Canad. Med. Ass. J.
Jan. 5, 1963, vol. 88

# Gastritis of the Herniated Stomach in Patients with Esophageal Hiatus Hernia

G. G. FORSTNER, M.D. and A. BOGOCH, M.D., *Vancouver, B.C.*

**ABSTRACT**

**Seven illustrative cases of gastritis of the herniated stomach in patients with sliding esophageal hiatus hernia are reported. Five had superficial gastritis (three mild, one moderate and one severe); two had atrophic gastritis. Gastritis was present in two patients whose mucosa appeared normal at esophagoscopy. Interstitial hemorrhage into the lamina propria was present in four of the seven biopsy specimens. The possibility that interstitial hemorrhage may be related to the development of gastric erosions is considered. The pathogenesis of this form of gastritis is discussed.**

GASTRITIS of the herniated stomach in patients with esophageal hiatus hernia has been recognized for many years. Not until recently, however, has the incidence of this condition been appreciated. In 1958, Dagradi, Killeen and Schindler[1] reported that 49 of 72 patients (68%) with sliding hiatus hernia had gross evidence of gastritis in the herniated stomach at gastroscopy, and that in 33 of the 72 patients (45.8%) gastritis was confined to the herniated sac. In a study of 214 cases of hiatus hernia, Palmer[2] found erosive gastritis, "due to or directly associated with hiatus hernia", in 25 cases (11%).

Several investigators[3-5] have shown that the correlation between the visual and histological appearance of diffuse gastritis is frequently poor. Doig and Wood[6] state that "the definitive diagnosis of gastritis can be made only by biopsy of the mucosa". It is, of course, difficult to quarrel with the visual diagnosis of gastritis when there is marked hyperemia with exudation and erosion. Hyperemia, however, may be due to simple venous engorgement as well as to gastritis; and without other evidence of inflammation, the differentiation can only be made by biopsy. It is well recognized that histological evidence of gastritis may be found when the mucosa appears normal.

The features of seven illustrative cases, in which histological evidence of gastritis was found in biopsies of the herniated sac taken during esophagoscopy, are presented in this report.

## CLINICAL DATA

All seven patients had radiological evidence of a sliding hiatus hernia. In all but one the cardio-esophageal junction was considered to be 5 to 8 cm. above the hiatus; in Case 4, it was 2 cm. above the hiatus. Reflux of barium into the distal esophagus was demonstrable in all, and none of the hernias reduced during radiological examination. There was no evidence of associated gastric or duodenal ulcer disease.

All were males ranging in age from 44 to 74 years. Some of the pertinent clinical data are summarized in Table I. The chief complaint was dysphagia in four cases (Cases 1, 2, 5 and 6) and severe retrosternal pain in three (Cases 3, 4 and 7). The duration of symptoms ranged from two months to 20 years. Three patients (Cases 2, 3 and 5) had evidence of previous mild gastrointestinal bleeding; one had had hematemesis and melena, and two an iron deficiency anemia. In one patient (Case 6) who had a past history of excessive alcohol consumption, biopsy was performed three weeks after admission to hospital. During this interval there were no symptoms to suggest gastritis. All were well nourished at the time of biopsy except one (Case 5) who had carcinoma of the lung, rheumatoid arthritis, generalized amyloidosis and nephrotic syndrome. Three others also had other unrelated conditions for which they were taking medication (Table I).

From the Department of Medicine, Shaughnessy Hospital, and the University of British Columbia, Vancouver, B.C.

### *Endoscopy and Biopsy: Methods and Findings*

Esophagoscopy was performed with an Eder-Hufford esophagoscope under local anesthesia. Biopsies of the terminal esophagus and of the hernial sac were taken by forceps shortly after entering the stomach, to avoid excessive instrumental trauma to the mucosa. Esophagoscopy and biopsy were performed prior to bouginage in all cases. The specimens were fixed immediately in formol-saline solution and embedded in paraffin. Sections were stained with hematoxylin and eosin. The remainder of the stomach was not studied endoscopically or by biopsy.

The histological findings were classified as normal, superficial gastritis, and atrophic gastritis. No examples of gastric atrophy were seen. In superficial gastritis the lamina propria is infiltrated with lymphocytes, plasma cells and polymorphonuclear leukocytes. The surface epithelial cells may be irregular and flattened, but normal pit and glandular structure is preserved. Superficial gastritis was designated as mild, moderate or severe, depending upon the severity of the inflammatory change. In atrophic gastritis, in addition to an inflammatory infiltrate consisting of plasma cells,

TABLE I.—CLINICAL FEATURES

| *Case No.* | *I* | *II* | *III* | *IV* | *V* | *VI* | *VII* |
|---|---|---|---|---|---|---|---|
| Age (years) | 61 | 61 | 44 | 68 | 74 | 71 | 55 |
| Duration of symptoms | 20 years | 1½ years | 17 years | 2 months | 5 years | 10 years | 13 years |
| Interval since initial x-ray evidence of hiatus hernia | 5 years | 10 days | 3 years | 1 month | 5 years | 5 years | 13 years |
| Main symptoms | Dysphagia. Epigastric pain after or unrelated to eating. Retrosternal pain on eating and bending | Dysphagia. Retrosternal pain on eating. Nausea, vomiting | Retrosternal pain after meals and on bending. Epigastric pain after or unrelated to eating. Acid regurgitation. Nausea, vomiting | Postprandial retrosternal pain. Nausea, vomiting | Dysphagia. Retrosternal pain on eating | Dysphagia. Retrosternal pain on eating. Acid regurgitation. Nausea, vomiting | Retrosternal pain on bending and unrelated to meals. Acid regurgitation. Nausea, vomiting |
| Associated illness | — | — | Generalized arteriosclerosis | Chronic bronchitis. Rheumatoid arthritis | Rheumatoid arthritis. Carcinoma of lung. Generalized amyloidosis. Nephrotic syndrome | Chronic bronchitis | — |
| Medication | — | — | Codeine, triiodothyronine | Amesec ®, aspirin | Digitalis, meralluride, aminophylline | Amesec® | — |

lymphocytes and occasionally polymorphonuclear cells, the glands are atrophic, are reduced in number, and show a paucity of acid and pepsin secreting cells. The surface epithelium may be flattened or partly composed of goblet cells. The pits are elongated, dilated and decreased in number. Interstitial hemorrhage may be a feature of both superficial and atrophic gastritis.

All seven patients had evidence of acute or chronic esophagitis, and in four the esophageal lumen was narrowed. As noted in Table II, five patients had superficial gastritis. It was mild in three, moderate in one (Fig. 1) and severe in one (Fig. 2). In three of these patients the gastric mucosa was hyperemic, while in two the mucosa appeared normal. Two patients had atrophic gastritis. The gastric mucosa appeared hyperemic at esophagoscopy in one, and was not seen in the other, in which a blind biopsy was taken (Fig. 3).

Interstitial hemorrhage in the lamina propria, as shown in Fig. 2, was a feature of Cases 1, 4, 5 and 7.

## DISCUSSION

In the four cases (Cases 1, 2, 3 and 4) in which the mucosa was hyperemic, the histological changes were those of mild, moderate and severe super-

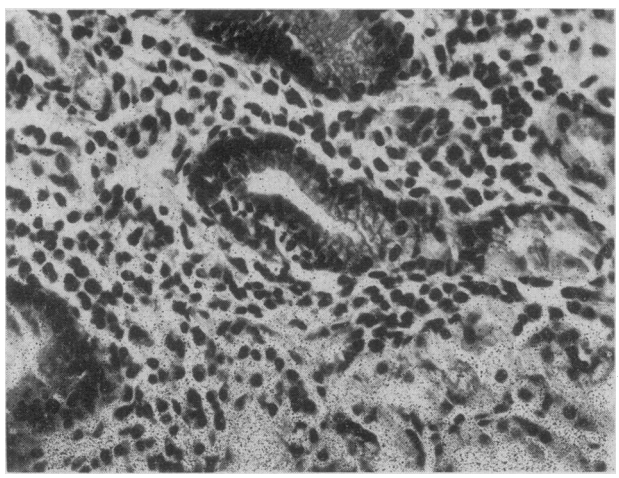

Fig. 1.—Case 3. Moderate superficial gastritis. There is leukocytic infiltration of the lamina propria, consisting chiefly of lymphocytes and plasma cells with a scattering of polymorphonuclear leukocytes. Several polymorphonuclear leukocytes are present in the wall of a gastric pit. Normal glands can be seen at the lower right. (H & E, × 350.)

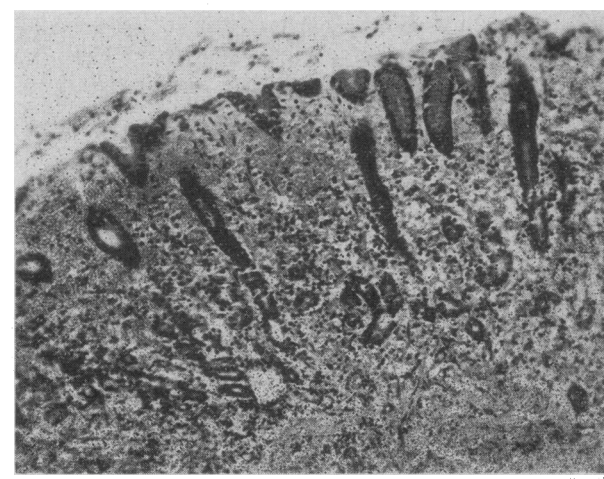

Fig. 2.—Case 1. Severe superficial gastritis. The cellular infiltrate is predominantly polymorphonuclear in type, and extends to the submucosa. There is a marked interstitial extravasation of erythrocytes into the lamina propria. Flattening and loss of surface epithelial cells is evident. (H & E, × 30.)

Canad. Med. Ass. J.
Jan. 5, 1963, vol. 88

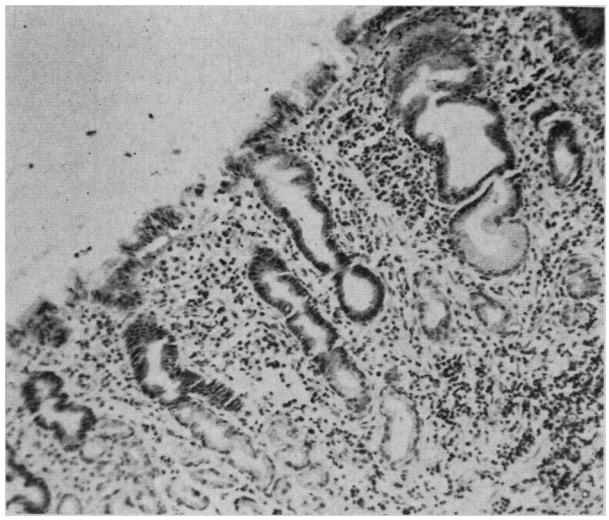

Fig. 3.—Case 6. Atrophic gastritis. The gastric pits are dilated and elongated, and glands are decreased in number. The cellular infiltrate consists of a moderate number of polymorphonuclear leukocytes, lymphocytes and plasma cells. (H & E, × 165.)

ficial gastritis and atrophic gastritis, respectively. Mild superficial gastritis was present in two patients (Cases 5 and 7) in whom the mucosa looked normal at esophagoscopy. These findings are consistent with the lack of correlation between gross and microscopic findings reported in patients with diffuse gastritis.[3-5]

It is impossible to differentiate between acute and chronic superficial gastritis histologically except by repeated biopsies. Atrophic gastritis, on the other hand, has been shown to result from prolonged superficial inflammation[1, 18] and represents a chronic change. The presence of atrophic gastritis was not suspected from the appearance of the mucosa in Case 4.

It is possible that the severe debilitating illness in Case 5 and alcoholism in Case 6 were responsible for the gastritis found in the hernial sac, or contributed to it. According to Palmer,[7] diffuse gastritis least frequently involves the gastric mucosa of the cardia and proximal fundus. This suggests the possibility that gastritis in the hernial sac may in some cases be a causally distinct entity. This concept is supported by the descriptions of gastritis confined entirely to the herniated stomach.[1, 10, 11]

Several theories have been advanced to explain the presence of gastritis in the herniated gastric pouch. According to Sahler,[8] gastritis may result from congestion of the blood vessels in the herniated portion of the stomach. Palmer[9] has accepted the postulation that erosions and ulcers in this area are of traumatic origin. Transient or prolonged incarceration of the hernia may cause mucosal congestion, and possibly increase the susceptibility of the mucosa to peptic digestion. The retention of food particles may provide another source of mucosal irritation. Aylwin[12] has shown that there is little flow of gastric juice from the stomach below the hiatus into the unreduced

TABLE II.—ESOPHAGOSCOPIC AND HISTOLOGIC FINDINGS

| | *Distal esophagus* | *Cardio-esophageal region* (*cm. from incisor teeth*) | *Hernial sac* | |
|---|---|---|---|---|
| | *Gross appearance* | | *Gross appearance* | *Histological findings* |
| I | Moderate narrowing, thick, granular mucosa | 35 | Hyperemic | Superficial gastritis, severe |
| II | Stricture. Grey mucosa for 2 cm. at 34 cm. | 34 | Hyperemic, edematous | Superficial gastritis, mild |
| III | Grey, slightly granular mucosa | 37 - 38 | Slight hyperemia | Superficial gastritis, moderate |
| IV | Slightly hyperemic and friable mucosa | 38 | Hyperemic | Atrophic gastritis |
| V | Moderate narrowing. Grey mucosa with ulcer at 35 cm. | 35 | Normal | Superficial gastritis, mild |
| VI | Moderate narrowing. Friable grey mucosa for 1 cm. at 37 cm. | 37 | Friable, grey mucosa for 1 cm. Not visualized below | Atrophic gastritis |
| VII | Hyperemic mucosa | 34 | Normal | Superficial gastritis, mild |

hernial sac during sleep, and concluded that gastric juice collected from the terminal esophagus at this time is chiefly produced by the mucosa of the herniated stomach. Hernial sacs which produced the largest volume of gastric juice with the lowest pH and highest peptic activity were usually associated with severe esophagitis. Marchand[11] has suggested that mucosal erosions in the sacs of rolling hiatus hernias are due to the effects of trapped, highly active gastric juices. It is possible that a similar mechanism is in part responsible for the gastritis in the unreduced hernial sacs of patients with sliding hiatus hernias.

Only two patients complained of pain which might have suggested the diagnosis of gastritis. This is roughly in accord with the experience of Joske, Finckh and Wood,[13] who found that about three-quarters of patients with diffuse superficial and atrophic gastritis had no symptoms related to this disease.

Approximately 20% of patients with hiatus hernia have overt or occult bleeding.[14-16] Bleeding in such instances is usually due to peptic ulcer disease, esophagitis or gastric erosions. Palmer's studies[2] suggest that erosive gastritis may cause bleeding in hiatus hernia almost as commonly as does esophagitis. It is of interest that hemorrhage was present in the lamina propria of four of the seven biopsy specimens described in this report. It is possible that this change was produced in part by the trauma of esophagoscopy and biopsy, but it has also been found in cases of diffuse gastritis when tissue is obtained by suction biopsy.[17] The possible pathogenetic relationship of interstitial hemorrhage in the mucosa to gastric erosions deserves consideration.

## SUMMARY

Seven cases of sliding esophageal hiatus hernia, in which gastritis of the herniated stomach was demonstrated by biopsy, have been described. Five patients had superficial gastritis. This was mild in three instances, moderate in one, and severe in one other.

Two patients had atrophic gastritis. Interstitial hemorrhage into the lamina propria was present in four of the seven biopsy specimens.

There was histological evidence of mild superficial gastritis in two cases, and atrophic gastritis in one other, when these conditions were not suspected from the gross appearance of the mucosa.

The pathogenesis and possible importance of gastritis in the herniated stomach in patients with sliding esophageal hiatus hernia have been discussed.

REFERENCES

1. DAGRADI, A. E., KILLEEN, R. N. AND SCHINDLER, R.: *Gastroenterology*, 35: 54, 1958.
2. PALMER, E. D.: *Amer. J. Dig. Dis.*, 3: 45, 1958.
3. ATKINS, L. AND BENNEDICT, E. B.: *New Engl. J. Med.*, 254: 641, 1956.
4. SELESNICK, S. AND KINSELLA, E. D.: *Ibid.*, 248: 842, 1953.
5. YLVISAKER, R. S. *et al.*: *Gastroenterology*, 28: 88, 1955.
6. DOIG, R. K. AND WOOD, I. J.: *In*: Modern trends in gastroenterology, edited by A. F. Jones, Butterworth and Co., London, 1958, p. 208.
7. PALMER, E. D.: *Medicine (Balt.)*, 33: 199, 1954.
8. SAHLER, O. D. AND HAMPTON, A. O.: *Amer. J. Roentgenol*, 49: 433, 1943.
9. PALMER, E. D.: *In*: The esophagus and its diseases, Paul B. Hoeber, Inc., New York, 1952, p. 131.
10. STEMPIEN, S. J. AND SILK, A. D.: *Calif. Med.*, 78: 499, 1953.
11. MARCHAND, P.: *Brit. J. Surg.*, 47: 515, 1960.
12. AYLWIN, J. A.: *Thorax*, 8: 38, 1953.
13. JOSKE, R. A., FINCKH, E. S. AND WOOD, I. J.: *Quart. J. Med.*, 24: 269, 1955.
14. JACOBS, W. H.: *J. Mount Sinai Hosp. (N.Y.)*, 26: 464, 1959.
15. FELDER, S. L., MASLEY, P. M. AND WOLFF, W. I.: *A.M.A. Arch. Int. Med.*, 105: 873, 1960.
16. JOHNS, T. N. P. AND CLEMENTS, E. L.: *J. Thor. Cardiov. Surg.*, 41: 737, 1961.
17. WOOD, I. J. AND TAFT, L. I.: *In*: Diffuse lesions of the stomach, Edward Arnold Ltd., London, 1958, p. 50.
18. *Idem*: *Ibid.*, p. 65.

# Congenital Hypofibrinogenemia in Five Members of a Family

R. HASSELBACK, M.D., F.R.C.P.[C], RITA B. MARION, A.R.T.
and J. W. THOMAS, M.D., *Vancouver, B.C.*

**ABSTRACT**

**The bleeding tendency in five members of one family with fibrinogen levels ranging from 58 mg. % to 158 mg. % was mild and chiefly related to dental extractions. Abruptio placentae in one patient produced severe bleeding. Reports of menstrual bleeding patterns in patients with defects of hemostatic mechanisms suggest that normal platelets, vascular function and extrinsic and possibly intrinsic coagulation systems, except for fibrinogen, control menstrual blood loss. An autosomal dominant gene with variable penetrance may determine fibrinogen levels.**

OVER 30 cases of congenital afibrinogenemia have been reported to date.[11, 14, 29] Hypofibrinogenemia, sometimes referred to as fibrinogenopenia, has been reported much less frequently and only a few cases of this disorder have been described in the literature.[10, 31, 35, 43] This report concerns five members of one family with moderately low levels of fibrinogen.

## HISTORY OF PROPOSITI

CASE 1.—M.N., a 27-year-old married woman, was admitted to the Vancouver General Hospital in February 1960, for excision of a Bartholin cyst. From the age of 20 she had noted excessive bruising after minor trauma, bleeding lasting up to two days after dental extractions, and a postpartum hemorrhage at the age of 24 which required two transfusions after the birth of her only child. She had had no bleeding which could be described as spontaneous, no joint hemorrhages, and her menses had always been lighter than average. After excision of the Bartholin cyst, she developed a large hematoma of the vulva that had to be evacuated 36 hours postoperatively; no transfusions or blood products were administered. Excision of skin tags that were causing dyspareunia was carried out five months later, without excessive bleeding. The results of the investigations of this patient are shown in Table I.

CASE 2.—J.B., the 19-year-old sister of M.N., presented at another hospital with vaginal hemorrhage during the 32nd week of her first pregnancy. Her previous history of bleeding was limited to one to two days' oozing after dental extractions on two occasions; two heavy, prolonged menstrual periods at the ages of 12 and 13; and one very heavy period at age 18 which required the transfusion of two bottles of blood. Otherwise her menses had been normal. Neither the patient nor her mother had noted any other bleeding episodes. On admission, a diagnosis of abruptio placentae was made. The addition of thrombin to the patient's plasma gave only a delayed poor clot suggesting an associated defibrination syndrome. After 16 hours of ineffective labour, a classical Cesarean section was carried out. At operation, there were multiple small, blue hematomas in the uterus, with a large hematoma involving the right tube and ovary. Liquid blood, to 1500 ml., was free in the uterine cavity, external to the amniotic sac. A 1420-g. female infant was delivered live, but expired almost immediately. The patient received 2500 ml. of blood, 500 ml. of polyvinylpyrrolidone, and 2.2 g. of fibrinogen concentrate before and during the procedure. Moderate oozing but no extensive

From the Department of Pathology, Vancouver General Hospital, and the Department of Medicine, The University of British Columbia, Vancouver, B.C.