Ansa Pancreatica, an Uncommon Cause of Acute, Recurrent Pancreatitis

Danial H. Shaikh\textsuperscript{a, b}, Ahmed Alemam\textsuperscript{a, b}, Jennifer von Ende\textsuperscript{c}, Haider Ghazanfar\textsuperscript{a}, Anil Dev\textsuperscript{a, b}, Bhavna Balar\textsuperscript{a, b}

\textsuperscript{a}Division of Gastroenterology, BronxCare Health System, Icahn School of Medicine, Bronx, NY, USA; \textsuperscript{b}Department of Medicine, BronxCare Health System, Icahn School of Medicine, Bronx, NY, USA; \textsuperscript{c}School of Medicine, American University of the Caribbean, Cupecoy, Saint Martin

Keywords
ANSA pancreatica · Acute pancreatitis · Recurrent pancreatitis · Endoscopic ultrasound

Abstract
The pancreatic duct is vulnerable to developmental anomalies which may produce variations in its course and/or its configuration. Ansa pancreatica is the least common anatomic variant. It is characterized by the formation of an “S-shaped loop” from the main pancreatic duct to the minor papilla. Ansa pancreatica has been implicated as a cause of recurrent acute pancreatitis. We review existing literature on pancreatitis secondary to the ansa deformity and present a case of recurrent acute pancreatitis in a patient who was ultimately found to have the ansa deformity on endoscopic ultrasound.

Introduction
The pancreatic duct is vulnerable to developmental anomalies which may lead to variations in its course resulting in a descending, sigmoid, vertical, or a loop-shaped path. Developmental irregularities may also alter its configuration leading to a bifid duct with either dominant drainage from the duct of Wirsung or the duct of Santorini, an absent duct of Santorini, pancreas divisum, or ansa pancreatica [1].

Ansa pancreatica, first described by Dawson and Langman [2], is the least common anatomic variant. The word ansa translates to “handle” in Latin. It is characterized by the obliteration of the accessory pancreatic duct at the proximal end near its junction with the main pancreatic duct, and the replacement of this portion by a branch of the main pancreatic duct that forms an “S-shaped loop” between the 2 ducts, initially descending and then ascending to eventually drain into the minor papilla (Fig. 1) [3, 4].
Ansa pancreatica has been described as 2 distinct subtypes by some authors [3, 5]. These include the classic "S shaped loop" (described above) and a second form that describes a looping of the distal part of the duct of Wirsung that joins a patent duct of Santorini [6]. However, other authors have refuted this claim and include the latter type in a group of disorders classified as meandering main pancreatic duct [7].

True prevalence of ansa pancreatica is not known. A study with over 3,000 subjects who underwent an endoscopic retrograde cholangiopancreatography (ERCP) in Tokyo, Japan, reported the prevalence of 0.5% [3]. A separate study from Turkey showed that only 1.2% of the 1,158 patients who underwent magnetic resonance cholangiopancreatography (MRCP) had evidence of ansa pancreatica [8]. Another Japanese study by Hayashi et al. [9] included 587 patients who also underwent MRCP, and 0.85% were found to have ansa pancreatica.

Recently, ansa pancreatica has been implicated as a cause of recurrent acute pancreatitis [5, 9–11]. We present a case of recurrent acute pancreatitis in a patient who was ultimately found to have the ansa deformity on endoscopic ultrasound.

**Case Presentation**

A 49-year-old woman presented to our emergency department with complaints of nausea, vomiting, and abdominal pain for the past 3 days. The abdominal pain was in epigastric area, sharp in character, and radiating to the back. Pain was associated with multiple episodes of vomiting and it worsened with eating. Patient took metoclopramide and ondansetron the nausea and vomiting without much relief of symptoms. She denied any fever, chills, shortness of breath, early satiety, unintentional weight loss, or change in bowel habits. Her medical history included hypertension, diabetes mellitus, migraine headaches, chronic kidney disease, depression, and anxiety. She was admitted at our hospital four times in past with acute pancreatitis. She denied any previous surgical history. Her family history was unremarkable and she denied any alcohol, smoking, or illicit drug use. Her daily medications included insulin, esomeprazole, aspirin, atorvastatin, nifedipine, labetalol, buspirone, and sertraline.

Upon arrival to the emergency department, her vital signs were stable. Physical examination was remarkable for tenderness in the epigastric area; however, the abdomen was nondistended and had no signs of peritonitis. Initial laboratory work-up revealed a lipase of 794 IU/L and the rest of the work-up is as summarized in Table 1. An ultrasound of the abdomen showed a normal gallbladder with no gallstones and normal bile ducts. The patient was diagnosed with acute pancreatitis since she fulfilled 2 of the 3 Atlanta criteria. She was treated with intravenous fluids and analgesics. A magnetic resonant cholangiopancreatography...
| Investigation            | Value                      |
|--------------------------|---------------------------|
| White blood cell         | 12.3 (4.8–10.8 K/µL)      |
| Hemoglobin               | 12.5 (12.0–16.0 g/dL)     |
| Hematocrit               | 37.6 (42.0–51.0%)         |
| Platelet                 | 310 (150–440 K/µL)        |
| Sodium                   | 132 (135–145 mEq/L)       |
| Potassium                | 2.9 (3.5–50 mEq/L)        |
| Bicarbonate              | 35 (24–30 mEq/L)          |
| Glucose                  | 354 (70–120 mg/dL)        |
| Blood urea nitrogen      | 29 (70–120 mg/dL)         |
| Creatinine               | 2.3 (8–26 mg/dL)          |
| Calcium                  | 8.5 (0.5–1.5 mg/dL)       |
| Albumin                  | 3.5 (3.2–4.4 g/dL)        |
| Bilirubin, total         | 0.3 (0.2–1.2 mg/dL)       |
| Alkaline phosphatase     | 94 (42–98 U/L)            |
| Aspartate transaminase   | 36 (9–36 U/L)             |
| Alanine aminotransferase | 11 (5–40 U/L)             |
| Total protein            | 6.5 (6.0–8.5 g/dL)        |
| Cholesterol              | 252 (162–240 mg/dL)       |
| Triglyceride             | 200 (25–150 mg/dL)        |
| High density lipoprotein | 68 (34–82 mg/dL)          |
| Low density lipoprotein  | 144 (≤160 mg/dL)          |
| Ethanol                  | <10 (≤10 mg/dL)           |
| Lipase                   | 794 (≤61 U/L)             |
| Immunoglobulin G subclass 4 | 47.7 (4.0–86.0 mg/dL)   |

Table 1. Initial laboratory values on presentation

(MRCP) showed no biliary obstruction or common ductal stones. The pancreas appeared intact, and the pancreatic duct was tortuous but nondilated (Fig. 2).

Due to the recurrent nature of her condition, the decision was made to perform an endoscopic ultrasound (EUS) to better evaluate the pancreas and biliary tree. EUS findings were significant for an ansa deformity of the pancreatic duct. No focal pancreatic lesion was identified (Fig. 3). Her clinical condition improved over the following days. Treatment options, including a minor papilla sphincterotomy were discussed with the patient, who opted for a conservative approach, and she was eventually discharged with an outpatient gastroenterology follow-up appointment.

**Discussion**

Acute pancreatitis secondary to congenital anomalies of the pancreas, such as annular pancreas and pancreas divisum, is well known [7]. However, the association between ansa pancreatica and pancreatitis is less concrete. Hayashi et al. [9] demonstrated that compared with a control group, patients with recurrent acute pancreatitis had a significantly higher frequency of ansa pancreatica (5/587 [0.85%] vs. 2/18 [11.1%], \( p = 0.016 \)). It has been proposed that oblique angle of ansa pancreatica origination from the main pancreatic duct
causes anatomical obstruction of the flow of pancreatic secretions [12]. This loop can obstruct the normal outflow of pancreatic enzymes, causing them to backup, become activated and digest the pancreatic tissue, causing acute pancreatitis. This obstruction can lead to increased ductal pressure which can lead to recurrent attacks of acute pancreatitis [4]. Studies have also described a nonpatent opening of the ansa loop at its termination in the minor papilla [2, 6, 13].

During our search of existing literature on ansa pancreatica and pancreatitis, indexed in PubMed Central, we found 14 such cases (Table 2). The mean age of the patient was 50 years, 8 were males, 4 were females, and 2 reports did not disclose the patient’s gender. Of the 14 cases, 7 (50%) had recurrent pancreatitis, 4 had an initial episode of pancreatitis, 1 had obstructive jaundice, 1 was asymptomatic, and 1 had no information. Five case reports mentioned the number of prior episodes of pancreatitis with an average of 4 episodes; however, it is worth noting that one case had 15 prior episodes. The mean lipase level on presentation was 2,586. Nine of the 14 patients were diagnosed with ansa pancreatica at MRCP, four at ERCP. Only 1 patient had concomitant gallstones present, and 2 had coexisting intraductal papillary mucinous neoplasm. Three cases were managed with sphincterotomy alone, 2 were managed with stent placement alone, and 3 were managed with both stent placement and sphincterotomy. Of the remaining, 3 were managed conservatively and only 1 eventually underwent surgery.

To our knowledge, this is the first case of ansa deformity diagnosed with EUS. As EUS is associated with less complications than an ERCP, it should be considered in the work-up of patients with recurrent pancreatitis and a negative MRCP. Our patient was managed conservatively prior to discharge at her last hospitalization. Ansa pancreatica can be diagnosed with EUS, MRCP, or ERCP. It has been shown that MRCP can underdiagnose ansa pancreatica as it
| Study reference | Age, years | Gender | Recurrent pancreatitis | Prior episode | Serum lipase, IU/L | Other findings on imaging | Diagnostic modality | Treatment |
|-----------------|------------|--------|------------------------|---------------|-------------------|--------------------------|-------------------|-----------|
| [4]             | 53         | –      | –                      | –             | –                 | –                        | ERCP              | –         |
| [5]             | 24         | Male   | Yes                    | –             | 4 × ULN           | No gall stones           | MRCP              | Sphincterotomy |
| [6]             | 68         | Male   | Yes                    | ×1            | 5,399             | No gallstones           | MRCP              | Stent and sphincterotomy |
| [10]            | 11         | Male   | Yes                    | ×2            | 1,972             | No gallstones           | MRCP              | Sphincterotomy |
| [11]            | 45         | Female | Yes                    | ×1            | 1,250             | No gall stones normal CBD | MRCP              | Conservative |
| [14]            | 72         | Male   | Yes                    | –             | –                 | –                        | MRI               | Sphincterotomy and stent |
| [15]            | 70         | Male   | –                      | –             | –                 | –                        | MRCP              | –         |
| [16]            | 53         | Female | –                      | –             | –                 | No gallstones           | CT scan/MRCP      | Pancreatic enzymes |
| [17]            | 35         | Male   | Yes                    | ×15           | 3,670             | Absent GB, santorinicoe | MRCP              | Sphincterotomy and stent |
| [18]            | 79         | Female | –                      | –             | –                 | IPMN                     | ERCP              | Whipple’s |
| [19]            | 80         | Female | No                     | –             | –                 | IPMN                     | MRI/MRCP          | Conservative |
| [20]            | 65         | –      | –                      | –             | –                 | Walled of pancreatic necrosis | ERCP              | Pancreatic stent |
| [21]            | 28         | Male   | Yes                    | ×2            | –                 | Gallstones, dilated CBD | MRI/MRCP          | Sphincterotomy |
| [22]            | 21         | Male   | No                     | –             | –                 | Pancreatic necrosis/abscess, no gall stones | ERCP              | Stent |

IU/L, international units/liter; ULN, upper limit of normal; MRCP, magnetic resonance cholangiopancreatography; ERCP, endoscopic retrograde cholangiopancreatography; CT, computed tomography; MRI, magnetic resonance imaging; IPMN, intraductal papillary mucinous neoplasm; CBD, common bile duct.
only detects cases with severe dilation of the duct. ERCP is considered the gold standard in evaluation of the pancreatic ductal system and therefore diagnosing ansa pancreatica [9]. In ERCP, the sigmoid branch of ansa pancreatica may be wrongly identified as annular pancreas. However, differentiation can be done on the basis of a pancreatogram. In annular pancreas, the looping branch when approaching the minor papilla can be seen crossing the duodenum whereas in ansa pancreatica the branch does not cross the duodenum [10].

Not all patients with ansa pancreatica will experience acute pancreatitis, rather it is an anatomic predisposition. In a patient with recurrent acute pancreatitis, the first objective remains to rule out all other causes, such as alcohol use, gallstones, and medications. As with any other cause of pancreatitis, the mainstay of treatment is adequate fluid resuscitation, early enteral nutrition, and correction of the underlying issue. Once ansa pancreatica is diagnosed, the patient may undergo sphincterotomy of the minor papilla to relieve the obstruction, or in severe cases, endoscopic ligation of the ansa deformity may be needed. Cannulation of the minor papilla can be challenging, and in such situations, an ultrasound-assisted rendezvous technique via the ansa has been performed [14].

Patients with recurrent pancreatitis with an obscure etiology must undergo a complete investigative work-up, including a pancreatogram, in order to exclude the rare anatomic variations of the pancreas and the pancreatic duct. Treatment of these conditions can prevent future episodes of pancreatitis which in rare instances can become severe and life threatening. We propose EUS in cases of negative MRCP before subjecting the patient to ERCP. Further studies are needed to validate this modality as a diagnostic tool for ansa pancreatica.

Acknowledgement

This manuscript does not include any nonauthor contributors to acknowledge.

Statement of Ethics

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. This study protocol was reviewed and the need for approval was waived by the institution review board at BronxCare Health System.

Conflict of Interest Statement

The authors of this manuscript do not have any conflicts of interest to declare.

Funding Sources

This manuscript did not receive any funding.

Author Contributions

D. Shaikh and A. Ahmed searched the literature, wrote and revised the manuscript. J. von Ende and H. Ghazanfar edited and revised the manuscript. A. Dev and B. Balar revised and approved the final version, and B. Balar is the article guarantor.
References

1. Türkvatan A, Erden A, Türkoğlu MA, Yener Ö. Congenital variants and anomalies of the pancreas and pancreatic duct: imaging by magnetic resonance cholangiopancreatography and multidetector computed tomography. Korean Radiol. 2013;14(6):905–13.

2. Dawson W, Langman J. An anatomical-radiological study on the pancreatic duct pattern in man. Anat Rec. 1961;139:59–68.

3. Ishii H, Araki K, Fukushima M, Maruoka Y, Hoshino M, Nakamura A, et al. Fusion variations of pancreatic ducts in patients with anomalous arrangement of pancreatobiliary ductal system. J Hepatobiliary Pancreat Surg. 1990;5(3):327–32.

4. Jarrar MS, Kenissi A, Ghirissi R, Hamila F, Letafie R. Ansa pancreatica: an anatomic variation and a rare cause of acute pancreatitis. Surg Radiol Anat. 2013;35(8):745–8.

5. Guerroum H, Rami A, Kassimi M, Habij I, Imane R, Chikhaoui N, et al. Ansa pancreatica: a rare cause of acute recurrent episode in chronic pancreatitis. BJR Case Rep. 2020;7(1):20200044.

6. Kosirog IS, Boulai BR, Yazici C. Ansa pancreatica: a rare cause of acute pancreatitis: clinical relevance and review of the literature. JOP. 2018;19.

7. Takuma K, Kamisawa T, Hara S, Tabata T, Kuruma S, Chiba K, et al. Etiology of recurrent acute pancreatitis, with special emphasis on pancreatobiliary malformation. Adv Med Sci. 2012;57(2):244–50.

8. Adibelli ZH, Adatepe M, Imamoglu C, Esen OS, Erkan N, Yıldırım M. Anatomic variations of the pancreatic duct and their relevance with the Cambridge classification system: MRCP findings of 1158 consecutive patients. Radiol Oncol. 2016;50(4):370–7.

9. Hayashi TY, Gonoi W, Yoshikawa T, Hayashi N, Ohtomo K. Ansa pancreatica as a predisposing factor for recurrent acute pancreatitis. World J Gastroenterol. 2016;22(40):8940–8.

10. Fatima Hussain SN, Malik MI, Khan SA. Case report on ansa pancreatica: an uncommon cause accounting for recurrent acute pancreatitis. J Pak Med Assoc. 2019;69(11):1759–61.

11. Gandhi V, Gautam P, Pai N. Recurrent acute pancreatitis and the reverse “S”-shaped pancreatic duct. BMJ Case Rep. 2019;12(6):e226492.

12. López-Durán S, Zaera C, González-Martín JA, Forony JR, Albillos A, Vázquez-Sequeiros E. The endoscopic ultrasound-assisted Rendez-Vous technique for treatment of recurrent pancreatitis due to pancreas divisum and ansa pancreatica. Rev Esp Enferm Dig. 2017;109:798–800.

13. Tabata T, Ichiba Y, Miura Y, Itoh H, Doki K. Variations of the pancreatic ducts as a cause of chronic alcoholic pancreatitis; ansa pancreatica. Pancreas. 1992;87(6):806.

14. Tabata T, Kamisawa T, Takuma K, Anjiki H, Egawa N. A patent accessory pancreatic duct prevents pancreatitis following endoscopic retrograde cholangiopancreatography. Dig Surg. 2010;27(2):140–3.

15. Carrascosa-Mirón T, León-Ledesma R, Manuel-Vázquez A, Gorosabel-Calzada M. Uncommon anatomic variation of the pancreatic duct: ansa pancreatica. Cir Esp. 2020;98(4):236.

16. Lee SW, Davidson CJ, Kia Y, Devereaux B, Godinho S, Appleyard M, et al. Recurrent pancreatitis in the setting of gallbladder agenesis, ansa pancreatica, Santorinicoele and eventual intraductal papillary mucinous neoplasia (IPMN). Ann Hepatobiliary Pancreat Surg. 2020;24(3):381–7.

17. Magulick JP, Salem R, Jamidar P. Intraoperative pancreatoscopy for surgical planning in a patient with ansa pancreatica and mixed-type intraductal pancreatic mucinous neoplasm. Gastrointest Endosc. 2020;92(4):968–70.

18. Giarraputo L, Savastano S, Napetti S. Trifidum anomaly of the main pancreatic duct. Pancreatology. 2020;20(3):569–70.

19. Jagielski M, Smočzyński M, Drelich-Górecka B, Adrych K. Transduodenal drainage of symptomatic walled-off pancreatic necrosis in a patient with ansa pancreatica anatomic variation. Arch Med Sci. 2017;13(1):267–9.

20. Ayari H, Rebii S, Ayari M, Hasni R, Zoghlami A. [Ansa pancreatica: a rare cause of acute pancreatitis]. Pan Afr Med J. 2012;13:33.

21. Bhasin DK, Rana SS, Nanda M, Gupta R, Nagi B, Wig JD. Ansa pancreatica type of ductal anatomy in a patient with idiopathic acute pancreatitis. JOP. 2006;7(3):315–20.