Clinical Department.

ACUTE PRIMARY "IDIOPATHIC" PHLEGMONOUS GASTRITIS. REPORT OF A CASE WITH BACTERIOLOGICAL FINDINGS.*

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By this term is generally understood a purulent inflammation of the stomach wall, which most often localizes in the submucosa, and there leads to more or less extensive pus formation, but which may also affect the mucosa, muscularis, submucosa and serosa.

On the basis of etiology, phlegmonous gastritis is divided into two classes.

1. The primary or idiopathic forms, in which a direct cause is not demonstrable.

2. The secondary forms. The latter are again divided into three sub-groups.

- a. Direct extension phlegmons, *i.e.*, those following oesophagitis.

- b. Local secondary phlegmons which follow ulcer, carcinoma, or an operation.

- c. Metastatic secondary phlegmons, in which infection of the stomach follows infection of some other part of the body.

On the basis of pathologic anatomy two forms are distinguished: the diffuse phlegmonous gastritis, in which the pus infiltration spreads diffusely over a considerable area, and the circumscribed form or gastric abscess. Strictly speaking, only the former should be included among the cases of phlegmonous gastritis.

The primary or idiopathic phlegmonous gastritis (Hosch¹) is the most frequently encountered, and our case also belongs to this class. It is not our purpose to give an extensive review of the literature, as several excellent reviews are on record, among the more recent of which is that of Bossart² in 1912, which consists of a comparative study of all cases which the author was able to find with a complete bibliography. Among the earlier papers may be mentioned those of Schnarrwyler,³ Robertson,⁴ Adams,⁵ Koenig,⁶ Jensen,⁷ in 1911, recorded the detailed histories of ten Danish cases, and found 121 on record elsewhere. In the literature before 1911, Bossart was able to collect 110 cases of circumscribed and diffuse phlegmonous inflammations of the stomach wall. Emge,⁸ in 1914, thought that only one-half the cases reported, or about 50, were true primary, or idiopathic phlegmonous gastritis. It is difficult to make a correct estimate without running down each case recorded, but most authors are agreed that the idiopathic variety is the one most commonly observed.

The history of this case was kindly furnished me by Dr. Paul Oliver of the Surgical Staff, of the Cook County Hospital.

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The patient was a laborer, 58 years old, of Swedish nationality. He entered the hospital Dec. 24, 1915, because of a large inguinal hernia on the right side, which had slowly grown in size during a period of eight years. It had never been painful or irreducible, and he wished to have it attended to because of some discomfort and interference with heavy work. He also stated that a swelling of the left forearm had developed and persisted, following a fall upon the hand one week previously. He had noted some edema of the ankles during the past year. About a year ago the patient had been in the hospital on account of broken compensation. He further stated that at one time he had been a heavy drinker, but had not taken liquor in recent years. He had had some nocturnal frequency of urination. Otherwise the history was negative.

Upon physical examination, the man appeared to be obese, with ruddy complexion and a moderate acne rosacea. The mouth was in bad condition; teeth decayed, pyorrhoea alveolaris, tongue swollen and covered with a brown coat. The tonsils showed some enlargement. Except for a few mucous râles the lungs were negative. The heart dulness was increased. There was a systolic murmur transmitted to the axilla and an accentuated second pulmonic. The abdomen was negative, except for the liver, which was palpable two fingers' breadth below the costal margin. In the inguinal region there was a large hernia which passed down the inguinal canal and was easily reducible. A smaller hernia was present on the left side. There was a soft edema of both ankles. There was swelling and discoloration of the left forearm and hand which the Roentgen plate showed to be due to an incomplete fracture of the radius. The urine showed a specific gravity of 1.012, a trace of albumin and granular and hyaline casts. From these findings a clinical diagnosis of right complete indirect, inguinal hernia, left incomplete hernia, decompensated heart lesion, chronic nephritis and recent left Colles fracture was made.

After a period of appropriate medical treatment the patient improved, so that on Jan. 4, 1916, a repair of the right hernia under local anesthesia was made by Dr. Ryan. The post-operative course was normal in every way until the night of January 15, about ten days after the operation, when he complained of a chill and his temperature rose to 101.6 with a pulse of 96. He then complained of pains in the abdomen, especially in the epigastrium. During the next three days the temperature ranged between 99.8 and 102, with the pulse becoming more rapid and the pains more severe. During the night of January 17, the patient moaned all night because of the severe pain. He did not vomit at any time.

Dr. Oliver first saw the case on the 18th of January and found the patient with flushed face and apparently in distress, with respirations rapid and shallow, temperature 101.2, pulse 130, irregular and weak. The abdomen was rigid and somewhat tender in the epigastrium. The patient pointed to the epigastrium as the source of the trouble. There seemed to be slight shifting dullness in the flanks. A probable diagnosis of general peritonitis was made, the source of which was difficult to determine, a perforated gastric ulcer being thought most likely. Immediate operation was decided upon, and under ether anesthesia, the abdomen was opened through a right rectus incision. As soon as the peritoneal cavity was entered a few ounces of a dark sero-pus escaped. Exploration of the lower

abdomen was negative, except for a hernia on the left which contained an appendix epiploicum. There was only slight injection of the peritoneum in this region, but the serosa of the bowel of the upper abdomen showed marked injection and some fibrinous exudate. The omentum was found firmly wrapped about the stomach, just as it is often found about an acute appendix. Upon its separation, a large amount of pus escaped and revealed the stomach a uniform angry red, having the appearance of a very acutely inflamed appendix. The wall was stiff, thick, indurated and edematous and the process seemed to involve the entire organ, stopping abruptly at the pyloric sphincter. The duodenum beyond could be felt thin walled, and apparently normal. The pylorus felt like a solid mass. Careful search was made for a perforation without success. Nothing further was attempted, the patient being put to bed with tubular drainage in the suprapubic stab wound and upper abdomen. The patient did not react after operation and died in about eight hours.

The post-mortem examination was made on the afternoon of the same day, January 19, 1916, by Dr. H. Gideon Wells, to whom I am indebted for the abstract from his autopsy records and for permission to work up the case.

The following is an abstract of the autopsy notes on this case:

External Appearance. The body is that of an obese man. There is a slight yellow coloration of the skin. The superficial lymph glands are not palpable, except those of the right groin. There is some edema of the legs. The scrotum is reddened, and in the right side is an indurated mass about 5 cm. in diameter, continuous with the testicle. There is a little hernial bulging on the left side. In the anterior abdominal wall are two openings, one above the pubic crest and extending to almost the costal margin above the outer border of the right rectus. There is another drain in the lower portion of the latter.

On palpation, there is an indurated mass along the course of the right inguinal canal. Above the umbilicus is a circular nodule in the subcutaneous fat, 7 mm. in diameter. There is a recently healed surgical wound parallel to the right Poupart's ligament.

Abdominal Cavity. There is a considerable amount of subcutaneous fat, about 5 cm. in thickness. The upper drainage tube passes between the liver and stomach, deeply into and over the lesser curvature to reach the retroperitoneal tissue. The peritoneal surface is very much reddened. The lower tubular drain runs into the cul-de-sac of Douglas in which there is a small amount (30-50 c.c.) of purulent fluid. A cigarette drain runs between the intestines. The peritoneal coat is injected, but for the most part is shiny. In the interstices of the peritoneum are pockets of purulent fluid but no large accumulations of fluid, and no fibrin. The left inguinal ring is open and admits two finger tips up to the external ring, after which only one finger tip is admitted. The right inguinal ring is closed. The mass consists here of fibrofatty tissues in which are small hemorrhages. There is no evidence of infection or suppuration here. The peritoneal surface in this region is puckered, but no infection is present.

The mesenteric lymph glands are not enlarged, but there are small pigmented spots and a small calcified fibrous strand in the peritoneum. The ap-

pendix is free but atrophied. Such pigmented areas are also found 20 cm. above the ileocecal valve. There is one particular area, one mm. in diameter, in which the peritoneum is necrotic and a necrotic tissue protrudes, but there is no perforation. There are no perforations in the small intestines. In the lesser peritoneal cavity there is a little purulent fluid but no perforations. The lesser peritoneum is free from adhesions. Some fibrous adhesions are present about the spleen; also a few about the sigmoid. The diaphragm is at the fourth interspace on the right side, and at the upper border of the sixth rib on the left.

Gastro-intestinal Tract. In the intestines there is a small pigmented area in the ileum, but no ulcers. The stomach shows a markedly hyperemic but smooth serosa, and on palpation shows diffuse thickening of the wall but no great induration. The thickening is most marked above the pylorus. On the lesser curvature is a point of irregular perforation limited to the serosa where the drainage tube was located. There are no adhesions about this perforation. Upon opening the stomach, the mucosa is seen to be diffusely hyperemic with hemorrhages beneath the mucosa. Folds of mucosa are missing. There is a purulent infiltration of all parts of the wall, and when cut, creamy yellow pus oozes out. The infiltration extends from the cardiac orifice to the pyloric valve, where it stops abruptly. The mucous membrane is everywhere intact. The purulent material forms a layer apparently in the submucosa, one to four mm. in thickness, the pus oozing out slowly from the cut surface everywhere. The stomach is heavy and boggy, weighing 440 grams, emptied. No ulceration or tumor masses are visible. The mucous membrane is swollen and edematous. There is some hemorrhage in the mesogastrium, but no adhesion about the stomach.

Other autopsy findings are indicated by the following:

Anatomical Diagnosis. Acute diffuse phlegmonous gastritis. Acute suppurative peritonitis. Two recent laparotomy wounds with drainage. Recent healed herniotomy incision. Remains of hernial sac in right inguinal canal with hematoma. Chronic interstitial nephritis (red granular kidney). Thrombotic occlusion of anterior coronary artery with anemic necrosis in left ventricle. Hypostatic congestion and edema with broncho-pneumonia in right lower lobe. Slight atrophic cirrhosis and passive congestion of the liver. Parenchymatous degeneration of the liver and spleen. Fibrous perisplenitis. Senile arteriosclerosis with atheroma. Edema of lower extremities. Obliteration of left tunica vaginalis testis. Necrotic masses in serosa of ileum. Lipomatous nodule in anterior abdominal wall. Diffuse fibrous myocarditis.

Histological Examination. The surface of the mucosa shows only post-mortem changes. Just beneath the surface there are small areas of hemorrhage. In the deeper portions of the mucosa, bordering on the muscularis, there is a diffuse infiltration of polymorphonuclear leucocytes. Scattered throughout this area are small masses of bacteria. The muscle bundles in the muscularis mucosa are widely separated and the spaces between are filled with polymorphonuclear leucocytes. In the upper layer of the submucosa there are two large circumscribed areas consisting of polymorphonuclear leucocytes and small masses of necrotic connective tissue and elastic fibers. Between these two areas of

suppuration the submucosa shows only a diffuse infiltration and some fibrin. The deeper layers present the same picture. The serosa is normal. Apparently the pus infiltration of the submucosa occurred in two bands separated by a wall which had not yet been replaced by pus, and only the upper layers of the submucosa were involved in the process. The diagnosis was acute interstitial phlegmonous gastritis, with marked suppuration of the submucosa and muscular coat, but no ulceration of the mucosa.

Bacteriological Findings. Microscopical examination of pus smears taken from the wall of the stomach and stained with methylene blue showed pus cells filled with chains of diplococci, four to six pairs to the chain. Scattered through the field were single diplococci. Careful search of several smears failed to reveal any other organism. Other smears stained with Giemsa's stain showed that the pus consisted almost entirely of polymorphonuclear neutrophils containing this same short-chain streptococcus, for the most part intracellularly.

Cultures were made on January 19, 1916, the day of the autopsy. After searing the surface of the stomach wall, pus was aspirated with a sterile Pasteur pipette. The contents were then smeared over the surfaces of agar and blood serum tubes, and also inoculated into broth. In addition, some of the material was plated on blood agar. Both aerobic and anaerobic cultures were made; the anaerobic cultures being placed in a Novy jar, and the pyrogallie acid method used to obtain anaerobic conditions.

After twenty-four hours the broth showed a uniform turbidity. On shaking, a faint cloud could be seen whirling through the medium. On slant agar there were discrete, dewdrop, moist colonies, each about 0.5 mm. in diameter. Growth was very poor. On Loeffler's blood serum, there was a pale, yellowish-brown slimy growth covering the surface. The surface of the medium had not been digested. Smears from these cultures showed the same organism that had been observed in the pus smears, and here, too, it occurred in short chains and diplococci. It retained the stain by Gram's method of staining. Subcultures made on all the ordinary laboratory media gave all the cultural characteristics of the streptococcus group. On blood agar (human blood), there appeared small pin-point colonies, with a clear zone of hemolysis about one mm. wide around them. The cultural characteristics were the same, both aerobically and anaerobically.

The fermentative reactions of this organism were studied, both aerobically and anaerobically. For this purpose three loopfuls of a twenty-four hour old broth culture were inoculated into tubes containing ten c.c. of sugar-free broth, and one per cent. of the sugar or carbohydrate to be tested. In addition, each tube contained enough of a one per cent. litmus solution to give it a distinct blue color, and a small inverted fermentation vial to show gas formation. The substances tested were dextrose, lactose, saccharose, raffinose, dulcitol, arabinose, salicin, sorbitol and xylose.

The aerobic culture fermented dextrose, lactose and salicin with enough acid formation to impart a distinct red color to the litmus, but without gas formation. The cultures containing dulcitol and sorbitol were very faintly acid, and showed no gas. Saccharose, raffinose, arabinose and xylose were unaffected.

Anaerobically the organism fermented all the sub-

stances tested with the formation of acid. In the cases of dextrose, lactose, saccharose, dulcitol, sorbitol and xylose enough acid was formed to completely discharge the color of the litmus. Gas was formed in all the tubes, varying in amount from a single bubble in those containing saccharose and salicin, to a tube-full in those containing dextrose, raffinose, dulcitol and sorbitol.

Experiments were also carried out in an attempt to reproduce this condition in animals. A rabbit was injected intravenously with a twenty-four hour broth culture and died the next day showing all the findings of an acute toxemia except an enlarged spleen. The organism was recovered in pure culture from the heart's blood. Several animals were thus treated with the same result.

Two guinea-pigs were injected intra-cardially with one c.c. each of a twenty-four hour culture and died the next day of acute toxemia. In none of the rabbits or guinea-pigs did the stomach show any lesions whatever.

An aseptic operation was performed upon a dog in which a twenty-four hour growth on agar suspended in sterile salt solution, was injected into the wall of the stomach, through a laparotomy wound. The animal recovered from the operation and the wound healed, but no symptoms of illness developed after two weeks. The same dog was then given an intravenous injection of another twenty-four hour growth, without showing any ill effects after forty-eight hours. A subsequent intra-cardiac injection of the same organism caused death within twenty-four hours. A streptococcus was isolated from the heart's blood, but the stomach was normal, with the exception of a small hemorrhagic area in the serosa, representing the site of injection.

A similar operation was performed on another dog, who also recovered, and after two weeks seemed to be perfectly normal. Because of the statements in the literature that alcohol seems to be a predisposing factor, we introduced 50 c.c. of 50 per cent. alcohol into the stomach of this dog on several successive days, by means of a stomach tube. A twenty-four hour broth culture of the same organism was then poured over the meat which was eaten by the dog at a single meal. The animal became ill, with spells of vomiting for two days, but later recovered and seemed to be as well as ever. One month after the operation this dog was given an intravenous injection of a twenty-four hour culture, and was killed the next day. A careful examination of the stomach at autopsy showed only a few punctate hemorrhages beneath the mucosa; otherwise it was normal.

In most of the cases thus far reported streptococci have been found commonly in pure culture, rarely associated with other organisms such as the staphylococcus, or the *Bacillus coli communis*. Muentzer⁹ reports a case in which the pneumococcus was apparently the causal organism.

As early as 1893 Pilliet¹⁰ states that according to Straus and Sebillon the infiltration of the stomach wall, and especially the connective tissue of the submucosa, is caused by death or injury to the mucous membrane, through which streptococci may gain access to the depths. He further states that in producing the disease, experimentally, mineral acids or organic acids are of no avail, since they lead to the formation of a scar or only to a hemorrhagic infiltration of

the wall. By introducing 5 gm. of zinc chloride (1-10), over a period of three days, into a rabbit, he succeeded in causing the formation of a scar and edematous swelling of the mucosa and submucosa, so that the thickness of infiltrate was 0.1 to 1 cm. No bacteria were demonstrable in sections. His conclusion is that acute fibrinous infiltration of the stomach wall is brought about only with the caustic solutions, such as zinc chloride and not with coagulating substances. This, according to him, is the first step in the production of phlegmonous gastritis. Our own experiments along these lines were negative, in the case of a dog. Kleineberger's patient¹¹ received 3.5 gms. of potassium iodide in three days, and this author believes that the iodide entered the stomach through defects in the mucosa and set up an inflammatory process which later led to phlegmonous gastritis. Bossart (loc. cit.) believes that idiopathic phlegmonous gastritis has its points of origin in the flat epithelium at the cardiac end of the oesophagus, and that this region is especially favorable for the localization of streptococci.

More recently, Cheinisse¹² has directed attention to the unappreciated occurrence of acute gastritis in infectious diseases. He has encountered several cases of erysipelas and mumps in which the gastric symptoms dominated the clinical picture and could be explained only by assuming a localization of the specific infectious process, the erysipelas or the mumps, in the stomach. Cheinisse had a case of phlegmonous gastritis in 1909 which developed in the course of an apparently mild erysipelas. Baird¹³ also reports a case which ended fatally shortly after the development of facial erysipelas. Vasilevsky¹⁴ also believes that phlegmonous gastritis is a secondary complication in the course of some general infection such as erysipelas. In his case the erysipelas spread to the mucosa of the mouth, pharynx, oesophagus and stomach.

We are inclined to believe that local injuries are not an important factor in the causation of this condition, but rather that it represents the localization of an organism, analogous to subcutaneous phlegmon, and to be explained on the basis of the anatomy of the organ, and possibly thrombosis of the vessels of the submucosa. As bearing upon the rôle of focal infections in the production of phlegmonous gastritis, the case of Schwarz¹⁵ is of interest. In his case the infection apparently spread from the tonsils, which were swollen and ulcerated. In the latter, as well as in the fauces and in the peritoneal exudate, numerous streptococci were found. Schwarz believes that a portion of the cases, at least those with unknown sources of infection, might be explained as due to infection from the tonsils. It should also be remembered that this condition is practically never diagnosed except at autopsy. It would be well to bear in mind that this condition is a possible complication in acute infectious diseases and to suspect it whenever acute stomach symptoms develop, especially when ac-

companied by sudden pain in the epigastrium, and severe vomiting of material containing pus.

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Medical Progress.

PROGRESS IN OPHTHALMOLOGY.

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ÆTIOLOGY OF GLAUCOMA.

R. H. ELLIOT has written on the ætiology of glaucoma, discussing various theories. We quote him as follows. We cannot base our ætiology on the pathology, but on clinical observations tempered by a study of the changes produced both in the eye and in the organism by those conditions of life which may be causative of the disease. Glaucoma being a label for a large group of conditions, the distinctive feature of which is increased tension, must have a widely different ætiology in different cases. Once increased tension is established, every sign and symptom finds a ready explanation.

The different factors cited as causes of glaucoma may be brought under one or the other of two headings: (1) Those which influence the balance of secretion and excretion of the intra-ocular fluid. (2) Those which directly or indirectly determine a change in the vascular conditions prevailing within the eye. Taking up the more interesting observations, we have, changes in the vortex veins. Ligature of these veins in animals produces increase in tension, but thickening and obstruction of these veins in glaucoma is rare. Disease of the vessels, brought forward as a cause, may usually be put down as a result, of the increased tension.

Schnabel in 1892 thought that cupping of the disc was due to an acute atrophy of the nerve itself. The lamina cribrosa was not pushed back by pressure, but pulled back by shrinking of the nerve. Small spaces were formed, and at length a large cup—a cavernous atrophy. But this cavernous atrophy has been met with in eyes that never have had increased tension, and no signs of it are found in most glaucomatous