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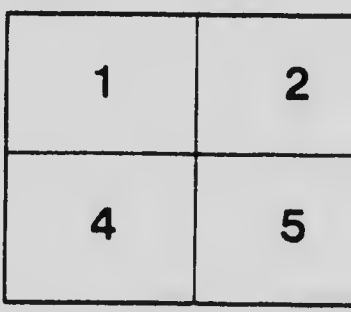

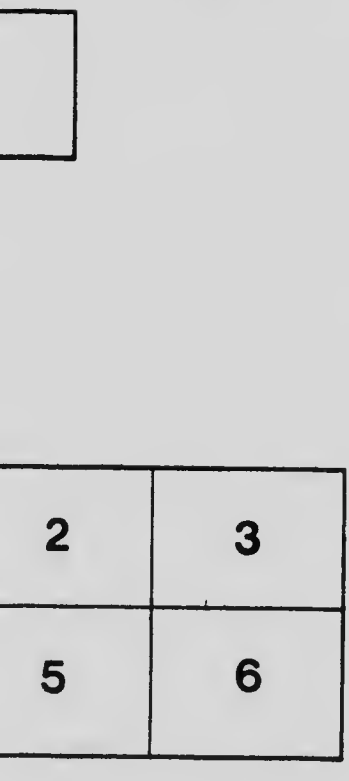

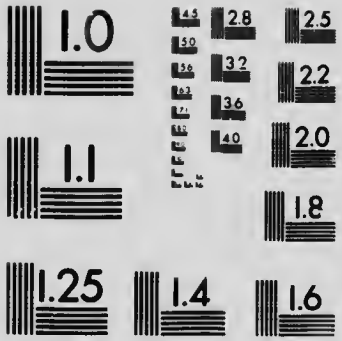

UNIVERSITY OF TORONTO STUDIES

PATHOLOGICAL SERIES

No. 2: A CASE OF ACUTE PHLEGMONOUS GASTRITIS

BY J. J. MACKENZIE

(REPRINTED FROM THE CANADIAN LANCET, VOL. XL.)

No. 3: PHLEGMONOUS GASTRITIS REPORT OF A CASE

BY CHARLES J. WAGNER

(REPRINTED FROM THE CANADIAN PRACTITIONER AND REVIEW, VOL. XXXII.)

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A CASE OF ACUTE PHLEGMONOUS GASTRITIS.

J. J. MACKENZIE, B.A., M.B.
Professor of Pathology and Bacteriology, Medical Faculty, University of Toronto.

ACUTE phlegmonous inflammations of the stomach are among the rarest of affections of that organ, between eighty and ninety cases only being recorded in medical literature.

Although the chance of diagnosis is in the majority of cases doubtful and the prognosis is exceedingly bad, only one or two possible cases of recovery being recorded, yet it is of interest to describe a case of this disease which came to autopsy in the Toronto General Hospital in May of the present year.

It is probable that the condition is somewhat more common than the literature records would lead us to suppose, since an autopsy is necessary to confirm any suspicions, and, as in our case, in the absence of a post-mortem examination the cases might be thought to be cases of perforative appendicitis.

In addition to this it seems most likely that all the recorded cases are not included in the more extensive articles on the subject, since we find in the article by Leith in 1897, in Allbutt's System of Medicine, 85 cases of the diffuse and circumscribed form, whilst in an article by Schaurwyler in the *Archiv für Verdauungs-Krankheiten* for 1906 only 83 cases are mentioned.

The condition occurs in two forms, a circumscribed and a diffuse form, the latter being about twice as common as the former. As Leith points out, the circumscribed form had been recognized as far back as the seventeenth century by Verandaeus in Tractatus de Morbis Ventriculi, 1620; the diffuse form, on the other hand, was first described by Cruveilhier in 1861.

The history of our case is as follows:—

M—— K——. Admitted to hospital Thursday, May 24th, 1906, under Dr. Gordon. He had been seen by Dr. Lehmann outside, who made a diagnosis of acute gastritis and sent him into the hospital.

Complaint: Pain all over abdomen and chest.

Family History not obtained.

Personal History: Patient is a laborer, age 28. Single, of Austrian nationality (no further information obtainable as patient unable to speak a word of English).

Physical Examination:

12 o'clock. On admission patient did not appear particularly distressed, but pulse was weak (rate 90), which, however, is much improved owing to an immediate administration of strychnine. Patient lies on his

face and appears very nauseated. He is able to turn without difficulty. The abdomen is soft with no special points of tenderness. There are confluent reddish purple blotches all over the body. The temperature is 101°. Examination of the chest revealed no abnormalities.

At 1 o'clock temperature was 101°.

At 2 o'clock a quantity of yellowish fluid was vomited.

Patient's pulse rose to 120 by 2.30.

At 3 o'clock patient again vomited and an hour later the respirations were 30. Temperature 102 (axilla) and pulse 128.

Patient vomited at 5 o'clock and again at 7 o'clock after having taken water, oz. iv.

At 8 o'clock the respirations were 36, pulse 152, and temperature 99. Milk, oz. viii., was given, but patient became very nauseated and suffered great pain in abdomen. The bowels moved and urine was passed. He seemed very restless. The pulse rate has dropped to 130.

At 10 o'clock the patient was in great pain and the pulse rate had risen to 152 and was of a weak and running nature. The abdomen was flat but rigid, especially on the right side, and was tender all over. The blotches previously noted had assumed a much deeper color. The patient was lying on his face. The face was clammy, but pinched; the features sharp, eyes sunken, the lips [illegible] tips of the fingers were cyanosed and patient very restless.

At 11.30 patient was examined by Dr. F. N. G. Starr, who found him in a critical condition, almost pulseless, feet and hands cold and facial expression indicating general collapse as often seen in peritonitis. Case was diagnosed as one of peritonitis and all hope of recovery abandoned.

The pulse became too feeble to be counted and the patient died at 12.40 a.m.

Autopsy Report.

There were no abrasions or sores on the surface of the body.

The thoracic organs, with the exception of some old pleuritic adhesions, showed nothing of importance. Omentum spread out over loops of intestine, which contains very little gas. Peritoneum moist over upper portion, becoming dry and sticky towards the mesentery. The right inguinal region contains purulent fluid in character slightly blood-stained. Pelvis contains quantity of this fluid. In region of the appendix there are a few old scars; the appendix itself is small and bent on itself, very slightly congested, somewhat more so at the tip. The mesenteric glands somewhat enlarged and soft. The wall of the lesser curvature of the stomach, on the anterior surface, is discolored and looks soft.

Stomach. Measuring along greater curvature, 48 c.m. Greatest width, 14½ c.m. The gastric wall, 1.4 c.m. in thickness, soft and boggy; on cutting into the wall, purulent fluid oozes from the sub-mucosa everywhere; the mucosa is of a mottled pink and yellow, darker in color towards the greater curvature. At the fundus, in one spot, there is a break of the mucosa, but this looks as if it might be post-mortem; underneath the serous coat one can notice the lymphatics filled with sero-pus.

Photograph of Stomach: Note the thickened pulpy wall.

Intestines. Small intestine nothing abnormal; large intestine normal; the appendix vermiformis normal; mucosa pale; lumen contains small amount of fæcal matter.

The other organs show nothing of importance.

Bacteriological examinations were made of the peritoneal cavity and gastric wall, heart's blood and spleen.

All the cultures showed a pure growth of staphylococcus pyogenes aureus. The blood infection was so intense that a single drop of blood upon a blood serum slant gave a confluent growth of the staphylococcus. A microscopic examination of smears from the gastric wall and from the peritoneal exudate showed only staphylococci.

Histologically, the thickening of the gastric wall is seen to be chiefly due to the tremendous infiltration of the submucosa with pus; the mucosa also shows some infiltration although not nearly so extensive as the submucosa. The tissue spaces of the muscular coat also show more or less purulent infiltration and in the serous coat the lymphatics everywhere contain pus.

The condition is fairly evenly distributed throughout the stomach, but is more marked about the middle of the greater curvature and becomes less extensive as we approach the cardiac orifice.

The œsophagus is quite free from infiltration. On the other hand the condition extends through the pyloric orifice and sections of the duodenum show the same condition as the stomach but to a less degree, which extends practically as far down as the papilla of Vater.

It will be seen from this short clinical history and anatomical study of the case that it was a very typical example of the diffuse type of acute phlegmonous gastritis.

The clinical features were practicaly the same as those described by Leith and other writers on the subject, perhaps the only point of difference being the presence of the pronounced purpuric rash. The occurrence of a rash is the more interesting as, although it is not mentioned in any of the histories I have seen, it was a prominent feature of a case of Dr. Charles Wagner's which occurred at St. Michael's Hospital about six months earlier. In his case the rash was scarlatiniform.

The subject of the etiology of acute phlegmonous gastritis is a difficult one. In a certain number of the cases recorded the question was definitely settled since the gastric condition was secondary to other conditions such as gastric carcinoma, puerperal infection or pyæmia.

In the majority of cases, however, the gastric condition was primary and no point was discovered where the infecting organisms gained entrance. It seems most probable that the stomach wall itself was in this case the point of entrance into the tissues.

The fact that the condition is more common in men than in women and that a number show a history of alcoholism, has led some writers to give prominence to this factor, but personally I am inclined to think that this is overestimated. Dietetic errors is also given as a possible important factor. Leith points out that in a certain number of cases the disease followed immediately after a meal and in two the patients were in the habit of eating to excess after periods of abstinence.

This seems to me to be much the most important factor and in our case, and possibly in Dr. Wagner's, it seems the most likely explanation.

Both cases occurred in a colony of foreigners who were living under the most unfavorable conditions in regard to food. Dr. Wagner informs me that he made enquiries in regard to the food habits of these people and they seemed to regularly purchase meat which was of the very worst character on account of its cheapness; one member of the colony stated that they never paid more than five cents a pound for meat and as they probably often kept it until almost putrid it is most likely that conditions of gastro-intestinal intoxication would result which would favor, if not actually lead, to infection of the gastro-intestinal tract.

The organism in our case was, as stated above, the staphylococcus aureus. This has been found in other reported cases, but the majority of those which were examined bacteriologically showed the presence of the streptococcus, either pure or mixed with other forms.

PHLEGMONOUS GASTRITIS—REPORT OF A CASE.*

By Charles J. Wagner, M.B., Toronto.
Demonstrator of Pathology, Toronto University.

This rare disease was described as early as 1656 by Bevel. In our own time cases have been recorded by various observers under many different names. Of these the more noteworthy are: Inflammation of the submucosa (Rokitansky), gastritis submucosa (Dittrich, Klebs and others), phlegmon ventriculi, suppurative interstitial gastritis, submucous suppurative phlegmon, and phlegmonous gastritis. The term phlegmonous gastritis is that most in favor with English writers, although perhaps not so descriptive as others.

Etiology.—The disease is due to the development within the gastric wall of pyogenic organisms. Of these the streptococcus pyogenes is the organism most frequently found. The disease occurs in both sexes and at all ages. Males are much more commonly affected than females in the ratio of four to one. Alcoholism predisposes to the disease, one-fourth of the cases occurring in persons addicted to the excessive use of alcohol. This undoubtedly explains to some extent the more frequent occurrence in males.

The disease may be primary or secondary. The primary cases occur in apparently healthy individuals or follow traumatism, ulcer or growth. The secondary cases occur in the course of such acute infectious fevers as pyemia, septicemia, typhoid fever and variola.

Mayo Robson and Moynihan, who have collected histories of 85 cases, state that in all cases of primary and probably in all cases of secondary disease there is a superficial denudation of the mucous membrane which permits of the entrance of the organism.[1] It appears to me that this statement would be difficult to prove or disprove, as we know that the most minute abrasion may suffice for the entrance of bacteria into the tissues, whereas, in these cases, secondary ulceration due to sloughing is very frequently found. Again in the infectious fevers, secondary infections of the blood by pyogenic organisms not infrequently occur, and it would be unreasonable to hold that in cases of phlegmonous gastritis secondary to such diseases hematogenous infection might not occur.

* Paper read before The Pathological Society of Toronto.

Pathological Anatomy.—The condition may occur as a diffuse cellulitis affecting the whole of the stomach submucosa, or it may be localized to form one or more large or small abscesses. The diffuse form is probably that more frequently met with. Cases of this type show enormously thickened stomach walls, particularly towards the pyloric end, with pus diffused throughout the submucosa. When compressed between the fingers, pus wells up from the cut surface.

The mucosa may appear normal, or may be greatly congested, even hemorrhagic. Large ulcers due to sloughing may be present. Occasionally there are many minute perforations through the mucous membrane out of which pus oozes when the stomach is compressed.

The microscope shows necrosis and purulent infiltration of the submucosa, and leucocytic infiltration and edema of the mucosa, muscularis and peritoneum.

In both forms general peritonitis is almost invariably present at autopsy. There may be thrombosis of vessels of the stomach, liver or lungs. Occasionally there is an acute pleuritis.

Clinical Symptoms.—The disease is frequently ushered in by a chill. Pain in the region of the stomach, vomiting of partially digested food or bile stained fluid, accompanied by a moderate rise of temperature and increased frequency of pulse, are frequently the only symptoms for the first day or two, and being very similar to the initial symptoms of such conditions as, so-called, acute indigestion, gastric fever, etc., are not particularly alarming.

The symptoms, however, instead of ameliorating, become worse: the pain in stomach region becomes intense, the vomiting frequent and most distressing. There is great thirst. The temperature may be high and the pulse is rapid and thready. The expression is pinched and anxious, and, altogether, the symptoms are suggestive of sepsis, with localization in the stomach region. Physical examination usually reveals nothing excepting tenderness in the stomach region, if seen early, or general abdominal tenderness and distention, if seen during the period of general peritonitis. In a few of the localized cases a tumor may be felt. Auscultation occasionally reveals a friction sound behind the left costal cartilages or just below them. The bowels are usually constipated.

In the later stages of the disease, symptoms of general peritonitis almost invariably supervene. The vomiting may cease for a short period before death. Death is seldom deferred

more than fourteen days, and frequently occurs on, or about, the fourth.

The symptoms of individual cases may differ somewhat widely from the above. The temperature may remain normal, as in a case reported by Asverus,[2] or it may be subnormal. Although constipation is the rule, there may be diarrhea. The abdominal pain may be absent throughout, or, as in a case reported by Habershon, of a nurse who suffered with malaise and intense aching of back and limbs for fourteen days before abdominal pain and vomiting developed, it may be greatly delayed.[3] In rare instances pus has been vomited, and sometimes in large quantities, as the result of the bursting of a large localized abscess. These localized abscesses occasionally rupture into the peritoneal cavity.

Diagnosis.—The symptom complex is not characteristic. A positive diagnosis may rarely, if ever, be made. Some of the conditions most difficult to differentiate from phlegmonous gastritis are perigastritis following ulcer, purulent cholelithiasis, acute pancreatitis, peritonitis, hepatic abscess and poisoning by caustics.

The previous history may reveal alcoholism, traumatism, ulcer or carcinoma, or, as in Kelynack's case a stricture of the esophagus.[4]

However, in any case where epigastric pain and frequent vomiting are accompanied by grave general symptoms suggestive of sepsis the possibility of phlegmonous gastritis must be considered.

Prognosis.—The prognosis is extremely grave. Only a few cases are known to have recovered, and these were of the localized variety.

Treatment.—Little more can be done than to allay the more distressing symptoms: opium to relieve the pain and saline enemata to relieve the distressing thirst. Hot fomentations applied to the abdomen may be grateful to the patient.

Case.—F. G., a brewer, aged 30. Had previously been addicted to alcohol, otherwise normal.

April 8th, 1905. Felt chilly and unwell.

April 10th. Still very unwell, with nausea, but still able to work.

April 11th. Too ill to work.

April 12th. On this day I saw the patient for the first time, when I found the following symptoms present: Pain and tenderness in the region of the stomach, sharply localized; anorexia; frequent vomiting of bile-stained fluid; furred tongue;

great thirst; the pulse was running at about 100, full and strong; temp., 101 deg. F.

April 13th. No change.

April 14th. Decided change for the worse. Expression drawn and anxious. Pulse smaller and more rapid. Pain most intense and vomiting very distressing.

Over the entire body with the exception of the face were large and small patches of the most intense erythema, sharply defined from the normal intervening skin. This rash closely resembled that of a marked case of scarlatina, differing only in that the skin was not uniformly affected. It was also most intense upon the mucous membrane of the soft palate and pharynx.

Believing that the rash was the expression of an intense sepsis, I had the patient removed to St. Michael's Hospital because of the inadequate nursing at home, and because I thought it possible that surgical measures might later be necessary.

When the patient arrived at the hospital there was some doubt expressed as to the nature of the rash, and for safety the patient was isolated. Shortly after, my attention was directed to a friction sound over the stomach just below the cartileges on the left side, by Dr. Graham Chambers, who had been asked to see the case. This disappeared within an hour, and could not again be heard. The day following admission, the pain became diffuse over the abdomen, tympanites developed, and the diagnosis of general peritonitis could be positively made.

The tongue, which at first had been heavily furred, became clean, red and glazed.

The patient became gradually weaker, and died from collapse on the tenth day of his illness. Consciousness remained until the end.

Autopsy.—The erythema, so intense before death, was still plainly visible. Over the rash areas the epidermis was peeling off in large patches.

In the thorax both pleural cavities were obliterated by fibrous adhesions. A small calcareous fibrous scar was found immediately beneath the pleura at the apex of the right lung.

The lungs were both congested. On separating the base of the left lung from the diaphragm, pus was found in the interstices of the adhesions.

The lung tissue at the base was intensely congested, but not consolidated.

In the abdominal cavity we found an acute fibrino-purulent

peritonitis, most intense upon the anterior surface of the stomach. No perforation could be found.

The stomach appeared large and felt very heavy, as though there were considerable substance within its cavity. When opened, it was found entirely empty, the unusual weight being due to its immensely thickened walls, which were in places one-half of an inch in thickness. The cut surface was very pale, and when squeezed creamy pus welled up from the submucosa. The interior of the organ presented a mottled appearance, the mottling being due to patches of intense congestion of the mucosa. There were several large superficial ulcers, which I believe were the result of sloughing.

Under the microscope the stomach wall presented the following appearances:

The mucosa was greatly congested and infiltrated with leucocytes.

The submucosa was necrotic, the few cells not entirely disintegrated could be recognized as a polymorphonuclear leucocytes. The muscularis and subperitoneal tissues were infiltrated with leucocytes and serous fluid.

Stained for bacteria, streptococci and bacilli were found.

Cultures from the stomach wall were examined by Dr. Brefney O'Reilly, who isolated two organisms, a proteus and the colon bacillus, the streptococcus so evident in the stained section having evidently died out in the culture.

At a meeting of the American Medical Congress, in May, 1900, Kennicut, of New York, showed a specimen from a very similar case. A streptococcus and a bacillus, which he believed my case, a friction sound had been heard over the stomach wall. This case was discussed by Welch and others. As in my case, a friction sound had been heard over the stomach. This friction sound is due to the roughening of the peritoneal surface of the stomach upon the development of peritonitis. Its early disappearance in my case I ascribe to the exudation of pus upon the peritoneal surface acting as a lubricant.

The rash which was such a prominent feature in my case would appear to be unique in connection with this disease, and beyond the suggestion of sepsis can have no diagnostic significance.

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