



## Polycystic Renal Disease Presented by Anuria and Nephrolithiasis Associated Progressive Kidney Damage: A Case Report

Müge KARACAKAYALILAR<sup>1</sup>, Selman CANDAN<sup>2</sup>, Mehmet Çağatay ÇİÇEK<sup>3</sup>, Abdülmecit YILDIZ<sup>4</sup>,  
Münevver İrem KÖK<sup>5</sup>

<sup>1</sup> Bursa Mustafakemalpaşa State Hospital, Internal Medicine, Bursa, Turkey

<sup>2</sup> Bursa Uludağ University, Department of Radiology, <sup>3</sup>Department of Urology, <sup>4</sup>Department of Internal Medicine, Division of Nephrology, Bursa, Turkey

<sup>5</sup> Tokat Zile State Hospital, Tokat, Turkey

*Turk J Int Med* 2021;3(Supplement 1):S44-S45

DOI: [10.46310/tjim.866999](https://doi.org/10.46310/tjim.866999)

**Keywords:** Anuria, Nephrolithiasis, Polycystic renal disease

Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited kidney disease.<sup>1</sup> Kidney stones develop in 20-30% of patients with ADPKD and urinary stones are usually treated with conservative methods (urinary alkalinization, spontaneous stone passage, extracorporeal shock wave etc.).<sup>2,3</sup> Uric acid stones and calcium oxalate stones are the most frequently detected stones and seen in similar proportions.<sup>4</sup> Hydronephrosis, which is the most valuable radiological finding in the diagnosis of stone-related postrenal insufficiency, may be difficult to differentiate from common cysts in ADPKD patients.<sup>3</sup> When kidney dysfunction develops in ADPKD patients, glomerular filtration rate (GFR) loss reaches an average of 4.4 to 5.9 mL/min per year.<sup>1</sup> Faster deterioration in these patients requires investigation for prerenal factors such as dehydration that triggers acute kidney damage or stone-related postrenal factors.<sup>2</sup> In ADPKD, stone-related postrenal obstruction should be considered in rapid GFR losses despite negative ultrasound report for hydronephrosis as there is frequent occurrence of kidney stones and the difficulty in detecting hydronephrosis with ultrasonography (US) in these patients.<sup>5</sup> Here, we presented a case of ADPKD who developed stone-related renal dysfunction during chronic follow-up, and renal function improved after the intervention.

The 73-year-old patient, who was diagnosed

with ADPKD and was in outpatient follow-up with basal creatinine level of 1.6 mg/dL and GFR: 62 mL/min, had a history of type 2 diabetes, hypertension and occlusive type of cerebrovascular events. During the follow-up, the creatinine level increased from 1.6 mg/dL to 3.6 mg/dL. In the evaluation, there was no new drug use or fluid loss suggesting a prerenal event. The patient, who had no signs of pain, bleeding, and urinary tract infection, was considered to have the accelerated natural course of ADPKD, considering the high creatinine level and accompanying diseases. In the evaluation one week later, the patient's general condition was worsened, the creatinine value increased to 7.1 mg/dL and GFR was 10 mL/min then patient has hospitalized for further investigation and treatment. No postrenal pathology was detected in US performed to detect hydronephrosis for the diagnosis of postrenal renal insufficiency in the patient who has been on emergency hemodialysis due to uremic symptoms. In order to explain the current situation, noncontrast computed tomography (CT) has been performed and it showed dilatation in the pelvicalyceal system of both kidneys and stones in the distal ureters on the both sides, which were 9.5x4.5 mm and 5.5x3 mm, respectively. In addition, right indirect inguinal hernia was observed simultaneously in the case. In the emergent bilateral ureterorenoscopy performed by the urology clinic, the stones causing complete



Received: January 30, 2021; Accepted: March 6, 2021; Published Online: March 6, 2021

**Address for Correspondence:**

Müge KARACAKAYALILAR

Bursa Mustafakemalpaşa State Hospital, Internal Medicine, Bursa, Turkey

E-mail: [mugekaracakaya@uludag.edu.tr](mailto:mugekaracakaya@uludag.edu.tr)



obstruction at the bilateral distal ureter level were observed. Bilateral Double J stents were placed to the bilateral collecting system in the same session. After the procedure, urine output is observed, and the creatinine level decreased to 1.7 mg/dL, which is the basal level.

After development of the renal failure in ADPKD, progression is usually rapid and varies according to genetic and clinical risk factors.<sup>6</sup> It should be kept in mind that stones are observed with high frequency in these patients and unexplained accelerated progression in deterioration may be caused by obstruction in the urinary system.<sup>2,7</sup> Cysts in both kidneys that disrupt the normal anatomy can easily be confused with pelvic enlargement due to obstruction. Therefore, in patients with a diagnosis of ADPKD who are investigated for postrenal kidney failure, pelvic enlargement may not be differentiated by US because of the abundant cysts. In US, which is inexpensive, practical, lack of radiation exposure and the first choice in the evaluation of postrenal pathologies, pelvicalyceal dilatation can easily be evaluated as a cyst in these patients. Obstruction due to stones in these patients may result in irreversible renal failure when not diagnosed in the early period.<sup>5,8</sup>

Stones may cause acute renal failure in ADPKD, usually due to infection or urosepsis, persistent pain, vomiting and rarely bilateral obstruction in the ureteropelvic region, and urgent intervention is required.<sup>9</sup> In the present, extracorporeal shock wave (ESWL), ureteroscopy, percutaneous nephrolithotomy (PCNL) and retrograde intrarenal surgery (RIRS) are the procedures commonly used in the treatment of urinary stone disease.<sup>5,10</sup>

The prevalence of stones in patients with ADPKD has been reported in variable rates in the literature, and the prevalence of urinary stones varies between 3% and 59%. In cases with urolithiasis, the rate of patients who undergo interventional treatment is between 1-8%, but there is no meaningful data on the rate of stone-related interventions in adults with ADPKD.<sup>2,7</sup> Stone-related pain in ADPKD can be confused with pain due to cyst rupture. However, it should be considered in patients with acute GFR losses under chronic follow-up, as in our case, and advanced imaging with low-dose non-contrast CT should definitely be performed in these patients if the diagnosis cannot be made by US.<sup>3</sup>

Imaging methods, especially US, may be insufficient to distinguish pelvicalyceal stones because some of the cysts may contain wall or septa calcification or calcium milk. Diagnostic uncertainty may occur in the detection of kidney stones. Therefore, the best imaging method

is high-resolution CT that detects stones and calcifications. Studies suggest that non-contrast CT is the preferred imaging method for the diagnosis of suspected nephrolithiasis in ADPKD.<sup>5,8</sup>

We presented this case in order to explain that nephrolithiasis accompanying polycystic kidney disease may lead to clinical picture of postrenal acute renal failure, anuria and progressive kidney damage, and that it should be accurately diagnosed with appropriate imaging technique in order not to be overlooked, as this problem can be reversed with appropriate intervention.

### Conflict of Interests

Authors declare that there are none.

### Acknowledgment

This study has been presented in 17<sup>th</sup> Uludag Internal Medicine National Winter Congress, 6<sup>th</sup> Bursa Family Medicine Association National Congress, 11<sup>th</sup> Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

### References

- Torres VE, Harris PC, Pirson Y. Autosomal dominant polycystic kidney disease. *Lancet*. 2007 Apr 14;369(9569):1287-1301. doi: 10.1016/S0140-6736(07)60601-1.
- Kalatharan V, Grewal G, Nash DM, Welk B, Sarma S, Pei Y, Garg AX. Stone prevalence in autosomal dominant polycystic kidney disease: A systematic review and meta-analysis. *Can J Kidney Health Dis*. 2020 Jul 4;7:1-19. doi: 10.1177/2054358120934628.
- Gaur P, Gedroyc W, Hill P. ADPKD-what the radiologist should know. *Br J Radiol*. 2019 Jun;92(1098):20190078. doi: 10.1259/bjr.20190078.
- Veser J, Özsoy M, Seitz C. Congenital and acquired diseases related to stone formation. *Curr Opin Urol*. 2018 Sep;28(5):414-9. doi: 10.1097/MOU.0000000000000522.
- Mallett A, Patel M, Tunncliffe DJ, Rangan GK. KHA-CARI autosomal dominant polycystic kidney disease guideline: Management of renal stone disease. *Semin Nephrol*. 2015 Nov;35(6):603-6.e3. doi: 10.1016/j.semnephrol.2015.10.012.
- Ghata J, Cowley BD Jr. Polycystic kidney disease. *Compr Physiol*. 2017 Jun 18;7(3):945-75. doi: 10.1002/cphy.c160018.
- Mufti UB, Nalagatla SK. Nephrolithiasis in autosomal dominant polycystic kidney disease. *J Endourol*. 2010 Oct;24(10):1557-61. doi: 10.1089/end.2010.0093.
- Chapman AB, Devuyst O, Eckardt KU, Gansevoort RT, Harris T, Horie S, Kasiske BL, Odland D, Pei Y, Perrone RD, Pirson Y, Schrier RW, Torra R, Torres VE, Watnick T, Wheeler DC; Conference Participants. Autosomal dominant polycystic kidney disease (ADPKD): executive summary from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. *Kidney Int*. 2015 Jul;88(1):17-27. doi: 10.1038/ki.2015.59.
- Grantham JJ. Clinical practice. Autosomal dominant polycystic kidney disease. *N Engl J Med*. 2008 Oct 2;359(14):1477-85. doi: 10.1056/NEJMcp0804458.
- Skolarikos A, Alivizatos G, de la Rosette J. Extracorporeal shock wave lithotripsy 25 years later: complications and their prevention. *Eur Urol*. 2006 Nov;50(5):981-90; discussion 990. doi: 10.1016/j.eururo.2006.01.045.

