Penetrating Trauma to the Diaphragm

Natural History and Ultrasonographic Characteristics of Untreated Injury in a Pig Model

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Hypothesis: Recent use of minimally invasive techniques to evaluate the chest and abdomen in patients with penetrating thoracoabdominal trauma has led to the discovery of many occult diaphragm injuries. Surgical repair of these injuries is relatively straightforward. However, diagnosis can be difficult, and the natural history of these injuries is controversial. By developing a penetrating diaphragm injury model, the ultrasonographic characteristics and natural history of this injury can be better understood.

Setting: Surgical laboratory of a tertiary care hospital.

Subjects: Seven pigs (Sus scrofa), weighing between 55 and 80 kg, received a 3-cm right-sided (n=3) or left-sided (n=4) diaphragm injury via thoracoscopy.

Interventions: Thoracoabdominal x-ray and ultrasonographic examinations were performed preoperatively; at 2, 4, 8, and 12 weeks postoperatively; and when symptoms related to the diaphragm injury occurred. At 12 weeks, or at the time of earlier death, a postmortem thoracoabdominal examination was performed.

Main Outcome Measures: x-Ray and ultrasonographic characteristics, and evidence of wound healing, in a penetrating diaphragm injury model.

Results: Perioperative recovery occurred in all pigs. No pigs had radiographic evidence of immediate postoperative herniation. Pigs in the right-sided injury group died early (≤10 days postoperatively). At the time of death, x-ray and ultrasonographic examination revealed hollow viscus herniation into the thorax (n=2). Pigs in the left-sided injury group remained asymptomatic, without radiographic evidence of herniation, although subtle ultrasonographic signs of diaphragm injury were seen at the 2-week (n=2), 4-week (n=2), and 8-week (n=3) intervals. Postmortem examination of the right-sided injury group revealed the liver afforded no protection against herniation. Right-sided defects (n=3) did not change size or character despite small-bowel herniation. Conversely, the left hemidiaphragm was well protected by the relatively fixed liver, spleen, and large stomach. The 4 left-sided defects (100%) spontaneously healed.

Conclusions: We developed a penetrating diaphragm injury model with high and low risk of herniation. Ultrasonography may prove to be an important diagnostic adjunct in evaluating diaphragm injuries with and without herniation. Moreover, since the “protected” diaphragm injuries in our model healed spontaneously, a role may exist for the nonoperative treatment of diaphragm injuries in clinical practice. This pig model may prove useful in further defining future management and repair techniques for such injuries.


Because of its large size and variable position, the diaphragm is often injured in penetrating thoracoabdominal trauma. Surgical repair of these injuries is relatively straightforward. However, the method for diagnosis and the natural history of diaphragm injury remain controversial.

Suspected diaphragmatic injury has been evaluated with radiologic techniques, diagnostic peritoneal lavage, and induced pneumoperitoneum with notoriously low sensitivity and specificity. With the advent of minimally invasive videoendoscopic techniques, however, many occult diaphragmatic injuries are being found. Recently, it was reported that 42% of penetrating left-sided thoracoabdominal trauma resulted in diaphragmatic injury when aggressively evaluated by laparoscopy.1 This suggests surgeons are likely to face an increased number of clinically occult diaphragm injuries from penetrating trauma to the lower chest.

Moreover, the true natural history of these defects is relatively unknown. It has been stated that there has never been a case of a spontaneously healed diaphragm injury.2 To support this contention, how-

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MATERIALS AND METHODS

ANIMAL USE

Seven pigs (Sus scrofa) (age, 7-9 months; weight, 55-80 kg) were divided into 2 groups: a left-sided diaphragmatic injury (LS) group (n=4) and a right-sided diaphragmatic injury (RS) group (n=3). The protocol was reviewed, approved, and monitored by the David Grant Medical Center Institutional Animal Care and Use Committee.

ANESTHESIA AND INITIAL PREPARATION

All animals were placed under general anesthesia by first administering xylazine hydrochloride, 2 mg/kg, intramuscularly and atropine sulfate, 0.04 mg/kg, intramuscularly as a preanesthetic agent. A combination of tiletamine hydrochloride and zolazepam hydrochloride (Telazol), 10 mg/kg, was then administered intramuscularly to facilitate endotracheal intubation. Intubation was performed with a cuffed 5- to 6-mm endotracheal tube, and maintenance anesthesia was given with gaseous isoflurane at approximately 2% to 3% in combination with 100% oxygen. The animals were maintained on positive pressure ventilatory control with a tidal volume of 10 mL/kg and a respiration rate of 15/min. Antibiotic ophthalmic ointment was placed in each eye to help preclude drying of the cornea. Placed in a decubitus position, the animals were prepped and draped in aseptic fashion with 70% isopropyl alcohol followed by 10% povidone-iodine (Betadine) solution. Continuous monitoring of heart rate and mean airway pressure was performed.

OPERATIVE PROCEDURE

Animals in the LS group were approached from the left hemithorax, and animals in the RS group were approached from the right hemithorax. A small skin incision was made into the thorax through the lateral working port. Positive pressure ventilation to 35 mm Hg was applied for 2 to 3 minutes to aid in lung expansion. The 10-mm port and thoracoscope were removed when the lung was fully expanded. The port site was closed primarily using skin staples. The red rubber catheter was placed on 20-mm Hg suction and slowly removed. On complete removal, the final port site was closed primarily using skin staples.

RADIOLOGIC EVALUATION

All animals underwent thoracoabdominal ultrasonography, anteroposterior chest x-ray, and lateral chest x-ray preoperatively; immediately postoperatively; and at 2-, 4-, 8-, and 12-week postoperative intervals. All chest x-rays were performed with 95 kilovolt (peak) and −1 density settings. Ultrasonographic examinations were performed using an ultrasound imaging system (128 XP; Acuson Corporation, Mountain View, Calif) with a vector 328 and 3.5-MHz frequency transducer. For the 2-, 4-, 8-, and 12-week postoperative examinations, the animals were given tiletamine hydrochloride-zolazepam hydrochloride, 5 mg/kg, intramuscularly for anesthesia. Perioperative radiologic examinations were performed during induction anesthesia and the operative recovery period.

POSTMORTEM EXAMINATION

At 12 weeks postoperatively, or at the time of earlier death, each animal received tiletamine hydrochloride-zolazepam hydrochloride, 10 mg/kg, intramuscularly followed by an intracardiac injection of pentobarbital (“B-Euthanasia”), 300 mg/mL, at 1 mL/5 kg. Postmortem examination was performed using a ventral midline thoracoabdominal incision. Diaphragm injury sites were harvested if no hollow viscus herniation was present. Wound size and characteristics were recorded.

RESULTS

The results of preoperative ultrasonography were normal in all animals (N=7), and the diaphragm was seen as an echogenic line (Figure 1). Perioperative recovery occurred in all animals. No animals had ultrasonographic or cause of “protection” from herniation afforded by the large fixed liver.

Nevertheless, the true natural history and radiographic characteristics of penetrating diaphragm injury remain ambiguous. This investigation describes the ultrasonographic features and natural history of untreated penetrating diaphragm injury in a pig model.

MODEL DEVELOPMENT AND RADIOGRAPHY

The results of preoperative ultrasonography were normal in all animals (N=7), and the diaphragm was seen as an echogenic line (Figure 1). Perioperative recovery occurred in all animals. No animals had ultrasonographic or
chest x-ray evidence of abdominal visceral herniation into the thorax on immediate postoperative examination.

All animals in the RS group (n=3) died early (≤10 days postoperatively) from respiratory distress (n=2) or symptomatic bowel obstruction (n=1). At the time of death, chest x-ray revealed hollow viscus herniation into the thorax of the animals available for study (n=2). Ultrasonographic examination results at this time revealed disruption of the echogenic diaphragmatic line (DEDL) with bowel located in the thorax (n=1) and absence of the echogenic diaphragmatic line, replaced with heterogeneous echogenic material (n=1) (Figure 2 and Figure 3).

All animals in the LS group (n=4) completed the study with uneventful postoperative courses. Chest x-ray revealed no evidence of diaphragm disruption or visceral organ herniation at the 2-, 4-, 8-, or 12-week examinations. Serial ultrasonographic examinations revealed DEDL at the 2- (n=2) and 4-week (n=1) intervals, abnormal contour of the echogenic diaphragmatic line at the 4- (n=1) and 8-week (n=1) intervals, and attenuation of the echogenic diaphragmatic line at the 8-week (n=2) interval (Figures 4, 5, and 6). No evidence of visceral herniation was seen on serial ultrasonographic examinations in the LS group.

POSTMORTEM EXAMINATION

Postmortem examination of the RS group (n=3) occurred immediately at the time of death (postoperative days 1, 6, and 10). Inspection of the right side of the abdominal cavity revealed the liver afforded no protection...
against herniation due to the lack of lateral ligamentous attachments on the right side. Herniated small bowel compressing the right lung was seen in all RS animals (Figure 7). The herniated bowel was strangulated above and below the diaphragm in 2 of the 3 RS animals. Postmortem wound length (2.7, 2.9, and 3.0 cm) or wound characteristics did not change in any of these animals despite small-bowel herniation.

Postmortem examination of the LS group occurred at 12 weeks in all animals (n=4). Review of the left side of the abdominal cavity revealed a relatively fixed liver, spleen, and large stomach protecting the left hemidiaphragm from contact with bowel (Figure 8). No visceral herniation was seen in any of the LS animals. All LS animals had complete healing of the diaphragmatic injuries (Figure 9). The results are summarized in the Table.

**COMMENT**

The diaphragm is a thin musculotendinous structure with a peripheral muscular component and a central avascular tendinous component. Because of the diaphragm's large size and variable position, it is often injured in penetrating thoracoabdominal trauma. Until recently, the incidence of diaphragm injury from penetrating trauma was estimated to be 15% for stab wounds and 46% for gunshot wounds, but these estimates are based on unstable patients with injuries diagnosed at laparotomy. Recently, the use of videoendoscopic surveillance in stable patients with penetrating thoracoabdominal trauma has identified many occult diaphragmatic injuries. The incidence of diaphragm injuries in series using videoendoscopic surveillance is much higher, ranging from 21.7% to 32% for stab wounds and up to 59% for gunshot wounds. Therefore, the diagnosis of diaphragmatic injury is becoming more commonplace, and surgeons are more likely to face occult diaphragm injuries in patients with penetrating thoracoabdominal trauma.

However, diagnosis of penetrating diaphragm injury without videoendoscopy is notoriously difficult. In the trauma setting, chest x-ray results can be normal 50% to 80% of the time, and the remainder usually demonstrate only hemotorax or pneumotorax. False-negative rates of up to 40% have been reported using computed tomography. Induced pneumoperitoneum, magnetic resonance imaging, and contrast instillation into the abdominal and thoracic cavities in an attempt to find abnormal communication have all been tried, but are usually not appropriate in the trauma setting. Diagnostic peritoneal lavage can be particularly insensitive when the avascular central tendon is involved in the injury.
Ultrasonographic diagnosis has also been described in the trauma setting. The diaphragm is normally seen as a curvilinear echogenicity separating the abdomen from the thorax. Diagnosis of injury is suggested by a DEDL, bowel peristalsis in the chest indicating visceral herniation, or free-flapping wound edges in a fluid collection. However, since most injuries diagnosed immediately are repaired, ultrasonographic characteristics of long-term untreated injury have never been reported. In our model, ultrasonography identified diaphragm injury in the trauma and long-term settings. Two of the animals with early postoperative herniation showed DEDL, absence of the echogenic diaphragmatic line with heterogeneous echogenicity, and loops of bowel in the chest cavity. These findings are consistent with clinical ultrasonographic descriptions of penetrating diaphragmatic injury. Moreover, in 4 of the animals whose injuries subsequently healed, ultrasonography identified the area of injury as a DEDL in the early examinations (2 weeks postoperatively) and as abnormal contour or attenuation of the echogenic line in the later examinations (4-8 weeks postoperatively). A DEDL most likely represents separation in the diaphragm surface, while attenuation and abnormal contour of the echogenic line most likely represent an intact, healed diaphragmatic wound. The heterogeneous echogenicity presumably represents mesenteric or omental fat and decompressed bowel. Future evaluations using our model may help define these results further.

It is thought that diaphragmatic defects heal poorly, and abdominal viscera tend to migrate into the thorax. There are several theories in the literature that attempt to explain the pathophysiological features of this natural history. Marchand3 describes a pressure gradient between the abdomen and thorax of 7 to 20 cm H2O at rest, and up to 100 cm H2O with maximal inspiration. This pressure gradient may force viscera into the thorax and preclude wound edge apposition. The thin nature, constant movement, and radial forces of the diaphragm have also been implicated in nonhealing of injuries, but no scientific data exist to support these theories. Many researchers cite cases of late visceral herniation into the thorax after a remote history of penetrating or blunt trauma to the thoracoabdominal region as evidence for the nonhealing nature of the diaphragm. Morbidity of up to 30% and mortality of up to 10% for this complication have been reported, which has driven most surgeons to repair all documented diaphragm injuries. Recently, however, a trend toward conservative management of right-sided penetrating thoracoabdominal trauma is evolving. Renz and Feliciano recently reported on nonsurgical management of stable patients with gunshot wounds to the right thoracoabdominal region. Five of their patients had documented diaphragmatic injury by computed tomography, with no reports of long-term complications. In addition, surgeons have argued that minor right-sided penetrating diaphragmatic injuries are of little consequence because right-sided injury is rarely associated with herniation and is “protected” from herniation by the large fixed liver. The only way to delineate the true natural history of penetrating diaphragm injury is to observe documented, untreated injury, a practice not commonly done. However, in our diaphragm injury model, untreated injury was followed up for 3 months. Healing of the injury occurred in animals with left-sided injury. Moreover, the left hemidiaphragm in a pig is well protected by fixed organs (spleen, liver, and stomach), unlike the left hemidiaphragm in humans. Right-sided injury, on the other hand, resulted in small-bowel herniation, and the liver afforded little protection of the right hemidiaphragm surface due to the lack of “bare area” liver attachments. In our animal model, the natural history of small penetrating injury to a protected diaphragm is spontaneous healing with a low risk of herniation, whereas injury to the “unprotected” diaphragm results in early herniation with resultant cardiorespiratory compromise, bowel strangulation, and nonhealing of defects.

Moreover, the natural history of diaphragm injury in our model, with a high and low risk of complications, lends itself well to future studies involving optimal repair tech-
niques for penetrating diaphragm injury. Traditionally, thoracoabdominal penetrating injury in an unstable patient is managed with laparotomy. When the diaphragm is injured in such trauma, associated injury has been reported to occur in 15% of those with stab wounds and in 46% of those with gunshot wounds. A laparotomy approach allows the surgeon to inspect and repair these injuries. However, with the recent trend toward videoendoscopic surveillance of penetrating thoracoabdominal trauma in stable patients, the optimal approach to repair of diaphragm injuries is in question. If a diaphragm injury is found during laparotomy, and the patient has no other indication for laparotomy, it logically follows that a laparoscopic repair would be the least morbid option, if technically feasible. Moreover, some believe thoracoscopic surveillance in stable patients with penetrating thoracoabdominal trauma offers better exposure and less chance of induced pneumothorax from the laparoscopic pneumoperitoneum crossing the diaphragm barrier through the suspected diaphragmatic defect. Again, thoracoscopic repair offers less chance of morbidity than a standard thoracotomy, but has not been aggressively evaluated. The first report of successful videoendoscopic repair of diaphragmatic injury was in 1994. Thoracoscopy and laparoscopy were used to repair diaphragmatic injuries in 2 hemodynamically stable patients. The immediate results were promising, but long-term follow-up was not available. Few subsequent reports have evaluated the long-term success of endoscopic repair. The low-risk group (LS group) in our model provides a useful tool to evaluate different repair approaches for diaphragmatic injury without visceral herniation. Moreover, the high-risk group (RS group) in our model tends to herniate at an early stage. Laparoscopic and thoracoscopic techniques for reducing herniated viscera and repairing diaphragm injury could be evaluated and compared with the standard laparotomy or thoracotomy reduction and repair.

CONCLUSIONS

We have developed a penetrating diaphragm injury model with a high and low risk of herniation. This model can be used to better delineate ultrasonographic findings of diaphragmatic injuries in immediate high-risk injuries (right sided in our pig model) or low-risk injuries (left sided in our pig model). Ultrasonography may prove to be a useful adjunct for observing patients with suspected or low-risk penetrating diaphragmatic injuries. It is believed that diaphragm injuries heal poorly, and can result in late complications with increased morbidity and mortality. However, the natural history of protected penetrating diaphragm injury in our model is spontaneous healing, while those injuries that are not protected by solid organ viscerum result in visscus herniation at an early stage. These new data on the natural history of diaphragm injury in an animal model may be helpful in defining future management of occult diaphragmatic injury in patients who experience trauma and who are also at a high or low risk of herniation. Last, the optimal approach to repair occult, asymptomatic diaphragmatic injuries found during videendoscopic surveillance has not been established.

Our model is a useful tool for future studies to evaluate less morbid repair techniques of small, uncomplicated diaphragmatic injuries.

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