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Biliary Stricture

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INDEX

C O U R T E S Y O F C I T I Z E N S A I D S O C I E T Y

I. CASE REPORTS

1. BENIGN STRICTURE OF COMMON BILE DUCT

Female ( ), age 44, admitted 4-3-34 and expired 4-21-34 (18 days).

Cholecystectomy - Postoperative Biliary Fistula

11- -31 - Cholecystectomy and common duct drainage for definite attacks of cholecystitis, characterized by jaundice, right upper quadrant pain radiating to the right scapular region, acholic stools, chills, fever and pruritis. Relief from pain and jaundice following operation. Drained considerable amount of bile from wound.

Jaundice

5- -32 - Bile drainage stopped. Became jaundiced and complained of weakness. No pain, chills or fever. Attack lasted 6 weeks.

Chills, Fever, Jaundice

12- -32 - Attack similar to those previous to operation. Jaundice, chills, fever, pain and pruritis. Acute attack lasted 2 weeks. Never free from jaundice from this time until admission.

Weakness

2- -34 - Called physician because of weakness. Very drowsy. Appetite poor. No nausea, vomiting or pain. Stools acholic and urine dark. In bed until admission.

Past History

Appendectomy 1916, tonsillectomy 1909, four "stillbirths" at 8th month.

Jaundice, Enlarged Liver

4-3-34 - Admitted. Physical examination - emaciated, deeply jaundiced, numerous excoriations over entire body. Pupils - regular and equal, react to light and accommodation. Thyroid - barely palpable. Heart - not enlarged, no murmurs, regular; blood pressure 108/58. Chest - normal breath sounds, no rales, percussion note normal. Abdomen - two operative scars, one upper midline, other right lower rectus; spleen enlarged. Extremities - numerous excoriations. Reflexes - normal. Laboratory: urine - negative, urobilin and urobilinogen negative. Blood - Hb. 88%, wbc's 8,950, Pmn's 84%, L 16%. Coagulating time - 8 min., bleeding - 1' 30". Icterus index - 85.6. Stool - stercobilin present. Stool - urobilinogen 5 mgs. Urine - urobilinogen - 3.9 mgs. per day. Interpretation: High grade but not complete biliary obstruction. Fairly marked diffuse liver disease because of relatively high urine urobilinogen.

4- 4-34 - Icterus index - 65.5. Surgical impression - obstructive jaundice, probably due to benign stricture of common bile duct. Medical impression - biliary cirrhosis, secondary to extrahepatic biliary obstruction. Preoperative preparation - transfusion, glucose with calcium chloride.

Operation

4-6-34 - Numerous adhesions of inferior surface of liver to stomach and duodenum, and the latter to anterior abdominal wall. Common duct could not be found so dissection was carried transversely to its course in the hope of thus forming an external biliary fistula. Biopsy of gastro-hepatic ligament shows fibrous tissue and small glandular structures like small biliary radicals.

4-9-34 - Bleeding from wound.

4-10-34- Icterus index 65.5. Thought to be some bile from drainage site. Color concentration of wound drainage 4174 icterus index units.

4-11-34 - Bile drainage from wound. Given 3 transfusions, calcium chloride and glucose.

4-19-34 - Urea nitrogen - 425 mg. Plasma chlorides - 620 mg. CO_2 - 50 vol. In spite of transfusions, etc., patient became progressively worse and died on 15th postoperative day.

Wound separation

Autopsy

Body is poorly developed, very poorly nourished, white female, 44 years of age, measuring 154 cm. in length and weighing about 90 lbs. Rigor present. Hypostasis purplish and posterior. No edema. Questionable

cyanosis present. Jaundice 3 to 4+. Each pupil measures 3 mm. in diameter. Operative incision through right upper rectus muscle when separated exposes loops of bowel in wound. Great deal of bloody discharge.

Bleeding and Infection

General Peritoneal Cavity contains a considerable amount of bloody exudate and a few clots in right upper portion of abdomen surrounding operative site. In remainder of cavity, there is purulent exudate covering the serous surface and there is about 1000 cc. purulent fluid which gravitated into the culdesac. Appendix not examined.

Empyema

Pleural Cavities: On left side, there is about 1000 cc. purulent fluid. Right side shows few adhesions at base and in apex; no fluid. Pericardial Sac shows no change.

Heart weighs 300 grams. Musculature is firm. No infarction or softening. No evidence of endocarditis. Coronaries are soft and patent throughout. Root of Aorta shows minimal amount of atheromatous change.

Atelectasis

Right Lung weighs 450 grams, Left 500. There is a massive collapse involving at least 90% of both lower lobes and posterior part of upper lobe. These areas are soft, purplish and flabby. Bronchi can be palpated through the lung parenchyma. No nodules or infiltration to suggest bronchopneumonia.

Spleen weighs 350 grams, is soft and shows large amount of pulp.

Cirrhosis

Liver is deep green in color, shows very slight granular roughening of surface; weighs 2100 grams, cuts with increased resistance. Radicles of biliary system are slightly dilated. Liver substance appears separated with fine strands of fibrous tissue.

Segment of common duct absent.

Biliary Apparatus.

There is a large amount of fibrosis, exudate and blood in the hilus of the liver and adjacent area. Fibrosis makes dissection very difficult. Gall-bladder is absent. Portal vein and hepatic artery can be isolated and appears normal. No common duct found. Dissection is carried on in the hilus of the liver and the upper end of the common duct likewise cannot be found.

Dissection of the fixed specimen reveals the following in addition to that already noted above.

The bile ducts in the liver substance are followed toward the hilus in an attempt to find the upper end of the obstructed bile duct. The right and left common ducts are isolated within the liver substance and unite together to form a single duct within the liver tissue adjacent to the hilus. The left and right hepatic ducts show the same moderate degree of dilatation noted in the small ducts within the liver substance. As the two ducts unite together, the common duct at this point is dilated to a diameter of slightly more than 1 cm. It continues within the liver substance for a distance of about 2.5 cm. and ends abruptly in a blind end within scar tissue at the hilus of the liver adjacent to the old gall-bladder bed. The lower end of the common duct is identified by finding the pancreatic duct and dissecting this into the ampulla of water. It measures about the normal diameter, is somewhat fibrotic, has a smooth mucosa. The duct penetrates through the head of the pancreas and enters the heavy scar tissue at the hilus of the liver and at this point is lost. The segment between the lower and upper ends cannot be identified. It may have been broken across during the pervious dissection. The lower segment of the common duct had a lumen throughout the full length of the portion finally identified.

Hemorrhages into bowel.

Gastro-Intestinal Tract: shows hemorrhages in mucosa of stomach and in several points along small bowel.

Pancreas is quite firm. Head is imbedded in fibrosis in right upper part of abdominal cavity. Duct not dilated. Pancreas itself, particularly in body and tail, show no significant change.

Adrenals well developed. No hemorrhage.

Each Kidney weighs 160 grams. Surfaces slightly granular. Substance does not appear to be decreased in amount. Pelvis on left side is moderately dilated.

Bladder shows some cystitis along trigone and a moderate degree of trabeculation.

Genital Organs: Uterus is of normal size, shows no polyps. Ovaries contain no cysts. Broad ligaments are free of fibrosis. Cervix is transversely lacerated and shows numerous small retention cysts.

Aorta shows no evidence of any arteriosclerosis.

Organs of Head and Neck - not examined.

Diagnoses:

1. Absence of common bile duct, probably due to inflammatory and fibrous replacement, postoperative.
2. Obstructive cirrhosis.
3. Generalized peritonitis.
4. Postoperative evisceration.
5. Postoperative abdominal hemorrhage.
6. Hemorrhages in bowel and stomach.
7. Empyema.
8. Pulmonary atelectasis.
9. Splenitis.
10. Hydronephrosis, probably old pyonephrosis.
11. Cystitis.

2. CONGENITAL STRICTURE OF BILE DUCT

Male ( ), age 4 months, admitted 4-6-34.

Jaundice at Birth

1-5-34 - Born at University of Minnesota Hospitals. Birth weight 8 lb. 2 oz. Had deep jaundice beginning on 3rd day after birth. Stools clay-colored. Urine dark brown. By discharge, jaundice had cleared slowly but majority of stools showed only slight amount of bile pigment.

Infection - Jaundice Persisting

1-22-34 - Umbilical stump became reddened, swollen and discharged considerably. Swelling extended about 3 cm., radially, from infected stump. Jaundice decreased. 1-30-34 - Readmitted. Weight 7.92 lbs. Icterus index - 64.8 units.

2-5-34 - Discharged. Gained weight consistently while in hospital and infection of umbilical stump subsided. Jaundice decreased but persisted very slightly.

Jaundice more intense, age 4 months

3-1-34 - Jaundice increased. Vomiting.

4-6-34 - Readmitted. Weight 11 lb. 4 oz. Intensely jaundiced. Vomiting every day. Physical Examination - fairly well-nourished, jaundiced, white male. Eyes - sclera jaundiced, otherwise negative. Ears, nose and throat - normal. Neck - few palpable precervical nodes. Chest - clear. Negative heart. Abdomen - liver down 2½ fingers below right costal margin; spleen not enlarged. Extremities - negative. Impression: 1. Congenital stricture of biliary duct. 2. Hepatic cirrhosis.

4-11-34 - Stools clay colored with no bilirubin or urobilin. 24 hour urine contains 2.86 mg. urobilin. Losing weight. Transfused. Given glucose and calcium gluconate.

Operation

4-12-34 - Liver enlarged. Spleen enlarged. Common duct could not be found. Tissue in which place common duct and artery lay divided completely across. No trace of duct or gallbladder. Proximal portion of the tissue dissected medially well up into the liver and hepatic artery came well into view. Drains were inserted with the hope that an atresic bile duct may have been cut across.

External Bile Drainage

4-13-34 - Icterus index - 133.3 units. Draining bile.

4-14-34 - Transfused.

4-17-34 - Draining bile. Condition good.

4-25-34 - Temperature 105. Dressings more saturated with bile than before. Taking feeding well.

5-1-34 - Temperature decreasing.

Icterus index 55.9. Oxbile given every day.

5-10-34 - Icterus index 32 units. Draining less bile. Temperature 102.

5-19-34 - Temperature lower. Still in hospital. (At present has abundant bile draining from wound. Taking feedings well. Improving).

II. ABSTRACT

BENIGN OBSTRUCTION OF BILE DUCTS

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Classification

(1) Congenital.

(2) Acquired.
   a. Traumatic
   b. Gall-stones, inflammatory
   c. Diffuse stenosing, cholangitis.

Congenital Benign Stricture of Bile Ducts

Manifests itself at or soon after birth, or within three weeks, by symptoms of biliary obstruction. Traces of bile may be in stool, due to oozing from desquamated intestinal mucosa. There is an enlargement of the liver, two to three times normal. Spleen is enlarged due to an increase in size and number of endothelial cells suggesting an attempt on part of spleen to assume the phagocytic function of the liver cells which have been damaged.

Etiology

Thomson - a developmental anomaly of

ducts giving rise to hepatic cirrhosis by obstruction.

Rolleston - Primary mixed cirrhosis of liver of the fetus due to toxin from internal blood with a descending obliterative cholangitis that involves extrahepatic ducts in a process similar to obliterative appendicitis.

Feldman and Lawson - Present evidence contrary to latter theory. Reports a case in which congenital obliteration of common duct occurred in one only of twins.

Types

(1) Ducts between liver and duodenum obliterated.
(2) Gallbladder and cystic duct absent.
(3) Hepatic and cystic ducts obliterated.
(4) Common bile duct partially or completely obliterated. Commonest site is in cystic and hepatic ducts. Holms states that in 16% of the recorded cases, the hepatic and cystic ducts were normal, and communicated with each other so that cholecystenterostomy should give relief in this group. Ladd stated that 40% revealed an anatomical situation that would be amendable to surgical intervention. He collected 11 operations, only 2 of which showed complete atresia and were cured by cholecystenterostomy. In all other operative survivals the surgical procedure was only dilation of stenosed ducts.

Differential Diagnosis

Must be differentiated from icterus neonatorum, chronic interstitial hepatitis and syphilitic hepatitis, icterus of septicemia, cholelithiasis and acute catarrhal jaundice.

Duration and Prognosis

A certain number die from hemorrhage within a few days. Thomson reported 49 cases, 30 of which lived more than one month. Only 3 survived more than 8 months. Laverson recorded only 5 cases out of 63 that survived for 8 months.

Treatment

In view of the uniformly fatal outcome and the possible change of surgical relief in a certain few it is felt that all cases should be explored with a full understanding of the high fatality.

Acquired Benign Strictures

Etiology

Majority of strictures are due to injuries during cholecystostomy or cholecystectomy. Some are due to cholelithiasis and some extensive contractures have been observed apparently due to cholangitis without a previous operation or without the presence of stones.

In the first group, 48 cases were reported from the Mayo Clinic in 1925, and again in 1923, 62 cases, and 17 more cases in 1929.

Conditions predisposing to Operative Damage to Duct

1. Abnormally short cystic duct and short pedicle.
2. Abnormal coursing cystic duct, parallel and closely united to common duct.
3. Short pedicle due to inflammation or dilation.
4. Clamping a retracting bleeding cystic artery.
5. Obliteration of all anatomical landmarks.
6. Difficult exposure viz; thick abdominal wall.

Clinical Types - After Cholecystectomy

1. Jaundice shortly after cholecystectomy with never any drainage of bile and early convalescence seems normal. In a few weeks, patient presents a typical picture of complete jaundice. Generally no pain. Usually in this type the duct is found to be completely severed or entirely closed by stricture.

2. Bile drains profusely a few hours after cholecystectomy and continues throughout convalescence. Complete external drainage can sometimes be demonstrated. In a few days, drainage decreases and jaundice develops. Generally the duct is found to be completely severed or strictured.

3. Normal cholecystectomy convalescence and entirely well for 6 months or more. Then jaundice intervenes and eventually becomes constant. This group complains of pain, fever and chills. At times portion of duct is found to be missing, explained possibly by adhesions contracting and necrosing a segment of duct (our case).

After Cholelithiasis without Operation

Strictures seldom result even if stones are implanted so tightly as to cause complete mucosa ulceration. In a large series of cases at Mayo's Clinic, stricture developed in only 4 cases due to this cause. These strictures are generally localized lesions limited in extent and most frequent in the lower part of the common duct.

After Cholangitis without Operation

History not distinctive in these cases. Patients come to operation with diagnosis of common duct obstruction either due to stone or neoplasm. Often a longstanding history of recurrent attacks of biliary colic precedes the onset of jaundice because the stricture must be far advanced before the resulting stenosis will be sufficient to cause jaundice. This type of case has been reported by Delbut 1924, Lafaurcode 1925, Miller 1927, and others.

The extreme rarety of non-traumatic stricture of the common bile duct in spite of the great frequence of inflammation impresses one with the peculiar resistance of bile ducts to cicatrical stenosis.

Opening of the common bile duct which is widely practiced does not result in strictures. Complete severance of the common duct has been done in dogs with spontaneous union in a large percentage.

Effect of Obstruction of Bile Ducts on Liver and Bile Passages

As is well-known, biliary cirrhosis is consequent to obstruction of the biliary tract. McMaster, et al., demonstrated that a dilation of the biliary tracts analogous to the hydronephrosis occurring in the urinary tract and coined the term hydrohepatitis. Counsellor and McIndoe pointed out that the most important causes of hydrohepatosis are stones in the common duct, strictures following operation, and neoplastic obstructions. Accompanying this ectasia of the ducts, there is fibrosis which extends out into the liver parenchyma. Often the intralobular fibrosis is not in proportion to the degree of obstruction. Infection and slowly progressing obstruction probably induces greater degrees of portal cirrhosis. The liver parenchyma is injured so that the usual conversion of bile salts is no longer carried out.

Site: In order of frequency.

1. Junction of cystic and hepatic ducts.
2. In supraduodenal portion.
3. At papilla.

Diagnosis

In many cases, the differentiation between primary cirrhosis and postoperative stenosis of the ducts can be made only at exploratory operation.

Infection of opaque media into the fistula when present may be of value. Bismuth was first used for this purpose but this was later replaced by lipiodol and thorotrast. The position and the degree of the obstruction can sometimes be visualized and occasionally the most favorable type of operative procedure to be done may be determined.

Chemistry of Bile in Diagnosis

(C.J.W.)

Bilirubin formed from hemoglobin in reticulo-endothelial cells of spleen, liver and bone marrow is normally excreted into bile by the liver cells. If these are diseased, or if there is obstruction along the bile passages,

or if bilirubin formation is increased, jaundice results. Last named cause (increased bilirubin formation) is associated with "hemolytic" jaundice and there is no bilirubin in the urine. Other types are associated with bilirubinuria. Bilirubin goes to bowel with bile and is reduced there to urobilinogen. At least one-half of this is resorbed and goes back to the liver. Normal liver uses it, probably to make new hemoglobin, although a little goes back again in the bile. Diseased liver refuses it and it goes to the general circulation and appears in urine as a sign of liver injury. If there is obstruction to the passage of bilirubin from liver to bowel, proportionately less urobilinogen is formed and less goes back to the liver. Therefore, to be of value in diagnosing liver disease, one must know the relative amounts in the feces and urine. It is best to know the amounts passed per day.

Examples	Feces	Urine
Normal	150 mg.	1 mg.
Cirrhosis of liver	120 mg.	5 - 20
Common duct stone (short duration)	5 -15 mg.	1 - 3
Common duct stone (long duration with biliary cirrhosis)	5 -15 mg.	10 - 15
Carcinoma of head of pancreas	1 - 2 mg.	0
Catarrhal jaundice	5 -20 mg.	10 - 20

Treatment - Prophylactic

1. Care in dissection of cystic duct.

2. Thorough and accurate technique and complete primary operation when possible.

3. Early recognition and early surgical treatment of diseases of the gall-bladder and bile duct.

4. Preference of cholecystectomy over cholecystostomy.

Treatment is based on fact that immediate drainage of the bile is imperative and permanent passage to the intestine must be established before a cure is affected.

Technique

Hepaticoduodenostomy.
Resection of stricture with end to end anastomosis.
Simple reconstruction over a tube.
Transplantation of an established external biliary fistulae into duodenum.

Summary

1. Benign strictures of the bile ducts may be congenital or acquired.

2. Congenital obstructions manifest themselves at birth or within 3 weeks by jaundice. Bleeding, enlarged liver and splenomegaly usually is present.

3. The congenital obstructions probably are developmental errors although some workers suggest intrauterine inflammatory processes.

4. There may be any degree of absence of the duct system. From 16 to 40% of the cases have had some remnant of duct outside the liver in which surgical procedures are possible.

5. In this group of cases, the differential diagnosis includes icterus neonatorum and the various forms of jaundice of the new born secondary to infections of various types.

6. The duration of life in untreated cases is between 1 and 8 months. In 49 cases, 30 lived over 1 month and only 2 over 8 months.

7. Operative mortality is very high but on the basis of 40% operability (Ladd) exploration is recommended.

8. Acquired strictures are nearly

always due to complications after gall-bladder operations and rarely after cholangitis with or without stones.

9. The postoperative obstructions result either from direct injury to the ducts at the time of operation or from a progressive stenosis due to inflammation.

10. Anatomical abnormalities, such as a very short cystic duct, abnormally coursing duct or inflammatory obliteration of landmarks increases the chance for operative injury to the main duct.

11. The course of events after the operation is variable. In the cases with operative injury, bile drainage may begin immediately and continue for a few weeks. After closure of the fistula, jaundice supervenes. In other cases, the convalescence may be normal or there may be temporary bile drainage and jaundice occur a few weeks or months after the operation. This latter type may be due to contraction of scar tissue secondary to local inflammation about the ducts.

12. Strictures due to impaction of stones in the common duct are rare. In one large series, only 4 cases were of this type.

13. Only a few cases of obstruction due to long standing cholangitis (without stone) have been reported. The bile ducts apparently have a peculiar resistance to stenosis due to lesions within the duct itself. Traumatic tears and surgical incisions are not followed by stricture.

14. The secondary changes in the liver consist of dilation of the ducts and cirrhosis. The two conditions frequently do not progress together. In some cases, enormous ectasia of the biliary tree can occur. In others, a fine fibrosis of the liver parenchyma extending out from the periphery of the lobules is present. The damage to the liver influences the transformation of bile pigment and becomes a factor in differentiation between primary cirrhosis and these secondary changes.

15. The common site of injury is, of course, near the point of the operative procedure, at the junction of the cystic and hepatic ducts. Other areas of stricture may be at the supra-duodenal portion of common duct and finally at the papilla.

16. When a biliary fistula is present, injection of the sinus tract with lipiodol or thorotrast sometimes visualizes the site and extent of the stricture and may be a value in planning the operative procedure.

17. A study of the relative daily amounts of urobilinogen in the urine and feces frequently gives an impression regarding the degree of the obstruction and the severity of the liver injury.

18. When the common bile duct is obstructed, bilirubin cannot reach the bowel and therefore there is a low percentage of urobilinogen in the feces. When the liver cells are damaged, the reabsorbed urobilinogen is not utilized by the liver cells and is retained with the general circulation and appears in the urine. It is this proportion between stool and urine urobilinogen which is of diagnostic value. The quantities per day are of most value.

19. Probably most cases of stenosis of the bile duct occur after difficult cholecystectomy due to long standing gall-bladder disease. Other cases are due to the residual postoperative inflammation. Early treatment of the primary gall-bladder disease and especial care in the handling of the cystic duct in technically difficult operations are the most valuable prophylactic measures.

20. The treatment of the stenosis consists of reestablishment of drainage into the bowel by whatever method is possible depending on the anatomical position of the upper end of the ducts.

--Prepared By:

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III. ANNOUNCEMENTS

1. PHYSIOLOGICAL-PHARMACOLOGICAL SEMINAR

Physiological-Pharmacological Seminar will meet Friday, May 25, at 12:30 in room 116 Millard Hall.
F. W. Hoffbauer: "Hypoglycemia."

2. SENIOR LECTURE

"Value of X-ray Examination in Diagnosis of Intracranial Conditions" by Dr. John D. Camp of the Mayo Clinic, Eustis amphitheater at 3 P.M. on Friday, May 25. All seniors are required to attend. Anyone interested is welcome.

3. RADIOLOGY SEMINAR

"Details in the Roentgen Diagnosis of Brain Tumors" by Dr. John D. Camp of the Mayo Clinic, University Hospital, X-ray department, room M-515, Friday, May 25 at 5 P.M. Anyone interested is welcome.

4. WANTED!

Internes to take

locum tenens.

See Dr. O'Brien