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Data Article

Data on the assessment of LV mechanics by speckle tracking echocardiography in ADPKD patients

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ABSTRACT

In this article, we report anthropometric, clinical and laboratory data from Autosomal Dominant Polycystic Kidney Disease (ADPKD) patients with mild to moderate renal dysfunction and normal LV ejection fraction and from age- and sex-matched healthy controls and renal controls. Factors influencing LV untwisting rate in the group of ADPKD patients are also reported. For further interpretation and discussion please refer to the research article "Left ventricular dysfunction in ADPKD and effects of Octreotide-LAR: a cross-sectional and longitudinal sub study of the ALADIN trial" (Spinelli et al., 2018) [1].

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Specifications table

Subject area	*Medicine*
More specific subject area	*Echocardiography*
Type of data	*Tables and figures*
How data was acquired	*Two-dimensional echocardiography was performed by using a digital ultrasonic device system (Vivid 7, GE Vingmed Ultrasound, Horten, Norway)*
Data format	*Analyzed*
Experimental factors	*Speckle-tracking echocardiography analysis was performed offline by using dedicated software (EchoPac PC version 110.0.0; GE Vingmed Ultrasound).*
Experimental features	*All patients underwent speckle-tracking echocardiography at baseline. ADPKD patients were further evaluated after 3 years of treatment with somatostatin-analogue or placebo*
Data source location	*Naples, Italy*
Data accessibility	*Data is within this article*
Related research article	*Left ventricular dysfunction in ADPKD and effects of Octreotide-LAR: a cross-sectional and longitudinal substudy of the ALADIN trial, Spinelli* et al., *Int J Cardiol, 2018,* [1].

Value of the data

- This data provides information on association between clinical and echocardiographic parameters of left ventricular diastolic function and potential influence on such parameters of the treatment with somatostatin-analogue octreotide long-acting-release in patients with Autosomal Dominant Polycystic Kidney Disease and normal ejection fraction.
- To assess reproducibility of speckle-tracking measurements, intra-observer and inter-observer variability values are provided.
- These data are valuable as they prove the usefulness of speckle-tracking echocardiography in Autosomal Dominant Polycystic Kidney Disease to detect very early impairment of left ventricular function that is missed with conventional echocardiography and may stimulate further research.

1. Data

Anthropometric, clinical hemodynamic, laboratory and therapy data from and healthy controls, renal controls and Autosomal Dominant Polycystic Kidney Disease (ADPKD) patients, categorized according to treatment with octreotide-long acting release (LAR) or placebo, are provided in Tables 1 and 2. Diabetes mellitus was found only in renal controls (6 out 34 patients, 17.6%), while the prevalence of systemic arterial hypertension was comparable between ADPKD and renal controls. Laboratory data were similar in ADPKD patients and their healthy and renal controls, by excluding higher fasting serum glucose levels and urinary protein excretion in renal controls. Likewise, anthropometric, clinical and laboratory characteristics were very much the same between the 16 ADPKD patients randomized to octreotide-LAR therapy and the 18 randomized to placebo.

Data on association of clinical and echocardiographic variables with left ventricular (LV) untwisting rate in the group of ADPKD patients by univariate and multivariate linear regression analyses are provided in Table 3. LV early diastolic untwisting rate was associated with left ventricular mass (LVM) index and diastolic blood pressure (BP) at univariate analysis. These associations did not change at multivariate analysis.

Data on adjusted linear regression analysis showing the relationships, in ADPKD patients, between LV untwisting rate at final visit and the following variables: age, body surface area, glomerular

Table 1
Anthropometric and clinical parameters from Healthy Controls, Renal Controls and ADPKD Patients (overall and categorized according to treatment with octreotide-LAR or Placebo).

	Healthy Controls ($n = 34$)	Renal Controls ($n = 34$)	ADPKD patients		
			All patients ($n = 34$)	Octreotide-LAR ($n = 16$)	Placebo ($n = 18$)
Age (years)	34.8 ± 6.9	38.2 ± 8.6	35.8 ± 8.4	33.2 ± 8.4	38.1 ± 7.8
Men, n (%)	13 (38.2)	13 (38.2)	13 (38.2)	6 (37.5)	7 (38.8)
Height (cm)	170 ± 5	169 ± 8	167 ± 9.5	167 ± 10	168 ± 9
Weight (kg)	71 ± 8	71 ± 10	74 ± 16	73 ± 18	75 ± 13
Body Mass Index	25.0 ± 3.6	25.2 ± 4	25.8 ± 3.8	25.06 ± 3.8	26.5 ± 3.9
Diabetes mellitus, *n* (%)	0	6 (17.6) [*,b]	0	0	0
Hypertension, *n* (%)	0	24 (70.6)	22 (64.7)	10 (62.5)	12 (66.6)
Active smoker, *n* (%)	11 (32.3)	15 (44.1)	15 (44.1)	7 (43.7)	8 (44.4)
Family history of hypertension, *n* (%)	11 (32.3)	14 (41.2)	19 (55.9)	8 (50.0)	11 (61.1)
Family history of CAD, n (%)	8 (23.5)	15 (44.1)	14 (41.2)	6 (37.5)	8 (44.4)

Values are means ± SD for continuous variables, and absolute numbers (%) for categorical variables.
ADPKD = autosomal dominant polycystic kidney disease; LAR = Long-Acting-Release; CAD = coronary artery disease.
[*] $p < 0.01$versus healthy controls,
[b] $p < 0.01$ versus ADPKD,

filtration rate,heart rate, systolic BP, diastolic BP, octreotide-LAR treatment, are reported in Table 4. Details regarding the changes of untwisting rate in ADPKD patients are described and commented in Ref. [1].

2. Experimental design, materials, and methods

2.1. ADPKD patients selection criteria

All ADPKD patients consecutively included in the ALADIN trial [2] at the Outpatient Clinic of the Federico II University of Naples were evaluated for participation. They had to fulfill the selection criteria of the ALADIN trial [2]. For the specific purposes of the present dataset, patients with previous myocardial infarction, atrial fibrillation, cardiac valve disease or obstructive pulmonary disease were also excluded.

Data on ADPKD patients selection, are reported in Fig. 1. ADPKD patients were compared with age- and gender-matched healthy controls or equally-matched renal controls with non-cystic chronic kidney disease. LV function was assessed by speckle tracking echocardiography. For further details see Ref. [1]. Changes in LV function were compared in the 16 and 18 ADPKD patients randomized respectively to octreotide-LAR or placebo for three years [2].

2.2. Echocardiography

Two-dimensional echocardiography was performed by using a digital ultrasonic device system (Vivid 7, GE Vingmed Ultrasound, Horten, Norway) with patients lying in left lateral decubitus. Gray scale 4-chamber, 2-chamber and long- axis LV apical views as well as basal and apical LV short axis views were obtained at a high frame rate (60–80 frames/s). The basal level was marked as one showing the tips of mitral valve leaflets, the apical level was defined just proximal to the level with LV luminal obliteration at end systolic period.

Table 2
Heart rate, blood pressure, laboratory data and therapy from healthy controls, renal controls and ADPKD patients (overall and categorized according to treatment with octreotide-LAR or Placebo).

	Healthy Controls ($n = 34$)	Renal Controls ($n = 34$)	ADPKD Patients				
			All patients ($n = 34$)	Octreotide-LAR		Placebo	
				($n = 16$)		($n = 18$)	
				Baseline	Final visit	Baseline	Final visit
Heart rate (b/min)	66.7 ± 5.0	72.3 ± 8.1	74.2 ± 11.6	72.0 ± 13.0	74 ± 9.0	76.2 ± 10.0	71.0 ± 6.0
Systolic BP (mmHg)	119.2 ± 8.6	133.7 ± 21.6*	120.6 ± 11.3	122.2 ± 9.3	124.0 ± 15.0	119.2 ± 12.9	123.0 ± 13.0
Diastolic BP (mmHg)	74.3 ± 7.7	81.8 ± 11.0*	79.0 ± 8.0	79.4 ± 7.5	81.0 ± 10.0	78.6 ± 8.5	78.4 ± 6.0
Mean BP (mmHg)	89.3 ± 7.5	99.1 ± 13.9 *	92.8 ± 8.0 †	93.6 ± 6.5	94.0 ± 7.0	92.1 ± 9.3	93.0 ± 9.0
Fasting serum glucose (mg/dl)	84.0 ± 4.3	96.6 ± 8.5*	84.2 ± 4.7 †	84.8 ± 4.2	79.0 ± 7.0	83.6 ± 5.2	83.0 ± 7.0
Hemoglobin (g/dl)	14.1 ± 1.3	13.8 ± 1.4	13.9 ± 1.4	14.1 ± 1.4	13.0 ± 1.1	13.8 ± 1.4	12.6 ± 3.0
Hematocrit (%)	42.2 ± 3.9	41.4 ± 4.3	41.7 ± 3.8	41.8 ± 3.9	38.7 ± 3.8	41.6 ± 3.7	39.2 ± 3.8
Serum creatinine (mg/dl)	0.82 ± 0.19	1.23 ± 0.59‡	1.03 ± 0.37	0.94 ± 0.35	1.08 ± 0.6	1.11 ± 0.39	1.78 ± 1.10
Serum calcium (mg/dl)	9.5 ± 0.4	9.5 ± 0.4	9.5 ± 0.4	9.5 ± 0.4	8.7 ± 2.1	9.5 ± 0.3	8.8 ± 2.0
Serum phosphorus (mg/dl)	3.4 ± 0.6	3.3 ± 0.4	3.3 ± 0.5	3.4 ± 0.6	2.5 ± 1.6	3.2 ± 0.4	2.8 ± 1.2
Total cholesterol (mg/dl)	167.1 ± 21.1	182.6 ± 30.1	170.7 ± 35.4	180.1 ± 27.9	184.3 ± 28.6	162.3 ± 39.8	171.9 ± 29.6
HDL cholesterol (mg/dl)	57.2 ± 7.3	52.4 ± 10.0	51.6 ± 14.6	52.3 ± 17.2	56.6 ± 19.3	51.1 ± 12.4	51.6 ± 11.1
Triglycerides (mg/dl)	106.5 ± 55.9	114.1 ± 57.1	108.2 ± 51.0	119.3 ± 68.6	104.2 ± 50.0	98.3 ± 25.9	101.7 ± 49.4
PTH (pg/ml)	30.2 ± 7.6	34.1 ± 8.8	47.9 ± 24.0*,‡	49.0 ± 25.2	75.5 ± 65.9	46.9 ± 23.5	92.2 ± 116.6
GFR (mL/min/1.73 m^2)	120.8 ± 8.6	70.7 ± 28.4*	82.1 ± 26.2*	86.4 ± 23.6	80.7 ± 28.6	78.3 ± 28.5	71.1 ± 30.9
Urine proteins (g/24 h)	0	0.9 (0.72–1.15)	0.14 (0.03- 0.26)‡	0.12 (0.02–0.26)	0.14 (0.03–0.29)	0.14 (0.01- 0.56)	0.32§ (0.05–0.69)
ACE inhibitor	0	21 (61.8)	15 (44.1)	7 (43.8)	7 (43.8)	8 (44.4)	8 (44.4)
ARB	0	13 (38.2)	12 (35.3)	6 (37.5)	6 (37.5)	6 (33.3)	6 (33.3)
Statin	0	21 (61.8)	2 (5.9) ‡	1 (6.2)	1 (6.2)	1 (5.5)	1 (5.5)

Values are means ± SD or medians (interquartile range) for continuous variables, and absolute number (%) for categorical variables.
ADPKD = autosomal dominant polycystic kidney disease; LAR = long-acting-release; BP = blood pressure; HDL = high-density lipoprotein; PTH = parathyroid hormone; GFR = glomerular filtration rate; ACE = angiotensin converting enzyme; ARB = angiotensin II receptor blockers.

* $p < 0.05$ versus healthy controls.
‡ $p < 0.001$ versus healthy controls.
‡ $p < 0.001$ versus renal controls.
† $p < 0.05$ versus renal controls.
§ $p < 0.05$ final visit vs. baseline.

Table 3
Univariate and multivariate linear regression analysis showing associations of clinical and echocardiographic variables with LV untwisting rate in the group of ADPKD patients at inclusion in the ALADIN Trial.

	Univariate analysis		Multivariate analysis*	
	β (95% CI)	***p* Value**	**β (95% CI)**	***p* Value**
Age	0.015 (-0.746 – 0.811)	0.932		
Body Surface Area	0.005 (-28.310 – 29.079)	0.978		
GFR	-0.165 (-0.359 – 0.132)	0.352		
Systolic BP	-0.222(-0.920 – 0.208)	0.208		
Diastolic BP	-0.342 (-1.547 – -0.007)	0.048	-0.337 (-1.441 – -0.093)	0.027
LV end-diastolic volume	0.168 (-0.170 – 0.477)	0.342		
LV end-systolic volume	0.181 (-0.413 – 1.280)	0.305		
LVM index	0.481(0.183 – 0.879)	0.004	0.478 (0.201 – 0.854)	0.002
Global longitudinal strain	0.231 (-1.164 – 5.677)	0.188		
LV twist	0.015 (-2.368 – 2.569)	0.934		

LV = left ventricular; ADPKD = autosomal dominant polycystic kidney disease; CI = confidence interval; GFR = glomerular filtration rate; BP = blood pressure; LVM = left ventricular mass.

* Multivariate model includes only diastolic BP and LVM index as covariates (i.e. only those with $p < 0.1$ at univariate analysis).

Table 4
Adjusted linear regression analysis showing associations of octreotide-LAR treatment with final LV untwisting rate in the group of ADPKD patients.

	β (95% CI)	***p* Value**
Age	-0.044(-1.474 – 1.192)	0.829
Body Surface Area	0.144 (-28.715 – 62.766)	0.451
GFR	0.281(-0.142 –0.647	0.199
Heart rate	-0.122 (-1.308 – 0.744)	0.577
Systolic BP	0.015 (-1.038 – 1.107)	0.948
Diastolic BP	-0.127 (-1.966 – 1.107)	0.570
Octreotide-LAR treatment	-0.504 (-46.905 – -6.367)	0.012

LAR = long-acting-release; LV = left ventricular; ADPKD = autosomal dominant polycystic kidney disease; CI = confidence interval; GFR = glomerular filtration rate; BP = blood pressure.

2.3. Echocardiography analysis

According to the American Society of Echocardiography guidelines [3], LV mass (M) and ejection fraction were calculated by Devereux formula and biplane Simpson's algorithm, respectively. LVM was divided by body surface area in order to calculate LVM index. The measurement of left atrium volume was obtained using the apical 4-chamber and 2-chamber views and modified Simpson's rule. LV filling was assessed by mitral flow velocity curves and mitral annulus tissue Doppler imaging. Mitral early (E) and late (A) diastolic peak flow velocities, E deceleration time, isovolumic relaxation time and peak mitral annulus velocity during early diastolic filling (Ea) were measured. Details regarding speckle-tracking echocardiography analysis are described in Ref. [1].

2.4. Reproducibility of speckle-tracking echocardiography measurements

To assess reproducibility of speckle-tracking measurements the echocardiographic studies were repeated by the same observer in 15 randomly selected patients (5 from each group of the cross-sectional study) within one month after completing the initial analysis and before ADPKD randomization in the ALADIN trial. The same studies were also analyzed by an independent observer to determine inter-observer variability. Intra-observer and inter-observer variability values were calculated as the absolute difference between the corresponding two measurements in terms of percentage of their mean. Intra-observer

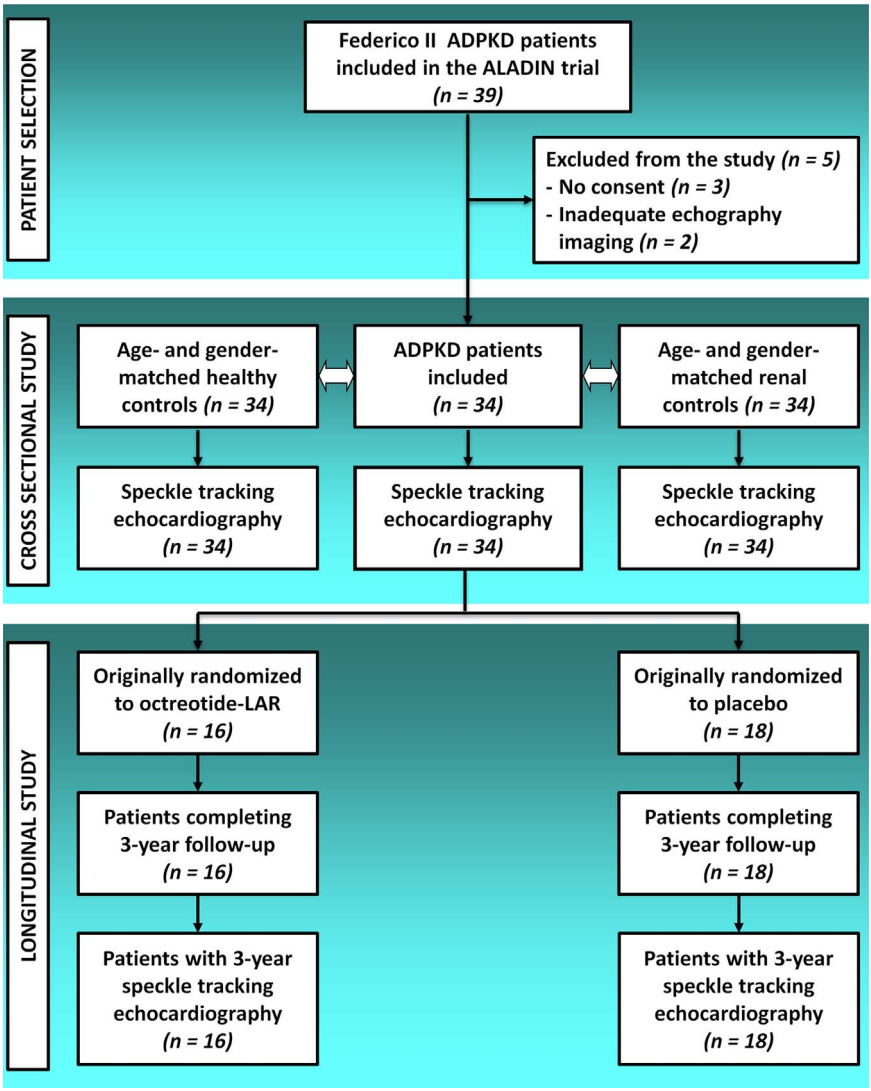

Fig. 1. Data set process flow chart.

variability was $r = 0.95$ for global longitudinal strain (SE of estimation: 4.1%), and 0.91 for twist (SE of estimation 4.9%), $r = 0.90$ for LV untwisting rate (SE of estimation: 5.1%). Inter-observer variability was $r = 0.93$ for global longitudinal strain (SE of estimation: 4.9%), $r = 0.90$ for twist (SE of estimation: 6%), $r = 0.89$ for untwisting rate (SE of estimation: 6.2%).

Transparency document. Supporting information

Transparency data associated with this article can be found in the online version at https://doi.org/10.1016/j.dib.2018.11.041.

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