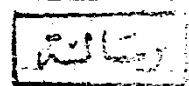


MANAGEMENT OF CHOLELITHIASIS

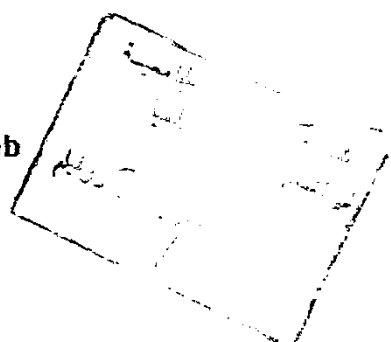
ESSAY

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INTRODUCTION

INTRODUCTION

The history of gall stone disease has ancient roots. Hippocrates described biliary symptoms in patient with pain in the right upper quadrant who eventually develop jaundice. In the 16th century, the first autopsy studies on patient with gallstone were done. cholelithiasis first became of practical importance to surgeons towards the end of the last century, following the first cholecystectomy by Carl Langenbuch in 1882.

Recently, it has been found that gallstone disease affects 6.4% of men and 10.4% of women between 30-39 years of age. Unfortunately, Knowledge of risk factors for gallstone formation is still anecdotal because of the lack of incidence studies. **(Luigi Barbara et al., 1991),**

There is a considerable progress in the understanding of the pathophysiology of both cholesterol and noncholesterol biliary stones has been made in the last decades. **(Enrico Roda. et al., 1991).**

Diagnosis of gallstone, often depends on radiographic data. The stone size, number and composition are critical for the most suitable technique for treatment. **(James Brink et al., 1991).**

Ultrasound has overtaken cholangiograph as the primary modality for the evaluation of the gall bladder and biliary tree stone disease with accuracies of 99% (Kathleen Reagan et al., 1991)

Surgical treatment of symptomatic gallstones has become institutionalized in modern medical practice. In recent years, however, alternative treatments have been developed, such as oral bile salt chemolytic therapy, endoscopic and percutaneous lithotomy in combination with contact solvent dissolution or with various direct fragmentation techniques, and extra-corporeal shock-wave lithotripsy (ESWL). (Vincenzo Speranza, 1991).

Laparoscopic cholecystectomy was first introduced in France in 1987 by Dubois et al. since then the procedure has changed our approach to gallbladder disease.

The aim of this essay is to review the whole subject, a special attention will be paid to the ongoing development and clinical use of the new technology and therapeutic modalities.

CHAPTER ONE

PATHOGENESIS OF GALL STONES

AETIOLOGY OF GALL STONES

Epidemiology :

The epidemiology of cholelithiasis is complex but it holds the key to the aetiology. Descriptive epidemiologic studies have demonstrated that the distribution of gallstones disease varies remarkably according to age, sex, and racial background. Geographic and time-trend analysis have suggested that variations in the occurrence and composition of gall stones may be related to factors such as diet or genetic predisposition. Analytic epidemiologic studies, which have systematically explored possible aetiologies of gallstones disease, have elaborated other possible risk factors for gallstones disease including parity, obesity, medical conditions and drug use. (Strom and west, 1985).

AGE :

Gallstones are rarely found under 10 years of age. The prevalence of gallstones among adults appears to increase with age, the peak occurrence of cholesterol stones is in the fourth decade of life, whereas the peak occurrence of pigment gallstones is in the eighth decade.

About 25% of children with gallstones have haemolytic disease, other possible predisposing factors are cystic

fibrosis, liver disease, bowel resection and heart disease. **(Henschke and Teele, 1983).**

Sex :

Cholesterol gallstones occur twice as frequently in females than males while pigment gallstones do not demonstrate a similar female preponderance.

Hormonal differences appear to account for the sex related incidence of gallstones. A number of studies have suggested that endogenous oestrogens and progesterones mediate this phenomenon through an effect on bile saturation and smooth muscle function of the gallbladder and intestine during the phases of menstrual cycle and pregnancy. **(Sali, 1990).**

Racial Background :

Gallstones are common among the white races, particularly in western Europe and North America. Pigment gallstones predominate in the Far East. **(Sali, 1990)**

Diet :

Several studies have noted an association between the increasing incidence of cholesterol gall bladder disease in Japan and a Westernisation of the traditional Japanese diet, that is, excessive consumption of highly purified carbohydrates and animal fats, together with a decreased

intake of dietary fiber and vegetable fats. However, because most gallstones are asymptomatic, we have no information on the dietary habits of patients without symptoms. **(Luigi Barbara et al., 1991).**

Genetics :

Families of patients with gallstones have an increased frequency of gallstones, irrespective of their age and weight. **(Gilat et al., 1983).**

Parity :

Multiparous women have a greater incidence of cholelithiasis than nulliparous women. Progesterone appears to cause relaxation and impaired emptying of the gallbladder. **(Sali, 1990).** Gallbladder relaxation and dilatation may progressively increase with each pregnancy resulting in increased gallbladder volume and diminished degree of emptying. **(Strom and West 1985).**

Obesity :

Incidence of gallstones in obese people has been estimated to be about three times that of the general population.

A number of studies have shown that a very high percentage of obese patients have bile supersaturated with cholesterol from an absolute and relative increase in

cholesterol in bile, without changes in bile acid secretion. **(Sali, 1990).**

Drugs :

There is some indication that various drugs raise the saturation of bile and hence the danger of gallstones. The principal substances are oral contraceptives and clofibrate-type derivatives.

a- Oral contraceptives :

There is a biologic basis for the relationship between gallstone formation and the use of oral contraceptives. It lies in the fact that oral contraceptives increase cholesterol secretion, decrease bile acid secretion, and hinder gall bladder motility.

b- Clofibrate:

A drug used to lower serum lipid levels, enhances the saturation of bile with cholesterol by increasing biliary secretion and decreasing bile acid synthesis. **(Luigi Barbara et al., 1991).**

Other illnesses :

Several disorders may predispose a patient to gallstone disease. Among these are diabetes, hyperlipidemia, ileal disease, liver cirrhosis and hemolytic anemia.

a- Diabetes :

The increased cholesterol saturation which occurs in the bile of diabetics is the consequence of associated obesity and hypertriglyceridaemia. Gallbladder atony due to autonomic neuropathy may favour stone formation in supersaturated bile.

b- Hyperlipidemia :

The presence of gallstone disease is associated positively with type IV and type II b hyperlipoproteinemia, both are characterized by an increase in the concentration of triglyceride-rich very-low density lipoproteins [VLDL]. In a cross-sectional study of patients with gallstone disease, there is an inverse relationship between gall bladder disease and high-density lipoprotein [HDL] level. A possible mechanism is that free cholesterol in HDL is metabolized preferentially to bile acids.

c- Ileal disease :

Cholelithiasis is more than four times as frequent in patients with a disorder of the terminal ileum. The biologic

explanation for this association is that patients with ileal disease and those with ileal resection have an increased loss of bile salts in the faeces and diminished bile-acid pool size (Luigi Barbara et al., 1991).

D- Haemolytic anaemias :

Haemolytic anaemias as well as patients with liver cirrhosis are associated with gallstone disease of the pigment type

A possible explanation is the increased production and biliary secretion of bilirubin which can induce gallstone formation. (Luigi Barbara et al., 1991). The gallstones in haemolytic disease tend to be asymptomatic. Ultrasonic screening of a population with haemolysis indicates that around 12% of two to four old children have stone and the prevalence rises to 42% in the 15-18 year old group (Sarnaik et al., 1980)