CONGENITAL ABSENCE OF GALLBLADDER

REPORT OF THREE CASES

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IT IS QUITE STRIKING that congenital absence of the gallbladder without other anomalies of the biliary system should be so rare, whereas other anomalies of the biliary system are present in 10 per cent of autopsy cases.[1] Courvoisier[2] collected 25 such cases in 1890. In 1928, Bower[3] reviewed the literature and found 31 cases; the earliest of these cases having been reported by Bergman in 1701. Gross,[4] in 1938, published 38 cases. In 1945, Dixon and Lichtman[5] reviewed 50 cases since 1900, and reported ten additional cases. Our review of the available literature revealed 71 such cases. To this number we wish to add three case reports.

The incidence of congenital absence of the gallbladder is about 0.065 per cent, as calculated from 18,350 autopsies by Talmadge.[6] Kirshbaum[7] reported the incidence of such anomaly to be about one case in 3,661 (0.03 per cent); while Smith, Hall, Mentzer, and Nägel found six cases (0.075 per cent) in 7,919 autopsy specimens.

Syphilis, catarrhal inflammation of the biliary tract, fetal peritonitis, maternal toxins, and mechanical factors all have been advanced by various authors as theories of origin of this anomaly. A more easily defended theory involves the embryology of the biliary tree. The embryologic origin of the biliary tract is intimately associated with that of the liver. The entire biliary system, including the liver, arises as a ventral outgrowth from the gut entoderm in the region of the anterior intestinal portal.[8] From the floor of the future duodenum arises the hepatic diverticulum; a cranial portion which is the anlagen of the bile ducts, and a caudal portion which is to become the gallbladder and cystic duct. Though the hepatic diverticulum is originally tubular, the biliary passage tends to become solid, and it is not until the seventh week that a lumen has been reëstablished throughout most of its length. Thus, failure of the gallbladder anlagen to form, or, as Gross[4] suggests, failure of the solid anlagen to become tubular, would result in an absent gallbladder. Miller[9] presents strong evidence that the lumen may sometimes fail to form; as he demonstrated tubular tissue in the hepatoduodenal ligament.

In our review of the literature we found many case reports to be incomplete and a few were available to us only in abstract form. An analysis of the data with respect to age and sex, symptoms, preoperative diagnosis, whether found at operation or autopsy, and attempts to prove an intrahepatic gall-

bladder was made. This material was obtained from a total of 71 cases, 34 of which were reported from surgical procedures while 38 were autopsy reports—one case appearing in both series.

The average age of all cases reported was 46 years at the time of discovery. Females were twice as frequent as males in the entire series. These figures agree with those of Melville.[10] However, when viewed as to autopsy or operative reports, the ratio in autopsied specimens is one to one; while those operated upon have a three and three-tenths to one ratio. This would tend to show that occurrence of the anomaly has little relation to sex; but that symptoms provoking exploration of the biliary tree in this entity follow the same sex ratio as does all surgical biliary disease.

Of the 71 cases 34 gave histories indicating biliary tract disease. Due to the brevity and incompleteness of the earlier reports, this analysis is not necessarily valid.

Of the 34 cases (Table I) operated upon, the preoperative diagnosis in 13 cases was cholecystitis; in eight it was obstruction of the common bile duct; five cases were listed only as exploratory celiotomies; four were found incidental to operations for nonassociated intra-abdominal pathology; and in four cases a preoperative diagnosis was not recorded.

In the series upon whom operations were performed, the gallbladder was reported as absent in all 34 cases, while the cystic duct was reported absent only 15 times. Twenty-one reports noted the condition of the common duct; it being dilated 11 times, containing stones in 11 and appearing normal in seven. The liver was described 13 times. In three cases it was atrophic, in two, hypertrophic, and in two reports anomalies were cited—one being a large Riedel lobe and the other a complete absence of the left lobe.

In the series where the gallbladder was found to be congenitally absent at postmortem examination (Table II), the cystic duct was recorded as absent in 18 cases; the common duct as dilated in four cases, containing stones in only one case; and as normal in seven instances. Ten cases exhibited a pathologic liver, seven of the atrophic variety, and three of the hypertrophic type. Four reports noted anomalies of the liver. In two the quadrate lobe was indistinct; while in a third case the quadrate lobe was absent. The remaining case exhibited nonseparation of the liver lobes.

Visualization of the gallbladder by cholecystograms was attempted in 12 instances. In several patients the attempt was repeated using different methods of administration of the dye. All failed to be visualized.

In one autopsy specimen (Mackmull[11]—1930) and two postoperative patients (Drenckhahn and Rogers[12]—1943; and Dixon and Lichtman[5]—1945), the biliary trees were injected, *via* the common duct, with radiopaque material in an attempt to visualize an intrahepatic gallbladder if present. All failed to reveal a shadow which could be interpreted as a gallbladder.

CASE REPORTS

Case 1.—Mrs. R. A., a 52-year-old white female, was admitted to Wesley Memorial Hospital, September 13, 1945, because of severe cramping pain in the epigastrium, nausea,

TABLE I

SYNOPSIS OF CASES OPERATED UPON

Author		Age and Sex	Symptoms and Duration Of		Preoperative Diagnosis	Why Operated	Findings at Surgery	Proven Not Intrahepatic-
Bower	1928	50 F.	Severe pain in R. U. Q. Jaundice.	1 day	Acute pancreatitis; perforated ulcer	Acute abdomen.	Absent gallbladder. Left liver lobe absent.	No.
Danzis	1935	53 F.	Belching, with vomiting postcibal. Pain in R. U. Q. radiating to back.	3 mos.	Cholecystitis, with calculi.	Cholecystectomy.	Absent gallbladder and cystic duct. Com. duct dilated and containing stones, with inspissated bile.	Proven at autopsy. Death from peritonitis.
Davis	1944	56 F.	Anorexia, nausea, abdominal pain.	5 days		Celiotomy	Absence of gallbladder and cystic duct. Liver normal.	No.
Dixon & Lichtman	1945	(1) 61 F.	Jaundice, chills, fever, and pain in L. U. Q. Postcibal distress.	8 yrs.	Cholecystic dis., with biliary obst.	Biliary disease	Absent gallbladder and cystic duct. Stones in dilated com. duct. Liver normal.	No. Neg. X-ray.
		(2) 68 M.	Jaundice, chills, fever, and epigastric pain. Weight loss of 60 lbs.	6 wks.	Obst. Cholecystic disease.	Biliary disease.	Absent gallbladder. Com. duct normal. Liver normal. No stones.	No. Neg. X-ray.
		(3) 29 F.	Chills, fever, epigastric pain. Vomiting or eating relieved pain.	4 yrs.	Perforating gastric ulcer.	Acute abdomen.	Gallbladder absent. Com. duct normal.	No.
		(4) 39 F.	Nausea, vomiting, food intolerance, and right-sided pain.	3 yrs.	Susp. gallbladder disease.	Biliary disease.	Gallbladder and cystic duct absent. Com. duct moderately dilated.	No.
		(5) 53 M.	Bloating and tenderness R. U. Q.	10 yrs.	Duod. ulcer and cholecystic dis.		Absent gallbladder. Com. duct normal.	No.
		(6) 52 F.	Jaundice, chills, fever, nausea, vomiting. Intolerance to foods. Colicky R. U. Q. pain.	4 yrs.	Cholecystitis, with cholelithiasis.	Biliary disease.	Absent gallbladder. Normal com. duct. Liver normal.	No. Neg. X-ray.
		(7) 57 M.	Jaundice, chills, fever, nausea, vomiting. R. U. Q. pain.	12 yrs.			Absent gallbladder and cystic duct. Com. duct and liver normal. Lymphosarcoma of stomach.	No.
		(8) 50 F.	Jaundice, food intolerance, pruritis acholic stools, epigast. pain.			Celiotomy.	No gallbladder. Com. duct dilated. Hepatitis. Duod. distended.	Neg. X-ray. Retrograde study revealed no G. B.

TABLE I – *Continued*

SYNOPSIS OF CASES OPERATED UPON

Author		Age and Sex	Symptoms and Duration Of		Preoperative Diagnosis	Why Operated	Findings at Surgery	Proven Not Intrahepatic-
Drenckhahn & Rogers	1943	43 F.	Postcibal burning, with bloating. Food intolerance.	10 yrs.	Chronic cholecystitis.	Biliary disease.	Gallbladder and cystic duct absent. Liver grayish-brown with fibrous markings. Com. duct moderately dilated.	Neg. X-ray. Retrograde study revealed no G. B.
Emmert	1931	20 F.	Pain in right abdomen.	3 yrs.	Appendicobiliary synd.	Biliary disease.	No gallbladder. Normal ducts. Liver enlarged and red.	No. Neg. X-ray.
Bwers	1914	45 F.	Pain in hypogastrium.			Exp. appendicectomy.	Gallbladder and cystic duct absent. Panc. head enlarged and hard.	No.
Fowler	1917	42 F.	Jaundice.		Com. duct obst.	Biliary disease.	No gallbladder or cystic duct. Panc. hard and indurated.	No.
Golob	1927	59 F.	Belching and epigast. pressure. Pyrosis.	4 yrs.	Cholecystitis.	Biliary disease.	Gallbladder not located.	No. Neg. X-ray.
Giuliani	1924	35 F.	Sympt. of common duct obstruction.		Com. duct obst.	Biliary disease.	Gallbladder absent. Stone in com. duct.	No.
Hinder	1909	60 M.	Jaundice and dyspepsia. Apparent cholecystitis.			Acute abdomen.	No gallbladder or cystic duct. Pancreas hard.	No.
Ishiyama	1927	50 F.	Jaundice. Pain in R. U. Q.	20 yrs.	Com. duct obst.	Biliary disease.	Gallbladder absent. Com. duct enlarged, with stones. Liver swollen and icteric.	No.
Leopold	1911	45 F.	Jaundice. Gallstone colic.		Cholecystitis, with stones.	Biliary disease.	Absent gallbladder. Stones in common duct.	No.
Lintz	1927	48 F.	Pain in R. U. Q. radiating to back.	5 yrs.		Biliary disease.	No gallbladder. Large Riedel lobe.	No. Neg. X-ray.
Melville	1937	48 F.	Epigastric pain. Jaundice and obst. symptoms. Vomiting.	7 yrs.	Chronic cholecystitis.	Biliary disease.	No gallbladder located.	No. Neg. X-ray.
Meyer	1929	21 M.	Subicteric sclerae. Epigastric distress and colicky pain.	5 yrs.	Cholecystitis.	Biliary disease.	Absent gallbladder. Band between usual G-B site and duodenum.	No. Neg. X-ray.

TABLE I—*Continued*

SYNOPSIS OF CASES OPERATED UPON

Author		Age and Sex	Symptoms and Duration Of		Preoperative Diagnosis	Why Operated	Findings at Surgery	Proven Not Intrahepatic-
Naegeli	1921	65 F.	Gallstone colic. Vomiting. Icterus.	30 yrs.			No gallbladder or common duct. Liver cirrhotic.	No.
		34 F.	Vomiting, jaundice, acholic stools. Pain in R. U. Q.	5 yrs.			No gallbladder. Stone in common duct. Com. duct dilated.	No.
Niemach	1908	60 F.	Nausea, vomiting, chills, fever. Abd. pain. Acholic stools.	2 wks.	Com. duct obst.	Biliary disease.	Gallbladder absent. Stone in common duct.	No.
Robertson, Robertson, & Bower	1940	45 M.	Postcibal distress. Jaundice and acholic stools.	2 yrs.	Cholecystitis and com. duct obst.	Biliary disease.	Gallbladder and cystic duct not identified. Dense adhesions about gallbladder area.	Yes, at autopsy.
Sarma	1941	41 F.	Abd. pain and vomiting.	3 mos.	Cholecystitis.	Biliary disease.	Gallbladder absent. Liver small, with dense adhesions.	No. Neg. X-ray.
		47 F.	Typical gallbladder symptoms, with calculi.		Cholecystitis, with calculi.	Biliary disease.	Absent gallbladder and cystic duct. Com. duct dilated, with stones.	No Neg. X-ray.
Schmidt	1928	30 F.	Pain in epigastrium.	6 yrs.	Cholelithiasis, with pericholecystitis.	Biliary disease.	Gallbladder and cystic duct absent. Common duct normal.	No.
Schulz	1914	64 F.					Absent gallbladder and cystic duct. Com. duct dilated, with stones.	No.
Stone	1908	54 F.	Jaundice and epigastric pain.	5 yrs.	Cholelithiasis.	Biliary disease.	Gallbladder absent. Hepatic and com. ducts dilated, with stones.	No.
Torrence	1920	38 M.	Digestion poor, with constipation. Chronic appendix symptoms.	3 yrs.	Appendicitis.		Absent gallbladder and cystic duct.	No.
Whipple	1921	52 F.	Nausea, vomiting, jaundice, pruritis. Bloating and belching postcibal. Epigastric pain.	2 yrs.	Obst. of com. duct.	Biliary disease.	Gallbladder and cystic duct absent. Com. duct markedly dilated, with stones. Pancreatic lymphangitis.	No.

vomiting, jaundice, and clay-colored stools. The pain had been present for one week, radiating through to the back and up to the right shoulder. It was not related to meals nor foods and was relieved only temporarily by hypodermic injections of opiates. Nausea and vomiting were persistent. Jaundice appeared three days after the onset of pain. She had noted clay-colored stools for two days prior to entry into the hospital. Similar episodes, though less severe, had occurred once ten years ago and again one month prior to her hospital admission.

Physical examination revealed a well-nourished white female appearing moderately ill and having a marked icteric tint to her skin and sclerae. The heart was not enlarged but there were frequent extrasystoles and a systolic murmur was present at the apex. Pulse 68; blood pressure 104/65. Examination of the abdomen revealed no scars, rigidity or masses. Tenderness on deep palpation in both upper quadrants was present.

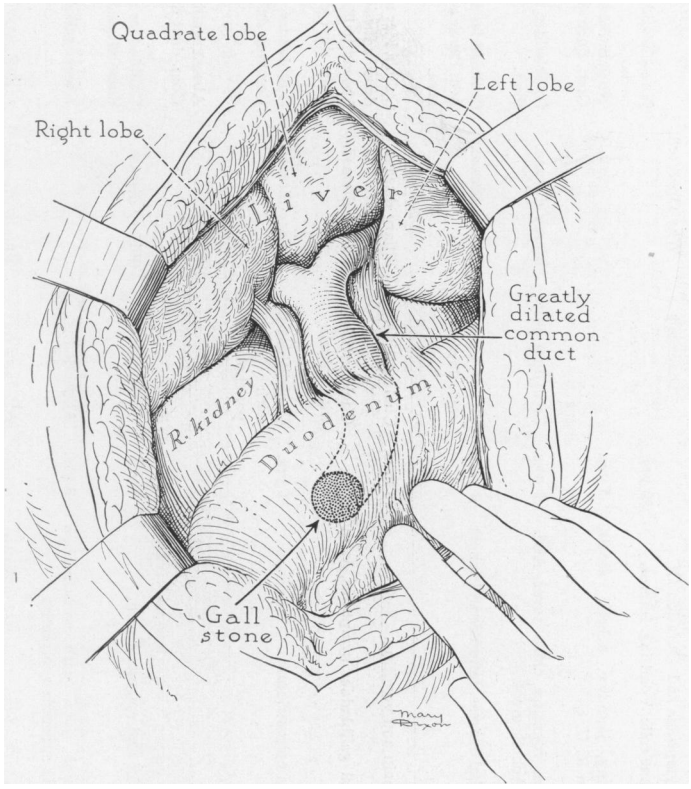

FIG. 1.—Case 1: Drawing of findings at time of operation.

The erythrocyte count was 5,790,000 cells per cm., the hemoglobin 15 Gm. per 100 cc. of blood; and the white cell count was 5,800 cells per cm. Urinary urobilinogen was absent, while bile was present in the urine; otherwise the urine was normal. The icteric index was 123.5 units, and the van den Bergh test revealed a prompt direct reaction with a 9.45 mg. per cent quantitative indirect reaction. The stools were clay-colored and contained no bile. The blood proteins on admission were 6.80 Gm. of total protein per 100 cc., with 3.87 Gm. albumin fraction and 2.93 Gm. globulin fraction. An intravenous

TABLE II

SYNOPSIS OF CASES AUTOPSIED

Author		Age and Sex	Symptoms Presented	Autopsy Findings	Proven Intrahepatic	Remarks
Arnissat	1831	24 yrs. F.	Abdominal-chest disease.	Gallbladder absent; cystic duct present. Pancreas enlarged.	Autopsy only.	
Baker	1835	Nm.	Suppurative process.	Gallbladder absent; cystic duct not mentioned.	Autopsy only.	Additional information about symptoms not given.
Bergman	1701	Nm.	Not mentioned.	Gallbladder absent; numerous small cystic ducts. Liver showed two lobes not separated.	Autopsy only.	
Bergman	1836	60 yrs. F.	Mental disease.	Liver enlarged. Gallbladder replaced by small fibrous mass.	Autopsy only.	This case is questionable, as gallbladder may have been present.
Bubenhoefer	1905	66 yrs. M.	Cardiac failure.	Gallbladder and cystic duct absent. Liver small and cirrhotic.	Autopsy only.	
Canton	1847	65 yrs. F.	Cerebral hemorrhage.	Gallbladder and cystic duct absent. Liver two-thirds normal size. Common duct twice normal size.	Autopsy only.	
Danzis	1935	53 yrs. F.	Belching, with vomiting postcibal. Pain R. U. Q. radiating to back.	No evidence of gallbladder. No cystic duct found. Common duct dissected back into hepatics and liver.	Autopsy only.	Death following operation.
Droste	1853	74 yrs. F.	Pulmonary disease.	Gallbladder absent. Cystic duct not mentioned. Liver atrophic.	Autopsy only.	
Eiben	1910	48 yrs. M.	Pneumonia.	Gallbladder and cystic duct absent. Common duct not dilated. Normal liver.	Autopsy only.	
Eiben	1910	20 yrs. F.	Tuberculosis.	Gallbladder and cystic duct absent.	Autopsy only.	
Elvert	1780	Nm.	Not mentioned.	Gallbladder absent. Cystic duct not mentioned	Autopsy only.	

TABLE II—*Continued*

SYNOPSIS OF CASES AUTOPSIED

Author		Age and Sex	Symptoms Presented	Autopsy Findings	Proven Intrahepatic	Remarks
Eschner	1894	2 yrs.	Pulmonary signs and symptoms of ricketts. No biliary disease.	Gallbladder absent.	Autopsy only.	
Gaultier	1829	60 yrs. M.	Pulmonary tuberculosis.	No gallbladder. Cystic duct entered liver.	Autopsy only.	Cystic duct was traced into liver substance.
Gordon & Dragutsky	1942	76 yrs. M.	Urinary complaints. Anorexia, with constipation.	Gallbladder absent. No cystic duct. Common duct one cm. in diameter.	Multiple microscopic sections of liver.	
Harle	1856	50 yrs. F	Not mentioned.	No gallbladder or cystic duct found.	Autopsy only.	
Hoffman & Jackson	1910	65 yrs. F.	Pneumonia.	No gallbladder or cystic duct found. Common duct dilated.		
Kehr		Adult M.	Not mentioned.	Absent gallbladder. Cystic duct unusually large.	Autopsy only.	Cystic duct present and enlarged.
Latham	1897	49 yrs. M.	Pulmonary tuberculosis.	Gallbladder absent.	Autopsy only.	
Lenain	1853	74 yrs. F.	Pulmonary disease.	Gallbladder and cystic duct absent.	Autopsy only.	
Lockhart	1927	24 yrs. F.	Symptoms of lung abscess.	Only pouch of common duct at level of gallbladder site.	Autopsy only.	Outpouching of common duct present.
Loreta	1888	40 yrs. F.	Not mentioned.	No gallbladder or cystic duct found.	Autopsy only.	
Knox	1922	1 yr. F.	Bronchopneumonia	Absent gallbladder.	Autopsy only.	
Mackmull	1930	57 yrs. M.	Pulmonary tuberculosis. Constipation with attacks of nausea.	Gallbladder and cystic duct absent.	Retrograde lipiodol injection of liver.	Retrograde examination of the biliary tree with X-ray carried out in cadaver.
Miller	1936	84 yrs. M.	No gastro-intestinal or biliary complaint. Myocardial failure.	Absent gallbladder. Small, blind cystic duct in fibrous tissue. Quadrate lobe of liver indistinct.	Autopsy only.	Remnant of cystic duct present.
Montault	1829	Adult M.	Carcinoma of pylorus.	Absent gallbladder and cystic duct. Liver atrophic.	Autopsy only.	

TABLE II—*Continued*

SYNOPSIS OF CASES AUTOPSIED

Author		Age and Sex	Symptoms Presented	Autopsy Findings	Proven Intrahepatic	Remarks
Mǫller	1920	28 yrs. F.	Bronchopneumonia	Absent gallbladder and cystic duct. Common duct normal.	Autopsy only.	
Patterson	1864	35 yrs. M.	Asthma death.	No gallbladder or cystic duct present. Liver hypertrophic.	Autopsy only.	
Robertson & Robertson & Bower	1940	45 yrs. M.	Indigestion, with postprandial burning. Jaundice and clay-colored stools.	No gallbladder or cystic duct located. Common duct dilated. Cirrhosis of hypertrophic type. Carcinoma of common duct.	Autopsy only.	Operated for removal of common duct obstruction.
Sands	1865	29 yrs. M.	Pulmonary tuberculosis.	Absent gallbladder. Liver atrophic. Absent quadrate lobe of liver.	Autopsy only.	
Schachner	1916	2 yrs.	Not mentioned.	Absent gallbladder. Syphilis of liver.	Autopsy only.	
Tambault & Schachman	1882	Nm.	Paretic dement. No gallbladder symptoms.	Gallbladder absent. Liver small.	Autopsy only.	
Talmadge	1938	51 yrs. M.	None.	Gallbladder absent. Small cystic duct present. Hepatic and common ducts dilated. Liver normal but quadrate lobe poorly developed.	Careful dissection of liver and biliary system.	
Trimble	1850	55 yrs. F.	Infectious disease, with jaundice.	No gallbladder or cystic duct. Liver atrophic. Pancreas contained stone.	Autopsy only.	
Vergne	1826	26 yrs. M.	Accidental death.	Absent gallbladder and cystic duct.		
Konjetzny	1913	64 yrs. F.	Attacks of gallstone colic. Died from perforation.	Absent gallbladder. Stone in common duct.	Autopsy only.	
Dixon	1945	74 yrs. M.	Epigastric distress. Diabetes. Hemochromatosis.	Gallbladder absent. Com. duct normal. Liver normal.	Autopsy only.	
Dixon	1945	38 yrs. M.	Generalized miliary tuberculosis.	Gallbladder and cystic duct absent. Liver normal. Common duct normal.	Autopsy only.	

liver function test revealed only 0.3241 Gm. of hippuric acid recovered, compared to 0.5 Gm., or more, for the normal. This figure indicates considerable liver damage—a conclusion supported by gross examination of the liver at operation. The electrocardiogram, taken September 14th, showed changes interpreted as minimal evidence of myocardial damage. A plain film of the abdomen revealed no evidence of opaque biliary calculi. The right diaphragm was elevated. An upper gastro-intestinal tract roentgenologic study revealed no evidence of intrinsic pathology.

With a provisional diagnosis of cholelithiasis and obstruction of the common duct, the patient was prepared for operation. The preoperative management consisted chiefly

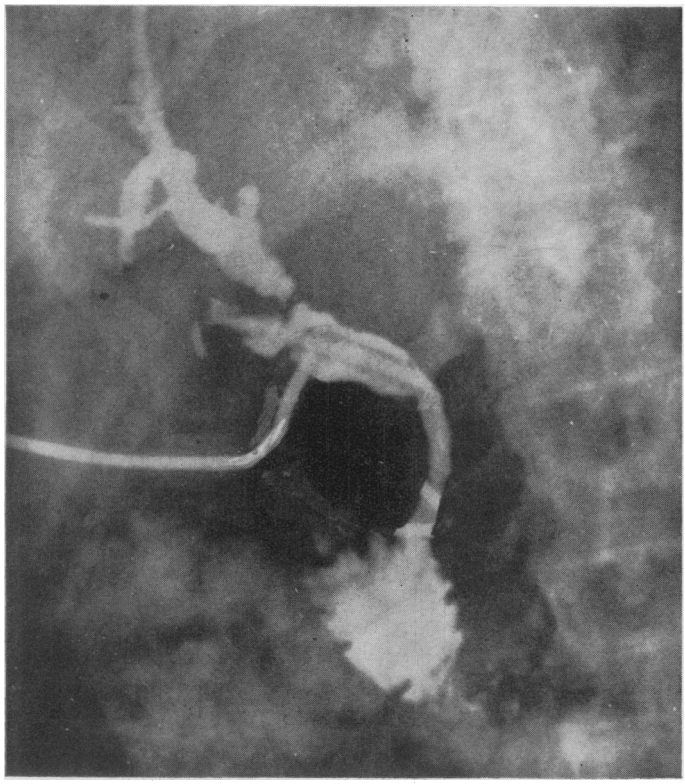

FIG. 2.—Cholangiogram of Case 1.

NOTE: The roentgenogram has been retouched for sake of clarity. The extra coil of the external limb of the "T"-tube has been obliterated.

of a high carbohydrate-high protein-low fat diet plus ten mg. daily of intramuscular vitamin K. Following this regimen the patient's prothrombin time was 24 seconds, the control being 25 seconds.

On September 22nd, using spinal anesthesia, the patient was operated upon. The liver was atrophic, nodular, yellow-brown in color, with streaks of scar tissue between the nodules. No gallbladder could be seen. The stomach was normal, as was the head of the pancreas. The duodenum was adherent to the liver and common duct. The common duct was markedly enlarged, being approximately three centimeters in diameter (Fig. 1). "White bile" was obtained from the common duct by aspiration. A large

stone, approximately two centimeters in diameter, could be palpated in the region of the ampulla of Vater. The extrahepatic ducts were thoroughly searched, but no structure resembling a gallbladder or cystic duct was discernible.

The stone was removed, and a "T"-tube inserted into the common duct after carefully probing the right and left hepatic duct.

A cholangiogram, October 3, 1945, showed the common duct to be patent, as the radiopaque material flowed into the duodenum. No structures resembling cystic duct or gallbladder were seen (Fig. 2). The patient was discharged from the hospital on the 17th postoperative day, having made an uneventful recovery. Following removal of the "T"-tube, cholecystography was attempted by the oral method, on November 23rd, to further rule out the possibility of an intrahepatic gallbladder, but no such structure was visualized.

Case 2.—Through the courtesy of Dr. J. A. Shacter, we are reporting a second case of congenital absence of the gallbladder. On March 20, 1946, Mrs. M. P. entered Wesley Memorial Hospital complaining of pain in the right upper quadrant, and an intolerance to greasy foods. These symptoms had been constantly present during the past three years, but the patient also stated that even as a small child she was unable to eat the high-fat diet of her family table. The pain began as epigastric distress gradually increasing in severity. The onset was in the late evening with the pain lasting 12 to 48 hours. Bicarbonate of soda gave only partial relief. Nausea, belching, and flatulence accompanied these episodes. A weight loss of ten pounds in the past six months was noted. The past history revealed an appendicectomy had been performed in 1928. The patient had been pregnant 13 times, and had been delivered of six living children. On physical examination, this 62-year-old white woman appeared to be in no apparent distress. Blood pressure 140/90; normal pulse. A few small, nontender lymph nodes were palpated in the left submaxillary area. The heart was of normal size, shape, and rhythm. The abdomen was obese, with a right lower quadrant scar. There was no tenderness or rigidity; and no masses or viscera were palpable.

The urine was normal. The erythrocyte count was 4,710,000 cells per cm., and the hemoglobin was 14 Gm. per 100 cc. The leukocyte count was 6,050 cells per cm.; and the blood Wassermann was negative. An oral hippuric acid test was within normal limits. The nonprotein nitrogen was 44.7 mg. per 100 cc. of blood. The total proteins were 7.05 Gm. per 100 cc., with the albumin and globulin fractions 3.70 Gm. and 3.35 Gm., respectively. Cholecystograms, made elsewhere, failed to visualize the gallbladder. The prothrombin time was within normal limits.

A preoperative diagnosis of chronic cholecystitis was made, and the patient operated upon on March 25, 1946. Careful inspection of the entire inferior surface of the liver failed to reveal a gallbladder. A small accessory liver lobe, 5 x 4 cm., was found at about the normal site of the gallbladder. The entire inferior and superior surface of the liver was readily palpated. Inspection of the liver surface itself failed to reveal what might have been a gallbladder buried within the liver substance. No cystic duct was found. Since the common duct was not opened, a "T"-tube was not utilized in an attempt to visualize an intrahepatic gallbladder.

Case 3.—Mrs. M. W., a patient of Dr. Paul Rhoads and Dr. Arthur Metz, entered Wesley Memorial Hospital May 16, 1946. She complained of yellow color of the skin, intense itching, nausea, and malaise, all of four weeks duration. Although she had been troubled with "biliousness" for many years, the first right upper abdominal colic occurred in May, 1938. This episode subsided, and the patient was symptom-free until April, 1946, at which time a painless jaundice was noticed. The inventory of systems revealed selective dyspepsia to fatty foods, clay-colored stools, and dark-colored urine.

On physical examination, this 60-year-old, white female, appeared deeply jaundiced and superficial excoriations of the skin were noted. Except for an old right lower quadrant scar (removal of suppurative appendix and drainage of abscess in 1938), the only positive physical findings were those mentioned above.

The laboratory reported a normal blood count and hemoglobin level. The urine was dark in color, with urobilinogen present on several occasions. The stools were clay-colored and positive for occult blood. The icteric index was 148 units, with a four plus prompt van den Bergh reaction. On admission, the prothrombin time was 50 seconds, with 25 seconds for the control. Following vitamin K therapy the prothrombin returned to 20 seconds, with a control of 20 seconds. The albumin-globulin ratio was reversed. An upper gastro-intestinal study revealed no intrinsic pathology, and the electrocardiogram was within normal limits.

A preoperative diagnosis of neoplasm involving the extrahepatic biliary system was made. At operation, May 23rd, a thorough search failed to reveal a gallbladder or cystic duct. The extrahepatic ducts were slightly enlarged and very firm, as if invaded by a new growth. The pancreas appeared normal except for evidence of passive congestion. The surgical impression was primary neoplasm invading the common and hepatic bile ducts. Absence of the gallbladder and cystic duct was a coincidental finding. Since the common bile duct was not incised, cholangiograms were not possible.

COMMENT: For evident reasons, a preoperative diagnosis of congenital absence of the gallbladder is extremely difficult, if not impossible. This fact is well illustrated in our survey by the ratio of cases found at autopsy to those discovered at surgery. There is no group of symptoms or signs which are of value in making such a diagnosis. Roentgenographic visualization of the biliary tract is the most accurate diagnostic method known at present. The accuracy of this test, when properly conducted, is about 90 to 95 per cent.[13] However, when this method fails to produce any shadow whatsover, congenital absence of the gallbladder is so rare that such a condition is not suspected, there being no reported case of the diagnosis made before operation or postmortem examination.

Should surgical procedure fail to reveal the gallbladder in or about its normal location, the presence of an intrahepatic location of this viscus should be ruled out. This can best be done, we believe, by retrograde cholangiography. Such procedure was reported in only three cases in the literature. Mackmull[11] injected a cadaver specimen; while Drenckhahn and Rogers,[12] and Dixon and Lichtman[5] illustrated the use of this method in the patient. This procedure of retrograde visualization of the biliary tree was carried out in one of the cases satisfactorily, and no evidence of the gallbladder or cystic duct was seen. Kehr mentions needling the liver and aspirating bile as proof of an intrehepatic viscus. We agree with Talmadge[5] who contends that aspiration of bile from the liver does not prove the presence of an intrahepatic gallbladder, inasmuch as bile may be aspirated from a dilated intrahepatic bile duct.

SUMMARY

1. The literature pertaining to congenitally absent gallbladder is reviewed.

2. A total of 71 cases of congenital absence of the gallbladder were reviewed and analyzed; 34 at operation and 38 at autopsy—one case appearing in both series.

3. We present three additional surgical case reports of congenital absence of the gallbladder. The absence of an intrahepatic gallbladder was proven by cholangiography in one case.

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