“LIMY BILE” — A CASE REPORT

C. R. THAMBI DORAI

SUMMARY

A case of limy bile, i.e. bile in the gall bladder rendered radio-opaque due to excessive concentration of calcium carbonate, is reported and its pathogenesis discussed.

INTRODUCTION

Limity bile is a condition where calcium, usually in the form of carbonates and rarely as phosphates and bilirubinates, accumulates in the gall bladder in excess quantities, rendering it opaque to X-rays.1 The consistency of bile may vary from fluid to a putty-like mass or even solid, and the colour from white through yellow to brown, depending on the quantity of bile pigments.

CASE HISTORY

A 44-year-old Malay female was referred from a district hospital with upper abdominal pain of two years duration. The pain was dull aching, periodic and felt in the epigastrium and right hypochondrium. There were no aggravating or relieving factors. The pain had progressively increased in severity over a two-year period. There was no history of jaundice or symptoms suggestive of acute infection. Past history did not indicate any significant illness.

Clinical examination revealed an obese lady who was afebrile and not jaundiced. There was no tenderness in the abdomen. Liver, gall bladder and spleen were not palpable. The basic investigations and liver function tests were normal.

The urinary calcium excretion on a normal diet was 180 mg in 24 hours (30 – 250). Total serum cholestrol was 3.8m mol/L (3.6 – 7.3). A plain X-ray of the abdomen showed over the right upper quadrant a pear-shaped opaque object with multiple filling defects within it (Fig. 1). Intravenous cholangiogram did not show any increase in gall bladder opacification compared to the plain X-ray. The main hepatic ducts and common bile duct were visualised and normal. The dye was seen in the duodenum. Ultrasound examination of the abdomen showed that the gall bladder area was full of echoes with acoustic shadowing behind.

At laparotomy, the gall bladder was found to be slightly enlarged and tense. The greater omentum was adherent over its fundus. The wall of the gall bladder was thickened. The cystic duct was long and narrow. The gall bladder was filled with pultaceous material and some stones could also be felt within it. A large stone was felt in the neck region. This stone was mobile but could not be pushed back into the body of the gall bladder, suggesting partial obstruction of the outlet, also accounting for the tenseness of the gall bladder. The common bile duct was normal in calibre and no stones were felt within it. The liver, pancreas and other viscera were normal. Cholecystectomy was done, and the operation was completed after ascertaining the patency of the common bile duct.

The specimen of gall bladder showed the wall to be thickened. The thickening was most pronounced over the neck producing narrowing of the lumen and revealed a stone 1 cm in diameter in its lumen. The gall bladder was filled with pasty material of greyish white colour (Fig. 2). There were many calculi within the pasty material. The
Photography of plain abdominal X-ray. (The radio-opaque gall bladder in X-ray appears black in photograph, with filling defects in it due to the stones.)

surface of the stones was covered with greyish white material while the core was more blackish.

The post-operative course was uneventful. Histopathological examination of the gall bladder revealed chronic cholecystitis. There were no calcified deposits in the gall bladder wall. The limy bile was alkaline in reaction and contained calcium carbonate with cholesterol and bile pigments.

DISCUSSION

The pathogenesis of limy bile is not definite. Partial or complete obstruction to cystic duct, long cystic duct with stasis, superadded infection, abnormalities in calcium metabolism, abnormal alkaline pH in gall bladder, insoluble calcium complexes formed in gall bladder (due to abnormalities in bile salts secondary to gall bladder disease) have been suggested as possible causes.

Calcium carbonate is known to crystallise from a solution when the pH is above 6.6. The mean pH of normal hepatic bile is 8 and that of the gall bladder is 7.3, with a range of 6.5 to 9. As the pH of the bile is therefore always greater than 6.6 in a normal biliary system, alkaline pH alone is unlikely to be the reason for limy bile formation.

There is no characteristic symptomatology. Most cases present with abdominal discomfort or pain.

The limy bile is usually confined to the gall bladder. Jaundice is uncommon but can occur when the limy bile gets into the common bile duct. This passage of the limy bile from gall bladder, where it is formed, into the common bile duct explains the occurrence of documented cases of spontaneous disappearance of limy bile in serial X-rays.

Limy bile is to be differentiated from "porcelain gall bladder". In the latter condition, there is calcification of the gall bladder wall and is a late result of chronic fibrous cholecystitis.

The rarity of the limy bile can be judged from the fact, that till 1972 only 70 cases had been reported in the literature. From the University Hospital, Kuala Lumpur, only one case was recorded out of 745 cholecystectomies, done over a period of 15 years from 1967.

The diagnosis of limy bile can be made on plain radiograph of the abdomen. Ultrasonogram is supplementary. Cholecystogram is not necessary. Cholecystogram may be harmful as it may be responsible for limy bile moving into the common bile duct.

CONCLUSION

In the case reported there was no evidence of abnormal calcium metabolism. Liver function tests were within normal limits. pH as explained earlier, may not be a very important factor. Probably alterations in bile salt binding capacity secondary to cholecystitis, along with partial...
obstruction to the outlet might have contributed to limy bile formation in this case.

ACKNOWLEDGEMENT

The author wishes to thank Tan Sri Datuk Dr Abdul Khalid bin Sahan, the Director General of Health Services, Malaysia for permission to publish the case report.

REFERENCES