

## CASE REPORT

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# Solitary Main Pancreatic Ductal Calculus of Possible Biliary Origin Causing Acute Pancreatitis

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### ABSTRACT

**Context** Pancreatic ductal calculi are most often associated with chronic pancreatitis. Radiological features of chronic pancreatitis are readily evident in the presence of these calculi. However, acute pancreatitis due to a solitary main pancreatic ductal calculus of biliary origin is rare.

**Case report** A 59-year-old man presented with a first episode of acute pancreatitis. Contrast enhanced computerized tomography (CT) scan and endoscopic retrograde cholangiopancreatography (ERCP) revealed a calculus in the main pancreatic duct in the head of the pancreas causing acute pancreatitis. There were no features suggestive of chronic pancreatitis on CT scanning. The episode acute pancreatitis was managed conservatively. ERCP extraction of the calculus failed as the stone was impacted in the main pancreatic duct resulting in severe acute pancreatitis. Once this resolved, a transduodenal exploration and extraction of the pancreatic ductal calculus was performed successfully. Crystallographic analysis revealed the composition of the calculus was different to that seen in chronic pancreatitis, but more in keeping with a calculus of biliary origin. This could be explained by migration of the biliary calculus via the common channel into the main pancreatic duct.

Following the operation the patient made an uneventful recovery and was well at two-year follow up.

**Conclusion** Acute pancreatitis due to a solitary main pancreatic ductal calculus of biliary origin is rare. Failing endoscopic extraction, transduodenal exploration and extraction is a safe option after resolution of acute pancreatitis.

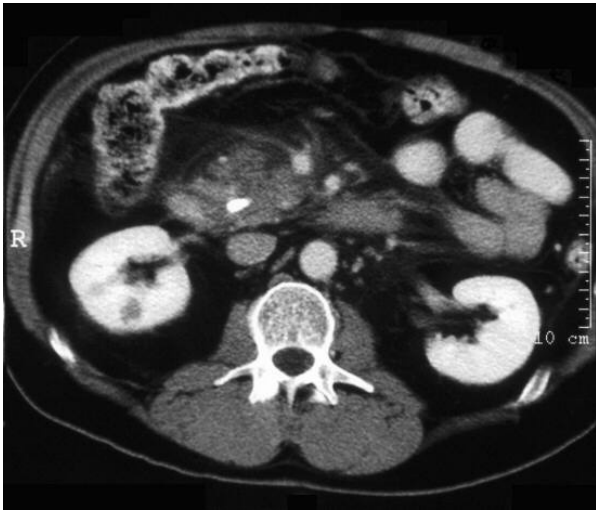
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### INTRODUCTION

Pancreatic calculi are a feature of chronic pancreatitis (CP). The most common cause for CP in the UK is alcohol. Other causes of CP are tropical, hereditary or idiopathic. The prevalence of calculi cannot be separated from the prevalence of the etiological factors, the most common being alcohol [1, 2, 3, 4, 5, 6].

Sarles suggested that all forms of CP are calculous disease irrespective of radiological studies showing presence or absence of calculi [2]. It is generally believed that pancreatic calculi visible on radiography usually occur in the late stages of chronic pancreatitis [6]. More recently, abdominal CT scanning has revealed a comparatively larger number of intraductal calculi [6].

We wish to report a case of a solitary main pancreatic duct (MPD) calculus of possible



**Figure 1.** CT scan showing normally enhanced pancreas with no features of chronic pancreatitis.

biliary origin causing acute pancreatitis with no radiological features of CP in the remainder of the gland.

## CASE REPORT

A 59-year-old Caucasian man presented with acute onset upper abdominal pain radiating through to the back, associated with nausea but no vomiting, following a large meal. There was no history of alcohol intake. There were no similar episodes of pain in the past. The patient had a long standing history of musculoskeletal back pain in the lower thoracic area and had undergone facet joint injections for pain relief. On examination the patient was not jaundiced and was tender in the epigastric region. The rest of the examination was unremarkable.

Blood tests revealed an elevated amylase serum level (greater than 1,000 IU/L; reference range 0-110 IU/L), a leukocytosis ( $23.7 \times 10^9/L$ ; reference range  $4.0-11.0 \times 10^9/L$ ), hyperbilirubinemia ( $41 \mu\text{mol/L}$ ; reference range  $5-21 \mu\text{mol/L}$ ) and a normal alkaline phosphatase. Abdominal ultrasound scan demonstrated changes of acute pancreatitis around the pancreatic head, with no gallstones and normal caliber bile ducts.

Contrast enhanced abdominal CT scan demonstrated an edematous and inflamed pancreatic head, as well as an impacted stone at the ampulla within the distal bile duct,

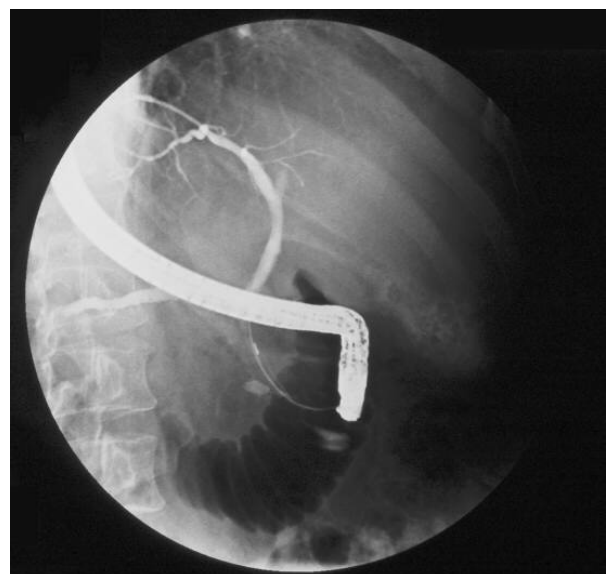
causing obstruction. The remainder of the pancreas enhanced normally with no associated features of CP (Figure 1). As the scan suggested an impacted stone in the distal bile duct at the ampulla, an ERCP was performed.

Initial ERCP suggested a radio-opaque calculus within the MPD in the head of the pancreas, with no abnormality of the biliary tree (Figure 2). Therefore, pancreatic duct sphincterotomy was undertaken. Attempted endoscopic extraction of the MPD calculus was unsuccessful.

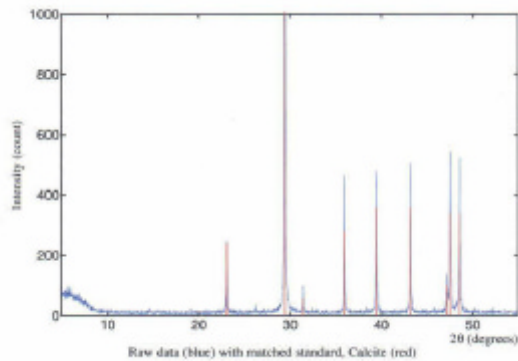
Following ERCP the patient had a further attack of upper abdominal pain with an increase in serum inflammatory markers. Abdominal CT scan revealed severe acute pancreatitis associated with sterile necrosis within the head and body of the pancreas. The patient was managed conservatively with supportive therapy and nasojejunal feeding.

Following resolution of the acute episode 8 weeks after initial presentation, the patient underwent a transduodenal exploration of the pancreatic duct with extraction of calculus ( $1 \times 0.75 \text{ cm}$ ), pancreatic and biliary sphincteroplasty and a cholecystectomy.

Crystallographic analysis of the calculus revealed the composition to be pure calcite with no amorphous or proteinaceous material, which was not in keeping with typical



**Figure 2.** ERCP: radio-opaque calculus in the head of the pancreas and not in the CBD.



**Figure 3.** Crystallographic analysis of the calculus composed of pure calcite with no amorphous or proteinaceous material.

pancreatic calculi (Figure 3). The patient made an uneventful recovery and has been asymptomatic with no recurrence of pain or pancreatitis for the past two years.

## DISCUSSION

Pancreatic concretions have been a subject of investigation since the 17<sup>th</sup> century. Since they were described in 1667 by DeGraaf, several workers have elaborated on their clinical, biochemical, biophysical and behavioral characteristics [6, 7].

Previously, two primary patterns of calcification were believed to exist: an intraductal pattern, representing true stones and a parenchymal calcific pattern, representing “false stones” or calcifications [6, 8, 9]. The current consensus is that the only mode of calcification in the excretory portion of the pancreas is the formation of intraductal calculi [10]. In the industrialized nations of the West, alcoholism stands out as the cause for chronic calcific pancreatic disease [6]. Tropical pancreatitis is one of the most common causes of CP in the developing world [11].

In the present case, CT scans had suggested a stone impacted at the ampulla, however this was disproved at ERCP which revealed a 1 cm calculus in the MPD within the pancreatic head. This was solitary with no calculi or strictures in the remainder of the pancreatic duct and no calcification within the parenchyma. Despite this large solitary

calculus the patient had no previous episodes of pancreatitis and no history of alcohol abuse. He presented with a first episode of acute pancreatitis, with no radiological features of chronic pancreatitis.

A clue to the diagnosis of pancreatic calculi in CP by ERCP is the presence of radiolucent areas in the dilated MPD. The circular translucent areas denote protein plugs or precalcified stones [6]. Contrary to this, our patient had a radio-opaque shadow in the head of the pancreas on CT and ERCP. The lack of amorphous material as revealed in the chemical analysis of the calculus from this patient may explain the radiological findings.

Studies have shown that stones in the pancreas are formed primarily from calcium carbonate, proteins and polysaccharides. They may be single or multiple existing as small concretions or well developed calculi 1 to 2 cm in diameter. A sliced section of a pancreatic stone usually shows single or more often multiple cavities, occupied by proteinaceous material in vivo. The core is composed of a very fine network of fibres (amorphous substance). The outer shell shows a wavy spiral pattern with small, tiny particles scattered around. These particles represent immature calcite crystals. The elemental composition of calculi in patients from different geographic regions appears to remain the same [6].

Crystallographic analysis of the pancreatic calculus in our case revealed no amorphous material in the sample although it did show calcite. This composition is distinct when compared to the well known two layered structure of pancreatic calculi seen in chronic pancreatitis, thus suggesting a possible different mechanism of lithogenesis. We propose that the calculus may have originated in the gallbladder and possibly migrated into the MPD through the common channel. We believe the calculus migrated at an earlier date via the common channel and lodged in the MPD, over time attaining the size of 1 cm. Crystallographic analysis of the calculus supports this hypothesis. Arguably, the point against this hypothesis would be the absence

of calculi in the gallbladder. However, solitary biliary ductal calculi in the absence of multiple gallstones have been described [12]. Endoscopic extraction, with or without extracorporeal shock wave lithotripsy (ESWL), is the preferred method of extraction of MPD calculi [13]. However, failing this, transduodenal exploration and calculus extraction from the MPD is a safe procedure.

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Received June 22<sup>nd</sup>, 2005 - Accepted July 1<sup>st</sup>, 2005

**Keywords** Lithiasis; Pancreatic Ducts; Pancreatitis

**Abbreviations** CP: chronic pancreatitis; MPD: main pancreatic duct

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**References**

1. Edmondson HA, Bullock WK, Mehl JW. Chronic pancreatitis and lithiasis; pathology and pathogenesis of pancreatic lithiasis. *Am J Pathol* 1950; 26:37-55. [PMID 15399220]

2. Sarles H. Chronic calcifying pancreatitis: chronic alcoholic pancreatitis. *Gastroenterology* 1974; 66:604-16. [PMID 4595185]
3. Pitchumoni CS. Special problems of tropical pancreatitis. *Clin Gastroenterol* 1984; 13:941-59. [PMID 6386245]
4. Sarles H, Muratore R, Sarles JC. Etude anatomique des pancreatites chroniques de l'adulte. [Anatomical study of chronic pancreatitis of the adult]. *Sem Hop* 1961; 37:1507-22. [PMID 13746538]
5. Comfort MW, Steinberg AG. Pedigree of a family with hereditary chronic relapsing pancreatitis. *Gastroenterology* 1952; 21:54-63. [PMID 14926813]
6. Pitchumoni CS, Mohan AT. Pancreatic stones. *Gastroenterol Clin North Am* 1990; 19:873-93. [PMID 2269523]
7. Rockwern SS, Snively D (citing DeGraaf). Pancreatic lithiasis associated with pancreatic insufficiency and diabetes mellitus. *Arch Intern Med* 1940; 65:873-5.
8. Lagergren C. Calcium carbonate precipitation in the pancreas, gallstones and urinary calculi. *Acta Chir Scand* 1962; 124:320-5. [PMID 13928053]
9. Mayo JG. Pancreatic calculi. *Mayo Clin Proc* 1936; 11:456-7.
10. Sarles H, Sahel J. Pathology of chronic calcifying pancreatitis. *Am J Gastroenterol* 1976; 66:117-39. [PMID 788498]
11. Geevarghese PJ. Calcific Pancreatitis: Causes and Mechanisms in the Tropics Compared with Those in Subtropics. Trivandrum (Thiruvananthapuram), India: St Joseph's Press, 1986: 67-75.
12. Schumann C, Notzold K. Der Solitarstein der Gallenblase: ein harmloser Befund? [The solitary biliary calculus: a harmless condition?]. *Z Arztl Fortbild (Jena)* 1981; 75:1043-5. [PMID 7340186]
13. Delhaye M, Matos C, Deviere J. Endoscopic technique for the management of pancreatitis and its complications. *Best Pract Res Clin Gastroenterol* 2004; 18:155-81. [PMID 15123090]