Acute Pancreatitis in Polycystic Kidney Disease: Possible Etiologic Factor?: Case Report

Polikistik Böbrek Hastalığında Akut Pankreatit Gelişimi: Olası Etiyolojik Neden?

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Yazışma Adresi/Correspondence: Selim SERTER, MD Celal Bayar University, Faculty of Medicine, Department of Radiology, Manisa, TÜRKIYE/TURKEY serterselim@gmail.com **ABSTRACT** Autosomal dominant polycystic kidney disease (ADPKD) is the most frequent inherited poly-cystic disease characterized by multiple cysts in kidneys and other organs. Cysts are usually observed in both kidneys which can be in variable size and scattered out by renal cortex and medulla. Cysts can also be seen in liver, spleen, pancreas and other organs . In autopsy series, the prevalence of pancreatic cysts in patients with ADPKD is about 10 %. It has been a few case reports showed that, pancreatic cysts are rare causes of chronic pancreatitis in ADPKD patients. Herein we report a patient previously diagnosed for ADPKD presenting with acute pancreatitis and no cyst in pancreas.

Key Words: Polycystic kidney, autosomal dominant; pancreatitis, acute necrotizing; computed tomography

ÖZET Böbrekler ve diğer organlarda çok sayıda kist varlığı ile karakterize olan otozomal dominant polikistik böbrek hastalığı (ODPBH) kalıtımsal polikistik hastalıklar içinde en sık görülendir. Her iki böbrekte korteks ve medullaya yayılmış çok sayıda tüm parankimi dolduran farklı boyutlarda kistler olabilir. Böbrek dışında karaciğer, dalak, pankreas ve diğer organlarda da kistler görülebilir. Otopsi serilerinde ODPBH ile birlikte pankreatik kistlerin görülme sıklığı yaklaşık %10 kadardır. Literatürde ODPBH olan hastalarda pankreatik kistlere bağlı pankreatit gelişimi birkaç olgu sunumunda bildirilmiştir. Bu olgu sunumunda, akut pankreatitle başvuran, daha önceden ODPBH tanısı almış ve pankreasta kisti olmayan bir olgunun sunulması amaçlandı.

Anahtar Kelimeler: Polikistik böbrek, otozomal baskın; pankreatit, akut nekrotizan, bilgisayarlı tomografi

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utosomal dominant polycystic kidney disease (ADPKD) is the most frequent inherited poly-cystic disease characterized by multiple cysts in kidneys and other organs. ¹⁻³ Ultrasonography is the most important diagnostic imaging method and Ravine criteria are practically applied. Computed tomography and magnetic resonance imaging methods are much more useful in complicated cases and extrarenal involvement. ⁴ Cysts are usually observed in both kidneys which can be in variable size and scattered out by renal cortex and medulla. Cysts can also be seen in liver, spleen, pancreas and other organs. ^{3,4} In autopsy series, the prevalence

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of pancreatic cysts in patients with ADPKD is about 10%. It has been reported that, pancreatic cysts are rare causes of chronic pancreatitis in ADPKD patients.² Herein we report a patient previously diagnosed for ADPKD presenting with acute pancreatitis and no cyst in pancreas.

CASE REPORT

A 34-year-old woman was admitted with acute epigastric pain and vomiting. She had been suffering from this pain for 2 days. Autosomal dominant polycystic kidney disease (ADPKD) had been diagnosed at the age of 24 on systemic familial screening. Physical examination revealed normal vital signs and there had been no abnormal finding except an epigastric tenderness. She had mild hypertension which was treated by perindopril. She was not taking any other drugs, and had no history of abdominal injury, gallstones or chronic alcohol intake. A mild leukocytosis (12.000/mm³) and elevated serum amylase (ranging between 3 and 4 times the upper limit of normal value) and lipase (ranging between 5 and 8 times the upper limit of normal value) and urinary amylase (ranging between 2 and 3 times the upper limit of normal value) were the only abnormal laboratory findings. Informed consent was obtained before computed tomography (CT) and ultrasonography (US) examination. Abdominal ultrasound showed enlarged kidneys including multiple cysts (Figure 1). The pancreatic

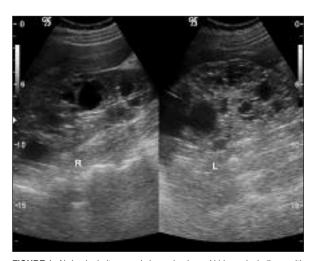


FIGURE 1: Abdominal ultrasound showed enlarged kidneys including multiple cysts.

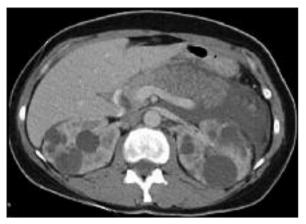


FIGURE 2: The pancreatic tail was edematous and surrounded by peripancreatic fluid neighboring the largest cyst of the left kidney in ultrasound and CT. The left peri-pararenal fluid collection may reflect a possibility of a cyst rupture located at the middle port of left kidney.

tail was edematous and surrounded by peripancreatic fluid neighboring the largest cyst of the left kidney in ultrasound and CT (Figure 2). The left peri-pararenal fluid collection may reflect a possibility of a cyst rupture located at the middle port of left kidney (Figure 2). No gallstone or choledocholithiasis was seen. Abdominal CT ruled out pancreatic calcifications or biliary tract abnormalities. Endoscopic retrograde cholangiopancreatography was normal.

The final diagnosis was acute pancreatitis and there was not any etiologic factor. Hospitalisation outcome was uneventful and there was not any complication improve. After six mounths follow up the patient remained asymptomatic and there has been no evidence of pancreatitis periodic laboratory tests and ultrasonographic examination.

DISCUSSION

ADPKD is inherited autosomal dominantly and may have a subtle clinical course. Because the disease is usually asymptomatic and has a slow progression, diagnosis delays until the early and middle adult life. Ultrasonography is the most important diagnostic imaging method and Ravine criteria are practically applied. CT and magnetic resonance imaging methods are much more useful in complicated cases and extrarenal involvement The disease may lead to chronic renal failure and

hypertension.^{2,4} Most of the cases suffer from flank or back pain. Pain is mostly related to the compression of the enlarged kidney and liver. Infection or hemorrhage of cysts and nephrolithiasis are the other causes the acute severe pain.⁵⁻⁷ In our case, pain was caused by acute pancreatitis which is a rare extrarenal complication of polycystic renal disease. Although serum amylase levels may increase in renal pathologies, it can never reach to the level of acute pancreatitis. Taken together, high serum amylase-lipase levels and radiological findings, the patient was diagnosed for acute pancreatitis.

All causes of pancreatitis were ruled out appropriately. There was no history of alcohol, drug and herbal medicine, surgery, trauma. Furthermore we ruled out hyperlipidemia and hypercalcemia, biliary stone, and structural abnormalities of the common bile duct.

The possible reason of acute pancreatitis in ADPKD patients is the obstruction of the pancreatic canal by a pancreatic cyst. Another etiologic factor is the divisium of pancreas. No cyst in the pancreatic parenchyma and any abnormality in the pancreatic duct in ERCP was observed in our patient. The rupture of a renal cyst causing a chymic reaction in the pancreatic tissue was the most plausible reason of acute pancreatitis. Irregularity and pararenal extension of a cyst which is located in upper pole of left kidney may support our argument.

In conclusion, in an ADPKD patient presenting by acute abdominal pain, acute pancreatitis should be considered in the differential diagnosis. Although pancreatic cysts and pancreatic divisium were the common causes of pancreatitis reported previously, herein we speculated rupture of renal cyst as an etiological factor in pathogenesis of pancreatitis in a patient with ADPKD.

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