MANAGEMENT OF ACUTE PANCREATITIS

AN ESSAY

SUBMITTED FOR PARTIAL FULFILLMENT
OF THE MASTER DEGREE IN GENERAL SURGERY

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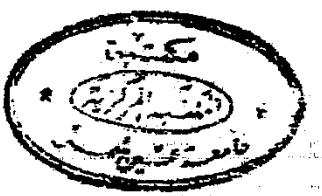

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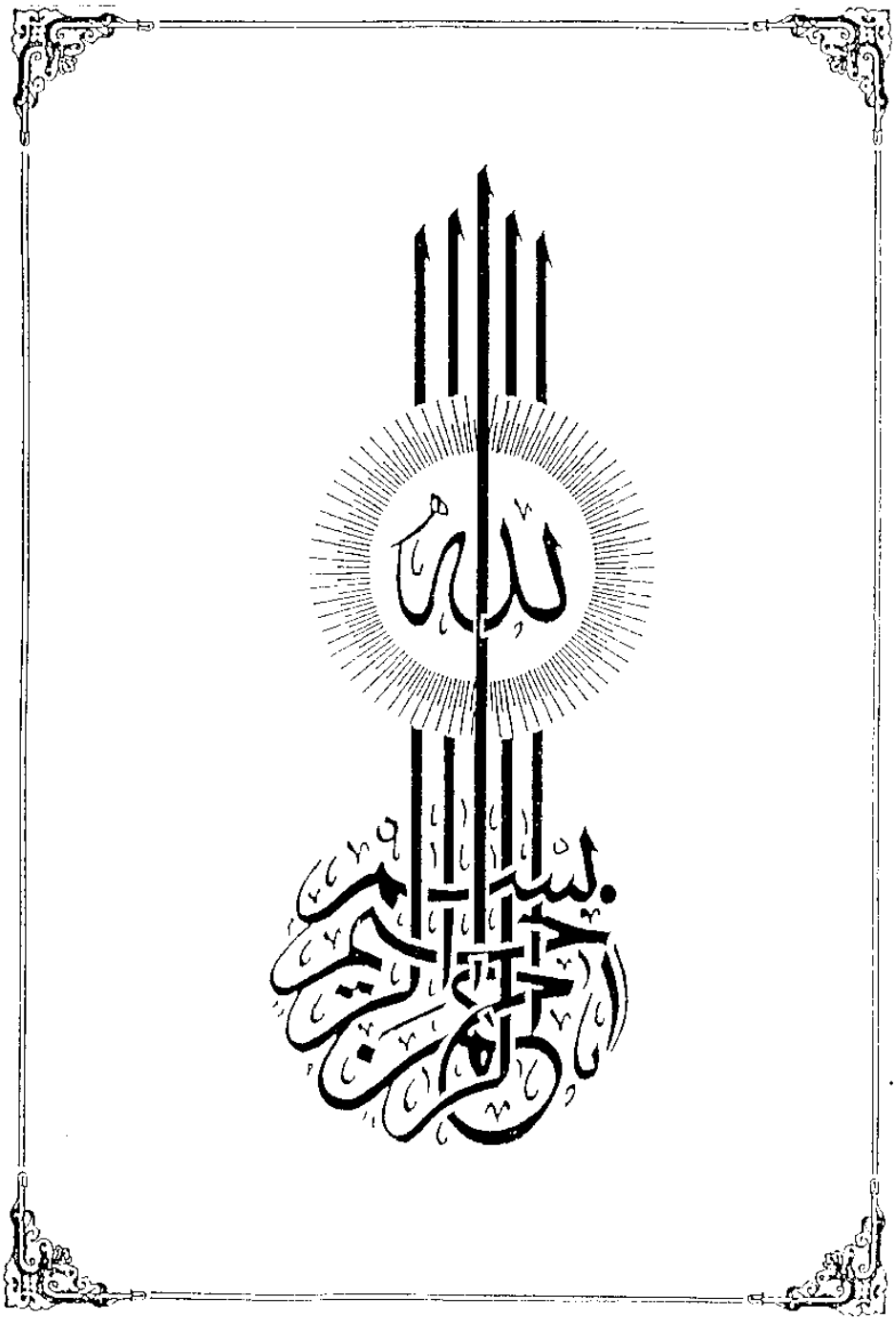
(1992)

AKNOWLEDGMENT

I wish to express my deep thanks and sincere gratitude to Professor. Dr. ADEL AIN SHOUKA , Professor of General Surgery ,Faculty of Medicine, Ain Shams University, for the kind advice encouragement and supervision he offered throughout the course of this work .

Special grateful thanks and appreciation to Dr.AWAD ALKAYYAL Lecturer of General Surgery ,Faculty of Medicine ,Ain Shams University for continuous help,encouragement and supervision provided me all facilities during the conduction of this work.

To every one who participated in some way or the other, to let this work come to such a final picture, I owe my thanks and gratitude.



INTRODUCTION

Acute pancreatitis has been defined as pancreatic inflammation that may be followed by clinical and biological restitution of the gland if the primary cause is eliminated. It includes spectrum of clinical illness that range from mild selflimiting symptoms to rapid deterioration and death. The initiating etiologic factors are multiple and diverse and the pathological finding may range from pancreatic edema to hemorrhagic infarction.

* Much is known about the causes of acute pancreatitis but despite the accumulation of a considerable amount of experimental data, our understanding of the pathogenesis of acute pancreatitis is still incomplete.

* Patient suffers clinically from many symptoms and signs similar to other acute abdominal conditions or with chest or cardiac complaints. SO, it is realy important to bear the diagnosis of acute pancreatitis always in mind.

* Great advances have occurred recently in the laboratory, radiological and scanning techniques. Such advances facilitate a proper aetiological diagnosis of acute pancreatitis and subsequentley to a proper management.

EMBROLOGY OF THE PANCREAS

-The pancreas develops at the begining of the fourth week of gestation from the caudal part of foregut as dorsal and ventral pancreatic primordia .

-The dorsal pancreatic primorduim arises from the dorsal side of the duodenum.

-The ventral pancreatic primordium arises slightly later from the base of the hepatic diverticulum.

-The ventral pancreas shows a slower growth rate than the dorsal primordium producing a large dorsal and a small ventral embryonic pancreas.

-The ventral pancreas is shifted to a dorsal position by rapid growth of the left side of the duodenum.

-The ventral and dorsal pancreatic primordia then fuse into a single glandular structure at the end of the sixth week.
-The ventral primordium contributes the uncinate process and part of the head of the pancreas.The dorsal primordium

ANATOMY OF THE PANCREAS

ANATOMY OF THE PANCREAS

The pancreas is a soft ,flattened gland and its surface is finely lobulated ,the whole being over six inches long. It lies transversely in the retroperitoneum of the upper abdomen. It consists of head ,neck,body and tail. (Last,1988)

THE HEAD OF PANCREAS:-

Is firmly fixed to the medial aspect of the second and third parts of duodenum . The head of the pancreas together with duodenum is suspended from the liver by the hepato-duodenal ligament,the head lies at the level of the second lumbar vertebrae.It is flattened and has TWO surfaces.

ANTERIOR SURFACE

Is related to the pylorus and transverse colon.

POSTERIOR SURFACE

Is related to the hilus of the right kidney,inferior vena cave,right crus of diaphragm.

(Skandalakis et al .,1979)

The posterior surface is deeply indented and sometimes tunneled by the terminal part of the common bile duct. The lower part of posterior surfaces is prolonged,wedge-shaped to the left behined the superior mesentric vessels and in front of the aorta this is the uncinate process. (Last,1988)

*** THE NECK OF THE PANCREAS:-**

Is prolonged to the left from the upper part of the anterior portion of the head. It is roughly that portion of the pancreas lying over the superior mesentric vessels. The neck of pancreas lies in front of the superior mesentric vein and its direct upward continuation, the portal vein. (Last , 1988)

***THE BODY OF THE PANCREAS :-**

Passes from the neck to the left at the level of the first lumbar vertebra and sloping gently upward across the left renal vein and the aorta, left crus of the diaphragm, the left psoas muscle and the lower pole of the left suprarenal gland to the hilum of left kidney. The body of the pancreas has 2 borders and 2 surfaces .

(A)UPPER BORDER:

Is related to the aorta at the origin of the short coeliac axis. The tortuous splenic artery passes to the left along upper border of body and tail.

(B)LOWER BORDER:

Superior mesentric vessels emerges from under the lower border alongside of the neck.

(2)ACCESSORY PANCREATIC DUCT:-

It opens in the duodenum at a small papille situated 2 cm proximal to the duodenal popilla. It drain the uncinate process and lower part of the head of the pancreas.

(Last , 1988)

THE ARTERIAL BLOOD SUPPLY OF THE PANCREAS

* The pancreas is supplied with blood from both the coeliac trunk and the superior mesentric artery.

* The head of pancreas and the concave surface of duodenum are supplied by 2 pancreatico-duodenal arterial arcades:-

(1) ANTERIOR PANCREATICO DUODENAL ARCADES:-

Is formed by anastomosis of the antero- superior pancreatico- duodenal artery (branch from gastroduodenal) and the antero-inferior pancreatico- duodenal artery (branch from superior mesentric artery).

(2)POSTERIOR PANCREATICO-DUODENAL ARCADES:-

Is formed by anastomosis of postero-superior pancreatico duodenal artery (branch from gastroduodenal artery)and postero-inferior pancreatico duodenal artery(branch from superior mesentric artery).

THE DORSAL PANCREATIC ARTERY:-

It lies posterior to the neck of the pancreas.Its most common origin is from the proximal 2 cm of the splenic artery it gives off the TRANSVERSE PANCREATIC ARTERY and it supplies the body and tail of the pancreas.

(Skandalakis et al.,1979)

THE VENOUS DRAINAGE OF THE PANCREAS

The venous drainage of the pancreas flows into the portal system.The anterior and posterior pancreatico-duodenal veins pass over the ventral and dorsal aspects of the pancreas near the duodenum draining the head and uncinate process and emptying into the superior mesentric vein as well as into the portal vein ,tributaries of the portal vein drain the neck also the splenic vein drains tributaries from the body and tail.

Inferior pancreatic vein lie within the glandular parenchyma and empty into the left side of the superior mesentric vein,inferior mesentric vein or occassionally to the splenic vein. (Skandalakis et al .,1979)

PATHOLOGY OF ACUTE PANCREATITIS

DEFINATION:-

Acute pancreatitis has been defined as pancreatic inflammation that may be followed by clinical and biological restitution of the gland if the primary cause is eliminated. It includes spectrum of clinical illness that range from mild self limiting symptoms to rapid deterioration and death.

The initiating etiologic factors are multiple and diverse and the pathological finding may range from pancreatic edema to hemorrhagic infarction. (Ranson J.H.C ,1990)

MORPHOLOGICALLY:-

Acute pancreatitis presents a great variety of morphologic pictures:-

(1)An edematous interstitial pancreatitis :

In the majority of cases, acute pancreatitis takes the course of an edematous interstitial inflammation characterized by a periacinar, interstitial edema that causes an acculumation of inflammatory cells.This may lead to the release of an inflammatory exudate containing biologically active compounds,generating spots of fatty tissue calcification.This form of the disease is generally causes low morbidity and the hospital mortality rates are below 2% in most published clinical series.

(2)NECROTIZING PANCREATITIS:

In 8 to 15%,a necrotizing course of acute pancreatitis develops.

Morphologically it shows interstitial edematous inflammation combined with necrosis of the pancreatic and peripancreatic tissues.

The necrotizing process tends to spread diffusely into the retroperitoneal tissue,the pancreas is enlarged soft and may show multiple areas of reddish black haemorrhage with a numerous small or larger greyish areas of fatty necrosis.

Hospital mortality rates in patients with hemorrhagic necrotizing pancreatitis exceed 20% in most recently published series. (Beger et al.,1989)

ETIOLOGY OF ACUTE PANCREATITIS:-

Although the cellular events that underlie the development of acute pancreatitis are not clear,a number of factors and disease states have been associated with the development of pancreatitis in table 1.

TABLE (1)ETIOLOGIES OF PANCREATITIS

* Biliary tract stone disease	* Lipid abnormalities
* Ethanol abuse	* Postoperative
* Ductal obstruction	* Traumatic
* Infections	* Miscellaneous
* Drugs	* Idiopathic

(Steer et al., 1989)

PATHOLOGY OF ACUTE PANCREATITIS

* Biliary tract stone disease and ethanol abuse together account for the origin of 60 to 80% of all cases ,the other etiologies account for only 10-20% of the cases leaving 10 to 15% of patients who have no identifiable etiology that is referred to idiopathic pancreatitis.

(1)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 2 patients dying of acute pancreatitis who at autopsy were found to have stones impacted in the terminal common bile duct. (Steer ,1989)

Several theories have been advanced to explain the relation between stones and an attack of acute pancreatitis .

I-COMMON-CHANNEL THEORY OF OPIE

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

However,an objection has been raised:

Pancreatic secretory pressure exceeds that the biliary secretory pressure So distal obstruction should favor flow of pancreatic juice into the biliary tree ,rather than flow of bile into pancreatic ductal system . (Cuschieri et al ., 1984)

ii-DUODENAL REFLUX THEORY

Which suggested that passage of a stone through the sphincter of oddi,rendered that muscular barrier to reflux incomptent and in this manner led to pancreatitis. The most serious objection to this theory is the observation that pancreatitis does not follow surgical precedure that either render the sphincter incompetent e.g Sphincteroplasty or bypass the sphincter entirely (Pancreatico-Jejunostomy). (Steer , 1989)

iii-THE FINAL THEORY,SUGGESTS THAT

The stone will triggers pancreatitis by obstruction of pancreatic duct which lead to continuous secretions into the obstructed ductal system and so leading 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.

Experimental observation that ligation of the pancreatic duct causes pancreatic atrophy but not acute pancreatitis.

Further more because the pancreatic ductal fluid normally contains inactive digestive enzymes,so duct obstruction and rupture will lead to extravasation of inactive proteolytic enzymes and thus parenchymal injury would seem unlikely.But a recent study suggested that pancreatic ductal obstruction could lead to activation of digestive enzymes within acinar cells. In that case, pancreatitis might follow ductal obstruction as activated enzymes would be released within the gland. (Steer , 1989)

2-ALCOHOL

Pancreatitis is recognized in 0.9-9.5% of alcoholic patients. Symptoms are usually first noted only after 6 to 10 years period of heavy alcohol ingestion. Chronic alcohol administration leads to changes in pancreatic exocrine secretion with development of precipitates within the pancreatic ducts. It is postulated that these precipitate may lead to ductal obstruction and inflammation. (Ranson ,1990)