PANCREATITIS

An Essay

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of
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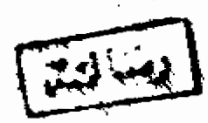

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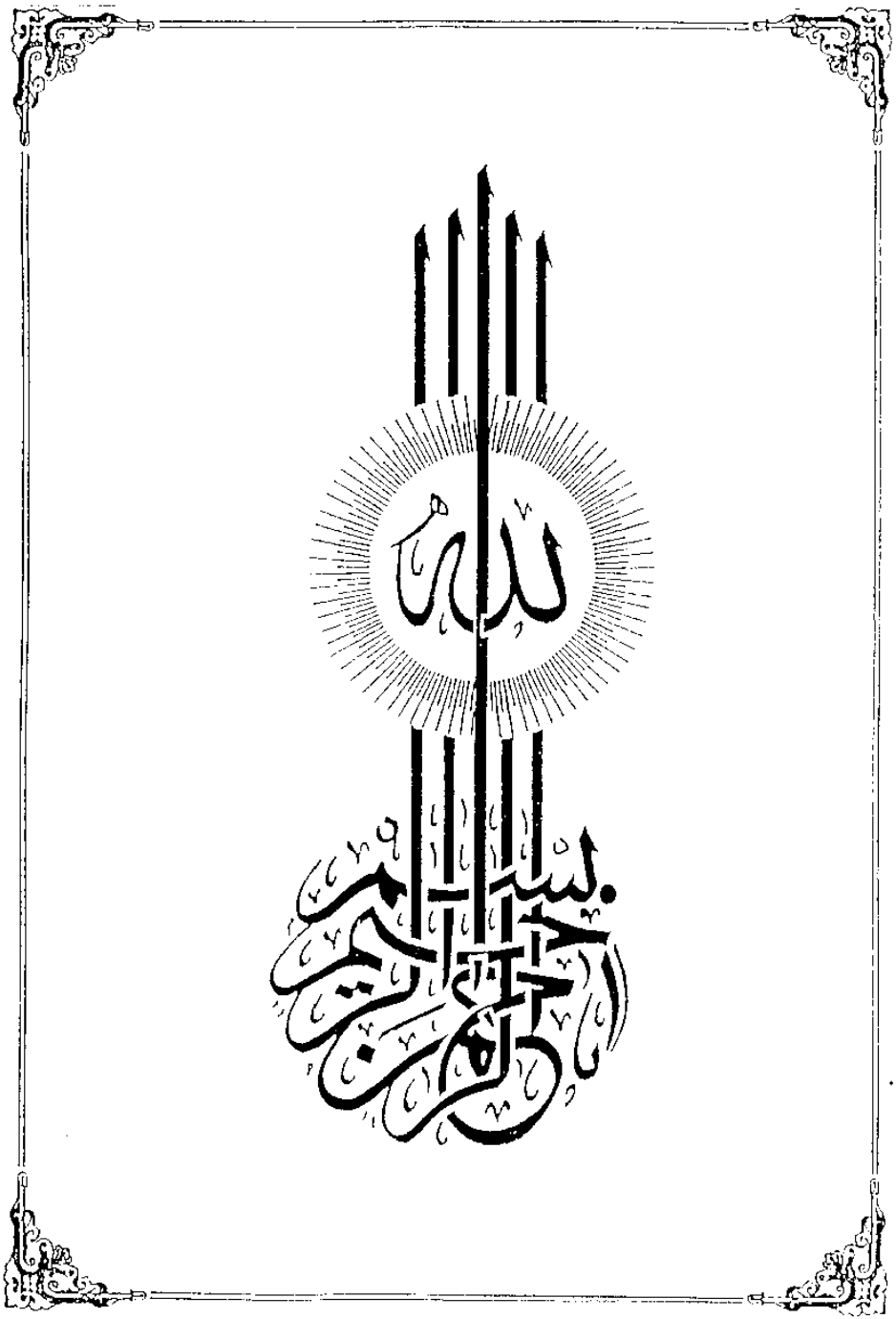

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INTRODUCTION

Acute and chronic pancreatitis has been represented by high rate of mortality and morbidity due to difficulties in diagnosis, laboratory investigation, hence the subsequent differential diagnosis and treatment in the proper time and technique.

In the present study, the various forms of acute and chronic pancreatitis with their manifestations and management are dealt with aiming at a good approach to the subject from the various points of view.

ANATOMY OF THE PANCREAS

The pancreas is an elongated flattened organ located in the epigastrium and left hypochondrium behind the lesser omentum. The weight of the adult human pancreas ranges from 90 to 120 gram. The gland is normally pinkish in colour and varies in length from 10 to15 cm. The surface is lobulated and is anteriorly covered by the peritoneum and the antral portion of the stomach, liver, tranverse colon and small intestine. It is conveniently divided into the head, body, and tail and extend from the loop of the duodenum to the hilum of the spleen. The head with its inferior extension, the uncinate process, is closely attached to the duodenal loop. The common bile duct passing through a groove within the parenchyma of the organ. The posterior surface of the head is in close proximity to the inferior vena cava, the left adrenal vein and the aorta. Anteriorly it is crossed by the superior mesenteric vessels. From the head, a slightly constricted portion, the neck bends slightly forward and upward and then extends to the left, where it forms the body, which then tapers off to form a short tail *(Volk and Allen, 1986).*

BLOOD SUPPLY

The arterial supply consists of a vertically directed system of arteries (from the gastroduodenal and superior mesenteric arteries) around the head of the pancreas and a horizantally directed system (from the splenic or coeliac arteries), around the body and tail of the pancreas. There is a rich anastomosis between the two systems. Most of the veins draining the pancreas are tributaries of the splenic vein. Other veins empty into the superior mesenteric and portal veins *(Lee McGregor's, 1986).*

NERVE SUPPLY

The sympathetic nerve supply is from the splanchnic nerves and the parasympathetic supply is from the vagus *(Lee McGregor's, 1986).*

THE DUCTAL SYSTEM

The duct of wirsung, the major pancreatic duct, begins in the tail, lying nearer its posterior than anterior surface. It takes its origin from confluence of numerous small ducts of the lobules crossing the gland. They forming a herring bone pattern, enter the main duct almost at a right angle. The duct traverses the body and reaches the neck where it turns downwards and backwards and approaches the common bile duct or joins it as it courses through the parenchyma of the pancreas. Both ducts form a short dilated hepatopancreatic ampulla, which empties into the major duodenal papilla (papilla of vater) situated at the junction of the medical and posterior walls of the duodenum about 8 to 10cm distal to the pylorus. The ampulla varies from 1.5 to 4.5 mm in width, and from 1 to 14 mm in length. Usually the 2 ducts do not unite until they arrive close to the major papilla. At times they enter separately into the duodenum. The ampulla is surrounded by the sphincter of oddi, which is presumed to prevent reflux of bile into the pancreas or pancreatic secretion into the biliary system. The other pancreatic duct (duct of santorini) originates in the upper anterior portion of the head. It runs upward in front of the main duct to which it is connected by communicating duct in 33% to 90% of the cases. Eventually, it opens by way of the minor papilla into the duodenum, 2 cm above and slightly ventral to the a major duodenal papilla. Both ducts may fuse and form a common channel outside the duodenum. In 10% of the cases, the main duct drains into the accessory papilla and has no connection with common bile duct. As stated, the duct of wirsung is the major duct in 90% of the cases and drains the bulk of the pancreatic parenchyma.

Only a small upper anterior portion of the head is drained into accessory duct. In about 9% of cases, the duct of santorini is the major pancreatic duct.

Occasionally, additional small accessory ducts may enter the duodenum or the common bile duct *(Volk and Allen, 1986)*

CONGENITAL ANOMALIES OF THE PANCREAS

1) PANCREAS DIVISUM

The condition referred to as pancreas divisum results from the failure of the dorsal and ventral pancreatic primordia to fuse. As a result, the usual anastomosis between the dorsal and ventral ducts does not take place, with the result that the dorsal duct (duct of santorini) becomes the dominant duct draining the body and tail of the gland. The ventral duct (duct of wirsung) remains short and rudimentary, draining only the lower portion of the pancreatic head and the uncinate process that arise from the ventral anlage *(Moossa, 1985).*

The Usual Explanation for the development of pancreatitis in patients with pancreas divisum is that the minor papilla acts as a point of relative obstruction to the flow of pancreatic juice since its small size is unable to accommodate the bulk of pancreatic secretion *(Moossa, 1985).*

The over all prevalence of pancreas divisum appears to be between 2% and 3% in the general population *(Gregg, 1977).*

2) ANNULAR PANCREAS

Annular pancreas is an uncommon congenital anomaly consisting of histologically normal pancreatic tissue partly or completely encircling the duodenum in ring like fashion *(Griboski, 1975).*

It is generally believed that the formation of the annulus of the pancreas is a result of failure of the tip of the ventral pancreatic bud to rotate completely to the right and posteriorly with the duodenum. Presenting symptoms in infancy were commonly related to duodenal obstruction. About 70% of adults are started to have symptoms as intermittent epigastric fullness, symptoms of duodenal ulceration and jaundice *(Kiernan et al., 1980).*

CLASSIFICATION OF PANCREATITIS

During the Meeting held in Marsielles, France in 1963, four types of pancreatitis were identified.

Acute and relapsing acute were defined as forms of the disease in which morphological and functional recovery of the gland could be expected once responsible factors had been eliminated. Biliary tract stone disease was most often the cause of these forms of pancreatitis. In contrast, chronic and chronic relapsing pancreatitis, most frequently associated with ethonal abuse, were defined as forms of the disease in which morphologic and functional derangements persisted even after the initiating causes or factors were eliminated *(Steer, 1989)*.

In 1984, additional international meetings were held in Marseilles to classify and describe the various forms of pancreatitis into Acute reversible & chronic progressive which is a simplified classification scheme *(Singer et al., 1985)*.

PATHOLOGY OF PANCREATITIS

1-ACUTE PANCREATITIS

Two main Morphologic forms of acute pancreatitis are generally distinguished; edematous and necrotizing forms. Since haemorrhage and suppuration are striking features commonly associated with necrosis, the term haemorrhagic or suppurative pancreatitis are often used to designate the necrotizing form *(Ammann and Warshaw, 1986).*

In the Milder form of edematous pancreatitis, the characteristic features consist of a grossly enlarged and congested pancreas without appreciable necrosis or haemorrhage. In the fulminant form of pancreatic necrosis, by contrast, the pancreas and peripancreatic tissue are grossly transformed into an inflammatory. tumour mass with diffuse hemorrhagic changes. These sometimes simulate a large hematoma. The entire gland is usually involved but in some cases the lesion is confined to one or several subsegments of the pancreas. Spotty fat necrosis in the adjacent adipose tissue is a frequent finding. A small amount of blood tinged effusion in the peritoneal cavity is also found. The histologic aspect of acute pancreatitis varies according to the basic nature and the stage of evolution of the process. Varying degrees of edema and leucocytic infiltration are common and may or may not be attended by necrosis, haemorrhage, suppuration, cyst formation, and fibrosis *(Ammann and Warshaw, 1986).*

2. CHRONIC PANCREATITIS

Classic chronic pancreatitis would appear to be due to protein precipitation in the duct leading to duct obstruction and, subsequently to duct dilatation, acinar cell atrophy, fibrosis and eventual calcification of the protein plugs *(Marks et al., 1986).*

Macroscopically, the gland is hard and usually shrunken, duct dilatation with calculi and cysts of various sizes may be evident on palpation. Histologic examination shows localized or diffuse fibrosis with destruction of glandular parenchyma and duct dilatation. Calcification, if present, is almost invariably intraductel. End stage chronic pancreatitis may show little scattered islets of langerhans in area of fibrosis *(Marks et al., 1986)*.

ETIOLOGY OF PANCREATITIS

Although the cellular events that underlie the development of pancreatitis are not clear, a number of factors and disease states have been associated with the development of pancreatitis (table 1). Billiary tract stone disease and ethonal abuse together account for the origin of 60 to 80 percent of all cases of pancreatitis. The other etiologies of pancreatitis account for only 10 to 20 percent of the cases, leaving 10 to 15 percent of patients who have no identifiable etiology, that is referred to idiopathic pancreatitis

Table (1): Etiologies of pancreatitis.

Billiary tract stone disease	lipid abnormalities
Ethonal abuse	post operative
Ductal obstruction	Trauma
Infections	Miscellaneous
Drugs	Idiopathic

(Steer, 1989).

I. Biliary Tract Stone Disease

An association between biliary tract stone disease and acute pancreatitis has been recognized since 1901, when in a classic report Opie described two patient dying of acute pancreatitis who at autopsy were found to have stones impacted in the terminal common bile duct *(Steer, 1989).*

More recent reports, most notably those of acosta and his associates demonstrated that acute pancreatitis is associated with the passage of stones through the terminal bile duct into the duodenum *(Acosta et al., 1977).*

Several theories have been advanced to explain the relation between stones and an attack of acute pancreatitis. The earliest was the common-channel theory of

Opie, which suggested that the stone became impacted at distal end of a common biliopancreatic channel permitting bile to reflux from. The biliary tree into pancreatic duct. Bile could trigger pancreatitis either by activating pancreatic digestive enzymes or by injuring pancreatic cells directly *(Opie, 1901).*

Indeed, more recent experimental observations by Reber and coworkers have shown that bile salts can damage the pancreatic duct mucosal barrier, and that as a result molecules as large as digestive enzymes can diffuse from the duct into the gland parenchyma *(Reber et al., 1982).*

However, a number of objections to the common channel theory have been raised. For example, the pancreatic secretory pressure exceeds the biliary secretory pressure, so distal obstruction should favour flow of bile into the biliary tree rather than flow of bile into pancreatic ductal system. Finally, bile itself does not activate pancreatic digestive enzymes *(Robinson et al., 1963).*

For these reasons, the common channel theory has not been widely accepted *(Steer, 1989).*

In experimental animals, if the segment of duodenum into which the pancreatic duct empties is surgically converted to a closed loop, reflux of duodenal juice initiates severe pancreatitis (*pfeffer loop). Pancreatitis associated with acute afferent loop obstruction after Billorth II gastrectomy is probably the result of similar factors *(Reber and Way, 1988).*

Advocates of this duodenal reflux theory suggested that, passage of stone through the sphincter of oddi render that muscular barrier to reflux incompetent and in this manner led to pancreatitis. The most serious objection to the duodenal reflux theory is the observation that pancreatitis does not usually follow surgical procedures that either render the sphincter incompetent (e.g., sphincteroplasty) or by pass the sphincter entirely (pancreaticojejunostomy) *(Steer, 1989).*

The final theory advanced to explain the association between biliary stones and pancreatitis suggests that the offending stone triggers pancreatitis by

obstruction of the pancreatic duct. Presumably, continued secretion into the obstructed ducted system leads to ductal hypertension, rupture of small ducts, and extravasation of pancreatic juice into the parenchyma of the gland. Several objections to this theory have been raised. These include the experimental observation that ligation of the pancreatic duct cause pancreatic atrophy but not acute pancreatitis. Further more, because the pancreatic ductal fluid normally contains inactive digestives enzymes, ductal hypertension and rupture would be expected to cause only extravasation of inactive proteolytic enzymes, and thus parenchymal injury would seem unlikely. This latter issue might be resolved by recent studies, that suggest that pancreatic ductal obstruction could lead to activation of digestive enzymes within acinar cell, in that case, pancreatitis might follow ductal obstruction, as activated enzymes would be released within the gland *(Steer,* 1989).

2. Alcohol

Long-standing ethonal abuse is perhaps the most common cause of chronic pancreatitis. Epidemiologic studies indicate that the incidence of this type of chronic pancreatitis is directly related to the duration of alcohol abuse and to the total amount of alcohol consumed *(Steer, 1989).*

Dietary factor interact with alcohol as a cause; population with high protein intake seem statistically to be most likely to develop alcohol induced chronic pancreatitis, *(Steer, 1989).*

Most patients with alcohol-related pancreatitis develop symptoms after many years of ethonal abuse and when first studied already have significant irreversible morphologic and functional derangements. Occasionally, however, patients are encountered who have a sudden onset of symptoms after only one or two exposures to ethonal, and morphologic as well as functional recovery occurs after the symptoms have resolved. The means by which alcohol causes this acute form of pancreatitis is entirely unknown but it is likely that this form results from either a direct toxic effect of alcohol or one of its metabolites on the pancreas.

Alternatively, alcohol might cause ductal hypertension as a result of secretory stimulation combined with alcohol-induced spasm of the sphincter of oddi *(Pirola, 1970).*

3. Ductal Obstruction

Pancreatic tumours, strictures, or other lesions that interfere with the drainage of secretions including duodenal tumours, penetrating peptic ulcers can cause pancreatitis. Thus, an attack of acute pancreatitis may be the first presentation of a periampullary tumour, and such a tumour must be considered as the cause of pancreatitis when evaluating a non alcoholic patient whose attack of pancreatitis is not the results of biliary stone disease *(Steer, 1989).*

In pancreas divisum, most of the pancreatic secretion drains via the duct of santorini through the lesser papilla. It has been suggested that these patients have a relative stenosis of the lesser papilla not enough to accommodate the volume of juice that must drain by this route in pancreas divisum, *(Warshaw et al., 1983).*

Occasional patients with pancreas divisum have been identified to have changes typical of chronic pancreatitis confined to the distribution of the duct of santorini *(Blair et al., 1984).*

4. Infections and Infestations

Mumps and Coxsackie viruses and mycoptasma pneumoniae have been reported to cause pancreatitis *(Imrie et al., 1977).*

Certain parasites, including ascaris have been associated with pancreatitis by physically blocking the pancreatic duct *(Steer, 1989).*

5. Drugs

Exposure to a number of drugs has been associated with attacks of pancreatitis Greutz feldt and *(Lankisch,* 1985).