

Cardiac Tamponade: A Rare and Insidious Surgical Complication of Hiatal Hernia Repair

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Hiatal hernia is defined as a protrusion of abdominal contents through the hiatal foramen into the thoracic cavity. Etiology is presumed to be a congenital malformation, trauma, or iatrogenic like prior surgical dissection of the hiatus during surgery for esophageal or gastric etiology. Age, sex, hormonal changes, body habitus (i.e., kyphosis, scoliosis), and increased body weight are key risk factors. Most hiatal hernias are asymptomatic and discovered incidentally. Surgical repair of hiatal hernia is indicated in symptomatic patients with dysphagia, weight loss, respiratory symptoms such as aspirations, and recurrent pneumonia events [1].

Complications arising from laparoscopic repair of hiatal hernia are generally minor and do not typically necessitate surgical intervention. Major complications include pneumothorax, splenic laceration, esophageal rupture, and pericardial injury. Other complications include recurrence of hernia, vagal nerve injury, gastroesophageal reflux disease,

and gastroparesis. The utilization of mesh in repair procedures introduces additional complications such as mesh migration and mesh infection. Previously reported recurrence rates following the repair of a hiatal hernia with mesh range from 10–30%. In this case communications, we presented a case involving the early recognition and treatment of postoperative cardiac tamponade.

PATIENT DESCRIPTION

A 77-year-old female arrived for elective recurrent hiatal hernia repair surgery. She had undergone laparoscopic hiatal hernia repair with Toupet fundoplication 8 years prior to the index admission with resolution of her symptomatic dysphagia for 6 months followed by repeated symptoms. She was followed by her gastroenterologist. Following worsening of her symptoms she was referred to our clinic. At her clinic visit, she reported severe dysphagia to solids and liquids and severe heartburn. The physical exam was unremarkable with normal appearing laparoscopic scars. Her preoperative body mass index (BMI) was 25.6 kg/m². Chest abdomen computed tomography (CT) scan demonstrated recurrent hiatal hernia containing the stomach fundus and gastroesophageal

junction. Esophagogastroduodenoscopy demonstrated hiatal hernia with gastroesophageal junction height of 27 cm. Preoperative anesthesia assessment was unremarkable with an ASA score of 2.

The patient underwent elective laparoscopic repair of the recurrent diaphragmatic hernia. The patient was placed supine, an infraumbilical skin incision was made, and an 11 mm trocar was placed to achieve pneumoperitoneum. Additional trocars were placed as follows: 12 mm in the left upper abdomen, 5 mm in the left lateral abdomen, and 5 mm in the right upper abdomen. A liver retractor was used to retract the left lobe of the liver. Gastroesophageal junction dissection was performed bluntly and sharply using ultrasonic shears (HARMONIC ACE™+7 Shears, Ethicon, USA). Following adhesiolysis at the area of the gastroesophageal junction, the herniated stomach was found with sutures from the prior surgery.

During the mediastinal dissection, the pleurae were violated bilaterally. The hiatal crura were approximated with non-absorbable braided 2-0 suture (Vloc®; Medtronic, USA), and non-absorbable mesh (Ultra-pro® Advanced, Ethicon, USA) was placed and fixed to the diaphragm with laparoscopic absorbable tucks

(SECURESTRAP® Absorbable Strap Fixation Device, Ethicon, USA). Nissen fundoplication was completed, and a Jackson Pratt drain was left transabdominally in the mediastinum. Operative time was 144 minutes. Throughout the surgery the patient remained hemodynamically stable. No anesthesia nor surgical complications were noted during surgery and the immediate postoperative period. During the first postoperative morning, the patient complained of weakness and dyspnea, and an urgent bedside evaluation was performed. A physical exam revealed a stuporous patient with tachypnea and hypotension. Lung auscultation revealed reduced bilateral breath sounds. Her heart rate was 85 beats per minute with normal heart rhythm, and no murmurs or pericardial rubs were auscultated. Bilateral distention of jugular veins was noted. Her abdominal exam was appropriate for early postoperative day. An electrocardiogram recorded sinus rhythm with no signs of acute ischemia and normal amplitudes. A chest X-ray showed no pneumothorax, atelectasis, or pleural effusion. Urgent bedside echocardiography noted large pericardial effusion with cardiac tamponade. The right ventricle appeared small and congested.

Urgent bedside pericardiocentesis via apical approach was performed with extraction of 60 ml of bloody fluid. Following pericardiocentesis, the patient immediately recovered; however, after an additional 30 minutes, her hemodynamic status deteriorated again. Repeated bedside echocardiography noted re-accumulation of the pericardial fluid. Due to a concern of active bleeding, the patient was transferred urgently to the operating room. In the operating room, an urgent sternotomy with

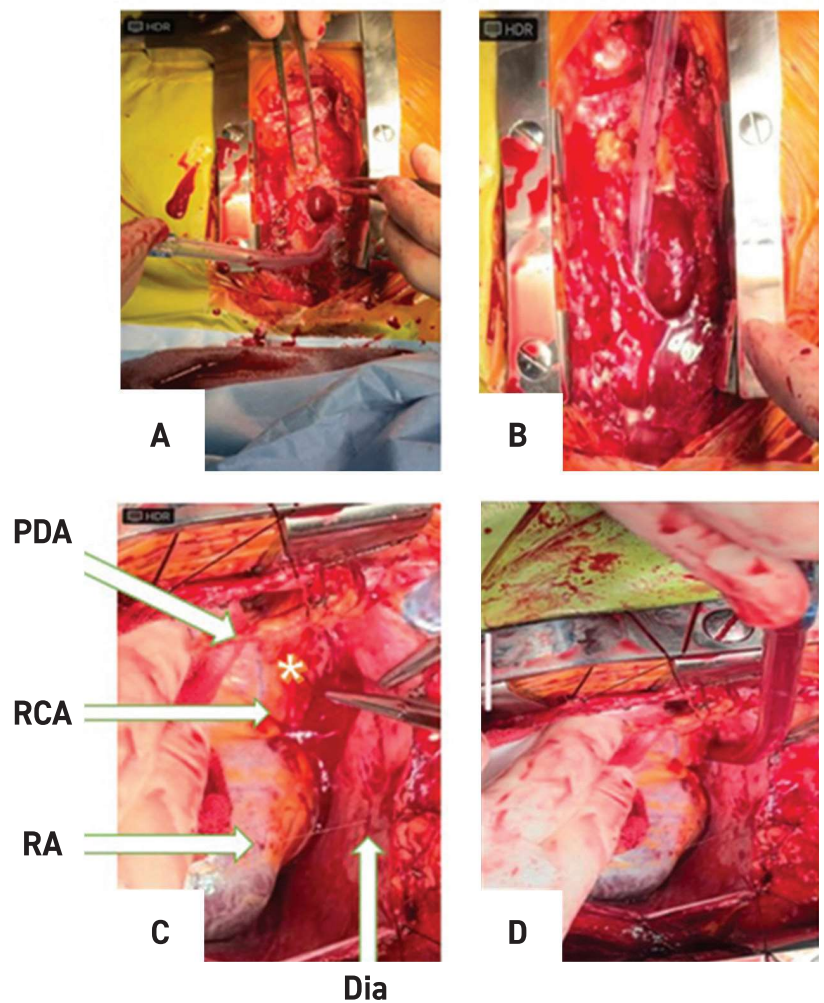
pericardiotomy was performed with evacuation of a large amount of blood and blood clots surrounding the heart, as shown in Figure 1. Exploration revealed a 5-mm diaphragmatic defect with active bleeding from the posterior descending artery (PDA). Primary repair of the injured vessel was performed, and the bleeding vessel was sutured with Prolene 4-0 suture with pledgets. A sealant was added for ad-

ditional hemostasis (Tachosil hemostatic patch®, Baxter, USA). The distal injury site of the PDA artery and the fact that this territory is supplied by other coronary vessels resulted in low probability of injury and reduced the risk of impairing heart perfusion. Surgical drains were placed, and the patient was transferred to cardiothoracic intensive care unit (ICU). The patient was extubated on postopera-

Figure 1. Intraoperative view of the pericardial drainage

[A] Pericardial sac opened, **[B]** pericardial sac filled with blood and clots, **[C]** Posterior view of the heart with bleeding point from PDA marked with asterisk. **[D]** heart after evacuation of hematoma

Dia = diaphragm, PDA = posterior descending artery with laceration, RA = right atrium, RCA = right coronary artery



tive day 1. The remaining postoperative course was unremarkable. She was discharged home on postoperative day 3. Since her discharge the patient has been asymptomatic. Echocardiography performed 2 months following the surgery post-sternotomy showed good left ventricular function and right ventricular function with no recurrent pericardial effusion and no wall motility abnormality. Her preoperative dysphagia resolved.

COMMENT

Postoperative mortality rates following elective laparoscopic repair of hiatal hernia are low and previously reported as 1.7% in 30 days. Postoperative major morbidity (Clavien Dindo > 3) rates reported in a retrospective study were 5% and included ICU admission due to pulmonary edema, reoperation for delayed gastroesophageal perforation, reoperation for incarcerated incisional hernia, reoperation for intestinal perforation, reoperation for mediastinal hematoma, reoperation for dysphagia, dysphagia treated with endoscopic dilation, and percutaneous drainage of liver abscess [2].

Following hiatal hernia repair, cardiac complications are scarce but present fatal outcomes if missed. A review published by Çalikoğlu and colleagues [3] reviewed 30 cases of cardiac tamponade following peri-hiatal surgery with 33.3% mortality rate, primarily caused by laparoscopic anti-reflux surgery and graft fixation. Cardiac complications described previously in literature include cardiac tamponade, arrhythmias and pericarditis [4]. Cardiac tamponade is the most serious adverse effect. The causes are usually direct injury to cardiac tissues or the pericardium with staples during fixation of the mesh or sutures during

the fundoplication. Sugumar et al. [5] reported two cases of pericardial effusion following surgery. One patient presented 5 weeks post-laparoscopic adjustable gastric banding combined with hiatal hernia repair surgery with non-hemorrhagic cardiac tamponade and required immediate drainage. The second patient presented 13 days following laparoscopic hiatal hernia repair with acute pericarditis treated medically with non-steroidal drugs and later 37 days post-surgery represented with acute cardiac tamponade requiring drainage. Fernandez and colleagues [4] described their experience with immediate complications after large hiatal hernia repair. Three cases of pericardial hematoma following laparoscopic repair of large hiatal hernias were presented with two requiring drainage in the early postoperative course. One patient underwent pericardiocentesis on postoperative day 2 and the second patient was taken to surgery on postoperative day 5 for pericardial drainage with evacuation of approximately 2500 ml of bloody fluid from a pericardial hematoma. The authors advocated for risk awareness of injury to the surrounding structures, specifically the heart and major diaphragmatic vessels during the anchoring stage of the mesh. They urged rapid and prompt early recognition of surgical complications.

Our case report describes early postoperative bleeding with direct cardiac injury to the PDA. Fortunately, the patient recovered well with normal cardiac function. Anchoring the mesh to the crura only and not to the diaphragm might reduce the likelihood of cardiac injury. In addition, if fixation is performed, meticulous stitching should be prioritized over fixators. This maneuver reduces the risk of cardiac injury. Postoperative anticoagulation may precipitate cardiac hemor-

rhage and should be used with caution due to potential cardiac injury. Treatment of cardiac tamponade depends on its size and on the hemodynamic condition of the patient. Treatment may be conservative, pericardiocentesis, or surgical. Persistent hypotension during or following peri-hiatal surgery should be considered an alarming sign of cardiac tamponade. Increased awareness among caregivers is essential for prevention and for improved survival rates in these unique cases.

CONCLUSIONS

Postoperative cardiac tamponade following hiatal hernia repair is a serious and life-threatening condition. Intraoperative awareness and high clinical suspicion for cardiac complications following hiatal hernia repair can improve patient outcomes.

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References

1. Kohn GP, Price RR, DeMeester SR, et al. Guidelines for the management of hiatal hernia. *Surg Endosc* 2013; 27 (12): 4409-28.
2. Sorial RK, Ali M, Kaneva P, et al. Modern era surgical outcomes of elective and emergency giant paraesophageal hernia repair at a high-volume referral center. *Surg Endosc* 2020; 34 (1): 284-9.
3. Çalikoğlu İ, Özgen G, Toydemir T, Yerdel MA. Iatrogenic cardiac tamponade as a mortal complication of peri-hiatal surgery. Analysis of 30 published cases. *Heliyon* 2019; 5 (4): e01537.
4. Fernandez MDC, Diaz M, López F, Martí-Obiol R, Ortega J. Cardiac complications after laparoscopic large hiatal hernia repair. Is it related with staple fixation of the mesh? Report of three cases. *Ann Med Surg (Lond)* 2015; 4 (4): 395-8.
5. Sugumar H, Kearney LG, Srivastava PM. Pericardial tamponade: a life-threatening complication of laparoscopic gastroesophageal surgery. *Heart Lung Circ* 2012; 21 (4): 237-9.