DIAPHRAGMATIC HERNIA AND ASSOCIATED ANEMIA: RESPONSE TO SURGICAL TREATMENT

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From 1985 to 1993, 49 patients (35 women and 14 men) with diaphragmatic hernia and associated anemia underwent surgical repair. The median age was 64.5 years (range 24 to 84 years). Hematologic and gastroenterologic evaluations revealed no other potential cause of bleeding. Each patient had a diaphragmatic hernia. The median time between the diagnosis of anemia and surgical repair was 36 months (range 1 to 334 months). Forty-five patients (91.8%) had received replacement therapy, including iron for 43 and blood transfusions for 32 (median 6 units; range 2 to 70 units). Forty-six patients (93.9%) had symptoms: heartburn in 28, early satiety with bloating in 19, regurgitation in 11, dysphagia in 7, and aspiration in 4. Preoperative upper gastrointestinal endoscopic evaluation demonstrated gastric erosions at the level of the hiatus in 22 patients (44.9%), esophagitis in 7, stenosis in 1, and Barrett's disease in 1. An uncut Collis-Nissen fundoplication was performed in 44 patients, Belsey fundoplication in 2, a cut Collis-Nissen fundoplication, Nissen fundoplication, and Hill repair in 1 each. There was one operative death (2% mortality). Complications occurred in 18 patients (36.7%). Follow-up was complete and ranged from 4 to 103 months (median 63 months). Forty-five patients (91.8%) had resolution of their anemia. Functional results were excellent in 40 patients (81.6%), good in 2 (4.1%), fair in 4 (8.2%), and poor in 3 (6.1%). In most patients with diaphragmatic hernia and associated anemia refractory to medical treatment, surgical repair can result in successful resolution of the anemia. (*J Thorac Cardiovasc Surg* 1996;112:1340-5)

Diaphragmatic hernia (DH) at the esophageal hiatus is the most common abnormality of the upper gastrointestinal tract, occurring in approximately 10% of the North American population.¹ Although acute or chronic blood loss may occur as a complication of reflux esophagitis, it is unusual. Chronic anemia has been associated with DH, but reports of results after surgical repair are rare.²⁻⁸ These patients frequently have a chronic history of anemia without obvious cause, despite extensive and repeated diagnostic evaluations. To better under-

stand the benefit of hernia repair in patients with DH and associated anemia, we reviewed the cases of all patients whose conditions were surgically managed at our institution during the past decade.

Patients and methods

Between January 1, 1985, and December 31, 1993, 39,112 patients seen at the Mayo Clinic had a diagnosis of DH. Associated anemia was diagnosed in 349 patients (0.89%), and 49 (0.13%) underwent surgical repair. Anemia was defined as a hemoglobin concentration less than 12.0 gm/dl in women and less than 13.5 gm/dl in men; the values were determined at the Mayo Medical Laboratories. All patients had associated DH, with at least 25% of the stomach above the diaphragm as determined by means of upper gastrointestinal roentgenography or esophago-gastroduodenoscopy. All patients underwent complete hematologic and gastroenterologic evaluations, which revealed no other cause of bleeding, and most had anemia that was refractory to medical treatment.

The records of these 49 patients were analyzed for demographic data, presenting signs and symptoms, associated medical conditions, evaluation and duration of DH and anemia, operative indications, operative procedure, complications, and long-term outcome. Follow-up was accomplished by review of our outpatient records, a

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Table I. Complications occurring after repair of diaphragmatic hernia

Complication	No. of patients
Major	9 (18.4%)
Pneumonia	2
Respiratory failure	2
Leak*	2
Bleeding	2
Lacerated left main stem bronchus	1
Minor	9 (18.4%)
Atrial fibrillation	2
Respiratory depression due to narcotics	2
Atelectasis	2
Left pleural effusion requiring thoracentesis	1
Transient global amnesia	1
Small bowel ileus	1

*Postoperative death.

telephone interview with the patient, or both. The last hemoglobin value recorded was used to determine the degree of anemia. Functional results after repair were considered "excellent" if the patient was eating a general diet without experiencing symptoms or requiring medication, "good" if symptoms were minimal and neither medication on a regular basis nor dilatation was needed, "fair" if symptoms were improved but medication on a regular basis or dilatation was required, and "poor" if symptoms were unchanged or worse or the patient required another operation. Operative mortality included all deaths within the first 30 days of the operation or during the same hospitalization if it was longer than 30 days.

Clinical findings. The patients studied comprised 35 women and 14 men. The median age was 64.5 years, and the range was 24 to 84 years. All patients were initially seen with a history of anemia that had existed for a median of 36 months (range 1 to 334 months). The median preoperative hemoglobin level was 11.0 gm/dl (range 6.4 to 15.2 gm/dl). Twelve patients (24.5%) initially had with a hemoglobin level of less than 10.0 gm/dl before hernia repair. Forty-five patients (91.8%) had received replacement therapy before surgery. Forty-three of these patients were receiving chronic oral iron replacement, and 32 patients had received blood transfusions. The median number of units transfused was 6 (range 2 to 70 units).

Symptoms were present in 46 patients (93.9%) and included heartburn in 28 (57.1%), early satiety with bloating in 19 (39%), regurgitation in 11 (22.5%), dysphagia in 7 (14.3%), and aspiration in 4 (8.2%). Twelve patients (24.5%) were receiving antireflux medical therapy. Three patients had previous operations; two had antireflux procedures, and one had an exploratory laparotomy to evaluate anemia with small-bowel resection and cholecystectomy.

Upper gastrointestinal roentgenograms were obtained for 42 patients (85.7%). All patients had evidence of DH; 24 had most or all of the stomach contained within the chest. Upper gastrointestinal endoscopy was performed in all patients, and all were found to have a DH. Other

Fig. 1. After repair of the diaphragmatic hernia, 45 patients (91.8%) had resolution of their anemia.

endoscopic findings included linear gastric erosions at the level of the hiatus in 22 patients (44.9%), esophagitis in 7 (14.3%), and stenosis and Barrett's esophagus in 1 patient (2.0%) each. Esophageal manometry and motility testing were performed in 42 patients (85.7%). Normal motility was documented in 25 patients. Twelve patients had abnormal examination findings, which included reduced lower esophageal sphincter pressure in six, weak peristalsis in four, and reduced lower esophageal sphincter pressure plus weak peristalsis in two. In five patients, the motility examination was incomplete because of the inability to pass the catheter into the stomach. No patient had achalasia or any other defined motility disorder. No patient underwent a 24-hour acid reflux test.

Operative indications included anemia in all patients, obstructive symptoms in 13, reflux esophagitis in 10, and combined obstruction and reflux esophagitis in 12. Surgical repair included an uncut Collis-Nissen fundoplication in 44 patients, Belsey fundoplication in 2, cut Collis-Nissen fundoplication in 1, Nissen fundoplication in 1, and Hill repair in 1.

Results

Median hospitalization was 8 days (range 6 to 28 days). There was one operative death (2.0% mortality). A left subphrenic abscess from a gastric leak developed in this patient. After repair and drainage, a Swan-Ganz catheter was placed to monitor cardiac status. Two days after reoperation, massive pulmonary hemorrhage occurred from a rupture of the left pulmonary artery, and the patient died shortly thereafter. Thirty-one patients (63.3%) had no postoperative complications. Of the 18 patients (36.7%) with complications, 9 had major and 9 had minor complications (Table I).

Follow-up was complete for all surviving patients and ranged from 4 to 103 months (median 63 months). Five patients died. The cause of death was myocardial infarction for three patients (at 42, 53, and 83 months), and death occurred after aortic

Fig. 2. Upper gastrointestinal roentgenograms of a patient with a large diaphragmatic hernia in the (A) supine position and (B) when standing upright. These views depict the movement and constriction that can occur at the diaphragmatic hiatus. (From Cameron AJ, Higgins JA. Linear gastric erosion. A lesion associated with large diaphragmatic hernia and chronic blood loss anemia. *Gastroenterology* 1986;91:338–42.)

valve replacement (26 months) for one and heart failure (3 months) for one. At last follow-up, 45 patients (91.8%) had resolution of their anemia (Fig. 1), and none was receiving replacement therapy. Two patients are alive with persistent anemia. One of these patients has chronic ulcerative colitis, and the other has diverticulosis. Neither has had rectal bleeding or guaiac-positive stools, and the cause of the anemia is still unknown. A third patient who died 3 months after DH repair of heart failure was still anemic at the time of death.

Functional results were excellent for 40 patients (81.6%), good for 2 (4.1%), fair for 4 (8.2%), and poor for 3 (6.1%). Eight patients had dysphagia; it was transient in six, moderate in one, and severe in one. Three patients underwent postoperative esophageal dilatations; one had one dilatation, one had two, and one had 35. Five patients had a history of taking antireflux medication; three took H₂ blockers, one took a proton pump inhibitor, and one took antacids. Three patients had poor results. Two of these had revisions of the hernia repair for dysphagia. The third patient died postoperatively. Other late postoperative findings included occasional bloating in 7 patients, excessive gas in 6, ventral

hernia in 1, persistent incisional pain in 1, and pulmonary embolus in 1.

Discussion

DH is the most common upper gastrointestinal abnormality.¹ Most patients do not have symptoms. When symptoms develop, the cause is usually an incompetent lower esophageal sphincter leading to reflux esophagitis or a large diaphragmatic or paraesophageal hernia leading to an intrathoracic stomach with obstruction. A subset of patients have DH and associated anemia.³ Cameron compared 259 patients with DH with an equal-sized age- and sex-matched control group without DH and found 18 patients with anemia in the first group (6.9%) and 1 (0.4%) in the second ($p < 0.001$).⁹

Although difficult to prove, the pathophysiology of anemia is thought to result from mechanical irritation of the stomach as it passes back and forth through the diaphragmatic hiatus or intermittent torsion in the case of intrathoracic stomach (Fig. 2).^{3,7} This ultimately leads to mucosal erosion with chronic bleeding (Fig. 3).⁷ Cameron and Higgins⁷ described an increased incidence of linear gastric erosions in a group of 55 patients with DH and

anemia compared with a similar group of 54 patients with anemia but without DH. It is hard to determine what role associated reflux esophagitis may play in the cause of anemia. Acute or chronic blood loss may occur, but it is rare.

Examination of patients who have anemia and DH must be thorough. A careful hematologic evaluation to exclude other forms of anemia is indicated. A lower gastrointestinal evaluation, including colonoscopy, barium enema with proctoscopy, or both procedures, is mandatory. Upper gastrointestinal roentgenography examination and upper gastrointestinal endoscopy are indicated to find other causes of blood loss. Symptoms of reflux mandate further evaluation, including manometry, motility testing, and a 24-hour pH test for selected patients. Occasionally, intraabdominal angiography is indicated to rule out bleeding disorders, such as angiodysplasia, from the small or large bowel.

If the patient is anemic and all preoperative evaluations of the upper and lower gastrointestinal tracts and hematologic studies reveal no obvious site of blood loss, it is likely that the anemia is related to the DH. The anemia is initially treated with replacement therapy, including oral iron preparations. Some patients may require intermittent blood transfusions because the degree of anemia can be severe and quite symptomatic.¹⁰ In our study 91.8% of patients were receiving replacement therapy of some form before repair of the hernia. Moskovitz and colleagues¹¹ suggested that iron therapy plus H₂ antagonists may promote healing of linear gastric erosions and resolution of the anemia. If the anemia persists, we recommend surgical repair of the diaphragmatic hernia. Associated reflux esophagitis or obstructive symptoms may also be indications for repair.

Repair should reduce the stomach into the abdominal cavity and maintain a competent lower esophageal sphincter. We advocate repair of the DH with an antireflux procedure.⁸ However, some have argued against including an antireflux procedure for a pure paraesophageal hernia.¹² We and others believe the chance of having occult reflux disease is significant enough to include an antireflux repair.¹³ Equally important, mobilization of the hernia, resection of the sac, and repair of the DH may produce an incompetent lower esophageal sphincter. The Nissen fundoplication with or without a Collis gastroplasty performed through the chest is our procedure of choice if the degree of esophageal

Fig. 3. Upper gastrointestinal endoscopy shows mucosal irritation at the hiatus resulting in linear gastric erosions. (From Cameron AJ, Higgins JA. Linear gastric erosion. A lesion associated with large diaphragmatic hernia and chronic blood loss anemia. *Gastroenterology* 1986;91:338–42.)

peristalsis is within normal limits. If peristalsis is impaired, a Belsey repair is performed. The risks of such a procedure must be weighed against the benefits of resolving the anemia and associated symptoms. The risks of an antireflux procedure should be less than 1%.

Although none of our patients was managed with a laparoscopic Nissen fundoplication, this procedure may be beneficial in future series. This possibility will be determined by the results of long-term follow-up. The selection of which patient to repair laparoscopically depends on several factors, including the size of the hernia; only small hernias will be repaired at first. If the laparoscopic technique survives the test of time, it may become a less invasive way to manage this problem.

Reports of surgical results for patients with anemia and DH, although infrequent, have supported the idea of resolution of anemia after repair.^{2–8} In the current series, 45 (91.8%) of 49 patients experienced resolution of anemia after repair, and 85.6% of the patients had excellent or good functional results.

Anemia associated with DH may be difficult to diagnose and may have significant medical consequences, especially if it is refractory to treatment. A knowledge of the underlying pathophysiology and a high index of suspicion are required to make the

diagnosis. We believe that surgical repair is indicated for this group of patients because it usually leads to resolution of the anemia.

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Discussion

Dr. Nassar Altorki (*New York, N.Y.*). The term diaphragmatic hernia encompasses a variety of gastric herniations through the esophageal hiatus. The axial or the sliding type is the most common and is rarely, if ever, associated with occult or frank hemorrhage, except perhaps in the case of a penetrating Barrett's ulcer. However, some degrees of herniation of the gastric fundus can be associated with a sliding hernia and gradually progress until the entire stomach has migrated cephalad into the thorax. It is these types of hernia, as well as the infrequent pure paraesophageal hernia, that present the complications associated with a true internal visceral hernia, including obstruction, torsion, and hemorrhage. This is a problem that most of

us are familiar with and that unfortunately sometimes is not fully appreciated by our medical colleagues.

What is the incidence of anemia in the subgroup of patients with a paraesophageal or an intrathoracic stomach, excluding the axial type of hernia? I believe it is not a rare event.

Dr. Trastek and his colleagues have documented the source of bleeding in these patients, and it appears to result from mechanical irritation of the stomach as it rides up and down on the crus of the diaphragm. Another mechanism that we have observed is diffuse punctate erosions within the supradiaphragmatic gastric pouch as it intermittently undergoes torsion and venous congestion.

The authors outlined their operative strategy, with which I am in complete agreement, including the addition of an antireflux procedure to the operation. I believe that the pros and cons of an antireflux operation have been discussed at length in the literature. I think the pros far exceed the cons and an antireflux repair should be included within the repair.

Why did you choose to perform an esophagus-lengthening procedure in most of these patients? We have found it to be unnecessary, particularly with the use of the transthoracic approach that practically allows the surgeon to mobilize the esophagus from the hiatus to the arch, providing sufficient length to reposit the repair into the abdomen without any tension on it. I think it is possible that introduction of a staple line may add to the morbidity in the form of a leak.

Repair an internal hernia is proposed to correct the anemia. Do we have any information on the intermediate or long-term recurrences of these large hernias, which can sometimes be challenging to repair?

Dr. Trastek. We did not subdivide this already small group into patients with intrathoracic stomachs or pure paraesophageal hernias and those with routine sliding diaphragmatic hernias. I agree with you that it is likely that this subgroup has a higher incidence of anemia than those who have a sliding type of hernia, but these patients present in many different ways, and not all of them could be categorized into pure categories.

The lengthening procedure we use at our institution is called an uncut Collis-Nissen. It may add a little time to the procedure, but it also adds some anchoring abilities to the wrap. It is one we have used for about 15 years. I think your questions about adding something to the procedure that may precipitate other postoperative problems such as a leak is a fair one. We had two in this series and others in previous series. Because we feel this maneuver does add to the longevity of the operation, we continue to do it.

There were no known recurrences, but not all the follow-up data is not objective. There was not a repeated set of upper gastrointestinal studies or endoscopies for all patients to prove whether there were recurrences in this entire group or even in the subgroup of those with intrathoracic stomachs. According to the subjective follow-up data, and chest x-ray films, there were no known recurrences.

Dr. Antoon Lerut (*Leuven, Belgium*). I would like to comment on the technical difficulties alluded to by

Dr. Altorki, especially the sometimes very large size of the hiatus. Did you encounter any difficulties in narrowing the hiatus, and did you have to use prosthetic material, which is increasingly reported by surgeons who are doing this kind of surgery through the laparoscope?

Dr. Trastek. I do not recall that we had any hernia defects that were unable to be closed. I can tell you that none required prosthetic material. At the end of the procedure all of them had closure, allowing a fingertip to slip in around the esophagus with a dilator in place.