Phlegmonous Gastritis *

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THE HIGH MORTALITY RATE and the extreme difficulty in recognizing phlegmonous gastritis prior to operation makes a periodic review of this subject mandatory.[18] The excellent results of gastrectomy in two recent patients will be reported. Since an analysis of current methods of therapy in a large group of patients is not available in the literature, all cases reported in the last decade will be reviewed.

INCIDENCE AND ETIOLOGY

This uncommon bacterial infection of the gastric wall was first described by Cruveilhier in 1820. By 1947 the total number of cases reported had reached 335.[11] The present series of 25 cases brings the total to 360. Phlegmonous gastritis is more common in males than females by a ratio of three to one. Eighty per cent of the cases have occurred in patients between 30 and 60 years of age. The hemolytic streptococcus is the offending organism in the majority of instances and pneumococci, staphylococci, Proteus vulgaris, Escherichi coli, Clostridium welchii account for the remainder. Alcoholism, hard labor and gastric hypoacidity are well known predisposing conditions. It may occur as a complication of chronic ulcer, gastritis, gastric carcinoma or gastric surgery. It has also been reported following the acute exanthemeta and in association with furunculosis.[5, 7, 10, 11, 23, 24]

PATHOLOGY

The pathologic appearance is that of a diffuse or less commonly localized inflammation with injection, thickening and induration of the gastric wall. When diffuse, the lesion is sharply confined to the stomach, rarely going beyond the cardia or pylorus. When localized, it may present as a mass in the gastric wall whose inflammatory nature is not readily apparent unless needle aspiration yields purulent material. The stomach may be coated with fibrin and adherent to other organs. Perforation of the gastric wall may occur and there may be an associated pleural effusion or pericarditis. A characteristic feature is the thickening of the gastric wall on cut section, most marked in the submucosa. The mucosa is flattened with loss of the normal rugae and less commonly superficially ulcerated. In emphysematous gastritis, a rare form of phlegmonous gastritis, the gastric wall is infiltrated with gas produced by the invading organism. Although scarring and deformity of the stomach has been reported, recovery is usually followed by a normal appearing gastrointestinal x-ray.[28] Microscopic examination reveals extensive infiltration of the submucosa with polymorphonuclear leucocytes with scattered areas of necrosis, thrombosis and hemorrhage. The muscle layers may show infiltration with inflammatory cells and granulation tissue.

CLINICAL COURSE

Phlegmonous gastritis may follow a rapidly fulminating course with sudden onset, marked toxemia, early peripheral circulatory collapse and death within a few hours. The more usual picture, however, is that of an acute upper abdominal inflammatory process with high fever, abdominal pain, nausea and vomiting. Rarely, gross pus is found in the vomitus. The pain is usually

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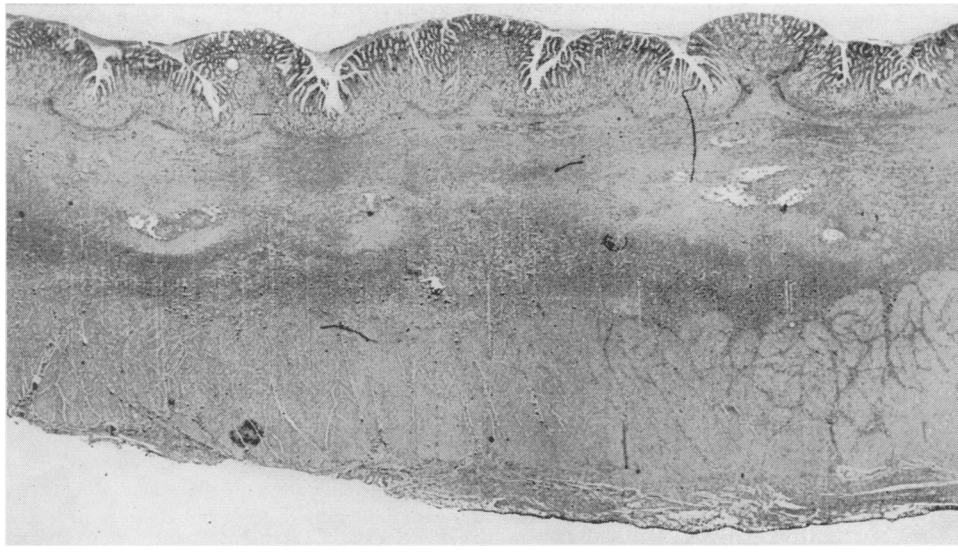

FIG. 1. Case 1. Section of gastric wall demonstrating the intact mucosa with marked thickening of the submucosa due to edema and infiltration with inflammatory cells. The muscularis is less extensively infiltrated and the serosa is congested and coated with fibrin.

most marked in the epigastrium and is nonradiating. Tenderness and muscle guarding may be localized to the epigastrium or the physical findings of generalized peritonitis may be present. There is usually a marked leucocytosis of from 15,000 to 30,000 white blood cells. Gastric analysis commonly reveals low or absent acid, and positive cultures of the offending organism can frequently be obtained by gastric aspiration. A gastrointestinal x-ray performed during the illness reveals the absence of mucosal folds and marked atony. In rare instances gas can be seen in the gastric wall on a plain roentgenogram of the abdomen. Occasionally, the disease follows a more chronic course in which, following localization of a circumscribed abscess, the patient may be afebrile, complaining of only mild epigastric pain and anorexia. This picture can closely mimic an obstructing ulcer or gastric carcinoma, both before and during operation. In evaluating therapeutic results, this type of disease must be clearly separated from the acutely ill, toxic, fulminating case.

CASE REPORTS

Case 1. M. R., a 49-year-old Puerto Rican woman, was admitted to the First Surgical Service of Bellevue Hospital in 1954 with the chief complaint of severe abdominal pain of 2½ days' duration. The patient had a history of mild hypertension and diabetes mellitus. Two and one-half days prior to admission the patient noted the sudden onset of severe steady upper abdominal pain radiating to the precordium and the left shoulder. She had vomited bile stained material repeatedly following the onset of her illness and on the day prior to admission she had developed shaking chills and fever. She denied any previous digestive difficulties.

Physical examination revealed an acutely ill, dehydrated, obese woman with a temperature of 39.2° C. (102.6° F.), pulse 120, respirations 30 a minute and blood pressure 160/90. The tongue was dry and coated. Examination of the chest revealed only slight enlargement of the heart to the left on percussion. Examination of the abdomen revealed marked upper abdominal tenderness and guarding, maximal in the right upper quadrant. There was upper abdominal rebound tenderness, and what seemed to be a vague palpable mass just below the right costal margin on a line with the outer border of the rectus sheath. Pelvic and rectal examination were negative.

Laboratory studies revealed a hemoglobin of 14.5 Gm. and a white cell count of 22,000. Urinalysis revealed four plus glycosuria and a three plus acetone. X-ray of the chest disclosed slight left

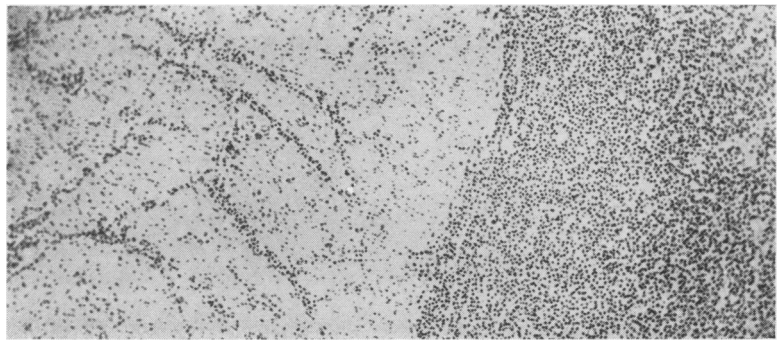

FIG. 2. Case 1. Section through junction of muscularis and submucosa demonstrating the more intense infiltration of the submucosal layer with polymorphonuclear leucocytes.

ventricular enlargement. A plain roentgenogram of the abdomen was negative with no evidence of calculi or free air under the diaphragm. An electrocardiogram was normal.

Insulin, intravenous fluids, penicillin, streptomycin and gastric suction were started. Ten hours after admission the patient was explored through a right subcostal incision with the preoperative diagnosis of acute cholecystitis. Upon opening the peritoneal cavity a few ml. of purulent material escaped. The gall bladder was found to be normal. The mass previously noted was found to consist of a portion of the stomach, the distal half of which was grossly thickened, indurated and hyperemic. The gastrocolic and gastro hepatic ligaments on either side of the pylorus and pyloric antrum were thickened and edematous. The lesser sac was partially obliterated by numerous adhesions. After extending the incision, approximately 75 per cent of the stomach was resected and a posterior Polya gastrojejunostomy was performed. The proximal line of resection was above the palpable area of induration of the gastric wall. At the site of anastomosis, however, there was slight noticeable thickening of the submucosal layer.

Gross examination of the specimen revealed a shallow 3 mm. ulcer on the lesser curvature 3 cm. from the pyloric ring. There was loss of the usual mucosal folds and marked thickening of the gastric wall to 5 cm. in some areas. Microscopic examination revealed massive infiltration of the entire submucosa with polymorphonuclear leucocytes with multiple sites of abscess formation. The inflammatory pattern was also present, though less marked, in the muscle and serosal layers. The minute ulceration did not extend past the mucosa.

Postoperatively, the patient was continued on penicillin and streptomycin. She was afebrile on the fifth postoperative day and she was discharged on the nineteenth postoperative day after a benign course.

Comment: This patient demonstrates the more common, acute form of phlegmonous gastritis and the difficulty in distinguishing it from other acute inflammatory conditions of the upper abdomen. The patient recovered although the suture line of the gastrojejunostomy involved inflamed stomach. This was probably due to the early removal of the bulk of the infected stomach and excellent supportive therapy.

Case 2. I. B., a 54-year-old white man, was admitted to the First Medical Service of Bellevue Hospital on February 2, 1950 with the chief complaint of hematemesis 4 hours prior to admission. The patient had had previous admissions, during the preceding 5 year period, for various complaints including known chronic alcoholism of many years' duration, and in the past 3 years for recurrent episodes of epigastric fullness, burning and vomiting. An exploratory laparotomy in 1945 had revealed an enlarged liver but no other pathologic lesion. Several gastro-intestinal X-rays taken after the onset of the episodes of epigastric pain in 1947 had been reported as negative.

The patient was hospitalized for a brief period on the First Medical Service in January 1950 for what was diagnosed as gastroenteritis secondary to alcoholism. Following this he had repeated episodes of post prandial epigastric pain associated with occasional nausea and vomiting. He had noted once or twice weekly for a period of several weeks the passage of tarry stools. On the day of admission the patient suddenly developed nausea and vomited an estimated 2 liters of bright red bloody fluid.

Physical examination on admission revealed a pale, anxious, well-developed, moderately obese white man with a temperature of 36.7° C. (98° F.), a pulse of 120, respirations of 20, and a blood pressure of 160/90. Examination of the heart and lungs was negative. The liver edge was palpable one and one half finger breadths below the costal

margin. The tip of the spleen was palpable just above the left costal margin.

The hemoglobin was 8 Gm. per cent and the leukocyte count 8,800 with 87 per cent polys, 11 per cent lymphocytes, 1 per cent monocytes and 1 per cent eosinophiles. Urinalysis was negative. A stool guaiac test was positive.

A complete medical workup was started and blood replacement begun. A chest X-ray study, esophagogram, an upper gastro-intestinal and small bowel X-ray, a barium enema and a cholecystogram were reported negative. Studies of hepatic functions revealed an icteric index of four, total proteins of 5.8 Gm. with an AG ratio of 3.8/2.0, a two plus cephalic flocculation, no bromsulfalein retention, and a prothrombin time of 52 per cent. An esophagoscopy was negative.

The patient had two further episodes of hematemesis on March 6, 1950 and March 22, 1950. On April 27, 1950 an exploratory laparotomy was performed. An area of induration in the lesser curvature of the pars media of the stomach and in the adjacent gastrohepatic omentum was found. On gastrotomy, adjacent to this area of induration in the gastric mucosa there was an area about 1 cm. in diameter which had the appearance of a healing ulcer. The patient also had an enlarged nodular liver and a spleen 3 times normal size. Portal pressure measured 18 cm. of water. Because of the findings in the gastric wall, a subtotal gastrectomy and an antecolic Hofmeister type gastrojejunostomy were performed. Postoperatively, he received penicillin and streptomycin.

Gross examination of the resected specimen revealed diffuse thickening of the gastric wall along the lesser curvature. The wall in the fixed specimen was 2 cm. thick and appeared to be mainly dense fibrous tissue. The mucosa was intact throughout. Microscopic examination of sections of the gastric wall along the lesser curvature revealed an intact mucosa under which there was an extensive area of edematous organizing fibrous tissue containing a diffuse infiltration of polymorphonuclear leucocytes, eosinophiles and lmyphocytes which markedly thickened the submucosa. There was hypertrophy of the muscularis and edema of the serosa. The serosa was covered by an extensive fibropurulent exudate. The pathologic diagnosis was phlegmonous gastritis with fibrosis of the gastric wall.

The patient's postoperative course was complicated by a right subhepatic abscess which required incision and drainage on May 12, 1950. The patient was discharged on June 12, 1950. When last seen in October of 1953, he had no gastro-intestinal complaints.

Comment: This patient demonstrates the less frequent chronic form of phlegmonous gastritis related perhaps to alcoholic gastritis and gastric ulcer. Survival following gastrectomy is not unusual in this stable form of the disease.

DISCUSSION

The success of gastrectomy in these two instances prompted a review of the literature to determine current thought on the therapy of phlegmonous gastritis. Twenty-five hitherto uncollected cases reported in the last decade are tabulated in Table I. Only cases reported after 1945 were selected to insure that antibiotics and modern concepts of fluid and electrolyte therapy were available. It is important in the evaluation of these data to differentiate between the acute and chronic form of the disease. Cases were classified as chronic if they met the following criteria: (1) Their history revealed a prolonged illness of more than a few weeks without rapid or sudden progression of symptoms; (2) they had minimal or no fever, tachycardia or leukocytosis. All other cases were classified as acute.

There were 12 deaths in this series, a mortality of 48 per cent. The mortality was much higher in the acute cases with ten out of 17 dying of their disease. In eight chronic cases there were only two deaths. This represents significant improvement over the 92 per cent mortality rate reported by Sundberg in 1919, and the 84 per cent mortality rate reported by Eliason in 1938.[7, 24, 25]

The multiplicity of treatments recommended for phlegmonous gastritis is evidence that none are completely satisfactory. Gerster in 1927 found eight successful gastric resections in the literature and although the two patients in his own series who were operated upon died, he recommended resection as the procedure of choice.[10] Eliason and Wright suggested gastrotomy with transmucosal incision and drainage of the abscess. Two of their five survivors had such a procedure; one had a subtotal gastrectomy and two recovered spontaneously after laparotomy. Cutler in 1940[5] stated that any operative procedure undertaken

TABLE I. *Summary of Data of 25 Cases of Phlegmonous Gastritis 1946–1955*

Author	Year	Age	Sex	Type	Treatment	Result	Comment
Joyeux	1948	50	F	acute	subtotal gastrectomy	died	The patient expired on the fifteenth day of renal failure after an otherwise benign course.
Birch	1946	64	F	acute	subtotal gastrectomy	alive	The operation was performed 24 hours after the onset.
Authors	1955	49	F	acute	subtotal gastrectomy	alive	The operation was performed soon after the onset.
Forster	1947	36	F	acute	gastrotomy	alive	The patient developed a gastro-cutaneous fistula.
Guzzetta	1947	18	F	acute	gastrotomy	alive	The patient also received penicillin.
Bandmann	1950	58	M	acute	exploratory laparotomy penicillin	died	The diffuse type was found at the operation. The patient died three months following operation of a liver abscess.
Bendixen	1947	37	F	acute	exploratory laparotomy penicillin	alive	
Kohler	1949	43	M	acute	exploratory laparotomy penicillin	died	The patient was explored late in the course. Penicillin was started the tenth post-operative day. The patient died in three weeks.
Miller	1952	42	M	acute	exploratory laparotomy penicillin	died	Death was due to a suspected perforation. No autopsy was performed.
Henry	1952	1 mo.	M	acute	penicillin	died	The patient died 9 hours after the onset of emphysematous gastritis.
Weitz	1949	72	M	acute	supportive	died	The patient died 3 days after admission. Antibiotics were not mentioned.
Siudmak	1954	53	M	acute	penicillin, streptomycin	died	The patient died 40 hours after admission of peritonitis without perforation.
Welch	1947	15	F	acute	penicillin, sulfadiazine	alive	The patient had emphysematous gastritis diagnosed radiologically.
Stenstrom	1951	64	F	acute	penicillin	died	The patient died in 43 hours of shock.
Laser	1953	77	M	acute	terramycin, penicillin	alive	The disease was diagnosed by x-ray.
Weens	1946	37	M	acute	penicillin, sulfadiazine	died	The patient had emphysematous gastritis following HCL ingestion. He died in three months.
Rutherford	1953	39	F	acute	medical	died	The patient died 17 hours after fulminating course. She was getting cortisone for lupus.
Bandmann	1950	55	M	chronic	subtotal gastrectomy	alive	Antibiotics were not mentioned.
Dias	1950	35	M	chronic	subtotal gastrectomy	alive	Antibiotics were not mentioned. The lesion was localized about the antral ulcer.
Lorenzo	1947	51	M	chronic	subtotal gastrectomy	alive	He also received penicillin. The patient had a localized lesion.
Authors	1955	54	M	chronic	subtotal gastrectomy	alive	The patient was an alcoholic with a gastric ulcer.
Bonorino	1948	53	M	chronic	subtotal gastrectomy	died	The patient had a localized lesion, but was a poor risk because of chronic illness.
Wessel	1949	61	M	chronic	exploratory laparotomy	died	A cholecystojejunostomy was performed because of an error in diagnosis.
Mouter	1949	56	F	chronic	penicillin	alive	The disease was diagnosed gastroscopically.
Mouter	1949	49	M	chronic	penicillin	alive	The disease was diagnosed gastroscopically

for acute phlegmonous gastritis is hazardous and that gastric resection is not indicated. Miller and Nushan in 1952 stated that if the disease is encountered at laparotomy, nothing further should be done. The patient they report died of perforation after exploration without definitive attack.[19]

Analysis of Table 1 and the report of Case 2 reveal that subtotal gastrectomy is a relatively safe and effective means of treating the chronic case. In the two chronic cases in patients surviving after penicillin alone, the diagnosis of phlegmonous gastritis was not established beyond doubt. Gastrectomy is often mandatory in this group because of the difficulty of excluding gastric carcinoma or chronic gastric ulcer. The usual localization of the lesion to the pyloric region makes resection feasible.

A review of the acute cases in Table 1 reveals that there was one death among five patients treated by gastrectomy or gastrotomy for drainage of submucosal abscesses. There were nine deaths among 12 patients treated without definitive surgery. The presence of survivors in both groups implies that the problem is more than one of medical versus surgical management. The common factors in all of the survivors were the early recognition of acute phlegmonous gastritis by laparotomy or other means, and the prompt use of intensive supportive measures. The removal of infected tissue by gastrectomy or the drainage of purulent

collections in the stomach wall are important ancillary measures. While not essential to survival, experience in the last decade indicates that these procedures may significantly reduce the mortality.

SUMMARY

1. Two patients surviving gastrectomy for phlegmonous gastritis are presented.

2. All cases of phlegmonous gastritis reported in the last decade are reviewed.

3. The mortality during the past ten years has dropped to half that of previous reports.

4. The importance of separating the acute fulminating case from the chronic case in analyzing mortality statistics is stressed.

5. Subtotal gastrectomy appears to be safe and feasible in the chronic form.

6. Early recognition and prompt intensive supportive therapy are the common factors in survival of the acute form.

7. Subtotal gastrectomy or drainage procedures, though not essential to survival, may significantly reduce the mortality in the latter group.

BIBLIOGRAPHY

1. Bandmann, F.: Phlegmonous Gastritis. Zentralblatt für Chirurgie, **75**: 1606, 1950.
2. Bendixen, K.: Phlegmonous Gastritis with Recovery after Penicillin. Nordisk Medicin, **33**: 780–781, 1947.
3. Birch, C. A.: Phlegmonous Gastritis. Clinical Journal, **75**: 1748, 1946.
4. Bonorino, C.: Purulent Gastritis. Revista Clínica España, **28**: 287, 1948.
5. Cutler, E. C. and J. H. Harrison: Phlegmonous Gastritis. S. G. O., **70**: 234, 1940.
6. Dias, R.: Phlegmon of Gastric Antrum. Boletín Clínico Hospital Civas de Lisbon, **14**: 363, 1950.
7. Eliason, E. L. and V. W. Murray-Wright: Acute phlegmonous Gastritis. Surg. Cl. N. A., **18**: 1553, 1938.
8. Fink, H.: Phlegmonous Gastritis. Am. J. Surg., **35**: 599, 1937.
9. Forster, E.: Acute Phlegmon of Gastric Wall. Schweiz. med. Wchnschr., **77**: 541–542, 1947.
10. Gerster, J. C. A.: Phlegmonous Gastritis. Ann. Surg., **85**: 668, 1927.
11. Guzzetta, Jr., P. C. and H. W. Southwick: Acute Phlegmonous Gastritis. Surg., **22**: 453, 1947.
12. Henry, G. W.: Emphysematous Gastritis, **68**: 15, 1952.
13. Joyeux, R.: Staphylococcic Phlegmonous Gastritis. Académie de Chirugie, **74**: 412, 1948.
14. Kohler, H. and G. Kitzerow: Clinical Picture of Acute Diffuse Phlegmonous Gastritis. Ztschr. f. Arztl. Fortbld., **43**: 466, 1949.
15. Laser, S. Blochlinger: Gastritis Phlegmonosa. Gastroenterologia, Basel, **79**: 94, 1953.
16. Lorenzo, Lozadu and Piouano: Gastritis Phlegmonosa. Bol. Soc. de Cirugía de Uruguay, **18**: 507, 1947.
17. Marshall, C. J.: Phlegmonous Gastritis. British Journal of Surgery, **22**: 629, 1935.
18. Miller, B. and H. Nushan: Phlegmonous Gastritis. American Journal Roentgenol., **67**: 781, 1952.
19. Mouter, F., A. Corent and R. Wolfsohn: Penicillin Therapy of Phlegmonous Gastritis. Arch. de Maladies l'app. Digestif, **38**: 779, 1949.
20. Rutherford, P. S. and J. A. Berkeley: Phlegmonous Gastritis. Can. Med. Ass. J., **69**: 1, 68, 1953.
21. Stenstrom, J. D. and G. H. Hoehn: Fulminating Diffuse Phlegmonous Gastritis. Can. Med. Ass. J., **64**: 317, 1951.
22. Siudmak, J. J. and I. Kempner: Acute Phlegmonous Gastritis. N. Y. State J. of Med., **54**: 1189, 1954.
23. Sundberg, H. Heber: Gastritis Phlegmosa. Nord. Med. Arkiv. Stockholm, **51**: 303, 1919.
24. Sundberg, H.: Phlegmonous Gastritis. J. A. M. A., **73**: 802, 1919.
25. Weens, H. S.: Emphysematus Gastritis. Am. J. Roent., **55**: 588, 1946.
26. Weitz, G.: Phlegmonous Gastritis in Superficial Cancer of Gastric Mucosa. Der Chirurg., **20**: 490, 1949.
27. Welch, C. E. and C. M. Jones: Emphysematous Gastritis. N. E. Journal of Medicine, **237**: 983, 1947.
28. Wessel, M. S., E. L. Wiebur and R. A. Burger: Acute Phlegmonous Gastritis. Gastroenterology, **12**: 884, 1949.