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Portal Vein Thrombosis

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I. ABSTRACT

PORTAL VEIN THROMBOSIS

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Terms

A thrombus in the portal vein may be infected, or non-infected. When infected, the process is termed "pylephlebitis"; if not infected, a "bland thrombus."

Incidence

1886, Fitz found pylephlebitis in 11 of 257 cases of appendicitis (4.3%) at necropsy.

1897, Armstrong reported pylephlebitis in more than 5% of 546 cases of appendicitis.

1903, Gerster reported 1,189 operative cases of acute appendicitis in which pylephlebitis occurred 7 times or 0.6%.

Colp, reviewing all the cases of appendicitis at the Mt. Sinai Hospital from 1916 to 1925, found 9 instances of pylephlebitis in 2,841 cases, or 0.3%.

Koster and Kasman reported 3 cases of pylephlebitis in 1,027 cases of acute appendicitis, an incidence of 0.3%, and 1 case in 112 cases of acute cholecystitis; an incidence of almost 0.9%.

At the University of Minnesota Hospitals, 4 instances of pylephlebitis, in addition to the one presented with this article, have been found in the necropsy records since 1933. Two of these were the result of an acute gangrenous process in the appendix which had ruptured and formed an appendiceal abscess prior to admission to the hospital. In the third instance, the source was not determined but was believed to be in the sigmoid. The fourth was associated with an arteritis of the splenic and mesenteric arteries. Multiple liver abscesses were present in three instances.

The decrease in incidence has been attributed to earlier diagnosis, as well as to better surgical handling of acute appendicitis. Statistics as to the incidence of this condition, which rely solely on clinical findings, and are not based upon operative or necropsy records are not reliable, and no attempt was made to correlate these.

Weir and Beaver, in reviewing all the necropsy records at the Mayo Clinic, found 127 cases in which suppurative and bland thrombotic processes involving the portal vein or its tributaries occurred. They were divided as follows:

1. Terminal bland partial thrombosis..... 54
2. Suppurative portal thrombosis.... 32
3. Bland portal thrombosis associated with disease of the liver, spleen or blood..... 32
4. Significant bland partial thrombosis associated with cardiac disease..... 7
5. Significant bland portal thrombosis associated with biliary disease..... 2

Exciting causes

In 32 cases of suppurative thrombosis of the portal vein mentioned above (2), the following conditions were listed as the underlying causes:

Infection of gallbladder and common duct.....	9
Appendicitis.....	6
Malignant disease of colon.....	4
Perforated peptic ulcer.....	3
Malignant disease of pancreas.....	2
Uncertain.....	2
Abdominal trauma.....	1
Pelvic abscess.....	1
Umbilical abscess.....	1
Gastric carcinoma.....	1
Pancreatic suppuration.....	1
Diverticulitis of sigmoid.....	1

The conditions found with terminal bland thrombosis (1-above) of the portal vein and its tributaries in 54 cases listed as follows:

		Total
Benign disease of biliary tract	5)	
" " " pancreas	2)	
" " " stomach	3)	13
" " " colon	3)	
Malignant disease of pancreas and bile ducts	6)	
" disease of stomach	5)	
" " " colon	13)	34
Miscellaneous malignant condition in abdomen	10)	
Cardiovascular disease	7)	7

Pathogenesis

A. Suppurative thrombosis of the portal venous system may occur in 3 ways:

1. Extension into portal vein from infectious process in contiguous tissue.
2. Secondary invasion of bland thrombus in the portal vein.
3. Propagation of infected thrombus or embolus from any tributary of portal vein.

It should be noted that pylephlebitis and abscess of the liver cannot be regarded as synonymous, the latter being almost invariably a sequel to the former. But not all abscesses of the liver result from suppurative thrombosis of the portal vein, as they may arise through one of four channels, hepatic artery, portal vein, bile ducts and lymphatics. As a rule, it is only when the infection travels by way of the portal vein that both pylephlebitis and hepatic abscess are present, and even then these two need not be associated. If the abscess is single and the result of a septic extension from appendiceal or superior mesenteric veins, it is usually found in the right lobe of the liver. This localization in the right lobe is said to be due to the presence of a dual current in the portal vein, first described by Serege and Glenard, who showed that blood from the splenic vein entered the left lobe of the liver, and blood from the superior mesenteric vein entered the right lobe.

B. The formation of bland portal vein thrombi is dependent on numerous factors, as varied as the associated pathologic states, but the important etiological factors have been enumerated by Weir and Beaver:

1. Injury to wall of portal vein from invasive malignant growth of contiguous tissue, from inflammation, from operative procedures, or other trauma.

2. Slowing of current of blood within portal vein, due to:

   (a) General circulatory failure.
   (b) Compression of portal vein.
   (c) Inhibition of natural propelling forces generally responsible for maintenance of portal circulation.
   (d) Cirrhosis of liver.

3. Propagation of thrombi from splenic or mesenteric veins.

4. Syphilis, polycythemia vera and migratory phlebitis may give rise to aseptic thrombosis of portal vein as complication in course of disease.

5. Isolated changes in wall, such as atherosclerosis of portal vein or branches.

C. Any constitutional disease which predisposes individual to venous thrombosis may result in thrombosis of portal vein. In polycythemia vera, leukemia and in certain infections, the accompanying rise in blood platelets is regarded as one of the etiological factors.

Pathology

Only the local and closely related findings will be enumerated here.

There is usually a moderate amount of ascites present. Generalized peritonitis is not an uncommon finding. The liver is enlarged and its surface may show numerous small light spots. On section, many small abscesses are seen scattered along the portal radicles; a variable number of larger abscesses may be present. Hepatic abscess [illegible] and

in all but one of the 32 cases in the series reported by Weir and Beaver. The portal vein is usually distended and free pus escapes on incision. No admisture of blood with the pus indicates that circulation has ceased within the vein. Softened, adherent and grayish-white thrombi are usually found on the wall of the vein.

Microscopically, the liver shows areas of necrosis scattered along the portal radicles and the picture of acute inflammation in the areas of the abscesses. Section through the portal vein shows a variable degree of acute inflammatory involvement of the wall of the vein. An adherent suppurating thrombus is invariably present, with some peripheral organization, and a central portion containing an abundance of polymorphonuclear leucocytes, areas of necrosis, and, often, colonies of bacteria.

The spleen is soft and enlarged, weighing up to 1200 grams and may show old and recent infarcts.

In the cases of bland portal thrombosis, e.g., those associated with splenic anemia, the thrombi may be of varying ages, with varying degrees of canalization and organization. If this process has existed for some time, there will be evidence of establishment of collateral venous circulation, as indicated by the dilated veins in the gastrosplenic ligament, on the abdominal surface of the diaphragm, in the lesser omentum, and in the falciform ligament of the liver. The umbilical vein in the round ligament may be patent, and the esophageal, left suprarenal, hemorrhoidal and prostatic veins may all show conspicuous dilation. The obstruction is entirely extrahepatic so that in chronic portal thrombosis, there are developed two sets of anastomoses. One is between the distal part of the portal vein and the inferior vena cava (the "hepatofugal" veins); the other is between the proximal portal or mesenteric veins and the liver, i.e., "hepatopetal" veins.

Symptoms

A definite diagnosis of pylephlebitis is difficult before the entire picture has developed but the following are of importance:

1. A history of antecedent infection in the abdomen.

Cases of appendicitis which develop pylephlebitis are usually advanced cases. The appendix in these cases is usually found to be gangrenous or badly inflamed. The average time of onset of the symptoms of phlephlebitis after the start of the history of appendicitis, in one series of cases, was 13.3 days or 6.7 days after appendectomy.

2. A history of chills, past or present. (40% in the series of Synder, Hall and Allen).

3. The patient is very ill, appears quite septic, has an elevated temperature varying between 101-102 to 104 to 105 daily. Sweating is quite marked, the pulse rapid, the patient complains of abdominal pain, and there is tenderness in the right hypochondrium or epigastric region.

4. The liver is enlarged and tender. Jaundice is an important diagnostic sign and is present in about one-half of the cases. Ascites and dilatation of the abdominal veins are not infrequent occurrences. The appearance of jaundice following an appendectomy should always arouse suspicion that pylephlebitis is developing as a complication. The spleen is usually palpable.

5. Malaise, vomiting, weakness and wasting are all present. Mental sluggishness and disorientation are not uncommon.

6. The diagnosis of bland portal vein thrombosis is often difficult because the most conspicuous symptoms, ascites without generalized edema, is more commonly the result of an uncomplicated cirrhosis of the liver or of a peritoneal or retroperitoneal neoplasm.

7. X-ray of the abdomen will show a high diaphragm on the right, with limitation of motion and is of value in differentiating a subdiaphragmatic abscess.

8. Laboratory findings:

(a) The leucocyte count varies between 9,000 to 40,000 with 60 to 80% polymorphonuclear leucocytes. In itself, it cannot be considered to be of great diagnostic importance.

(b) The urine shows bile, albumin and blood cells.

(c) Blood cultures are seldom positive. Koster and Kasman state that cultures of the blood in these cases are usually negative as in most instances the invading organism is E. Coli, which, when it breaks into the blood stream, is quickly destroyed there.

Differential Diagnosis

1. It is often difficult to distinguish pylephlebitis from primary hepatic abscess. A history of etiological factors, as described above, and the early appearance of jaundice will suggest pylephlebitis. An associating history of amebiasis or other causes of liver abscesses will often enable the proper diagnosis to be made.

2. Hepatic vein thrombosis is characterized by pain in the upper right quadrant of the abdomen, radiating to the back or to the right or left scapular regions, a huge tender liver, the simultaneous appearance of splenomegaly and the sudden development of ascites.

3. Bantis disease, cirrhosis of the liver and neoplasm of the peritoneum may all be very difficult to differentiate from bland portal vein thrombosis. Here again, the history of sepsis will be of utmost importance in making the differentiation.

4. Echinococcus cyst of the liver may be differentiated by means of the history, complement fixation test and exploratory puncture.

5. Suppurative cholangitis may be differentiated on the basis of a history of previous attacks of gallstones and an earlier and more pronounced jaundice.

6. X-ray is of considerable help in differentiating from a subdiaphragmatic abscess.

7. In warm climates, malaria is sometimes confused with this condition but a careful examination of the blood for plasmodia and a therapeutic test of quinine will often enable the correct diagnosis to be made.

Other pathological states which have been diagnosed as pylephlebitis are:

1. Acute yellow atrophy of the liver.
2. Acute pancreatitis.
3. Bronchopneumonia with pyelitis.
4. Coronary occlusion.
5. Pus in the lesser peritoneal cavity.
6. Empyema
7. Tuberculous peritonitis

Prognosis

The mortality in pylephlebitis is very high. Almost all observers report a mortality of at least 90%.

Synder, Hall and Allen report the mortality from pylephlebitis to be 92% in their series.

Brutt reported a mortality of 95%.

Recovery has occurred in cases believed to be pylephlebitis. Barnes and Pearson reported a case of recovery in which laparotomy was performed, a suppurative appendix removed and liver abscess drained. This patient's temperature subsided to normal in 8 days and following this he made an uneventful recovery.

Barlow reported an apparently fulminating case of pylephlebitis which recovered with only intravenous injection.

Treatment

A. Prophylaxis: The prevention of this condition is part of the entire question of surgical treatment of suppurative abdominal conditions, especially appendicitis. It has been

suggested that if during the course of an appendectomy the surgeon finds that there is no bleeding on cutting the meso-appendix because of thrombosis in the appendiceal vein, he is entitled to consider this an instance in which pylephlebitis may develop later, and it is here that ligation of the ileocolic vein may be practiced. If a frank suppurative phlebitis of the mesentery is evident at operation, ligation of the ileocolic vein should be performed before removing the appendix. These methods have been described by Gerster, Wilms and Braun.

Colp has reviewed the literature on the value of ligation of the portal vein in this condition and concluded that such an operation is probably never indicated. The studies of Newhof and Beer have demonstrated that the "hepatopetal" veins in the gastrohepatic omentum which anastomose between the portal and the mesenteric veins may, in certain persons, efficiently carry on the portal circulation after complete occlusion of the portal vein by ligation. If, however, the process has extended for any appreciable distance into the liver, this procedure will be in vain.

B. After a liver abscess has formed, exploration and drainage should be performed in two stages, either through the chest wall with attachment of the pleura to the diaphragm or by laparotomy with attachment of the peritoneum overlying the liver to the parietoperitoneum. Halstead reported a case treated successfully by the latter maneuver.

C. Supportive treatment consists in maintaining the proper level of the nutrient and fluid intake, and in giving numerous small blood transfusions.

SUMMARY

1. Thrombi in the portal vein may be infected (pylephlebitis) or non-infected (bland thrombi).

2. Any suppurative process in the abdomen, situated in a region drained by the portal vein, may give rise to pylephlebitis. The most common underlying causes of this condition are:

(a) Infections of the gallbladder and common duct, (28% of cases).

(b) Appendicitis (19% of cases).

3. Present day statistics place the incidence of pylephlebitis complicating acute appendicitis at 0.3%.

4. Bland thrombi in the portal vein are dependent upon varied associated pathological states:

(a) Injury to the wall of the vein.
(b) Slowing of the blood stream within the vein.
(c) Propagation of thrombi from the tributaries of the portal vein.
(d) Syphilis, polycythemia, or migratory phlebitis.
(e) Atherosclerosis of the portal vein or its branches.

5. Pylephlebitis may occur in 3 ways:

(a) By extension from a nearby infectious process.
(b) By secondary invasion of a bland thrombus.
(c) By propagation of an infected thrombus or embolus from a tributary of the portal vein.

6. Not all abscesses of the liver result from a pylephlebitis as they may develop through the hepatic artery, the bile ducts and the lymphatics.

7. Suppurative processes in the region drained by the superior mesenteric vein usually give rise to abscesses in the right lobe of the liver. Those in the region drained by the splenic and inferior mesenteric veins usually abscess in the left lobe of the liver. This is explained by the presence of a dual current in the portal vein.

8. Pathology: liver abscesses are almost invariably present. The portal vein is distended and free pus is present. Softened, adherent, grayish-white thrombi are found on the wall of the vein. Microscopically, the picture is that of acute inflammation with multiple abscesses.

9. If thrombosis of the portal vein has existed for some time, there will be evidence of a collateral venous circulation, between the portal and systemic systems as well as between the liver and the mesenteric veins.

10. The clinical picture of pylephlebitis:

(a) History of antecedent abdominal infection accompanied or followed by chills, malaise, vomiting and weakness.

(b) Patient is very ill and septic, temperature is elevated, pulse is rapid and sweating is marked.

(c) The liver is enlarged and tender and jaundice is present in about one-half of the cases and usually appears early. The appearance of jaundice following appendectomy should always arouse the suspicion that a pylephlebitis is developing.

(d) X-ray shows an elevation and limitation of motion of the diaphragm.

(e) Leucocytes vary from 9,000 to 40,000 with 60 to 80% polymorphonuclears.

(f) The urine shows bile, albumin and blood cells.

(g) Blood cultures are seldom positive as the usual invading organism, E. coli, is quickly destroyed in the blood stream.

11. Bland thrombosis of the portal vein is frequently confused with cirrhosis of the liver or abdominal neoplasm because the most conspicuous symptom is ascites without generalized edema.

12. The more important conditions in differentiate portal vein from are:

(a) Thrombosis of hepatic vein.
(b) Banti's disease.
(c) Echinococcus cyst of liver.
(d) Hepatic abscess.
(e) Cirrhosis of liver.
(f) Subdiaphragmatic abscess.
(g) Malaria
(h) Suppurative cholangitis.
(i) Abdominal malignancy.

13. The mortality in pylephlebitis is above 90%. A case of spontaneous recovery has been reported.

14. Prevention, if possible, is said to consist in the earlier diagnosis and proper surgical treatment of suppurative abdominal conditions.

15. Ligation of the ileocolic vein is advocated at appendectomy when there is evidence of thrombosis of the appendiceal vein. Ligation of the portal vein is probably never indicated.

16. If a liver abscess has formed, exploration and drainage should be performed whenever possible in two stages.

17. There is no specific treatment. Numerous small blood transfusions are indicated as a supportive measure.

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II. CASE REPORT

PORTAL THROMBOPHLEBITIS. SUPPURATIVE CHOLANGITIS.

Case is white male, 41 years of age, admitted to University of Minnesota Hospitals 4-27-33 and expired 8-13-33. Total stay - 108 days.

Indefinite abdominal pain

4-22-33 - Awakened in morning with indefinite pain over abdomen. 4 A.M. - Pain became more severe, intermittent in character, seemed localized in right epigastrium. Did not radiate to back or shoulders. Up and about and did work on this day.

Jaundice

4-23-33 - Pain more constant, intermittent and sharp. Slight jaundice noted. Constipation marked. Vomited several times. Definite anorexia. Given pills for constipation but obtained no relief. Enema given. No change in color of stool. Fevernish. Urine dark brown.

Increased jaundice

4-25-33 - Jaundice definite. Pain not severe. Vomited once.

4-27-33 - Admitted. Pain diminished in severity. Jaundice increased. Appetite improved slightly, although he did not eat much. Continues to feel feverish. No chills. Physical examination: well-developed and well nourished; moderate icterus. Throat - slight injection and enlarged tonsils. Chest - negative, blood pressure 120/74, cardiac rhythm, rate and sounds apparently normal. Abdomen - slightly distended, midline scar (4" long) from umbilicus to pubis; small incisional hernia present in lower third of scar; possible slight amount of fluid present; slight rigidity along inferior costal margin on right side (most marked toward midline); one small area of tenderness below costal margin in nipple line; no rebound tenderness or palpable mass; liver palpable 3 cm. below costal margin.

Laboratory

Blood - Hemoglobin 82%, white blood cells 13,500, neutrophiles 79%, lymphocytes 19%, basophils 1%, monocytes 1%. Blood chemistry - icteric index 32 units, Van den Bergh - biphasic reaction, 2 - 5% maximum color in 30 seconds, 10 - 15% maximum color in 60 seconds. Stool - greenish brown, unformed, contains mucus, benxidine negative, urobilin and urobilinogen present. Urine - increased urobilin. Blood - Wassermann and Kline - negative. Urine - occasional red blood cell, few white blood cells, urobilin and bile salts +. X-ray of gallbladder - calcified density in region of gallbladder of very irregular type, may represent calcification in gallbladder although this is not definite.

Progress

Temperature 102. Pulse 95. Respirations 26. Weight - 165 lbs.

Improved - chills, fever

5-6-33 - Regular rise in temperature every day, ranging as high as 102.6. Chill. Temperature 103.6. Duodenal tube in place. Magnesium sulphate injected from time to time and nasal suction instituted. White blood cells ranged around 11,000. Icteric index slowly dropped, readings are 40, 24 and 22. Hemoglobin dropped to 70%. Bleeding time, 45 seconds; clotting time, 4-3/4 minutes.

Chills, fever - Operation - Cholecystotomy

5-19-33 - Temperature more septic. Several chills, during these temperature rose to 103. For past 3 days, had chill every day. Icteric index - 16 units. Blood cultures, negative. Agglutination for typhoid, paratyphoid, B. melitensis and tularemia - negative.

Operation

Findings - Liver practically normal in color. Edges rounded and soft. Gall-bladder soft, normal in color and appearance, shows no definite evidence of infection or pericystitis. Stomach normal. No gall stones palpated in gall-bladder or common ducts. Pancreas moderately firm, possibly slightly enlarged, does not have appearance of carcinoma. Some very large lymph nodes in region of common duct. Otherwise exploration of common duct by palpation is normal. No attempt made to explore common duct itself. Cholecystostomy done by fixing rubber tube into gall-bladder and bringing it out through a stab wound. Following operation, transfusion of 500 cc. whole blood given.

5-22-33 - Temperature dropped slightly since operation. No chills. No drainage of bile through catheter although there is some drainage around drain left through wound.

No improvement

6-1-33 - Temperature now similar to that before operation. Chill and temperature (reaches 103.8). Hemoglobin 67%, white blood cells 11,000. Icteric index - 12 units.

Septic

6-5-33 - Some bile drainage appeared through catheter. Temperature more septic than previously.

Purulent drainage

6-12-33 - Temperature continues as before. Hemoglobin 55%. Blood cultures and agglutinations - negative. White blood cells 12,000. Purulent drainage around catheter. 10 cc. thick pus obtained. X-ray of chest - some elevation of diaphragm but no parenchyman disease.

6-22-33 - Pus still draining from around wound.

6-25-33 - Given 750 cc. citrated blood. Temperature still septic, ranging up to 102. Blood cultures - negative. Liver function - 1st specimen 15%, 2nd 10%. Icteric index, 20 units. Hemoglobin 50%. On one occasion, icteric index dropped as low as 8 units.

Weaker

7-1-33 - Condition unchanged. Losing weight. Becoming much weaker. Temperature lower. Icteric index, 32 units.

7-6-33 - NPN - 28.5 mgs. Van den Bergh - 40 to 50% maximum color in 30 seconds, 65 to 75% in 60 seconds. Icteric index, 32 units. Temperature subnormal a good deal of time, occasional rises to 100.

Ascites

7-17-33 - Ascites. X-ray of abdomen confirms. Paracentesis yields bile stained ascitic fluid.

Paracentesis

7-18-33 - 5 quarts ascitic fluid removed. Temperature subnormal with occasional rises to 102. Hemoglobin 66%.

7-25-33 - Parecentesis done again, 1200 cc. obtained. Temperature subnormal. Appears very ill, rapidly losing weight, becoming extremely weak.

Progressive decline

8-5-33 - Condition approximately same. Pus still draining from operative

incision. Wound open and Dakin's irrita-tion begun. Rapidly losing weight and going downhill. Temperature almost always subnormal. Fecal fistula at site of one paracentesis wound.

Death

8-13-33 No change except weight loss. Temperature has been subnormal throughout. Mentally confused. Respirations shallow. Rapid pulse. Chest hyper-resonant. Blood pressure 80/70. Expired at 1 P.M.

Emaciation - draining sinuses

Autopsy

Body is well-developed, poorly nourished, white male, 41 years of age, measuring 170 cm. in length and weighing approximately 100 lbs. No malformations. Marked emaciation. Rigor disappearing. Hypostasis purplish and posterior. No edema or cyanosis. Jaundice 2+. Pupils equal, each measuring 6 mm. in diameter. Three scars on abdomen: one below umbilicus extending to symphysis, 2 crusted draining sinuses; one more recent scar in right rectus region in upper part of which is drainage of some foul purulent material; stabwound lateral to this through which a catheter is protruding and pus exudes around this. Numerous petechiae over body. Over trochanters and bony parts of sacrum, there are several reddened areas (not ulcerated). Subcutaneous fat practically absent. Musculature pale.

Multiple peritoneal abscesses

Peritoneal Cavity is practically obliterated. Subdiaphragmatic spaces, areas around spleen, around colon and greater portion of small bowel are obliterated by fine web-like adhesions (almost universal.) Between webs of these adhesions, there is thin, greenish fluid with a few flakes of fibrin. Small bowel, colon and under-surface of liver are fused into a continuous mass which is firmly bound down to the under-surface of abdominal wall around operative site. About midline, a coil of bowel is attached to anterior abdominal wall. As this is cut away, a sinus between the crusted draining area on outside of skin, through abdominal wall into bowel is revealed. There are 2 crusted areas on the skin but only one sinus is found in the bowel. A second sinus may have been present; because of very dense scar tissue, it is not recognized. As the bowel is separated away and the dense adhesions are broken down, there is a large quantity of pus liberated from between the coils of bowel plastered together under the liver. About 200 cc. of pus is released. Pus is very thick and of greenish color, has foul odor. No sulphur granules. Pus is in innumerable loculations among coils of bowel. It has penetrated through the abdominal wall along the operative incision and along the stab wound. Appendix is fused into lower edge of mass of bowel and adhesions, described in right upper quadrant. When dissected, it is intact and has not been perforated as far as can be determined. Wall is rather thin and flabby. Lumen contains some thickened feces. Appendix is not swollen, shows no scar-ring or abscess, and appears to be separate away from abscess in remaining portion of abdomen.

Left Pleural Cavity contains 600 cc. slightly greenish, clear fluid. Right Pleural Cavity is obliterated by adhesions. Pericardial Sac contains no excess fluid or adhesions.

Heart weighs 325 grams, is brown and somewhat soft. Musculature not hypertrophied. Cavities not dilated. Mural endocardium smooth. Valves well formed. Myocardium shows no infarction or softening. Vessels at base are well-formed and have normal relation to each other. Root of Aorta shows no evidence of syphilis. Very little atheromatous change present. No ectasia. Coronaries soft and show no atheromatous plaques.

Emphysema, Atelectasis

Left Lung weighs 550 grams, Right 600. Base of right lung shows considerable edema. Upper lobe of right side shows emphysema. Bronchi contain considerable mucus in larger branches. Left lung is about 50% collapsed in lower lobe, probably compression collapse due to fluid. Ghon tubercle in lateral portion of lower half of upper lobe. No active or old healed tuberculosis seen in apices or in hilar glands. Lymph nodes of hilus and bifurcation of trachea not appreciably

enlarged.

Passive congestion

Spleen weighs 300 grams, is large, has rounded edges and firm consistence. On cross section, practically no pulp can be scraped away. Appears fleshy and somewhat fibrous.

Portal Thrombophlebitis. Liver Abscesses

Liver, Gall-Bladder and bile ducts: Under-surface of liver is fused with numerous abscesses among bowel, described above. Hilum of liver and region of gall-bladder and bile ducts is very fibrotic and extremely difficult to dissect. Dissection of this system is begun by opening duodenum starting at lower end of ducts. As soon as the duodenum is opened and pressure is applied on the liver and ducts, thick green pus oozes from ampulla of vater and from accessory pancreatic duct. Common bile duct is opened lengthwise. It is incorporated within heavy fibrous tissue, described above. Mucous surface is red and puffy. No stones present. Duct not dilated. No stricture found. Duct filled with thick, green pus. Cystic duct small, not obstructed, contains no stones. Gall-bladder has been opened and attached to skin at site of cholecystostomy. Wall heavy. Mucosa is bright red. Pus present in its lumen. No stones. Hepatic duct is incorporated within a mass of fibrous tissue. It is likewise inflamed and filled with pus. No stones.

Portal Vein

Portal vein is dissected along with remaining structures. It is extremely thick walled, measuring 2 to 3 mm. in thickness. Inside of portal vein is a dirty black-brown color and covered with shaggy exudate and lumen is filled with pus. Dissection of tributaries of portal vein toward bowel shows that not all of the branches are involved. Splenic vein near spleen and toward tail of pancreas is not thickened. Some of branches of vein from bowel near the upper part of small bowel likewise do not appear involved. Branches to middle and terminal parts of bowel are thrombosed and infected similar to main trunk itself. Veins near sigmoid colon are involved. Numerous abscesses are present adjacent to vein in mesentery.

Liver weighs 2000 grams. On superior surface of right lobe, there are 2 projecting areas in which numerous yellowish foci are visible through capsule. On cross section, interior of liver contains unnumerable abscesses which range up to 2 cm. in diameter but most are less than 1 cm. The radicals of both the portal vein and bile duct are dilated, filled with pus and present about the same characteristics as the common duct and portal vein, respectively. The areas about these sutures are fibrotic and infiltrated with pus. Some of the abscesses in the liver are not liquefied. The contents are semi-solid and like caseous tubercles. The liver substance is of a dark brown color with exaggeration of the liver markings.

Secondary Changes (?)

Gastro-Intestinal Tract: Esophagus and stomach show only postmortem change. Duodenum is extremely red, and pus drips out of the ampulla of Vater and accessory duct to the pancreas. Small bowel is thick, edematous, discolored and has a great deal of exudate on its surface. This exudate is purulent where the coils of bowel have been involved in the abscess and shaggy and fibrous in the remaining portions. The surface of the bowel shows no tubercles that can be recognized grossly as such.

The colon, in general, presents approximately the same characteristics. The cecum is quite red and inflamed; no ulceration or tuberculosis. The transverse and descending colon are very edematous. Sigmoid is red. Rectum is bright red. About the rectosigmoid junction, there is an abscess in the wall. This apparently has come through from the mesentery. No ulceration, diverticulae, Meckel's diverticulum or polyps seen. Mesentery of practically all of bowel and colon contains abscesses. Most of these abscesses appear to be broken-down lymph nodes or abscesses within the vessels. Abscesses are most marked at root of mesentery and in mesentery of sigmoid colon.

Abscesses. Pus in ducts.

Pancreas shows several foci of fat necrosis. Remainder of pancreas is somewhat glassy and edematous. Fibrous

numerous abscesses throughout, particularly on the posterior surface adjacent to the attachment of the mesentery. Ducts contain pus.

Adjacent to the cecum in the base of the mesentery and near the head of the pancreas, there are collections of large calcified lymph nodes. The nodes at the cecum are the largest. In one plaque, there are 7 or 8 which measure about 2 cm. in diameter, calcified but not caseous.

Adrenals are imbedded in fibrous tissue. On the right side, an abscess is adjacent to the adrenal but the gland is separate and apparently not involved. Appears somewhat thin. Medulla and cortex are well demarcated. No abscesses present.

Pyelitis

Capsules of Kidneys strip easily. They are not involved in the inflammatory reaction within the peritoneum. Surface of kidneys is smooth, deeply congested and very swollen. Each kidney weighs 190 grams. Cut surface is well demarcated in pyramids and cortices. Marked congestion present. No abscesses noted. Pelvis of right kidney somewhat dilated. Urine somewhat turbid. Mucosa of pelvis not injected. Ureter on right side is dilated to about 1 cm. in diameter.

Bladder is dilated and shows a slight amount of trabeculation and some cystitis at the base.

Prostate is small and soft. No adenoma present. No metastases. Prostate appears somewhat edematous and greenish. Seminal vesicles show no inflammation.

Aorta is of good caliber throughout. Minimal atheromatous plaque present in dorsal retion.

Organs of Head and Neck - not examined.

Musculature about Spine is examined for a possible focus of infection which may have extended into abdomen but none is found.

Conclusions

1. Portal thrombophlebitis (source indefinite).
2. Secondary suppurative cholangitis (?).
3. Multiple abscesses of liver, bowel, peritoneum and mesentery.
4. Emphysema.
5. Atelectasis.
6. Splenitis, acute.
7. Pyelitis, acute.
8. Fistula of bowel.
9. Empyema.

Bacteriology

Search for actinomyces in pus and tissue is negative. Flora is mixed cocci and coli aerogenes group.

III. CASE REPORT

THROMBOPHLEBITIS OF PORTAL VEIN.

Case is white male, 80 years of age, admitted to University of Minnesota Hospitals 7-29-35 and expired 7-31-35 (2 days).

Indefinite History. Pain, Jaundice.

No history obtainable from patient. Relatives state that he had been ill for about one week prior to admission, but character of illness is not very certain. Apparently had had some pain in abdomen and became jaundiced. Also dyspnea and palpitation of heart.

7-29-35 - Admitted. Physical examination: Appears seriously ill, complains of abdominal pain. Marked icterus. Heart is irregular, rate about 120. Fibrillating. Chest hyperresonant and shows crepitant rales in left base. Liver palpable one finger below costal margin. Slight tenderness over most of abdomen, more pronounced in right upper quadrant. Rigidity in this area. Slight edema of the ankles. X-ray of chest shows diffuse increased density in the lungs, suggesting congestion. There is diffuse dilation of aorta. Progress: Temperature 105.6

7-30-35 - Condition worse. Complains of abdominal pain. Pulse irregular. Respirations shallow and labored.

7-31-35 - 1:45 A.M. - Expired.

Autopsy

Body is poorly nourished, white male, 80 years of age, measuring about 179 cm. in length and weighing approximately 140 lbs. Rigor present. Hypostasis purplish and posterior. Slight edema of ankles. No cyanosis. Jaundice 2 +. Pupils equal.

Blood Tinged Fluid

Peritoneal Cavity contains about 200 cc. of blood-tinged fluid. Appendix hangs free. Pleural Cavities free of adhesions and excess fluid. Pericardial Sac negative. Heart weighs 360 grams. Musculature of good color. No fibrosis or softening. Some calcification of base of mitral valve, extending into base of aortic valve. Edges of valves are thin. Root of Aorta shows moderate atheromatous change. Coronaries are beaded with atheromata in the main trunks, the smaller tributaries are open. Right Lung weighs 660 grams, Left 450. Both lungs are emphysematous. In base of left small patch of early bronchopneumonia. At right base, several such scattered areas. The Spleen, soft, weighs 220 grams.

Intrahepatic Portal Phlebitis

The Liver weighs 1530 grams. It is yellow, soft and friable. When cut across, thick, purulent, blood material exudes out of all branches of portal vein. In some of the branches exudate is bright yellow pus and in others, hemorrhagic material. Around each of the veins, there is a zone of red, semi-necrotic appearing liver tissue.

The Gall-Bladder negative. Wall thin. No stones. Mucosa smooth.

Proctitis - Thrombophlebitis

Gastro-Intestinal Tract: Stomach, duodenum, small bowel and colon as far as the sigmoid show no change. No areas of inflammation, granuloma, ulcer, diverticula, or tumor. At the junction of the sigmoid to the upper two-thirds or one-half of the rectum, there is a zone in which the bowel is red, edematous and there are superficial ulcerations over the tips of the edematous folds. There are no old ulcers, tumors or diverticulae. This zone involves the entire circumference of the bowel, extends up and down for a distance of about 5 inches. Beginning at this point, the inferior mesenteric vein is thrombosed throughout its length and at its junction with the portal; the main portal itself is thrombosed from this point on through into the liver. The other branches of the portal are not involved. In the mesenteric vein, the lumen contains pus and the entire tissue around the vein is hard and indurated. The portal vein is filled with soft, jelly-like, red pus. The pancreas is soft. No pancreatitis. No tumors or cysts.

Adrenals well formed.

Kidneys weigh 320 grams together. Capsules smooth. Cortices somewhat pale. Pelves not dilated or reddened.

Bladder shows no cystitis or tumors.

The prostate is relatively small and soft. No adenomas or tumors.

The aorta is markedly tortuous due to arteriosclerosis. Intima is cracked here and there over the atheromatous plaques. No evidence of syphilis.

Head and Neck - not examined.

Diagnosis

1. Cellulitis of rectum.
2. Thrombophlebitis of portal vein.
3. Multiple abscesses of liver.
4. Emphysema of lungs.
5. Bronchopneumonia.

IV. MOVIE

Title: Historic Mexico City

Released by: Metro-Goldwyn-Mayer.

V. LAST WEEK'S MEETING

Date: October 10, 1935.

Place: Recreation Room,
Nurses' Hall.

Time: 12:15 to 1:15.

Program: Movie (The Land of the Eagle)
Influenzal Meningitis
Remarks (Director Amberg)

Present: 100

Discussion: W. Thompson
R. W. Koucky
H. A. Reimann
J. C. McKinley

VI. NEXT WEEK

The Staff Meeting will not start until 12:30 P. M. to allow every one to hear Dr. Morris Fishbein speak at Convocation from 11:30 to 12:20.

Next week's subject will be "Annual Review of Appendicitis Problem at University of Minnesota Hospitals".

Visiting physicians will be our guests.

An invitation has been extended to Dr. Fishbein to attend the Staff Meeting.

VII. HOME-COMING CLINICS for MEDICAL SCHOOL ALUMNI and OTHER MINNESOTA PHYSICIANS

October 24 and 25,
-1935-

MINNESOTA ALUMNI!

You are cordially invited to attend a series of clinics which will be held at the University of Minnesota Hospitals during Home-Coming Week. Many of you will no doubt wish to attend the game with Northwestern and this plan will enable you to combine professional advancement with your joy in the game. The clinics, which will be conducted by leading Minnesota physicians, are under the joint auspices of the University of Minnesota Medical School, the Minnesota State Medical Society, and the Minnesota Medical Alumni. A program of the sessions and details of registration will be found on the pages following.

Offered to All Minnesota Physicians.

HOME-COMING CLINICS TO BE HELD AT UNIVERSITY OF MINNESOTA HOSPITALS

Thursday, October 24, 1935

A.M.		EUSTIS		TODD
9:00- 9:25	Dr. P. F. Dwan	Rheumatic Heart Disease	Dr. I. McQuarrie	Nutritional Problems in Childhood
9:25- 9:50	Dr. M. Barron	Anemia	Dr. J. C. Litzenberg	Obstetrics
10:00-10:25	Dr. G. E. Fahr	Treatment of Congestive Heart Failure	Dr. C. A. Stewart	Childhood Tuberculosis
10:25-10:50	Dr. M. H. Nathanson	Coronary Disease	Dr. H. M. N. Wynne	Remarks Concerning Hysterectomy
11:00-11:25	Dr. H. L. Ulrich	Hypertensive Heart Disease	Dr. E. J. Huenekens	Infections in Childhood
11:25-11:50	Dr. J. A. Myers	Tuberculosis	Staff	Neurology

P.M.				
12:30- 1:30	Staff Meeting, University of Minnesota Hospitals, Dr. W. A. O'Brien, chairman. Luncheon will be served.			
2:00- 2:25	Dr. A. V. Stoesser	Allergy in Childhood	Dr. E. L. Meland	Bladder Tumors
2:25-2:50	Dr. L. G. Rigler	Radiographic Interpretation	Dr. W. A. Fansler	Rectal Disease
3:00- 3:25	Dr. C. D. Wright	Gastro-Intestinal Diseases	Dr. A. A. Zierold	Surgical Abdominal Problems
3:25- 3:50	Dr. R. Newhart	Ear Infections	Dr. W. H. Cole	Fractures
4:00- 4:25	Dr. W. R. Shannon	Pseudo Birth Injury in the Newborn	Dr. O. J. Campbell	Disease of the Breast
4:25- 4:50	Dr. S. E. Sweitzer	Dermatological Diagnosis and Treatment	Dr. V. L. Hart	Orthopedics

A.M.		EUSTIS		TODD
9:00- 9:25	Dr. C. D. Creevy	Prostatic Disease	Dr. C. J. Watson	Jaundice
9:25- 9:50	Dr. R. Swanson	Obstetrics	Dr. H. Reimann	Pneumonia
10:00-10:25	Dr. H. E. Michelsen	Dermatological Treatment	Dr. F. H. Schaaf	Newer Therapy
10:25-10:50	Dr. R. V. Ellis	Allergy	Dr. F. C. Rodda	General Pediatrics
11:00-11:25	Dr. S. B. Solhaug	Ovarian Cysts	Dr. T. A. Peppard	"The So-Called Irritable Bowel"
11:25-11:50	Staff, Department of Ophthalmology	Iritis, Diagnosis and Treatment	Dr. A. H. Beard	Diabetes Mellitus

P.M.

12:30- 1:30 Luncheon will be served in hospital dining room, followed by a meeting of the University of Minnesota Medical School Alumni Association.

2:00- 5:00 Surgical operative clinics, 5th floor University of Minnesota Hospitals. Drs. O. H. Wangensteen, W. T. Peyton, M. H. Manson, H. Carlson, N. L. Leven, and associates.

3:00- 4:00 Open house: Department offices, Medical School and University of Minnesota Hospitals.