Letter to the editor

The Measurement of Inferior Vena Cava Diameter for Assessing Volume Status in Autosomal Dominant Polycystic Kidney Disease

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Dear editor,

Hypertension is an early and frequent manifestation in ADPKD, being the presenting clinical finding in 13 up to 81% of patients [1,2]. Although the underlying mechanisms for the rise in blood pressure in individuals with ADPKD are still unclear, two principal mechanisms as elevated plasma volumes [3] and increased plasma renin activity [4] are reported. Since invasive methods for evaluation of fluid status such as measurement of central venous pressure cannot be used routinely, the measurement of the inferior vena cava diameter (IVCD) by echocardiography has been suggested to reflect the intravascular volume in adult haemodialysis patients and to correlate well with the other methods for estimation of fluid volume [5,6]. Also a significant correlation was found between IVCD and blood pressure [7]. The present cohort study was undertaken for assessing the correlation of volume status with IVCD in our ADPKD patients.

A hundred ADPKD patients included in the study were divided in two groups: first group of 52 patients without hypertension (normotensive patients) and second group of 48 patients with hypertension (hypertensive patients). Hypertension was defined as BP greater than or equal to

140 mmHg for systolic BP and greater than or equal to 90 mmHg for diastolic BP [8]. Eighty patients had normal renal function (GFR > 60 ml/min). All hypertensive patients were treated with one or more antihypertensive drugs. Only three patients received diuretics in small doses. All vasoactive medications were stopped 48 hours prior the IVCD measurement. Knowing that patients with ADPKD can be complicated with valvular abnormalities, in this study we didn't include patients with these abnormalities. IVCD was visualized two-dimensionally and measured by Doppler-echocardiography three times during a period of 18 months (every six months, divided in three periods). The anteroposterior IVCD was measured using 2-dimensional and Doppler recordings 1.5 below the diaphragm in the hepatic segment in the supine position after 5-10 min of rest during normal expiration and inspiration. The same examiner performed all the measurements of IVCD. Mean IVCD was expressed as (IVCD in inspiration+IVCD in expiration)/2. The referee values for IVCD were those defined by Mandelbaum et al. (normal range 8-11 mm/m²) [6]. Results are reported as mean±SD. P values less or equal to 0.05 were considered statistically significant.

Table 1. Demographic and clinical data of patients

Demographic variable	Normotensive patients	Hypertensive patients	
Number of patients	52	48	
Gender (M/F)	23/29	22/26	
Age (years)	46.4±5.7	49.1±8.9	
Smoking (Yes/No)	12/40	19/29	
Renal function			
- normal renal function	42 patients	38 patients	
(GFR > 60 ml/min)	12 patients		
- chronic renal failure	10 patients	10 patients	
(GFR < 60 ml/min)	10 patients	10 patients	
Mean blood pressure values			
(mmHg)			
Mean systolic pressure	131.2	162.5	
Mean diastolic pressure	88.4	97.7	
Body weight (kg)	74.2±5.7	71.6±4.3	
Body height (cm)	162.7 ± 9.4	164.8 ± 10.2	

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The demographic and clinical data are presented in Table 1. The mean IVCD result was $9.8\pm2.3~\text{mm/m}^2$ in the first group of normotensive patients, while in the second group of hypertensive patients it was $12.4\pm0.9~\text{mm/m}^2$ (p< 0.005) (Table 2). An interesting fact was that in 18 patients of the first group (normotensive patients), the mean IVCD result increased and 13 of these patients developed hypertension after a mean period of $2.4\pm1.2~\text{years}$. In

addition, five patients from the last group developed left ventricular hypertrophy (LVH) after some years.

These results indicate the importance of fluid overload in the pathogenesis of hypertension in ADPKD patients. Since the volume expansion is a pathogenetic mechanism for hypertension in ADPKD patients, IVCD would be accurate assessments of the volume state in ADPKD patients. It was proved that hypertension plays an important role in cardiovascular morbidity including LVH and mortality [9].

Table 2. IVCD measurements

	Normotensive patients	Hypertensive patients	P
Mean values of IVCD	$9.8 \pm 2.3 \text{ mm/m}^2$	$12.4 \pm 0.9 \text{ mm/m}^2$	< 0.001
First measurement (mean values)	$9.3 \pm 2.5 \text{ mm/m}^2$	$11.9 \pm 1.3 \text{ mm/m}^2$	< 0.001
Second measurement (mean values)	$9.5 \pm 1.8 \text{ mm/m}^2$	$11.4 \pm 1.5 \text{ mm/m}^2$	< 0.01
Third measurement (mean values)	$10.1 \pm 1.9 \text{ mm/m}^2$	$13.1 \pm 1.4 \text{ mm/m}^2$	< 0.001

The development of LVH after some years in normotensive patients supports the fact revealed from Timio *et al.* that LVH in ADPKD patients can be caused by hemodynamic initial burden [10].

Although promising, this method has several limitations. One of the obstacles is the lack of normal values for IVCD in adults. In a study of 86 healthy adults the diameter of IVCD varied widely and did not correlate with the height, weight or body surface area (BSA) [6]. Although it is reasonable that IVCD correlates with BSA, the precise relationship is not known because other factors such as the heart rate, blood pressure and treatment with antihypertensive drugs may influence IVCD.

In conclusion, IVCD is a non-invasive and relatively convenient method for obtaining a good correlation with the intravascular fluid status in ADPKD patients. It may serve as an additional reliable parameter in estimation of the hydration status in ADPKD patients, but it cannot be used as a single parameter for fluid status. The increased IVCD could be an early predictor of developing hypertension and sometimes aslo LVH.

Conflict of interest statement. None declared.

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