AETIOLOGY AND MANAGEMENT OF OBSTRUCTIVE JAUNDICE

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THESIS

Submitted for the partial fulfilment of the Master degree in General Surgery

PRESENTED BY :

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Ain Shams University

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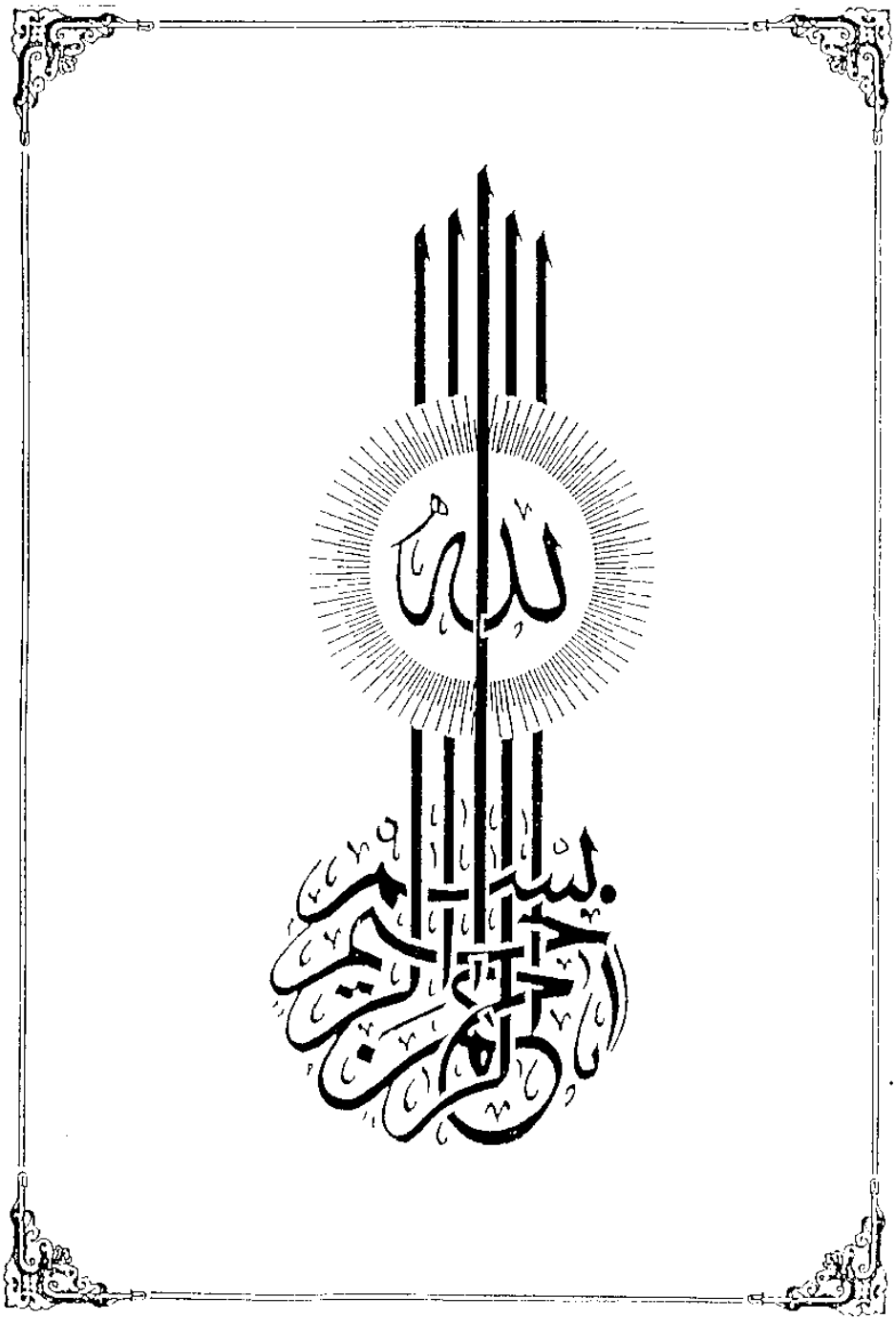

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Introduction

Time was when only two factors were necessary for jaundice before laparotomy: yellow skin and clinical suspicion of mechanical biliary obstruction. The simplicity of those days are gone. The evaluation of the advanced new tools of investigations mainly physical and radiological, has changed the situation from a matter of guess into an objective, mostly accurate way of solving the diagnostic problem.

It is not enough to diagnose patients with surgical jaundice accurately before laparotomy, but preoperative regimes now well recognized, must be meticulously followed if a promising outlook is sought in the prognosis of patients treated surgically.

The aim of this work is to study the different aetiological factors and up to date methods of diagnosis and treatment in patients with obstructive jaundice.

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EMBRYOLOGY & ANATOMY

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Embryology of the Biliary system:

The liver appears in the middle of the third week of intrauterine life as an outward growth of the endodermal epithelium at the distal end of the foregut called "The hepatic diverticulum" on the ventral aspect of the future duodenum.

The septum transversum here is immediately dorsal to the heart and contains paired vitelline viens medially and umbilical veins laterally (Craigmyle and Presley, 1975).

The hepatic diverticulum which consists of rapidly proliferating cell strands penetrate the septum transversum, and is formed of a large cranial portion (this forms the hepatic parenchyma and the intrahepatic bile ducts), and a small caudal portion designed to form the gall bladder.

The cranial part divides into right and left branches, from which columns of endodermal cells grow out into the vascular mesoderm of the septum transversum. These cells continue to grow and branch and anastomose with each other.

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The paired vitelline veins and umbilical veins that course the septum transversum become broken up by the invading columns of liver cells thus forming the liver sinusoids. The main hepatic duct and its right and left branches become canalized forming the hepatic ducts and the main hepatic duct. Further canalization of the intrahepatic duct system takes place, so that the duct system eventually joins the biliary capillaries. The liver cells start to secrete bile during the fifth month of development.

The candal part expands to form the gall bladder while the narrow stem remains as the cystic duct. It opens into the common hepatic duct (CHD) forming the common bile duct (CBD).

As the result of the positional changes of the duodenum, the enterance of the C.B.D. gradually shifts from its initial anterior position to a posterior one, and consequently the C.B.D is found passing behind the duodenum. The C.B.D and the main pancreatic duct join one another and pass obliquely through the wall of the second part duodenum, to open on the summit of the duodenal papilla, which is surrounded by the

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sphincter of Oddi. In about one third of individuals they pass separately through the duodenal wall, although in close contact, and open separately on the summit of the duodenal papilla. In a small number of cases the two ducts join and form a common dilation , the "Ampulla of Vater" opening on the summit of the duodenal papilla (Craigmyle, and Preseley, 1975).

Congenital Anomalies:

The gall bladder may be congenitally abscent, double, (where the hepatic bud bifurcates giving two gall bladders) septate or hour glass shapped fovouring gall stone formation. Normally about one third of the circumference of the gall bladder lies in a fossa on the under surface of the liver. In some cases it is entirely covered by serosa and connected to the liver by a mesentry giving a floating gall bladder. This facilitates cholecystectomy, but is liable to kinking of the cystic duct and twisting of the mesentry giving haemorrhagic infarction of the gall bladder. The phrygian cap appears in 2-6% of cholecystographies due to folding of the fundus of the gall bladder and may be mistaken for a pathological deformity of the organ.(Shields, 1977).

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Congenital atresia of the bile ducts may occur and jaundice appears soon after birth with clay coloured stools and very dark urine The liver is enlarged and dark green in colour. Surgical correction should be attempted as soon as possible, if not, the child will ultimately die of liver failure.

Congenital choledochus cyst results from a specific weakness in a part or the whole of the wall of the C.B.D. The distension rarely involves the gall bladder, cystic or hepatic ducts. The distended cyst may contain as much as 1-2 litres of bile, and may press on the C.B.D. causing obstructive jaundice accompanied by upper abdominal pain and pyrexia due to infection.

The cystic duct itself may be the seat of various anomalies, it may be short or absent, this may cause injury and latter stricture formation of the C.B.D after cholecystectomy. It may join the common hepatic duct at a low level, running parallel to it for a considerable length. Sometimes a sheath of strong connective tissue encircles them, and a stone impacted in the cystic duct may compress the common hepatic duct leading to obstructive jaundice. (Netter,1967).

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Surgical Anatomy of the Biliary System:

The right and left hepatic ducts emerge from the liver, and unite near the right end of the porta hepatis to become the 2-3 cm. long common hepatic duct which joins the cystic duct at an acute angle forming the common bile duct or ductus choledochus. It is 10-15 cm. long, 5-8 mm.wide and descends in the free margin of the lesser omentum. On its left is the hepatic artery and behind both is the portal vein. It continues behind the pars superior of the duodenum with the gastroduodenal artery on its left side. It then passes through the pancreas, in a groove on its posterior surface, infront of the inferior vena cava, in a downward and slightly rightward direction. It enters the medial side of the second part of the duodenum either separately or joins first the pancreatic duct.(Last, 1978). The C.B.D could be thus divided into a supraduodenal, retroduodenal, infraduodenal and an intraduodenal portion.

Hand, (1963) found that, throughout its course, the C.B.D is relatively wide with relatively thin walls, composed almost entirely of fibrous tissue and scanty

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muscle fibres. Some 2 mm. before the duct enters the duodenum, its lumen shows an abrupt narrowing as a result of a sudden increase in the thickness of its wall. This is due almost entirely to the appearance of copious, circular smooth muscle fibres. The duct retains its thick coat throughout its passage through the duodenal submucosa.

In the majority of cases Hand,(1963) found that the CBD is at its narrowest just before its junction with the pancreatic duct. The C.B.D. can thus be divided into 2 segments, a proximal, has a wide lumen and a thin fibrous wall, and a terminal segment, has a narrow lumen and a thick muscular wall. The junction between the proximal and the terminal segments is easily recognized macroscopically.

In radiologic studies of the C.B.D. it appears as a sudden narrowing of the duct. In the past, this sudden narrowing of the C.B.D has often been interpreted as a pathological stricture. A clear appreciation, that this abrupt narrowing represents normal anatomy is essential to the correct interpretation of any form of cholangiograms.

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The gall bladder is an elongated pear-shaped sac (vesica fellea). It is attached to the inferior surface of the right and quadrate lobes of the liver. It is about 10 cm. long, 3-5 cm. in diameter and about 30-50 ml. in capacity. Areolar tissue, in which blood vessles, lymphatics and nerves run, fills the gall bladder bed, otherwise, it is covered by peritoneum. The bulbous blind end, the fundus, projects a little beyond the sharp anterior margin of the liver and touches the parietal peritoneum of the anterior abdominal wall at the tip of the ninth costal cartilage, at the lateral border of the right rectus abdominus muscle. Posteriorly, the fundus is in relation with the transverse colon near its commencement.

The body is narrower than the fundus, and passes backwards and upwards towards the right end of the porta hepatis. The body is in contact with the second part of the duodenum and transverse colon. (Last, 1978). The infundibulum, or Hartmann's pouch, located at the free edge of the lesser omentum, is a dilation of the right wall of the neck and bulges forward towards the cystic duct,

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