



Case Report

Reversing the Polycystic Kidney Disease Using Dietary Modification: A Case Report

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ABSTRACT

Polycystic Kidney Disease (PKD) is an inherited disorder in which the anomalous composition of the renal tubules results in the buildout of multiple cysts within the kidney. The genetic PKD mainly occurs between 30 to 40 years of age but acquired PKD can occur because of obesity, persistent high blood pressure, irregular eating patterns, and a sedentary lifestyle at any stage of life. The severe side or back pain, abdomen fullness, edema, hematuria, and weight gain are most common in both females and males. Several therapies are presented for PKD, including weight management, blood pressure control, medicaments like Tenormin, Dietary Approaches to Stop Hypertension (DASH) diet, and the use of cumin along with ajwain in place of salt. **Case Summary:** This case reported a 30-years-old female approached for evaluation of hypertension, severe side or back pain, and high blood pressure as well as overweight with a sedentary lifestyle. **Conclusion:** Cortisol hormone levels were elevated with an increased level of creatinine and urea in the blood. Tenormin and DASH diet, and cumin and ajwain were the best treatment choices for PKD patients that need additional investigation before being recommended on a long-term basis and fruitful treatment result.

INTRODUCTION

Polycystic Kidney Disease (PKD) is a genetic disorder of the kidneys with the development of cyst clusters, resulting in the kidney enlarging and dropping down functioning with time. The fluid retention in the noncancerous sacs causes the kidneys to enlarge. The different sizes and characteristics of cysts can damage the kidneys. PKD complications can result in cysts developing in the liver and other organs in the body. There are a lot of associated problems, including high blood pressure, hypertension, obesity, and even kidney failure [1,2]. Signs and symptoms can develop at any age depending upon whether the disease is genetic or acquired. Improved diet is the basic

element for the treatment of PKD and its advancement, controlling the damaging impact of PKD problems, including hypertension, hyperkalemia, and metabolic acidosis [3]. In addition, PKD can give rise to flank pain, cyst hemorrhage, nephrolithiasis, Intracranial Aneurysms (ICA), biliary tract disease, intestinal diverticulosis, and cardiac valve defects [4,5]. Several studies related to PKD treatment by Modification in Diet in Renal Disease (MDRD), Dietary Approaches to Stop Hypertension (DASH), healthy blood sugar levels, and healthy weight. There are further studies needed to examine the management of PKD. Here we report a case in which an adult woman with PKD, who

has been treated with Tenormin (01 year) and DASH diet (3 months) along with *Cuminum cyminum* (cumin) and *Trachyspermum ammi*(ajwain)as taste enhancer instead of salt.

Case Report

In January 2021, a 30-year-old female presented for evaluation of high blood pressure, hypertension, and obesity. She feels the fullness of the abdomen most of the time. She had not been treated with any diet therapy. She was referred from the other treatment center and suffered from ICA, severe headache, and high blood pressure. There is no family history of PKD and she has no practice of alcohol use or cigarette smoking.

Patient Assessment

She was suffering from hypertension and obesity with a height of 64.8”(inches)and weight of 95 kg, BMI (Body Mass Index) of 35 kg/m², an ideal body weight of 56.5 kg, blood pressure of 160/95, disturbed history of urination and Hematuria, and having the habit of fast and junk food in a company a sedentary lifestyle which causes to pile the disease. While the patient avoids most of the fruits, vegetables, and carbonated drinks in her daily life. The patient suffered from severe headaches, yellow teeth, pale color of skin, severe, or sometimes side pain with an enlarged abdomen mass due to edema or water retention in the cysts observed during clinical assessment. Cortisol hormone levels were elevated along with increased creatinine and blood urea level. The blood glucose level was on average and the hemoglobin level was low (Table 1). The albumin level was high, as well as the lipid profile was seen as abnormal during lab test examination. Whole abdomen USG showed; there is an anechoic large round cyst in the upper pole with thin walls 14.2x12.4cm, absent calcification, internal septations, or echoes, showing acoustic enhancement, no mass was seen in the left kidney and three anechoic round cysts showing acoustic enhancement, located at inferior pole 9.6x7.6cm at superior pole medially 4.2x4.9cm and at superior lateral pole 7.6x5.9cm, no calcification, internal septations or echoes, no mass was seen in the right kidney. The patient pelvic ultrasound was done (Figure 1). The patient was evaluated by a doctor, physiotherapist, and nutritionist, which guide her lifestyle modification and increase physical activities to manage the weight gain and bring down the weight. The patient already uses Tenormin (atenolol) 50mg/day to control her blood pressure and hypertension, patient recently started a DASH diet to improve her condition. The nutritionist also suggests cumin and ajwain instead of salt to control the severe headache and elevated blood pressure. Within 3 months, the patient was only able to manage the blood pressure by using Tenormin and a slightly improved lipid profile. After 6

months of treatment with Tenormin and DASH diet and cumin and ajwain, weight was 84kg and BMI decreased. The patient lipid profile improved and whereas blood pressure and hypertension were also controlled. The PKD remains but now the headache is not the problem. The side pain or back pain is not the problem as well as no edema or water retention in the body. The patient is still on the DASH diet and Tenormin therapy and cumin and ajwain because she is still overweight [6–10].

Test	Normal Range	Result
RFT (Renal Function Test)	0.6-1.1 mg/dL1	0.9 mg/dL
GFR (Glomerular Filter Rate)	40 mL/min/1.73m	<140 mL/min/ 1.73m ²
LFT (Liver Function Test)	27-56 units/liter for ALT 10-34 IU/L for AST	34 units/L25IU/L
GGT (gamma-glutamyl transferase)	0 to 51 IU/L	35IU/L
Total Protein	6 and 8.3 gm/dL	8.1 gm/dL
Albumin	3.4 to 5.4 g/dL	5.1 g/dL
Bilirubin	0.3 to 1.9 mg/dL	1.4 mg/dL

Table 1: Biochemistry of blood samples of the patient

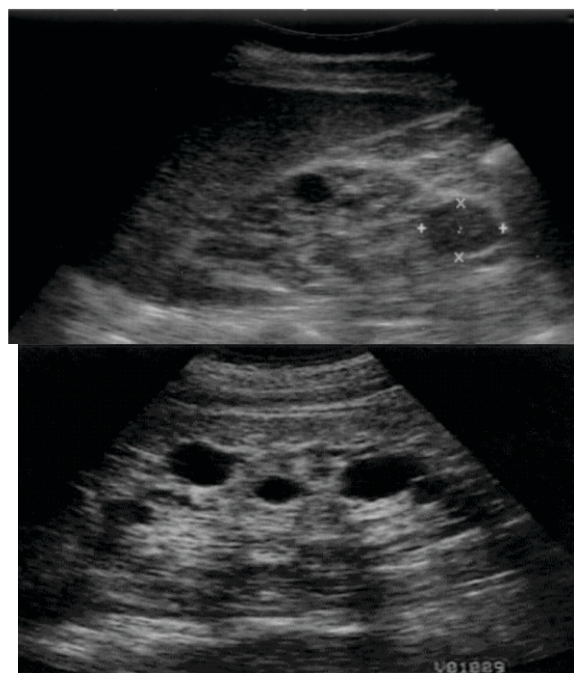


Figure 1: Pelvic Ultrasound report

DISCUSSION

Kidney disease belongs to a prevalent hereditary disorder: polycystic kidney disease. It results in 5% to 10% of end-stage renal disease in most individuals. It shows that PKD1 (chromosome 16p13.3) and mutations of PKD2 (chromosome 4q13q23) produce effects of 85% and 15%, respectively in patients. Performance of mutations analysis has not been done in our case. Instead of acquired cystic kidney disease (AKCD), In most cases, recessive inheritance and autosomal dominance are the basic

reasons behind polycystic kidney disease. Most patients with chronic kidney failure produce AKCD, which belongs to the extended period of dialysis [6]. Polycystic kidney disease acquired by inheritance contributes to several other disorders like tuberous sclerosis, nephronophthisis, ADPKD, and von-hippel-lindau syndrome along with these, it participates with other genetic diseases which are not common [7]. Diagnosis can be made clinically. The conclusive factors are as follow: family history having such favorable disorders, age, numerous cysts present on both sides of kidneys with increased in size, and at the time of diagnosis restricted functioning of kidneys occur. Mainly, this disease occurs in individuals aged 30-50 years. The disorders like refractory pain, arterial hypertension, and cyst infection are the most common complications associated with renal disease. Excessive intra-abdominal pressure usually causes flank pain, which develops infections and cyst rupture [8]. Those individuals having missed or adverse family history is diagnosed with the help of liver cysts detection and extra renal appearances. The exact elucidation has not been done in which a precise mechanism explains the origin of renal injury in polycystic kidney disease. In PKD, at least two mechanisms are present that intervene in renal injury and the growth of cysts, named cellular proliferation and fluid secretions [9]. Excessive development of fluid accretion can be involved by cyst expansion in PKD, which is supposed to require cystic fibrosis transmembrane conductance regulator protein, CFTR, that transports cAMP-mediated activation of chloride. The changes of cAMP do not mediate any effect on the appearance of FR, along with this, it shows that reduction of glucose accessibility or the suppression of the SIRT-1 pathway either does not mediate any effect on FR [10]. Mimic several effects of FR and Reduction of IGF-1 levels can be done by restricting a single amino acid called methionine restriction. Also, MR increases metabolic health and life span in numerous animal models and in vitro. Moreover, with the help of a presumed vegan diet, IGF-1 suppression and MR can be achieved. Amazingly, some studies have shown that protein load is the major factor in the progression of ADPKD in animal models. Although many types of research have shown that there are no beneficial results on the restriction of protein in patients with progressive ADPKD and kidney failure. Certainly, a trend has been demonstrated for a higher ratio of injuries consuming low protein. According to this, in further studies, there is a need to obtain the crucial role of macronutrients and calories in consequence of nutritional management presented here. Moreover, it is imperative to accomplish the time management of dietary manipulations in humans with ADPKD, where nutrient limitations may have conflicting effects before and after

the development of the disease [11]. In addition, it can also happen that nutrient management like FR can play a role in enhancing the effects of pharmacologic suppressors of the mTOP pathway, such as rapamycin. Antihypertensive, broncho-dilating activity and antispasmodic T. ammi has an antihypertensive effect that can be organized intravenously in vivo, and the broncho-dilating actions and antispasmodic can be administered in vitro. It has been found that plant material shows antispasmodic effects mediated by studies of calcium channel blockage and reviewed that this mechanism participates in their experimental conclusions and reinforced the typical usage of T. ammi diseases like hypertension and states of the gut like diarrhea and colic [12]. According to our study, it has been concluded that preventive effects have appeared with the pre-treatment of black cumin on renal reperfusion injury (I/R) of a kidney. Histological examination and functional parameters have shown this evidence. In this research, worsening kidney functions such as the rise of serum urea, creatinine, and uric acid characterize kidney tissue injury demonstrated by reperfusion injury in animals. According to enhance histopathological destructions these changes might occur like interstitial edema, tubular necrosis, cellular vacuolization, glomerular changes, and hemorrhage, and hyperemia. Though, in the reperfusion rats, black cumin with pre-treatment, histopathological and functional changes were reversed just as biochemical remarks were maintained by a histopathological test of renal stones [13]. Glomerular filtration rate has indicator markers such as Blood Urea Nitrogen (BUN), Serum Creatinine (Scr), and uric acid. Rather than sham operated rats, I/R rats have significantly higher levels of BUN, Scr, and uric acid. It shows that after I/R operations, renal dysfunction occurred. Our study concluded that black cumin pre-treatment lowers the levels of uric acid, BUN, and Scr which were persuaded by the ischemia-reperfusion process. It proves that IR-induced renal dysfunction can be helpfully prevented by pre-treatment with black cumin in a dose-dependent manner. Current research also exposed that the ratio between the weight of the kidney and the weight of the body significantly enhanced and decreased urine production significantly. It is supposed that after renal reperfusion outcomes, interstitial edema by the result of an increased ratio of kidney weight to body weight, and edema is improved by the pre-treatment with black cumin. In adults, oliguria is defined as a urine output of less than 400ml or 500ml per 24 hours [14]. Oliguria is induced by acute tubular necrosis, which is done by different mechanisms. In the first process, the urine flows from the damaged or necrotic tubules within the basement membrane in the renal interstitium due to the loss of

epithelium damaged by necrosis. Other than that, the renin-angiotensin process that affects the glomerular blood flow, filtration, and cortical nephrons is significantly reduced, resulting in less urine formation. Inflammation of various renal tissues, damage to the tubular system, cell necrosis, and glomerular structure injury with the cell debris are all characteristics of acute renal failure damaged by reperfusion and ischemia. Most of the dysfunction and destruction process occurs during the reperfusion (re-oxygenation injury) along with anoxia (hypoxia) & Reactive Oxygen Species ROS (Reactive Oxygen Species) gas being the major contributing factor in reperfusion injury. It is also responsible for cell death due to lipid peroxidation of biological membranes [15]. The cellular pathogenic activity of I/R42 includes free radical scavengers produced during I/R support. The generation of reactivating oxygen species ROS 43,44 plays a role in the kidney injury during the I/R process, they are the result of renal reperfusion and have different toxic effects for example damage to the DNA, oxidation of the protein, apoptosis induction, and nitrosylation. Black cumin's effect on lipid peroxidation was measured in terms of MDA assessed as a stable metabolite of the free radical-mediated lipid peroxidation process [16]. Our research demonstrated enhancement in the renal MDA and antioxidant enzyme pool reduction demonstrated by animals subjected to renal I/R. To some noticeable extent, the increase of MDA is reversed by black cumin, approving their antioxidant role in I/R, representing that protein oxidation and lipid peroxidation are prevented by black cumin in renal I/R. In our research, renal ischemia reperfusion reduction also occurs in GSH which will also be called depleted following an ischemic insult. Rather than their respective controls, pretreated rats demonstrated higher GSH content, representing that in replenishing the GSH pool black cumin helped. The statistic that in comparison with the I/R group, a significant increase was caused by black cumin in GSH-Px, CAT, and, SOD activities. It represents that it may reflect its antioxidant nature in enhancing enzyme activities like GSH-Px, CAT, and SOD. Therefore, the antioxidant activity of black cumin can significantly explain its protective role mechanism in renal I/R injury [17]. The rennin angiotensin system plays a crucial role in hypertension and blood pressure regulations. To form Angiotensin-I, renin acts on angiotensinogen, with the support of the angiotensin converting enzyme, it is converted into angiotensin-II. By gathering all pieces of evidence, intracellular formation of ROS i.e., hydrogen peroxide and superoxide anion (associated with renal damage) stimulated by angiotensin-II. Reperfusion damage includes many causes in which the generation of ROS is one of the main causes. Augmented ROS production

or reduced ROS sifting ability can cause oxidative strain. The inefficiency of cell organization and function can cause by peroxidation. Peroxidation is the process in which ROS link to the polyunsaturated fatty acids in the tissue fats. After re-oxygenation and reperfusion, the inequity between the renewal of oxygen source and mitochondrial respirational function concludes in the immense generation of superoxide anion in mitochondria [18]. Below these circumstances, the protective system, which is called antioxidant enzymes or antioxidants, can't escape the outflow of ROS, specifically in mitochondria, and their impacts on other intracellular sites. This cataract of proceedings is recognized as reperfusion injury. In this research, renal I/R maximized oxidative strain products comprising tissue MDA and washed out the antioxidant enzymes pool, as is obvious from the decreased action of superoxide dismutase, glutathione peroxidase, reduced glutathione, and catalase. It can be shown that pretreatment with black cumin prohibited renal I/R injected lipid peroxidation and prevented the kidneys from the acute raising of ROS products, and reduced glutathione peroxides, superoxide dismutase catalase, and decreased glutathione in rats uncovered to the renal I/R. Taken in all, the usage of this plant in reperfusion injury/renal ischemia is then sustained but the specific active substances of cellular, sites, molecular mechanisms, and black cumin of its pharmacological impact and likely harmfulness and collaboration with other medications are still to be considered. Correspondingly, because of the extended span of providing black cumin, before three weeks of ischemia, this can postpone treatment of stones and renal tumors and be intolerable for kidney harvesting for patients with kidney trauma and cadaveric donors [19]. The various segments of black cumin seeds in different dosages must be measured through animal modeling for their quick therapeutic potential. Though in an animal model, records favor black cumin's role in reducing kidney harm after reperfusion injury/ischemia of the kidneys, however, upcoming research and trials to find the effectiveness and finest doses of this plant in human bodies are essential [20]. In PKD, the mechanism that can slow CKD progression is diet, a disease modifier according to clinical and preclinical evidence. Compliance has been a critical issue with the dietary management of the disease, especially when immediate adverse consequences are not resulting from non-compliance, apart from the utopian reports from a few patients followed for a few weeks. For instance, sodium restriction is recommended by many guidelines and how to monitor sodium intake. No provision is made. It seems realistic as it would probably be required 24hr urine collections. In this regard, compliance may be facilitated by periodic assessment of targets. Regarding calorie

restriction, in all the tested species so far, the only maneuver that has increased life expectancy is the concern about compliance that has led to developing drugs that impersonate the effects of calorie restriction [21]. To help combat monotony and enhance compliance, low osmole/high water diets food restriction to high fat ketogenic diets, or time-restricted ketogenic diets may offer the variety that allows switching from one regime to another and design diverse dietary approaches that slow down PKD progression. Additionally, to promote compliance, the active participation of the patients in guideline development can help to create patient-centered recommendations. So far, Autosomal Dominant Polycystic Kidney Disease (ADPKD) patients that are specially tailored by no dietary intervention trial, have assessed the impact on ADPKD progression. Currently, both with a target urine osmolality of ≤ 270 mOsm/kg, ongoing or recently completed clinical trials are only exploring an increased water intake (Prevent-ADPKD and Drink). Unfortunately, we are not optimistic about compliance based on prior trials in general CKD patients [22]. However, salt restriction, which provided benefits despite rampant non-compliance with salt intake targets, there is a margin for eventual clinical success. Therapeutic relationships are highly emphasized by the contribution to compliance and how much the clinician relies on and transmits the efficacy of the dietary manipulation. From a worldwide perspective, to further pursue potential dietary approaches for ADPKD care, the lack of access to drug therapy for most of the human population emphasizes this need. Potential indications for specific dietary manipulation in this context will be appropriate and should be stressed as given by the known pathophysiology of ADPKD [23]. Moreover, the fact that in the absence of renal function, ADPKD diagnosis can be made, decline offers the important possibility to act in a preventative way with a specific dietary manipulation. This is independent of the classical protein intake restriction typical of advanced CKD stages, coming from preclinical studies provided by clinical trials, which confirm the promising results. For this reason, the need of the hour for these clinical studies in this field should be emphasized.

CONCLUSION

This case was distinctive of a patient complicated with hematuria, higher intra-abdominal mass, hypertension, and severe headache who secured recovery results with adequate therapy and diagnosis. This intimate devotion to the low-calorie DASH diet, cumin, and ajwain pose a favorable curative potential for PKD patients.

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