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CASE REPORT ON AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE

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Abstract

The most prevalent hereditary renal cystic disease, is a set of conditions defined by the formation of renal cysts and a variety of extra renal symptoms, is known as autosomal dominant Poly-cystic kidney disease(ADPKD). ADPKD is sometimes referred to as "adult PKD." It is typically diagnosed between the ages of 30 and 50 when signs and symptoms first arise. Two genes have been found to cause ADPKD, is PKD1 (chromosome 16p13.3) and PKD2 (4q21).Urinary blood in the urine, hypertension, anaemia brought on by CKD, and liver cysts may all be risk factors for ADPKD. Hypertension, impaired renal function, palpable kidneys, microscopic or gross hematuria, recurrent urinary tract infections, lower back pain, and shortness of breath are the most typical clinical manifestations of ADPKD. In our study we are aimed to summarize the case report is to mainly slows the cyst growth and Urine accumulates within cysts and delays ensuing loss of kidney function, which ultimately delays the need for renal replacement therapy and improves patients' quality of life. It has been observed that a variety of treatments can reduce the symptoms and progression of ADPKD.

Keywords: Autosomal polycystic kidney disease, Chronic kidney disease, Polycystic kidney disease.

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Introduction

S. NO	Study related to	Reports / Findings	Author
1	ADPKD	The purpose of this study was to evaluate the role of dietary intervention in reducing the osmotic load on urine volume and its effects on patients with ADPKD using tolvaptan's quality of life (QOL).	[1]
2	ADPKD	The purpose of this trial was to determine whether the PPAR-agonist pioglitazone may be used to treat the potentially fatal ADPKD in addition to being a major therapeutic agent for T2DM.	[2]
3.	ADPKD	They came to the conclusion that the use of tolvaptan in quickly advancing ADPKD can result in an increase in dialysis-free renal life of approximately 1.5 years up to 7.3 years, depending on the baseline eGFR and the time of treatment beginning.	[3]
4.	ADPKD	In the end, they came to the conclusion that early treatment of ADPKD patients who are largely asymptomatic is associated with a therapeutic burden but slows cyst growth and delays ensuing loss of kidney function, which ultimately delays the need for renal replacement therapy and improves patients' quality of life.	[4]
5	ADPKD	The goal of the review was to provide an overview of the variety of medications now used in clinical practise as well as the most promising compounds being investigated in human, animal, and lab-grown cell investigations.	[5]
6.	ADPKD	ADPKD is related to growth factors, signalling pathways, cell proliferation, apoptosis, inflammation, the immune system, structural abnormalities,	[6]

		epigenetic mechanisms, micro RNAs, and more, according to a review that tried to summarise the condition. It has been observed that a variety of treatments can reduce the symptoms and progression of ADPKD.	
7	ADPKD	The long-term management of ADPKD for patients and their families is anticipated to change as a result of the renewed focus on patient-centered research goals and the development of novel medicines.	[7]

Case Study

A38 yrs Old Male Patient was admitted in the hospital with the complaints of Fever, shortness of breath, A/W vomiting

Past History

HTN, and He is an Ex-alcoholic [stopped since 3yrs]

Family History

His mother has ADPKD.

His vitals on admission:

Temperature: 100 F

B.P- 120/70mmHg

P.R- 102/min

CVS- S1+S2+

R.S – B/LAE +

Advice

Haematology, Bio-chemistry, USG Abdomen,2D-ECHO

Laboratory Investigation

Haematology

Examination	Observed Value	Normal Range
HAEMOGLOBIN	7.2g/dl	14-18 g/dl
WBC	8800/cu.mm	4400-11000/cu.mm
RBC	5.42m/cu.mm	4.50-6.50/cu.mm
HEMATOCRIT	44.1%	42-54 %

Biochemistry Examinations

Examination	Observed Value	Normal Range
BUN	93.00mg/dl	7-20mg/dl
SERUM CREATININE	10.6mg/dl	0.80 - 1.30mg/dl
PLASMA GLUCOSE - RANDOM	101mg/dl	70-140mg/dl
SERUM SODIUM	135.0mmol/l	134-145 mmol
SERUM POTASSIUM	4.5mmol	3.6 - 5.2mmol
SERUM CHLORODE	97.7mmol/l	95-105 mmol

USG of the Abdomen

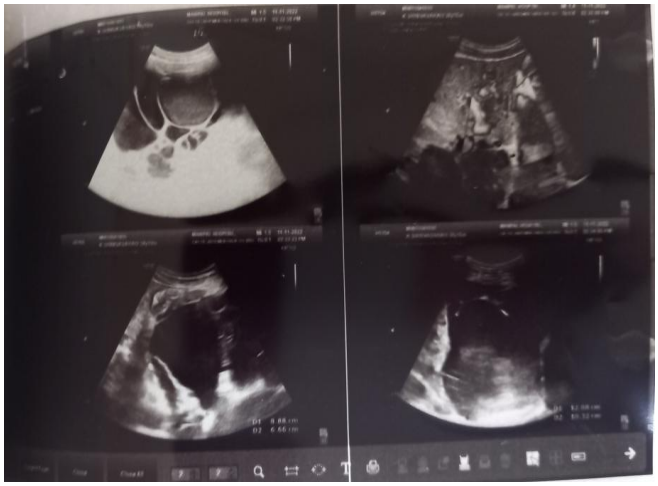
B/L Polyctstic kidney disease completely replacing the entire parenchyma.

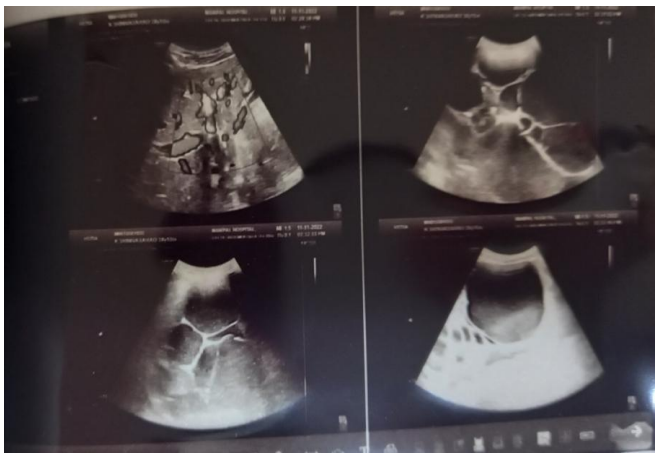
Simple hepatic cysts.

Minimal intrahepatic bilary dilation

Few portal varices noted.

Minimal ascites.





Pathophysiology

95% Inherited autosomal dominant mutations; 5% spontaneous mutations.

↓

Due to dysfunctional calcium channels on epithelial cells are the result of Mutations in the polycystic gene.

↓

Expensive cell proliferation, increased fluid secretion, abnormally expandable basement membranes.

↓

Urine accumulates within cysts, and cyst growth will be occur.

Diagnosis

Autosomal Polycystic Kidney Disease

Standard Treatment

There is presently no treatment for autosomal dominant polycystic kidney disease (ADPKD), and kidney cyst development cannot be prevented. But some potentially helpful drugs, such Tolvaptan, Pioglitazone can occasionally be used to slow cyst formation. Two primary methods of treatment Dialysis, or a kidney transplantation

Treatment

Patient is treated with following Medications

DRUG	DOSE	ROA	Frequency	NO.OF PRESCRIBED DAYS
TAB.CALCITROL	0.25MG	PO	OD	9 DAYS
TAB.CILNIDIPINE	20MG	PO	BD	9 DAYS
TAB.PRAZOSIN	5MG	PO	BD	9 DAYS
TAB.TORSEMIDE	20MG	PO	OD	9 DAYS
INJ. PANTOPRAZOLE	40MG	IV	OD	9 DAYS
INJ .ONDANSETRON	4MG	IV	BD	4 DAYS
TAB.CLONIDINE	100MG	PO	TID	9 DAYS
TAB.VELSARTAN	400MG	PO	TID	9 DAYS
TAB.CARVEDILOL	20MG	PO	OD	9 DAYS
TAB.TRANEXAMIC ACID	250MG	PO	TID	9 DAYS
TAB.LEVOFLOXACIN	750MG	PO	OD	5 DAYS
TAB.PARACETMOL	650MG	PO	OD	SOS
INJ ERYTHROPOIETIN	4000U	IV	MOTHLY THRICE	-

Life Style Modifications:

- Eating smaller portions of high quality protein
- Limiting salt, or sodium, in your diet
- Avoiding too much potassium and phosphorous in your diet
- Limiting alcohol consumpti

Actual Outcome

Based on patient's renal illness, the main prescribed drug is torsemide (a loop diuretic), and the anti-hypertensive drugs are still being used. He is undergone five times of hemodialysis .After hemodialysis , he developed hematuria and his haemoglobin levels dropped to 4.9 g/dl, and tranexamic acid was prescribed to stop the bleeding. Therefore, the patient's quality of life was improved by the dialysis extension in the patient.

Better Therapeutic Outcome

If they prescribed these drugs to the patient JYNARQUE (Tolvaptan), a Vasopressin antagonist, and Pioglitazone, a PPAR- Antagonist, along with surgery, the progression of ADPKD may be slowed down.

- And also lifestyle changes to help lower blood pressure and promote kidney health
- Maintaining fluid intake sufficient to reduce arginine vasopressin (AVP) secretion has been hypothesized to slow kidney cyst growth in ADPKD.

Conclusion

Aimed to summarize the case report is to mainly slows the cyst growth and Urine accumulates within cysts and delays ensuing loss of kidney function, which ultimately delays the need for renal replacement therapy and improves patients' quality of life. It has been observed that a variety of treatments can reduce the symptoms and progression of ADPKD.

Conflict of Interest

All authors are declared that no conflict of Interest.

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Inform Consent

Taken from the Patient

Ethical Consideration

Not Applicable

Author Contribution

All authors are contributed equally.

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