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Low Fat and Protein Therapy for Divisum Relapsing-Pancreatitis

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Abstract

Background: Acute Pancreatitis (AP) is the leading gastrointestinal emergency. It results from premature intracellular activation of trypsinogen or intraductal obstruction of pancreatic enzymes. Gall stones, alcohol and hypertriglyceridemia are the leading causes with Pancreatic Divisum (PD) representing a rare one. **The case**: A 58-year-old man presented with severe and progressive epigastric pain following ingestion of a fatty meal and red meat. The patient had a similar attack 3 months ago which was associated with high amylase and lipase, disclosing a self-limited AP. He did not have a previous history of medical, surgical disease, allergy, cigarette smoking, alcohol consumption or family history of pancreatitis. On admission he had high serum amylase at 724 IU/L (Normal: 28 - 100) and lipase at 15000 IU/L (Normal: 13 - 60). Investigations excluded other culprits: infections, autoimmune diseases, IgG4-disease, and genetic disorders. Magnetic Resonance Cholangiopancreatography disclosed PD with complete separation of dorsal and ventral parts and severe pancreatitis limited to the ventral part (head & uncinate process) with its duct draining into minor pancreatic papilla while dorsal part drains into the major papilla. There was no evidence of biliary disease, obstructive lesion, malignant disease or pseudocyst formation. Since the mechanism of such recurrent AP in PD is inadequate drainage of pancreatic enzymes stimulated by cholecystokinin, the patient was treated conservatively and subsequently with a diet low in fat and red meat. He improved within a few days and remained stable clinically and on laboratory testing up to 2 years. **Conclusion**: Mild recurrent AP associated with PD is amenable to a low-fat and protein diet.

Keywords

Acute Pancreatitis, Divisum, MRCP, Diet

1. Introduction

Acute Pancreatitis (AP) is the leading gastrointestinal emergency in the United States accounting for 275,000 hospital admissions annually [1]. It is associated with high mortality, morbidity and impairment of life-quality [2]. It presents with the cardinal features of acute upper abdominal pain radiating to the back, elevated levels of the pancreatic enzymes amylase and lipase, and characteristic features on imaging [3]. It results from inflammation of the pancreas due to premature activation of enzyme precursors, particularly trypsin, in the acinar cells triggering a self-digestive inflammatory cascade or pancreatic duct obstruction [4]. Pancreatic enzymes such as trypsinogen are synthesized and stored in an inactive—hence harmless—form, and are activated upon release into the lumen of the duodenum via the action of enterokinases [5]. AP can result from: (a) premature activation of pancreatic enzymes due to genetic predisposition, toxins (alcohol and organophosphate insecticides), drugs, infections (mumps), metabolic (hypercalcemia, obesity, hypothyroidism and hypertriglyceridemia) as well as (b) pancreatic-duct obstruction, viz. biliary stones, tumor, IgG4-related disease, post-ERCP and congenital duct anomalies [6]. In the present case report, we describe our management of an adult patient who developed recurrent attacks of AP due to Pancreatic Divisum (PD).

2. The Case

A 58-year-old man presented with severe and progressive epigastric pain radiating to the back for 1 week. The patient had similar attack 3 months ago which was associated with high serum amylase and lipase. Hence, it was diagnosed as self-limited AP. Both episodes followed an excessive intake of fatty meal with meat. He did not have significant previous medical or surgical history nor chronic intake of cigarettes, alcohol or drugs. He did not have family history of hyperlipidemia, pancreatitis, or allergy. His body weight was 70 kg. He was afebrile with blood pressure at 110/70 mm Hg with postural hypotension. He had tender and rigid epigastrium, with diminished bowel sounds. Laboratory investigations showed peripheral leukocytic count at 16×10^9/L with 90% neutrophils with normal platelet counts and hemoglobin. Serum sugar, urea, creatinine, electrolytes and liver functions were normal including albumin and bicarbonate. Lipid profile (cholesterol and triglycerides) as well as TSH were normal. Serum amylase and lipase were elevated at 724 IU/L (Normal: 28 - 100) and 15000 IU/L (Normal: 13 - 60), respectively. Urine routine and microscopy were normal. Furthermore, specific investigations were negative for infections (cultures, mumps IgM), autoimmune markers (ANA & ANCA), high-IgG4-level, genetic mutations in (PRSS1, SPINK-1, CFTR, CTRC, CPA1 and CASR genes). Ultrasound of the abdomen showed an enlarged and edematous pancreas with ill-defined margins, yet without pancreatic pseudocyst. The gall bladder and common bile duct as well intrahepatic bile ducts were normal. There was no evidence of gall stones. Magnetic Resonance Cholangiopancreatography (MRCP), with secretin protocol, showed bulky head and uncinate process of pancreas with heterogeneous signal intensity and enhancement after

contrast administration with surrounding fat stranding, free fluid and small lymphadenopathy indicating AP limited only to ventral part of pancreas (Figure 1). There was no evidence of fibrosis, calcifications, tumor or pseudocyst formation. The ventral pancreatic duct was completely separate from the dorsal duct and neither was dilated. The ventral duct entered the duodenum and communicated with common bile duct via the major papilla while the dorsal one communicated via a minor papilla indicating type I PD as shown by MRCP (Figure 2) and in a diagrammatic representation (Figure 3). The patient was treated conservatively with initial nasogastric suction, intravenous fluids, and narcotics for pain control. Within a few days, the patient had improved clinically and his serum amylase and lipase had decreased to normal levels. By the third week, CT scan of the abdomen showed marked improvement in his AP and absence of local complications including pseudocyst formation. On discharge, he was instructed to adhere to (a) low-fat and protein (red meat) diet, (b) excessive carbohydrates and sweats, (c) Omeprazole to decrease stomach acidity, and (d) an oral supplement of pancreatic enzymes (containing combination of lipase, amylase and protease). After 2 years of follow-up, he remained stable and without clinical or laboratory evidence of AP-relapse. Moreover, he remained active, with stable body weight and muscle mass as well as without clinical or laboratory evidence of malabsorption or diabetes mellitus.

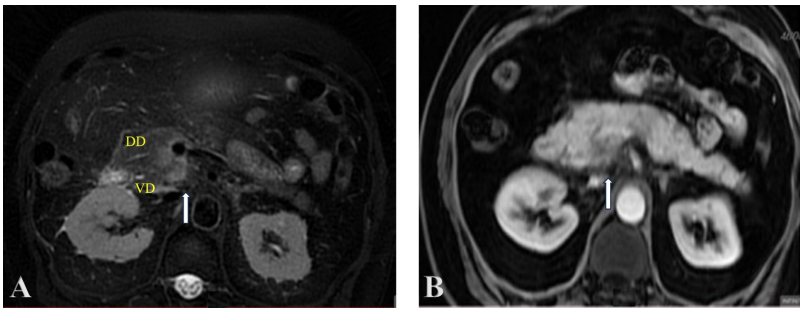

Figure 1. Magnetic resonance cholangiopancreatography of the pancreas showing complete separation of the dorsal and ventral pancreatic ducts (DD & VD) with edema limited to head and uncinate process of pancreas (Arrows) in STIR images (A) with reduced enhancement postcontrast in T1 FAT SAT images (B) indicating ventral pancreatitis.

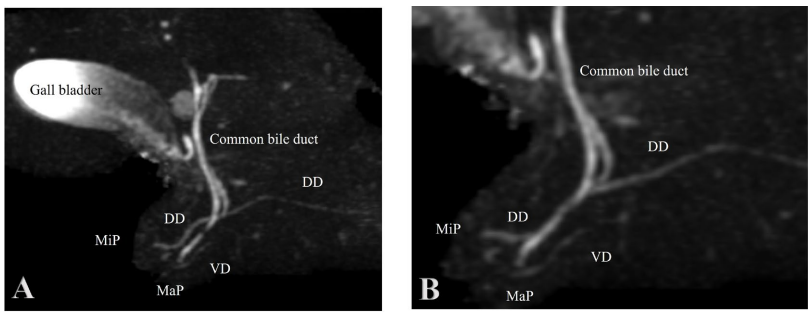

Figure 2. Magnetic resonance cholangiopancreatography images (A&B), without contrast, of the patient with complete separation of the 2 pancreatic ducts (type I divisum); dorsal (DD) which opens in duodenum via minor papilla (MiP) and ventral (VD) via major papilla (MaP). The VD is small and without focal stricture.

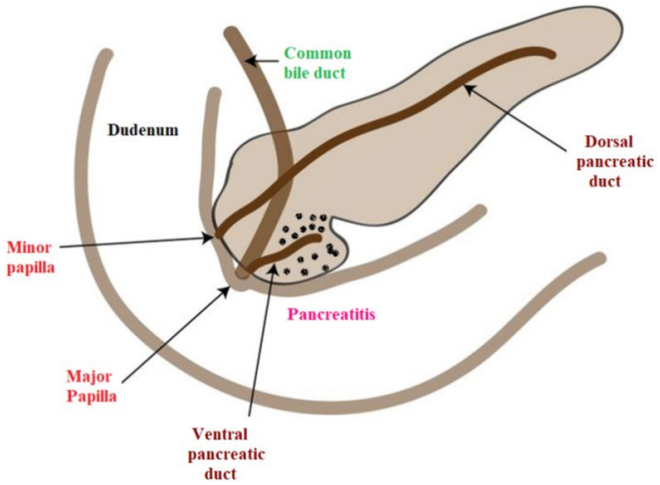

Figure 3. Diagrammatic representation of the pancreatic divisum and ventral pancreatitis.

3. Discussion

The pancreas develops from the posterior foregut endoderm. At approximately 4 weeks of gestation, this endoderm gives rise to dorsal and ventral buds, which gradually elongate. Around week 6, the ventral bud rotates around the developing duodenum and ultimately fuses with the dorsal bud at approximately the 17th week of gestation to form the pancreas. The dorsal bud forms the upper portion of the pancreatic head, the body, and the tail, whereas the ventral bud forms the lower portion of the pancreatic head and the uncinate process [7]. Congenital anomalies of the pancreas are numerous and include its (a) ducts, viz. Pd, Wirsungocele and Santorinicele, anomalous Pancreaticobiliary Junction, and (b) parenchyma viz. annular pancreas, circumportal, dorsal agenesis, contour variants, ectopic, intrapancreatic splenic tissue and congenital cysts [8]. PD represents the most common developmental anomaly of the pancreas. Its prevalence is 10% - 15% of the general population and results from failure of fusion of the 2 pancreatic buds [9]. Consequently, the tail, body, and part of the head of the pancreas drain, into duodenum, through the accessory duct of Santorini rather than the major duct of Wirsung. Historically, PD is associated with recurrent pancreatitis, in 5% of cases, due to stenosis of the sphincter obstructing ventral pancreatic outflow [10]. According to the Revised Atlanta Classification, our patient had recurrent attacks of AP which were mild due to absence of organ failure [11]. Moreover, since the mechanism of his AP is inadequate release of pancreatic enzymes due to stenosis of his ventral duct, we elected to treat him conservatively with measures that suppress stimulation of cholecystokinin that leads to pancreatic enzyme secretion [12]. The latter entails: (a) a diet low in fat and protein (red meat), (b) avoidance of excessive carbohydrates, (c) Omeprazole to decrease stomach acidity, and (d) an oral supplement of pancreatic enzymes (containing a combination of lipase, amylase and protease). Over 2 years of follow-up, it was effective with (a) prevention of further attacks of AP, (b) did not induce malnutrition or weight loss, and (c) remained active and with good quality of life. Assessment of severity

of pancreatic ductal stenosis can be achieved by MRCP with gadoxetate disodium (Primovist or Eovist) from Bayer Health-Care and secretin-enhanced MRCP [13] [14]. Patients with moderate and severe AP due to PD or those who fail our conservative approach should be subjected to endoscopic or surgical papillotomy with/without stent placement [15].

4. Conclusion

Divisum pancreatitis results from inadequate drainage of its pancreatic duct due to relative stenosis and mild cases can be ameliorated with acceptable dietary restriction.

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Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare that they have no potential conflict of interest related to the contents of this article.

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