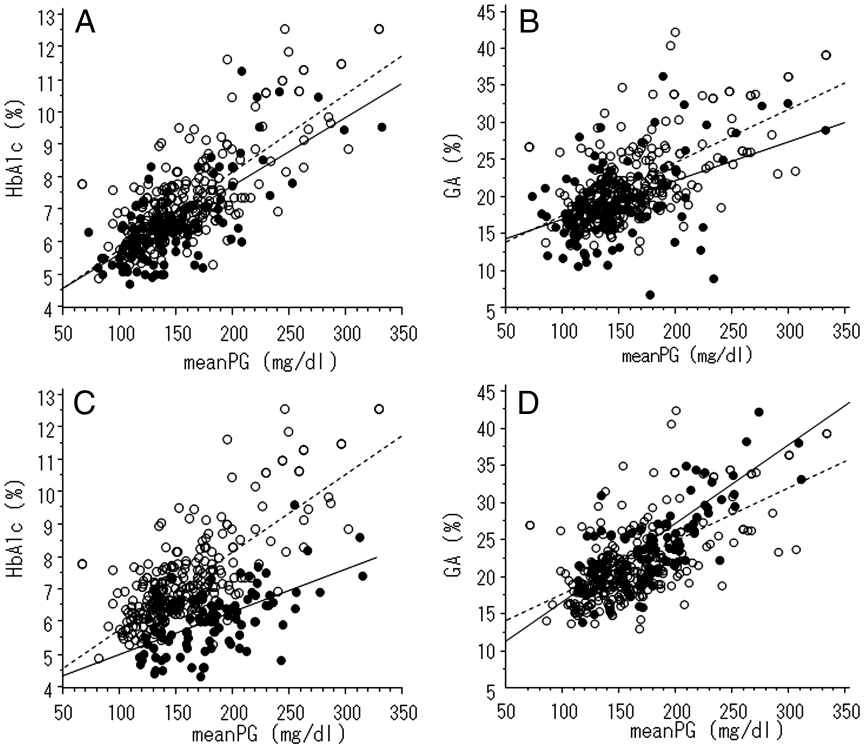

Fig. 1. Correlation between mean PG and HbA1c or GA in each group of patients. Pearson's correlation coefficients were used for statistical analysis. Group N patients were indicated as open circles with dotted line. Group RD patients were indicated as closed circles with solid line in (**A** and **B**) and Group HD patients as closed circles with solid line in (**C** and **D**).

A robust correlation between HbA1c and mean PG in RD patients was conserved even after the stratification according to CKD stages. In opposition to our results, Freedman *et al*. reported that GA appeared to reflect more accurately recent glycaemic control than HbA1c in pre-dialysis diabetic patients [1]. One possible explanation for these discordant results is that our study included many CKD stage 4 or 5 patients with massive proteinuria, and their serum albumin concentration was 3.57 ± 0.64 (CKD stage 4) and 3.3 ± 0.59 g/dL (CKD stage 5) which was much lower than that in the previous report (3.7 ± 0.57 g/dL). Many patients with nephrotic syndrome in our Group RD might affect such a weaker correlation between GA and mean PG.

Our results are interpreted to mean that HbA1c can be stringently used for RD patients so long as they are not treated with EPO or HD, and that GA could be substituted for HbA1c once HD has been started, and keeping in mind the possibility of a falsely high value. These findings are to be further confirmed and elucidated in future studies composed of a larger scale of patients recruited from multiple institutes.

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