ABO BLOOD GROUPS AND GALL STONES IN EGYPTIANS

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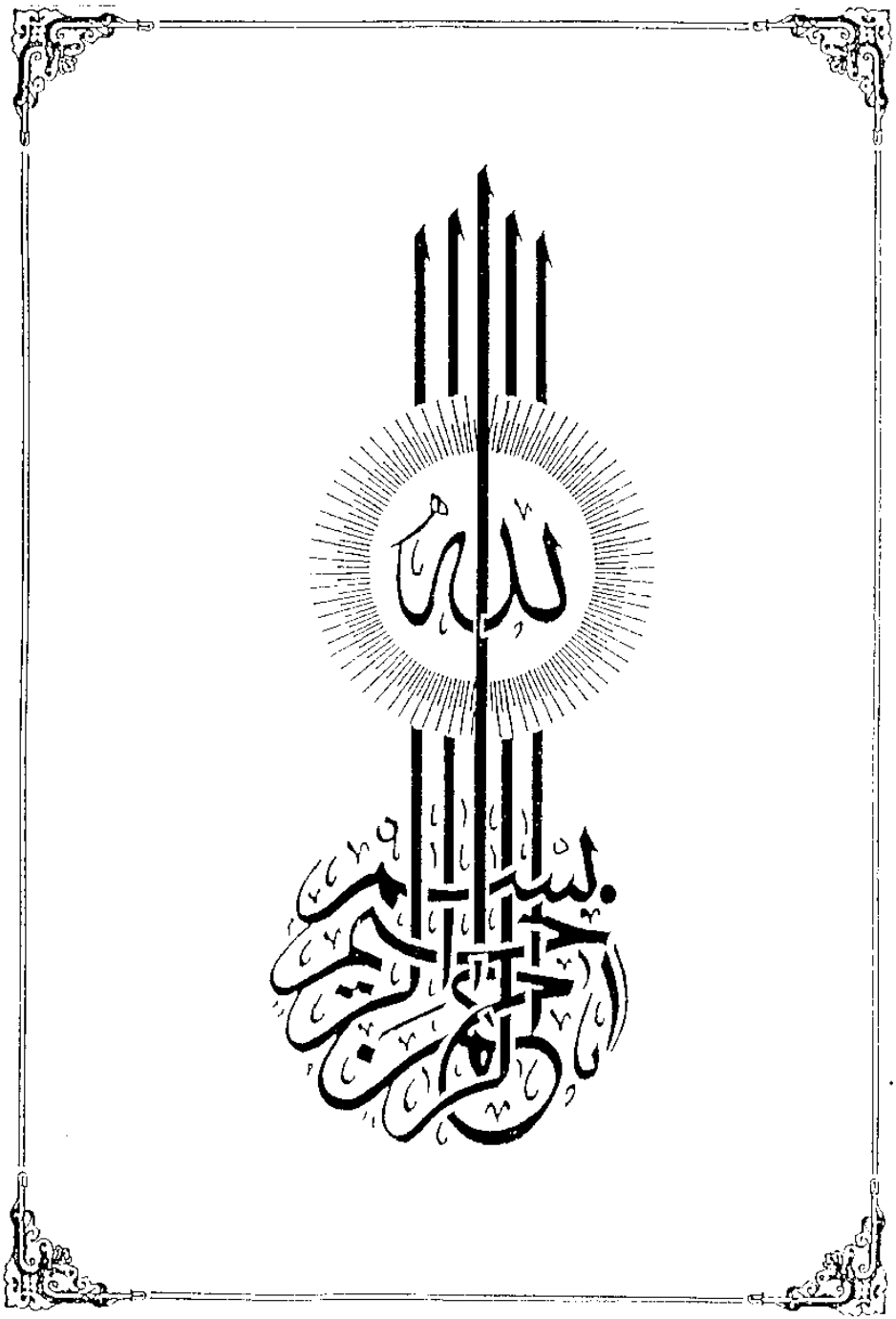

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Introduction:

ABO blood groups have been shown to be associated with various diseases such as duodenal ulcer, tumours of the stomach and pancreas as well as ischaemic heart diseases. ABO genes also seem to be differently distributed in various socioeconomic, geographical and ethnic groups. Epidemiological investigations into gall stone disease have shown that its incidence varies according to age, sex, racial background and geographical area, and other postulated factors include parity, obesity and diet.

ABO blood groups were also studied in metabolic processes including cholesterol metabolism, but the relation between ABO blood group and stone composition has not received.

Aim of the Work:

The aim of this study is to determine the relation between gallstones and ABO blood groups in Egyptians, taking into consideration the factors of obesity and hyperlipidemia.

REVIEW OF LITERATURE

PHYSIOLOGY OF THE BILIARY SYSTEM

Biliary Secretions:

Bile is secreted continuously by hepatic cells into the bile canaliculi, small bile ductules and large bile ducts. The interalobular bile ducts coalesce to form right and left hepatic ducts, which in turn, unite to form the common hepatic duct, which is joined by the cystic duct to form common bile duct ***(Samson, 1984)***.

Bile contains both secretory products important in digestion and excretory products such as bile pigments and cholesterol. It also contains hormones and drugs, which are either metabolized or conjugated within the hepatic cells ***(Arthur, 1985)***.

About one liter of bile is secreted each day. It is produced continuously, but between meals the contraction of sphincter of Oddi causes it to accumulate in the gall bladder (volume 30-50ml). In the gall bladder, an iso-osmotic electrolyte solution is reabsorbed leaving behind a concentrated solution of bile salts, bile pigments, lecithin and cholesterol. Bile is ejected by contraction of gall bladder and enters the duodenum after relaxation of the sphincter of Oddi ***(Jahn, 1986)***.

Enterohepatic Circulation of Bile Salts:

Approximately, 94% of the bile salts are reabsorbed by an active transport process through the intestinal mucosa in the distal ileum. They enter the portal blood and pass to the liver. On reaching the liver, these salts are absorbed almost totally on the first passage through the venous sinusoids into the hepatic cells and then resecreted into the bile. In this way, about 94% of all the bile salts are recirculated into the bile, so that on the average, these salts make the entire circuit some 18 times before being carried out in the faeces. The small quantities of bile salts lost into thefaeces are replaced by new amounts formed continuously by the liver cells. This circulation of the bile salts is called the enterohepatic circulation **Arthur, 1991)*.

The quantity of bile secreted by the liver each day is highly dependent on the availability of bile salts. The greater the quantity of bile salts in the enterohepatic circulation (usually a total of about 2.5 g), the greater the rate of bile secretion. Indeed, ingestion of an excess of bile salts can increase bile secretion by several hundred milliliters per day.

If a biliary fistula empties the bile salts to the exterior for several days to several weeks, so that they cannot be reabsorbed from the ileum, the liver increases

its production of bile salts as much as tenfold, which increases the rate of bile secretion approximately back to normal. This demonstrates that the daily rate of bile salt secretion is actively controlled by the availability (or lack of availability) of bile salts in the enterohepatic circulation ***(Arthur, 1991)***.

Constituents of Bile:

The composition of hepatic bile differs from that of gallbladder, which is more concentrated (table 1) ***(Arthur, 1991)***.

Table (1): The composition of hepatic and gallbladder bile ***(Arthur, 1991)***

	Liver Bile	Gallbladder Bile
Water	97.5 g/dl	92 g/dl
Bile Salts	1.1 g/dl	6 g/dl
Bilirubin	0.04 g/dl	0.3 g/dl
Cholesterol	0.1 g/dl	0.3-0.9 g/dl
Fatty Acids	0.12 g/dl	0.3-1.2 g/dl
Lecithin	0.04 g/dl	0.3 g/dl
Na^{++}	145 mEq/L	130 mEq/L
K^{+}	5 mEq/L	12 mEq/l
Ca^{++}	5 mEq/L	23 mEq/L
Cl^{-}	100 mEq/L	25 mEq/L
HCO_3^{-}	28mEq/L	10 mEq/L

Bile is a solution containing four primary ingredients, these include:

I. Bile salts:

These are synthesized by the liver cells. Choleic acid is formed from cholesterol and the acid side chain is conjugated either with taurine or glycine to form taurocholic acid and glycocholic acid respectively.

At the pH of bile (7.3-7.7), taurocholate and glycocholate exist as anions, but they do not contribute to the osmotic pressure of bile despite their concentration (10-20 mmol/L ***(Arthur, 1985)***.

Arthur (1985) also stated that, the bile salts together with some cations form aggregates, which are osmotically inactive and that bile salts which enter the duodenum are reabsorbed into the portal vein and return to the liver, the so called enterohepatic circulation.

Function of Bile Salts:

The hydrotropic action of bile salts, that is their characteristic property of lowering the surface tension of aqueous solution and therefore, allowing the formation of stable solution or emulsion of many fatty materials is due to the

structure of the bile salts molecule, being at one side hydrophilic (water attracting) and the opposite side hydrophobic (water repelling or lipid attracting) ***(Arthur, 1985)***.

II. Electrolytes:

Sodium, potassium and calcium are the main cations. Chloride and bicarbonate are the important anions ***(Jahn, 1986)***.

III. Cholesterol:

The biliary content of cholesterol is 0.6-0.7 g/l. The blood concentration of cholesterol is 0.2 g/L. The concentration of bile which occurs in the gallbladder may lead to further rise in the cholesterol concentration, and thus to its crystallization ***(Samson, 1984)***.

IV. Bile Pigments:

The major bile pigment is bilirubin, a breakdown product of the haem protein of haemoglobin, which is formed and released into the blood when macrophages destroy red blood cells. The cells of the liver extract bilirubin from the plasma and secrete it into the bile by an active process. After entering the intestinal tract via

bile, the bile pigments which are yellow, are modified by bacterial enzymes to form the pigments which give the faeces their brown colour.

Some of these yellow pigments are absorbed into plasma during their passage through the intestinal tract and are entering their passage through the intestinal tract and are eventually extracted in urine giving it yellow colour ***(Arthur, 1985)***.

Regulation of Biliary Secretion:

When food enters the mouth, the resistance of sphincter of Oddi decreases. Fatty acids in the duodenum release cholecystokinin, which causes gallbladder contraction. Acids, the products of protein digestion and calcium ions also stimulate the secretion of cholecystokinin.

The production of bile is increased by vagal stimulation and by hormone secretion, which increases the water and bicarbonate content of bile.

The bile salts themselves are among the most important physiological choleretics ***(Ganong, 1985)***.

LIPID METABOLISM

Function of Lipids:

1. Supply 20-25% of daily caloric requirements.
2. Essential fatty acids and fat soluble vitamins.
3. Render food palatable.
4. Preserve body temperature.
5. Support of internal organs.

Digestion and Absorption of Lipids:

The bile salts in the duodenum assist in the emulsification of triglycerides and stabilize the emulsion formed.

Pancreatic lipase hydrolyses triglycerides into free fatty acids and monoglycerides.

With the aid of bile salts, fatty acids and monoglycerides are dispersed into smaller particles known as micelles, which are acted upon by intestinal lipase and liberate glycerol and free fatty acids. Glycerol carried by portal blood to the liver,