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A 45 Year Old Male with Jaundice

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Mr. A. R. 45 years male, admitted on 10-11-86 with complaints of:-

Yellow discoloration of sclerae	8 weeks
Loss of appetite	7 weeks
Loss of weight	6 weeks
Fever, low grade	3 weeks
Nausea/vomiting	2 weeks

Past History

No blood transfusion, injections, or childhood history of jaundice.

Family Hsitory

Mother died of "Blood cancer"

Personal History

No history of alcohol abuse. Smoker for 20 years.

Drugs on admission

Deltacortil 5 mg 1 t.d.s.
Litrison tablets 1 b.i.d.

He gave no history of malena or hematemesis. His bowel habits were regular. His urine had turned dark brown since the onset of jaundice and his stools had been pale intermittently.

His weight loss had not been documented, though he claimed to have lost considerable weight. (10 to 20 lbs in 2 months).

On Examination

Intelligent, cooperative with some abdominal distension.

BP 100/60 mg H.g., RR 18/minute, Pulse 100/minute, Temp, 98.6°F JVP not elevated, no lymphadenopathy, asterixis, erythema. jaundice present, Spider angioma none.

G.I. Tract

Abdomen: Umbilicus was central, no fullness of flanks or visible veins, soft on palpation. Liver span was 17 cm in midclavicular line. Spleen normal, ascites was negative, bowel sounds audible.

CVS and respiratory system were unremarkable.

Central nervous system: Well oriented, normal speech, no sensory or motor deficit. No cranial nerve palsies; no neck rigidity.

Lab review on admission

Hb 9.7G, W B C 14,109/cu mm, polymorph 85 % lymphos-11 % Na 135 mcq/I, K 2.7 meq/I FBS 87 mg %, SGPT 168 (5-30)IU, SGOT 330 (5-40)IU, ESR 76 mm platelets 600,000. PT 15/15 sec, APTT 30/36 sec, Alk. Phos. 174(20-40) U/I, total bilirubin 20 mg, conjugated 10 mg, unconjugated 10 mg, albumin 2.3 g (3-5.5)/dl, stools positive occult blood, HBs Ag negative.

The lab review suggested a conjugated cholestatic hyperbilirubinemia. An ultrasound of abdomen was done to distinguish between intrahepatic cholestasis and extrahepatic obstruction.

The abdominal ultrasound read as follows

Liver enlarged 7.2 cm below right costal margin, no focal lesion seen but gall bladder distended with dilated intra hepatic and common bile ducts. Unfortunately lower end of common bile duct and pancreas was not seen clearly

due to bowel gas. No definite pancreatic lesion seen. No calculus in biliary tree, no mass or ascites seen.

The ultrasound had shown that there was some form of mechanical obstruction. However, the site and nature of obstruction is indeed valuable to a surgeon contemplating biliary surgery and therefore we decided to go ahead with either a PTC or ERCP.

The choice between a PTC and ERCP is determined largely by the following features:-

	PTC	ERCP
Technique	Easy	Difficult
Time taken	30 minutes	30-50 minutes
Anatomical difficulties	Few	Many
Cost	Low	High
Complications	5 %	5 %
Complications, a	bile Leakage	Pancreatitis
b,	Cholangitis	Cholangitis
Success		
Overall	95 %	75% to 85%
In dilated ducts	100 %	80%
Non dilated ducts	60 %	80%
Pancreatic ducts	0 %	80 %

Our inclination was to do a PTC. His P.T. was 19 seconds against control of 15 seconds two days before the PTC. He was given VIT. K therapy. On 7th November 1986 the day of the PTC, we gave him 32 units of fresh frozen plasma and went ahead with the procedure. Before going on to the description, let us discuss the subject of jaundice.

Jaundice is simply divided into:-

Heptocellular	1. Acute
	2. Chronic
Cholestatic	1. Dilated ducts
	2. Undilated ducts

– Hemolytic
– Non-hemolytic familial hper-bilirubinemia. The familial non-hemolytic hyperbilirubinemias are as follows :-

Unconjugated

1. Gilbert's Disease: (Familial) S. bilirubin increases with fasting and falls with phenobarbital. Liver biopsy is normal but conjugating enzyme glucoronyl transferase is reduced.

2. **Crigler-Najjar:**

Type 1. No. conjugating enzyme in liver. No response to phenobarbital. Usually die young with kernicterus.

Type 2. Deficient conjugating enzyme in liver. Response to phenobarbital is good.

Conjugated: characteristic features.

1. **Dubin-Johnson Syndrome**

Black tissue on liver biopsy. Non-visualization of gallbladder on cholecystography. Secondary rise in BSP test is seen.

2. **Rotor Syndrome**

Normal liver biopsy, cholecystography is normal, BSP test is abnormal.

1. **Pre-hepatic Jaundice**

Increased serum bilirubin (unconjugated; normal serum transaminases, alkaline phosphatase and proteins. The circulating serum bilirubin is unconjugated. No bilirubin in urine.

Reticulocytosis, positive coomb's test or splenomegaly are present.

2. **Hepatocellular:**

Increased serum bilirubin (conjugated & unconjugated, increased serum transaminases, prolonged PT; in chronic cases low albumin, varying degree of liver failure.

3. **Cholestatic**

Increased serum bilirubin (conjugated), raised biliary alkaline phosphatase; increased cholesterol, pale stools, pruritus, increased skin pigmentation, osteomalacia, reversal of prolonged PT after vit K 10 mg 1.M. daily for three days. Information that is essential in establishing the case of jaundice is as follows :-

1. **Serum Biochemistry:**

SGPT, SGOT, alkaline phosphatase, total bilirubin, conjugated bilirubin, globulin, albumin; A/G ratio.

2. CBC, Hb, platelets, WBC, reticulocytosis, PT (before and after vit. K injection. Blood film (target, spur cells, micro angiopathic hemolytic anemia).

3. Urine/stools examination

4. X-ray chest & abdomen.

5. Oral cholecystography and i.v. cholangiography are mentioned here to discredit them in the setting of jaundice. They fail to visualize the biliary system when the bilirubin is 2.5-3 mg and carry a real risk of precipitating acute renal shutdown.

6. Abdominal Ultrasound.

7. Percutaneous Trans-hepatic Cholangiography (PTC).

8. Endoscopic retrograde cholangio-pancreatography. (ERCP).

9. HIDA scan.

In context of our patient, we planned to do PTC on him which was done without any problem. The injected dye clearly outlined the intrahepatic biliary radicles, the common hepatic duct, cystic duct, gallbladder and part of the common bile duct. There was a sharp cut-off point in the CBD below which the dye would not be visualized.

PTC had still not identified the site and the cause of the lesion. It was imperative, that the whole biliary system be visualized before a confident diagnosis could be reached as to the cause of the obstruction.

The ERCP was done next day. This showed a normal ampullary region and a normal 2.5 cm of CBD with a stricture above it (1.5 cm) and then the dye spilled into the dilated upper portion of the CBD.

The ERCP had indeed been rewarding, it clearly mapped out the architecture as the size of the obstruction and determined its length. A stone could not be seen. In the absence of previous biliary surgery (as was the case in our patient), this almost certainly meant a malignant stricture.

Patient was shifted to the surgical floor on 18th November, 1986. He had developed some malena and abdominal distension. He was given symptomatic treatment (blood transfusion, fluids, potassium supplements).

Cholecystostomy was done on 20th November, 1986. Biopsy was taken from the omentum. He did well for the first post-operative day. He then developed abdominal distension absolute constipation and his bowel sounds were absent. He also developed respiratory distress. Nasogastric aspiration revealed 200 cc of coffee ground material. While on the respirator he suffered cardiac arrest and inspite of all resuscitative efforts, failed to respond. He died in the the ICU on 23rd Novemebr, 1986.

I would now like to invite Dr. Qureshi to describe the histopathology of the specimen obtained during surgery.

DR. QURESHI (*Histopathologist*): The specimen was obtained from the omentum. The histopathology revealed a metastatic adenocarcinoma (non-mucinous) which suggested that it did not arise from the intestine. The most likely source was cholangiocarcinoma in a patient with such presentation. The primary tumor could not be obtained for examination.

Dr. Anwaar then further described the value of endoscopy in obstructive jaundice and elucidated this by refering to various slides on the subject.

DR. ANWAAR (*Gastroenterologist*) : PTC & ERCP have made a great advance in the management of patients with obstructive jaundice. These techniques will pick up the cause and site of obstruction thereby facilitating the planning in advance for surgical procedures in these patients.

He went on to show various slides with strictures and stones in CBD, which had been picked up only by these techniques. He also went on to add that in the absence of biliary surgery history, these strictures are almost always malignant. Benign stictures due to chronic pancreatitis show indentation of the CBD and not the ragged irregular string like stricture seen in this patient. In South East Asia benign strictures may also result from recurrent suppurative cholangitis.

Dr. Mahmood who had operated on this patient spoke next;

Prof. MAHMOOD (*Surgeon*) : When dealing with patients of obstructive jaundice and especially those that are malignant, the primary object is to drain the bile and not necessarily remove the obstruction. The cause of the obstruction may be dealt with at a later date when the condition of the patient is more stable. We opened his abdomen and found adhesions in R.U.Q. There was mild bile stained ascites. A cholecystostomy was performed and a portion of the omentum was also removed for histopathology. The patient was given I V Mannitol to prevent renal shutdown. However he succumbed to his primary illness, which subsequently turned out to be a metastatic adenocarcinoma.

Prof. Lt. GENERAL MOHYDIN (*Prof. Medicine*): The modern techniques in the work up of selected patients of jaundice is an invaluable aid to both the physician and the surgeon. Wroking within the time span dictated by the individual patient's disease state and the value of a cohesive, unified effort of both the physician and the surgeon should be emphasised. This man, however, was doomed at the outset owing to his disease in advanced stage and there was very little one could do in prolonging his life.