CASE REPORT

Tensioning diaphragmoplasty for treating bilateral phrenic nerve paralysis in a dog

S. Matsumoto*, K. Hosoya*†,1, S. Kim*† and M. Okumura*‡

*Small Animal Surgery Services, Veterinary Teaching Hospital, Graduate School of Veterinary Medicine, Hokkaido University, Sapporo, Japan
†Laboratory of Advanced Veterinary Medicine, Department of Veterinary Clinical Sciences, Graduate School of Veterinary Medicine, Hokkaido University, Sapporo, Japan
‡Laboratory of Veterinary Surgery, Department of Veterinary Clinical Sciences, Graduate School of Veterinary Medicine, Hokkaido University, Sapporo, Japan

[1]Corresponding author email: whitelittlesnow@aol.com

A spayed female Miniature dachshund presented with a large mediastinal mass. During thymectomy, the left phrenic nerve, which was encircled by the neoplastic tissue, was resected *en bloc* with the tumour and the right phrenic nerve was damaged by thermal coagulation from electrosurgery. Postoperatively, the dog exhibited significant respiratory distress and paradoxical respiratory movement. Bilateral diaphragmatic paralysis was confirmed on inspiratory radiographs, and lack of structural abnormalities on thoracic computed tomography was noted. The dog collapsed 5 days postoperatively from respiratory fatigue. Thus, salvage surgery and tensioning diaphragmoplasty were performed. Briefly, The diaphragm was partially resected, primarily closed and reinforced with a polypropylene mesh sutured on the abdominal surface. Thereafter, the dog's condition improved significantly. The dog did not present with respiratory issues until she died of urothelial carcinoma on day 375. In conclusion, this surgical technique can be considered to re-establish the function of the diaphragm.

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INTRODUCTION

Phrenic nerve paralysis is a common complication of thoracic surgery in dogs and humans (Mamada et al., 2020; Ricoy et al., 2019). The phrenic nerves comprise united nerve fibres that arise from the ventral branches of the 5th, 6th and 7th cervical spinal nerves in dogs (Kitchell, 2013). Within the thoracic cavity, the phrenic nerves run along each side of the mediastinum, ending on the respective crus of the diaphragm and innervating and controlling diaphragmatic muscle contraction for respiratory motion (Kitchell, 2013). They control active diaphragmatic contraction that is required for normal inspiration. Diaphragmatic paralysis can be caused by degeneration of these nerves, injuries to these nerves and diaphragmatic muscle myopathy (Ricoy et al., 2019).

Iatrogenic injury to one of the phrenic nerves in humans results in postoperative unilateral diaphragmatic paralysis, which is associated with decreased arterial blood oxygen saturation and prolongation of hospitalisation period (Fujimura et al., 1995; Ross Russell et al., 2008). In dogs, unilateral diaphragmatic paralysis is reported to cause no clinically symptomatic respiratory problem except in individuals with preexisting compensated respiratory dysfunction (Mamada et al., 2020). On the other hand, there is a paucity of information regarding how dogs adapt to bilateral diaphragmatic paralysis (Raillard et al., 2017; Santarelli et al., 2017), and very little information regarding a surgical management option for this condition has been reported.

Tensioning diaphragm plication is a procedure to treat congenital or acquired diaphragmatic eventration in humans that tacks the diaphragm to itself (plication) to reduce its area without removing a part of it. In people, this procedure has been performed via thoracotomy (Celik et al., 2010) as well as via laparoscopy or thoracoscopy (Freeman et al., 2009; Groth et al., 2010). This technique has been reported in one dog (Santarelli et al., 2017).

The following databases (PubMed and Google Scholar) were searched with the following keywords; phrenic nerve, paralysis, bilateral, surgery, canine, dog on 11/05/2024. The following textbook was consulted; Veterinary Surgery: Small Animal, 2nd edition (Johnston et al., 2017). Only one clinical case report was found that described a bilateral diaphragmatic paralysis in a dog with muscle degeneration, in which diaphragmatic plication was effective (Santarelli et al., 2017).

We describe a clinical case of iatrogenic diaphragmatic paralysis caused by bilateral phrenic nerve injury associated with thymectomy in a dog, which was treated with a newly developed surgical procedure, tensioning diaphragmoplasty.

CASE HISTORY

An 11-year-old spayed female Miniature dachshund with acute-onset respiratory distress and suspected acute intrathoracic haemorrhage caused by a large mediastinal mass lesion was urgently referred to X. Upon presentation (day 1), the dog was quiet, alert and responsive. Her respiration was mildly tachypnoeic (respiratory rate = 54 cycles/minute). However, her respiratory pattern was normal. Physical examination did not reveal a remarkable abnormality except moderate oedema and bruising over the ventral cervical area and cranial thorax. Complete blood count and serum chemistry profile showed moderate anaemia (haematocrit: 23.3%, reference range: 37.3%–61.7%), neutrophilia (neutrophil count: 19,630/µL, reference range: 2950–11,640/µL), monocytosis (monocyte count: 3020/µL, reference range: 160–1120/µL), thrombocytopaenia (platelet count: 74,000/µL, reference range: 148,000–484,000/µL) and hyperglycaemia (blood glucose level: 137 mg/dL, reference range: 75–128 mg/dL).

Thoracic radiography showed a large cranial mediastinal mass, displacing the heart caudally and the tracheal bifurcation dorsolaterally, and low-volume pleural effusion. Cytology of the mass revealed clustered epithelial cells, indicating neoplasia of thymic epithelial origin. There were no signs of megaesophagus or systemic sign of myasthenia gravis. Medical management with intravenous fluid therapy and prednisolone (1 mg/kg administered subcutaneously every 24 h) was initiated to potentially decrease the mass size even though lymphocytes were not observed upon cytology.

On day 4, the dog was anaesthetised with intravenous injection of 1% propofol (to effect, PropoFlo®28, DS Pharma Animal Health, Osaka, Japan) before undergoing a computed tomography (CT) scan (Aquilion PRIME™, Toshiba Medical Systems, Tochigi, Japan). Anaesthesia was maintained with inhalation of isoflurane (ds Isoflurane®, DS Pharma Animal Health) and oxygen. Following the precontrast scan, iopamidol (Iopamidol 150; Bayer, Osaka, Japan) was administered intravenously at a dose of 600 mgI/kg for the contrast study. Results showed a soft tissue large mass with a maximum diameter of 90 mm in the cranial mediastinum, displacing the cranial vena cava to the right and the heart to the left and caudal. Branches from the left internal thoracic artery supplied the mass. Vascular invasion was not noted. Large non-contrast enhancing areas were observed in the central part of the mass, indicating necrosis or intra-tumoral haemorrhage (Fig. 1A, B). The pleural effusion observed on the previous radiographs had resolved.

On day 7, curative-intent surgery was performed under general anaesthesia. Fentanyl (Fentanyl injection, Daiichi-Sankyo, Tokyo, Japan) at 5 µg/kg were injected intravenously for analgesia and sedation. General anaesthesia was induced with 1% propofol to effect, and the anaesthesia was maintained with isoflurane (Isoflu, Zoetis Japan, Tokyo, Japan). Fentanyl (5 to 10 µg/kg/minute) and ketamine (600 µg/kg/minute; Ketamin injection 5%, FUJITA, Tokyo, Japan) were administered intravenously by constant rate infusion (CRI) for analgesia. The thorax of the dog was approached via complete median sternotomy. The mass was cautiously dissected from the surrounding tissues with sufficient care taken to identify the two phrenic nerves on each side of the thorax. Grossly, the right phrenic nerve ran on the surface of the mass (Fig. 2A). Meanwhile, the left phrenic nerve was encircled by the neoplastic tissue. The right phrenic nerve was bluntly

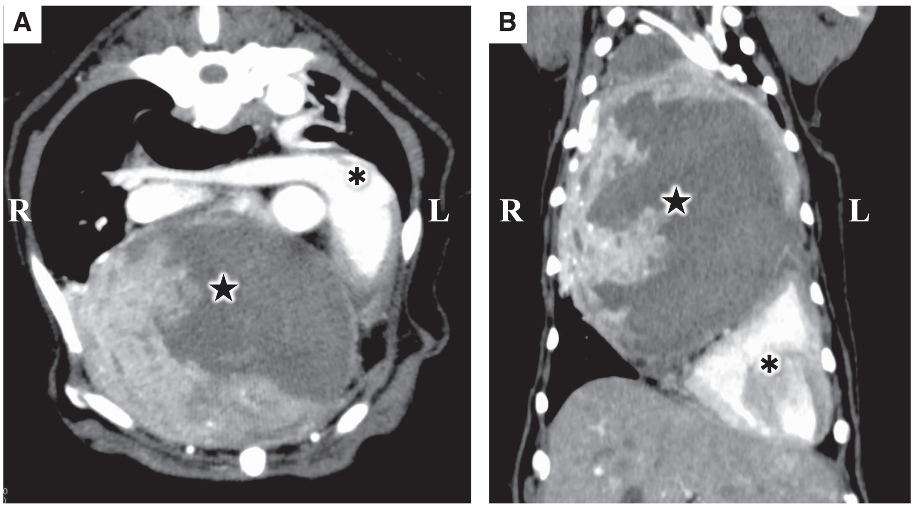

FIG 1. Representative slices of computed tomography scan images of the dog on day 12. (A) Axial view showing that the cranial mediastinal mass (as indicated by a star) displaced the heart (as indicated by an apostrophe) dorsolaterally. (B) Coronal view showing that the mass (as indicated by a star) displaced the heart (as indicated by an apostrophe) caudally.

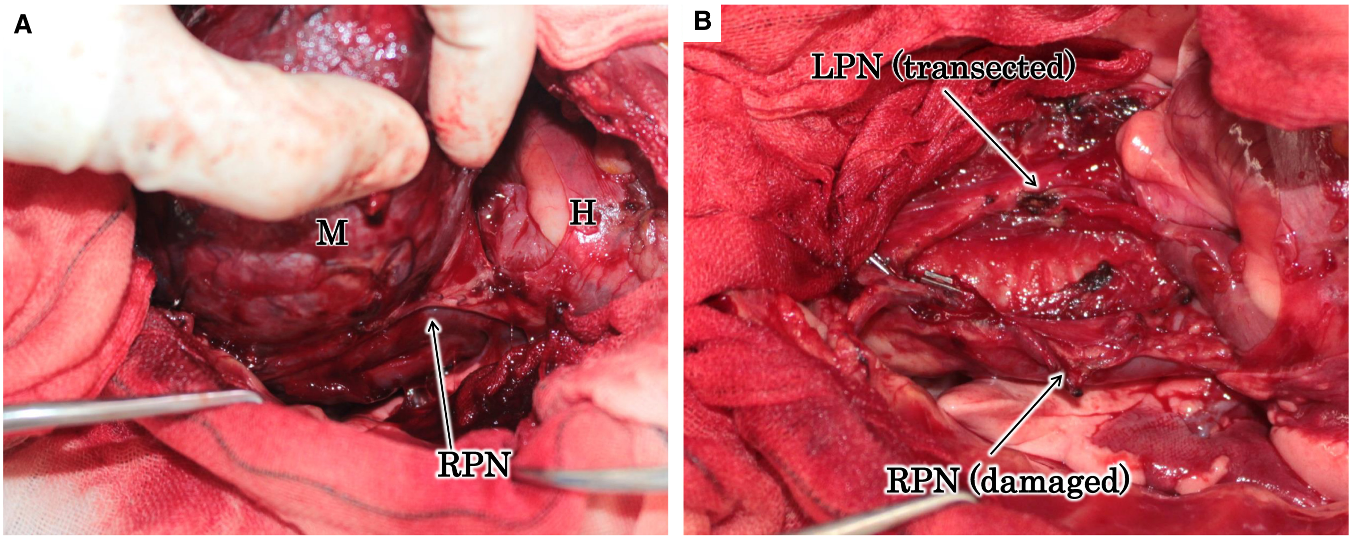

FIG 2. Intraoperative images during mass excision. (A) Image during tumour dissection. The cranial mediastinal mass (M) was retracted ventrally, and the firmly adherent right phrenic nerve (RPN) was visible. (B) Image during the completion of tumour excision. The RPN was preserved. However, it was partly damaged from electrosurgery, and the left phrenic nerve (LPN) was resected with the tumour.

dissected free from the tumour. However, thermal coagulation from bipolar electrosurgery accidentally reached the nerve. Thus, the intactness of the phrenic nerve on this side became questionable. The left phrenic nerve was segmentally resected for *en bloc* resection (Fig. 2B). The rest of the surgery was uneventful, and the sternum was closed routinely, including placement of a closed suction drain (The BD channel drain, C. R. Bard, Inc., New Jersey, USA.; placed as a information drain to monitor postoperative intrathoracic complications) after the mass was completely resected. Based on the histopathological examination, the mass was a malignant epithelial tumour of thymic origin (thymic carcinoma, type B3).

The dog presented with significant respiratory distress postoperatively, without evidence of haemothorax or pneumothorax. Further, paradoxical respiratory movement was observed (*i.e.* cranial movement of the diaphragm during the inspiratory phase, Fig. 3A, B), indicating the development of bilateral diaphragmatic paralysis. Radiography confirmed cranial displacement of both cruses of the diaphragm during the inspiration phase (Fig. 5B). Thus, a diagnosis of bilateral diaphragmatic paralysis was made. The right phrenic nerve injury presumably was caused by mild thermal damage, while the nerve itself was physically intact. The functional defect could potentially recover with time, hence, the dog was managed conservatively with oxygen supplementation.

On day 12 (5 days postoperatively), the dog exhibited evident respiratory distress and arterial blood gas analysis under room air revealed severe hypoxaemia, with an oxygen partial pressure of 47 mmHg, and normocapnia, with a carbon dioxide partial pressure of 41 mmHg, indicating respiratory failure, presumably caused by bilateral diaphragmatic paralysis and subsequent respiratory muscle fatigue. The dog was intubated and placed on mechanical ventilation after she collapsed shortly after the analysis. The hypoxia immediately resolved under artificial ventilation with physiological pressure/rate settings, indicating there was no functional defect in the pulmonary parenchyma. Thoracic CT scan did not reveal structural abnormality to explain the observed clinical signs in the thorax (*i.e.* presence of pneumothorax, signs consistent with aspiration pneumonia, re-expansion pulmonary oedema and other interstitial pulmonary pathologies), thereby supporting the diagnosis of diaphragmatic paralysis.

Tensioning diaphragmoplasty, which is a salvage procedure newly developed by one of the authors (X), was performed on the same day to counteract the inspiratory negative pressure in the thorax. The anaesthesia was maintained with 1% propofol, and fentanyl (5 to 10 µg/kg/minute, Fentanyl injection, Daiichi-Sankyo) and ketamin (600 µg/kg/minute, Ketamin injection 5%, FUJITA) were administered intravenously by constant rate infusion (CRI) for analgesia. The dog was placed in the dorsal recumbency, and the diaphragm was approached via midline celiotomy. On gross inspection, the diaphragm was subjectively flaccid. Thus, it was dissected along its ventral attachment, and the central one-third of the diaphragm was resected in a fan-like shape (Fig. 4A, B). The volume of the diaphragm resected was subjectively determined by retracting both cruses of the diaphragm medially and by determining the appropriate tension of the diaphragm to sustain the flat shape of the diaphragm during the inspiration phase. The remaining diaphragmatic cruses were apposed with each other on the midline and sutured with 3–0 polydioxanone with a simple continuous pattern (Fig. 4A). The ventral edge of the diaphragm was reattached to the thoracic wall using the same suture material. A sheet of polypropylene mesh (PROLENE® Mesh [hard-type], Echicon Inc., Somerville, NJ, USA), which was pre-shaped to match the shape of the diaphragm, was sutured to the caudal surface of the diaphragm, thereby covering the whole surface of the diaphragm ventral to the caval hiatus using a simple interrupted pattern with 3–0 polydioxanone (Fig. 4B). The abdomen was closed routinely.

Immediately after tensioning diaphragmoplasty, the dog's respiratory status remarkably improved. Hence, she could maintain regular daily activity without oxygen supplementation.

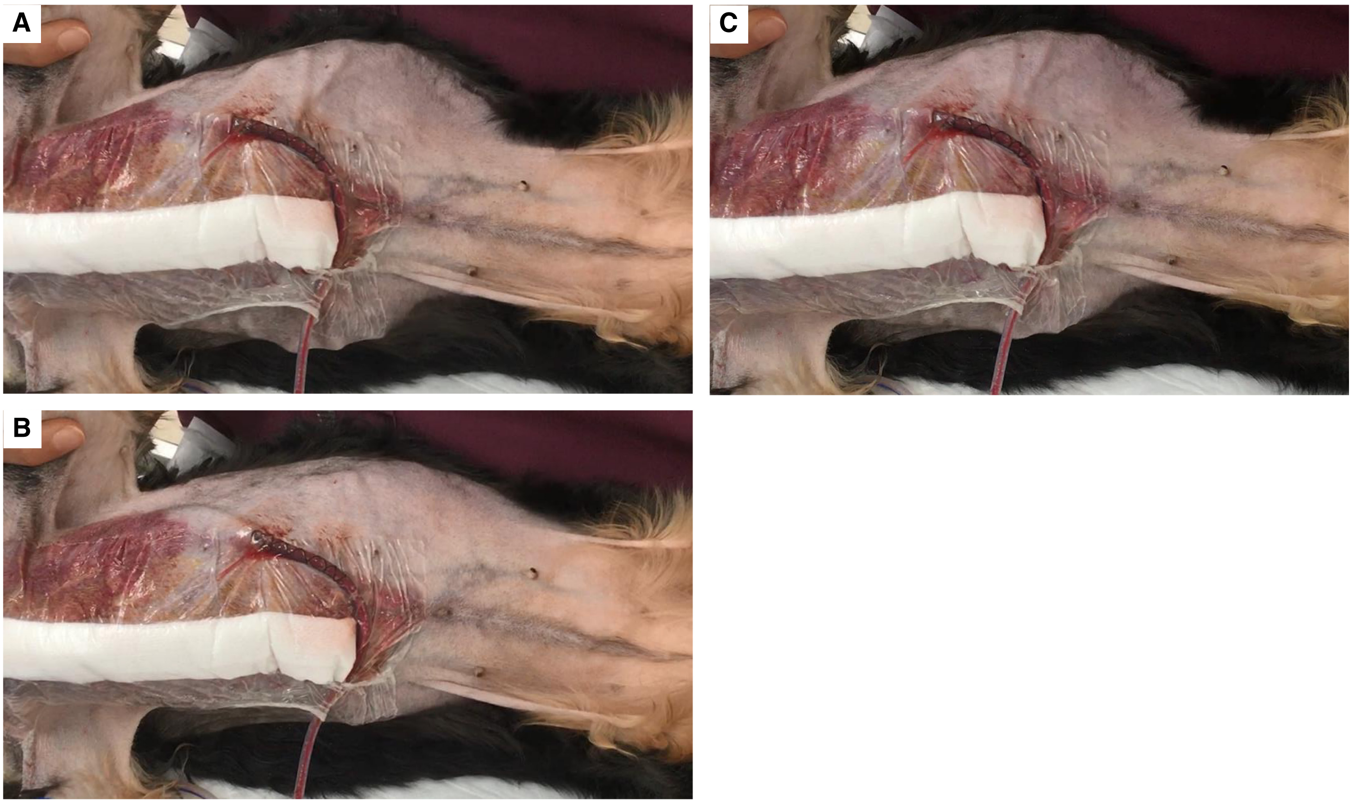

FIG 3. Respiratory pattern of the dog a day after mediastinotomy. (A) The inspiratory phase. The rib was extended laterally, and the abdominal contents were being pulled cranially. (B) The expiratory phase. The diaphragm was relaxed, and the abdominal contents were in the normal position. (C) A movie file of the respiratory pattern.

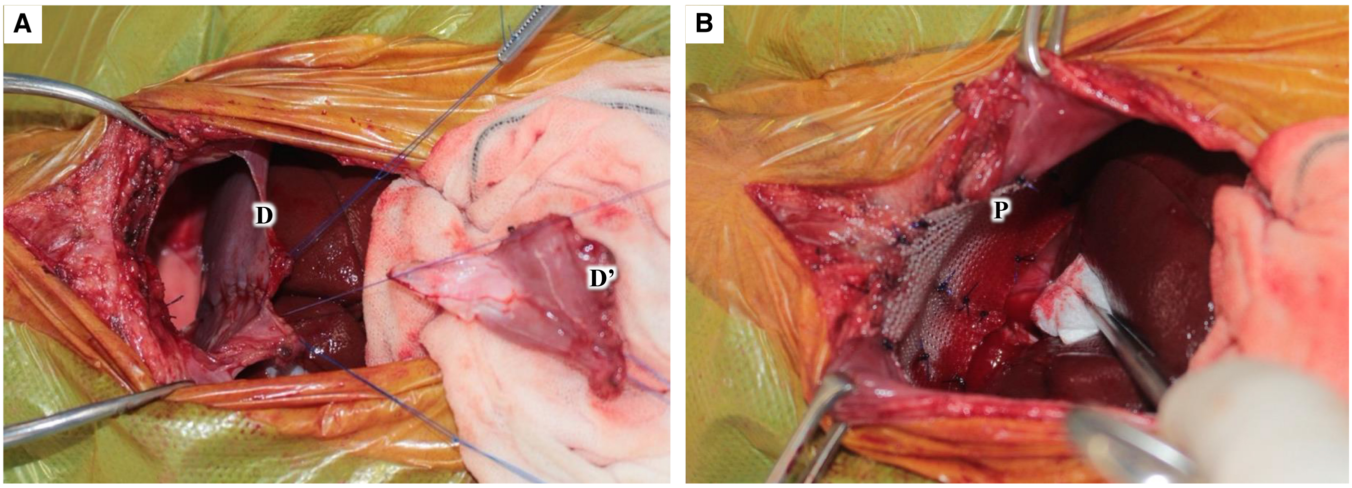

FIG 4. Intraoperative views of tensioning diaphragmoplasty. (A) The central one-third of the diaphragm (D) was resected in a fan-like shape (D′). The remaining diaphragmatic cruses were apposed on the midline and sutured with 3–0 polydioxanone using a simple continuous pattern. (B) A sheet of polypropylene mesh (P) was sutured to the caudal (abdominal) surface of the diaphragm to cover its whole surface ventral to the caval hiatus using a simple interrupted pattern with 3–0 polydioxanone.

Postoperative analgesia was provided by CRI of fentanyl (3 to 5 μg/kg/minute) and ketamin (600 μg/kg/minute) for 24 hours and by intravenous injection of buprenorphine thereafter for 2 additional days. For continued monitoring of respiratory status, the dog was kept hospitalised for 10 days after the tensioning diaphragmoplasty procedure without further event of dyspnoea and the dog was discharged on day 22 (10 days after the tensioning diaphragmoplasty). Radiography showed increased thoracic cavity function with caudal repositioning of the diaphragm (Fig. 5A–C).

On day 32, the dog's respiratory condition was re-evaluated. She was in good condition without any sign of respiratory

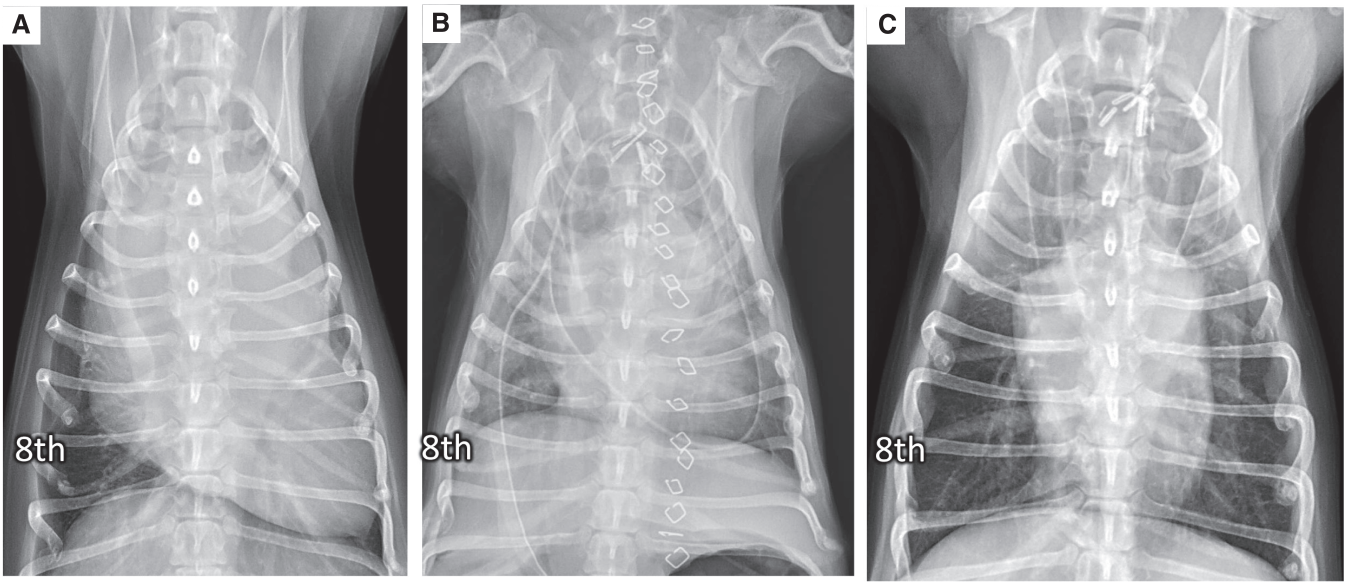

FIG 5. Ventrodorsal views of the radiographic images taken at the maximal inspiration point before the first surgery (A), after the first surgery (B) and after performing tensioning diaphragmoplasty (C). Note that the level of the diaphragm was displaced cranially after the first surgery (B) due to bilateral phrenic nerve paralysis, which was corrected after tensioning diaphragmoplasty (C).

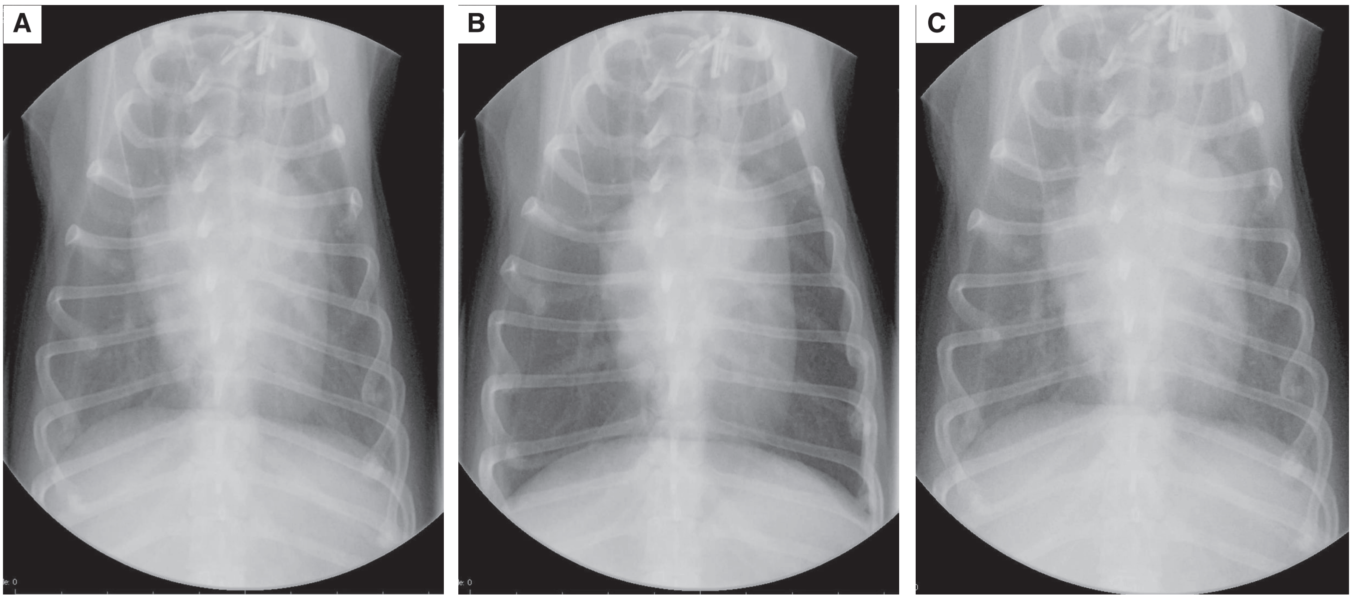

FIG 6. Fluoroscopic ventrodorsal view 20 days after tensioning diaphragmoplasty showing cranial movement of the diaphragm during the expiratory phase (A) and caudal movement of the diaphragm during the inspiratory phase (B), which indicated a near normal pattern of diaphragmatic motion during respiration. A movie file (C) is provided.

dysfunction. Continuous digital radiography with a flat panel detector (POPULUS So, Hitachi Medical Systems, Tokyo, Japan) revealed that the diaphragm's movement pattern was similar to that of a normal dog. That is, the diaphragm moved caudally during inspiration, and it was displaced cranially during expiration (Fig 6A, B).

The dog was followed-up until day 375, which is the day she died of urothelial carcinoma that originated from the urethra. Until the time of death, the dog remained clinically active, and the owner did not report any clinical symptoms related to respiratory issues. Necropsy was not performed.

DISCUSSION

Based on the literature search, this is the first case of iatrogenic diaphragmatic paralysis that was surgically treated in dogs. Other than diaphragmatic paralysis, the differential diagnoses of dyspnoea after thoracic surgery include pneumothorax, pleural effusion (including hydro-, haemo- and chylothorax), aspiration pneumonia and re-expansion pulmonary oedema (Brissot, 2016). These complications, particularly the latter two in cases of large thymic tumours, should not be overlooked before investigating the possibility of diaphragmatic paralysis. In the current case, parenchymal diseases of the lung had been ruled out before considering the unproved surgical procedure. The diagnosis of diaphragmatic paralysis as a cause of respiratory dysfunction could be difficult in non-surgical cases or post-surgical cases in which phrenic nerves are not clearly damaged. In dog with respiratory distress, it is difficult to take films at maximal inspiration which is essential when using radiographs to diagnose this condition. Other diagnostic tools may be efficient to diagnose this condition. Fluoroscopy and M-mode ultrasonography of the diaphragm, and electromyography have been used to confirm this condition (Santarelli et al., 2017). In the current case, the left phrenic nerve was resected and the right phrenic nerve was clearly damaged by thermal effect in operation. Since postoperative paradoxical respiratory movement was obvious, bilateral diaphragmatic paralysis was strongly suspected, after ruling out other possibilities.

The original concepts of tensioning diaphragmoplasty are as follows; first, sufficient tension should be provided to the unnerved, flaccid diaphragm and the inward motion of the diaphragm during inspiration, which is caused by negative intrathoracic pressure, must be prevented. Second, the elasticity of the diaphragm, which likely leads to eventual elongation in the long-term due to the repeated application of pulling force during each inspiratory phase, must be negated. For the latter purpose, a polypropylene mesh was sutured in a manner that the area of the diaphragm covered by the mesh must be as wide as possible to promote the sustained effect of diaphragmoplasty. However, the need for this step in this surgical procedure has not been confirmed. This mechanism of restoring normal caudal position of the diaphragm and counteracting against the inspiratory negative intrathoracic pressure likely played a role in the observed clinical improvement.

In the current case, tensioning diaphragmoplasty had an unexpected additional effect. That is, the diaphragmatic motion (functional reconstruction) was re-established. The flat panel detector clearly showed that the motion pattern of the diaphragm had been reversed from paradoxical to normal after tensioning diaphragmoplasty. The right phrenic nerve function was unlikely to have recovered. This was because both cruses of the diaphragm were positioned symmetrically and they moved in the same way despite resection of the left phrenic nerve. The effect of normalising diaphragmatic motion was likely created by a mechanism where the intercostal muscle force was effectively converted to the diaphragm force. The structural motion that creates thoracic volume to expand during inspiration comprises two independent mechanisms: caudal movement of the centre of the diaphragm (which is controlled by the phrenic nerves) and lateral movement of the ribs (which is controlled by the intercostal nerves) (Hermanson, 2013) (Fig 7A). In case of bilateral diaphragmatic paralysis, the former is compromised. However, the latter remains functionally intact (Fig 7B). Tensioning diaphragmoplasty creates tension across the diaphragm. Hence, the ribs separate laterally, and the diaphragm becomes flattened with its central dorm-shaped portion moving caudally, thereby essentially providing the net effect of converting the respiratory effort of the intercostal muscles to the force to pull back the centre of the diaphragm (Fig 7C). This mechanism likely contributed to the observed functional diaphragmatic reconstruction and significant clinical improvement.

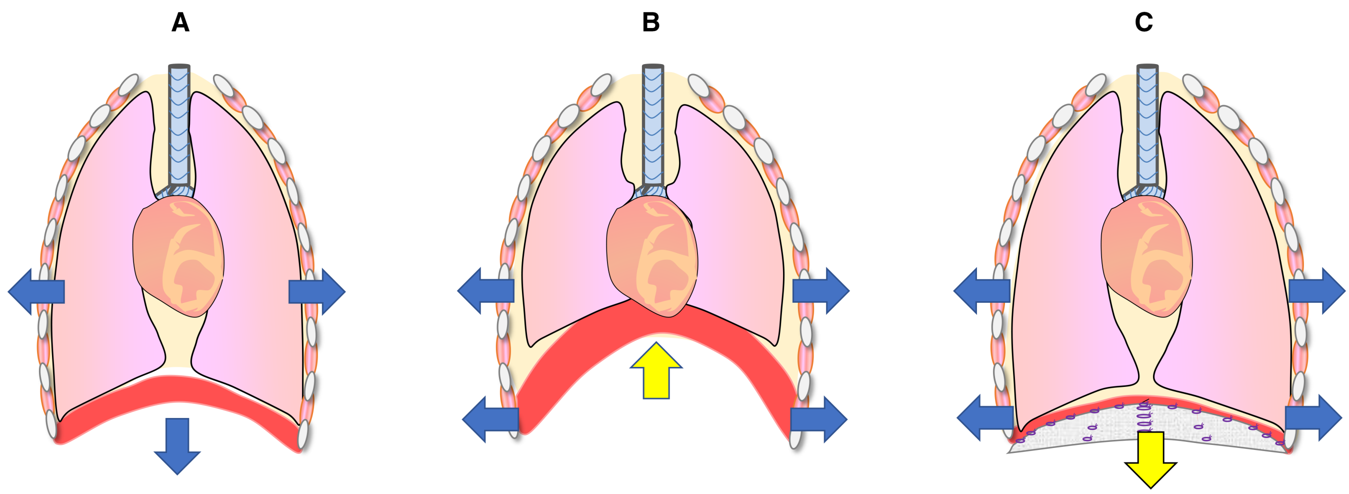

FIG 7. Images of the thoracic wall and diaphragmatic movement during the inspiration phase. The blue arrows indicate active motion of the two structures created by the contraction of the respiratory muscles. The yellow arrows indicate passive motion of the diaphragm caused by the active motion of the thoracic wall. (A) In the normal inspiratory phase, the intercostal muscles and diaphragmatic muscles contracted to expand the intrathoracic volume. (B) In case of bilateral phrenic nerve paralysis, the diaphragm passively moved cranially when the intercostal muscles contracted to expand the ribcage laterally, thereby negating their effect to increase intrathoracic volume. Note that the effort of the intercostal muscles increased to compensate for the effect of hypoventilation caused by the flaccid diaphragm. (C) With tensioning diaphragmoplasty, the increased effort of the intercostal muscles was converted into the force to pull a central portion of the diaphragm caudally, thereby effectively increasing the volume of the intrathoracic cavity.

In humans, the radial plication technique is reported to reduce the risk of iatrogenic damage to the phrenic nerve during the plication procedure (Steimer et al., 2022). In the tensioning diaphragmoplasty described here, we have also performed partial diaphragmectomy in a radial direction to avoid the areas of phrenic nerve innervation in each side of the diaphragm, although it was not necessary in case of complete phrenic nerve injury. Partially damaged phrenic nerve injuries have reported to recover their function in long-term. Mamada et al. (2020) reported that unilateral phrenic nerve injury associated with mitral valvoplasty recovered in 1 to 3 months after the initial surgery; whereas in humans it takes 5 to 12 months for injured phrenic nerves to recover. Vignoli et al. (2002) also reported that traumatic phrenic nerve paralysis in two cats recovered after 1 month of conservative therapy.

We could not follow-up the current case for a longer period due to unrelated death. However, the clinical effect of tensioning diaphragmoplasty lasted for at least 1 year. Therefore, the use of this newly developed procedure as a permanent and definitive surgical treatment for bilateral diaphragmatic paralysis should be further evaluated. This technique has several potential adverse effects. For example, it can cause expiratory dysfunction if excessive portion of the diaphragm is removed. Further, it can promote the development of re-expansion pulmonary oedema after the excision of a large intrathoracic mass and adhesion of the intraabdominal organs to the diaphragm, particularly the liver and gall bladder, which pose issues if abdominal surgery is required in a similar case in the future. In conclusion, the surgical technique described herein was effective, in this case, to re-establish the function of the diaphragm.

Author contributions

S. Matsumoto: Writing – original draft (equal). **K. Hosoya:** Conceptualization (equal); data curation (equal); formal analysis (equal); investigation (equal); methodology (equal); project administration (equal); resources (equal); supervision (equal); validation (equal); visualization (equal); writing – review and editing (equal). **S. Kim:** Supervision (supporting). **M. Okumura:** Supervision (supporting).

Conflict of interest

No conflicts of interest have been declared.

Data availability statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Video S1. A movie file of the respiratory pattern of the dog a day after mediastinotomy. Note that the diaphragmatic region is moving cranially during the inspiration phase and caudally during the expiration phase (a paradoxical respiratory pattern).

Video S2. A movie file of the fluoroscopic ventrodorsal view 20 days after tensioning diaphragmoplasty of the dog. Note that the diaphragm movement pattern has been reversed from paradoxical to normal.