surface-adjusted TKV (bs-TKV) or log-converted TKV (log-TKV), were compared from the standpoint of minimizing the differences between men and women. It remains unclear which adjusted TKV marker correlates best with renal function.

On the other hand, the results from three recent prospective clinical trials examining the effect of mammalian target of rapamycin inhibitors on disease progression of ADPKD have not demonstrated an association between changes in TKV and glomerular filtration rate (GFR) [6–8]. These studies might have used too short a period for examining the relationship between TKV and functional changes.

If TKV correlates with kidney function, it will be a useful clinical marker of renal function since (1) it can be measured reliably, and (2) it changes by a measurable amount during a relatively short period of time (mean % increase of TKV is 5–6 % per year) [9]. In contrast, kidney function, measured by estimated GFR (eGFR), decreases at a slow rate of 0–3 ml/min/1.73 m^2 per year depending on the chronic kidney disease (CKD) stage [10]. Taking the measurement variation of eGFR into consideration, it is difficult to detect a small change as significant, especially during early CKD stages when a relatively small amount of eGFR decreases from a relatively large baseline eGFR.

For the above reasons, we reappraised the relationship between kidney volume and kidney function (using eGFR). If a significant relationship between two parameters was confirmed, the characteristics of these parameters and their changes (slopes) in relation to age and CKD stage were examined.

Materials and methods

All patients fulfilled Ravine's diagnostic criteria of ADPKD. One hundred and eighty-eight patients with ADPKD gave informed consent to take part in an observational clinical study protocol measuring TKV once a year with simultaneous collection of 24-h urine for determination of creatinine clearance (Ccr) and urinary protein excretion between April 2007 and July 2012. Patients with end-stage renal disease (ESRD) underwent TKV measurement only. Of 188 patients, 70 underwent TKV measurement three times or more. Two patients who received laparoscopic cyst fenestration, one patient with a ureteral stone with hydronephrosis during the study period, and three patients with baseline ESRD were excluded from analysis.

Serum creatinine was measured enzymatically. Kidney function was estimated with Ccr using 24-h urine, reciprocal creatinine and eGFR. eGFR was calculated using the following formula—eGFR (male) = $194 \times Cr^{-1.094} \times Age^{-0.287}$, and eGFR (female) = eGFR (male) $\times$ 0.739. This equation is a Japanese coefficient of the modified Isotope Dilution Mass Spectrometry—Modification of Diet in Renal Disease (IDMS–MDRD) Study [11]. The staging of kidney function is based on the Kidney Disease Outcomes Quality Initiative Clinical Practice Guidelines for CKD [12] using the final eGFR measurement.

TKV was measured by high-resolution magnetic resonance imaging (MRI) using a volumetric measurement of cross-sectional imaging, as described in the report from the CRISP study [13]. Gadolinium enhancement was not used for safety reasons. TKV was adjusted by height (ht-TKV, ml/m), body surface area (bs-TKV, ml/m^2) and log-converted form (log-TKV, log[ml]). Kidney volume was measured by one radiologist (KK). Intrareader reliability was extremely high—the correlation coefficient was 0.999 for ten different single kidney volume measurements at different times when blind to first measurement. The mean of the % difference between two measurements was 0.29 ± 3.28 (SD) %.

Twenty-four-hour urinary protein excretion was expressed as the mean value of several measurements for each patient. The slopes of TKV, adjusted TKV parameters and kidney function parameters were calculated using linear regression analysis for each patient. %TKV was calculated with baseline TKV as 100 %.

The study protocol was approved by an institutional review board (09-56), and the study was conducted in accordance with the guidelines of the Declaration of Helsinki. All participants gave written informed consent to use their clinical data for medical research.

Statistical analyses

Analyses were performed with StatMate 4 and SAS 10 for Windows. Parametric variables are expressed as the mean and standard deviation in parentheses. Two-sided $p < 0.05$ was considered to indicate statistical significance. p values for differences between CKD stages were obtained using ANOVA or the Kruskal–Wallis test. Correlations between two variables were examined by linear regression analysis. The correlation coefficient (r) was obtained by the Spearman rank-order correlation coefficient.

Results

Between April 2007 and July 2012, 188 patients with ADPKD attending our clinic were followed annually by measuring TKV with MRI and 24-h urine collection. Among them, 70 patients repeated MRI and 24-h urine measurements three times or more. Six patients with a medical history affecting kidney volume, such as laparoscopic fenestration and baseline ESRD, were excluded

from the study, leaving 64 patients for analysis (67 % were female).

Four of the 64 patients had ESRD and one died of cerebral hemorrhage during this observation period. Baseline characteristics and the annual change rate (slope) of kidney function and volume are shown in Table 1. Mean slope of %TKV and eGFR were 5.9 % per year and −1.0 ml/min/1.73 m^2 per year, respectively.

Relationship between TKV and kidney function

TKV, ht-TKV, bs-TKV and log-TKV are all significantly correlated with eGFR (Fig. 1). Figure 1 illustrates the data measured at final observation, but qualitatively similar results were obtained using baseline observation. Among these parameters, log-TKV correlation was most significant. Baseline TKV and ht-TKV, but not bs-TKV and log-TKV, negatively correlated with the eGFR slope ($r = -0.2642$, -0.2476, -0.1811 and -0.2425, $p = 0.0349$, 0.0485, 0.1521, 0.0534, respectively, Fig. 2a). There was a weak but significant correlation between the eGFR slope and TKV slope ($r = -0.2593$, $p = 0.03853$, Fig. 2b).

Table 1 Baseline and annual change rate (slope) data of kidney volume and function

N (men/women)	64 (21/43)
Age (year)	47.0 (14.1)
Observation period (months)	39.7 (11.1)
Baseline data of kidney volume and function	
TKV (ml)	1,681.1 (1,001.1)
ht-TKV (ml/m)	1,023.8 (604.2)
bs-TKV (ml/m^2)	1,029.4 (615.2)
log-TKV (log[ml])	3.1588 (0.2357)
1/Cre (ml/mg)	109.8 (42.7)
eGFR (ml/min/1.73 m^2)	60.2 (27.38)
Ccr (ml/min/1.73 m^2)	90.01 (36.96)
Annual change rate (slope, b*) of kidney volume and function	
TKV slope (ml/year)	109.5 (123.8)
%TKV slope (%/year)	5.90 (4.38)
ht-TKV slope (ml/m/year)	65.9 (74.4)
bs-TKV slope (ml/m^2/year)	64.3 (71.6)
log-TKV slope (log[ml]/year)	0.022 (0.021)
1/Cre slope (ml/mg/year)	−0.948 (8.073)
eGFR slope (ml/min/1.73 m^2/year)	−1.020 (3.632)
Ccr slope (ml/min/1.73 m^2/year)	−3.753 (9.233)

Numbers are the mean and standard deviation (in parentheses).

*A linear regression line ($y = a + bX$) was obtained by regression analysis between each parameter and age (months) for the measurement of each patient and b is expressed as change rate per year (slope)

TKV total kidney volume, *ht-TKV* TKV divided by height (m), *bs-TKV* TKV divided by body surface area (m^2), *log-TKV* log-converted TKV, *eGFR* estimated glomerular filtration rate by Japanese MDRD equation, *Ccr* creatinine clearance measured by 24-h urine collection

Statistically significant correlations between eGFR and TKV-related parameters support the view of a clinically meaningful surrogate marker of TKV in ADPKD. The significant correlation between baseline TKV and eGFR slope (Fig. 2a) suggests the prognostic value of TKV for kidney functional deterioration.

TKV and function in relation to CKD stage

Individual data plotted as age-related TKV according to different CKD stages (Fig. 3) and Table 2 show that TKV increases faster and becomes larger as CKD stages advance. Age, systolic blood pressure, proteinuria, TKV, and TKV slope increase while eGFR slope decreases significantly ($p < 0.001$) as CKD stage advances (Table 2). Stages 1 and 2 are combined because TKV did not differ significantly (1264 ± 511 ml in stage 1 ($n = 7$) and 1492 ± 595 ml in stage 2 ($n = 24$), $p = 0.3666$).

In five of seven patients with CKD stage 5, TKV increased >3,000 ml. In contrast, only two of 46 patients with CKD stages 1–3 had TKV >3,000 ml (Fig. 1, $p < 0.001$).

In patients with advanced CKD stages, eGFR decreased faster, which was demonstrated by a significant correlation between final eGFR and the eGFR slope ($r = 0.4002$, $p = 0.0011$); however, no significant correlation was observed between baseline eGFR and the eGFR slope ($r = 0.1069$, $p = 0.4007$). There was a high correlation between baseline as well as final TKV and the TKV slope ($r = 0.7995$ and 0.8955, $p < 0.001$ $p < 0.001$, respectively), suggesting that patients with large kidneys have a rapid rate of kidney enlargement.

Changes in kidney volume and function in relation to age

As age advanced, eGFR, reciprocal creatinine and Ccr decreased significantly (Table 3). There was highly significant correlation between age and eGFR but the eGFR slope did not change significantly in relation to age.

TKV and TKV-related parameters had no significant correlation with age (Fig. 5). In contrast, the %TKV slope and log-TKV slope became smaller as age advanced (right panel of Table 3 and Fig. 5d). There was no significant correlation between function-related slopes and age.

The age-related results were not qualitatively different between baseline and final age.

Discussion

The present study confirmed the significant relationship between TKV and kidney function, which was reported by

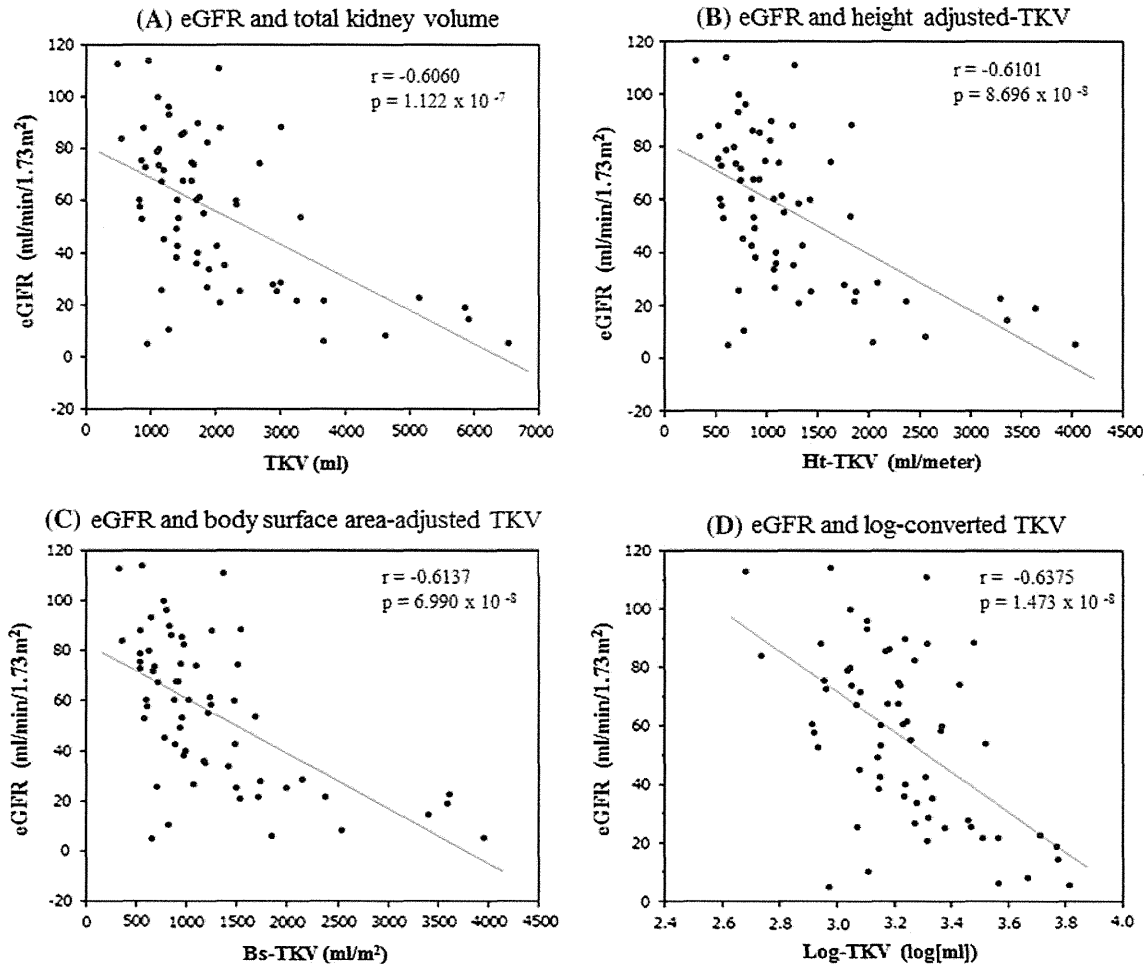

Fig. 1 Correlation of **a** total kidney volume (TKV), **b** height-adjusted TKV (ht-TKV), **c** body surface area-adjusted TKV (bs-TKV) and **d** log-converted TKV (log-TKV) to estimated glomerular filtration rate (eGFR). These values are final measurements. The correlation coefficients (r) of all TKV-related parameters are significant. Among them, r of log-TKV is most significant

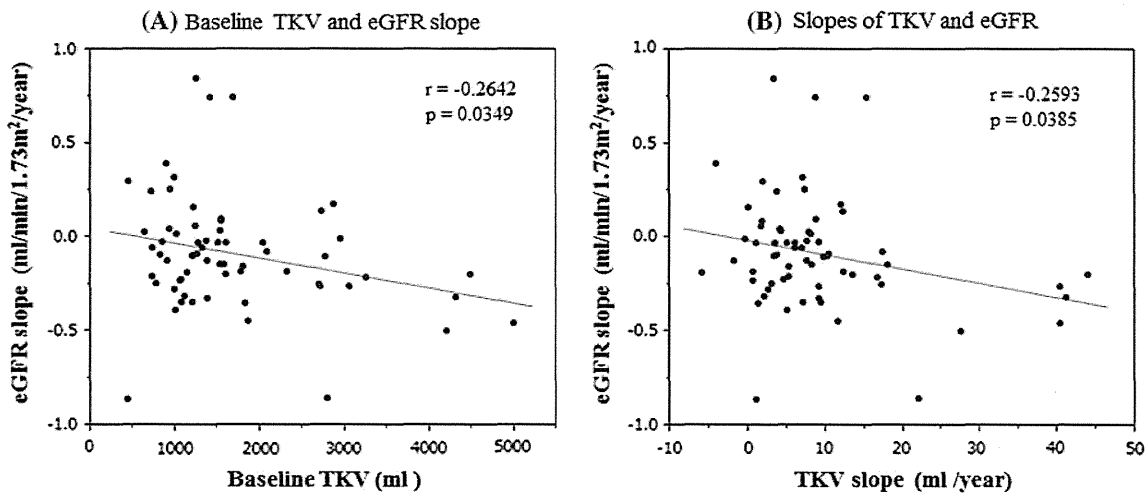

Fig. 2 **a** Correlation coefficient (r) between baseline TKV and eGFR slope is significant ($p = 0.0349$). **b** The correlation coefficient (r) between TKV slope and eGFR slope is significant ($p = 0.0385$)

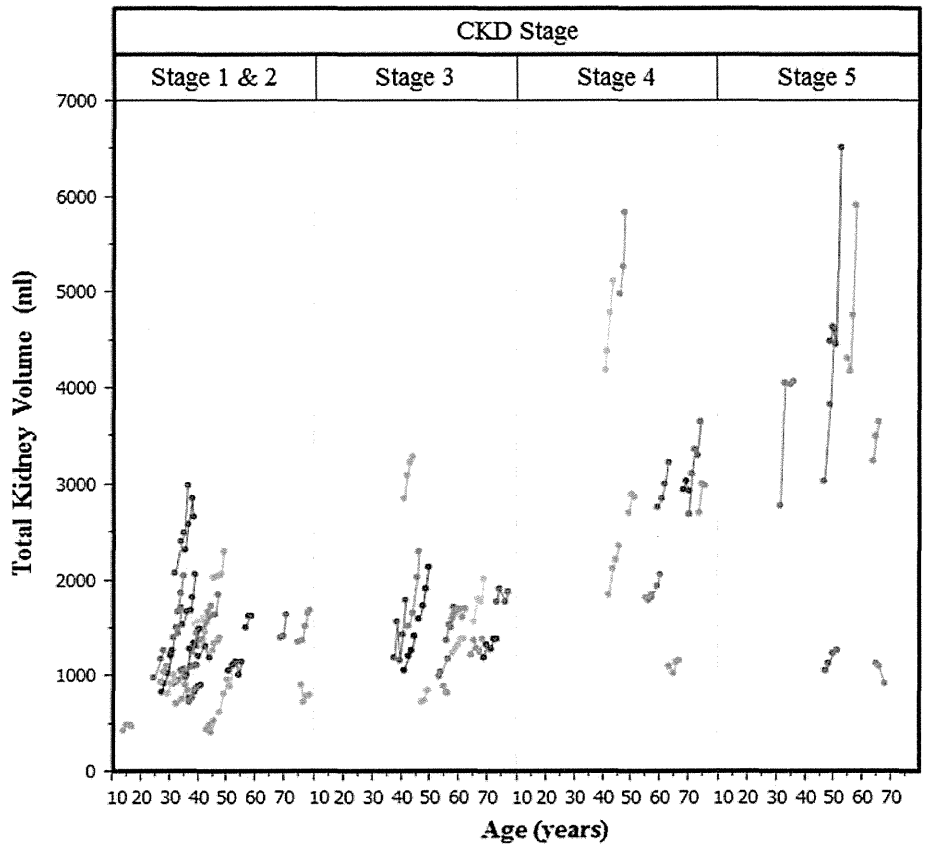

Fig. 3 Individual TKV data and the age at measurement are plotted and connected according to chronic kidney disease (CKD) stages. Finally measured eGFR was used to indicate the CKD stage category

CRISP studies [4, 5, 14–16]. Among adjusted TKV parameters, log-TKV correlated with eGFR most significantly. As the CRISP study showed that TKV increased exponentially and GFR decreased linearly [4], it is reasonable that log-TKV correlates with kidney function better than the other adjusted TKV parameters [14].

Final eGFR but not baseline eGFR correlated with the eGFR slope. This observation is in agreement with our previous report [10], in which the eGFR slope had no correlation with baseline eGFR. The kidney function remains well preserved for many years but decreases rapidly at a later stage [1, 17]. This characteristic profile of renal function progression is explained by a compensatory adjustment for the loss of GFR. Compensatory adjustments make the decline in GFR slow or close to zero until certain stages [1]. GFR is maintained within the normal range despite decreased renal plasma flow in children and young adult patients with ADPKD [18–20]. In early stages, the decrease in renal plasma flow due to structural distortion in ADPKD is partially compensated for by an increased glomerular filtration fraction to renal plasma flow, but these adaptations eventually prove inadequate and kidney function starts to decline at a faster rate [21]. Those observations and hyperfiltration hypothesis are collectively in accordance with the present finding that the eGFR slope becomes more negative as eGFR decreases (Table 2).

The eGFR slope is relatively constant in relation to age (Fig. 4b). In our previous study, changes of reciprocal creatinine in 106 patients plotted against age showed that the progression patterns of renal function deterioration were different among patients [10]. Individual variation in renal functional progression might be a parallel characteristic to the wide distribution of kidney size growth, as shown in Fig. 3. Due to individual differences, the mean yearly change in eGFR (eGFR slope) as a whole patient group seemed to be constant, at least after $\sim$30 years of age.

TKV increases each year in most patients with ADPKD (Fig. 3), but no significant correlation between age and TKV was seen in the present study (Table 3). Similar findings were reported in the CRISP study [4]. The reason for this insignificant correlation between TKV and age is probably the wide individual variation in TKV. It is interesting to note that the TKV slope was constant at all ages, but the %TKV slope and log-TKV slope decreased as age advanced (Table 3; Fig. 5d). This finding has already been reported with the slopes expressed as a percent per year being significantly lower in the older age group ($p = 0.02$) [4]. The mechanism of this saturation-like phenomenon is speculated as follows—the rate of kidney volume enlargement (ml/year) is constant throughout life (Table 3), but the growth rate (%/year) becomes lower

Table 2 Functional and volume parameters in relation to chronic kidney disease (CKD) stages according to the final measurement of the estimated glomerular filtration rate (eGFR)

	CKD stage according to the final eGFR (ml/min/1.73 m^2) measurement				*p* value
	Stages 1 and 2 ≥60	Stage 3 59–30	Stage 4 29–15	Stage 5 <15	
N (men:woman)	31 (10:21)	15 (5:10)	11 (3:8)	7 (3:4)	
Observation period (months)	40.2 (11.5)	42.3 (10.2)	34.5 (11.9)	40.0 (9.1)	NS
Baseline age (years)	39.8 (13.7)	53.3 (11.0)	56.4 (11.3)	50.7 (11.4)	<0.01
Systolic BP on treatment (mmHg)	118.9 (10.6)	133.2 (11.3)	133.5 (19.4)	137.1 (17.7)	<0.01
Diastolic BP on treatment (mmHg)	77.2 (6.6)	81.0 (4.9)	80.3 (10.2)	82.3 (11.3)	NS
Urine protein excretion (mg/day/1.73 m^2)	62.3 (96.1)	124.6 (119.1)	223.7 (267.6)	1,102.7 (1,727.6)	<0.01
Kidney function					
Baseline eGFR (ml/min/1.73 m^2)	82.1 (18.2)	52.7 (10.7)	33.0 (6.7)	21.9 (13.5)	<0.01
Final eGFR (ml/min/1.73 m^2)	82.5 (19.4)	46.5 (8.6)	24.2 (3.1)	7.8 (3.7)	<0.01
eGFR slope (ml/min/1.73 m^2/year)	0.18 (3.47)	−0.74 (3.95)	−2.95 (2.38)	−3.88 (2.89)	<0.01
Baseline Ccr (ml/min/1.73 m^2)	114.3 (30.7)	85.1 (17.8)	48.6 (7.0)	39.5 (19.4)	<0.01
Ccr slope (ml/min/1.73 m^2/year)	−2.11 (11.74)	−4.04 (3.49)	−4.62 (7.96)	−9.59 (3.67)	NS
Baseline 1/Creatinine (ml/mg)	143 (27)	103 (20)	70 (15)	42 (19)	<0.01
Kidney volume					
Baseline TKV (ml)	1,192.0 (457.9)	1,394.3 (499.9)	2,693.0 (1,112.8)	2,871.4 (1,362.4)	<0.01
Final TKV (ml)	1,440.9 (576.7)	1,689.1 (618.4)	3,103.7 (1,377.2)	3,855.3 (2,129.5)	<0.01
TKV slope (ml/year)	73.8 (51.8)	75.0 (68.0)	148.6 (146.9)	279.6 (234)	<0.01
% TKV slope (%/year)	6.25 (3.86)	5.16 (4.74)	4.80 (3.14)	7.69 (7.09)	NS
log-TKV slope (ml/year)	0.0240 (0.0140)	0.0244 (0.0260)	0.0116 (0.0268)	0.0273 (0.0277)	NS
Baseline ht-TKV (ml/m)	724.7 (279.3)	862.1 (268.6)	1,681.6 (718.7)	1,661.8 (787.9)	<0.01
Baseline bs-TKV (ml/m^2)	714.2 (267.4)	890.4 (257.0)	1,729.0 (764.8)	1,623.5 (784.9)	<0.01
Baseline log-TKV (log[ml])	3.044 (0.1759)	3.109 (0.1600)	3.396 (0.1825)	3.402 (0.257)	<0.01

Numbers are the mean and standard deviation (in parentheses).

Slopes are calculated by regression analysis of each patient. Urine protein excretion and Ccr were measured from 24-h urine. CKD stage 1 and 2 are combined. *p* values were calculated by ANOVA

BP blood pressure, *CKD* chronic kidney disease, *eGFR* glomerular filtration rate estimated by Japanese MDRD equation, *Ccr* creatinine clearance, *TKV* total kidney volume, *ht-TKV* TKV divided by height (m), *bs-TKV* TKV divided by body surface area (m^2), *log-TKV* log-converted TKV

Table 3 Correlation coefficient (*r*) between age and kidney volume, function and their slopes

r between parameters and age at final measurement			*r* between each parameter slope and age at final measurement		
	r	*p* value		*r*	*p* value
TKV (ml)	0.1264	NS	TKV slope (ml/year)	−0.0979	NS
% TKV (%/year)	–	–	% TKV slope (%/year)	−0.3923	<0.01
ht-TKV (ml/m)	0.1526	NS	ht-TKV slope (ml/m/year)	−0.0945	NS
bs-TKV (ml/m^2)	0.1894	NS	bs-TKV slope (ml/m^2/year)	−0.0545	NS
log-TKV (log[ml])	0.1774	NS	log-TKV slope (log[ml]/year)	−0.4002	<0.01
1/Cre (ml/mg)	−0.5097	<0.001	1/Cre slope (ml/mg/year)	−0.1585	NS
eGFR (ml/min/1.73 m^2)	−0.6027	<0.001	eGFR slope (ml/min/1.73 m^2/year)	−0.0809	NS
Ccr (ml/min/1.73 m^2)	−0.436	<0.001	Ccr slope (ml/min/1.73 m^2/year)	−0.1592	NS

Correlation coefficients (*r*) are calculated between each parameter and final age.

TKV total kidney volume, *ht-TKV* TKV divided by height (m), *bs-TKV* TKV divided by body surface area (m^2), *log-TKV* log-converted TKV, *Cr* creatinine, *eGFR* estimated glomerular filtration rate by Japanese MDRD equation, *Ccr* creatinine clearance

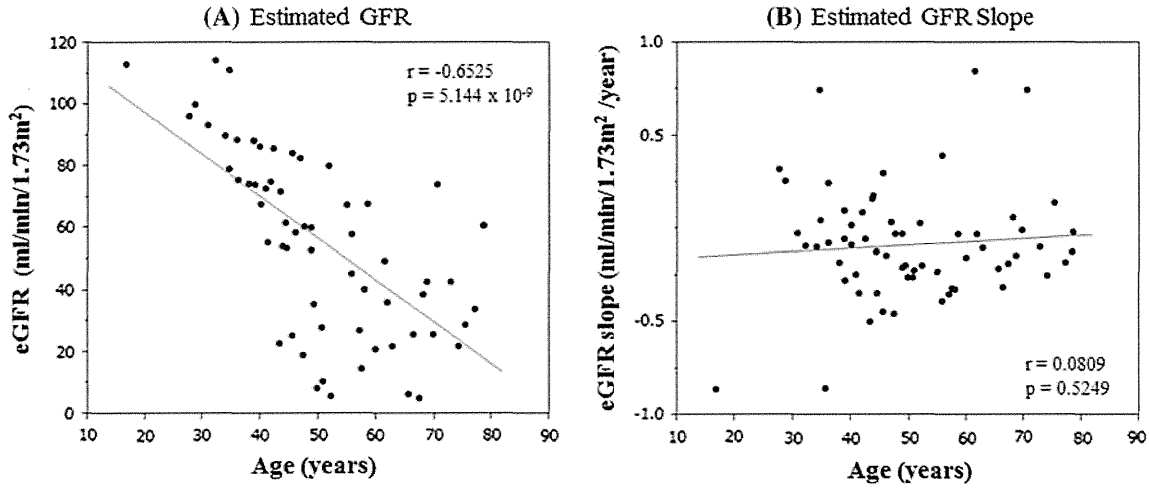

Fig. 4 **a** Correlation coefficient (r) between eGFR and age is highly significant. Age and eGFR are those measured at the final time. **b** There was no significant correlation coefficient (r) between age and the slope of eGFR. Age is at the final measurement

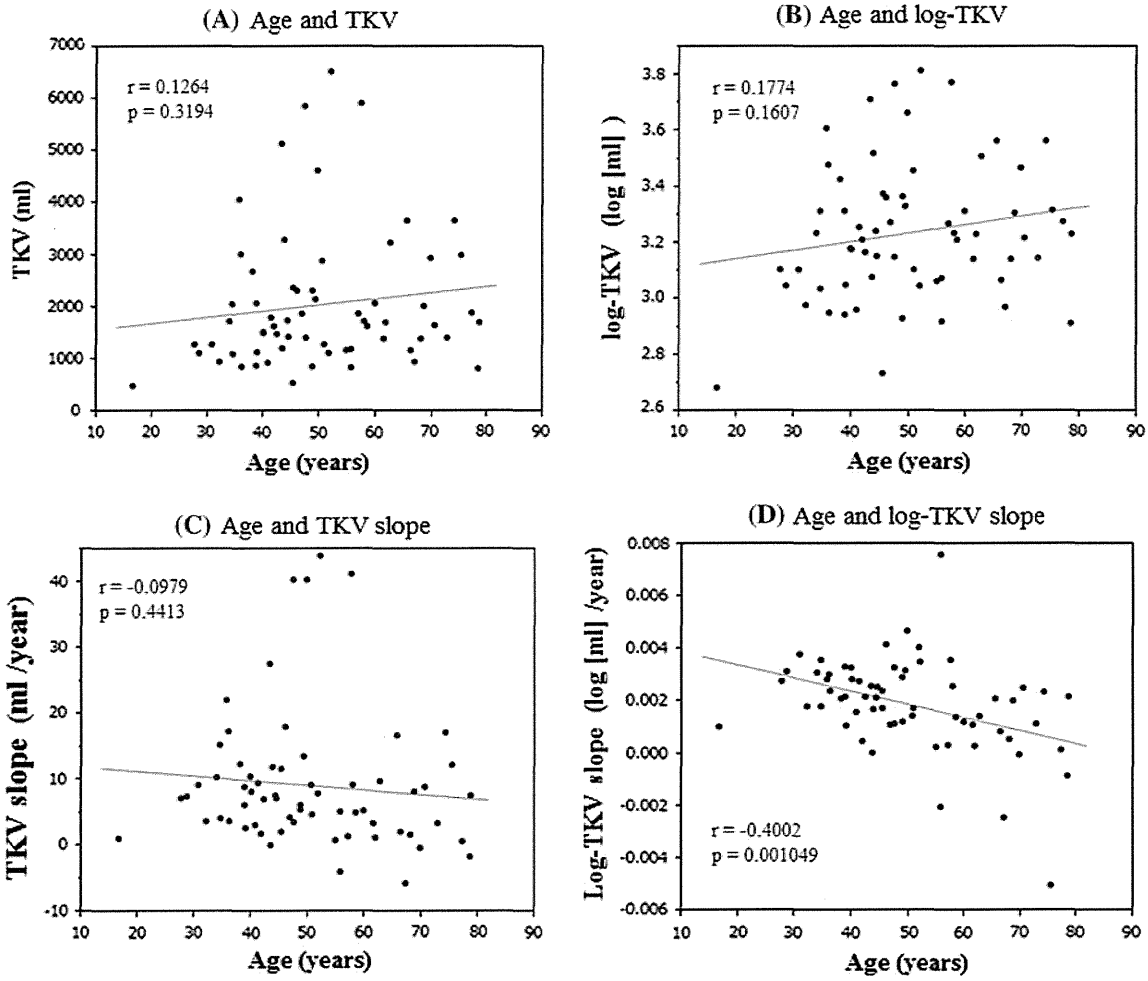

Fig. 5 The correlation coefficients (r) between age and TKV **a** and between age and log-TKV **b** are not significant. **c** The TKV slope tends to decrease as age advances, but r between age and TKV slope is not significant. **d** The log-TKV slope decreased significantly as age increased. The r between age and log-TKV slope is significant ($p < 0.01$). Age, TKV and log-TKV are final measurements

because the denominator (kidney volume) increases every year. The same explanation is applicable to log-converted kidney volume.

The highly significant correlation between baseline as well as final TKV and TKV slope is an obvious result of a large kidney being the consequence of a rapid increase in kidney volume. Although genotype was not determined in the present study, it is known that faster growth is generally associated with PKD1 genotype [4]. A large kidney volume was associated with a more rapid declining slope of iothalamate-measured GFR as well as of eGFR in the present study (Fig. 2a), indicating that a large kidney volume is associated with decreased kidney function [4]. Recently, Chapman et al. reported that baseline ht-TKV $\geq$600 cc/m predicted the risk of developing renal insufficiency within 8 years [5]. The present study is not long enough to quantitatively predict the risk of renal insufficiency but supports the view that TKV is a prognostic biomarker in ADPKD.

In summary, this study confirmed that TKV is a clinically meaningful surrogate marker in ADPKD because it correlates with kidney function and predicts functional disease progression. Patients with larger TKV are at higher risk of developing ESRD.

Limitations of this study

Kidney function was not measured directly, such as by inulin clearance. Twenty-four-hour urine creatinine clearance is known to have a relatively large variance due to method imprecision and tubular creatinine secretion [22]. eGFR and reciprocal creatinine are affected by non-GFR factors such as creatinine production and tubular secretion. The patient number is limited and the observation period is not long enough to predict disease progression.

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Conflict of interest All the authors have declared no competing interests.

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厚生労働科学研究費補助金　難治性疾患等克服研究事業（難治性疾患克服研究事業）

資　料

第１回日本腎臓学会／KDIGO ガイドラインカンファレンス
1st JSN/KDIGO Guideline Implementation Conference

開催場所： 東京医科歯科大学 鈴木章夫記念講堂
日　時： 2013年10月6日 (日) 10：00 ～17：00

9:00- 10:00　開場 / 受付

10:00- 10:15　開会の辞:　松尾清一（日本腎臓学会理事長・厚生労働省進行性腎障害調査研究班長）
塚本雄介（KDIGO 理事）
KDIGO
厚生労働省
国立保健医療科学院

Nephrotic syndrome #1　座長: **和田隆志（金沢大学）, Daniel Cattran (University of Toronto)**
10:15- 10:35　“日本におけるネフローゼ症候群患者の実態
-日本ネフローゼ症候群コホート研究の結果から-”
演者：丸山彰一（名古屋大学）
10:35- 10:55　“日本におけるネフローゼ症候群の治療戦略- KDIGO-糸球体腎炎ガイドラインと JSN-NS ガイドライン-”
演者：西慎一（神戸大学）
10:55-11:05　質疑応答(10 分)

Nephrotic syndrome #2　座長:**岡田浩一（埼玉医大), Bertram Kasiske (University of Minnesota)**
11:05- 11:25　“KDIGO Guideline Recommendations on the Management of Minimal Change Disease and Focal Segmental Glomerulosclerosis”
演者：Bertram Kasiske (University of Minnesota)
11:25- 11:45　“KDIGO Guideline Recommendations on the Management of Idiopathic Membranous Nephropathy”
演者：Daniel Cattran (University of Toronto)
11:45-11:55　質疑応答(10 分)

11:55-12:05　休憩 (10 分)

Nephrotic syndrome #3　座長: **和田隆志、岡田浩一**
12:05-12:30　症例提示１および討論
12:30-12:55　症例提示２および討論

12:55-14:00　昼食 (65 分)

Hypertension #1　座長: **内田信一（東京医科歯科大)、Vlado Perkovic (The University of Sydney)**
14:00- 14:20　“Target BP levels in JSN Evidence-based Clinical Practice Guideline for CKD (JSN-CKD GL 2013)”
演者：長谷部直幸（旭川医大）
14:20-14:40　“First-line anti-hypertensive drugs in JSN Evidence-based Clinical Practice Guideline for CKD (JSN)”
演者：田村功一（横浜市大）
14:40-14:50　質疑応答(10 分)

15:15-15:30　休憩 (15 分)

Hypertension #2　座長: 井関邦敏（琉球大)、**David Wheeler (University College in London)**
14:50- 15:10　“KDIGO Blood Pressure Guideline Presentation-1”
演者：Vlado Perkovic (The University of Sydney)
15:10- 15:30　“KDIGO Blood Pressure Guideline Presentation-2
演者：David Wheeler (University College in London)
15:30-15:40　質疑応答(10 分)

15:40-15:55　break (15 min)

Hypertension #3　座長: **内田信一、井関邦敏**
15:55-16:20　症例提示１および討論
16:20-16:45　症例提示２および討論

16:45- 16:50　閉会の辞:木村健二郎 （日本腎臓学会CKD 診療ガイドライン作成委員長)

開会あいさつ 腎障害の研究成果を社会で共有するために

腎機能を悪化させる主原因は「糖尿病」

名古屋大学 腎臓内科学 教授
松尾 清一先生

現在、厚生労働科学研究費補助金難治性疾患克服研究事業の一環として、進行性腎障害に関する調査研究が進められています。私は、その調査研究班の班長を務めさせていただいており、臨床研究をはじめ治療指針のガイドライン作成、市民の皆さんへの啓発活動などを行っています。こうした取り組みの背景には、歯止めがかからない腎臓病の増加があります。本日は私たちの研究の成果を発表いたしますが、真の目的は腎臓病について皆さんに理解を深めていただくことにあります。そのため「腎臓病・糖尿病とともに生きる」をテーマに掲げました。というのも腎機能を悪化させる一番の原因は糖尿病であり、腎臓病と糖尿病は切っても切れない関係だからです。健康な方は今の健康をできるだけ長く維持するために、そして腎臓を患っている方は明日からの病気療養に、ぜひ先生方のお話を役立てていただきたいと思います。（談）

講演1 そら豆形の腎臓は肝心かなめの臓器

慢性腎臓病（CKD）は新しい国民病

名古屋大学 CKD地域連携システム寄附講座 准教授
安田 宣成先生

腎臓は腰のあたりに2個あり、重さはひとつ約150グラムです。毎日150～200リットルほどの尿のもとを作り、老廃物を体外に排泄（はいせつ）して体の中をきれいにしてくれます。他にも水分や塩分の調節をはじめ、血圧を調整したり、ホルモンを分泌して貧血を防いだり、骨を健康に保つといった働きがあります。

実は、日本は世界第2位の透析大国です。日本透析医学会統計調査速報値によると、2012年末の透析患者数は30万9946人。原因となる疾患別では糖尿病性腎症が44.1％。減少傾向にあるものの、慢性糸球体腎炎が19.4％と続いています。

透析に至らないためには、慢性腎臓病（CKD）という前段階から適切な治療を受けることが大切です。①タンパク尿など腎臓の障害がある②腎臓の機能（GFR）が60％未満になった状態のどちらかが3カ月以上続くと、CKDと診断されます。国内のCKD患者数は1330万人、成人8人にひとりと推計されています。CKDは心筋梗塞（こうそく）や脳卒中などの危険が約3倍高くなりますが、自覚症状がほとんどないため、尿検査や血液検査などの健康診断が欠かせません。

もちろん血圧の管理も必要です。130／80ミリメートルHg未満を降圧目標にしてコントロールしてください。（談）

講演2 共通の願いは健康寿命を延ばすこと

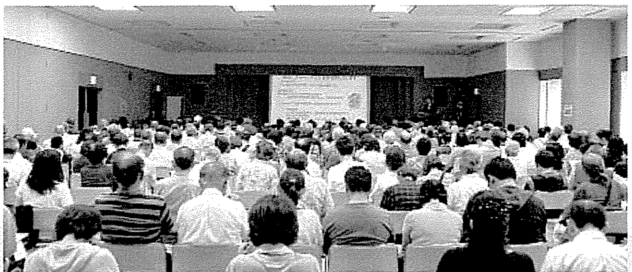
生活習慣の改善と、家庭での血圧測定のすすめ

金沢大学 血液情報統御学 教授
和田 隆志先生

人工透析に移行する腎臓病のうち、最も多いのが「糖尿病性腎症」です。糖尿病そのものの増加に加え、糖尿病性腎症の進行が十分にくいとめられていないことが主な原因です。糖尿病が強く疑われる人は890万人ですが、対して医療機関受診率は約半数。糖尿病性腎症は進行しないと自覚症状が現れないため、見過ごされているわけです。早期発見には、尿アルブミンの測定が必要です。アルブミンとは、腎臓のろ過機能が障害されると尿中に漏れ出るタンパク質の一種。3回中2回以上、30ミリグラム／グラムクレアチニン（mg/gcr）以上が検出されると、早期腎症と診断されます。

では、予防と進行を抑えるには何が重要なのか。まずは生活習慣の改善と血糖コントロールです。食事療法と運動療法を基本とし、効果不十分であれば薬物療法を追加します。運動については、近年「サルコペニア（ラテン語でサルコ＝筋肉、ペニア＝減少という意味）」が注目を集めています。筋肉量が減少すると転倒骨折だけでなく、動脈硬化や糖尿病などのリスクも高まります。歩行やジョギングなどのトレーニングは、毎日30分以上が目標です。

また、ぜひ実行していただきたいのが家庭血圧の測定です。病院では正常でも、家庭で血圧が上昇する「かくれ高血圧」「仮面高血圧」は少なくありません。糖尿病に高血圧が加わると、脳卒中や心筋梗塞を起こしやすくなります。3日坊主も、10回行えば1カ月。生活習慣を見直して、健康寿命をのばしましょう。（談）



～腎臓病・糖尿病とともに生きる～

生活習慣の見直しと定期検査で腎臓病を予防・改善しましょう

紙上採録｜9/29朝日ホールにて開催

市民公開講座

講演テーマ／講演者

- 講演1「あなたの腎臓、大丈夫?」 安田宣成先生
- 講演2「糖尿病からあなたの腎臓を守りましょう!」 和田隆志先生
- 講演3「腎炎・ネフローゼってどんな病気?」 丸山彰一先生
- 講演4「まんがで知る多発性嚢胞腎（のうほうじん）」 武藤智先生
- 講演5「腎臓にやさしい食事」 鈴木富夫先生

9月29日、朝日ホールにて市民公開講座「腎臓病・糖尿病とともに生きる」が開催されました。腎臓病の専門医を招き、腎臓の働きから慢性腎臓病の予防や治療、腎臓を守る食事のポイントなど、さまざまな視点から講演。専門的な内容もスライドを使って分かりやすくお話いただき、来場者の皆さんはメモをとりながら熱心に耳を傾けておられました。私たちにとって身近な腎臓病について、理解を深める有意義なセミナーとなりました。

＊主催：厚生労働省科学研究費補助金難治性疾患等克服（難治性疾患克服）研究事業「進行性腎障害に関する調査研究」班／厚生労働省科学研究費補助金難治性疾患等克服（腎疾患対策）研究事業「糖尿病性腎症ならびに腎硬化症の診療水準向上と重症化防止にむけた調査・研究」班
＊協賛：MSD、興和創薬、三和化学研究所、第一三共、大日本住友製薬、帝人ファーマ、バクスター　＊後援：朝日新聞社広告部

講演3 透析を回避するには早期発見・早期治療

「腎炎・ネフローゼ」は慢性腎不全の前兆

名古屋大学 腎臓内科 准教授
丸山 彰一先生

「腎炎・ネフローゼ」は血液をふるいにかける糸球体が壊れて、体に必要なタンパク質や赤血球が尿に漏れ出てしまう病気です。代表的な疾患には「IgA腎症」「ネフローゼ症候群」「急速進行性糸球体腎炎」があります。まず慢性糸球体腎炎で最も多いのが、IgA腎症です。通常は無症状ですが、風邪をひくとコーラ色の尿が出ることがあります。背中から細い針を刺して腎臓の一部を採取する腎生検の約35％は、IgA腎症と診断され、腎生検後20年で約40％が末期腎不全に至ります。ただし残り60％のうち、半分ほどは自然治癒します。多くは、健診で尿潜血やタンパク尿を指摘されて発見されます。

ネフローゼ症候群は、年間約4000例の新規発症が報告されています。症状はタンパク尿や低アルブミン血症、むくみ、高コレステロール血症など。治療は長期にわたりますが、7割近くはほぼ完治します。食生活では、タンパク質の過剰摂取を避けてください。

急速進行糸球体腎炎は、糸球体腎炎のうち最も重症な腎炎の一種です。短い期間で腎機能が低下して20～25％の方が2年以内に死亡、あるいは末期腎不全で透析になります。熱や倦怠（けんたい）感、しびれ、関節痛が数週間続いたら病院を受診してください。腎炎・ネフローゼも、早期診断と早期治療が重要です。（談）

講演4 脳動脈瘤（どうみゃくりゅう）や高血圧を合併しやすい難治性疾患

腎臓に水袋ができる、遺伝性の多発性嚢胞腎（のうほうじん）

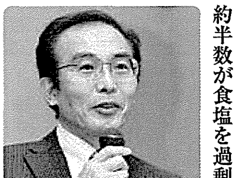
帝京大学 泌尿器科学教室 准教授
武藤 智先生

「多発性嚢胞腎」とは、両側の腎臓に嚢胞という水の入った袋が多発する遺伝性の病気です。年齢とともに嚢胞の数と大きさが増え、ある時点から老廃物を排泄する腎臓の働きが徐々に低下します。変異を起こす原因遺伝子は特定されていますが、同じ遺伝子変異を受け継いでも発症・進展には違いが生じます。しかしながら70歳までに半数の方が、人工透析を必要とするといわれています。

合併症は高血圧をはじめ、肝嚢胞、脳動脈瘤、臍（へそ）ヘルニアなどが挙げられます。特に高血圧は比較的早期から発症するため、多発性嚢胞腎では心臓血管病が最も多い死亡原因となっています。日常生活では塩分を制限し、尿路感染・結石を予防するために十分な水分を摂取するようにしてください。お子さんについては、一定条件を除いて早期診断はすすめられていませんが、状況を把握するための診断は検討してもいいでしょう。（談）

講演5 減塩食でタンパク質とエネルギーは適正に

約半数が食塩を過剰摂取。1食2グラムに



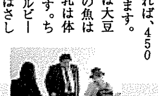
名古屋大学 栄養管理部 副部長
鈴木 富夫先生

腎臓にやさしい食事のポイントは、①塩分をできる限り少なめに②タンパク質の取り過ぎに注意③適切なエネルギー摂取の維持です。CKDの食塩摂取量は、1日3グラム以上6グラム未満。適正エネルギー量は、1日25～35キロカロリー／体重キログラム（※1）です。摂取タンパク質量はCKDのステージによって異なり、1日0.6～1.0グラム／体重キログラムと示されています。

中でも大切なのは、塩分制限です。高血圧を軽減して体内の水分バランスを保ち、降圧薬の効き目を向上させるといった効果があり、腎臓の保護につながります。実は世代別に食塩摂取量をみると、最も多いのが60代です。全体では、約半数の人が10グラム以上を摂取しています。減塩のコツは、①表面に味をつける（加工食品・漬物を控える）②めん類を控える（つゆは極力残す）③汁物は半量にして1日1回。調理法は煮込むより塩やしょうゆが直接、舌に触れる味付けのほうが有効です。

1日のご飯は、体重1キログラムを食品1グラムと見なし、標準体重の9～10倍程度が適量です。50キログラムの人であれば、450～500グラムとなります。副食は朝食の卵または大豆製品、昼食の肉、夕食の魚は体重と同量にし、牛乳は体重の2倍を摂取します。ちなみに肉60グラムはカルビー4枚ほど、魚60グラムはさしみ4～5切れが目安です。栄養失調にならないよう定期的に体重を測定して、無理のない食事を継続してください。（談）

※1／体重キログラム当たりでの記述における体重とは標準体重のことであり、現状の体重ではありません。標準体重＝身長（メートル）×身長（メートル）×22



腎臓にやさしい食品の試供品コーナーも盛況だった

減塩・低タンパクなどの健康食品も紹介

会場前のオープンスペースには、製薬メーカーや食品会社などの展示ブースが設置されました。生活習慣病の予防法や治療法をまとめたパンフレット類をはじめ、タンパク質の含有量を減らしたごはんなど、腎臓にやさしい健康食品やエネルギー補助食品などを紹介。減塩しょうゆや減塩だしつゆ、ノンオイルドレッシングといった試供品も提供され、セミナー開始前から来場者が長い列をつくるほどの盛況ぶりでした。皆さんの健康に対する意識の高さを実感しました。

厚生労働科学研究費補助金難治性疾患克服研究事業

進行性腎障害に関する調査研究

お問い合わせフォーム

文字サイズを変更 | 小 | 中 | 大 |

【IgA腎症・急速進行性糸球体腎炎・難治性ネフローゼ症候群・多発性嚢胞腎】症例数が少なく、原因が不明で、治療法が確立されていない疾患で、長期間生活に支障を及ぼすものについて、研究班を設置し、原因の究明・治療法の確立に取り組む、厚生労働省の事業。

検索

メインメニュー

最近のブログ記事

【病気の解説】

- IgA腎症
  IgA nephropathy
- 急速進行性糸球体腎炎
  Rapidly progressive glomerulonephritis; RPGN
- 難治性ネフローゼ症候群
  Nephrotic syndrome
- 多発性嚢胞腎
  Polycystic kidney disease

症例数が少なく、原因が不明で、治療法が確立されていない疾患で、長期間生活に支障を及ぼすものについて、研究班を設置し、原因の究明・治療法の確立に取り組む、厚生労働省の事業。

→ 詳細を見る

研究成果報告

＜平成23年度　研究成果報告書＞

→こちらをご覧ください。

＜平成24年度　研究成果報告書＞

→こちらをご覧ください。

→平成24年度研究報告書はこちらをごらんください。(PDFファイル)

2013年7月24日

患者様向けQ&A～急速進行性糸球体腎炎

【参考】解説スライド

急速進行性糸球体腎炎(RPGN)についての解説です。

進行性腎障害に関する調査研究班 班長 松尾清一

国民の皆さんや腎臓病と闘っておられる患者さんに貢献できるよう、活動しています。

→ 研究班班長のご挨拶

月別 アーカイブ

患者様向けQ&A