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CASE REPORT

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Chronic traumatic diaphragmatic hernia with pericardial rupture and associated gastroesophageal reflux

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Abstract Major thoracic and abdominal trauma damages the diaphragm 5% of the time. These injuries may be recognized when they occur but often are discovered months later during work up for related symptoms. Typically, the injury is to the left posterolateral aspect of the diaphragm. Rarely, rupture through the central diaphragmatic tendon into the pericardial space occurs and this results in different symptoms than the more common injury. We present the case of a patient who presented with chest pain, near syncopal episodes and refractory gastroesophageal reflux years after he was struck by a car and hospitalized. Radiographic imaging included a chest CT that demonstrated herniation of the transverse colon into the mediastinum. During exploration, a defect in the central diaphragm was found with free communication between the peritoneal and pericardial spaces. In this paper, we review our management of this unusual diaphragmatic hernia and the unique symptoms associated with it.

Keywords Diaphragm · Hernia · Trauma · Pericardial · GERD

Introduction

Traumatic diaphragmatic hernia (TDH) is a well-described entity. In the adult population, blunt thoracic and abdominal trauma results in acute diaphragmatic injury as often as 5% of the time [1]. The transverse interposition of the diaphragm between the abdomen and the chest, coupled with the pressure gradient that exists across it, makes it particularly susceptible to the sudden, dramatic increases in pressure that occur with blunt trauma [2]. Often these injuries are recognized acutely because they are associated with other, more compelling injuries that mandate exploration and result in recognition and repair of the TDH [3]. Less frequently, however, TDH occurs as an isolated injury that is not recognized until after the initial event. Instead it goes unnoticed for a period of time, until either symptoms are sufficient to lead to its discovery or it is found incidentally during work-up for an unrelated matter [2, 4]. The anatomic characteristics of the diaphragm and sub-diaphragmatic region are such that these hernias occur in areas of inherent weakness, typically in the left posterolateral region where the diaphragm inserts onto the chest wall, which is a common location for congenital diaphragmatic hernias as well [5, 6]. Very rarely, TDH occurs through the central diaphragmatic tendon with rupture into the pericardial cavity [6]. We present such a case and describe the unique symptoms associated with it.

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Case report

A 37-year-old man was referred to our institution for further evaluation and work-up after: (1) the development of intermittent, but increasingly severe, non-cardiac chest pain, dyspnea, frequent vasovagal symptoms and complaints consistent with gastroesophageal reflux disease (GERD), (2) a radiographic work-up, that included a chest X-ray (CXR) and computerized tomography (CT) of the chest, demonstrated herniation of abdominal viscera into the mediastinum and (3) documentation of symptomatic reflux poorly controlled with usual doses of oral acid suppressant medications.

Seven years prior to this referral, the patient had been involved in an accident in which he was struck by a car moving at moderate speed. He suffered blunt head, chest and abdominal trauma that resulted in a closed head injury, several minor orthopedic injuries and a prolonged hospitalization related largely to the initial

severity of his head injury. He did not require abdominal or thoracic surgery during the hospitalization and no thoracic or abdominal injuries were suspected. The patient subsequently made a full recovery from his initial injuries and was released from the hospital 3 months after the traumatic event. Soon thereafter, he began to experience episodes of pain in his substernal chest and difficulty in breathing that was best described as a ''strangling sensation''. He frequently became near syncopal with these episodes and often had to sit down until the symptoms subsided. He also developed frequent heartburn and regurgitation consistent with GERD. A trial of acid suppression therapy provided minimal relief and he continued to have breakthrough symptoms. A full cardiac work-up was negative but a CXR showed an air and fluid filled structure in the chest (Fig. 1a). A CT scan of the chest was then performed, which demonstrated a segment of transverse colon contained within the mediastinum (Fig. 1b). In addition, 24-h pH monitoring and esophageal manometry were performed to document GERD. These studies revealed normal esophageal motility, decreased lower esophageal sphincter pressure (LES) and abnormally high acid exposure in the distal esophagus.

Given the potential for life-threatening complications associated with this type of hernia, we recommended operative repair. In addition, the patient was quite frustrated with his refractory GERD and requested an anti-reflux operation at the time of the diaphragmatic hernia repair. The patient was explored through an upper midline incision. The entire transverse colon and a large portion of the greater omentum were herniated through the diaphragmatic defect. After reduction of the contents of the hernia, which was essentially free of chronic adhesions, a large defect of the central diaphragmatic tendon was noted. The hernia was approximately 10 cm from front to back and 16 cm across. The floor of the pericardium was absent, the heart was entirely visible through the defect and it was apparent that the contents of the herniation (colon and omentum) had been intrapericardial prior to reduction (Fig. 2a). The defect was then closed primarily with a running, non-absorbable suture and reinforced with a large section (13 × 16 cm) of dual-layered, bioimpregnated, expanded polytetrafluoroethylene (ePTFE) mesh (W.L. Gore, Flagstaff, AZ, USA) that was sutured to the peripheral edges of the overlying diaphragm in such a way that it generously overlapped the margins of the primary repair (Fig. 2b). Prior to full closure of the defect, a pericardial drain was placed to guard against postoperative tamponade. A Hill Repair, which is the anti-reflux operation of choice at our institution, was then performed.

Postoperatively, the patient did very well. The pericardial drain was removed 3 days after the operation without incident and the patient was discharged from the hospital on the fifth postoperative day. On subsequent return to the clinic, he reported full resolution of his preoperative symptoms, including the gastroesophageal reflux, and was not requiring acid suppression therapy. In addition, a CXR at that time was normal.

Discussion

TDH with pericardial rupture (PR) is a very rare injury [6, 7]. Fewer than 100 cases have been reported in the worlds' literature to date [7]. Like all traumatic diaphragmatic injuries, they can go unrecognized at the

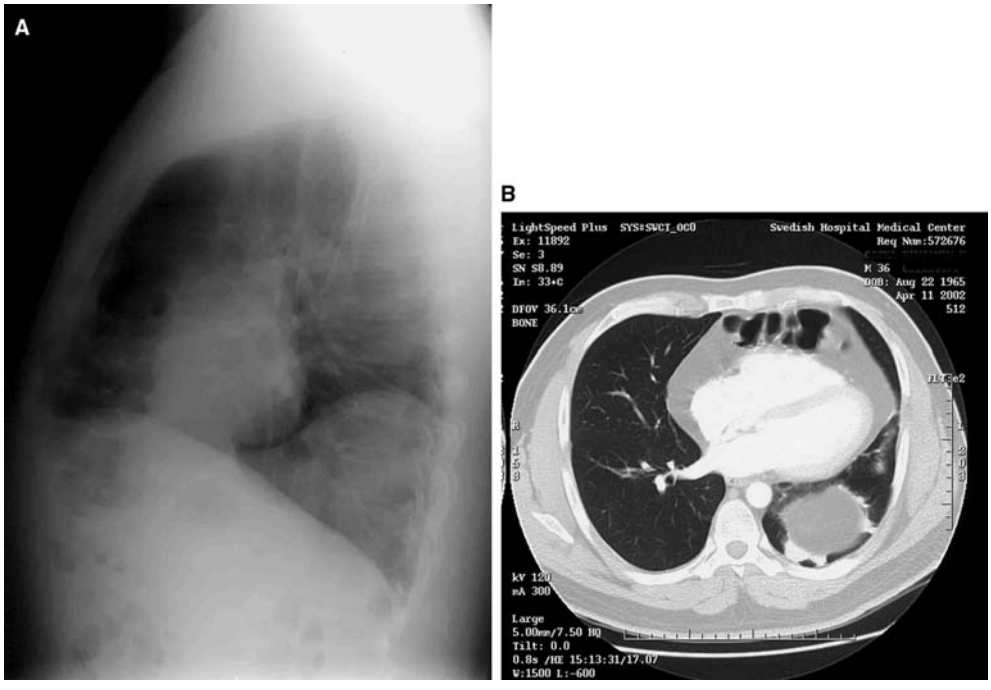

Fig. 1 *Panel A* Lateral plain film view of the chest demonstrating an air filled segment of colon above the level of the diaphragm. It was unclear on the posteroanterior view (not shown) if the herniation was into the right chest or the mediastinum. *Panel B* Computerized tomography of the chest clarifying that the segment of colon seen on plain film is herniated into the mediastinum and not the right chest

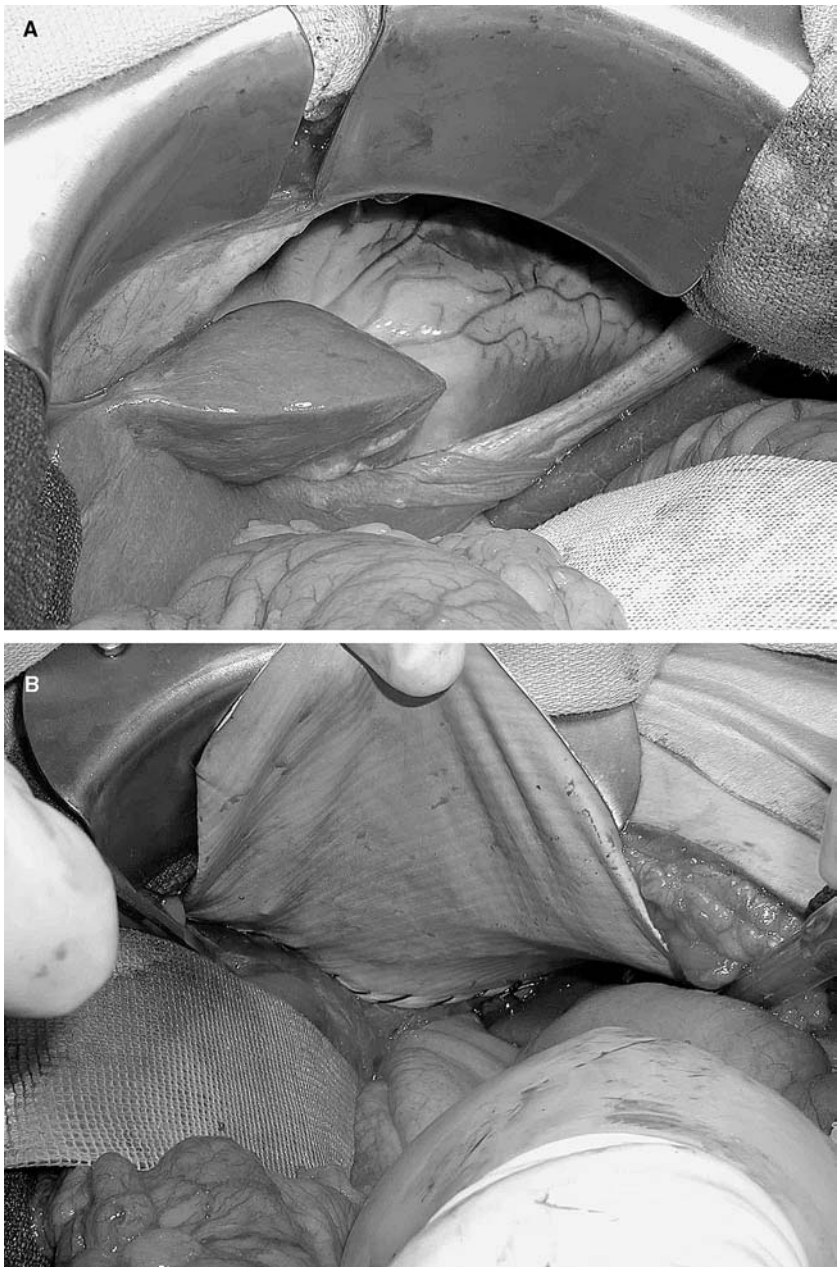

Fig. 2 *Panel A* Intraoperative photograph of the diaphragmatic defect after reduction of the omentum and the transverse colon from the pericardial cavity. The heart is clearly visible through the defect. *Panel B* Intraoperative photograph demonstrating the placement and positioning of the dual, bioimpregnated ePTFE mesh (W.L. Gore, Flagstaff, AZ, USA) to reinforce the repair after primary closure of the defect

time of the initial injury and present in a delayed manner, months or even years after the antecedent event [4, 6, 7, 8]. Unlike most other TDHs, however, that establish a peritoneal communication into the relatively large right or left pleural cavity, diaphragmatic injury with rupture into the pericardium creates this same communication into an extremely volume-limited space. The result is symptoms that are different than those seen in the more common defects. For instance, in our literature review, there were 26 cases in which preoperative cardiac symptoms were directly attributable to impeded diastolic filling from adjacent herniated viscera [7]. Transverse colon and omentum, as was the case in our patient, tend to fill the pericardium and surround the heart, abolishing the small potential space that allows normal cardiac function, creating a tamponade-like effect. In fact, there are at least six instances of reported acute pericardial tamponade related to TDH with PR and subsequent viscera herniation [7]. In 2001, Reina and colleagues authored a comprehensive review of the worlds' reported cases of TDH with PR (Table 1).

A large range of gastrointestinal symptoms have been reported in association with these diaphragmatic defects but few reports have associated traumatic absence of the central diaphragmatic tendon with intrapericardial herniation and symptomatic, refractory GERD. Problematic reflux is certainly common enough that the patient described in our report may well

Table 1 Summarization of 2001 literature review regarding traumatic diaphragmatic hernia with pericardial rupture (Reina et al. 2001)

Number of reported cases (1910–2000)	Dominant symptoms (%)	Associated injuries (%)	Herniated organs (%)
81 Cases	Breathing difficulties (40) Cardiac symptoms (32) Cardiac tamponade (7) GI/abdominal complaints (43) Asymptomatic (11)	Rib fractures (44) Pelvic fractures (25) Lower extremity fractures (20) Head injury (15) Splenic injury (11)	Transverse colon (48) Stomach (41) Greater omentum (38) Liver and/or spleen (14) Small bowel (11)

have developed GERD independently of his traumatic event but there are several aspects of this case that lead us to suspect a causal relationship between the presence of the TDH and the development of refractory GERD. Firstly, the onset of his reflux disease coincided with the onset of his other symptoms and was not present prior to his accident. Secondly, the reflux was poorly controlled with maximal dose acid suppression therapy but was fully corrected with repair of the hernia and GE junction—this is somewhat unusual. Typically, poor symptom control with appropriate acid suppression therapy is a negative indicator for outcome in anti-reflux surgery [9]. Thirdly, the central tendon of the diaphragm normally abuts the distal esophagus and GE junction. The absence of normal anatomy in this case, along with the presence of adjacent herniated viscera, likely led to a distortion of the GE junction that allowed intermittent reflux to occur. In fact, an interesting operative approach to this case might have been to simply repair the herniation with expectant resolution of the GERD postoperatively. We felt, however, that rather than risk the need for another operative procedure at a later date (if the reflux did not resolve), the patient was best served with a direct reconstruction to definitively treat the problem.

Regardless of the associated symptoms, the presence of TDH with PR, indeed any TDH, is an indication for repair [4, 6]. As already discussed, patients with a common communication between the pericardial space and the peritoneal cavity often have significant cardiac symptoms that lead to the diagnosis and can occasionally even be life-threatening [6, 10]. The specifics of repair are generally straightforward. The chronicity of the defect, coupled with inherent tension present at the central, domed aspect of the diaphragm, usually leads to a falling away of the surrounding diaphragmatic tissue such that the defect can often be large and difficult to close primarily without creating excessive tension at the repair. Mesh re-enforcement is commonly used to either buttress an overlying primary repair or to close a defect that is too large to bring together without an interposing prosthesis [6, 7]. The type of mesh chosen for repair is somewhat case dependent. Because we were able to close the defect primarily, a dual mesh was chosen to allow for mesh incorporation into overlying tissue to strengthen the repair without concern for adhesion to the heart. In those situations where the mesh will be used to bridge, rather than reinforce a primary closure of, the defect, a more inert material, such as simple Gore-Tex (W.L. Gore, Flagstaff, AZ), would help to avoid potential cardiac adhesions. The chronic TDH is usually approached through the chest rather than the abdomen due to concerns about adhesion formation between herniated abdominal viscera and adjacent lung, pleura and other structures [1]. Authors have advocated this approach for chronic TDH with PR as well, echoing some of these same concerns [6, 7]. After our experience in this case and, indeed, after reviewing the reported experiences of others, we recommend a transabdominal approach for virtually all TDH with PR [11]. It would seem that, unlike in the pleural cavity, adhesions seldom form between the inner lining of the pericardium or the heart and the herniated abdominal viscera. Furthermore, the margins of the defect are more easily delineated from an abdominal approach and the repair would seem more straightforward as well. Laparoscopic repair of certain diaphragmatic defects has also been well described [12]. Regardless of the operative approach chosen, there may be some difficulty preoperatively in distinguishing this type of hernia from some of the other more common diaphragmatic defects. In our case, for instance, the preoperative radiographic impression was a Foramen of Morgagni defect with resultant herniation into the anterior mediastinum rather than the pericardial sac.

In summary, TDH with PR is uncommonly seen. A significant number of these injuries will not be recognized at the time of the initial event but will instead be found a variable amount of time later. Prior to the advent of CT, these hernias were documented radiographically through the use of plain films and contrast studies, but are now best demonstrated through high-definition CT [10]. The potential for life-threatening complications mandates prompt repair in most patients.

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