Hypertension in Black Africans with Autosomal Polycystic Kidney Disease

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Abstract

Introduction: High blood pressure (HBP) that is a leading cause of end-stage renal disease (ESRD) in black African populations and is frequently associated to autosomal polycystic kidney disease (ADPKD). This study aimed to describe prevalence and severity of HBP in black Africans with ADPKD and to identify associated risk factors.

Patients and methods: We performed a retrospective study of 65 ADPKD patients regularly followed in outpatient nephrology clinic between 1995 and 2009. ADPKD was diagnosed according to recent unified criteria (2009). Statistical analyses were done with SPSS 16.0.

Results: We included 65 patients (36 males and 29 females) with a mean age of 47 ±5 years. Hypertension was found in 73.8 % of patients and it preceded diagnosis of ADPKD in 23 patients (median delay of 28 months). Mean systolic/diastolic blood pressure was 168 ±30 /96 ±16 mm Hg respectively. All hypertensive patients presented retinopathy and left ventricular hypertrophy. Two patients presented stroke. Fifty one percent of patients were treated with angiotensin converting enzyme inhibitors alone and 29% received combinations of anti-hypertensive drugs. One third of them had their blood pressure normalized. Patients with HBP at diagnosis showed a similar proportion of ESRD in comparison with normotensive patients (p=0.12). At univariate analysis, HBP was correlated with age, gender, BMI, GFR and proteinuria. Multiple regression analysis identified age (OR=1.95, p=0.05) and glomerular filtration rate (OR=2.33, p=0.001) as independently associated to HBP.

Conclusion: Hypertension with organ damage is frequent in Senegalese patients with ADPKD. Age and glomerular filtration rate at diagnosis were the main risk factors of HBP identified in our patients.

Keywords: Hypertension; Polycystic kidney disease; Black Africans

Introduction

Autosomal polycystic kidney disease (ADPKD) is the most common hereditary renal disease but it is rarely described in black African populations [1,2]. High blood pressure (HBP) is a leading cause of end-stage renal disease (ESRD) in this population and is frequently associated to ADPKD [3,4]. Patients with ADPKD usually develop HBP prior to impairment of glomerular filtration rate (GFR) [5,6] and its complex pathogenesis involves cyst growth, renal volume and many other hemodynamic and neuro-endocrine factors [4,7]. Moreover, once present, HBP can worsen the cardiovascular and renal prognosis [8].

This study aimed to describe prevalence and severity of HBP in black Africans with ADPKD and to identify associated risk factors.

Patients and Method

We performed a retrospective and descriptive study of 65 ADPKD patients regularly followed-up in outpatient nephrology clinic between 1995 and 2009. Socio-demographic, clinical and paraclinical data were collected from medical records that included first outpatient visit (or admission) and routine visits. Patients were followed-up on a yearly basis, or more frequently if required. All diagnosis of ADPKD were reviewed according to ADPKD unified criteria [9]. HBP was defined and stratified according to seventh Join National Committee report [10]. Patients with incomplete records and those who did not met ADPKD criteria were not included. Statistical descriptive and correlation analyses were done with SPSS11.0.

Results

We included 65 patients (36 males and 29 females) with a mean age of 47 ±5 years. Hypertension was found in 73.8 % of patients and it preceded the diagnosis of ADPKD in 28.8% of them (median delay of 28 months between HBP and ADPKD diagnosis). Levels of blood pressure in our patients are presented in Figure 1. Mean serum creatinine and
GFR were respectively 3.45±1.4 mg/dl and 45±13 ml/min. Differences between hypertensive and normotensive patients in clinical, biological and ultrasound findings at diagnosis are presented in Table 1.

Proliferative retinopathy was present among all 35 hypertensive patients who underwent fundoscopic examination. Two patients presented cerebral ischemia secondary to HBP. At univariate analysis, age (t=1.45; p=0.02), gender (t=0.98; p=0.17), BMI (t=0.85; p=0.02), GFR (t=2.56; p=0.001) and proteinuria (t=1.86, p=0.02) were significantly correlated to HBP. Multiple regression analysis identified age (OR=1.95; p=0.05) and glomerular filtration rate (GFR) (OR=2.33, p=0.001) as independently associated to hypertension.

Anti-hypertensive therapy for the majority of patients comprised ACE inhibitors alone or in combination with other drugs (Figure 2). However only 50% of them had their blood pressure in <140/90 mm Hg. After a median follow-up of 9.68 months, incidence of ESRD was not different between hypertensive and normotensive patients (respectively 60% versus 50%, p=0.12). But mortality was higher in the hypertensive group (17 versus 5 cases, p=0.03).

**Discussion**

ADPKD is probably an underestimated health issue in Senegalese population with an estimated hospital prevalence of 0.2% [2]. The high prevalence of hypertension in our patients was similarly found in previous studies where HBP concerned 50 to 75% of patients with ADPKD [3,4,6,11]. However, in Morocco where ADPKD represents 6.5% of dialysis patients, lower prevalence of HBP (11%) was reported [12]. This variability between studies is probably due to differences in genotype and diagnosis delay. In fact, frequency of HBP was higher in our patients compared to other series [11] probably because of delayed diagnosis of ADPKD (more cyst volume and lower GFR) [13] even though in 60% of patients increase of blood pressure precedes GFR decline years before [14]. Also, patients with PKD1 mutations or with an hypertensive ascendent are at higher risk to develop HBP and earlier in their life [4]. Occurrence of hypertension in ADPKD results from an interplay between many factors such as cyst growth and increase in renal vascular resistance and filtration fraction, activation of peripheral renin-angiotensin system, salt sensitivity, extracellular fluid overload, resetting of the pressure-natriuresis relationship [13]. An early decrease in renal blood flow is noticed in young ADPKD patients before development of HBP [15].

LHV was more frequent in hypertensives patients compared to normotensives. LHV may occur in ADPKD patients without HBP but HBP can also increase risk of LHV achieving 70% of ADPKD patients at ESRD [16]. However, the retrospective nature of our study could not help to distinguish whether LHV preceded or was secondary to HBP. Only 30% of our patients had their blood pressure in the normal range despite anti-hypertensive treatments. Poor blood pressure control increases the risk for LHV, proteinuria, ESRD and cardiovascular morbi-mortality [4]. The superiority of an anti-hypertensive drug in ADPKD is not yet clearly established. Blockers of renin-angiotensin system can efficiently help controlling blood pressure in ADPKD patients, regression of LHV [13] and might also reduce cyst progression and GFR decline [17]. Due to this hypothetical benefit, ACE inhibitors were the most prescribed anti-hypertensive drugs in our patients.

Kidney size was not a predictor of HBP in our patients but ultrasonography is not a recommended method to assess kidney volume. Magnetic resonance imaging is the best technique but it was not available in this study. In fact, many studies have demonstrated a correlation between hypertension, left ventricular hypertrophy, cyst volume progression and deterioration of GFR [13]. Albuminuria and GFR might be markers of underlying vascular damages in ADPKD like in general population [18].

Early detection and treatment of hypertension in ADPKD is an important clinical issue because it can reduce cardiovascular complications which are the main causes of death in the post-dialysis era [13,19].

**Conclusion**

Hypertension with organ damage is frequent in black African patients with ADPKD. In the majority of cases diagnosis is delayed with impaired renal function. Main risk factors are age, male sex and low GFR. Blood pressure control is poor but adherence to therapy can be a confounding factor in our patients with low economic level.

**References**


