Case Report

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Surgical Repair of Diaphragmatic Hernia-Related Small Intestinal Strangulation in a Neonatal Foal

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Abstract This case report describes the clinical presentation and successful surgical repair of a diaphragmatic hernia-related small intestinal strangulation in a neonatal foal. A nine-day-old foal presented with colic signs and respiratory distress. History taking showed that the dam of the foal experienced difficulty during delivery, and the owner assisted in delivery by pulling on the foal. Radiography and ultrasonography confirmed the diaphragmatic rent and the presence of a small intestine within the thoracic cavity. Surgical intervention was required to repair the diaphragmatic defect and address the intestinal strangulation. The diaphragm was reconstructed, and the nonviable incarcerated portion of the small intestine was resected and anastomosed using an end-to-end technique. This unusual case report provides insights into the surgical repair and outcomes of an acquired diaphragmatic hernia in a neonatal foal.

Key words anastomosis, colic, diaphragmatic hernia, foal, intestinal incarceration.

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Introduction

Diaphragmatic hernia (DH) is an uncommon cause of colic in neonatal foals with a poor prognosis (5,6,14,17). DH can be congenital or acquired, and identifying the cause of the defect is challenging (5,14). An acquired DH is typically found at the central part of the diaphragm, a probable weak point of the musculotendinous junction, and most cases are traumatic in origin (15). On the other hand, a congenital DH is commonly located in the left dorsal tendinous part of the diaphragm and is caused by the failure of the embryonic elements of the diaphragm to unite (15). The clinical signs of DH include abdominal pain, respiratory distress or dyspnea, and exercise intolerance (15-17). The severity of the symptoms varies depending on the portion and amount of herniated internal organs and whether there are strangulations on the affected organs (4,17).

This case report describes the clinical presentation and successful surgical repair of a DH-related small intestinal strangulation in a neonatal foal. Although several case reports on clinical cases of DH in horses have been published (2,3,12,13,16), detailed information is needed to manage surgical repair of DH-related small intestinal incarceration in a newborn foal. This report specifies the presentation, diagnosis, surgical repair, and postoperative care, as well as the limitations of the case, expanding the current knowledge of DH in horses.

Case Report

A 62 kg nine-day-old Thoroughbred filly was presented to the Jeju Stud Farm Equine Referral Clinic of the Korea Racing Authority for abdominal pain. The foal showed intermittent colic signs, including rolling and loss of appetite in the last two days. The foal was treated with a nonsteroidal anti-inflammatory drug (NSAID, flunixin meglumine, 1.1 mg/kg, IV), antibiotics (ceftiofur, 5 mg/kg, IV), and fluid (lactated Ringer's solution) by a referring veterinarian and the owner. On historical information, the dam of the patient had difficulty in the foaling process, and the owner pulled on the foal to assist delivery. The pregnancy was full-term. A physical examination on presentation revealed the foal to be weak and depressed and showed tachycardia (120 beats/min), tachypnea (60 breaths/min), irregular and labored breathing, and remarked respiratory noise on thoracic auscultation (Supplementary Video 1). The laboratory work revealed hyperglycemia (131 mg/dL; reference range 65-110 mg/dL) and an elevation of the blood lactate concentration (2.5 mmol/L; reference range $<$ 1.5 mmol/L), total bilirubin (3.5 mg/dL; reference range, 0.5-2.3 mg/dL), and creatine kinase (734 U/L; reference range, 120-470 U/L). Blood-packed cell volume (25.4%; reference range, 32-53%), total protein (4.8 g/dL; reference range, 5.7-8.0 g/dL), and globulin (1.8 g/dL; reference range, 2.7-5.0 g/dL) levels were decreased. An abdominal and thoracic ultrasonographic examination revealed distended loops of amotile small intestine in bilateral thoracic cavities (Fig. 1). An assessment of the radiography revealed the presence of gas-filled abdominal viscera within the thoracic cavity (Fig. 2A, B). Based on the history, clinical examination, and diagnostic imaging, DH-associated respiratory distress and colic were diagnosed, and immediate surgery was recommended to repair the DH.

Before surgery, a 16-gauge $\times$ 7.5 cm IV catheter (Milacath,

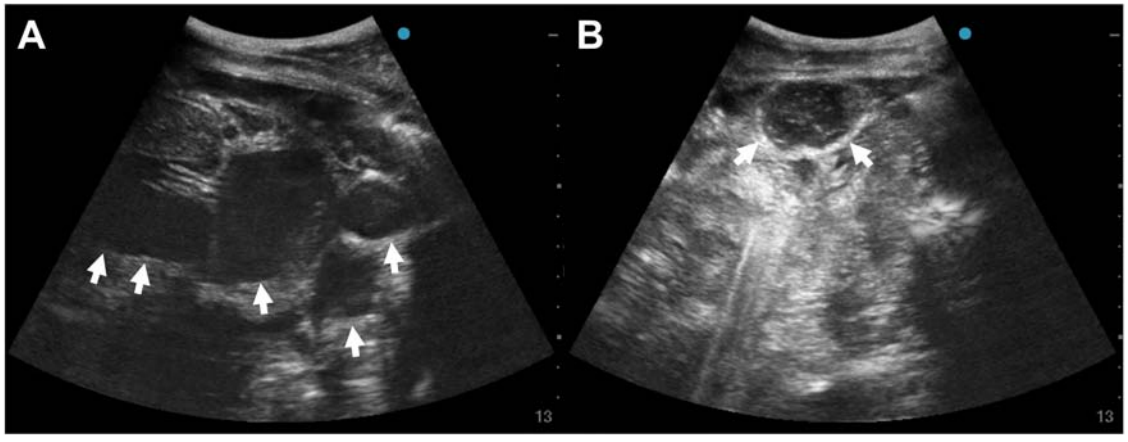

Fig. 1. Ultrasonographic images of the thoracic cavity in this study. (A) Depicts several distended and amotile small intestinal loops (white arrows) without lung visibility in the expected anatomical location. (B) Highlights the presence of the small intestine within the thoracic cavity (white arrows). In particular, the absence of typical pleural structures and A-lines is evident. Instead, a hyperechoic heterogeneous area surrounds the small intestine, replacing the usual pulmonary parenchyma. Scan depth is indicated by the scale on the right in centimeters.

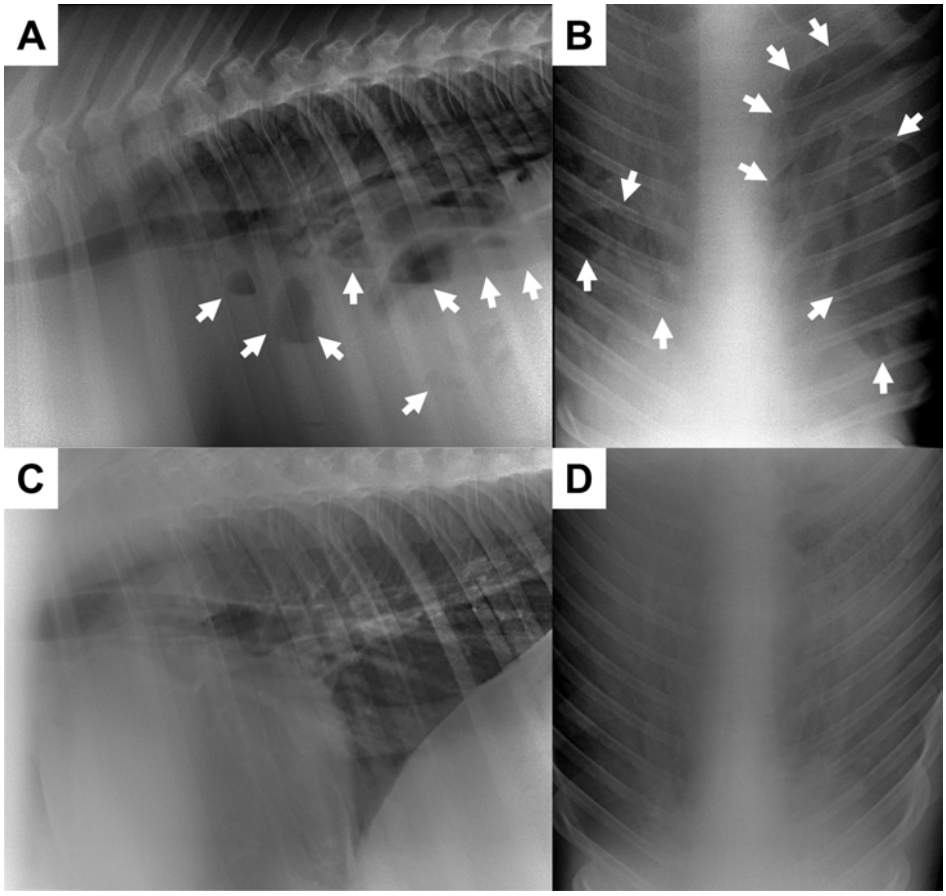

Fig. 2. Radiographic images of the thoracic cavity in this study. (A, B) Preoperative thoracic cavity images: the presence of pleural effusion and herniating loops of the small intestine into the left and right thoracic cavities is evident (white arrows). This is accompanied by the loss of the cardiac silhouette and diaphragmatic line. (C, D) Postoperative thoracic cavity images: the pleural effusion and herniating loops resolved after surgery, leading to the clear visibility of the cardiac silhouette and diaphragmatic line.

Mila International, USA) was placed in the left jugular vein. NSAID (flunixin meglumine, 1.1 mg/kg, IV) and prophylactic antibiotics (ceftiofur, 2.2 mg/kg, IV) were administered as preoperative medication. The general anesthesia was induced with diazepam (0.05 mg/kg, IV) and ketamine (2.2 mg/kg, IV) and maintained with isoflurane in 100% oxygen. The foal was positioned in dorsal recumbency, and the surgery table was tilted into a reverse Trendelenburg position (head up 30°) to facilitate the reduction of herniated viscera and improve the visibility of the diaphragm. A 15 cm craniocaudal midline incision was made using a No. 20 scalpel blade. After opening the peritoneum, the ascending and descending colon were exteriorized to improve accessibility to the diaphragm. The small intestines were fluid-distended, and sections of the jejunum were incarcerated in the thorax. The margin of the diaphragmatic defect and the involved small intestines were lubricated carefully with carboxymethylcellulose and manipulated to reduce the weight of the herniated viscera. The incarcerated sections were returned to the abdomen in gentle traction, and the diaphragmatic defect and viability of the strangulated intestinal lesion were evaluated. The opening to the hernia was 10 cm long, located in the muscular portion of the central and ventral aspect of the diaphragm, and the margins of the defect were smooth-edged (Fig. 3A). Omental adhesions were observed at the margin of the defect, and the heart and lungs were visible through the hernia opening (Fig. 3A). Approximately 2 m of entrapped jejunum were affected by strangulation with severe congestion and edema, and the color of the lesion was dark blue without a pulse and motility, indicating that the lesion was nonviable (Fig. 3D, E). Consequently, a decision was made to repair the diaphragmatic defect surgically and resect the nonviable strangulated jejunum using an end-to-end anastomosis procedure.

The diaphragmatic defect was closed directly using a #1 polyglactin 910 suture (Vicryl plus, Ethicon, USA) in a simple continuous pattern (Fig. 3B, C). The suture pattern started at the dorsal extent of the tear in the diaphragm, and the air was evacuated from the thorax after closure. The omentum adhered to the edge of the diaphragmatic defect was resected to prevent postoperative intestinal adhesion. For the resection and anastomosis of the small intestine, the exteriorized jejunum was draped carefully with sterile large laparotomy sponges, and the mesenteric vessels supplying nonviable

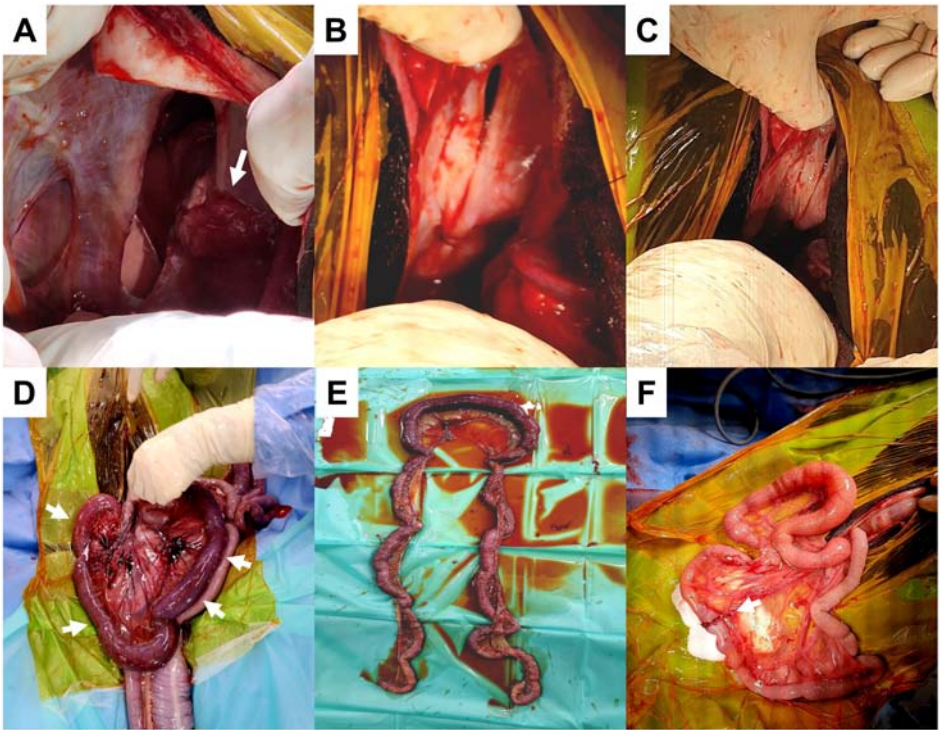

Fig. 3. Diagnostic and surgical procedures illustrated in the study. (A) The hernia opening situated in the central and ventral aspect of the diaphragm is depicted. Omental adhesions are observed at the defect's margin (indicated by a white arrow), providing visibility of the heart and lungs through the hernia opening. (B) The repair of the diaphragm using a simple continuous pattern. (C) Depicts the diaphragm after the repair was completed. (D) A section of strangulated jejunum with severe congestion and edema is shown. Lesions exhibit a dark blue coloration devoid of pulse and motility (white arrows). The nonviable nature of the lesions is determined. (E) Depicts the resection of the nonviable portion of the small intestine during the surgical procedure. (F) The small intestine after the successful completion of anastomosis. The anastomosis site is indicated by a white arrow.

loops were double ligated using a #2-0 poliglecaprone suture (Monocryl, Ethicon, USA). An incision was made in the middle of the nonviable lesion, and all ingesta and fluid in the small intestine were removed through the incision opening. The small intestinal transection was made on the viable area with the angled cuttings of the bowel ends. The intestinal ends were apposed to each other and closed using an appositional, simple interrupted pattern with #2-0 poliglecaprone suture (Monocryl, Ethicon, USA) (Fig. 3F). After the resection and anastomosis, the peritoneum and exteriorized bowel were irrigated with 10 liters of heparinized saline. The small intestine was coated with carboxymethylcellulose. The abdominal wall and skin were routinely closed after ensuring correct repositioning of the bowels. The foal recovered uneventfully from anesthesia.

Postoperatively, the horse received broad-spectrum antimicrobials (ceftiofur, 2.2 mg/kg, IV, once daily), NSAID (flunixin meglumine, 1.1 mg/kg, IV, once daily), antiacids (famotidine, 0.3 mg/kg, IV, twice daily), and fluids (lactated Ringer's solution with 5% dextrose) for six days. The abdominal bandage was maintained for 14 days postoperatively. In postoperative ultrasonography, fluid that appeared to have entered during surgery was observed in the thoracic cavity, but it was absorbed gradually, and respiratory distress was not present on postoperative day 2 (PO 2). The radiographic assessment revealed no abnormalities after surgery (Fig. 2C, D). The vital signs (heart rate, 68-80/min; respiratory rate, 24-30/min; body temperature, 38.1-38.3°C) and blood lactate level (1.1 mmol/L; reference range < 1.5 mmol/L) were improved compared to the values before surgery. As a complication, diarrhea occurred after surgery, but there were no abnormalities in appetite, vitality, hematology, and serum chemistry analysis. Anti-diarrheal agents (smectite and probiotics) were administered during the hospitalization period, and the diarrhea gradually improved. The foal was discharged on PO 15, and its vitality, appetite, and afebrile status were maintained until discharge. The foal remained healthy without clinical signs related to DH and gastrointestinal diseases throughout the five-month follow-up.

Discussion

This paper reported a case of small intestinal strangulation related to DH in a neonatal foal. Generally, the overall prognosis for horses with DH is poor, with only 16% of horses surviving until discharge and 27% of horses surviving after surgery, and the rate of survival is even lower in foals than in adult horses (6,14). The factors that appear to contribute to the survival rate include the size and location of the defect, as well as the amount of incarcerated small intestine (6). Anesthesia and gastrointestinal surgery techniques also affect the survival rates because intestinal incarceration in cases of

DH often necessitates a surgical resection and anastomosis under general anesthesia (3,6,11,14,16).

DH is a very unusual cause of colic in foals because it is found in less than 1% of neonatal foals exhibiting colic signs (11). On the other hand, most horses in DH presented with colic signs of varying severity and duration (6,14). Owing to the rarity of DH in horses, the differential diagnosis of DH as a cause of colic symptoms can be overlooked in the field. The foal in this case presented with colic symptoms and was brought to the clinic two days after the onset. A diagnosis for DH was possible through thoracic auscultation, revealing abnormal heart and lung sounds, as well as through ultrasonographic and radiographic examinations. Given the importance of early diagnosis and treatment to improve the prognosis of DH, the possibility of DH should be considered in horses showing colic signs, particularly when accompanied by respiratory symptoms, during the diagnostic process.

The signs of DH in horses range from nonspecific (e.g., inappetence, tachycardia, and tachypnea) to more demonstrative (e.g., respiratory distress with colic) and subtle (e.g., lying in lateral recumbency) (11). Careful observation and various methods are used to formulate a differential diagnosis list to distinguish DH from the various clinical signs (14,15,17). The non-invasive diagnostic methods include physical examinations, such as thoracic auscultation, rectal palpation, abdominal/thoracic ultrasonography, and radiography (14,15). Despite the utility of non-invasive methods, in cases where a diagnosis is challenging, more invasive approaches, such as laparoscopy, thoracoscopy, and exploratory celiotomy, are used for diagnostic purposes (14,15,17). On the other hand, these approaches are accompanied by an increased risk of complications. In this case, thoracic auscultation, along with abdominal/thoracic ultrasonography and radiography of the diaphragm, revealed evidence of DH. Other previous studies also reported that radiographic findings (eight out of 10 horses) and ultrasonographic findings (nine out of 10 horses) are consistent with DH (15). In this regard, diagnostic imaging tools are useful when DH is suspected in clinical cases.

Congenital and acquired DH have been reported, and identifying the cause of the defect is challenging (6,14,15,17). The acquired DH is associated primarily with traumatic events or caused by increased intra-abdominal pressure, such as foaling, trailer accidents, and injury during racing (7,11,14). The traumatic defect usually occurs in the central part of the diaphragm, a probable weak point at the musculotendinous junction of the diaphragm, and there is visible or histological evidence of trauma or lacerations with inflammation (4,17). On the other hand, congenital DH shows no evidence of scarring or adhesions at the edge of the defect (17,19). The colic associated with congenital DH can be asymptomatic for years (17). In this case, the defect was in the central part of the diaphragm, and there were omental adhesions at the smooth-edged margin of the defect. Considering the history of a problematic foaling process and the surgical findings, the defect was acquired.

Anesthesia of horses with DH is challenging because of the respiratory insufficiency, such as hypoxemia and hypercapnia (7). Therefore, it might be helpful to give 100% oxygen at 10 to 15 L/min before and after surgery (7,10). In the present case, the newborn foal maintained adequate ventilation and oxygenation with mechanical ventilation (maximum peak inspiratory pressure of 20 to 30 cmH_2O, 10 breaths per minute) during surgery. The foal received 100% oxygen at 10 L/min via a nasal tube in the recovery room right after the surgery.

Surgery for DH is challenging because of the limited access to the defect when the rent is located dorsally in adult horses (14,15,17). The ventral midline celiotomy is generally the most commonly applied method for correcting the defect, but alternative methods such as laparoscopy and thoracoscopy can be considered if access to the rent is limited (9,15). The alternative methods provide improved visualization and accessibility to the dorsal aspect of the diaphragm while the horse is standing (9,15). In this case, the diaphragmatic hernia could be repaired with direct conventional sutures since the rent was located ventrally and accessible without difficulties. On the other hand, applying a synthetic mesh over the defect is a potential option if direct suturing is not feasible because of the size or location of the rent and fragile surrounding tissues (8,14).

Postoperative complications of DH are commonly related to intestinal adhesions, hemoperitoneum due to diaphragmatic dehiscence, postoperative ileus, pneumothorax, and septic pleuritis (1,6,7,18). In the present case, serial postoperative radiography and ultrasonography were performed to confirm the integrity of the repair and assess the small intestinal motility. In addition, careful manipulation of the bowls, thorough postoperative abdominal lavage, and the application of carboxymethylcellulose for lubrication and coating were used to prevent adhesion. Postoperative antimicrobial agents were administered to reduce the risk of pleuritis and pneumonia, and the foal did not show any signs of complications.

Conclusions

This case report detailed the surgical repair of DH-related small intestinal strangulation in a neonatal foal. DH should be considered a potential cause of colic in neonatal foals, particularly when accompanied by respiratory distress. Radiographic and ultrasonographic examinations are valuable for

facilitating a DH diagnosis. This report outlines the presentation, diagnosis, surgical repair, and postoperative support, as well as the limitations of the case, contributing to the broader understanding of DH in horses.

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Conflicts of Interest

The authors have no conflicting interests.

Author Contributions

JY and AK contributed to the conceptualization, data curation, formal analysis, and manuscript writing. JY, JC, SK, and AK performed surgery, anesthesia, treatment, and aftercare. JY and AK supervised aftercare and revised the manuscript. All authors have read and agreed with the published version of the manuscript.

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