

## Effect on iron deficiency anemia of laparoscopic repair of large paraesophageal hernias

J. D. Hayden, G. G. Jamieson

*Department of Surgery, University of Adelaide and Royal Adelaide Hospital, North Terrace, Adelaide, South Australia, Australia*

**SUMMARY.** Patients with iron deficiency anemia sometimes have a large paraesophageal hernia and no other explanation for their chronic blood loss. The management of these patients can be a dilemma, especially when the hernia is otherwise asymptomatic. We aimed to determine whether a laparoscopic repair of the hernia could cure the anemia. We reviewed a consecutive series of 11 cases of iron deficiency anemia associated with a large paraesophageal hernia, many without associated linear gastric erosions, managed by laparoscopic repair and fundoplication. There was one conversion in a patient with dense adhesions from previous upper abdominal surgery. Another patient required a laparoscopic reoperation for an early recurrence. Major morbidity occurred in three patients and there was no mortality. There was no recurrence of anemia after a median follow-up of more than 2 years. Iron deficiency anemia in association with a large paraesophageal hernia can be treated by laparoscopic repair with acceptable morbidity and minimal mortality. The complications of a large paraesophageal hernia are also prevented.

**KEY WORDS:** fundoplication, hiatus hernia, iron deficiency anemia, laparoscopic procedure, outcome.

### INTRODUCTION

Some patients with iron deficiency anemia have an associated large paraesophageal hernia and no other identifiable source of bleeding.<sup>1–5</sup> Cameron's lesions are areas of linear gastric erosion that have been identified at the neck of the sac of large paraesophageal hernias in less than half of these patients.<sup>5</sup> Several studies have shown that the incidence of paraesophageal hernia in those undergoing investigation for iron deficiency anemia may be in the region of 9%.<sup>3,6</sup> The frequency of anemia in patients with a hiatus hernia ranges from 8% to 42%.<sup>3</sup> In the absence of gastric erosions and symptoms related to the paraesophageal hernia, the management of these patients poses a dilemma. In addition, the question of whether a giant paraesophageal hernia can cause blood loss without erosions remains unanswered.

Treatment options for this condition range from ferrous sulfate therapy with or without acid suppression medication to surgical repair of the hernia,

usually by an open trans-thoracic or trans-abdominal approach.<sup>7,8</sup> Here we describe our experience with managing a series of cases treated by laparoscopic repair of the paraesophageal hernia accompanied by a Nissen fundoplication or one of its recent modifications.<sup>9–11</sup>

### METHODS

Between 1997 and 2004, 11 patients were identified at the Royal Adelaide Hospital who presented with iron deficiency anemia and were found during investigation to have a large paraesophageal hernia with at least a third of the stomach above the diaphragm. A total of 120 patients had a laparoscopic paraesophageal hernia repair in the unit during this time. All 11 patients had symptomatic anemia and three also had mild reflux symptoms. The median age was 55 years (range 36–78). There were six male and five female patients. Hematological parameters are described as follows with a median value and range in parenthesis: hemoglobin 7.7 g/dL (6.1–10.1); mean cell hemoglobin concentration 266 g/L (256–313); mean cell volume 71.1 femtomoles/L (66.1–79.2); platelet count  $327 \times 10^9/L$  (272–510); ferritin 13 µg/L (7–15);

Address correspondence to: Professor Glyn G. Jamieson, Department of Surgery, University of Adelaide and Royal Adelaide Hospital, North Terrace, Adelaide, Australia 5000. Email: chris.batesbrownsword@adelaide.edu.au

**Table 1** Summary of preoperative investigations of patients with iron deficiency anemia

Investigation	Result
Gastroscopy	Large paraesophageal hernia ( <i>n</i> = 9); linear gastric erosions ( <i>n</i> = 3); oesophagitis ( <i>n</i> = 2)
Barium meal	Large hiatus hernia ( <i>n</i> = 11); intrathoracic stomach ( <i>n</i> = 5)
Colonoscopy	Sigmoid colonic diverticulosis ( <i>n</i> = 1); normal ( <i>n</i> = 7)
Barium enema	Sigmoid colonic diverticulosis ( <i>n</i> = 2); normal ( <i>n</i> = 1)
Small bowel meal	Normal ( <i>n</i> = 2)
Oesophageal motility	Normal ( <i>n</i> = 7), low amplitude contraction ( <i>n</i> = 1)

**Table 2** Summary of operative details of paraesophageal hernia repairs

Feature	Observation
Operative findings	Intrathoracic stomach ( <i>n</i> = 5); large paraesophageal hiatus hernia ( <i>n</i> = 11); dense adhesions ( <i>n</i> = 1)
Crural repair	Posterior and anterior sutures ( <i>n</i> = 8); posterior sutures only ( <i>n</i> = 3)
Fundoplication type	Anterior partial ( <i>n</i> = 5); 360° Nissen ( <i>n</i> = 5); none ( <i>n</i> = 1)
Major complications	Conversion ( <i>n</i> = 1); early reoperation ( <i>n</i> = 1); pneumothorax ( <i>n</i> = 1); oesophageal stricture ( <i>n</i> = 1)
Hospital stay	Median 3 days (range 2–10)
Follow-up	Median 25 months (range 3–54)
Post-operation hemoglobin	Median 13.5 g/dL (range 12.4–16.3)

iron 1 µmol/L (1–4), transferrin 45 µmol/L (43–56); transferrin saturation 1% (1–5); vitamin B<sub>12</sub> 289 pmol/L (179–442); and folate 26.7 nmol/L (4.7–28.4).

Table 1 shows a summary of remaining preoperative investigations. Six patients were taking regular ferrous sulfate tablets, and three had received at least one blood transfusion to correct the anemia. Five patients had been placed on acid suppression medication (proton pump inhibitor in four cases and H<sub>2</sub> receptor antagonist in one case). None of the patients were taking non-steroidal anti-inflammatory drugs, antiplatelet agents or anticoagulant medication. All but one patient was American Society of Anesthesiology class II, the remaining case being class III.

In addition to a large paraesophageal hiatus hernia, linear gastric erosions typical of Cameron's lesions were evident in only three cases. Even though esophagitis was identified in two patients, the referring gastroenterologist felt that this was unlikely to have caused the anemia. Because the anemia was an ongoing cause of symptoms and no other explanation had been identified, a decision was made to proceed with laparoscopic paraesophageal hiatus hernia repair. This was performed in a standardized fashion with reduction of the hernia by mobilization of the sac from the mediastinum, narrowing of the oesophageal hiatus using interrupted nonabsorbable monofilament sutures together with an antireflux procedure, either a partial or total fundoplication.<sup>9–11</sup> A partial fundoplication was offered if a patient was concerned about the side-effects of an antireflux procedure.

## RESULTS

Ten out of 11 patients underwent a successful laparoscopic repair of their large paraesophageal hernia.

There was no operative mortality. A summary of the operative details and outcome is shown in Table 2. There was one conversion in a 47-year-old man who had undergone an open Nissen fundoplication 15 years earlier. The operation was converted due to failure to make progress in the presence of dense upper abdominal adhesions.

Surgical complications included an early recurrence of the hernia on the third postoperative day in a 67-year-old man who had undergone a repair and a 360° Nissen fundoplication. Emergency laparoscopy identified posterior hiatal stitch disruption as the cause. This was successfully repaired laparoscopically. Recovery following that was then uneventful.

There was a single persistent pneumothorax that resolved with intercostal drainage. This occurred in a 78-year-old woman who had undergone a repair and an anterior 90° wrap. Her preoperative manometry showed 70% propagation of primary peristaltic waves. She went on to develop dysphagia after several months due to an oesophageal stricture. After several dilations her symptoms improved. There were also two minor chest infections that responded well to medical treatment.

After a median of 25 months follow-up, all of the postoperative hemoglobin measurements were within the normal range (Table 2). One of the 11 patients was still taking iron supplements at follow-up. The majority of patients were asymptomatic apart from two with occasional dysphagia, one with flatulence and another with minor reflux symptoms.

## DISCUSSION

The association of iron deficiency anemia with paraesophageal hernia is recognized but seldom reported.<sup>1–5</sup>

Cameron's lesions are linear gastric erosions that probably arise due to mucosal trauma by the diaphragm at the neck of the sac.<sup>5</sup> They are believed to be the underlying cause of chronic blood loss anemia associated with a paraesophageal hernia but have been found in only 42% of cases.<sup>5</sup> The precise cause of blood loss anemia in the remaining 58% of cases remains uncertain.

This retrospective study documents the successful treatment by laparoscopic repair and fundoplication of iron deficiency anemia associated with a large paraesophageal hernia with and without linear gastric erosions. The anemia remained corrected after a median follow-up of more than 2 years without iron supplementation in the majority of cases. In those patients without overt erosions seen on endoscopy, there may be subclinical mucosal lesions, or because gastroscopy is difficult in such patients, erosions may have been missed. It remains to be determined in a prospective study of a larger number of cases, and with longer follow-up, whether laparoscopic surgery provides a safe and robust treatment for paraesophageal hernia-associated iron deficiency anemia, but this study suggests this will be the case.

Patients who are treated with iron supplementation alone develop recurrent anemia once the treatment stops because the linear erosions do not heal.<sup>7</sup> However, short courses of H<sub>2</sub> receptor antagonists result in healing of the Cameron's lesions in most cases.<sup>7</sup> A more recent study using proton pump inhibitors alone in 11 patients or in conjunction with surgery in a group of 10 cases have shown a similar successful outcome after 12 months in terms of curing the anemia.<sup>6</sup> None of the medical treatments reduce the risk of complications such as cardio-respiratory compromise, visceral strangulation or perforation that may occur in up to 50% of patients presenting with a paraesophageal hernia.<sup>12,13</sup>

The results of open surgical repair of the hiatus hernia have been reported in a series of 49 patients with a mortality rate of 2%, morbidity rate of 37% and resolution of the anemia in 92% of cases.<sup>8</sup> The results for laparoscopic repair of large hiatus hernias are impressive with 0.5% mortality, 28% morbidity and a conversion rate of 1.5%.<sup>14</sup> The major problem appears to be recurrence of the hernia, that may occur in around 40% of cases.<sup>15</sup> It is possible that the use of a mesh prosthesis to reinforce the repair may prevent recurrence in the future.<sup>16</sup>

Our laparoscopic surgical results showed no major morbidity in most cases and no mortality. Although this series of cases is small and conclusions are difficult to draw, the results for laparoscopic repair of large hiatus hernias from our unit have recently been reported.<sup>17</sup> We have had no operative mortal-

ity, morbidity of 14% and an overall recurrence rate of 30% from 100 patients.<sup>17</sup> The patients in our iron deficiency anemia group were younger on average by 10–20 years compared to previous series<sup>5–8</sup> and it is our view that a surgical approach is a better long-term treatment in suitable patients than indefinite acid suppression and dealing with recurrent anemia in this group.

In conclusion, if a patient presents with refractory iron deficiency anemia and has a large paraesophageal hernia, and no cause for gastrointestinal blood loss can be found, even in the absence of gastric erosions, a laparoscopic repair of the hernia is indicated and will probably cure the anemia.

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