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Ayurvedic Management Of Polycystic Kidney Disease Patient – A Case Study

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Abstract: Polycystic kidney disease (PKD) is an autosomal dominant disorder. It is a multisystem and progressive disease with cysts formation and kidney enlargement along with other organ involvement (e.g., liver, pancreas, spleen). Cystic kidneys are common causes of end-stage renal disease, both in children and in adults. In the adult population, it is the most frequent genetic cause of renal failure, with 6% to 8% of patients on dialysis in the US. By the age of 60 years, 50% of them need renal replacement therapy. Cysts may be detected in childhood or in utero, but clinical manifestations appear in the third or fourth decade of life. Enlarging cysts destroy the structure of nephrons, ultimately resulting in the loss of renal function. Eventually, ADPKD develops into end-stage renal disease. Currently, there is no effective drug therapy that can be safely used clinically. The conventional approach of management includes dialysis and renal transplantation, which are not affordable by Indian population mainly due to economic reasons. Therefore, exploration of a safe and alternative therapy is needed, which proves to be helpful in reducing the requirement of dialysis and in postponing the renal transplantation. It is a case of Polycystic kidney disease (PKD) which is correlated with Mutravaha Stotodushtijanya Vyadhi. A patient 46 year old presented in the OPD with Complaints of loss of weight, loss of Appetite, fatigue and also brought the RFT report which showed high creatinine, and high blood urea. After clinical examination, she was treated with oral medication for 3 months. She had relief in symptoms and also improvement in RFT reports.

Index Terms - PKD, Cysts, Mutra Vaha Srotas, Medo Dushti, Vrukka Roga.

I. Introduction

In the present era some diseases are prevailing worldwide with a large number of complications. Among such disorders, chronic kidney disease is one. Chronic Kidney disease has become the most lingering disease caused by several factors like Diabetes type 1 and 2, Family history of renal disease, Autoimmune disease, Systemic infections, Urinary tract infections/stones Urinary tract obstructions, Recovery of acute kidney injury, Polycystic kidney Disease (PKD) Hypertensives Drug abusers: Nonsteroidal anti-inflammatory drugs, analgesics/ heroin, Neoplasia, and Low birth weight Reduced kidney mass etc¹.

Autosomal Dominant Polycystic Kidney Disease (ADPKD) is the most common hereditary kidney disorder, characterized by the progressive development of numerous fluid-filled cysts in the kidneys. These cysts gradually replace normal kidney tissue, leading to kidney enlargement and impairment of renal function over time. ADPKD affects both kidneys and can also involve other organs such as the liver, pancreas, and spleen².

ADPKD affects approximately 1 in 400 to 1 in 1000 individuals worldwide, making it a relatively common genetic disorder. The disease exhibits variable expressivity and age of onset, even among affected family members, due to genetic modifiers and environmental factors³.

ADPKD is primarily caused by mutations in two genes: PKD1 and PKD2. Mutations in the PKD1 gene are more common and generally associated with a more severe disease course compared to mutations in PKD2. These genes encode for polycystin-1 and polycystin-2 proteins, which play crucial roles in maintaining normal kidney structure and function, particularly in the regulation of tubular epithelial cell growth and differentiation⁴.

Clinical manifestations often become apparent in adulthood, typically between the ages of 30 and 50 years old. Common symptoms include flank pain, hypertension (due to activation of the renin-angiotensin-aldosterone system), hematuria (blood in the urine), and progressive renal insufficiency leading to chronic kidney disease (CKD) and potentially end-stage renal disease (ESRD)⁴.

Diagnosis of ADPKD is primarily based on imaging studies, such as ultrasound, CT scan, or MRI of the abdomen, which reveal the presence of multiple cysts in the kidneys. Genetic testing may also be utilized to confirm the diagnosis, especially in cases with atypical presentations or when family history is unclear⁴.

Management aims to control symptoms, slow disease progression, and manage complications such as hypertension and CKD. Regular monitoring of kidney function, blood pressure, and screening for associated complications (e.g., intracranial aneurysms) is essential. The prognosis varies widely, with some individuals remaining asymptomatic for many years while others progress to ESRD requiring renal replacement therapy (dialysis or transplantation)⁴.

Vrikka is comparable with kidney of contemporary science. *Vrikka* (kidney) regulate the removal of wastes from the blood in the form of urine.

मेदोवहानां स्रोतसां वृक्कौ मूलं वपावहनं च | [च.वि.५/८]

Vrikka is considered as Moolsthana of Medovaha Srotas⁵.

रक्तमेदःप्रसादाद्वक्कौ | [सु.शा.४/३१]

Vrikkaroga can be possible due to disequilibrium of *Shonita* and *Meda* as they form from it⁶. The kidney diseases mentioned in modern science and their common symptoms can be correlated with *Mutraroga* described in *Ayurveda*

The main function of *Mutravaha Srotas* is production of *Mutra*. Expelling out the liquid excretory products through *Mutra*. It carries the *Kleda tatwa* of the body - so " *Mutrasya Kleda Vahanam*. This *Mutra* does filling of *Basti (Basti Purana)* and gives *Mardavata* or *Mrudutwa* to *Basti⁷*. Whatever *Udaka* or *Kleda* is produced after the digestion is further gets transformed or metabolized into *Mutra* in *Mutravaha Srotas*. So "*Srotamsi khalu Parinama mapadyamananam Dhatunam abhivaheeni*" can be understood here⁸.

Acharyas have used the terms like Vrukka, Gavini, Basti, Mutra praseka, Mutravaha Srotas, Mutravaha Dhamani, Mutravaha Sira, and Mutra vaha Nadi in relation to explain Urinary system. Excess of kleda which is segregated from the udaka in the Shareera at Basti as Mutra. There are two source of kleda. They are Kosta/Amashaya and Shareeragata Kleda. 'Kittamannasyavin Mutram' the gross food which we consume has the fluid portion and the most portion of fluid which we consume is absorbed in to the system. The terms such as Rasakleda, Sonitakleda, Mamsakleda, Dhatukleda, Srotokleda, etc clearly suggest the formation and presence of kleda at different tissue level in the body.

The *mula* of *Ambuvahasrotas* is *Talu* and *Kloma*. But it is also rooted in *Amashaya/Kosta*. At *kosta*, it is in association with *Swedavahasrotas*, *Doshavahasrotas*, *Pachaka pitta*, *Kledaka kapha*, and *Samanavata*. Hence, *jaleeya dhatu* gets absorbed from *Kosta*. The mechanism of digestion, segregation of *sara kitta*, assimilation of *sara*, and facilitation of excretion is elaborated by *Sushrut Acharya*.

Anukta Vyadhi refers to diseases that are not explicitly described in classical Ayurvedic texts but are encountered in clinical practice. These unspecified diseases pose a unique challenge and opportunity for Ayurvedic practitioners, requiring a nuanced understanding of Ayurvedic principles & treatment approaches.

PKD is often associated with an imbalance in the dosha like *Vata, Kapha* and *Pitta, dhatus* like *Rasa, Rakta, Meda,* and *mala* like *Mutra, Vata* imbalance can contribute to the formation and growth of cysts, while *Kapha* imbalance may exacerbate fluid retention and swelling in the kidneys and *Pitta* imbalance may leads to *Rakta Dushti*. In *Ayurveda, Meda dhatu* (fat tissue) is closely related to *Kapha dosha* and also *Moola of medovaha* Srotas is *vrukka* so *Meda* and *Vrukka* can be affected in conditions where there is excess *Kapha*. Managing *Rasa Dhatu* through *Dipana & Pachana* and *Meda dhatu* through *Lekhana karma* (scraping or cleansing therapies) may help in reducing *Kapha* accumulation and supporting kidney health in PKD. Considering this, it has been planned to Treatment with *Dipana & Pachana* With *Vaishwanar Churna* For First 7 Days then *Rasayan Churna* with *Abhrak Bhasma, Gokshuradi Gugglu & Punarnavadi + Varunadi Kwath* also *Niruha Basti* of *Punarnavadi Kwath* Daily For 7 Day's.

II. CASE REPORT

Patient Information (Vedana Vruttanta)

A 46 years old female patient came to my Opd, with the complains of intermittent dull flank pain on both sides for the past year, with occasional episodes of haematuria, loss of weight, anorexia, insomnia, and fatigue. She also brought the RFT report, which indicates the Value of Serum Creatinine – 2.42 mg/dL and blood Urea – 51 mg/dL. There is no family history of kidney disease. She reports Low urinary frequency, decreased bowel movement, and average of 4-5 hours of sleep per night.

Clinical findings

On physical examination, pallor, icterus, clubbing, cyanosis and lymphadenopathy was absent. Her Pulse was 82/min and BP was 128/86 mm hg. Her height was 172 cm and weight was 52 kg. On abdominal examination shows bilateral palpable renal masses consistent with multiple cysts.

Ashtavidha Pariksha (eight-fold examination) was done which reveals Nadi Vata Pradhana Kaphaja, Mootra Pravruti was 2-3 times/day. Mala Pravruti 1 time a day, which was malabaddhata. Jihva was Saama (whitish-coated). Patient's Aakriti (general body built) was Avara.

Dashvidha Pariksha (tenfold examination) of the patient was done which illustrate that the patient was Vata-Kapha Prakriti (physical built), Vikriti – Vata-Kapha Pradhana, Sara and Samhanana were Madhyama, Satva-Avara. The patient had madhur and Lavana rasa Priyata with Madhyama Abhayvarana Shakti, Jaran Shakti, and Vyayama Shakti was Madhyama, which reveals that Roga Bala is Pravar and Rogi Bala was Madhyama.

Investigations

Blood tests suggested renal function with a serum creatinine of 2.82 mg/dL and a blood urea nitrogen (BUN) of 51 mg/dL. Urinalysis shows microscopic hematuria. Renal ultrasound (USG) confirms multiple bilateral renal cysts of varying sizes with no evidence of renal calculi or hydronephrosis.

Treatment Protocol

First Treatment start with *Dipana & Pachana* With *Vaishwanar Churna* Tablet 2 Tab 3 times/day For First 15 Days then also start *Rasayan Churna* + *Abhrak bhasma* with *Ushnodak* 2 Times Before Food, *Gokshuradi Gugglu* 2 Tab with *Ushnodak* 3 Times Before Food & *Punarnavadi* (15ml) + *Varunadi Kwath* (15ml) 2 Times Before Food also *Niruha Basti* of *Punarnavadi Kwath* Daily For 5 Day's.

Follow-up and outcome

Patient was assessed before and after the treatment, After Completion of 3 Months of Protocol Reduced Frequency of Flank Pain, no Hematuria, Weight Also Gain, No Anorexia and Insomnia. Laboratory Investigation Reports also improved.

Discussion

PKD is specific form of renal disease. According to *Ayurveda*, PKD is a disease of *Mutravaha Srotas*. Though all the three *Doshas* as well as all the *Dushyas* are involved in the disease, *Kapha* is responsible in blocking microvessels and developing microangiopathy with fluid retention and swelling in the kidney. *Vata* is responsible for degeneration of the structure of the kidney and formation and Growth of cysts. According to *Ayurvedic* principles of management of the disease, tissue damage can be prevented and repaired by *Rasayana* drugs because they have the capability to improve qualities of tissues and hence increase resistance of the

tissues. On the other hand, blockage can be removed by Lekhana drugs having scraping effect on blocked channels⁹.

Goksuradi guggulu¹⁰ (combined Ayurvedic preparation) is Rasayana for Mutravaha Srotas and it has also Lekhana (scraping) effect because of Guggulu (Commiphora mukul)¹¹. Varunadi kwath¹² is also helpful to relieve the Kapha and Vata doshas. Rasayan churna has Rasayan properties. Niruha basti is a minor alternative of dialysis. Reduction in fatigue, increase in appetite, and relief from nausea, and flank pain highly significant.

So to prevent the resistance of tissue, Rasayan drugs were chosen such as Gokshur, Rasayana Churna, Abhrak Bhasma, Punarnava etc. So as to prevent and repair the tissue damage and enhance the quality of tissue.

Also lekhana Dravyas Such as Varun, Sunthi, Haritaki etc. having Rooksha Guna which help in drying of Ama, bring out a clear Rasa Dhatu and Clear the Srotas, further helping in good circulation making way for proper *dhatu* nourishment.

Especially *Punarnava*, *Gokshura* are recommended exclusively in the disorders of *Mootravaha Samsthana*. These drugs should be accepted as Naimittika Rasayana for kidney and other organs of Mootravah Srotas. Rasayana drugs bear the property of antioxidant and work as free radical scavengers. Also, the above mentioned drugs have *Mutral* properties which resolve the problem of *Mutrakriccha*¹³.

Conclusion

The disorder of *Mutravaha* Srotas has resemblance with the description of urological disorders of modern parlance. On the basis of history and clinical presentation, the patient was diagnosed as case of PKD, it's treatment with Ayurveda was found encouraging¹⁴. In all types of urine disorders, Vata dosha is the prime causative factor collaborating with Pitta and Kapha. Hence, general treatment is applied considering Tridoshas, drug and disease¹⁵, especially Vata and Kapha Shamak Dravyas were chosen for management of this case to break the Samprapti Chakra, hence emitting the symptoms, thus preventing kidneys from its failure to perform its vital functions. Also, the drugs chosen showed promising results in parameters pertaining to quality of life. So, it shows

Patient consent details

Authors declare that they have obtained patient consent form, where the patient has given his consent for reporting the case along with the images and other clinical information in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal her identity.

Sr. No Name Of Medicine Dose **Time Anupana** No. Of Days Vaishwanar Churna Ushnodak 15 Days 1 2 Tab 3 **Times** Before Food 2 Gokshuradi 2 Tab 90 Days Times Punarnava + Guqqulu Before Food Varunadi Kwath 3 2 Ushnodak 90 Days Rasayan Churna 3 Gram Times 100 Gram + Abhrak Before Food Bhasma 5 Gram 2 Add 1 Punarnava 15 ml + 30 ml Times 90 Days Tsp Before Food Varunadi 15 Ghrita Kwath

Table 1 (Treatment Protocol)

Table 2 (Procedure)

Sr. No	Procedure	No Of Days
1	Punarnava Kwath Niruha Basti in Dose of 750ml Through Anal	5
	Route	

Table 3 (Follow-up & Outcome)

Laboratory Investigation	Before Treatment	During Treatment		After Treatment
Serum	2.42 mg/dL	2.2 mg/dL	2.1 mg/dL	1.30 mg/dL
Creatinine	(03/07/2023)	(03/08/2023)	(28/09/2023)	(31/10/2023)



Date:-03/07/2023 Date:-03/08/2023 Date:-28/09/2023 Date:-31/10/2023

Figure No. 01-04 S. Creatinine Report Improved With Treatment

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