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# A case report – Young age polycystic kidney disease

[1]Dr. Bhargav Reddy

**Corresponding Author:** Dr. Bhargav Reddy

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**Abstract**

An uncommon condition known as atypical or unilateral polycystic kidney disease is characterized by an imaging finding of an asymmetric or unilateral distribution of cysts restricted to the kidneys. We describe a case of a 72-year-old man who had atypical polycystic kidney disease that was discovered by accident. Only the kidneys displayed asymmetric cyst distributions on computed CT imaging, and the patient had no genitourinary complaints, normal renal function, or a family history of renal disease. Despite being benign, there have been a few isolated occurrences of atypical polycystic kidney disease progressing to bilateral polycystic kidney disease, which portends a worse prognosis. Clinicians must be aware of this phenomenon in order to periodically check on patients for illness development. Renal cysts are frequent conditions that can lead to renal failure or be clinically inconsequential. A disease syndrome with extra-renal symptoms such cerebral aneurysms, heart valvular disease, colonic diverticula, and extra-renal cysts may include them, or they may be singular incidental findings, have a genetic component, or be a result of genetics. The most prevalent form of polycystic kidney disease in adults is autosomal dominant polycystic kidney disease (ADPKD), which often manifests as a symmetric involvement of both kidneys and an abundance of cysts. In 50% of instances, ADPKD, which is categorized as a ciliopathy, develops to renal insufficiency and end-stage renal failure [1]. It is impossible to distinguish between conventional bilat eral polycystic kidney disease (PKD) and atypical polycy stic kidney disease (PKD) using histology or radiograph. There is no known genetic predisposition, it is thought to be benignit does not proceed to renal failure, and it is not known to have further renal symptoms.

**Keywords:** PKD, CT, Renal.

## Case Report

During the course of 4 days, a 48-year-old male patient developed a rectal abscess and complained of rectal pain. The patient disputed the existence of any fevers, chills, back discomfort, abdominal pain, or any other relevant symptoms. Physical examination revealed that the patient had a draining rectal abscess, but other than that, there were no noteworthy findings. The patient's vital signs were within normal ranges, and laboratory tests revealed that his Haemoglobin level was 7.4 g/dl, total white blood cell count was 17740, platelet count was 143000, urea level was 54, creatinine level was 7.7 mg/dl, and uric acid level was 4.1 mg/dl. LVH appeared mildly concentric on ECHO. LV systolic function that is normal,Grade 1 LV diastolic dysfunction, no anomalies in regional wall motion at rest.PCR in spot urine is 3062

mg/g. 111 mg/dl of urea, 12.9 mg/dl of creatinine, and 3.30 mEq/L of potassium. Urine analysis revealed many pus cells, RBCs of 10 to 12, and urine albumin +++. Bilateral polycystic kidney disease, polycystic liver disease, bilateral renal calculus, multiple calcified colon diverticulosis, and an umbilical hernia were accidentally detected on computed tomography (CT) scans of the abdomen and pelvis. The left kidney is 16.9 by 8.2 and the right kidney is 15.8 by 7.8 in size. The patient was released and given a standard outpatient follow-up appointment.

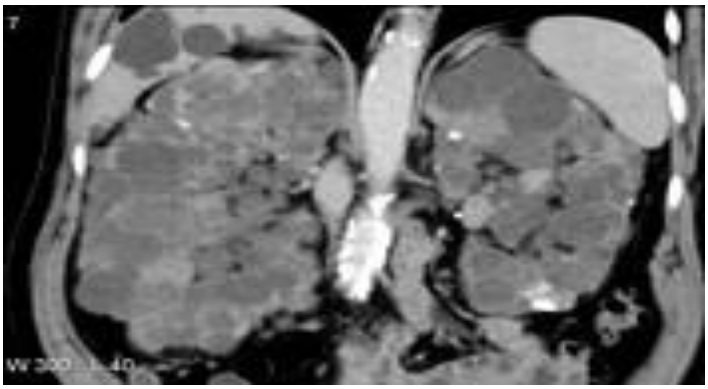

Figure 1: CT KUB of polycystic kidney disease.

**Discussion**

Atypical polycystic kidney disease (APKD) is a rare, benign condition that is indistinguishable from autosomal dominant polycystic kidney disease (ADPKD) both radiographically and histologically, but is currently recognized as a completely separate entity [2,3]. It can be differentiated from ADPKD in several ways. APKD is benign, non–progressive, does not have extra-renal manifestations, and is thought to be a non-genetic disorder whereas ADPKD is symmetric, bilateral,and monogenic [4,5]. There has been documentation of unilateral ADPKD, but is extremely rare [4,5]. Unlike ADPKD and some other polycystic kidney diseases, extra-renal cysts are not seen in APKD [6]. APKD is described as having multiple cysts involving the entire kidney or a specific region of the kidney with intervening normal parenchyma [3] The cortical and medullary locations, general simplicity, and tendency for renal cysts associated with ADPKD to grow in size and quantity over time [10] are true. The membrane proteins polycystin-1 and polycystic 2 are lost specifically in ADPKD due to mutations in the PKD1 and PKD2 genes, respectively. Along with other local or somatic variables, the absence of these proteins, which are typically responsible for fluid regulating activity inside primary renal cilia, is the main cause of cyst development. Up to 85% and 15%, respectively, of cases of ADPKD are caused by mutations in PKD1 and PKD2, with spontaneous de novo mutations accounting for 5%–15% of cases and having no known family history [10]. Other PKD cases in patients without a mutation are thought to be caused by somatic mosaicism. When a somatic mutation occurs during embryogenesis, it might result in the presence of two genetically different cell groups within a single individual. These mutations may affect somatic (body) cells as well as germline cells. For PKD cases with unusual kidney imaging patterns, such as asymmetric, unilateral, and lopsided patterns, somatic mosaicism is thought to be to blame [12]. Patients with APKD can exhibit signs like hematuria or flank pain, but they frequently show no symptoms at all, and the cysts are discovered by chance, as in our instance. Multiple simple cysts are shown on imaging, and they are distinguished from other renal cystic diseases like cystic nephroma by intervening normal increasing renal parenchyma and the absence of a capsule [13].The cysts typically exhibit localized involvement with a preference for the poles; complete kidney involvement is uncommon [13].Although only one kidney is often affected by APKD occurrences where the opposite kidney also contains a few cysts have been documented, as in our

case. These unusual occurrences can very rarely progress to bilateral illness [14].

We presented a case of APKD, an extremely uncommon condition. Despite the benign illness history shown by recent evidence, it is crucial for clinicians to recognize this diagnosis.As mentioned above, since cases of atypical PKD progressing to bilateral PKD have been described, patients may benefit from routine monitoring.

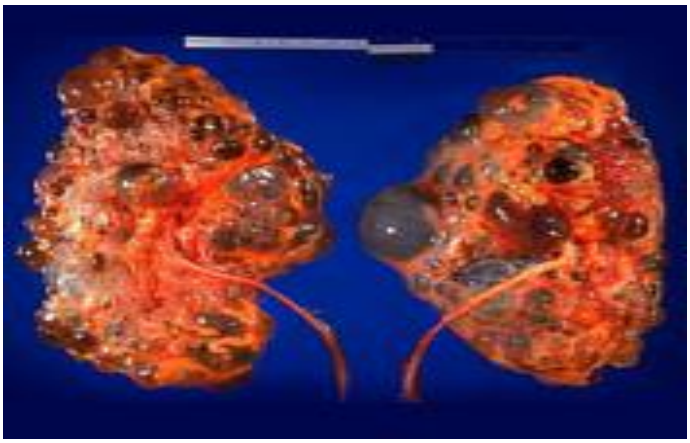

Figure 2: Gross picture of the polycystic kidney disease

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