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SEVERE HYPERBILIRUBINEMIA DUE TO BENIGN OBSTRUCTION OF THE COMMON BILE DUCT

CASE REPORT

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Icterus always presents an interesting problem of differential diagnosis. The "usual task is to determine whether jaundice is due to primary liver disease or to obstruction of the extrahepatic bile ducts by stone or tumor"[1]. The patient in this case report presented an unusual problem because of a remarkably high total serum bilirubin of 53 mg. per 100 cc. Bockus[2] states that the bilirubin rarely exceeds 10 mg. per 100 cc. of serum if the jaundice is associated with gallstones. When a neoplasm causes biliary obstruction, the bilirubinemia rises steadily to between 20 and 30 mg. per 100 cc. in most patients. In fatal viral hepatitis the jaundice may be extreme. The serum bilirubin concentration in our patient was the highest we have seen from any cause. Inasmuch as the etiology was benign, we are presenting this case report as an interesting exercise in differential diagnosis.

CASE REPORT

A 56 year old negro male was admitted to the hospital in October, 1956 with jaundice of four days duration. He had been seen regularly as an outpatient for fifteen years. In 1946, he had been hospitalized during an episode of transient jaundice with a provisional diagnosis of viral hepatitis. A review of the 1946 record does not confirm the etiology of his icterus at that time. He had been anorexic with chills and fever to 103 defrees Fahrenheit. He had been under treatment for prostatitis and was an alcoholic. In 1946, his cephalin cholesterol flocculation was four plus, thymol turbidity 4 units, and thymol flocculation negative. The leucocyte count was 7,900 per cu. ml., with 95 per cent polymorphonuclears. Cold agglutination determinations had been negative. His icteric index had elevated to 15 and the bromsulfalein retention was 19 per cent in forty-five minutes. The oral hippuric acid test was normal.

During the following ten years the patient had annual liver function studies. The forty-five minute bromsulfalein retention varied from 6 to 12 per cent. The cephalin cholesterol flocculation remained negative. The thymol turbidity was 8 units in 1949, but usually was below 4 units; and the thymol flocculation varied from negative to four plus. Abnormal liver function tests during this ten year period seemed to correlate with his intemperate use of alcohol. A right inguinal herniorrhaphy was performed in 1951, at which time there was no hepatomegaly.

One day prior to the present admission, the patient had been seen as an outpatient complaining of nausea and vomiting. The gallbladder was palpable and tender, and his sclerae were icteric. There had been a recent alcoholic debauch. He had not taken any medications and there had been no transfusions or needle injections. After the initial examination the gallbladder was not palpable, but the liver was felt 3 cm. below the right costal margin. The patient was able to eat most of his food, but his

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appetite was easily satiated. He had no chills and no fever.

The initial laboratory studies revealed a total serum bilirubin of 7 mg. per 100 cc. with a direct reacting bilirubin of 4.2 mg. per 100 cc. The urinanalysis was within normal limits. The hemoglobin was 14.1 gm. per 100 cc., leucocyte count 8,250 per cu. ml., polymorpho-neutrophiles 74, eosinophiles 4, lymphocytes 19, monocytes 3. The serum cholesterol was 222 mg. per 100 cc., with a 38 per cent ester fraction. The fasting blood sugar was 100 mg. per 100 cc., non-protein nitrogen 34 mg. per 100 cc. The serum alkaline phosphatase was 7 units and the prothrombin time 15 seconds (100 per cent). The transaminase was 22 and the serum iron 164 micrograms. His cephalin cholesterol was four plus, thymol turbidity 2 units, and the thymol flocculation one plus. The patient's stool gave a strong reaction for bile by the Schmidt test. In the transduodenal biliary drainage, microscopic stones and calcium bilirubinate pigment were seen in the "B" bile fraction. The "A" and "C" bile fractions were clear. His fasting gastric acidity was 8 degrees; the maximum free acidity being 64 degrees after a stimulating test meal.

During the first ten days in the hospital, the patient's bilirubin decreased to 5 mg. per 100 cc. The bile reaction in the stool remained strongly positive. A needle biopsy of the liver was done on the seventh hospital day. This revealed bile stasis with acute and chronic cholangitis. On the eleventh hospital day the patient was more icteric and the serum bilirubin had elevated to 31.6 mg. per 100 cc. with a direct reacting bilirubin of 15.6 mg, per 100 cc. The serum cholesterol was 262 mg. per 100 cc., and the alkaline phosphatase had risen to 26.4 units. The thymol turbidity had increased to 8 units. The quantitative urinary urobilinogen, which had been 0.8 units on admission, had now become elevated to 5.8 units per 100 cc.

The patient's icterus increased and on the twentieth hospital day the stool became negative for the presence of bile for the first time. The serum bilirubin was remarkably high. By dilution techniques it was determined to be 53 mg. per 100 cc. with 32 mg. of direct reacting bilirubin. At this time no bile was obtained by transduodenal drainage. The alkaline phosphatase was 31.8 units. A sickle test was negative. The leucocyte count was 12,450 per cu. ml., with 82 per cent polymorpho-neutrophiles. By serum electrophoresis the total protein was 7.2 gm. per 100 cc., gamma globulin 22, betta globulin 24, alpha 1 globulin 10, alpha 2 globulin 6, and serum albumin 39 per cent.

No calcification had been seen in a scout film of the abdomen at the time of admission. A roentgenologic study of the stomach and duodenal loop had been normal.

The differential diagnosis had included the possibility of hepatitis during the first three weeks of observation. Although the patient's liver flocculation studies were not greatly abnormal, the serum iron had elevated and the urinary urobilogen was increased. After the elevation of serum alkaline phosphatase and a rise in temperature to 100.5 degrees Fahrenheit, indicating the presence of obstructive jaundice with cholangitis, the need for surgery was evident.

Surgery was carried out on the twenty-first day. The liver was enlarged and deeply pigmented, but not necrotic. The omentum was adherent to the gallbladder, which showed mild distention. The common bile duct was dilated, and a stone could be felt

within its lumen. The duct was then opened, and was found to be filled with thick, inspissated bile. One stone approximately 1 cm. in diameter, and a number of very small stones were removed from the lower end of the common bile duct. The sphincter of Oddi was very stenotic. It was dilated with dilators to seven millimeters. The bile ducts were then irrigated thoroughly and golden bile appeared from the liver. There was no evidence of a malignant lesion in the pancreas or the ampullary region of the common bile duct. The gallbladder was opened and thoroughly irrigated. It did not contain stones, although it appeared that several small stones were flushed from the cystic duct. A T-tube was placed in the common bile duct, and a cholecystojejunostomy was performed.

The patient's postoperative convalescence was satisfactory. The jaundice cleared slowly. Cholangiograms made via the T-tube showed no abnormalities, other than the residual dilatation of the common bile duct. The media flowed freely into the duodenum. The T-tube was removed and the patient has remained well. There has been no icterus during the past year.

DISCUSSION

Early in the patient's illness the differentiation of obstructive jaundice from hepatocellular disease was not obvious. A prior viral hepatitis was not clearly proved. The normal bile reaction in the stool, a normal quantitative urinary urobilinogen, and absence of a alkaline phosphatase retention did not conform to the criteria for extrahepatic obstruction. The positive flocculation tests, the elevated serum iron, and the rise in urinary urobilinogen prevented an early exclusion of viral hepatitis. An improvement in the icterus during the first week of illness gave caution to surgical intervention. The later excessive elevation in serum bilirubin raised doubts of a possible benign etiology. Considerations of the pathogenesis and final diagnosis in this patient led to interesting speculations.

There are several factors to consider in evaluating hyperbilirubinamia. First, it must be recalled that bile pigment is formed from the disintegration of erythrocytes and more specifically the breakdown of hemoglobin by the reticulo-endothelial system[3,4]. The bile pigment, therefore, is formed largely outside the liver[5]. The degree of bilirubinemia will depend for the most part on:

1. The rate and degree of hemoglobin breakdown.
2. The urinary excretion rate of bilirubin. The normal renal threshold for bilirubin retention in serum is 1.5 to 2.0 mg. per 100 cc.
3. The degree of severity of liver damage or bile duct obstruction causing diminished excretion of bilirubin.

Thus, in complete obstruction of the extrahepatic bile ducts, the intensity of jaundice depends on the degree of hemoglobin wastage and rate of excretion of bile in the urine[6]. With loss of blood the intensity of icterus may decrease; but if the rate of blood destruction increases, as by hemolytic agents, icterus increases. An equilibrium between these various factors is reached and the level of hyperbilirubinemia stabilizes.

Extremely high levels of serum hilirubin have been reported in a few patients with liver atrophy or complete obstruction of the bile ducts. Kuwayte and associates[7] reported a total serum bilirubin of 152.7 mg. per 100 cc. in a patient having a carcinoma at the juncture of the cystic and common hepatic ducts. Bollman[8] has shown two

pigments that react directly with the van den Bergh reagent. One pigment predominates after uncomplicated obstruction of the bile duct. The second pigment appears in larger amounts if biliary obstruction continues. This is due probably to failure of the conversion of the first pigment to the second type if the hepatic cells are diseased. The high value of urinary uroblinogen in our patient indicates some impairment of the excretory function of the liver cells.

The complete biliary obstruction which occurred in the patient presented is thought to have been due to several causes: fibrosis of the sphincter of Oddi, stones in the common bile duct, and inspissation of bile in the bile ducts. Brush, et al[9,10] have emphasized organic structure of the sphincter of Oddi as a primary cause of the statsis and infection which leads to formation of biliary calculi. The sudden rise of the serum bilirubin to an unusually high level may have occurred when the bile in the ducts thickened or an inflammation caused edema of the common bile duct. Prior to the sudden rise, the serum bilirubin level was approximately that expected for obstruction due to stones in the bile ducts. To speculate further, an increase in hemoglobin breakdown may have occurred. Hanger[11] has outlined many of the baffling problems related to obstructive jaundice. Why serum bilirubin does not continue to rise with complete obstructive jaundice is not explained by its elimination through the kidneys. The extremely high serum bilirubin concentration in this patient serves to emphasize the need for further experimental investigation to understand the fate of bilirubin and its derivatives.

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