RECENT TRENDS IN THE MANAGEMENT OF PANCREATIC CYSTS

Essay

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List of Abbreviations

ACC	**A**cinar cell carcinoma
AIPD	**A**nterior inferior pancreaticoduodenal artery
ARDS	**A**cute respiratory distress syndrome
ASPD	**A**nterior superior pancreaticoduodenal arteries.
CBD	**C**ommon Bile Duct
CEUS	**C**ontrast-enhanced ultrasonography
CGRP	**C**alcitonin gene-related peptide
CNP	**C**ystic neoplasms of the pancreas
CT Scan	**C**omputed Tomography Scan
ERCP	**E**ndoscopic Retrograde Cholangiopancreato-graphy
EUS	**E**ndoscopic ultrasound
IMV	**T**he inferior mesenteric vein
IPMNs	**I**ntraductal Papillary Mucinous Neoplasms
LECs	**L**ymphoepithelial cysts
LOH	**L**oss of heterozygosity
MCNS	**M**ucinous cystic neoplasms
MDCT	**M**ultidetector computerized tomography
MPD	**M**ain pancreatic duct
MRCP	**M**agnetic resonance cholangiopancreatography
MRI	**M**agnetic Resonance Imaging
NETs	**N**euroendocrine Tumors
NOTES	**N**atural orifice transluminal endoscopic surgery
PANDA study	**T**he Pancreatic Cyst DNA Analysis study
PAS stain	**P**eriodic acid-Schiff stain
PET	**P**ositron-emission tomography

PFCs	**P**eripancreatic fluid collections
POPS	**P**eroral pancreatoscopy
pps	**P**ancreatic pseudocysts
PSPD	**P**osterior superior pancreaticoduodenal arteries.
SCA	**S**erous cystadenoma
SCNs	**S**erous cystic neoplasms.
SMA	**T**he superior mesenteric artery
SMV	**T**he superior mesenteric vein
SPDP	**S**pleen-preserving distal pancreatectomy
SPN	**S**olid pseudopapillary neoplasm
TPN	**T**otal parenteral nutrition.
US	**U**ltrasound
VHL	**V**on Hippel- Lindau disease
VIP	**V**asoactive intestinal peptide

INTRODUCTION

Cystic lesions of the pancreas have long posed diagnostic and treatment dilemmas to surgeons and patients. While many identified lesions may prove to be inflammatory pseudocysts or other benign conditions, the possibility of malignancy within a cystic lesion necessitates a thorough diagnostic work up. Advances in the medical disciplines of radiology, Pathology, gastroenterology and surgery have led to a recent reconsideration of the classification of cystic neoplasms of the pancreas, reflecting an improved understanding of diagnosis, prognosis, and treatment of these often challenging lesions ***(Christopher Sonnenday et al., 2007).***

Cystic lesions of the pancreas were first described in 1824. And can be either inflammatory or proliferative in nature. The natural history of such cysts has only begun to be fully appreciated in the last 20 years. Due to the Diagnostic uncertainty associated with these lesions, routine Excision of all pancreatic cysts has been recommended by some centers, the rationale for this stance being that differentiation between benign and malignant cysts can be difficult and the dire consequences of missing the window for a curative resection in patients who can tolerate such surgery.

This approach inevitably results in Patients with benign disease being exposed to the potential morbidity and mortality

of pancreatic resection and is becoming untenable with the increasing number of pancreatic cysts detected as an incidental finding on cross-sectional imaging ***(Garcea et al., 2008).***

These considerations have led to reports which argue that due to improvement in imaging techniques, identification of a group of patients with an extremely low risk of malignancy is possible and that these patients should be managed by observation alone ***(Garcea et al., 2008).***

Pseudocysts make up the majority of all cystic lesions of the pancreas, the remainder comprising cystic tumours and true cysts (true cysts accounting for a very Small percentage of these lesions). Hydatid cysts of the pancreas have also been described and should be considered as a differential diagnosis in countries where Hydatid disease is endemic. Pancreatic cystic tumours fall into one of three major groups; serous tumours (including serous cystadenoma and cystadenocarcinoma), Mucinous tumours (including mucinous Cystadenomas, mucinous cystadeno-carcinomas, intraductal Papillary adenomas and intraductal papillary Adenocarcinoma) and solid pseudopapillary tumours (SPT).

The majority of cystic tumours of the pancreas are slow-growing and asymptomatic. When symptoms do occur, they are usually secondary to a mass effect and tend to be vague and poorly localized in nature. Only intraductal papillary mucinous tumours (IPMTS) present with pain and may mimic chronic pancreatitis. Unfortunately, no formal classification of

pancreatic Cysts exists at present and there are other descriptions of them based on the nature of the cyst wall lining ***(Garcea et al., 2008).***

Pancreatic Pseudocysts are collections arising from around the pancreas, which lack an epithelial lining, and they occur following acute pancreatitis, chronic pancreatitis or secondary to pancreatic trauma. Pseudocysts normally contain necrotic fat and a mixture of necrotic cells, including Neutrophils surrounded by granulation tissue, which eventually matures to form a fibrotic pseudocapsule ***(Garcea et al., 2008).***

Pseudocysts developed in about 2 %of cases of acute pancreatitis, the cysts are single in about 85% of cases and the remaining are multiple ***(Gerand and Lawrence, 2003)***.

Primary cystic neoplasms of the pancreas are rare neoplasm, comprising about 10 – 15 % of all pancreatic cystic masses and only 1% of primary pancreatic lesions of unknown origin. The Importance of identifying those neoplasms became clear because of their latent or overt malignant potential ***(Christopher Sonnenday et al., 2007).***

Although the clinical, radiologic, and pathologic features of cystic pancreatic lesions are well known, preoperative diagnosis is difficult. Differentiation between a pancreatic pseudocyst and a cystic pancreatic neoplasm is crucial in determining the proper treatment. Careful evaluation of the patient's clinical history is important for accurate diagnosis of a pseudocyst ***(Kim et al., 2005).***

AIM OF THE WORK

The aim of this work is to highlight the recent trends in the diagnosis and management of the pancreatic true and pseudocyst.

Chapter (1)
EMBRYOLOGY

Development of the pancreas:

The pancreas is of endodermal origin and develops from ventral and dorsal pancreatic buds, the ventral bud arises from the hepatic diverticulum, and the dorsal bud arises from the developing duodenum ***(Mulholland et al., 2006).***

At stage 5 weeks differential growth of the wall of the duodenum results in movement of the ventral pancreatic bud and the bile duct to the right side and ultimately to a dorsal position. It is not clear whether there is a corresponding shift of mesenchyme during this rotation; however, the ventral pancreatic bud and the bile duct rotate from a position within the ventral mesogastrium (ventral mesodudenum) to one in the dorsal mesogastrium (dorsal mesodudenum) which is destined to become fixed onto the posterior abdominal wall ***(Williams et al., 2005)***.

The duodenal wall grows asymmetrically; the opening of the two ducts (the duct of the ventral bud and the duct of the dorsal bud), originally diametrically opposite, are thus carried around into line with each other, and the two parts of the gland fuse into the single adult pancreas (Figure 1) The duct systems of the two buds anastomose and there is eventually some interchange of drainage areas. The end result is that the duodenal end of the dorsal duct becomes the accessory

pancreatic duct, and the duct of the ventral bud joins with the remainder of the dorsal duct to form the main pancreatic duct ***(McMinn, 1994).***

During the shift of the ventral bud, the superior mesenteric vessels, which are extending from, the abdominal aorta, become trapped between the head and uncinate process of the pancreas. Initially the body of the pancreas extends into the dorsal mesogatrium and then cranially into the dorsal mesogastrium. As the stomach rotates, this portion of the dorsal mesogastrium is directed to the left forming the posterior wall of the lesser sac. The posterior layer of this portion of dorsal wall (peritoneum) and the pancreas becomes mainly retroperitoneal ***(Williams et al., 2005).***