## **CASE REPORT**

# Tale of a pseudocyst of pancreas with page kidney: A rare case report

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### Abstract

Page kidney, a rare clinical phenomenon, involves hypertension arising in a previously normotensive individual, typically caused by external compression of the renal parenchyma. Common etiologies include subcapsular hematomas or urinomas. We report an unusual case of a 35-year-old male with a history of chronic alcoholism who developed a pancreatic pseudocyst leading to Page kidney and secondary hypertension. This combination of conditions is exceptionally rare.

Keywords: Chronic Pancreatitis, Pseudocysts, Irvin Page

### Introduction

A true cyst is a fluid collection enclosed within an epithelial-lined capsule, whereas a pseudocyst is surrounded by fibrous and granulation tissue without epithelial lining. Pancreatic pseudocysts, is usually well circumscribed and located outside of the pancreas often located in the lesser sac, and frequently occurring as a complication of chronic pancreatitis and less commonly due to trauma [1]. Accounting for approximately 80% of benign pancreatic lesions, pseudocysts have a prevalence of 20% to 40% in chronic pancreatitis cases. Complications include infection, hemorrhage, bile duct obstruction, and gastric or duodenal stenosis [1-2]. Page kidney is a medical condition where compression of the kidney occurs from externally (usually by a hematoma, cyst, or tumor) leading to hypoperfusion and ischemia. This reduced blood flow stimulates the Renin-Angiotensin-Aldosterone System (RAAS), causing hypertension and potential kidney damage. The young patients are commonly affected by Page kidney and presents as hypertension and its complications, flank pain and

renal mass lesions. [3-4]. We present an extremely rare case of a pancreatic pseudocyst manifesting as Page kidney in a 35-year-old male. A comprehensive literature review revealed that this is the first documented case.

## Case Report

A 35-year male patient presented to surgical department with history of pain in abdomen, burning sensation in stomach, distension of abdomen in the last 20 days and vomiting for 15 days. Patient had history of similar complaints 5 years back and were relieved on taking medication. Patient was newly diagnosed to have hypertension 6 months back and on medication. Patient was a chronic alcoholic since 10 years. Pulse was 80 beats/min and BP-140/90 mmHg. On per abdomen examination, a mass measuring 12×15 cm was located in the left hypochondrium and left lumbar region, extending to the umbilicus and epigastric area. Its medial and lower borders were clearly identifiable upon palpation. The serum amylase and lipase levels were 484 IU/L (reference range:

25-125 IU/L) and 750 U/L (reference range: 40-290 U/L), respectively. The complete hemogram, liver function tests, and serum urea and creatinine were normal. Ultrasound abdomen showed bulky pancreas with irregularly dilated pancreatic duct, suggestive of chronic pancreatitis. Left kidney showed 9×8 cm hypoechoic cystic lesion noted with renal compression. Right kidney was normal in size and echogenicity.

Further Contrast-Enhanced Computed Tomography (CECT) abdomen and pelvis was done and it showed multiple calcific foci discretely throughout the pancreatic parenchyma. The main pancreatic duct also appeared dilated with few calcific foci within, largest measuring about 8.2 mm. Also noted were few cystic lesions near the tail of the pancreas, cranial to the spleen in sub-diaphragmatic location, measuring 10.6×8.2×7.6 cm (Figure 1). The CECT also showed a large cystic lesion in subcapsular region of left kidney with left renal compression. After all the radiological investigations patient underwent distal pancreatomy, wedge resection of stomach, splenectomy and nephrectomy and the resected specimen was sent for department of Pathology for histopathological examination.

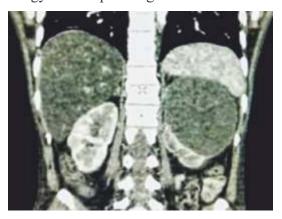


Figure 1: CECT abdomen large cystic lesion in subcapsular region of left kidney with left renal compression

On gross examination we received distal pancreatomy + wedge resection of stomach + splenectomy + nephrectomy + pseudocyst altogether measuring 27.5×23×12 cm (Figure 2). Identified part of the stomach adhered to the large cyst measuring 15×8×7 cm (Figure 3). External surface of the cyst was congested with exudate. Cut surface of cyst exuded 1000 ml of greenish fluid and cyst wall was grey white to grey brown. A thinned-out kidney was also noted, which appeared to be adhered to the large cyst (Figure 4).



Figure 2: Gross image showing pancreatomy, wedge resection of stomach, splenectomy & nephrectomy



Figure 3: Gross image showing page kidney (Black arrow)



Figure 4: Gross image showing cut section of pancreatic pseudocyst

The cut surface revealed a thinned-out cortex. Additionally, a duct measuring  $0.5 \times 0.5$  cm was identified. The spleen, measuring  $11 \times 6 \times 2.5$  cm, was found attached to the cyst. External surface and cut surface of spleen was unremarkable. A portion of the pancreas was also identified, showing calcification along with fibrosis. Microscopic examination of a section from the large cyst revealed thick fibrous tissue with lymphocytic infiltration, lacking a lining epithelium (Figure 5). Section studied from left kidney showed compressed cortex and medulla.

Few glomeruli showed mesangial cell proliferation and basement membrane thickening. Tubules were atrophied, showing thyroidisation (Figure 6). At places, interstitial fibrosis with chronic inflammatory cell infiltrates composed of lymphocytes and plasma cells were noted. Section studied from pancreas showed features of chronic pancreatitis. Section studied from attached stomach and spleen showed normal histology.

With all these features, final diagnosis of chronic pancreatitis with pancreatic pseudocyst with Page kidney having features of chronic pyelonephritis was given. Later patient was started on Angiotensin Converting Enzyme (ACE) inhibitors as a treatment for hypertension.

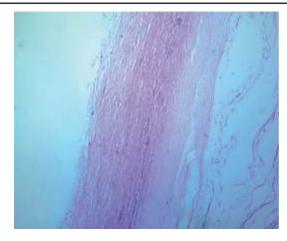


Figure 5: Microscopy of pancreatic pseudocyst showing fibrous wall (H&E stain 400X)

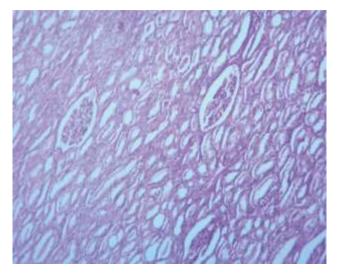


Figure 6: Microscopy of page kidney showing features of chronic pyelonephritis (H&E stain 400X)

## Discussion

The pseudocyst formation is attributed to the destruction of the main pancreatic duct or peripheral ductules, leading to leakage of pancreatic secretions. This leakage results in the activation and release of pancreatic enzymes, which in turn cause localized autodigestion and necrosis of the pancreatic parenchyma. The ensuing tissue damage

triggers an inflammatory response, culminating in the development of a distinct pseudocyst wall. This wall is composed of granulation tissue and blood vessels, which subsequently organize with connective tissue and fibrosis [5]. Regardless of the underlying cause, the overall incidence of pseudocysts remains low, at approximately 0.5 to 1 per 100,000 adults per year. Pseudocysts can develop in individuals of any age following an episode of pancreatitis. The incidence is slightly higher in males, reflecting the male predominance of pancreatitis. In line with this trend, our case also involves a male patient [5]. Pseudocysts are more commonly associated with chronic pancreatitis, with an incidence of 20% to 40%. In contrast, their incidence in acute pancreatitis is lower, ranging from 5% to 16%. Symptoms are often nonspecific and may include nausea, vomiting, or vague abdominal pain, as observed in our case [5].

Complications of pseudocysts include rupture, hemorrhage, infection, and disruption of the pancreatic duct system. While pseudocysts most commonly occur around the pancreas, they can rarely extend into surrounding structures such as the liver, transverse colon, spleen, retroperitoneum, anterior or posterior pararenal space, and even the mediastinum. In our case, the cyst extended to involve the spleen and part of the stomach while compressing the left kidney, resulting in Page kidney and subsequent hypertension [6]. The phenomenon of Page kidney was first described by Irvin Page in 1939. His groundbreaking experiments involved wrapping canine kidneys in cellophane, inducing hypertension and a perinephric inflammatory response in the test subjects [7]. The hypertension was attributed to compression caused by a subcapsular hematoma. Subcapsular hematomas most commonly occur as a complication of renal biopsy but may also result from sports injuries or motor vehicle trauma. Other less common causes include polyarteritis nodosa, warfarin therapy, and extracorporeal shockwave lithotripsy. Following a thorough literature review, our case appears to be the first reported instance of Page kidney caused by a pancreatic pseudocyst complication. In this phenomenon, renal compression leads to hypoperfusion and microvascular ischemia, triggering an increase in renin levels. This activates the RAAS, resulting in hypertension [5-6]. The diagnosis of pancreatic pseudocysts and Page kidney is primarily achieved through imaging techniques, with CECT being the diagnostic modality of choice.

Pancreatic pseudocysts vary in size, ranging from 2 cm to 30 cm. Pseudocysts larger than 10 cm are termed giant pseudocysts, which are extremely rare [5]. In the present case, the pseudocyst measured 15 cm. Grossly, a Page kidney appears flattened with a thinned-out cortex, while microscopically, a pseudocyst is lined by fibrous tissue without epithelial lining, and a Page kidney exhibits features of chronic pyelonephritis. The fate of pancreatic pseudocysts often involves spontaneous resolution, particularly in cases of acute pancreatitis. Stable, non-enlarging pseudocysts rarely cause symptoms, making conservative management the gold standard for uncomplicated cases. However, pseudocysts arising from chronic pancreatitis are less likely to resolve spontaneously. Cysts exceeding 6 cm in size typically require surgical management, as in our case. For page kidney, treatment involves surgical intervention followed by the use of ACE inhibitors to normalize blood pressure [6, 8].

## Conclusion

Pancreatic pseudocysts are very dangerous and as

a complication leads to Page kidney due to external compression of the kidney. Hence physicians and surgeons should be vigilant of rare complication that can occur, so that early

diagnosis can be made and timely intervention can be given so that it decreases the mortality and prevent development of hypertensive crisis and also helps to improve quality of life.

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