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CASE REPORT

A Case of Proceeding Polycystic Kidney Progress to the End Stage Renal Failure

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Abstract

Polycystic Kidney Disease (PKD) commonly an inherited monogenic disease, associated with severe morbidity and mortality. It is categorized by renal cyst formation and renal enlargement. Autosomal dominant polycystic kidney (ADPKD) causes end stage kidney disease and is most commonly seen in adults. Worldwide overall incidence is 1-2% and mutations in both the genes PKD1 and PKD2 causes ADPKD. Most of the ADPKD patients shows decline in renal function where as 70% leads to end- stage renal disease. An alarming non-communicable disease conditions currently with no cure causing a major public health concern as well as the economic burden on healthcare system. Here with we report a case of preceding polycystic kidney progress to the end stage renal failure, along with our understanding towards the management involved in the disease.

Keywords: *Polycystic Kidney Disease, Autosomal Dominant Polycystic Kidney Disease, End Stage Renal Failure, Hypertension.*

Introduction

PKD is characterized by multiple fluid filled renal cyst formation, leading to a progressive kidney enlargement, and gradually decreases renal function that advances to End Stage Renal Failure (ESRD). This condition is composed of various genetic, non-genetic and acquired manifestations. ADPKD is a common hereditary renal disease and the major genetic cause for kidney failure. At the mean age of 40 to 70 years, almost 70% of ADPKD patients develop ESRD [1]. The most common clinical manifestation is hypertension, as two third of ADPKD patients are hypertensive.

Case Report

A 50 years old male patient visited the casualty department of secondary care hospital with a chief complaint of oliguria and breathlessness from past ten days. After the physical examination, he was diagnosed with Chronic Kidney Disease (CKD) and got admitted to Intensive care unit for the further treatment. His vitals were found to be elevated such as blood pressure 180/100mmHg, pulse rate 120 beats per minute whereas cardiovascular system and Respiratory system were normal. Patient was a known case of CKD with a history of renal calculi for ten years. The Ultrasound was done one year prior at previous hospital and it revealed the impression of right polycystic kidney (5-6 cyst), with the largest measuring (6.6 x 4.5 cm) along with dilated ureter. He was having hypertension from past five years and was on medication Tab. Amlodipine 5mg BD. Laboratory investigation was done and it is presented in Table 1.

His GFR was calculated as 6 ml/min and he was prescribed with Antihypertensive and Diuretics. On second day his blood Pressure noted with 140/90 mmHg, pulse rate-98 beats per minute, respiratory rate- 22 per minute, and the urine input output chart was 100ml/50ml. He was continued with the same medication and planned to perform Dialysis on the same day. Hemodialysis was started at 2.30 pm and the recorded physical examinations at the beginning were: pre-weight-60.5 kg, weight gain- 1.5 kg and blood pressure- 160/90mm Hg. During Hemodialysis he received Inj. Heparin 8000

I.U. Hemodialysis was continued for 4 hours with ended post weight-58 kg, weight loss-2.5 kg, blood pressure-210/100mm/Hg. After Hemodialysis he received Inj. B_{12} 200 mcg (100mcg/ml), Inj. Erythropoietin 2000 I.U S/C and Inj. Iron Sucrose 100 mg (20mg/ml). Patient got discharged and advised to review after 10 days. (Patient consent was taken for publication)

Discussion

The persuasive nature of ADPKD towards the end stage renal failure and in addition ADPKD being fourth global leading cause for the renal replacement therapy represents a critical importance at nephrology. Gene mutation of PKD1 or PKD2 are the general cause, in which PKD1 mutation contributes almost 80% of ADPKD condition followed by ~ 15% ADPKD of PKD2 mutation with genetically unsolved ~5–10% of ADPKD cases respectively [2].

The Incidence of ADPKD is estimated over 600,000 individuals in the United States with a prevalence of 1/400 to 1/1000 individuals (141, 254) [3], along with 12.5 million people across globe [4]. At clinical practice the diagnosis can be done by using ultrasound examination as initial assessment technique and magnetic resonance imaging scan for the depth study.

According to Kidney Disease Improving Global Outcomes (KDIGO) determining the Total Kidney Volume (TKV) are recommended as TKV accurately estimates evading kidney failure along with the associated hypertension and loss of kidney function in ADPKD [5]. Kidney transplantation is most recommended for the renal impairment ADPKD patient who reached end stage renal failure disease.

Haemodialysis or peritoneal dialysis procedure is appropriate for those patients who are not suitable for transplantation. The vasopressin V2 receptor antagonist tolvaptan is known to slow down the kidney function decline in adults at risk of rapidly progressing ADPKD. In ADPKD condition it is well recognised that the patient is hypertensive and untreated hypertension may lead to increased mortality rate.

Angiotensin converting enzyme inhibitors (ACEi) are recommended as first line antihypertensive agents for the management of hypertension with ADPKD conditions and if intolerant to ACEi, Angiotensin receptor blockers (ARB) are considered as second line antihypertensive agents with a targeted blood pressure of less than or equal to 130/80 mmHg [6].

In our case the patient was on Antihypertensive agent i.e. calcium channel blocker (nifidepine and amlodipine) during and before admission despite ACEi and ARBs. Lipid lowering agent (LLA) such as 3-hydroxy-3-methyl-glutaryl-CoA reductase inhibitors therapies are recommended for the mortality prevention through reduction of cardiovascular risk among those with CKD. Non-pharmacological management includes less- salt diet, sufficient fluid intake, restriction of smoking and nephrotoxic agents such as nonsteroidal anti-inflammatory drugs and limitation of caffeine intake.

Despite being a major genetic disorder, approaches towards ADPKD treatment for the renal and extra renal manifestation has been limited towards the management of symptoms and complications. Informed consent was obtained from patient for publishing his data.

Table 1: Laboratory investigation values

Laboratory parameter	Observed Value	Normal Value
Haemoglobin (Hb)	6.1 g/dL	14-18 g/dL
Total Count (TC)	6.7 x 10^3 cells/mm^3	3.2-9.8 x 10^3 cells/mm^3
Polymorph	67%	54-64%
Lymphocytes	25%	25-33%
Monocytes	8%	3-7%
Platelet Count (Pt)	110 x 10^3/mm^3	130-400 10^3/mm^3
Red Blood Cells (RBC)	2.24 x 10^6/mm^3	4.3-5 x 10^6/mm^3
Haematocrit (Hct)	19.7%	39-49%
Mean Cell Volume (MCV)	87.9 fL	76-100 fL
Mean Cell Haemoglobin (MCH)	27.2 pg/cell	27-33 pg/cell
Mean Cell Haemoglobin Concentration (MCHC)	31 g/dL	33-37 g/dL
Blood Sugar (Random)	99mg/dL	200mg/dL
Sodium	137.5 mEq/L	135-147 mEq/L
Potassium	5.03 mEq/L	3.5-5 mEq/L

Chloride	111.8 mEq/L	95-105 mEq/L
Serum Creatinine (Sr.Cr.)	9.3 mg/dL	0.6-1.2mg/dL
SGOT	3 U/L	0-35 U/L
SGPT	7 U/L	0-35 U/L
Alkaline Phosphate (ALP)	170 U/L	30-120 U/L
Bilirubin -Total	0.6 mg/dL	0.1-1 mg/dL
Direct	0.3 mg/dL	0-0.2 mg/dL
Indirect	0.3 mg/dL	0.1-0.8 mg/dL

Conclusion

With this case we came to conclusion that there is a limited approach available for the treatment of ADPKD and life-style modification counselling can be given to patient for further benefits.

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