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ORIGINAL ARTICLE

Prevalence and Resolution of Anemia with Paraesophageal Hernia Repair

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Abstract
Background Paraesophageal hernias may produce a variety of clinical sequelae including anemia and esophagogastric ulcerations or erosions. We examined the prevalence of anemia in patients with paraesophageal hernias and frequency of anemia resolution with hernia repair.
Methods Patients undergoing paraesophageal hernia repairs from July 1996 to September 2010 were included. Data gathered included age, gender, type of hernia, presence of symptomatic anemia, presence of esophagogastric ulcer/erosion, type of repair, and anemia resolution.
Results One hundred eighty-three patients underwent paraesophageal hernia repair; of these, 68 (37 %) were anemic. Of these anemic patients, 39 (57 %) were symptomatic from their anemia or specifically referred for anemia, and 20 (29 %) had esophagogastric ulceration/erosion. Fifty-eight had documented follow-up. Overall, of these, 35 (60 %) had resolution of their anemia. Seventy percent of symptomatic patients had resolution of their anemia, compared to 48 % of asymptomatic patients (p=0.1). Of patients with esophagogastric ulceration/erosion, 85 % were symptomatic and 88 % had resolution of anemia, compared to 50 % of patients without ulceration/erosion (p=0.015).
Conclusions Anemia was a common finding in patients with paraesophageal hernia and most patients were symptomatic because of their anemia. Those patients with esophageal or gastric ulceration/erosion were very likely to have symptomatic anemia, and, interestingly, these patients were more likely to have their anemia resolve with paraesophageal hernia repair.

Keywords Paraesophageal hernia · Anemia · Esophageal ulcer · Gastric ulcer · Erosion

Introduction

Diaphragmatic hiatal hernias may produce a variety of clinical sequelae. The association between hiatal hernias and anemia has long been recognized.[1,2] Paraesophageal hernias (types II–IV), in which herniation of the gastric fundus through the esophageal hiatus occurs alongside the lower esophagus, account for approximately 5 % of all hiatal hernias.[3] Anemia has been shown to be three times more frequent in patients with the true paraesophageal variety as compared to the more common sliding type of hiatal hernia (type I).[4] The cause of this anemia has been attributed to linear ulcerations or erosions on the gastric mucosal folds at or near the level of the diaphragm.[4,5] In fact, the American Gastroenterology Association lists these so called Cameron's erosions in large hiatal hernias as a

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commonly overlooked lesion that may cause obscure bleeding.[6] The etiology of these lesions is thought to involve local mechanical trauma at the herniation site, though focal mucosal ischemia and gastric acid secretion has also been implicated.[7,8] The purpose of this study was to assess the prevalence of anemia in patients with true paraesophageal hernias as well as the frequency of resolution of anemia following surgical repair. Furthermore, we aimed to assess the frequency of ulceration or erosions in those found to be anemic, and whether the presence or absence of such lesions impacted resolution of anemia.

Methods

Patients with true paraesophageal hernias (types II–IV) undergoing either laparoscopic or open paraesophageal hernia repairs at Henry Ford Hospital from July 1996 to September 2010 were eligible for the study. Medical records of these patients were reviewed retrospectively. Epidemiologic and clinical data were collected, including age, gender, type of paraesophageal hernia, presence of symptomatic or asymptomatic anemia, specific surgical referral for anemia from a paraesophageal hernia, presence of esophageal or gastric ulcers or erosions by endoscopy, type of surgical repair, and resolution or persistence of anemia. Anemia was defined as a hemoglobin level <13.5 g/dl in males, <12.0 g/dl in females, or a symptomatic drop in hemoglobin. Nominal data were analyzed utilizing chi-square or Fisher's exact test with two-tailed p values. Nonparametric continuous data were analyzed by the Mann–Whitney U test. A p value of 0.05 was considered significant.

Results

A total of 183 patients underwent laparoscopic or open paraesophageal hernia repair between July 1996 and September 2010. Of these, 68 (37 %) were found to be anemic. Of these anemic patients, 39 (57 %) were symptomatic from their anemia or specifically referred for anemia, whereas the remaining 29 (43 %) were primarily worked up secondary to gastrointestinal symptoms, including dyspepsia, dysphagia, heartburn, regurgitation, abdominal or chest pain, nausea, and vomiting. Twenty (29 %) of the anemic patients had endoscopic evidence of esophageal or gastric ulcerations or erosions, compared to five (4 %) of the 115 non-anemic patients undergoing paraesophageal hernia repair ($p \leq 0.0001$). One of these five non-anemic patient's gastric ulcerations was attributed to chemical gastropathy secondary to nonsteroidal anti-inflammatory drug use. Seventeen (44 %) of the symptomatic anemic patients had evidence of esophagogastric ulcerations or erosions, while only three (10 %) of the patients with primarily GI symptoms had such lesions ($p=0.003$).

All patients underwent paraesophageal hernia repair, with 58 of the anemic patients having adequate documented follow-up. Overall mean length of follow-up was 52 months; 37.6 months for patients who had persistent anemia postoperatively and 61.5 months for patients whose anemia resolved postoperatively. The remaining 10 anemic patients had a mean length of follow-up of 7 days, insufficient to make determinations regarding postoperative resolution of anemia. Overall, of these 58 patients with sufficient follow-up, 35 (60 %) had resolution of their anemia. Seventy percent of patients with symptomatic anemia had resolution of their anemia postoperatively, compared to 48 % of asymptomatic patients ($p=0.1$) (Fig. 1). Of patients with esophagogastric ulceration or erosion, 85 % were symptomatic from their anemia, and 88 % had resolution of anemia postoperatively, compared to only 50 % anemia resolution in patients without ulceration or erosion ($p=0.015$) (Fig. 2).

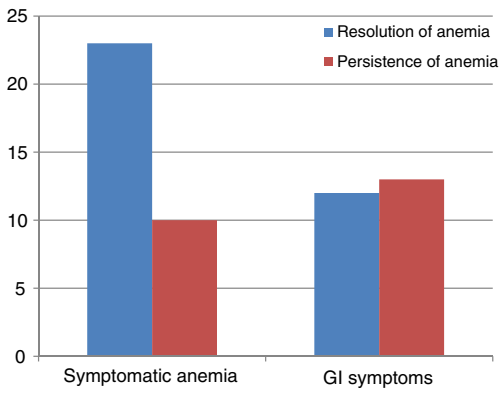

Fig. 1 Resolution of anemia following surgical repair of paraesophageal hernia in patients who presented with either symptomatic anemia or primarily with gastrointestinal symptoms

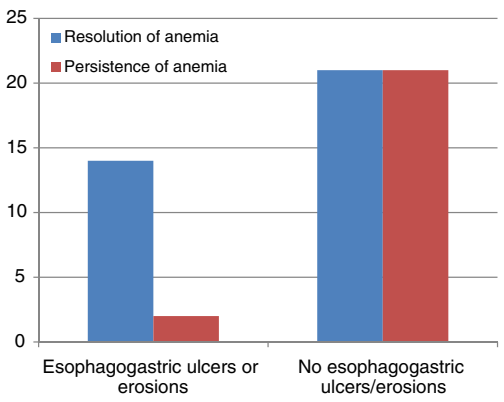

Fig. 2 Resolution of anemia following surgical repair of paraesophageal hernia in patients with and without evidence of esophagogastric ulcers or erosions

Discussion

Anemia is a relatively common finding among patients with hiatal hernias. Various studies have reported the prevalence of anemia among patients with paraesophageal hernias to be as high as 30–38 %.[3,4] The prevalence of anemia in our patient population was consistent with these previously reported rates at 37 %. It has been suggested and previously well demonstrated that the cause of anemia in such patients may be attributed to linear ulcerations or erosions in the gastric mucosa near the level of the diaphragm.[5] Our data supports this theory, in that patients with paraesophageal hernia and anemia were greater than seven times more likely to have evidence of these lesions than those patients who had paraesophageal hernias without evidence of bleeding and normal hemoglobin levels. Interestingly, however, these lesions have only been identified in an overall minority of anemic patients with paraesophageal hernias. Cameron's study in which these lesions were first described found them to be present in 42 % of patients who had a hernia and were anemic.[5] More recent studies have found Cameron's lesions in 28–33 % of such patients.[7–9] Our study similarly demonstrated these lesions in 29 % of anemic paraesophageal hernia patients (vs. 4 % of patients with normal Hgb). These lesions are occasionally missed on initial Esophagogastroduodenoscopy (EGD) and identified on repeat endoscopy.[10] This may be due to the technical difficulty in identifying what is often a very subtle lesion. Additionally, the erosions and ulcers may heal and recur over time, lending to the possibility that they could be missed on EGD during a healed stage.

Previous studies have demonstrated near complete resolution of anemia with diaphragmatic hernia repair. Trastek et al.[11] studied 349 patients with diaphragmatic hernia and anemia, of which 49 had surgical repair at the Mayo Clinic in a 9-year span. Though they did not differentiate between type I sliding hernias and true paraesophageal hernias, they did note resolution of anemia in approximately 92 % of those patients that underwent surgical repair. Hayden and Jamieson[9] studied 11 patients who underwent repair of paraesophageal hernia, and similarly found resolution of anemia in 91 % of these patients postoperatively. When we stratified our own study population into those who had evidence of Cameron's lesions on preoperative endoscopy, we demonstrated a similar rate of anemia resolution with 88 % of our patients achieving normal hemoglobin after surgical repair. We were only able to demonstrate such correction, however, in half of our patients without evidence of Cameron's lesions preoperatively (Fig. 2). Similarly, 70 % of patients who were symptomatic from their anemia demonstrated resolution of their anemia after surgery, compared with only 48 % of those patients who were asymptomatic from their anemia (Fig. 1). This difference can be attributed to the fact that symptomatic patients were much more likely to have evidence of Cameron's lesions than asymptomatic patients (44 % vs. 10 %). Patients with anemia and Cameron's lesions tend to be symptomatic from their anemia (85 %), and these patients are more likely to experience resolution of their anemia with surgical correction of their hernias.

The idea of elective surgical repair for asymptomatic paraesophageal hernias has been a controversial one. Dating back to the studies of Belsey[12] and Hill,[13] many surgeons have advocated an aggressive surgical approach to paraesophageal hernias irrespective of the presence of symptoms. The reasoning behind this is to provide prophylaxis against life-threatening complications such as obstruction, strangulation, and perforation. More recent data, however, has indicated that the risk of developing such complications in an asymptomatic patient may have previously been overstated. Stylopoulos et al.[14] determined that the available data does not support routine elective repair of asymptomatic paraesophageal hernias. They showed that prophylactic surgery to prevent future complications of a paraesophageal hernia would be more beneficial than watchful waiting in only one of five 65-year-old patients; thus, watchful waiting would be the preferred approach for patients with large but relatively asymptomatic paraesophageal hernias. In patients who are experiencing gastrointestinal or respiratory symptoms related to their hernias, however, the consensus remains that surgical repair in operable patients is the preferred choice of management.[15,16] Patients who are presumably anemic from their paraesophageal hernias present another treatment dilemma. While acid suppression with or without iron supplementation has been suggested as a treatment modality,[7,8] this requires indefinite therapy and does not address the aforementioned catastrophic complications that can occur. Furthermore, the majority of patients who are anemic from their paraesophageal hernias will also have accompanying gastroesophageal or respiratory symptoms, further supporting the decision to operatively correct their defects. In the hands of an experienced surgeon, both open and laparoscopic repair have proven to be safe and effective approaches.[3,16–18] Our data provides evidence to support the surgical repair of paraesophageal hernias in patients with symptomatic anemia especially those with esophageal or gastric ulcerations or erosions since the majority had resolution of their anemia.

There are several limitations to our study, including the retrospective nature of the data collection and lack of complete follow-up. Nearly all of the patients in this study were referred to us for gastrointestinal-related symptoms and not specifically for anemia. Therefore, once their gastrointestinal symptoms resolved, these

patients were sent back to their primary care physicians. Anemia specific follow-up on the patients whose anemia did not resolve was not elucidated for all patients. Another limitation is that postoperative endoscopy was not routinely employed to verify healing of the esophageal or gastric ulcerations or erosions. However, the vast majority (88 %) of those with ulcerations or erosions had resolution of their anemia postoperatively indicative of apparent healing.

Conclusion

Anemia is a common finding in patients with paraesophageal hernias and was present in over a third of our patients presenting for repair. Most of these patients were symptomatic because of their anemia. Patients who were anemic were more likely to have endoscopic evidence of esophageal or gastric ulcerations or erosions. Patients with esophagogastric ulcerations or erosions who underwent paraesophageal hernia repair were more likely to have resolution of their anemia postoperatively than anemic patients who did not have evidence of such lesions. Furthermore, anemic patients who have evidence of these so called Cameron's lesions are likely to be symptomatic from their anemia, and these patients specifically are very likely to experience resolution of their anemia with surgical repair. In summation, in the absence of other identifiable causes of blood loss, the presence of such lesions in patients with paraesophageal hernias should be considered the cause of the anemia, and these patients are likely to benefit from anemia resolution following surgical repair of their hernia.

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