

ADULT POLYCYSTIC KIDNEY DISEASE: WHO NEEDS HOSPITAL FOLLOW-UP?

Griveas I^{1,2}, Bishop K¹, World MJ^{1,3}

¹Department of Nephrology, Queen Elizabeth Hospital
Birmingham, Birmingham B15 2TH, UK

²Greek Army Medical Services, Athens, Greece

³Royal Centre for Defence Medicine, Selly Oak Hospital,
Birmingham B29 6JD, UK

BACKGROUND

- Autosomal dominant polycystic kidney disease (APKD) is one of the most common genetic disorders associated with a defect in a single gene and at the same time one of the most common causes of chronic renal failure.

BACKGROUND

- Previous studies have identified several factors to be associated with deterioration of renal function in patients with adult polycystic kidney disease (APKD). These include the nature of the mutation (PKD1 deteriorating faster than PKD2), male gender, the onset of hypertension below the age of 35y, haemoglobin concentration and hyperlipidaemia.

Aim

- The present retrospective study was undertaken further to examine the contribution of these factors to deterioration in renal function.
- Additionally, an attempt was made to determine whether hospital follow-up was necessary in the early decades of this disease in all patients or whether this could be more selective with beneficial cost savings.

METHODS

- The clinical data of 184 patients with APKD attending a dedicated clinic were reviewed. Of these, 120 of them satisfied criteria for inclusion in this study.

Exclusion criteria: age less than 18 years,
follow up for a period of less than 3
months,
less than four data points to calculate
change in estimated glomerular
filtration rate with time
($\Delta eGFR$, ml/min/1.73m²/y).

METHODS

- Gender and age were recorded.
- Patients were classified by race into white, black, indo-asian and oriental.
- Seated systemic arterial blood pressure (systolic, diastolic and mean), recorded at the time of first clinic attendance.
- Initial laboratory estimation of haemoglobin, cholesterol and creatinine concentrations.

METHODS

- In each case, estimated glomerular filtration rate (eGFR) was calculated using the 4-variable form of the Modification of Diet in Renal Disease (MDRD) formula. Using the same method, the eGFR at successive time points during follow-up of each patient was calculated.
- The patients were then classified into those who had a statistically significant ($p < 0.05$) average rate of deterioration in renal function ($-\Delta eGFR$) and those who did not.

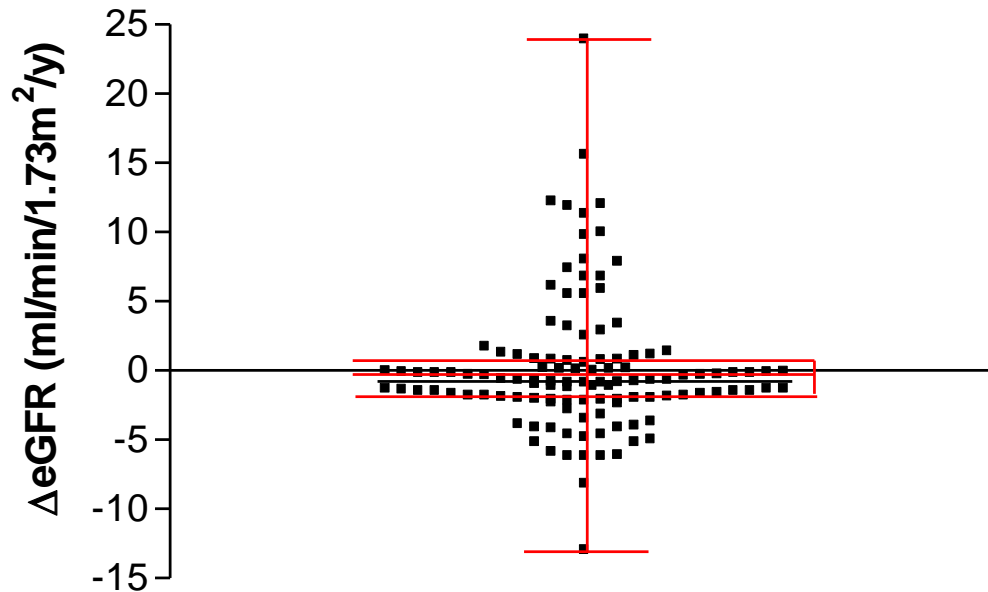
RESULTS

Characteristics of patients. Patients were subsequently split into two groups: those without and those with statistically significant annual deterioration in eGFR whose characteristics were then compared statistically.

Variable (units)	All patients	Those <i>without</i> a statistically significant annual deterioration in eGFR (=ΔeGFR)	Those <i>with</i> a statistically significant annual deterioration in eGFR (=ΔeGFR)	p value
Number (n)	120	94 (78%)	26 (22%)	-
Median ΔeGFR [range] (ml/min/1.73m ² /y)	-0.8 [-17.25 to +23.9]	-0.2 [-17.25 to +23.9]	-2.6 [-6.2 to -0.7]	p<0.0001
Male/Female	47/73	33/61	14/12	p=0.26
Mean age ±SD (y)	36.7±12.7	36.6±13.0	36.7±11.6	p=0.97
Race: White/Black/Asian (n)	103/7/10	80/6/8	23/1/2	p=0.32
Median duration of follow-up [range] (months)	58 [3 to 172]	46 [3 to 161]	86 [23 to 172]	p=0.002
Mean initial Hb±SD (g/dl)	13.6±1.5	13.7±1.4	13.4±1.7	p=0.42
Mean initial cholesterol±SD (mM/l)	4.86±1.1	4.88±1.1	4.81±1.0	p=0.84
Mean initial eGFR±SD (ml/min/1.73m ²)	74.2±18.2	73.2±17.4	78.0±21.0	p=0.25
Mean systolic BP±SD (mmHg)	146±21	148±21	128±11	p=0.02
Mean diastolic BP±SD (mmHg)	82±11	82±12	78±5	p=0.07
Mean MAP±SD (mmHg)	104±13	105±13	95±5	p=0.04

RESULTS

Distribution of $\Delta eGFR$ for all patients with APKD: median = $-0.8 \text{ ml/min/1.73m}^2/\text{y}$; (IQR= $-2.1 - 0.8$; range= $-17.2 - 23.9$).



RESULTS

- No statistically significant differences in initial haemoglobin or cholesterol concentrations or eGFR values were found between the groups.
- There was a trend for initial systolic, diastolic and mean arterial blood pressure values to be lower in the group of patients destined to develop deteriorating renal function but differences failed to achieve *a priori* levels of statistical significance.

RESULTS

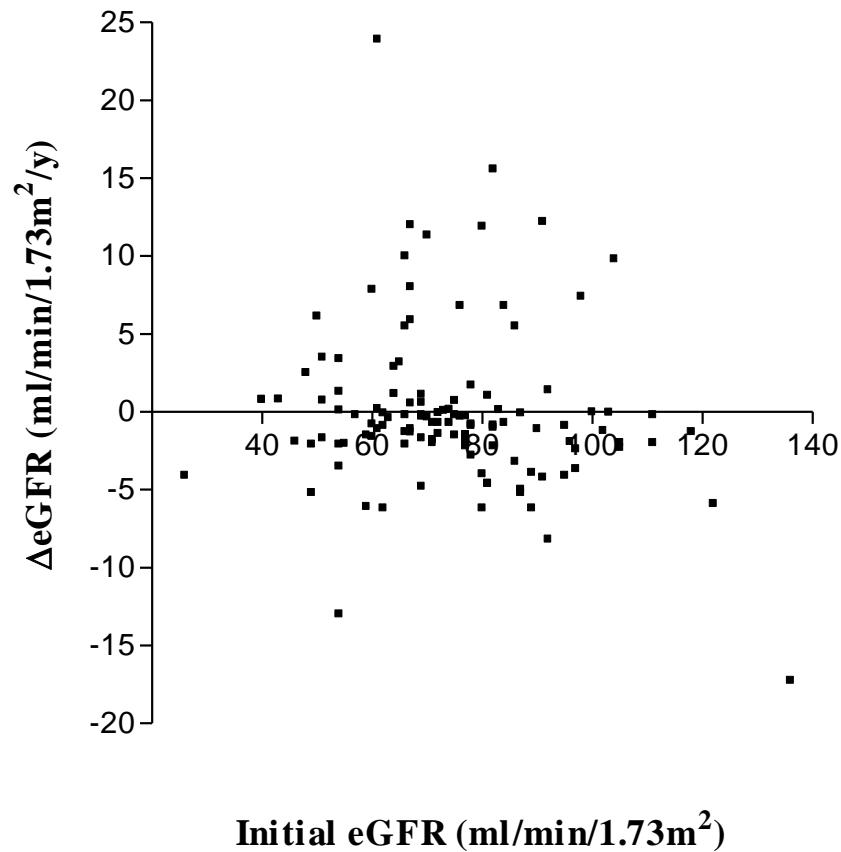
Rank correlations between Δ eGFR and other variables

Variable (Units)	Number of paired values	Spearman's <i>rho</i>	Probability
Duration of follow-up (months)	120	-0.07	p=0.48
Initial eGFR (ml/min/1.73m ²)	120	-0.19	p=0.04
Initial Hb (g/dl)	100	0.09	p=0.35
Initial cholesterol (mM/l)	53	0.05	p=0.74
Initial PTH (pg/ml)	7	0.22	-
Initial Systolic BP (mmHg)	55	0.11	p=0.44
Initial Diastolic BP (mmHg)	55	0.12	p=0.39
Initial MAP (mmHg)	55	0.18	p=0.18

RESULTS

Δ eGFR (ml/min/1.73m²/y) vs. initial eGFR (ml/min/1.73m²).

Spearman's ρ = -0.19, p=0.04



CONCLUSIONS

- There was no difference in initial age, gender or racial distribution or in initial eGFR between the groups destined to develop deteriorating renal function and those that would not develop this.
- Age at the time of presentation did not seem to predict subsequent deterioration.

CONCLUSIONS

- Only those patients with polycystic kidney disease with a statistically significant annualized decrease in eGFR may need to be referred for hospital follow up in the renal clinic.
- Some compromise could be necessary concerning control of blood pressure in some patients.
- Overenthusiastic antihypertensive treatment could accelerate deterioration in renal function while protecting against development of generalized vascular disease.

CONCLUSIONS

- In those patients with rather stable renal function over the follow-up period, no significant correlation was found between age of patients, duration of follow up and $\Delta eGFR$.
- That means that patients, who for unknown reasons do not develop significant decline in their renal function for a period of 5 years, probably do not usually need the care of a specialist.

References

- Peters DJ, Breuning M. Autosomal dominant polycystic kidney disease: Modification of disease progression. *Lancet* 2001;358: 1439–1444.
- Franz KA, Reubi FC. Rate of functional deterioration in polycystic kidney disease. *Kidney Int* 1983;23:526-529.
- Chapman AR, Schrier RW: Pathogenesis of hypertension in autosomal dominant polycystic kidney disease. *Semin Nephrol* 1991;11:653-660.
- Ecker T, Schrier RW: Hypertension in autosomal dominant polycystic kidney disease: Early occurrence and unique aspects. *J Am Soc Nephrol* 2001; 12: 194–200.