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CASE REPORT

End Stage Kidney Failure Secondary to Polycystic Kidney Disease Presenting With Hypokalemia

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Abstract

Polycystic Kidney Disease usually causes complications such as high blood pressure, formation of cysts in the liver, aneurysms and chronic kidney disease. Generally, chronic kidney disease (CKD) gives rise to high potassium levels but in patients of CKD with polycystic kidney disease, there is a significant lower potassium level and this is appreciated in CKD stages 1 to 3b. However, in end stage kidney disease potassium levels may be significantly high. Based on the case reported, a presentation of features of end stage kidney disease with significantly low potassium levels and kidney cysts on sonography may signify an underlying polycystic kidney disease.

Keywords: Polycystic Kidney Disease, Hypertension, Chronic Kidney Disease, Genetic Disorder, Diabetes, Hypokalemia.

1. Introduction

Polycystic Kidney Disease (PKD) is a genetic disorder characterized by the formation of cysts in the kidney, causing it to become larger than normal and fail in its ability to carry out its functions. Polycystic kidney disease cyst can profoundly enlarge the kidney while replacing much of the normal structure, resulting in reduced kidney function and leading to kidney failure(1).

The kidneys play an important role in excretion of waste products of metabolism, balance of electrolytes, homeostasis, osmoregulation, regulation of PH, production of hormones i.e. erythropoietin, renin, and activation of vitamin D3 (2)

Polycystic form the largest causes of end stage renal failure and are also amongst the common indications for dialysis and kidney transplant procedures. This disease state may come about sporadically as a developmental abnormality or maybe acquired in adult life. Majority are congenital. In some of the acquired forms, cysts develop in the kidneys as a results of aging, dialysis, drugs or hormones (3). Renal cysts also occur as secondary manifestations of proliferative disorders. Inherited l polycystic kidney diseases arising from single germ line mutations may be autosomal dominant (AD) or recessive polycystic renal diseases. Common presentations can also be nephronphthisis or medullary cyst diseases. Age at onset, symptomatology and progression to end stage vary very widely (4).

2. Case Report

2.1 History And Physical Examination

The patient MM, a 61 year old woman, of known HIV disease, diabetic for 13 years and diagnosed with

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hypertension 3 months prior date of history taking, presented with non-bilious, non-projectile vomiting for 4 months with a history of 8 episodes on the day of presentation, associated with burning chest pain. She had labour-like lower backache, which was non-radiating and intermittent with unknown relieving and worsening factors. She also had a history of dizziness, syncope, palpitations, dyspnoea, and oliguria and body weakness with no history of headache, cough, or body pain. She had history of smoking and drinking alcohol with no known allergies. The patient was on nifedipine for hypertension, cART (since 2010) and metformin, for diabetes. There was a positive family history from the younger sister and mother with diabetes.

2.1.1 Provisional Diagnosis

CKD in HIV stage 3 disease in a known diabetic and hypertensive patient r/o GI OIs

2.1.2 Differential Diagnoses

i. Malaria in HIV disease

ii. GERD in HIV disease

iii. UTI in HIV disease

On examination, her GCS was 15/15, she was afebrile, pale with no jaundice or cyanosis, and not in respiratory distress. The vesicular breath sounds were heard and normal. Cardiovascular system examination revealed clear S1 and S2, her abdomen was soft, non-tender, not distended and had no palpable masses nor organomegaly. There was oedema of grade 1.

The BP was 122/70mmHg, pulse was 97beats/min, respiratory rate was 24 beats per minute, and RBS was 13.2mmol/l

RDT for malaria tested negative

Urinalysis showed traces of blood (++), protein (++++) and glucose (+).

2.1.3 Provisional Diagnosis

Chronic kidney disease in a known diabetic and hypertensive patient r/o GI OIs in HIV stage 3

2.1.4 Differential diagnosis

Nephrotic syndrome

2.1.5 Investigations:

The blood workup revealed a haemoglobin level of 8.4g/dl, red cell count of $2.71*10^{12}$/l, HCT 23.8, MCV 87.8, and platelets of $323*10^{9}$/l. Her albumin was 34.6g/l, Urea was 16.49mmol/l and creatinine 1330.0. Sodium was 135mmol/l while potassium was 2.22mmol/l. Stool for modified ZN was done and was negative

Abdominal ultrasonography revealed hypertrophy in both kidneys with multiple cysts. The right kidney measured 15.03cm x 9.9 cm, the largest cyst on this kidney measures 4.3 cm x 3.77cm. The left kidney measures 18.20cm x 9.15cm, the largest cyst on this kidney measuring 6.74 cm x 9.3 cm

2.1.6 Diagnosis

Polycystic Kidney disease in a known diabetic-hypertensive with HIV stage 3.

3. Plan

The following pharmacological and non-pharmacological measures were indicated to manage the patient:

i. Enalapril to treat hypertension and ciprofloxacin to treat any cyst infections.

ii. Tolvaptan towards the management of hyponatraemia and to slow kidney function decline.

iii. Potassium supplementation as appropriate

iv. Serial urea/creatinine and electrolyte profile monitoring

v. Preparations for possible haemodialysis.

vi. Counsel patient and relatives on the condition and course of care.

vii. Consultation with the surgical unit in readiness for surgery as may be required

4. Discussion

Polycystic Kidney Disease is a genetic disorder characterized by the formation of cysts in the kidney, causing it to become larger than normal and fail in its ability to carry out its functions. The case being reported is that of an elderly woman who was diagnosed with polycystic kidney disease. Oliguria body weakness and hypertension were among the major presenting complaints. Haematuria, proteinuria and glycosuria only added to the confirmation of the diagnosis. Ultrasonography is the investigation of choice, as it is also ideal for screening patient's family members. Urinary proteomic biomarkers may carry the potential for diagnosis and prognosis of ADPKD (5).

In the approach to management, rigorous blood pressure control is essential as it prevents rapid deterioration of the already malfunctioning kidney (6). For young adults who may present with negative findings but are suspicious of PCKD, genetic testing

may be done. This has an accuracy rate greater than 95% for both ADPKD1 and ADPKD2 (7). As alluded earlier in the introduction, PCKD is one of the most common inherited disorders in humans. Being the most recurrent genetic cause of renal failure in the young and old adults

The typical presentation is by the 3 rd to 4th decade of life, but cysts may be detectable in childhood and in utero. It should be noted that ADPKD is associated with excessive angiogenesis which may be responsible for increased vascular permeability facilitating fluid secretion into the cysts (8).

Prognosis in patients with ADPKD: Patients with ADPKD may live a normal lifespan without knowing that they have the disease. Progressive renal dysfunction, grossly enlarged kidneys and eventually kidney failure by 40 to 60 years of age is a common presentation. However, studies have shown that there is an inverse proportion between size of polycystic kidneys and the level of glomerular filtration (1). Hypertension is known to be a common early clinical symptom in 50–75% of autosomal dominant polycystic kidney disease (ADPKD) patients (9). Further, the excessive production of aldosterone in primary aldosteronism (PA) is associated with a higher incidence of cardiovascular events than essential hypertension (10), while hypokalemia, a major characteristic of PA, can result in target organ damage (11) and aggravated hypertension.

5. Conclusion

This case report presents a rather unique manifestation of end stage kidney failure characterized by hypertension, deranged urea&creatinine and, a significantly low serum potassium level, features highly consistent with PCKD. In the given case, the autosomal dominant type being most probable.

Consent

Consent to assess, case write-up and publication was obtained from the patient.

Conflict of Interest

The authors declare that there was no conflict of interest regarding the publication of the manuscript

Author Contributions

All authors contributed towards the concept, design, reviews and ultimate write up of the manuscript

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