DO METABOLIC DERANGEMENTS IN END-STAGE POLYCYSTIC KIDNEY DISEASE DIFFER VERSUS OTHER PRIMARY KIDNEY DISEASES?

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OBJECTIVES

Autosomal dominant polycystic kidney disease (ADPKD) is a systemic disorder, arising from a mutation in genes encoding polycystins, and therefore ADPKD could be associated with metabolic derangements. Increased incidence of new-onset diabetes mellitus after transplantation in recipients with ADPKD have been reported. We here investigated markers of insulin resistance (IR), inflammation and nutritional status, and insulin-like growth factor-1 (IGF-1) in end-stage renal disease (ESRD) patients with ADPKD versus other primary kidney diseases.

METHODS

In a *post hoc* cross-sectional analysis in 254 non-diabetic CKD 5 patients (median age 54 (range 43-62) years; 58% males) starting on dialysis, glucose metabolism (fasting blood glucose, insulin, IGF-1, homeostasis model assessment of IR, HOMA-IR), inflammation (high sensitivity C-reactive protein, hs-CRP, interleukin-6, IL-6, and tumor necrosis factor), nutritional status (by SGA and leptin), and bone mineral density (BMD, by DEXA), were assessed. ADPKD was present n=42 (16 %) of patients. Survival was recorded for median time of 28 months (IQR 15–48 months).

RESULTS

Patients´ demographic and clinical characteristics differed in ADPKD group as compared to the -other etiologies of CKD as regards age, history of CVD, body mass index (BMI), hemoglobin (Hb) and white blood cells (WBC) count. Beside those mentioned, other clinical and biochemical characteristics, including plasma creatinine, residual renal function (measured, mGFR, or estimated glomerular filtration rate, eGFR) and medications, were not different in the ADPKD group as compared to the -other etiologies of CKD.

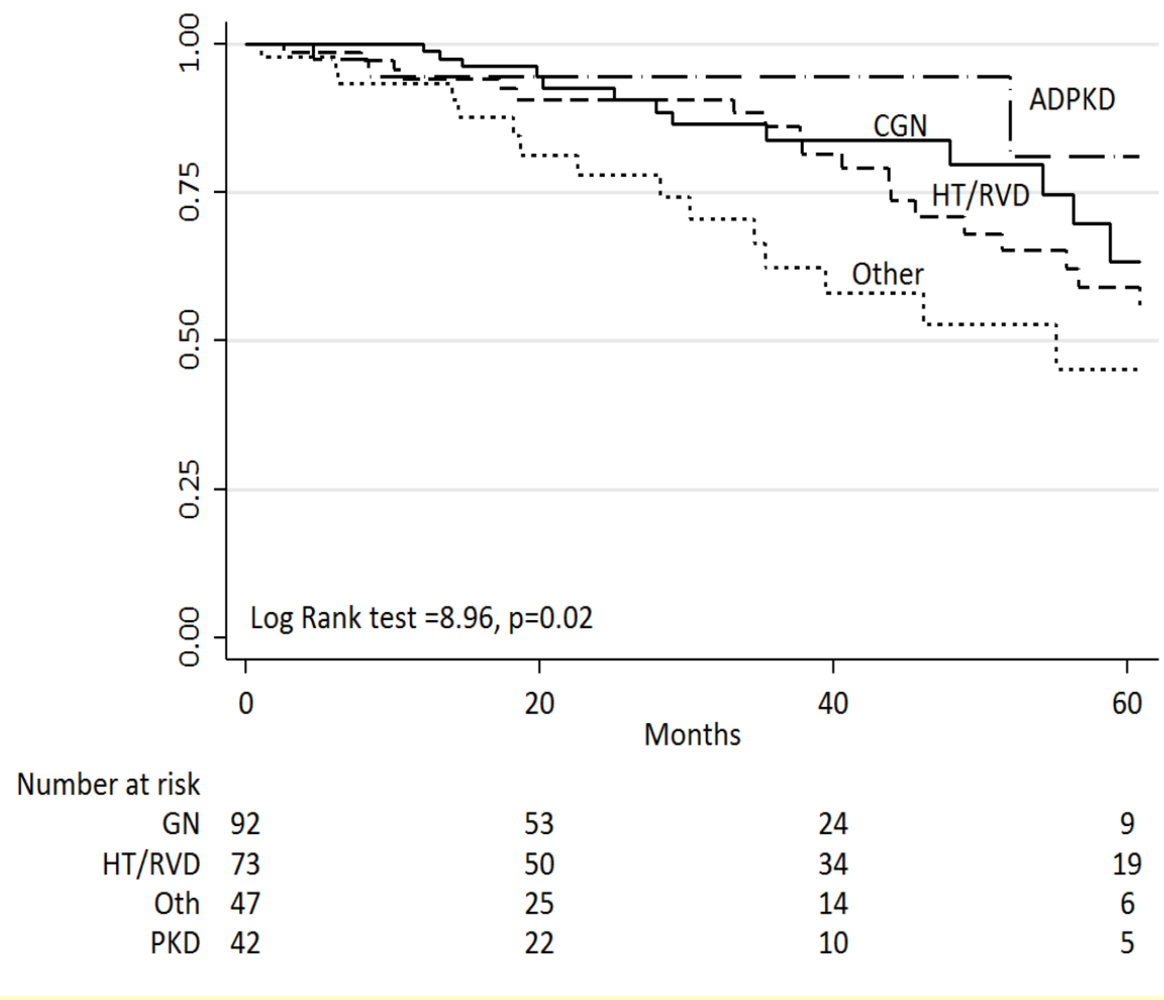
All parameters of glucose metabolism, except IGFBP-3, were comparable between the groups and IGFBP-3 was not different in the ADPKD group as compared with others. In **Table 1**, parameters of nutritional and inflammatory status are displayed. Again, despite significant differences between groups, in *post hoc* analysis those parameters were not different in the ADPKD group versus other primary causes of CKD. Survival from the day of examination up to the median time of 28 months (IQR 15 – 48) of follow up is presented in Kaplan-Meier curve in **Figure 1** showing that survival was better in ADPKD as compared to other primary causes of CKD

Table 1 Parameters of nutritional status, bone mass and inflammation in 254 non-diabetic CKD 5 patients starting on dialysis and grouped according to the etiology of CKD (* Available measurements (n=153) in ADPKD (n = 23), CGN (n=63), HT/RVD (n=39), and Other (n=28).

[a]Denotes the groups between which the difference occurred in post hoc analysis).

	ADPKD (n = 42)	CGN (n = 92)	HT/RVD (n = 73)	Other (n = 47)	P-value
Subjective global assessment (SGA) score	8 (6 – 10)	8[a] (6 - 11)	8 (6 – 12)	9[a] (7 – 14)	0.0278
SGA (% malnourished)	29	18[a]	26	49[a]	0.0034
Leptin (ng/ml)	16.5 (2.2 – 93.8)	9.9 (3.3 – 69.6)	13.9 (2.7 – 90.7)	8.0 (2.2 – 90.9)	0.478
Bone mineral density* (g/cm^2)	1.15 (1.02 – 1.31)	1.19[ab] (1.02- 1.31)	1.13[a] (0.97 – 1.28)	1.11[b] (0.96 -1.23)	0.0005
High sensitivity C-reactive protein (mg/l)	4.0 (0.8 – 26.5)	2.9[a] (0.6 – 23.1)	6.7 (1.2 – 56.0)	8.0[a] (0.6 -57.8)	0.049
Interleukin-6 (pg/ml)	6.1 (2.2 – 17.9)	3.9-------[a] (1.5 – 11.8)	6.7[a] (2.3 – 16.6)	6.7 (1.2 – 14.8)	0.0098
TNFα (pg/ml)	11.0 (6.1 -27.9)	9.5[a] (6.6 – 15.3)	12.2[a] (7.7 – 24.3)	12.6 (7.4 – 26.2)	0.019

Figure 1. Kaplan- Meier curves of survival in 254 non-diabetic patients with CKD stage 5 initiating dialysis therapy with ADPKD and other primary kidney diseases. Patients with ADPKD had the best survival followed by patients with chronic glomerulonephritis (CGN); hypertension/renovascular disease (HT/RVD); and other, or unknown causes (Others).



CONCLUSIONS

ADPKD was not found to contribute additionally to metabolic derangements characteristic for the ESRD phenotype. Thus markers of insulin resistance, inflammation and malnutrition as well as IGF-1 levels were not worse in non-diabetic ESRD patients with ADPDK versus patients with other CKD etiologies. ADPKD appears as a nephropathy with superior survival rate in CKD stage 5 patients, even after exclusion of patients with DM.

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