

## CASE REPORT

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# Ansa Pancreatica Type of Ductal Anatomy in a Patient with Idiopathic Acute Pancreatitis

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### ABSTRACT

**Context** Ansa pancreatica is a type of pancreatic ductal variation. The exact clinical significance of this ductal variation is not clear.

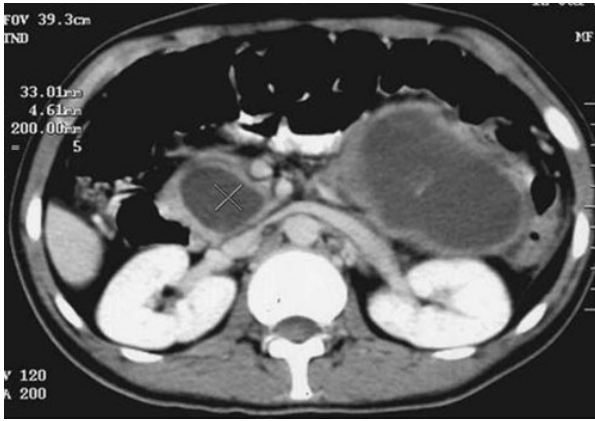
**Case report** We report the case of a 21-year-old male with acute idiopathic severe pancreatitis and extensive parenchymal necrosis who later developed a large pancreatic abscess. Subsequently, transpapillary drainage of the pancreatic abscess was attempted and on endoscopic retrograde pancreatography, disruption in the mid-body of the pancreas and the ansa pancreatica type of ductal anatomy was noted. A 7 Fr nasopancreatic catheter was placed across the disruption. However, due to the development of a new abscess, surgical drainage was performed. The patient has since been asymptomatic over a one-year follow up period.

**Conclusion** A pancreatic ductal variation such as ansa pancreatica may be a finding in severe acute pancreatitis; it is not clear if the presence of these two conditions is coincidental or if ansa pancreatica causes acute pancreatitis. Further studies are needed to clarify these points.

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### INTRODUCTION

A number of congenital anomalies and variants of the pancreas have been described; the majority of them are of no clinical significance and are detected incidentally at endoscopy, surgery or autopsy [1]. Pancreas divisum is the most common congenital pancreatic anatomic variant and its increased prevalence has been noted in patients with idiopathic pancreatitis [1, 2]. Most of the other variants are usually of no clinical significance. Ansa pancreatica is a rare type of ductal anatomical variation described in the literature, but its clinical significance is not clear [1]. The association of ansa pancreatica and acute pancreatitis is speculative. It has been reported that patients with the ansa pancreatica type of ductal variation, especially alcoholics, are vulnerable to pancreatitis [1, 3, 4, 5]. No reports of ansa pancreatica with idiopathic pancreatitis leading to pancreatic abscess have been reported in the literature. We report the case of a 21-year-old male with acute idiopathic severe pancreatitis and a pancreatic abscess which, on endoscopic retrograde cholangio-pancreatography (ERCP), revealed the ansa pancreatica type of pancreatic ductal anatomy.



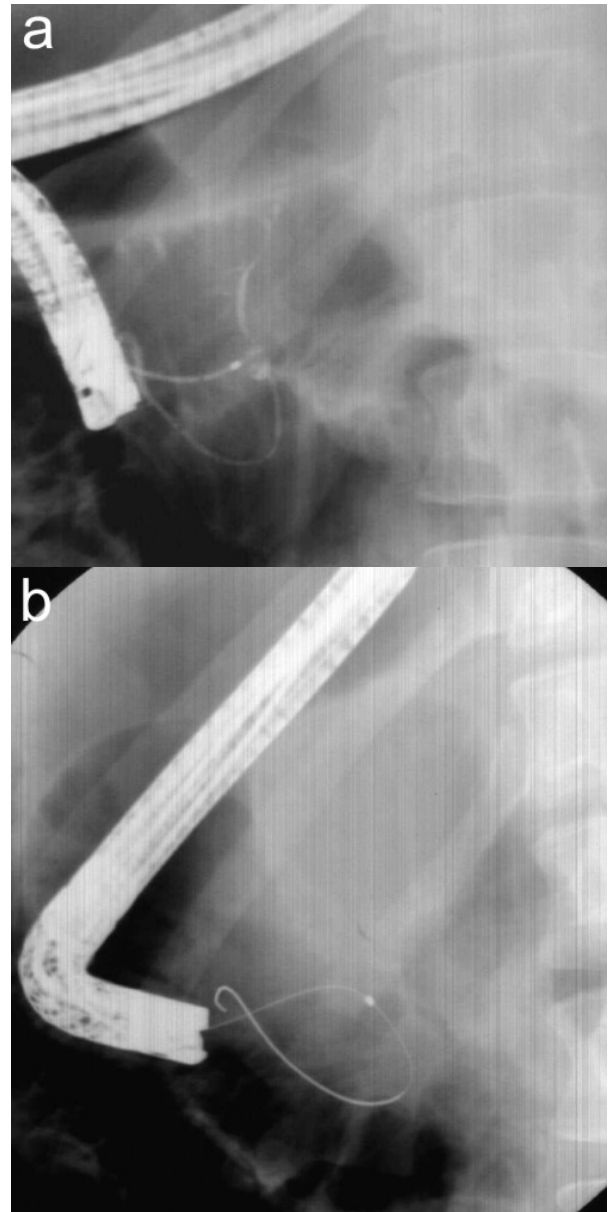
**Figure 1.** Contrast-enhanced computed tomography (CECT) of the abdomen showing two hypodense collections with a thick enhancing wall (pancreatic abscess).

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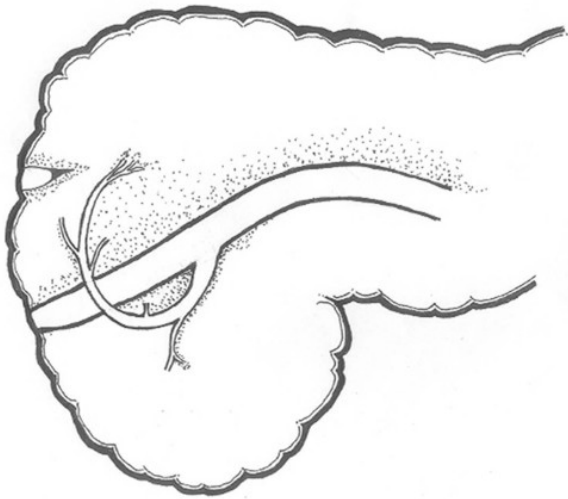
A 21-year-old male presented with severe pain in the epigastrium which radiated to the back and was associated with vomiting and abdominal distension. There was no history of alcohol ingestion or abdominal trauma. On examination, the abdomen was tender and bowel sounds were absent. Hemoglobin, total leukocyte count, liver function tests, renal function tests and serum electrolytes were normal. At admission, the serum amylase level was 640 IU/L (reference range: 0-160 IU/L). Serum calcium and triglycerides were within normal limits. An ultrasonogram (US) of the abdomen did not reveal gallbladder stones or sludge. Contrast-enhanced computerized tomography (CECT) of the abdomen revealed features of severe pancreatitis with more than 50% necrosis. The patient was hospitalized and managed with intravenous fluids and antibiotics. The patient showed gradual improvement and was discharged after 3 weeks.

Two months later, the patient presented with fever and pain in the abdomen. CECT of the abdomen revealed two hypodense collections with a thick enhancing wall (11x10 cm in size, in relation to the body-tail region of the pancreas and 5x4 cm in size, in relation to the head-body region of the pancreas) (Figure 1). US-guided needle aspirate from the larger collection yielded frank pus which grew *E. coli* on culture. Intravenous antibiotics were

started as per the sensitivity pattern and endoscopic transpapillary drainage of the pancreatic abscess was attempted. Informed consent was obtained and ERCP was performed. The major papilla was cannulated and the pancreatogram revealed a disruption in the pancreatic duct at the level of the body of the pancreas; a side branch arising from the main pancreatic duct and forming a loop to terminate near the minor papilla, was also noted (Figure 2a, 2b). This ductal variation corresponds to the ansa pancreatica type of



**Fig 2. a.** Endoscopic retrograde pancreatography showing a looping branch descending and then ascending upward towards the minor papilla. **b.** Guide wire into the looping branch.



**Figure 3.** Image depicting the ansa pancreatica type of ductal variation. Note that the looping branch terminates at or near the minor papilla.

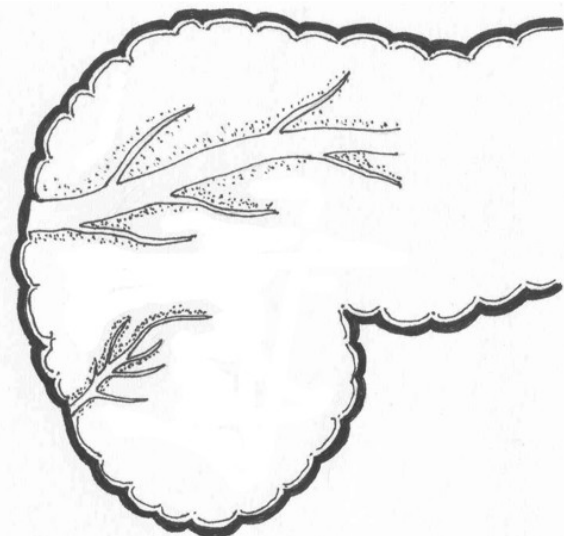
ductal anatomy (Figure 3). A 7 Fr naso-pancreatic catheter was placed across the disruption and antibiotics were continued. Following this, the abdominal pain as well as the fever subsided. Serial ultrasonograms revealed a progressive decrease in the size of both collections. Repeat CECT of the abdomen at 6 weeks revealed complete resolution of the smaller collection and the other decreasing to 7x7 cm in size. However, a new collection (5x3 cm) was noted in the pelvis. Subsequently, surgical drainage of the residual abscesses (abdominal and pelvic) was performed. The patient had an uneventful postoperative course and has been asymptomatic during a one-year follow up period.

## DISCUSSION

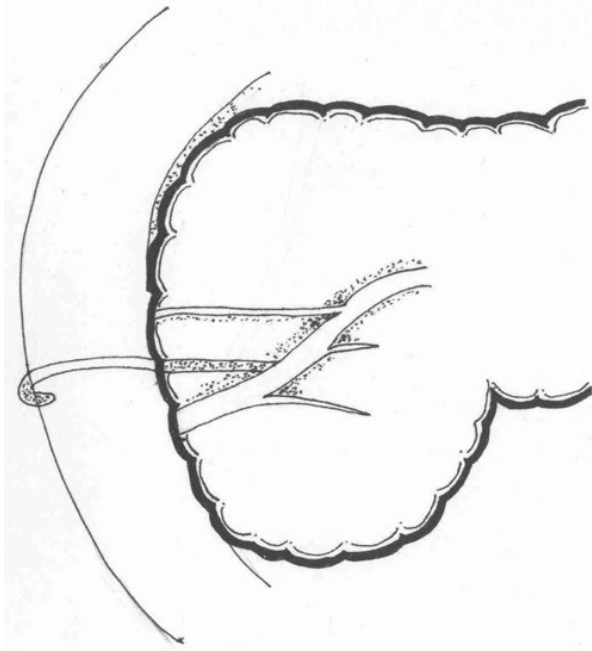
The pancreatic ductal system consists of the dorsal duct (or duct of Santorini) and the ventral duct (or duct of Wirsung). These two ducts commonly fuse in the head region with the majority of the drainage occurring through the major papilla via the duct of Wirsung [6]. The dorsal duct (duct of Santorini), usually plays a small role in the pancreatic drainage of normal individuals and if patent, it empties via the smaller accessory (minor) papilla. The embryological dorsal and ventral pancreatic buds come together medially as a result of

asymmetric rotation of the developing duodenum and, thus, the ventral pancreatic bud and the bile duct rotate 180° counterclockwise around the foregut arriving caudally to the dorsal pancreatic bud. The ventral and dorsal ductal systems then fuse during 6 to 8 weeks of gestation resulting in normal pancreatic duct anatomy at birth. However, as a result of abnormal ductal fusion during embryological development, a number of congenital ductal variations of the pancreatic ductal system such as complete or incomplete pancreas divisum, reverse pancreas divisum, functional pancreas divisum, annular pancreas, anomalous pancreaticobiliary junction and ansa pancreatica have been described [1,7].

Pancreas divisum, which results from malfusion of the pancreatic ductal systems, is the most common congenital variation of the pancreas and is seen in 5-8% of the population (Figure 4) [1, 7, 8]. As a result of this divided ductal system, the greater part of the pancreatic drainage occurs via the smaller orifice of the minor papilla through the dorsal duct of Santorini. The clinical significance of pancreas divisum is still being debated, as the majority of patients with this pathology have no pancreatic symptoms throughout their life [9, 10]. However, an increased frequency of pancreas divisum has been noted in patients with idiopathic acute recurrent and chronic



**Figure 4.** Image depicting complete pancreas divisum.



**Figure 5.** Image depicting the annular pancreas type of ductal anomaly. Note that the looping branch crosses the duodenum.

pancreatitis, and the data available suggests that patients with pancreas divisum are at increased risk of obstructive pancreatitis [1, 11, 12]. The proposed mechanism for pancreas divisum causing pancreatitis is that the flow of the pancreatic juice is obstructed at the level of an inadequately patent or stenosed minor papilla, resulting in ductal hypertension and consequent pancreatitis [13]. This is further supported by the fact that endoscopic or surgical decompression of the dorsal duct leads to a decrease in the rate of recurrent pancreatitis in patients having idiopathic acute recurrent pancreatitis with pancreas divisum and a decrease in pain in patients having chronic pancreatitis with pancreas divisum [14, 15, 16, 17, 18, 19, 20, 21].

During the course of embryological development, the accessory pancreatic duct or the duct of Santorini undergoes a variable degree of atrophy at its duodenal end. This variable degree of obliteration results in varying patency rates of the accessory duct where it enters the minor papilla, and also a variable course and shape of the accessory pancreatic duct. Ansa pancreatica is a type of ductal anatomy which was named by Dawson

and Langman [22] and is characterized by obliteration of the accessory pancreatic duct at its junction with the ventral pancreatic duct, and replacement of this duct by an additional arched communication between the dorsal and ventral duct systems. This arched duct is formed by the combination of the proximal duct of the dorsal pancreatic bud, the inferior branch of the duct of the dorsal pancreatic bud and the inferior branch of the duct of the ventral pancreatic bud. Thus, in the ansa pancreatica type of ductal variation, the accessory pancreatic duct arises from the main pancreatic duct and loops, running an arched course into the caudal portion of the pancreas, turning anterior to the main duct to terminate in or around the minor papilla (Figure 2) [1, 4, 5, 22, 23, 24].

At ERCP, the looping branch of ansa pancreatica may resemble the annular pancreas type of ductal variation. However, upon careful analysis of the pancreatogram, the two ductal variations can be easily differentiated. In contrast to the ansa pancreatica, the looping branch in the annular pancreas will course around the descending duodenum and therefore, at ERCP, it will be seen crossing the duodenum or side viewing endoscope if the scope is in situ, to the right of the papillary orifice (Figure 5). In ansa pancreatica, the looping branch courses towards the minor papilla and will not cross the duodenum or side viewing endoscope if the scope is in situ at ERCP as illustrated in the case presented (Figures 2 and 3).

It has been proposed that ansa pancreatica predisposes a patient to pancreatitis, although this is considered controversial [1, 3, 4, 5]. In contrast to the other tributaries of the main pancreatic duct which join the main pancreatic duct at right angles, the ansa joins the main duct at an oblique angle. Because of this ductal anatomy, it has been proposed that the areas served by the ansa have poor drainage of pancreatic juice. In conditions such as alcoholism or functional stenosis of the sphincter of Oddi, the drainage of pancreatic juice is further impaired, and such patients are vulnerable to the development of pancreatitis [4].

Kamisawa *et al.* [25] studied 180 patients with dye injection ERCP and found that the patency of the accessory pancreatic duct into the minor papilla was significantly lower in patients with acute pancreatitis (6%) as compared to healthy controls (41%). The authors thus postulated that a patent accessory pancreatic duct lowers the pressure in the main pancreatic duct by draining through the minor papilla and might therefore prevent acute pancreatitis. Kamisawa *et al.* [24] have reported the patency of the accessory pancreatic duct to be 20.7% in the ansa type of accessory pancreatic ductal variation. Dawson and Langman [22] have also reported patency rates of 33.3% (7/21) for ansa pancreatica type of ductal variation. They proposed that ansa pancreatica is formed post-embryologically at some stage during life to compensate for the absence of the duct of Santorini. However, as a consequence of low patency rates, the authors suggested that ansa pancreatica is non functional and, hence, cannot substitute the accessory duct in most of the patients with ansa. Whether these low patency rates in the ansa pancreatica type predispose to acute pancreatitis is speculative and needs to be confirmed by further studies. The patient in this report had idiopathic acute severe pancreatitis and the ansa pancreatica type of pancreatic ductal variation. The association between ansa pancreatica and pancreatitis is speculative and the presence of these two conditions in the report presented may be co-incidental or could be the result of a cause and effect relationship. More study is required in order to provide a definite answer to this hypothesized association. In conclusion, we have described the ansa pancreatica type of pancreatic ductal variation in a patient with severe idiopathic acute pancreatitis although the presence of these two conditions in the patient presented may be co-incidental or could be the result of a cause and effect relationship. However, further studies are needed to study this.

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**Abbreviations** CECT: Contrast-enhanced computerized tomography

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