Mallory-Weiss Syndrome *

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THE PATIENT with upper gastro-intestinal hemorrhage is a major challenge to the conscientious surgeon. A carefully performed history and physical examination and a minimum of laboratory study will uncover the 80 per cent with benign peptic ulceration of the stomach, the 5.0 per cent with benign and malignant gastric neoplasms, and the 5.0 per cent with portal hypertension and bleeding from esophageal varices. Esophageal tamponade will help delineate the latter group. Of the uncommon lesions comprising the remaining 10.0 per cent, it can only be said that even when great skill and care and experience are brought to bear on the problem, the diagnosis can elude discovery and a fatality may result from uncontrolled exsanguinating hemorrhage. When, as occurs occasionally, the pathologist is unable to discover the source of bleeding at autopsy, the magnitude of the problem is glaringly apparent.

Within the spectrum of causes of upper gastro-intestinal hemorrhage is an entity which, although it was described many years ago, is just recently coming into its own as a recognizable and curable lesion of the gastro-esophageal junction. The lesion is a cardio esophageal or esophageal laceration first described by Mallory and Weiss, in 1929,[7] and the clinical picture associated with this lesion is known as the Mallory-Weiss syndrome.

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Case Report

This 50-year-old, chronic alcoholic, white man was admitted to the hospital on September 6, 1959. Eighteen hours before admission during a bout of heavy drinking, he was overcome with nausea and vomited bright red blood. Six such episodes followed in the course of the day. Two bowel movements occurred during this period, the second of which was tarry. He was admitted to the hospital weak, dizzy, and on the verge of collapse. At no time did he complain of pain. The patient admitted to excessive alcoholic intake for years with frequent compromise of food intake. A 10- to 20-pound weight loss had occurred in the past year. He denied similar previous episodes or any serious previous illness in the past except for pneumonia many years ago.

Physical examination revealed a restless, pale, cooperative man with a right periorbital hematoma. The blood pressure was 90/50 and the pulse was 120 per minute and weak. The heart and lungs were normal. On examination of the abdomen the liver edge was 7.0 cm. below the right costal margin. The spleen was not palpable. There was no abdominal tenderness, no spider angiomata were present, and the palms were normal. Shortly before and during the interrogation and physical examination, the patient vomited 700 cc. of dark red blood.

On admission, the hemoglobin was 11.5 Gm. per cent and the hematocrit was 33 mm. After a relatively uneventful night in the hospital, hematemesis recurred and the blood pressure which had risen to 114/70 without transfusion suddenly fell to 86/50. During the course of the second hospital day, 2,500 cc. of blood were administered. A Sengstaken-Blakemore tube was passed but persistent tamponade failed to stop the bleeding. It was decided that the source of the hemorrhage was distal to the cardia and that the most likely diagnosis was peptic ulcer. Since it was evident that the bleeding was unrelenting, operative exploration was undertaken on the evening of the second hospital day.

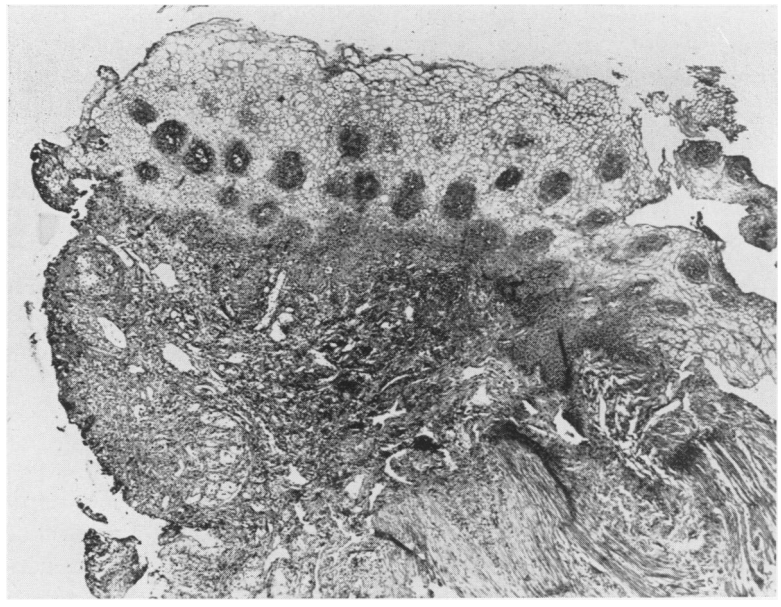

FIG. 1. Low-power photomicrograph demonstrating hemorrhage in the wall of the esophagus at the edge of the laceration which extends down to the muscularis (H. & E. stain, from × 40).

At operation the upper gastro-intestinal tract was filled with blood. After wide gastrotomy no duodenal or gastric lesion was found. Because blood was seen to emerge from the cardia, the esophagus was extensively mobilized and the lower third was easily delivered into the abdomen. The gastrotomy was then extended proximally to the cardia and onto the distal three inches of esophagus. In the long axis of the posterior wall of the esophagus there was a two and one-half inch long laceration ending at the cardia. The maximum width of the laceration was one half inch and the surrounding esophageal mucosa, including the edges of the laceration, appeared grossly normal. Small arteries were bleeding in its depth. There were no esophageal varices. A biopsy specimen was taken of one edge of the laceration and the latter was repaired with a running chromic catgut suture. The anterior esophageal wall and the stomach were repaired in layers and a specimen of the liver was removed for biopsy. Microscopic examination of the wall of the laceration showed it to extend into the hemorrhagic submucosa. There was also light round cell infiltration of the mucosa at one point (Fig. 1). The liver biopsy was normal.

The postoperative course was uneventful except for a left lower lobe pneumonia which eventually cleared. The patient has been seen at intervals. One month following the operation, oral barium X-ray study revealed slight irregularity in the distal esophagus. He continues to feel well but persists in his drinking habits. His sole complaint is discomfort in a ventral herniation which developed at the upper extremity of the midline abdominal incision. He has no pain and close questioning fails to reveal any evidence of dysphagia or esophageal difficulty.

Discussion

Spontaneous laceration of the lower esophagus and cardia is recognized infrequently as a source of massive upper gastrointestinal hemorrhage. The classic presentation by Mallory and Weiss[7] of 15 patients with this syndrome is an excellent example of a profitable collaboration of pathologist and clinician. These authors noted the characteristic history that after heavy drinking for days or weeks, their patients experienced persistent nausea, retching and vomiting. Four of these 15 patients came to autopsy and exhibited strikingly identical lesions at the cardiac opening of the stomach. These consisted of from two to four fissure-like lesions of the mucosa, characteristically arranged around the circumference of the cardiac opening, along the longitudinal axis of the esophagus. The size of the lesions varied from 3.0 to 20 mm. in length, and from 2.0 to 3.0 mm. in width. The edges were raised and slightly thickened, but not indurated. On microscopic

section, the lesions were found to be ulcerations of the mucosa extending as deep as the muscularis. The floor of the ulcers was composed of fresh fibrin and an exudate of polymorphonuclear leukocytes. In some of the sections, definitely ruptured arterioles were observed.

In 1932, the same authors [11] presented six additional cases and from this point on there was a complete void in the literature until 1953 when Decker, Zamcheck and Mallory [1] reported autopsy findings of 11 more cases of gastroesophageal lacerations. These authors emphasized again that vomiting was the common factor in the pathogenesis of the syndrome. They agreed with Mallory and Weiss' concept that the gastroesophageal lacerations were produced by violent retching movements which forced the gastric contents against a contracted cardiac sphincter. They also point out that atrophic gastritis is also a possible underlying factor in the production of lacerations at the cardia especially when there is associated alcoholism. It should be pointed out that the vomiting which initated the attacks of bleeding in the various reports available has been caused by different precipitating factors, including pernicious vomiting of pregnancy, motion sickness, postoperative nausea, uremia, pyloric stenosis, acute gastro-intestinal inflammation, intake of alcohol, severe migraine headache and marked emotional upset.[2]

In discussing their pathological observations at autopsy, Decker, Zamcheck and Mallory [1] pointed out that the lacerations observed conformed to the original descriptions of Mallory and Weiss. Most of them involved both the distal end of the esophagus and the proximal portion of the stomach, the lesion often being nearly bisected by the cardioesophageal junction. They noted that a few lesions were confined to the stomach alone immediately distal to the gastroesophageal junction, but *none* of the lacerations was limited to the esophagus alone. Review of the more recent literature provides evidence of a somewhat different picture as seen by the gastroscopist or surgeon in the live patient. Three groups of patients could be delineated when only the location of the laceration was considered:

Group I: Three cases. Laceration only in the esophagus (8, 9 and the present author's case).

Group II: Six cases. Laceration limited to the stomach (4—two cases [3-5, 12]).

Group III: One case. Laceration extends from esophagus across cardia into the stomach.[10]

It would appear then that in the live patient, lacerations are present either in esophagus or in stomach and in only one patient of ten is the cardia straddled by the ulcer as in the classical Mallory-Weiss syndrome predicated on observations at autopsy. An attempt to explain this difference is in order.

In discussing spontaneous rupture of the normal esophagus, Mackler [6] raises the question as to whether or not it is possible to generate a force sufficient to burst a normal, healthy esophagus by the effort of vomiting. He then suggests an affirmative answer by pointing out that rupture of the esophagus has occurred in numerous cases in which vomiting was stimulated by causes unassociated with this organ, and in which there was no history of any pre-existing esophageal disease. Thus, rupture has occurred during the vomiting caused by seasickness, anesthesia, small intestinal obstruction, cerebral stimulation, self-induction, peritoneal irritation, and large ovarian cysts. Mackler then deals with the problem of how a rupturing pressure could be built up in the esophagus in the absence of obstruction toward the outlet of the tube. He concludes that obstruction is necessary to build up the necessary hydrodynamic forces, but that the obstruction is usually functional rather than organic.

The act of vomiting is executed by a series of coordinated reflexes including a

forward movement of the larynx and hyoid bone and a relaxation of the cricopharyngeal sphincter to provide a wide exit for the contents of the stomach. In prolonged and repeated vomiting, ensuing fatigue of the vomiting center or fatigue of the muscular components with failure to respond promptly to nervous stimuli may result in incoordination. Reflex relaxation of the upper outlet may fail to coincide with the onrush of gas and liquid content from the stomach. The presence of previously regurgitated hydrochloric acid in this region, too, may promote spasm and delay in prompt relaxation. The sphincteric obstruction in the face of the generating forces from below may be responsible for the momentary development of sufficient pent-up pressure to burst the esophagus. This mechanism can be invoked to explain the cases of Mallory-Weiss syndrome in Group I in which the laceration is limited to the lower third of the esophagus. A force as described above might be insufficient to rupture the full thickness of the esophageal wall and yet be sufficiently strong to lacerate the mucosa and submucosa. The three lacerations in Group I were located on the posterior wall of the esophagus in the general location and direction of the classical esophageal rupture.

An explanation for those lacerations limited to the juxta-cardial region of the stomach (Group II) must invoke the not unreasonable association of irritative pylorospasm and cardiospasm at the moment of maximum increase of intra-abdominal pressure. This concatenation of circumstances would force a high pressure wave against the proximal stomach of sufficient strength to bring about partial disruption of the wall in this area.

To understand the mechanism of the one case in Group III and the cases with classical Mallory-Weiss syndrome, pylorospasm must again be invoked. At the same time the cardia must be open and the proximal or middle esophagus must be unable to relax. This would allow a sudden increase in pressure above, at, and below the cardia and could give rise to the type of laceration under discussion. This situation is closely akin to the mechanism in Group I with one main difference. The patients in the autopsy group were in the main elderly (average age of 63) and debilitated with various associated chronic diseases. It may be postulated that these weakened individuals were unable to generate the strong force necessary to rupture the esophagus and instead developed a moderate and diffused pressure while vomiting sufficient to produce the lacerations of the classical Mallory-Weiss syndrome.

Surgical Management

The surgeon must keep all possibilities in mind in a patient with massive upper gastro-intestinal hemorrhage in whom a definite preoperative diagnosis has not been possible. With the abdomen opened, wide gastrotomy is mandatory if there is no duodenal or gastric lesion. Blind gastrectomy at this juncture is contraindicated for bleeding from the esophagus or cardia will go undiagnosed and untreated. This latter point has been emphasized by Stahlgren.[10]

After the anterior wall of the stomach has been incised for a distance of five to seven inches, judicious application of packs will determine the origin of the hemorrhage. Mallory-Weiss lacerations of the proximal stomach will easily come into view and can be closed with a running catgut suture. If blood originates in the esophagus then more adequate exposure is required. Careful dissection of the esophagus in the region of the hiatus can deliver three inches of the esophagus from the mediastinum. This exposure can be facilitated by excision of the xiphoid process. Gastrotomy can then be extended across the cardia into the esophagus, or a separate incision may be made in the anterior esophageal wall. The laceration will easily be detected on the posterior wall. Bleeding arteries in the base of the laceration will explain the preopera-

tive failure of esophageal tamponade that is effective only in the case of bleeding varices.

Summary

Violent retching followed by massive upper gastro-intestinal hemorrhage should alert the attending physician to suspect Mallory-Weiss syndrome. Bleeding in this condition is caused by mucosal lacerations in the cardioesophageal region. The cardia should be carefully inspected when this diagnosis is entertained and bleeding can be controlled by suture of the lacerations.

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