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SHORT COMMUNICATION

Congenital Diaphragm Defect in a Neonatal Southern White Rhinocerus (*Ceratotherium simum simum*)

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ABSTRACT

The morphogenesis of the diaphragm requires a coordinated development of different components with potential of malformation. Diaphragmatic herniation has rarely been reported in large animals and has not been described in a rhinoceros. An 8-day-old- female southern white rhinoceros calf was diagnosed with congenital diaphragmatic hernia with large parts of the intestine translocated in the thoracic cavity. Gross necropsy revealed bilateral Bochdalek hernia, severe damage of the intestinal tract and congestion of both lungs. Histologic findings demonstrated lung hypoplasia and severe pneumonia due to regurgitation and aspiration of nutritional components.

1 | Introduction

The diaphragm serves a musculo-tendineous separation between the thoracic and abdominal cavity and is formed step-by-step during embryonic development from four different components: the septum transversum, the pleuroperitoneal membrane, the dorsal mesenterium of the oesophagus and muscle progenitors originating from the C3-C5 cervical somites (De Leon et al. 2022). Initially, pleuroperitoneal folds (PPFs) that derive from the lateral plate mesoderm form dorsolaterally, and leave the Ductus pleuroperitonealis open. Later, they merge with the Septum transversum (Persaud and Torchia 2025) and form the primitive diaphragm (Figure 1a).

Disruption of this concerted process leads to congenital diaphragmatic hernia (CDH), either retrosternally or dorsolaterally, known as Morgagni or Bochdalek hernia, respectively. A Bochdalek hernia arises from the failure to close the ductus pleuroperitonealis (Figure 1b) due to inadequate formation of the pleuroperitoneal membrane (García Cruz et al. 2023; Persaud and Torchia 2025).

Accounts on congenital defects in rhinoceros are rare. A review of the literature resulted in reports on Schistosoma-reflexus-like malformation (Lankton et al. 2014), a patent urachus (Langan et al. 2001; Bloch et al. 2014) in southern white rhinoceros (*Ceratotherium simum simum*), cleft palate and cardiac septal defects in a southern black rhinoceros (*Diceros bicornis minor*) (Lewis et al. 2016) and cranioschisis with cerebral aplasia in a Greater One-horned rhinoceros (*Rhinoceros unicornis*) (Schaftenaar et al. 2011). A review on main causes of death in black rhinoceros found four cases of congenital disease, including a Fallot's tetralogy, a spina bifida and cerebral herniation, a patent ductus arteriosus with ventricular septal defect and an encephalocele and gastroschisis (Radeke-Auer et al. 2023). However, some cases of congenital defects might be missed because of stillbirth without further investigation of the fetus (Hermes et al. 2020). Here, we describe for the first time a CDH in a rhinoceros.

2 | Materials and Methods

A female southern white rhinoceros (*Ceratotherium simum simum*) was born as the 2nd calf of a 13-year-old dam (Figure 2a).

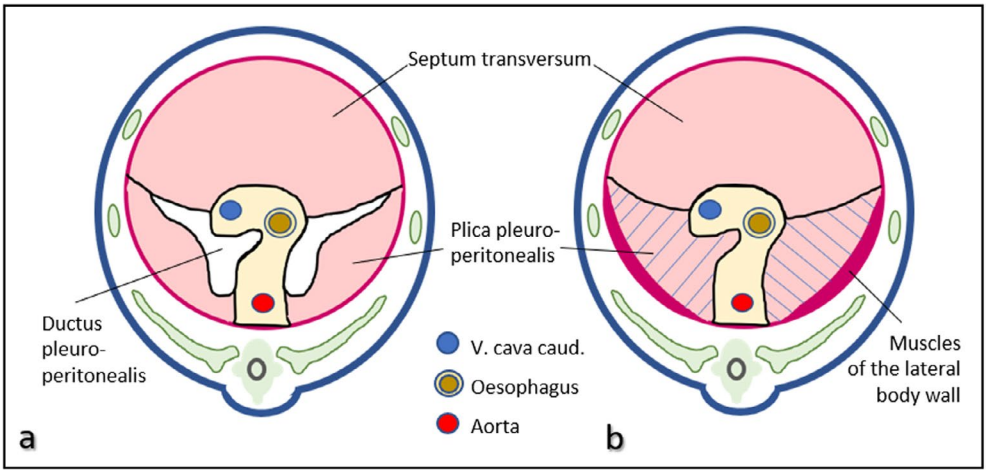

FIGURE 1 | Embryology: Development of the diaphragm with (a) Septum transversum, pleuroperitoneal folds and open Ductus pleuro-peritonealis potentially leading to malformation of Bochdalek hernia and (b) fully closed diaphragm with ingrowing muscle from the lateral body wall (adapted from Persaud and Torchia 2025).

FIGURE 2 | Newborn calf with mother (a) on day 1; Calf rolling and tumbling on day 3 (b).

Birth was without complications followed by an uneventful early postpartum period. On day 3 the calf showed various clinical signs: shortness of breath, increased breathing rate, signs of colic (rolling), lack of faecal heel, urine dribbling from the umbilical area (Figure 2b). On day 5 the calf was separated from the mother, and spasmolytic treatment (Buscopan comp. ad us. vet., 500 mg Metamizol und 4 mg Butylscopolamin), enema, and faeces manually removed lead to temporary improvement.

On day 7, the condition of the calf worsened, it showed regurgitation, was laterally recumbent with increased respiratory effort and was brought to the vet clinic and sedated (ketamine 0.7 mg/kg, diazepam 0.07 mg/kg, and medetomidine 0.016 mg/kg) for diagnostic imaging. X-Ray and CT-scans revealed a diaphragmatic herniation with large portions of intestine within the thoracic cavity (Figure 3). Surgery was performed under general anaesthesia (2%–3% isoflurane in oxygen) in an attempt to close the defect in the diaphragm. However, the animal died during the procedure.

Blood samples collected for whole blood count and blood chemistry revealed elevated HCT, haemoglobin, BUN, TP and GLOB, when compared to reference values (Miller et al. 2015), pointing to dehydration of the animal.

3 | Results

Necropsy was performed within one hour of death. The animal was in good body condition (weight 50 kg). At gross examination, large portions of the small intestinal tract were gas-filled and showed a dark bluish-red wall showing focally bloody imbibition and highly congested mesenterial blood vessels (Figure 4a).

Upon removal of abdominal organs, a bilateral herniation of the diaphragm was found and both lungs separated by the mediastinum were visible (Figure 4b). The pericardium was severely thickened and a tendon-like structure reached from the tip of the heart to the right thorax wall (Figure 4c). The lungs were compressed and the right lung appeared much smaller than the left (Figure 4d) due to the space-demanding abdominal organs that had penetrated the chest cavity.

H&E stained sections of the lungs showed hypoplasia, alveolar haemorrhage and edema (Figure 4e,f). Diffuse fibrino-necropurulent bronchopneumonia as a consequence of aspiration of food was detected with diffuse inflammation, intralesional bacteria and massive leucocyte and macrophage invasion. Alveolar epithelial cells showed reactive hyperplasia. Overall, airways

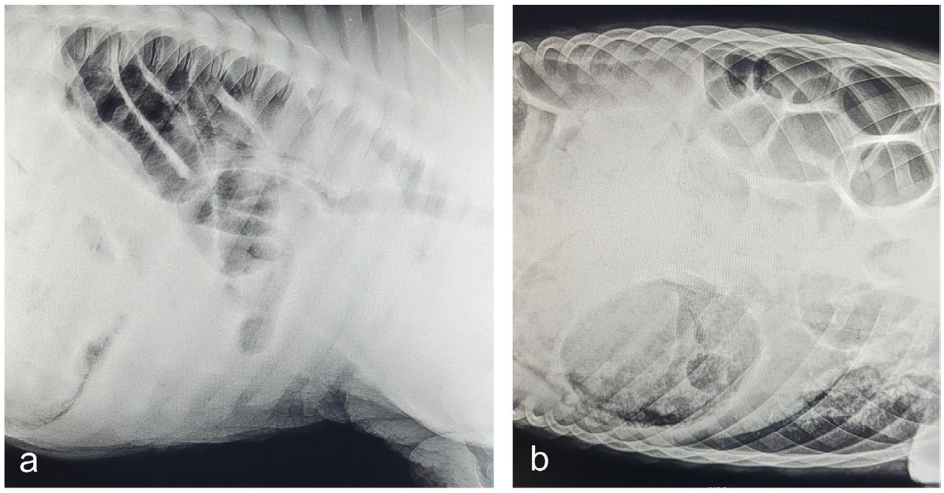

FIGURE 3 | Diagnostic imaging: X-Ray (a) and CT (b) showing gas-filled intestinal loops within the thoracic cavity.

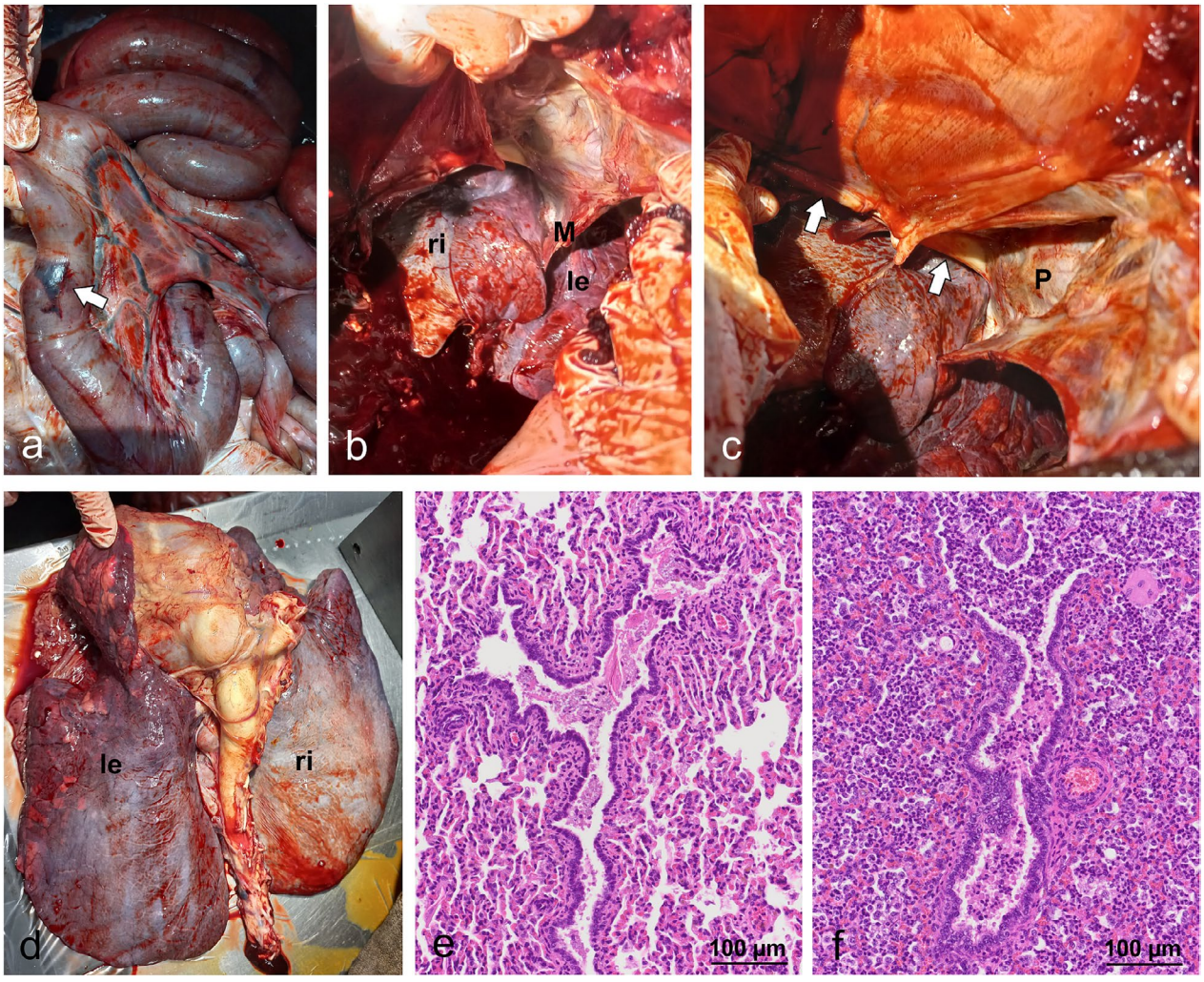

FIGURE 4 | Pathology and histology: Small intestine (a) with dark bluish-red wall showing focally bloody imbibition (arrow) and congested mesenterial blood vessels. Bilateral diaphragmatic hernia (b, c) separated by the mediastinum (M); tendon-like structure reaching from the pericard (P) to the lateral thorax wall (arrows); Left and right lungs (d), the right lung severely compressed; H&E stained sections of the left (e) and right lung (f) showing different degree of hypoplasia, inflammatory cell infiltration and nutritional components in the airconducting structures.

were filled with nutritional components and necrosis of the epithelial lining was found. The severity of the lesions suggests a long-lasting, chronically active process.

4 | Discussion

Bochdalek (or posterolateral) hernia, is the most common form of CDH in humans (De Leon et al. 2022), however, seems to be a rare condition in large animals (Williams et al. 2016). This case emphasises however, to consider congenital malformations such as CDH in newborns who present with abdominal and respiratory problems. With unclear and changing symptoms before separation from the mother and upon physical examination, only X-Ray and CT lead to the correct diagnosis. The extent of the defect prevented the damage from being repaired. In addition, the overall condition of the animal's intestinal and pulmonary tract was beyond potential recovery. Although the hernia turned out to be bilateral, the stomach probably prevented the passage of intestinal loops on the left side and subsequently less damage to the left lung. The weight of the protruding organs on the right side caused the development of a thick strand of connective tissue at the dorsal edge of the septum transversum.

The development of the diaphragm is complex due to the contribution of multiple structures contributing to the final diaphragm (Sefton et al. 2018). Disruption of this development leads to CDH however, the exact aetiology is not yet clear. Although complex malformations along with CDH occur, around 60% of CDH cases are isolated defects with low familial recurrence and not associated with other congenital anomalies (De Leon et al. 2022).

Studies on the early developmental events suggest that the lack of the expansion of the PPFs along with inability to recruit muscle progenitor cells which is linked with the projection of phrenic axons are crucial elements that lead to CDH (Sefton et al. 2018). Most CDH-related genes have been found to be expressed in the non-muscular PPF (Sefton et al. 2018) and therefore the formation of a diaphragmatic hernia is most likely a very early event during embryonic development.

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The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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