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CASE REPORT

Congenital bilateral laryngeal paralysis in a neonatal foal

L. De Maré | C. Loublier | H. Amory | L. Lecoq | C. Cesarini

Equine Clinical Department, Faculty of Veterinary Medicine, University of Liege, Liège, Belgium

Correspondence: C. Cesarini
Email: ccesarini@uliege.be

Summary

Laryngeal paralysis is rarely described in foals, despite being a known congenital condition in several dog breeds and the second most common laryngeal abnormality in human neonates. This report describes the case of a 3-h-old Warmblood colt referred for inspiratory stridor observed immediately after birth, which was diagnosed as bilateral laryngeal paralysis. The foal arrived in lateral recumbency, with an orotracheal tube in place, showing a weak suckle reflex and mild signs of dehydration. Blood analysis revealed an inverted neutrophil-to-lymphocyte ratio, increased creatine kinase activity, elevated haematocrit and higher blood lactate and creatinine concentrations. Airway endoscopy revealed bilateral laryngeal paralysis and intermittent dorsal displacement of the soft palate, without signs of inflammation. The foal was hospitalised in the neonatal intensive care unit; a nasotracheal tube was placed, and medical treatment was initiated (oxygen, intravenous fluid therapy with glucose and dimethyl sulfoxide, broad-spectrum antimicrobials, milk via nasogastric tube, and supplementation with B-complex vitamins, vitamin E and selenium). A diagnosis of neonatal encephalopathy was suspected, although nutritional myodegeneration could not be excluded. Laryngeal function progressively recovered, allowing removal of the nasotracheal tube 24 h after arrival and discharge from the clinic 10 days later, without the need for invasive surgical procedures. Follow-up 1 month after admission showed normal laryngeal function at rest. Bilateral laryngeal paralysis should be considered a differential diagnosis for neonatal stridor in foals. This case demonstrates that bilateral laryngeal paralysis can be fully reversible in foals and should not necessarily be associated with a poor prognosis.

KEYWORDS
horse, dyspnoea, encephalopathy, inspiratory, newborn, stridor

INTRODUCTION

Laryngeal paralysis is a well-known congenital condition in dogs and the second most common laryngeal abnormality in human neonates, after laryngomalacia (Friedman et al., 2018; Kitshoff et al., 2013; Kohn et al., 2021). However, it has rarely been described in foals, and little is known about its prevalence and underlying mechanisms in this species.

In dogs, congenital laryngeal paralysis is most often reported as a hereditary disorder with a guarded to poor prognosis, affecting several breeds (e.g. German Shepherds, Siberian Huskies, Bouviers des Flandres, Bull Terriers, Dalmatians, Cocker Spaniels, Miniature Pinschers, Dachshunds and Rottweilers; Kitshoff et al., 2013). It may occur as a mononeuropathy, involving degeneration of the recurrent laryngeal nerves (RLN) or as part of a broader polyneuropathy complex that includes oesophageal dysfunction. Affected puppies are profoundly dyspnoeic at birth and collapse easily after exertion.

In human neonates, vocal fold paralysis (VFP)—the second most common congenital laryngeal abnormality—shares similar functional consequences with laryngeal paralysis in horses and accounts for 10–20% of congenital laryngeal pathologies (Ridgway et al., 2021; Scatolini et al., 2018). Paralysis is bilateral in up to 73% of cases (Ridgway et al., 2021). Affected infants exhibit varying degrees of difficulty in breathing, phonation, and swallowing. VFP is most often

idiopathic (30–75.6%) but can also result from direct damage to the vagus nerve or its nuclei, secondary to brain anoxia, traumatic injury or medication side effects (Kohn et al., 2021; Ridgway et al., 2021; Scatolini et al., 2018). Depending on the nature and severity of the lesion, laryngeal reinnervation may take months to years (Kohn et al., 2021; Ridgway et al., 2021).

Transient unilateral pharyngeal dysfunction has occasionally been described in foals with prematurity or neonatal encephalopathy (NE) (Holcombe et al., 2012; Wilkins et al., 2003). A single publication describes a 2-day-old dyspnoeic foal diagnosed with bilateral laryngeal paralysis and intermittent dorsal displacement of the soft palate, secondary to a cerebral vascular hamartoma localised at the obex and suspected to cause compression of cranial nerve nuclei VII, IX and X (Borel et al., 2014).

Obstruction of the upper respiratory tract due to pharyngeal collapse, laryngeal paralysis and oedema has also been reported in Quarter Horse foals homozygous for the hyperkalemic periodic paralysis gene (Carr et al., 1996; Finno et al., 2009). Equine laryngeal dysplasia has also been reported as a cause of acute respiratory distress in a neonatal foal (Koskinen & Hewetson, 2017).

Other causes of bilateral laryngeal paralysis have been described in adult horses (Dixon et al., 2001), including hepatic encephalopathy, bilateral guttural pouch mycosis, lead or organophosphate intoxication and hyperextension of the neck during general anaesthesia (De Clercq et al., 2018). However, these aetiologies are unlikely to be responsible for bilateral laryngeal paralysis present at birth.

Respiratory distress at birth is a critical and stressful event that requires emergency intervention. This case report describes the diagnostic investigations, clinical management and successful outcome of a newborn foal presenting with severe respiratory distress at birth caused by bilateral laryngeal paralysis, a very rare condition in equine neonatology. Given the current scarcity of information in the literature, this paper also explores potential differential diagnoses and compares this disorder with existing knowledge regarding its aetiology and prognosis in other species.

CASE HISTORY

An 80-kg warmblood male born at term (338 days of gestation) after a very mild dystocia was referred 3 h after birth for severe respiratory distress and cyanotic mucous membranes. The veterinarian had placed an orotracheal tube for referral and administered dexamethasone and clenbuterol. The mare was multiparous and had no problems during gestation and after parturition.

CLINICAL FINDINGS

On admission, the foal was recumbent but bright, alert and strong, with the orotracheal tube still in place. Clinical examination revealed floppy ears and dry, pink oral mucous membranes with a prolonged capillary refill time (2–3 s). No other abnormalities were noted on general physical examination. Neurological assessment showed a weak suckle reflex. Upon removal of the endotracheal tube, the foal exhibited signs of severe respiratory distress with inspiratory stridor and the mucous membranes became cyanotic.

Blood analysis included white blood cell count, packed cell volume (PCV), serum amyloid A (SAA), fibrinogen, plasma total protein concentration, glucose, electrolytes, creatinine and lactate concentrations, as well as muscle enzyme activity. Abnormal results included a markedly increased PCV (0.58), an inverted neutrophil-to-lymphocyte ratio (2.17×10^9/L: 3.17×10^9/L), severe hyperlactatemia (>12 mmol/L) and moderate increases in plasma creatinine concentration (324 μmol/L) and plasma creatine kinase (CK) activity (11,440 U/L). Based on the history, clinical signs and bloodwork results, a modified sepsis score of 13 was retrospectively calculated.

Thoracic ultrasound revealed a few bilateral B-line artefacts cranioventrally over two intercostal spaces. Abdominal ultrasonography was within normal limits. Ultrasound of the umbilicus showed no evidence of thrombosis in the umbilical vein or arteries, which was considered normal given the age of the patient. Endoscopy of the upper airways, performed without sedation, revealed complete bilateral laryngeal paralysis and intermittent dorsal displacement of the soft palate (Figure 1). The remaining structures (nasal cavities, epiglottis, palatopharyngeal arch, guttural pouches) were within normal limits. Radiographs and ultrasound of the larynx did not reveal any structural damage or congenital abnormalities. The cricoid and thyroid cartilages were normal in appearance, as well as their joints and the position of the left and right crico-arytenoideus lateralis muscles.

TREATMENT

The foal was hospitalised in the neonatal intensive care unit (NICU). A nasotracheal tube was placed to ensure airway patency, delivering 6 L/min of humidified oxygen. An intravenous (i.v.) catheter was placed and a sterile blood sample was collected for hemoculture. Supportive treatment was initiated with i.v. fluid therapy (Lactated Ringer's solution) and a continuous rate infusion (CRI) of 50% i.v. glucose. During the first 24 h, dimethyl sulfoxide (DMSO 90%) was added to the fluids at 1 g/kg. Spurious hypercreatininemia was initially suspected, and parenteral broad-spectrum antimicrobial therapy was started with i.v. penicillin (22,000 IU/kg q. 6 h, Penicillin 5,000,000 Kela®) and gentamicin (6.6 mg/kg bwt q. 24 h, Genta-Equine®). The foal also received low molecular weight heparin (100 IU/kg SC q. 24 h, Clexane® 80 mg) and an injection of vitamin E and selenium (Myogaster®), followed by oral supplementation with vitamin E (500 IU q. 24 h, Equi-Vit E®). The mare's colostrum, along with 1 L of frozen bovine colostrum, was administered via a nasogastric feeding tube to optimise passive transfer of immunity. Twelve hours after birth, 2 L of hyperimmune plasma was administered i.v. due to complete failure of passive transfer (IgG <400 mg/dL; SNAP Foal Test, IDEXX).

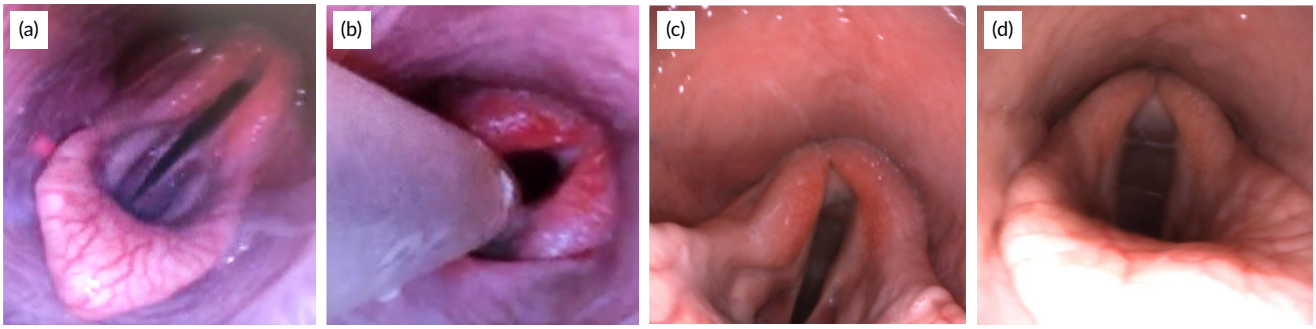

FIGURE 1 Endoscopic images of the larynx, showing on admission, (a) bilateral laryngeal paralysis and (b) placement of a nasotracheal tube. (c) On Day 10, persistent right laryngeal paralysis and (d) on Day 30, complete resolution of laryngeal pathology.

Initially, the foal became weaker, exhibited abnormal behaviour (e.g. episodes of agitation, weak suckle reflex, floppy ears) and was unable to stand or urinate properly, prompting placement of a sterile urinary catheter for 24 h. Over the following hours, the foal's hydration status improved and venous blood lactate decreased to 5.88 mmol/L.

On Day 2, the foal was able to stand with minimal assistance, and mental status improved. Respiratory auscultation revealed increased tracheal secretions. Repeat endoscopy of the upper airways showed partial, though incomplete, arytenoid movement and mild inflammation on the medial sides of both arytenoids, likely secondary to friction from the endotracheal tube. The foal only exhibited inspiratory stridor during excitement, without respiratory distress after withdrawal of the nasotracheal tube.

By Day 3, the foal exhibited a strong suckling reflex and only occasional stridor during excitement. Intranasal oxygen supplementation was discontinued. Repeated bloodwork showed improved CK activity (821 U/L), normal haematology and a decrease in creatinine concentration (232 μmol/L), although the SAA concentration had increased (215 mg/L). Thoracic radiographs and ultrasound revealed bilateral generalised interstitial opacification and multifocal discrete caudo-dorsal lesions (B-lines), suggesting of worsening pneumonia. Umbilical ultrasound showed increased diameters of the umbilical vein (1.4 cm) and arteries (left: 1.2 cm, right: 1.4 cm), with central hyperechoic foci, indicating suspected infection of the umbilical structures. The blood culture did not yield any bacterial growth. Due to the slow decline in creatinine levels despite fluid therapy, and imaging findings consistent with bronchopneumonia and omphalitis despite broad-spectrum antimicrobial treatment, penicillin and gentamicin were discontinued and replaced with ceftiofur (5 mg/kg i.m. q. 12 h).

On Day 4, controlled nursing from the mare was introduced while enteral feeding via the nasogastric tube was gradually reduced. Creatinine levels normalised (163 μmol/L), and inspiratory stridor continued to show consistent, gradual improvement.

Follow-up endoscopy on Day 10 showed full mobility of the left arytenoid (Figure 1) and no evidence of laryngeal inflammation, although incomplete abduction of the right arytenoid persisted. Haematology was repeated and remained within normal limits, while the SAA concentration had returned to the normal range (0 mg/L). Thoracic ultrasound revealed significant improvement of the pneumonia; however, the diameter of the umbilical structures had slightly increased after 7 days of ceftiofur treatment (vein: 1.5 cm; left artery: 1.2 cm; right artery: 1.6 cm). At that point, inadequate penetration of ceftiofur into the umbilical structures was suspected to account for the lack of response of the umbilical infection. For this reason, the foal was discharged with a 2-week course of oral trimethoprim-sulfamethoxazole (30 mg/kg bwt p.o. q. 12 h, Emdotrim 60%) and rifampicin (10 mg/kg bwt p.o. q. 12 h, Rifadine® 150 mg), a broad-spectrum antimicrobial combination with superior tissue penetration.

At the 2-week follow-up after discharge, the foal showed no signs of inspiratory stridor. Repeat endoscopy confirmed complete normalisation of laryngeal function and movement (Figure 1). Ultrasonography showed complete resolution of the omphalitis. The foal was reported to have continued normal growth without respiratory issues during the following 6 months.

DISCUSSION

Even though it is a rare condition, bilateral laryngeal paralysis should be included in the differential diagnosis for foals presenting with inspiratory stridor immediately after birth. Unlike in other species, where hereditary causes are common and often associated with a poor prognosis, this case report highlights that bilateral laryngeal paralysis in foals can be completely reversible within a short period of time, making it worthwhile to start supportive care in these patients before assuming a poor prognosis.

In this case, neonatal encephalopathy (NE) was mainly suspected, although nutritional myodegeneration could not be completely excluded. Neonatal encephalopathy, also referred to as hypoxic-ischaemic encephalopathy or dummy foal syndrome, can result in laryngeal dysfunction, particularly when it arises from hypoxic-ischaemic events. Inadequate oxygenation and blood flow may cause neuronal damage to the brainstem, potentially affecting the *nucleus ambiguus*. This nucleus, through the vagus and recurrent laryngeal nerves, controls the motor function of the laryngeal muscles.

Neonatal encephalopathy is well described in equine neonates and is often associated with in utero (e.g. placentitis) or

peripartum (e.g. premature placental detachment, dystocia) hypoxia and/or hypoperfusion (Magdesian, 2008). In this case, no information about the placenta was available, and the owner did not report any signs of premature mammary development or vaginal discharge during gestation. However, the marked elevation in creatinine concentration shortly after birth raised strong suspicion of placental insufficiency. Additionally, the clinical signs observed at admission and the foal's evolution during the first 24 h of hospitalisation supported the hypothesis of a hypoxic-ischaemic event. Finally, although the dystocia was described as mild, it may have exacerbated the situation.

Interestingly, NE is also the second most common cause of congenital laryngeal dysfunction in human neonates, following idiopathic forms (Bowe et al., 2019; Scatolini et al., 2018; Wang et al., 2011). In human infants with less severe birth asphyxia, selective involvement of certain brainstem regions has been reported (Quattrocchi et al., 2010, 2016). Clinical symptoms in these cases are consistent with brainstem lesions and include feeding difficulties, speech and communication issues and visual impairment (Quattrocchi et al., 2016).

Nutritional dystrophic myodegeneration (also known as white muscle disease) is a well-documented cause of profound muscle weakness and dysphagia in foals, which can be evident from birth (Lofstedt, 1997). Foals less than 30 days of age are primarily affected (Streeter et al., 2012). Oxidative damage to muscle tissue can involve the laryngeal and pharyngeal muscles, potentially leading to laryngeal dysfunction (Wilkins, 2009). Diagnosis typically relies on elevated muscle enzyme activities, particularly creatine kinase (CK), along with low selenium levels (and to a lesser extent, vitamin E deficiency) and decreased glutathione peroxidase activity (Lofstedt, 1997). In this foal, CK activity was markedly increased after birth, and generalised weakness was observed during the first 24 h of life. The foal's progressive clinical improvement coincided with normalisation of CK activity, which may support a diagnosis of nutritional myodegeneration. Measurement of serum vitamin E, selenium concentrations or glutathione peroxidase activity in the mare or foal was not performed in this case. However, because soils in our geographical area are historically deficient in selenium, the foal was empirically supplemented with vitamin E and selenium. In human medicine, vitamin E has demonstrated neuroregenerative properties via its antioxidant activity, reduction of neuronal apoptosis, enhancement of neuronal survival, increased myelin density and stimulation of neurotrophic factors (Icer et al., 2021).

Prematurity is another recognised cause of laryngeal paralysis in human infants (Kohn et al., 2021; Ridgway et al., 2021). The foal in this case exhibited some features suggestive of prematurity or dysmaturity, such as floppy ears and an inverted neutrophil-to-lymphocyte ratio (Magdesian, 2008). However, this diagnosis was considered unlikely given the normal gestational length, the appropriate size and weight of the foal for its breed, and the absence of additional clinical signs typically associated with prematurity (e.g. hypoglycaemia, difficulty maintaining body temperature, flexor tendon laxity). Unfortunately, radiographic evaluation of the carpal and tarsal joints, which could have provided further support for a diagnosis of dysmaturity, was not performed.

Sepsis was also considered a potential diagnosis due to the foal's generalised weakness and a retrospectively calculated modified sepsis score of 13 (Wong et al., 2018). Although the blood culture was negative, it is well established that a single blood culture has limited sensitivity (25.8–36%) for detecting septicaemia (Russell et al., 2008), whereas the modified sepsis score offers only moderate sensitivity (60%) and specificity (61%) (Wong et al., 2018). In this case, a total failure of passive transfer of immunity was documented, which is a known risk factor for neonatal sepsis. The presence of localised infections, such as pneumonia and omphalitis, may have resulted from systemic infection or could have developed as primary infections. Additionally, prolonged placement of the nasotracheal tube (48–72 h) likely facilitated tracheal contamination with upper airway commensal bacteria. Despite the inability to definitively exclude sepsis, it is considered extremely unlikely that sepsis was the primary cause of bilateral laryngeal paralysis in this foal.

Equine laryngeal dysplasia has been reported as a cause of acute respiratory distress in neonatal foals that present with weakness, recumbency, and inspiratory stridor from birth (Koskinen & Hewetson, 2017). This congenital disorder results from aplasia or hypoplasia of structures derived from the fourth and sixth branchial arches (Barakzai, 2015). Although the right side is more frequently affected, bilateral involvement has been documented (Lane, 2001). Typical endoscopic findings include paralysis and oedema of the arytenoid cartilages, rostral displacement of the palatopharyngeal arches and dorsal displacement of the soft palate. Palpation and ultrasonography typically reveal atrophy of the cricothyroid muscle. In the present case, endoscopic and ultrasonographic findings did not support laryngeal dysplasia, and spontaneous clinical improvement would not be expected with such congenital malformations without surgical intervention.

Birth trauma resulting in bilateral compression or stretching of the recurrent laryngeal nerves was considered unlikely, as the foal was delivered after only mild dystocia, which resolved easily with minimal manual assistance.

In equine medicine, the diagnosis of laryngeal paralysis is primarily based on endoscopic evaluation and laryngeal ultrasonography. In contrast, advanced imaging modalities such as CT or MRI and laryngeal electromyography (EMG) are used in human and canine patients to investigate underlying aetiologies and assess prognosis. In human neonates with hypoxic-ischaemic encephalopathy, selective brainstem lesions have been documented (Quattrocchi et al., 2016). The application of such diagnostic techniques to equine neonates could potentially provide valuable diagnostic and prognostic information in similar cases. Advanced diagnostic modalities like EMG and MRI were not used in the present case and were unlikely to have changed its management significantly. They are often associated with higher costs and are not routinely performed in foals.

Conservative management of laryngeal paralysis was selected in this case, consisting mainly of maintaining the presence of a nasotracheal tube for 24 h to ensure airway patency. This therapeutic approach was considered less invasive than a tracheotomy in such a small patient. Furthermore, in human infants, tracheotomy is correlated with a higher morbidity (12–14% of early complications and 26–62% of late complications) and mortality (up to 5.9%; Dal'Astra et al., 2017; De Gaudemar et al., 1996). There are no published data about the complication rate of tracheotomy in foals, although it remains a potential therapeutic option for managing life-threatening upper airway obstruction. Had the laryngeal paralysis persisted longer, other treatments such as electroacupuncture or nerve graft techniques could have been considered before resorting to more invasive surgical procedures (i.e. tie back, arytenoidectomy or permanent tracheostomy).

The foal received supportive treatments for neonatal encephalopathy, failure of passive transfer of immunity and potential sepsis. DMSO was administered for its neuroprotective properties (Bulama et al., 2022).

It is important to mention that antimicrobial choices in this case did not fully comply with current antimicrobial stewardship principles. Over recent years, new regulations governing the use of critically important antimicrobials have been progressively implemented across Europe (Ryan et al., 2023). The use of fluoroquinolones and third- or fourth-generation cephalosporins should always be supported by bacterial culture and susceptibility testing.

In the present case, penicillin and gentamicin were selected as first-line broad-spectrum therapy. Because the hypercreatininemia was initially considered spurious, the potential nephrotoxicity of gentamicin was deemed acceptable. However, owing to the slow normalisation of creatinine concentrations and the emergence of umbilical infection and pneumonia during the first days of treatment, nephrotoxic drugs were discontinued and the regimen was switched to sodium ceftiofur. As ceftiofur is classified as a critically important antimicrobial, its use should ideally be guided by culture and sensitivity results. Current legislation allows an exception for foals when the choice of a critical antibiotic can be justified. A blood culture was obtained but yielded no growth; sampling the infected umbilical remnants was impossible because the lesions were deep and lacked a discrete accumulation of pus. Moreover, given the high likelihood of nasopharyngeal contamination after 48 h of nasotracheal intubation, a tracheal wash was not performed. Under present guidelines, and in the absence of clear signs of sepsis at presentation, parenteral trimethoprim–sulfonamides or amoxicillin–clavulanic acid would have been safer and more appropriate first-line options, particularly with regard to renal safety, although resistance to potentiated sulfonamides can be high (Floyd et al., 2022).

Little is known about the long-term prognosis of bilateral laryngeal paralysis in foals. It is reasonable to assume that the prognosis is closely linked to the extent and underlying cause of the neural damage. Foals affected by neonatal encephalopathy generally have a favourable outcome, with reported survival rates ranging from 70 to 80%, particularly when born at term with normal birthweight and in the absence of major complications (Abraham, 2023; Wilkins, 2015). Neurological signs in these cases typically improve rapidly during the first week of life, and long-term deficits are uncommon. However, certain factors (seizures, sepsis, lack of improvement within 5 days) have been associated with a poorer prognosis and can reduce the survival rate to approximately 50–60% (Gold, 2017; Gold et al., 2016; Taylor, 2015).

In the present case, laryngeal function began to recover as early as Day 2, with progressive improvement over the following days. This rapid resolution contrasts with the timeline described in human infants, where recovery from bilateral vocal fold paralysis may take weeks to months—or even years in some cases (Kohn et al., 2021). In canine medicine, congenital laryngeal paralysis is often associated with a poor outcome. Most affected puppies are euthanised within the first 10 weeks of life due to progression of clinical signs and associated complications (Davies & Irwin, 2003). Nonetheless, a study involving Siberian Huskies reported spontaneous improvement in up to 40% of affected puppies by the time they reached maturity (von Pfeil et al., 2018).

In human medicine, compression neuropathies—when not accompanied by axonal damage—are associated with favourable outcomes and complete functional recovery (Ridgway et al., 2021). In more severe cases with axonal disruption, reinnervation may occur, particularly in paediatric patients. However, this process may lead to abnormal, nonselective laryngeal reinnervation, which can impair functional recovery (Flores et al., 2000).

Overall, bilateral laryngeal paralysis carries a less favourable prognosis than unilateral involvement. Reported spontaneous recovery rates vary between 32 and 65% (Kohn et al., 2021). Idiopathic cases are generally associated with the best outcomes, followed by neurogenic cases, whereas traumatic or iatrogenic etiologies tend to have a worse prognosis. In paediatric patients, approximately 50% of idiopathic cases recover spontaneously within 1–2 years, with an additional 10% recovering by 5 years (De Gaudemar et al., 1996). Recovery beyond 18–24 months is considered rare (Jabbour et al., 2014; Kohn et al., 2021; Zur, 2018), and it is generally recommended to delay invasive surgical intervention for at least 1–2 years (De Gaudemar et al., 1996; Kohn et al., 2021).

In humans, laryngeal EMG can help predict recovery. The presence of active motor unit potentials suggests a better chance of improvement, while their absence 6 months after injury indicates a poor prognosis (Munin et al., 2016; Wentland et al., 2016). However, its routine use in infants is limited due to the lack of reference values, variability in results, and the need for general anaesthesia (Wentland et al., 2016; Ysunza et al., 2007). A similar situation exists in neonatal foals, as no EMG reference values are currently available for this species (van Wessum et al., 1999; Wijnberg et al., 2004).

CONCLUSION

Although uncommon compared to other species, laryngeal paralysis can occur in newborn foals and should be considered as a differential

diagnosis for respiratory distress and stridor. Although a definitive cause could not be confirmed here, neonatal encephalopathy and equine nutritional myodegeneration were considered the most likely underlying conditions. In this case, the paralysis appeared to be reversible without the need for invasive surgical intervention. Therefore, bilateral laryngeal paralysis should not necessarily be associated with a poor prognosis in neonatal foals, and early endoscopic evaluation combined with supportive care may improve outcomes in similar cases.

AUTHOR CONTRIBUTIONS

L. De Maré: Writing – original draft; conceptualization; investigation. **C. Loublier:** Writing – review and editing. **H. Amory:** Writing – review and editing; resources. **L. Lecoq:** Writing – review and editing; supervision. **C. Cesarini:** Supervision; writing – review and editing; conceptualization.

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CONFLICT OF INTEREST STATEMENT

No conflicts of interest have been declared.

ETHICS STATEMENT

No Ethical Approval was required for this case report. Written owner consent was obtained to use the patient's clinical data for scientific purposes.

ORCID

L. De Maré https://orcid.org/0009-0007-2298-2403
C. Loublier https://orcid.org/0000-0002-8442-2766
H. Amory https://orcid.org/0000-0002-2285-0319
L. Lecoq https://orcid.org/0000-0002-3373-3740
C. Cesarini https://orcid.org/0000-0002-4367-3497

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