

CHOLEDOCHOLITHIASIS

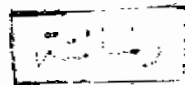
ESSAY

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TO MY MOTHER

IF THERE WAS EVER LOVE IN THE WORLD
I LOVE HER



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INTRODUCTION

Stone in the common bile duct is one of the most common and most serious complication of gall stones . The great majority of stones present in the common duct originally formed in the gall bladder, later passing down the cystic duct into the common duct. Usually patients with a stone, or stones, in the common duct also have calculi in the gall bladder ; however, such is not always the case, although in these latter patients the gall bladder almost invariably shows chronic inflammatory changes, suggesting that it previously harboured stones.

Stones or soft concretions of biliary mud may form in the bile duct in the presence of stasis and infection. The incidence of such primary duct stones is possibly higher than has been previously thought, and the mechanisms by which such concretions develop has been reviewed by Scott (1971). Stones can also form in the bile ducts in cases of suppurative cholangitis seen in certain Asian countries. In this latter condition, the stone formation has been attributed to infestation of the bile ducts with the parasite *clonorchis sinensis*. This parasite is not always present and infection of the ducts with *Escherichia coli* or enterococci may be the critical cause of the ductal stone formation.

In a more recent study, King (1971) was only able to isolate *C. sinensis* in one of 44 patients with oriental cholangitis, and he also emphasizes that the clinical and pathological features of this condition differs from those of the usual Western type of gall stone disease. Stones in the common duct may be single or multiple, 100 or more sometimes being present in a dilated duct. Very commonly the origin of the stones in the gall bladder is shown by their facettted shape. From the narrow calibre of the cystic duct, it is evident that, in the absence of fistula formation, only relatively small stones can pass into the common duct but, once in this duct, they may enlarge as the result of the deposition of soft pigment and debris on their surface. When this occurs, the stones may become barrel-shaped and mutually congruent. On section, such stones will usually be found to contain in their cetnres a small, hard, and often facettted calculus, betraying their origin from the gall bladder.

INCIDENCE :

The incidence of a stone in the common duct is difficult to determine with accuracy, partly because many such stones do not give rise to symptoms ; At operation,

stones in the common duct may be overlooked ; and comparison of reported series of patients may be misleading owing to the differing composition of the various series. This latter point is particularly important in relation to the age of the patient ; in any, as there is evidence that the incidence of stones in the common duct increases with age (Appleman, Priestley, and Gage, 1964). Stones are present in the common duct in 10 to 15% of all patients subjected to cholecystectomy for gall stones.

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AETIOPATHOLOGY

AETIOLOGY

In 1970 Small, in an attempt to clarify how cholesterol gall stone form, define abnormal bile as bile that contains excess cholesterol, with time give up it as cholesterol crystals. He suggested that abnormal bile production is the first process in production of cholesterol stones.

Admirant and Small (1968) have shown that solubility of cholesterol depends only on the relative concentration of bile salt. The solubility of cholesterol in various aqueous mixtures of bile salt, and lecithin is best represented on the triangular coordinate (Small, 1968).

The line 10% represent the solubility of cholesterol in lecithin in bile salt micelles in aqueous system containing 10% solid and 90% water. The line falling below refers to the calculated solubility of cholesterol in more diluted solution containing 3%, 2%, 1.5% and 1% total solid. The curve has been calculated assuming a critical micellar concentration for the conjugated bile salt mixture in bile of 2 mM (Small, 1968).

Theoretically there would be no solubilization of cholesterol below the critical micellar concentration of bile salt. Small, (1970) redefined an abnormal bile as one which, depending on its relative concentration of lecithin, bile salt and cholesterol, falls above the appropriate line in Fig. (A).

The bile contains 70% bile salt and 10% lecithin (point P) falls in the zone of abnormal bile, that is, it contains more cholesterol than can be held in solution under equilibrium condition. This bile could be present in at least 2 physical states. It could be a mixture of micellar phase saturated with cholesterol and a solid phase of cholesterol crystal.

However, bile of 5% cholesterol, 15% lecithin, 80% bile salt (point R) would be a stable micellar phase even in diluted micellar solution (containing 99% water). The production of abnormal bile was the problem that suggested by Small (1970) to be by liver and gall bladder.

Role of Liver in the Production of Abnormal Bile :

Liver secretes bile saturated with cholesterol in subjects with cholesterol stones (Small and Vlachovic, 1970).

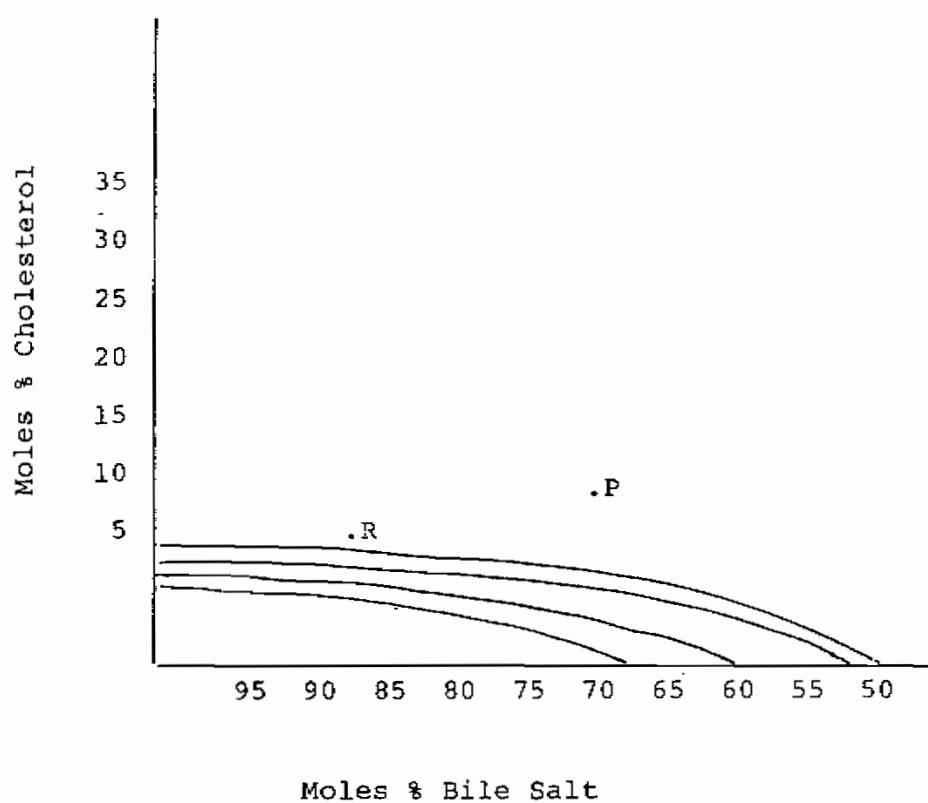
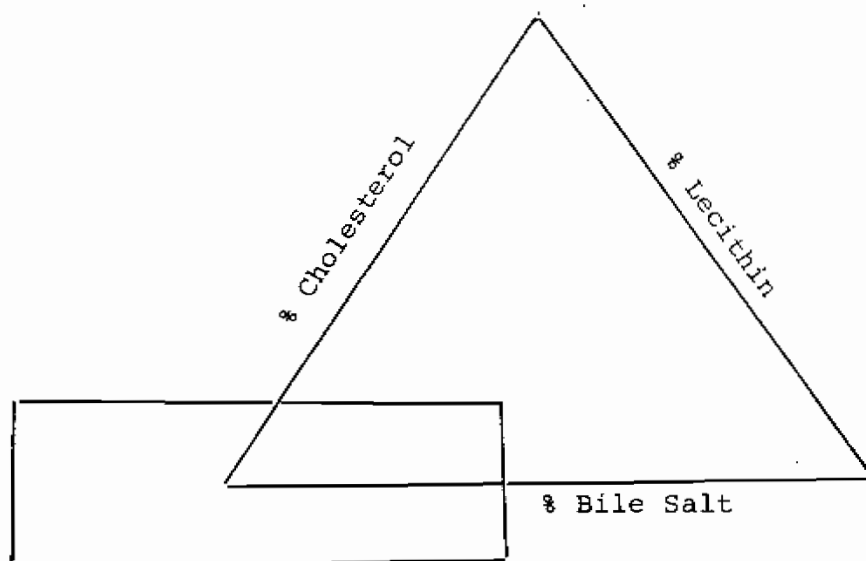


Fig. (A)

Normal subject, at least part of time, do the same (Northfield et al. and Metzger et al., 1973).

Dam and Co-workers (1964) and Shioda (1965) have been concluded that free diet used to form gall stone in young hamsters not only cause liver abnormalities but also formation of abnormal hepatic bile supersaturated with cholesterol. Since cholecystectomy tends to prevent stone formation in these animals, a responsible assumption that micelles supersaturated with cholesterol secreted by the liver are nucleated in gall bladder and precipitate the excess cholesterol in that organ to form gall bladder stones.

Small and Rapo (1970) proved the occurrence of abnormal bile in hepatic bile collected at time of gall bladder surgery for American Indian. Thus if liver is responsible for making abnormal bile than bile coming directly from the liver would be expected to have abnormal composition before entering the gall bladder.

The mechanisms possible for the formation of abnormal liver bile:

Bile salts are excreted by hepatocyte while bile lecithin comes from a different pool than plasma lecithin (Balint et al., 1967).

Small (1970) assumed that the lecithin and cholesterol found in bile came from these canaliculus and cholangioles membranes. Swell et al. (1968) strongly suggested that neither microsomes nor mitochondria are the sources. Using the figure of Wheeler (1968) for total canalicular surface and that of Dowling et al., (1968) for daily output of phospholipid and cholesterol, One can calculate that the canalicular membrane must turnover, and this turn-over is related to the amount of bile salt returning to the liver through enterohepatic circulation. The more bile salt returning, the greater the output of phospholipid and cholesterol (Hardison and Francis et al., 1969) and the ratio of cholesterol to lecithin at high or low bile salt is constant. These suggest that both lipid come from the same place , and bile salts cause their excretion by physical process . (Small, 1970).

Small, (1970) suggested that bile salt penetrate the membrane from the interior of the cell and dissect out specifically lecithin and cholesterol leaving the membrane protein and other structural lipid.

How might abnormal hepatic bile be produced ?