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Polycystic kidney disease - A case report

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ABSTRACT

This report presents a case report of a 29 year old Saudi Arabian patient who has been recently diagnosed with inherited polycystic kidney disease. The patient suffered from hypertensive symptoms and severe abdominal pain. Haematology, chemistry and microbiology examination was performed on the patient, alongside an ultrasound examination. The patient appeared to have hypertension, high uric acid and high lipid profile. The ultrasound results showed enlarged fatty liver, two cysts in the left liver (1.28cm and 1.32cm in width) and one cyst in the right liver (0.85cm in width). Currently, the patient's renal function is normal, but suffers from severe abdominal and kidney pain regularly, alongside hypertensive symptoms. The current treatments provided are Olmetec for controlling hypertension and intravenous analgesic such as xefo, morphine and paracetamol. There is currently no treatment for polycystic kidney disease. Therefore, patient is continuously monitored on a monthly basis.

Keywords: Polycystic Kidney disease, Kidney disease, Inherited Kidney disease, Case report.

1. INTRODUCTION

Polycystic kidney disease (PKD) a genetic disease, where the kidneys get packed with cysts or fluid filled sacs causing the kidneys to expand abnormally, and causing renal failure over time, alongside hypertension and urinary stasis (Sullivan et al., 1998). The collection system can be also compressed by the expanding cysts, causing urinary stasis, which in some cases leads to kidney stones. As enough nephrons are affected as time pass, renal insufficiency would be caused leading eventually to renal failure (Masoumi et al., 2008). ARPKD can be detected via prenatal ultrasound which shows large kidney with cysts and oligohydramnios (Zhou et al., 2013).

2. CASE REPORT

We present the case of a 29-year-old Saudi Arabian patient who was referred from the Urology Department and underwent additional laboratory tests and urinalysis after complaining of acutepain. According to the patient's family history, both his father and aunt had chronic dialysis; also his father was diagnosed with PKD. In addition, Kidney stone illness was diagnosed in many of his family members. Physical examination was abnormal

anthropometric measurements and the patient had hypertension reading of 148/94 mmHg. It was treated with a combination of medications, Adalat (30 mg/day) and Atacand (8 mg/day) for one year, and then the dosage increased for Adalat (60 mg/day) for one month but not fit with his hypertension. The patient also used Sevikar (40 mg/) for two days only, which caused body pain and excess sweat. Currently, the patient is only taking Olmetec (40 mg/day) to control hypertension. Uric Acid was also high reaching upto 750 umol/L; thus Agout (40mg /day) was given as a treatment. The patient does not have heart valve disease.

Lab. Section Hematology

The test for complete blood cells shows as normal except high significant for red blood cells 6.29 10^{12}/L (Table 1). Serum biochemistry including abnormal renal (Creatinine 131.1 umol/L) and liver functions (BUN 9.3mmol/L, ALT 59 U/L) Also, uric acid 750umol/L was high and lipid profile include Triglycerides 2.41 mmol/L and LDL Cholesterol 5.12 mmol/L, electrolytes and calcium were normal (Table 2). Urinalysis showed negative red blood cells, 5 g/l protein, trace Epithelial Cells, traceMucus Threads with negative isomorphic erythrocytes in the microscopic evaluation of the sediment (Table 3).

Table 1 Hematology laboratory data

Lab. Section Hematology	Results	Unit	Normal Ranges
WBC	9.42	10^9/L	4-11
Neutrophils	5.41	10^9/L	2-7
Lymphocyte	2.30	10^9/L	1-4
Monocyte	0.833	10^9/L	0.2-1
Eosinophils	0.781	10^9/L	0.02-0.5
Basophil	0,095	10^9/L	0.02-0.1
Red Blood Cells	6.29	10^{12}/L	4.5-5.5
Hemoglobin	16.7	g/dL	13-17
Hematocrit test (HCT)	49.4	%	40-50
Mean corpuscular volume (MCV)	78.6	fl	80-95
(MCH)`	26.5	Pg	27-32
MCHc	33.8	g/dL	31.5-34.5
RDW	10.5	%	11.5-14
Platelet	248	10^9/L	150-450
mean platelet volume (MPV)	7.90	fl	11.5

Table 2 Chemistry laboratory data

Lab. Section Chemistry	Results	Unit	Normal Ranges
TROP I High Sensitive	<0.010	ng/mL	Apparently Healthy Population: less than 0.03 : Diagnostic Cutoff for AMI = more than 0.05 ng/ml
BUN	9.3	mmol/L	1.8-6-4
Creatinine	131.1	umol/L	53-115
CPK (CK)	84	U/L	-
Sodium	129	mmol/L	135-145
Potassium	4.3	mmol/L	3.5-5.1
Uric Acid	750	umol/L	Male = 210 - 420 Female = 150 - 350
Urine Microalbumin Quant.	24.3	mg/L	Reference Range for UAE: Normal Condition = less than 30 mg/L
Triglycerides	2.41	mmol/L	0.50-1.70
Calcium	2.53	mmol/L	2.1-2.55
LDL Cholesterol	5.12	mmol/L	0.5-3.3
Cholesterol	7.14	mmol/L	1.14-5.2
25 Hydroxy Vitamin D.	13.6	ng/mL	Deficiency < 10.0: Insufficiency 10.0- 30.0 : Sufficiency 30.0-100 : Toxicity >100.0
Albumin	46	g/L	35 – 52
ALT	59	U/L	0 - 55

Table 3 Microbiology laboratory examination (Urine analysis)

Lab. Section Microbiology Urine analysis	Result	Unit	Normal Ranges
Color	Yellow	-	Yellow
APPEARANCE	Clear	-	Clear
Glucose Urine	Normal	-	Normal
Bilirubin	Negative	-	Negative
Ketone	Negative	-	Negative
Specific Gravity	1.025	-	1.005-1.030
Erythrocytes/Hemoglobin	Negative	-	Negative
Ph	5.0	-	5.0-8.0
Protein	5 G/L	-	Negative
Urobilinogen	Normal	-	Normal
Nitrite	Negative	-	Negative
Leucocytes	10/UL	-	Negative
Red Blood Cells (RBC)	0	/HPF	< 3 /HPF
White Blood Cells (WBC)	2-3	/HPF	< 5 /HPF
Epithelial Cells	Trace	-	Nil
Bacteria	Few	-	Nil
Mucus Threads	Trace	-	Nil
AMORPHOUS	Nil	-	Nil
Crystals	Nil	-	Nil
Casts	Nil	/LPF	Nil

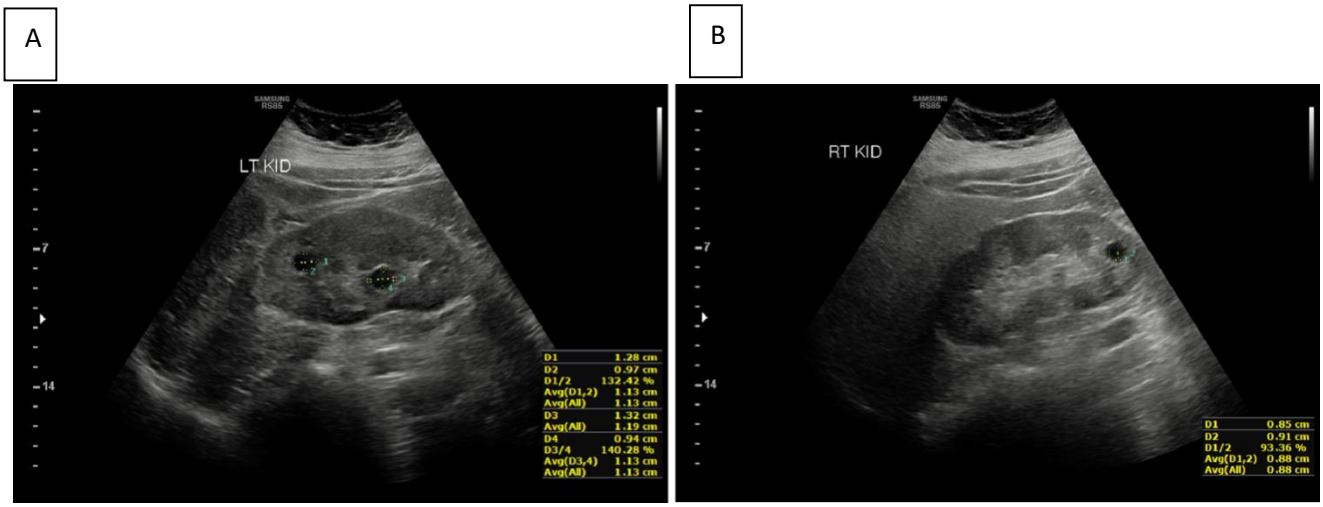

Figure 1 Ultrasound of both the left and right kidney with polycystic measures (cm). A) The left kidney has two dark circles (cysts) the top left cyst is 1.28cm in width (D1) and 0.97cm in height (D2). The central cyst has a width of 1.32cm and the height is 0.94cm. B) The right kidney has one cyst with a width of 0.85cm and the height is 0.91cm.

Ultrasonography for whole abdomen clinical diagnosis finding

Liver: mild enlarged in size, displaying bright echo pattern. No intra or extra hepatic biliary radicles dilatation.

Gall bladder: showed normal wall thickness with no definite sizable stone. Normal caliber of the portal vein and C.B.D.

Spleen: normal size and homogenous echogenicity.

The prostate is the average size, no sizable focal lesion and no sizable fluid collection. Left kidney is normal size and parenchymal thickness, increase echogenicity grad I with two cysts: the width is 1.28cm and 1.32cm (Figure 1A). Right kidney: normal size and parenchymal thickness, increase echogenicity grad I with one cyst with a width of 0.85cm (Figure 1B).

3. DISCUSSION

PKD is a hereditary disease that causes 5-10% of adult end-stage renal disease. PKD1 (chromosome 16p13.3) and PKD2 (chromosome 4q13q23) mutations have been shown to be responsible in 85% and 15% of patients, respectively. The most common form of polycystic kidney disease is theautosomal dominant polycystic kidney disease (ADPKD) (Bleyer and Hart, 2004; Ng et al., 2000). The onset of the disease is usually between the ages of 30 and 50. The detection of liver cysts and other external symptoms may contribute to the diagnosis in the absence of a family history or a negative family history (Hergan and Fellner, 2018). The development of cystic kidney can lead to chronic kidney failure, which is associated with long term kidney dialysis.

The following factors are critical: age, positive family history, enlarged kidneys with multiple cysts on both sides, and mild impairment of renal function at diagnosis. The most common kidney problems include heat discomfort, cyst infections, and arterial hypertension. Besides cyst rupture and inflammation, increased intra-abdominal pressure is usually what causes flank pain (Bajwa et al., 2004). In our case study the ultrasonography for abdomen clinical diagnosis finding; the bilateral renal simple cortical cysts, bigger in the left kidney with two cysts and one cyst at the right kidney. Over time, the cysts can enlarge due to fluid influx. Eventually, the cyst detaches from the nephron. Often PKD develop mild enlarged fatty liver (similar to our case), pancreatic cysts and heart valve changes (Gansevoort et al., 2016).

ADPKD can only be treated with a kidney transplant. Therefore, management of patients with PKD is the first action taken, such as blood pressure control, analgesics and antimicrobials for infections (Patel et al., 2009). Currently, only one drug has been approved to treat ADPKD, the V2 receptor antagonist vasopressin (tolvaptan) (Hergan and Fellner, 2018).

4. CONCLUSION

In conclusion, patients with mild PKD can suffer continuous abdominal pain due to cysts development, which leads to fatty liver and hypertension. Currently there is no specific treatment to prevent cysts development in PKD patients. Therefore, analgesics and hypertensive control drugs are provided. Our report presents the incident of an early developed PKD who suffer from extreme abdominal pain, enlarged fatty liver and hypertension. Therefore, PKD Patients require continuous hospital monitoring and good healthcare.

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Authors' contributions

JAA summarized the laboratory outcomes, was organizing the paper and giving guidance to co-authors on writing the case report. HJA wrote the discussion and edited the whole work for correct representation of the data. RJA wrote the introduction and summarized patient's current health status. AZA collected the data from the patient and placed them into tables and a figure. Also, gave detailed information of the current health of the patient. MAB done the final review and edit of the article

Informed consent

Written & Oral informed consent was obtained from all individual participants included in the study. Additional informed consent was obtained from all individual participants for whom identifying information is included in this manuscript.

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Conflicts of interest

The authors declare that there are no conflicts of interests.

Data and materials availability

All data associated with this study are present in the paper.

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