Mallory-Weiss Syndrome

Evolution of Diagnostic and Therapeutic Patterns Over Two Decades

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During a 19-year period ending December 1978, we treated 40 patients with upper gastrointestinal bleeding secondary to the Mallory-Weiss syndrome. Thirty patients had the triad of vomiting, hematemesis and alcoholism. The presence of lacerations within the gastric cardia was associated with the presence of hiatal hernia ($p = 0.03$). Endoscopic examinations demonstrated 32 of 38 additional upper gastrointestinal lesions associated with the syndrome that could have been mistaken as the actual source of hemorrhage. During the second decade, as compared with the first decade, widespread use of fiberoptic esophagogastroscopy led to the identification of the bleeding lacerations in 71% of the patients (versus 47% in the first decade) and in 80% (versus 0% in the first decade) of the patients who required an operation to control the bleeding. Although there was a 7.5% mortality rate in the two decades, the incidence of operative treatment tended to decrease (42–24%; $p = 0.13$). More impressive were the decreases in transfusions (14 units to 5 units per patient) and in delays before surgery (38 hours to 17 hours) ($p < 0.05$). Improved endoscopic diagnosis facilitates prompt and economic treatment.

FIFTY YEARS AFTER Mallory and Weiss[1,2] described a syndrome of acute postemetic lacerations of the gastric cardia as a source of major hemorrhage following an alcoholic debauch, confusion remains about several major aspects of the syndrome. These are 1) the physiologic events producing the syndrome, 2) the differentiation from postemetic rupture of the esophagus, and 3) the role of vomiting provoked by hiatal hernias in causing gastric lacerations in nonalcoholic patients. Even the frequency of the Mallory-Weiss syndrome as source of gastrointestinal bleeding is argued, with reported incidences ranging from 1–13%.[3–13] Although it cannot resolve all these issues, a review of the experience from the past two decades at this hospital has clarified them for us and has emphasized the key role of fiberoptic esophagogastroscopy in directing therapy.

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Materials and Methods

Patient Population

All cases of Mallory-Weiss syndrome diagnosed from January 1960, through December 1978, were reviewed. Only cases in which mucosal tears were clearly visualized at endoscopic examination, surgery, autopsy, or upper gastrointestinal contrast study[14] were included. In this 19-year period, 40 patients had one or more mucosal lacerations at the esophagogastric mucosal junction or near it. They represented 2.25% of 1780 patients admitted to the hospital for diagnosis and treatment of upper gastrointestinal bleeding.

Statistical Techniques

Ordinary parametric techniques were used to analyze continuous data. Mean values are reported plus or minus the standard error of the mean. When indicated, nonparametric methods for unpaired measurements (Mann-Whitney test) were used. Comparison of the percentages of discrete data was done by chi square and Fisher's exact tests. Survival curves were constructed according to Cutler and Ederer's life-table method.[15]

Results

Nineteen patients (48%) were admitted to the hospital between 1960 and 1969, and 21 patients (52%) were admitted from 1970. Thirty-two of the 40 patients (80%) were men (Table 1). The average age of the female patients was 21 years older than the average age of male patients. Bright red blood in the vomitus was present in 85% of the patients. In three patients, hematochezia was the only symptom.

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Major Identifiable Factors

The major identifiable inciting factors were chronic excessive intake of alcohol and ingestion of acetylsalicylic acid. Moderate or heavy ingestion of alcohol during the last hours immediately preceding hemorrhage was admitted to by 15 patients (38%). Seven of the 17 patients who mentioned epigastric pain as a symptom had a history of duodenal ulcer.

Hiatal Hernia

Hiatal hernia was diagnosed in 17 cases (42%), 16 times by endoscopic or radiologic methods and once at laparotomy.

Thirty-one patients had 38 mucosal abnormalities of the upper gastrointestinal tract other than gastric lacerations, of which 34% were bleeding, but none to a material degree. The vast majority (84%) of these additional abnormalities were identified by fiberoptic esophagogastroscopic examination. (Table 2).

Diagnosis

The diagnosis of the lacerations themselves was made by endoscopic examination in 24 patients (60%), at laparotomy in ten patients (25%), by upper gastrointestinal contrast study in four patients (10%), and at autopsy in two patients (5%). The length of the tears ranged from 0.5 to 5 cm, and the number per patient ranged from one to five.

Of the 24 patients in whom location of the tears was determined accurately by endoscopic examination, 13 patients had hiatal hernias also demonstrated by endoscopic examination. In the six patients in whom the lacerations were confirmed to the lower esophagus, hiatal hernias were present in two patients (33%); in the ten patients with lacerations at the cardioesophageal junction, hiatal hernias were present in four patients (40%). By contrast, hiatal hernias were diagnosed in seven (87%) of the eight patients with lacerations at the gastric cardia (Table 3).

TABLE 1. *Clinical Features of Mallory-Weiss Syndrome*

	Number of Patients	Per Cent
Sex		
female	8	20
male	32	80
	40	100
Age (Years ± S.E.)		
female 73 ± 5.0*		
male 52 ± 2.9		
Features		
alcoholism	30	75
aspirin	5	12
epigastric pain	17	42
emesis	35	87
hematemesis	34	85
melena	16	40
shock	9	22
hiatal hernia	17	42

* $P < 0.005$.

TABLE 2. *Upper Gastrointestinal Lesions Associated with Mallory-Weiss Syndromes*

Types of Lesions	Number of Lesions
Esophageal	
esophagitis	3
Schatzki's ring	1
Zenker's Diverticulum	1
diffuse esophageal spasm	1
submucosal hematoma	2
varices	0
Gastric	
erosive gastritis	4*
gastritis	6*
ulcer	0
atrophic gastritis	2
Duodenal	
duodenitis	3*
ulcer	15
	38

* Associated lesions that were also bleeding.

Treatment

Twenty-five patients (62%) required transfusions. The severity of the gastrointestinal bleeding was characterized by an average of 8.6 ± 1.7 units of blood administered per patient (range: 1–39; median: 7). Of the 40 patients with Mallory-Weiss syndrome, only 13 patients (32%) required operations. The operations generally consisted of oversewing the lacerations with a running suture of 2-0 chromic catgut through an anterior gastrostomy. One patient sustained both a spontaneous perforation on the left side of the distal esophagus (Boerhaave's syndrome) and a laceration of the cardia (Mallory-Weiss syndrome) on the right side of the stomach; he required suturing of the esophageal perforation through a left thoracotomy.

Although seven of the 13 patients (54%) treated by an operation were in hypovolemic shock when admitted to the hospital (systolic blood pressure less than 90 mmHg), only two of 27 patients (7%) treated medically were hypotensive ($p = 0.002$). The average volume of blood required for transfusions for the patients who were operated on was 10.8 ± 2.8 units, as compared with only 2.8 ± 0.9 units in the medically treated group of patients ($p < 0.001$).

Comparison of the clinical data of the 40 cases of Mallory-Weiss syndrome by decade 1960–69 versus 1970–78 showed that eight of 19 patients (42%) were

TABLE 3. *Association of Hiatal Hernia with Location of Mallory-Weiss Laceration Determined by Endoscopy*

Location of Laceration	Number of Patients	Hiatal Hernia	
		Number	(%)
Esophagus	6	2	33*
Cardioesophageal junction	10	4	40†
Stomach	8	7	87‡
	24	13	

* Esophagus versus stomach: p = 0.06.
† Cardioesophageal junction versus stomach: p = 0.05.
‡ Stomach versus combined esophagus and cardioesophageal group: p = 0.03 (Fisher's exact test).

operated on before 1970 and five of 21 patients (24%) were operated on after 1970 (p = 0.13). The average volume of blood required for transfusions before initiation of surgical treatment was 14.5 units before 1970, but only 5.0 units after 1970 (p < 0.05, Mann-Whitney test). This difference in the volume of transfusion between the two decades is probably related to the fact that four out of five patients (80%) operated on after 1970 underwent emergency diagnostic endoscopy, leading to an earlier operation (17.7 hours after admission to the hospital versus 38.7 hours before 1970; p < 0.05). None of eight patients (p = 0.01) operated on before 1970 underwent esophagogastroscopy during the acute episode of bleeding. The overall incidence of diagnostic endoscopic procedures increased from 47% (9/19) before 1970 to 71% (15/21) after 1970 (p = 0.08).

Medical treatment consisted of carrying out the necessary resuscitative measures and of initiating volume or blood replacement if the patient was in shock. Washing out the stomach with ice water and cleansing the stomach for subsequent endoscopy was, itself, successful in 27 of 31 cases for controlling the gastrointestinal bleeding. The medical management of continued bleeding from the lacerations included the use of intravenous vasopressin in only two patients. One of the two patients had Laennec's cirrhosis and finally required oversewing of four Mallory-Weiss lacerations.

Before 1970, three patients underwent balloon tamponade procedures with Sengstaken-Blakemore tubes in a desperate attempt to control the bleeding without surgical intervention. These maneuvers were unsuccessful in all three patients, who were finally operated on after delays of 30, 48 and 72 hours, respectively, providing another explanation for the discrepancy in the volume of transfused blood and the delay before surgical treatment between the two decades.

Mortality Rates

The overall mortality rate in this series was only 7.5% (three of 40 patients). The ten-year survival rate was 73 ± 11% (Fig. 1). There was no documented recurrence of Mallory-Weiss syndrome.

The three deaths were all in patients not operated on. One patient had Laennec's cirrhosis, but did not bleed from any other source than the gastroesophageal lacerations. This patient required 20 units of blood to maintain his hematocrit at 30%; he died before surgical treatment could be initiated. The two other fatal cases of Mallory-Weiss syndrome were diagnosed at autopsy. In one case, autopsy demonstrated presence of acute mediastinitis, but no perforation of the esophagus could be identified to explain this mediastinitis. The second patient had sustained 80% body surface area third degree burns four hours before admission and died in septic shock.

Discussion

According to recent reports,[8,11,12] the cause of acute postemetic lacerations of the gastroesophageal junction has shifted from alcohol-induced vomiting exclusively to vomiting induced by coughing paroxysms, uremia, pregnancy, and the acute development of high intra-esophagogastric pressures secondary to heavy lifting, straining at bowel movement, hiccoughing under anesthesia, and blunt abdominal trauma. Nonetheless, 75% of our patients manifested with the classic

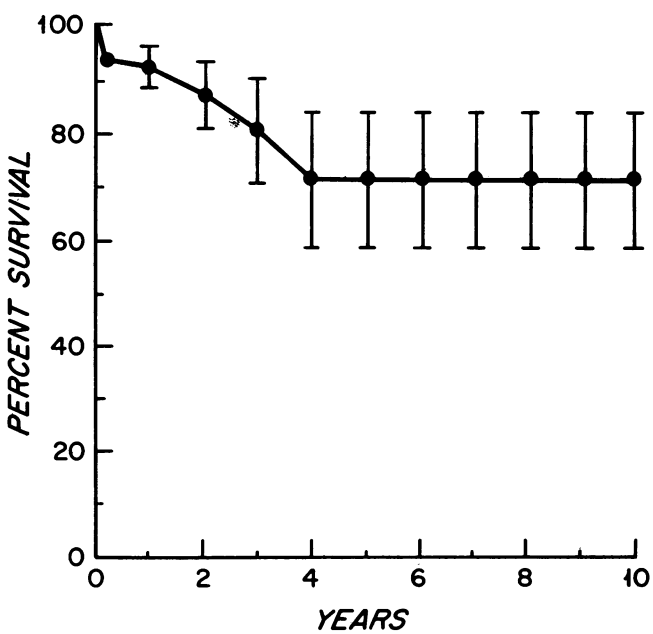

FIG. 1. Ten-year survival rate after treatment for Mallory-Weiss syndrome calculated by the actuarial method.

triad of vomiting, hematemesis, and alcoholism, as described by Mallory and Weiss in their initial report.

Incidence and Importance of Upper Gastrointestinal Lesions Associated with Mallory-Weiss Syndrome

Associated upper gastrointestinal mucosal lesions were found in 77% of the patients we treated. Such an association has been reported in other series.[7,11,16,17,18] In one series,[7] the incidence of associated lesions amounted to 83%. Indeed, these lesions may be the instigating cause of the retching or vomiting and produce the Mallory-Weiss syndrome indirectly. Furthermore, since these lesions could be mistaken as the source of bleeding, the value of thorough and early panendoscopy in patients who actually have the Mallory-Weiss syndrome is obvious if surgery is contemplated.

Effect of Hiatal Hernia on the Pathogenesis and the Location of Mallory-Weiss Lacerations

The Mallory-Weiss syndrome seems to occur more frequently in patients with hiatal hernias. The reported incidences range from 35 to 100%.[7,17,19–23] The intraluminal pressure in the herniated portion of the stomach corresponds with that in the subdiaphragmatic portion of the stomach rather than with the intrathoracic pressure.[20,24] The pressure gradient existing across the wall of the herniated portion can reach 100 torr or more during retching or vomiting, with consequent forceful stretching of the wall and eventual laceration. We agree with Watts[23] that an hiatal hernia may influence the site of the mucosal tear associated with emesis. The tears occuring in the esophagus or overlying the cardioesophageal junction are more often associated with the absence of demonstrable hiatal hernia; in contrast, tears located within the gastric cardia are generally associated with the presence of hiatal hernia.

The Role of Fiberoptic Esophagogastroscopy in Changing the Diagnostic and Therapeutic Approach of Mallory-Weiss Syndrome

Review of the literature[5,7,12,17,25] shows an increasing trend toward the use of nonoperative treatment of the Mallory-Weiss syndrome (Table 4). Seventy-nine per cent of the cases reported since 1969[5,7,12] were treated without operation, for a 98% survival rate. This changing clinical picture has been attributed to widespread use of the fiberoptic endoscope.[10,12]

Fiberoptic endoscopy has clearly led to a major improvement in the diagnosis of upper gastrointestinal hemorrhage, one aspect of this improvement being the increasing frequency of identification of Mallory-Weiss syndrome.[9,26,27] Less serious forms of lacerations are also recognized and treated medically now, as compared with the era before fiberoptic endoscopy. In addition, gastric lavage with ice water or iced saline solution to clean the stomach before emergency endoscopic study will often, by itself, stop the bleeding from a gastric laceration.

Other Therapeutic Modalities

Balloon tamponade for the treatment of Mallory-Weiss syndrome is scarcely justified if an early diagnostic approach is undertaken to determine what disease is present and which lesion is responsible for the bleeding. Furthermore, because hiatal hernia is frequently associated with the Mallory-Weiss syndrome, balloon tamponade is actually contraindicated since the inflated balloon may cause gastric tears in the herniated portion of the stomach.[28]

The rationale for the use of aqueous vasopressin in the treatment of gastrointestinal bleeding is based on its ability to constrict splanchnic arterioles. Successful use of intravenous vasopressin to control the bleeding from a Mallory-Weiss syndrome was reported in 1971,[29] and has since been used in two of our patients, with one success.

TABLE 4. *Type of Treatment and Survival Rate in Collected Series of Mallory-Weiss Syndromes (1929 through 1978)*

Period	Number of Cases	Per Cent of Patients Treated by Operation	Per Cent of Patients Who Survived Operation	Per Cent of Patients Treated Medically	Per Cent of Patients Who Survived Medical Treatment
1929–1966[25]	121	52	81	48	38
1966–1969[17]	108	47	90	53	86
1969–1978[5,7,12]	220	21	96	79	98
Massachusetts General Hospital Series					
1960–1969	19	42	100	58	91
1970–1978	21	24	100	76	88
Total	489				

Recently, transcatheter therapeutic embolization, introduced for control of bleeding from esophageal varices, has been reported to be successful also in four patients with Mallory-Weiss syndrome.[30] Massive bleeding was controlled by Gelfoam® embolization of the left gastric artery.

Distinction Between the Mallory-Weiss Syndrome and the Boerhaave's Syndrome

Both the Mallory-Weiss and Boerhaave's syndromes are manifested by upper gastrointestinal lesions due to vomiting, but the term "Mallory-Weiss syndrome" has been used in some reports to describe both hematemesis from lacerations of the esophagogastric junction and spontaneous rupture of the terminal esophagus irrespective of the presence or absence of hematemesis.[19,28] The term "Mallory-Weiss syndrome" should be restricted to patients with lacerations of the esophagogastric junction, the clinical presentation being massive hematemesis with or without shock, and generally without pain. Reported mortality rates for Mallory-Weiss syndrome range from 0 to 25%. In our series, the mortality rate of 7.5% corresponds to the 7.9% rate reported by Palmer[9] in a 23-year prospective study of 1,400 patients with upper gastrointestinal bleeding.

Patients with postemetic spontaneous perforations of the esophagus are properly classified as examples of the Boerhaave's syndrome. Surgical treatment is generally indicated and has to be performed as early as possible. Mortality rates are higher if surgery is delayed (30–70% in various series). In addition to the 40 patients with Mallory-Weiss syndrome treated between 1960 and 1978, ten patients with Boerhaave's syndrome were treated at the Massachusetts General Hospital. The mortality rate for the group of patients with Boerhaave's syndrome was 40%, versus a 7.5% mortality rate for patients with Mallory-Weiss syndrome ($p = 0.03$).

Despite differences in management and prognosis between these two syndromes, there is good reason to believe that they share a common pathogenesis.[24,28] The fact that one patient in our series had an unruptured laceration of the cardia on the right side of the stomach, in addition to a linear rupture of the cardioesophageal junction of the left side, lends weight to the argument that a laceration is an incomplete rupture and that rupture is a complete laceration.[2,24,28,31]

Recommendations for the Management of the Mallory-Weiss Syndrome

Patients who present themselves with upper gastrointestinal bleeding and have an history of vomiting or retching, especially when associated with recent alcohol intake, should be examined for presence of the Mallory-Weiss syndrome.

Since the bleeding will be controllable by nonoperative supportive therapy in the majority of these patients, and since it is not always possible to make the diagnosis by the patient's history alone, early esophagogastroscopy is indicated. Endoscopic diagnosis will accurately reveal the severity of the bleeding, the location of the bleeding and the presence of other upper gastrointestinal mucosal lesions. It will help direct the surgeon to the responsible lesion when a surgical procedure is required to control the hemorrhage.

The resuscitative measures that should be carried out before and during the emergency diagnostic procedures include: immediate assessment of the clinical and hemodynamic status, maintainence of ihtravascular volume, and gavage with ice water using a No. 36F Ewald® tube. In many instances, bleeding from Mallory-Weiss lacerations is so minor that gavage with ice water controls the bleeding.

Balloon tamponade is contraindicated.

Ten units of vasopressin diluted in 10 ml of saline solution can be injected intravenously over a ten-minute period. The vasopressin injection can be repeated at intervals of one to two hours, or preferably a constant intravenous infusion of 0.3 to 0.4 units per minute can be started. However hemodynamic function and urine output must be monitored carefully, since tachyphylaxis and an antidiuretic effect can develop.

Transcatheter embolization for control of massive bleeding from Mallory-Weiss tears is a promising new technique, but can not yet be recommended as primary treatment.[30]

The indications for the surgical treatment of Mallory-Weiss syndrome are decreasing, and no definite study has demonstrated the value of one surgical procedure over another. The surgical technique generally used to control massive bleeding from a Mallory-Weiss laceration that remains refractory to all conservative measures is oversewing the lacerations with a running suture of 2-0 catgut through an anterior gastrotomy. The best exposure is obtained through gastrostomy made in the middle one-third of the stomach and by using a large Deaver retractor while pulling upward on the nasogastric tube. This allows the cardia to be brought into view and exposes the folds at the esophagogastric junction.[32]

For lacerations that extend higher into the lower esophagus, left thoracotomy may be necessary to expose the bleeding tears by opening the esophagus. If unremitting hemorrhage from associated gastritis complicates the situation, truncal vagotomy should be coupled with a generous gastrectomy.[33]

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