Received: 12 April 2025 | Revised: 28 June 2025 | Accepted: 5 September 2025

DOI: 10.1002/vrc2.70208

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CASE REPORT

Companion or pet animals

Juvenile-onset bilateral laryngeal paralysis in a Hungarian Vizsla

Yolanda López Barroso | **William Robinson**

Willows Veterinary Centre & Referral Service, Solihull, UK

Correspondence
Yolanda López Barroso, Willows Veterinary Centre & Referral Service, Solihull, UK.
Email: Yolanda.lopezbarroso@willows.uk.net

Abstract

A 13-month-old entire male Hungarian Vizsla was referred with a 6-week history of non-productive cough and exercise intolerance, primarily during and after walks. Physical and neurological examinations were unremarkable; however, severe stridor and inspiratory dyspnoea were noted after minimal exertion, resolving promptly with rest. Complete blood count, serum biochemistry, thyroid function tests (total free thyroxine and thyroid-stimulating hormone) and an in-clinic antigen test for *Angiostrongylus vasorum* were within normal limits. Computed tomography of the head, neck and thorax did not identify any abnormalities. Laryngeal function assessment under light anaesthesia revealed bilateral laryngeal paralysis. Surgical management via unilateral cricoarytenoid lateralization resulted in marked clinical improvement, with only intermittent coughing reported at a 1-year follow-up. This report describes a case of juvenile-onset laryngeal paralysis in a Hungarian Vizsla and underscores the importance of including this condition in the differential diagnosis for young dogs presenting with signs of upper airway obstruction.

BACKGROUND

Laryngeal paralysis is defined as the failure of the arytenoid cartilages, and consequently the vocal folds, to actively abduct during inspiration, resulting in upper airway obstruction.[1,2] Both congenital and acquired forms of canine laryngeal paralysis (LP) have been extensively reported in the literature. A juvenile-onset laryngeal paralysis-polyneuropathy complex (LP-PNC) has been described in canine breeds, such as Dalmatian, Rottweiler, American Staffordshire Terrier, Pyrenean mountain dog, white-coated German Shepherd and Miniature Schnauzer.[3–8]

In certain canine breeds (e.g., Greyhound, Rottweiler, Leonberger, Saint Bernard, Alaskan sled dogs, Alaskan Malamute, Black Russian Terrier, Bull Terrier, and Miniature Schnauzer) affected by inherited polyneuropathy, a specific gene mutation associated with LP has been identified. [8–16] Congenital mononeuropathy of the recurrent laryngeal nerves has also been recognized in Alaskan husky dogs.[17] Additionally, the disease has been documented in young dogs of other breeds, either in isolation or in conjunction with other neurological abnormalities, including Bouvier des Flandres, Tibetan Mastiff, Siberian Husky, Cocker Spaniel, Miniature Pinscher and English Springer Spaniel.[18–21]

This report describes a case of juvenile-onset LP in a Hungarian Vizsla.

CASE PRESENTATION

A 13-month-old entire male Hungarian Vizsla was referred with a 6-week history of non-productive cough and exercise intolerance, primarily during and after walks. The cough had first appeared 6 weeks before referral and was primarily observed during and after walks. A 1-week course of meloxicam (Metacam, Boehringer Ingelheim; at 0.1 mg/kg, administered orally once daily) did not result in any improvement. Exercise intolerance, characterized by reduced tolerance to brief periods of physical activity, had acutely developed and worsened over the 2 weeks preceding presentation.

Investigations performed at the referring veterinary clinic included a complete blood count (CBC), serum biochemistry and three-view thoracic radiographs, all of which were unremarkable. A rapid in-clinic antigen test for *Angiostrongylus vasorum* (Angio Detect Test; IDEXX) yielded a negative result.

On physical examination, the patient was bright, alert and responsive. No respiratory abnormalities were noted at rest. However, severe stridor and inspiratory dyspnoea were observed after a few meters of trotting, resolving promptly upon cessation of exercise. Neurological examination did not reveal any abnormalities.

DIFFERENTIAL DIAGNOSIS

Physical examination was highly suggestive of upper respiratory tract obstruction, with a focus on the larynx. The main differential diagnoses included acquired or congenital unilateral or bilateral LP. Laryngeal collapse, laryngeal or pharyngeal neoplasia, laryngeal or pharyngeal foreign body and epiglottic retroversion were also considered.

INVESTIGATIONS

The dog was premedicated with butorphanol (Torbugesic, Zoetis; 0.3 mg/kg) administered intravenously (IV). A light plane of anaesthesia was induced using propofol (Propofol,

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Braun), given IV slowly to effect. A laryngeal function examination revealed bilateral LP, characterized by paradoxical medial movement of both arytenoid cartilages during inspiration. The dog was then maintained under anaesthesia with isoflurane (Isoflo, Zoetis) delivered in 100% oxygen.

A computed tomography (CT) scan of the head, neck and thorax ruled out underlying causes of LP (e.g., cervical or cranial mediastinal masses) and comorbidities associated with generalized polyneuropathy (e.g., megaoesophagus or tracheal hypoplasia). Serum total free thyroxine (T4) and thyroid-stimulating hormone (TSH) levels were within normal ranges (T4: 21.4 nmol/L, reference range: 13.5–50 nmol/L; TSH: 0.08 ng/mL, reference range: <0.60 ng/mL). Electrodiagnostic testing was offered to assess peripheral nerve function, but was declined by the owner.

LEARNING POINTS/TAKE-HOME MESSAGES

- Congenital laryngeal paralysis is considered in young dogs with upper airway obstruction.
- Hungarian Vizsla can now be added to the list of breeds potentially affected by this condition.
- Bilateral congenital laryngeal paralysis can occur in the absence of generalized polyneuropathy.
- Surgical treatment can provide excellent long-term outcomes in cases of juvenile-onset laryngeal paralysis without associated polyneuropathy.

TREATMENT

A unilateral left-sided cricoarytenoid lateralization was performed 1 week after diagnosis. The patient was premedicated with methadone (Comfortan, Dechra; 0.2 mg/kg IV) and medetomidine hydrochloride (Sedator, Dechra; 0.004 mg/kg IV). General anaesthesia was induced with propofol (Propofol, Braun) given IV to effect and maintained with isoflurane (Isoflo, Zoetis) in 100% oxygen.

The dog was positioned in left lateral recumbency, and a skin incision was made just dorsal to the linguofacial vein. After blunt dissection through the subcutaneous tissue and platysma muscle, the thyropharyngeus muscle was separated along its fibre pattern. A stay suture was placed through the wing of the thyroid cartilage to maintain ventrolateral retraction. The cricoarytenoideus dorsalis muscle was dissected near its insertion at the muscular process, and the cricoarytenoid joint surface was exposed. Two cricoarytenoid sutures of 2-0 polypropylene (Prolene, Ethicon) were placed, and adequate abduction was confirmed upon extubation.

OUTCOME AND FOLLOW-UP

Postoperative analgesia was provided with buprenorphine (Vetergesic, CEVA; 0.02 mg/kg IV every 8 hours) and meloxicam (Metacam, Boehringer Ingelheim; 0.2 mg/kg loading dose SC, followed by 0.1 mg/kg PO every 24 hours for 7 days). The patient recovered uneventfully in the intensive care unit and was discharged the following day with instructions for strict rest and a soft food diet (shaped into meatballs) for 2 weeks, after which a gradual return to normal activity was advised.

Follow-up assessments were conducted via telephone at 2 and 4 weeks postoperatively. The owner reported marked clinical improvement, with no further episodes of respiratory distress; however, an intermittent non-productive cough persisted.

At the 12-month follow-up, the patient returned for a scheduled physical and neurological examination, both of which were unremarkable. The dog was able to perform normal levels of physical activity without respiratory distress or stridor. The frequency of coughing had significantly decreased but remained intermittently present.

DISCUSSION

This report describes a case of suspected bilateral juvenile-onset LP in a young Hungarian Vizsla. Laryngeal paralysis should be considered in the differential diagnosis for any young patient presenting with inspiratory stridor, hoarseness and/or exercise intolerance.[20]

Contraction of the cricoarytenoideus dorsalis muscle induces abduction of the arytenoid cartilages during inspiration. The motor supply to this muscle is provided by the caudal laryngeal nerve, the terminal branch of the recurrent laryngeal nerve, which originates from the vagus nerve in the thorax.[22] The vagus nerve itself arises from the caudal nucleus ambiguous in the medulla. Damage to or disease affecting any level of the cricoarytenoideus dorsalis muscle's motor supply, or the muscle itself, can result in LP.[1]

In patients suspected of having LP, a thorough physical and neurological examination, along with a CBC, biochemical profile, thoracic radiographs and thyroid function screening, is essential to rule out underlying disease or concomitant comorbidities.[1] A definitive diagnosis of LP, however, requires direct visualization of the larynx to confirm the absence of arytenoid abduction during inspiration.[20]

A thorough understanding of laryngeal anatomy and its physiological movement is essential for an accurate diagnosis. Anaesthetic drugs can cause transient suppression of laryngeal motion, leading to apparent dysfunction that may mimic true LP.[20] A recent systematic review on the effects of anaesthetic drugs on laryngeal function found that most studies showed no significant differences in laryngeal motion between various sedatives, such as butorphanol, acepromazine and dexmedetomidine, or between induction drugs such as alfaxalone, propofol and thiopental.[23] However, since most of the dogs in these studies were healthy, it is important to recognize that the impact of anaesthetic drugs on laryngeal movement may be more pronounced in dogs with LP.[23] Therefore, patients suspected of having LP should be evaluated as they approach a near-conscious state, regardless of the anaesthetic protocol used.

Doxapram has been widely evaluated and used as a respiratory stimulant and has been shown to aid in distinguishing dogs with LP from those without, with no significant adverse effects reported in healthy individuals.[23] Given the potential for paradoxical laryngeal motion and increased airway resistance in affected patients, it is recommended that facilities for

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prompt endotracheal intubation are readily available during doxapram administration.[24]

Misinterpretation of paradoxical arytenoid movement as normal function may lead to an underdiagnosis of LP. In paradoxical motion, the arytenoid cartilages are drawn into the rima glottidis during inspiration due to the negative intraglottic pressure generated by breathing against an obstruction. During expiration, the passive phase of the respiratory cycle, the cartilages return to their original position, giving the appearance of abduction.[20] In the case presented here, the diagnosis was made based on the evidence of paradoxical movement observed during the laryngeal function evaluation.

Juvenile-onset LP-PNC is a condition where LP occurs alongside other neurological defects, such as gait abnormalities and megaoesophagus. While most dogs present for evaluation during their first year of life,[2–7] in Leonbergers, the age of onset can range from 1 to 9 years.[25] Similarly, in Alaskan Malamute dogs, clinical signs have been reported up to 19 months of age.[26] The majority of dogs with juvenile-onset LP-PNC exhibit a combination of respiratory signs (e.g., inspiratory stridor, cyanosis, syncopal episodes, gagging or coughing) and neurological signs (e.g., megaoesophagus, hyporeflexia, paresis, muscle atrophy, muscle fasciculations or hypermetria) at the time of presentation.

However, signs of neurological dysfunction may also appear before or after the onset of LP. A recent study evaluating the disease in American Staffordshire Terrier dogs found that some dogs experienced a delay of up to 3 months before developing locomotor defects.[5]

Electrodiagnostic testing is routinely performed as part of the diagnostic work-up for dogs suspected of having either congenital or acquired LP to rule out a polyneuropathy—juvenile-onset LP-PNC in congenital cases and generalized progressive idiopathic polyneuropathy in acquired cases.[1] Although further electrodiagnostic evaluation was offered, it was declined by the owners of the dog described in this case. However, the absence of associated neurological signs both at the time of presentation and at the 1-year follow-up makes an association with a generalized neuropathy less likely.

Determining the precise onset of LP in this patient remains challenging, as early unilateral paresis is frequently subtle and can be difficult to detect. While it is possible that the condition developed before 1 year of age—potentially classifying it as congenital—the authors consider juvenile-onset LP (without evidence of polyneuropathy) to be a more appropriate designation, given the age at which clinical signs first appeared and the relatively short duration of their progression.

The prognosis for dogs with congenital LP varies between breeds with recognized LP-PNC, but it is generally considered guarded to poor. In a case series of young Dalmatian dogs, all but one dog were either euthanized or died due to aspiration pneumonia.[3] Likewise, Rottweiler and Pyrenean mountain dogs have been reported to have a poor prognosis, with all affected dogs euthanized shortly after clinical signs were first noted due to the rapid progression and severity of the disease and the development of fulminant aspiration pneumonia.[4,6] In the American Staffordshire Terrier, the disease seems to have a slower progression, and the prognosis is considered guarded to fair.[5]

As demonstrated in the present case, anecdotal reports suggest that young dogs presenting with LP, but without additional neurological signs, generally have a favourable prognosis.[21,27] A similar pattern has been reported in Alaskan Husky dogs, where LP has been characterised as a mononeuropathy of the recurrent laryngeal nerve. In this population, all dogs that underwent surgical intervention showed significant postoperative improvement in both respiratory function and overall activity levels. Among those managed conservatively, approximately 40% exhibited spontaneous resolution of clinical signs between birth and skeletal maturity.[17]

To conclude, this report describes a case of suspected juvenile-onset LP in a young Hungarian Vizsla. The dog improved immediately after surgery, exercising normally at 1-month postoperatively and demonstrating only an intermittent cough at 1-year follow-up. LP-PNC was not suspected based on the clinical signs, physical and neurological examinations at both presentation and the 1-year follow-up. However, electrodiagnostic tests and/or histological analysis of muscle biopsies would be required to fully rule out a generalized neuropathy.

AUTHOR CONTRIBUTIONS

Yolanda López Barroso: drafted the manuscript and approved the final version to be published. William Robinson: reviewed the manuscript both grammatically and intellectually, and approved the final version to be published.

FUNDING INFORMATION

The authors received no specific funding for this work.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

ETHICS STATEMENT

The authors confirm the agreement from the pet owner in using the medical records of the patient with scientific purpose. The authors also state the patient described here was managed according to contemporary standard of care.

ORCID

Yolanda López Barroso https://orcid.org/0000-0003-0464-4447

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How to cite this article: Barroso YL, Robinson W. Juvenile-onset bilateral laryngeal paralysis in a Hungarian Vizsla. Vet Rec Case Rep. 2025;e70208. https://doi.org/10.1002/vrc2.70208

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