

SHOEMAKER (G.E.)

SUPPURATIVE PYLEPHLEBITIS AND HEPATIC
ABSCESS SECONDARY TO APPENDICITIS:

With a Report of Two Cases.

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GEORGE ERETY SHOEMAKER, A.M., M.D.,
VISITING SURGEON TO THE METHODIST HOSPITAL OF PHILADELPHIA.



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**SUPPURATIVE PYLEPHLEBITIS AND HEPATIC
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BY GEORGE ERETY SHOEMAKER, A.M., M.D.,
VISITING SURGEON TO THE METHODIST HOSPITAL OF PHILADELPHIA.

SUPPURATIVE pylephlebitis with hepatic abscess is so unusual during life and frequently so obscure a condition before death that it is often overlooked and seems to receive but little attention among clinicians, particularly those of England and America. Two cases that occurred in my practice within the past few months have been reported to the College of Physicians, of this city,¹ and are of more than usual interest. It may be worth while narrating them, to call renewed attention to a disease the literature concerning which is widely scattered and not abundant.

CASE I. *A common pin in the vermiform appendix; secondary retro-peritoneal infection; liver-abscess.*—H. P., aged eighteen years, a furniture polisher, when admitted to the surgical ward of the Methodist Hospital, under my care, was suffering from urgent symptoms of bowel-obstruction, fecal vomiting, and subnormal temperature, with well-marked peritonitis. He died about thirty hours after an

¹ Nov. 2, 1892; Feb. 3, 1893.

operation undertaken for abscess about the appendix. The condition of the liver and retro-peritoneal tissues was unknown at this time and was completely masked; otherwise no operation would have been undertaken, as death was inevitable.

The history previous to admission was meager and unsatisfactory. The man had been ill twelve days, chiefly under home treatment. The trouble began with a chill and coincident pain in the right hypochondrium, which his sister since states he then referred to as "that old pain." When first seen, about an hour before the operation, he had had complete obstruction of the bowel for five days, and obstinate constipation for several days before that. There had been fecal vomiting and subnormal temperature for twelve or fifteen hours, with evident peritonitis.

The abdomen was very tense; the walls edematous, dusky, and more resistant in the right iliac region, though from the great tension the presence or absence of a tumor could not be made out. The breathing was thoracic, 36 to the minute; the temperature, 95.6° ; the pulse, from 90 to 118; the mind clear, though there had been delirium. There was no pain, but a gulping up rather than a vomiting of a thin, brown, offensive fluid, with decided fecal odor. Well-applied efforts to move the bowels with calomel, salines, and gravity enemata of glycerin, magnesium sulphate, and turpentine, had failed. There was no discoverable evidence of liver-disease; no jaundice, soreness, redness, venous obstruction, or demonstrable enlargement of the organ; while there was evident obstruction of the bowels, with peritonitis, which the dusky and edematous appearance referred to the right side.

Incision in the right semilunar line disclosed pus and thick, yellow lymph as soon as the peritoneum was cut. Two or three ounces of pus were washed out from a cavity within the peritoneum, limited

outwardly by the pelvic wall and inwardly by adherent coils of intestine. The intestines were tightly distended, and as adhesions appeared to limit the pus-cavity on all sides, it was not considered wise to search for and remove the appendix, which could not be readily felt. Taking into consideration the man's condition, such an attempt at this time would have been highly dangerous. The abscess-cavity was flushed with boiled water and packed with iodoform-gauze about a glass drain, the incision being left open. This procedure gave great relief. The temperature rose to normal, and in a few hours reached 99.8° . The vomiting and hiccough stopped at once, while large quantities of urine began to be excreted, no less than eighty-seven ounces being obtained by catheter at short intervals in the next twenty-four hours. The improvement did not, however, continue beyond this period. The bowels did not move and the patient sank rapidly on the evening of the day following the operation, the pulse having never been over 120, the temperature never over 100.6° .

Post-mortem examination showed the cavity at the seat of operation to be clean and sweet. The parietal surface of the great omentum was bathed in pus, which had apparently come from a ruptured abscess of the liver. There was no pus upon the intestines, but a very recent general peritonitis had caused slight general bowel-adhesions and a widespread deposit of fresh, translucent coagulated lymph, which, immediately below the liver and in the left flank, was yellowish and in semi-solid masses. The appendix extended downward, and, though much distended, inflamed, and surrounded by recent plastic deposit, was apparently not perforated; nor was it gangrenous. It contained a common pin, head downward. There was purulent inflammation of the connective tissue and veins behind the peritoneum and along a path, two or three inches

wide, from the root of the mesentery upward along the front and to the left of the spinal column. This area, infiltrated with grayish pus, extended a short distance between the layers of the gastro-hepatic omentum and involved the structures at the transverse fissure of the liver. This organ contained within its substance an abscess, apparently acute, situated near the surface of the right lobe at its highest point. The pus, which was yellow, occupied a single irregular cavity, in size 2 by 2 by 3 inches. The liver was easily separated from the diaphragm, pus appearing as soon as this was attempted. As has been said, the front surface of the great omentum was covered with fluid pus, which had not yet reached the intestinal surfaces in visible quantities.

It is probable that the *general* peritonitis was caused by the rupture of the hepatic abscess and leakage in front of the liver and omentum after the operation. Had the whole attack, including the obstruction of the bowel at the time of operation, been due to a general and not to a localized peritonitis, it seems improbable that merely washing out the circumscribed area, which was shut off in the right side, would have caused the improvement noted for the first twenty-four hours.

It is also probable that the pin in the appendix was the original cause of trouble, and that it had, by piercing the appendix-wall and the adjacent peritoneum, infected the underlying connective tissue and veins. These veins belong to the portal system and reach the liver directly, though the neighboring lymphatics enter the general venous circulation by way of the thoracic duct, which enters the left subclavian vein. The abscess was single and was large

for one of secondary infective origin. In size, however, it has a parallel in the following case: ¹

A French soldier, previously healthy, had pylephlebitis following appendicitis. At the autopsy there were found several abscesses of the liver, one of them large, involving the whole thickness of the left lobe of the liver. The portal vein was filled with pus to its smallest ramifications. There was jaundice, but no tympany and no general peritonitis.

It may be remarked in passing that the finding of a genuine foreign body in the appendix, such as this pin, is very rare; as almost all the bodies so found are fecal concretions, like that in the case about to be narrated. They may bear general resemblance to "cherry-stones" or "grape-seeds," but are not such in reality.

CASE II. *Gangrene of appendix; acute suppurative inflammation of branches belonging to portal system; multiple liver-abscesses.*—This was almost a typical case of pylephlebitis suppurativa as described by Frerichs.²

W. S., seventeen years of age, a packer, had been previously healthy. His father died of enteric fever and the mother of apoplexy, though a year before her death I removed a sarcoma of the breast and axilla.

On November 12th, after eating indigestible food for breakfast and dinner, the patient worked for some hours in an unusual draught of cold air, and was seized with pain just below the navel, together

¹ Archives de Médecine et de Pharmacie Militaires, Paris, 1891, xviii, 62.

² Clinical Treatise on Diseases of the Liver, 1861.

with nausea, followed in the evening by vomiting. Two solid stools passed that day.

On the 13th the abdominal pain became localized in front of the anterior superior spinous process of the ilium on the right side, passing a little backward along the iliac crest. There was some difficulty in fully extending right thigh. He had a severe chill in the evening, followed by sweating and vomiting. He had one solid stool.

On the 14th localized pain continued. There was some nausea and gulping, but no vomiting; he had two severe chills (shaking his bed distinctly) in the evening, and two in the night, followed by profuse sweating. He had one loose stool.

I saw him for the first time on the 15th, finding him up and dressed at 9 A.M., with a temperature of 99° and a pulse of 90; the conjunctiva was yellowish; there was decided tenderness over the appendix, with resistance of muscles, but no tremor and no pain, even on full breathing; there was no tympany, and his mind was unusually clear. The diagnosis of appendicitis was announced, and the family informed that operation would have to be considered, though not at that moment demanded.

The patient now passed out of my hands, and I am permitted to report his subsequent history by courtesy only. He was admitted to one of the out hospitals, where he had immediately a violent chill, followed by a temperature of 104° and sweating. Until this time the diagnosis did not appear at all obscure, but when seen next day by the visiting surgeon, the presence of appendicitis was not deemed positive, because local tenderness had disappeared from the right iliac fossa; nor was it developed by rectal pressure. There was not even thickening to be felt by the rectum, and the resistance of the abdominal muscles was slight. The abdomen was not only flat, but scaphoid.

From this time on until death, eight days later, the obscurity of the diagnosis deepened. Jaundice set in and soon became intense; there was almost complete insomnia, but no delirium. Full doses of quinine seemed to moderate the chills, while small doses of calomel were followed by frequent, thin, greenish stools. There was a very irregular temperature, reaching a little over 103° or dropping to 99° . The liver was somewhat enlarged but not tender; the spleen was also enlarged. Rapid emaciation followed, with brown, dry tongue; dulness succeeded the abnormal brightness of intellect, and the patient died a few hours after he had been transferred from the surgical to the medical ward, and just twelve days after the onset of the disease.

Autopsy. Emaciation was deep and there was universal jaundice. The heart was normal; the lungs were moderately adherent at the apices, without abscesses. The kidneys were large, congested, the capsule stripping easily; no pus was present. The spleen was about twice the normal size and contained at least one hemorrhagic infarct as large as a pea. The intestines were not distended. There was no sign of general peritonitis; white gauze dipped in the scanty serum at the bottom of pelvis stained clear yellow. On lifting the cecum and separating extremely slight adhesions, about two drams of thin, gray, flocculent pus appeared about the appendix, which was thickened and gangrenous in its middle third, a slough about three-quarters of an inch in length having dropped out of the posterior wall. A concretion, one-quarter of an inch in diameter, afterward shown to be fecal and not a gall-stone, occupied the upper and healthier third of the appendix, completely filling its lumen. An area of parietal peritoneum upon which lay the appendix was grayish-black and gangrenous, showing a small point of perforation. Behind the peritoneum and extending

obliquely upward from the root of the appendix toward the front of the spine was an infiltrated area of loose connective tissue and veins, which could be picked up within the abdominal cavity like a section of bowel. This, when cut across, was seen to be inflammatory, the distended veins within it having thickened walls with a dirty-gray lining, and containing a grayish fluid. The liver was somewhat enlarged, and was studded throughout with abscesses of from a line to an inch in diameter. These were very numerous about the transverse fissure. The gall-bladder was distended by a mucoid fluid. No gall-stones were found. The brain was normal. No pus was found elsewhere than described. There were numerous ulcerations of the mucous coat of the cecum, the majority about two lines in diameter and the largest a third of an inch. One of these encircled the opening of the appendix into the cecum. This opening barely admitted a probe.

The points of interest in this case are two: Its obscurity from a diagnostic point of view after the liver-symptoms appeared; and the secondary venous and hepatic involvement.

The clinical history emphasizes the fact, well known to many, that the most serious disease of the appendix, going on to gangrene and perforation, may exceptionally exist without the traditional signs of peritonitis; without tenderness on deep pressure, without a demonstrable tumor, without tympany and without pain, at least in the later stages. There can be no reasonable doubt that operation on the third or fourth day would have saved this man's life by forestalling the infection of the veins, just as it would have killed him, or, at least, would have done no good, after the pyemic symptoms had set in. It

is such cases as these, growing more obscure as the patient grows worse, that add force to the reasoning of those who would operate at once in every case as soon as the diagnosis of appendicitis is made. While there is, however, so large a preponderance of recoveries from attacks which do not go on to pus-formation, that position seems to me to be extreme.

The diagnosis of suppurative inflammation of the portal vein, or pylephlebitis suppurativa, is a matter on which there is no small difference of opinion. Some writers consider a diagnosis during life to be a mere matter of guesswork, while others consider it quite feasible. As has been suggested by Von Schüppel, this discrepancy can be accounted for by the obscurity or clear character of the cases individual writers may have seen. As few men see more than two or three cases, they are likely to generalize from these. The following points are given¹ as the most important in recognizing the condition :

1. "The presence of an affection which we know from experience may act as a starting-point . . . especially perityphlitis, a purulent focus, or an ulceration of the stomach, intestine," etc.

2. "Pain in the epigastrium, above the umbilicus, or in the right hypochondrium, or any other situation in which the pylephlebitis may start."

3. "Violent chills, which are repeated at irregular intervals, and are followed by great heat and profuse sweats, while the temperature in the intermission remains abnormally high (and pyemia in the ordinary surgical sense is excluded)."

¹ Ziemssen: *Cyclop. of the Practice of Medicine*, ix, 822 et seq.

4. "The recent, uniform, and painful enlargement of the liver. Enlargement of the organ is not constant, but tenderness is always present."

5. "Considerable enlargement of the spleen, especially when we can follow its development."

6. "The icteric color of the skin and urine in addition to the biliary diarrhea."

7. "Rapid emaciation of the body and profound loss of power."

8. "The occasional development of diffuse peritonitis and typhoid symptoms in the later stages of the disease."

Jaundice is said to occur in three-fourths of the cases by Von Schüppel. Eichhorst¹ says that it is almost always present, while in liver-abscess, not pyemic, it is one of the rarer symptoms. Rouis² found it to be present in 17 per cent. of the 258 tropical cases included in his statistics. Waring³ found it in somewhat less than 6 per cent. of his cases. All observers note the violence of the chills. There is seldom time for the development of ascites or of enlargement of the superficial abdominal veins. Only one termination is known for the disease, and that is death. This occurs within two weeks as a rule; sometimes after five or six.

The abscesses of the liver are usually very numerous and small, from the size of a pin-head to that of a cherry, though they may reach a much larger size.

¹ *Specielle Pathologie und Therapie*, 4th ed., 1890, ii, 452.

² *Recherches sur les Suppurations endémiques du Foie*, etc. Paris, 1860; p. 189.

³ *An Inquiry into the Statistics and Pathology*, etc. Ed. John Waring, Resident Surgeon of Travancore, 1854, iii.

The walls of the portal vein or of any contributing branches which may be involved are thickened, reddish or grayish; the intima is of a dirty gray, swollen, and at times much softened or completely broken down. The contents of the inflamed veins are described as ichorous, putrid, dirty-gray or reddish-brown fluid. The connective tissue adjacent is also frequently inflamed and infiltrated.

All abscesses of the liver were supposed by Budd¹ to be secondary to inflammation or ulceration somewhere in the digestive tract, as in dysentery, ulcer of the stomach, or proctitis. This theory, though for a long time received with hesitation (Flint,² Frerichs³), is gradually gaining ground, and is probably, in the main, correct. At the time of examination the original disease may not be present, or it may not be found, while the embolus, which was the means of transfer, has long before disappeared in the resulting abscess. Many of these cases of ordinary liver-abscess run a prolonged course, as is well known, and often give rise to little disturbance of the system. They either rupture spontaneously, or are drained by operation, with a resulting cure in a considerable proportion of cases.

Very different, however, is the clinical picture when from the same starting-point—an appendicitis, for example—the portal vein or its contributing branches become involved in a suppurative inflammation, as in the last case narrated. These cases all die (Osler, Eichorst, Von Schüppel), and die quickly, with symptoms of the most violent charac-

¹ Diseases of the Liver, 1853.

² Practice of Medicine, p. 604, 1880.

³ Loc. cit., p. 116.

ter. The abscesses which form in the liver are multiple, and before large areas have time to break down death occurs from septic intoxication. It is not always that the inflammation spreads to the portal vein by continuity along a contributing branch, but a septic embolus may be the means, as in the tropical abscess. In each case the embolus traverses the same vessels. In the tropical abscess there is at some period abundant involvement of intra-hepatic portal radicles. What is the cause of the different result? It seems to me that it must be sought in the different characters of the infective material. There must be different ptomaines involved, probably different bacteria, so that in the one case a comparatively mild and circumscribed hepatitis is set up, and in the other an infective inflammation of all points of contact in liver and vein, which rapidly destroys life by a species of pyemia. It is these cases which are comparatively rare. An examination of late volumes of the *Index Medicus* will disclose from one to two cases per year reported as pylephlebitis, but seldom in American or English periodicals, the German predominating. For the same period the literature of ordinary hepatic abscess is enormous. References to some typical cases are given below.¹

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¹ Sonnenfeld: Wiener med. Presse, 1885, xxvi, 1259. Colquhoun: Lancet, London, 1887, ii, 606. Aufrecht: Berlin. klin. Wochenschr., 1869, S. 308. Payne: Path. Trans., 1871. Many references to German and French sources are given in Ziemssen's Cyclopaedia of Practice of Medicine, ix, 805.

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