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CASE REPORT
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Jugular Foramen Syndrome in a Dog - Imaging Features

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ABSTRACT

***Background*:** Cranial nerves IX, X, and XI share a common anatomical pathway associated with the jugular foramen. Therefore, the peripheral nerve sheath tumors around the jugular foramen may lead to concurrent dysfunction of these nerves. However, peripheral nerve sheath tumors of cranial nerves IX, X, and XI are often challenging to diagnose due to their deep anatomical location and nonspecific clinical signs. This report describes a rare case of a peripheral nerve sheath tumor originating from cranial nerves IX, X, and XI, diagnosed through a combination of clinical signs and imaging findings from computed tomography (CT) and magnetic resonance imaging (MRI).

***Case*:** A 5-year-old, intact male, Spitz was referred with chronic vomiting, left head tilt, abnormal gait and suspicion of portosystemic shunt. On CT, multiple collateral vessels were observed. However, the association between collateral vessels and the current symptoms appeared to be low. Accordingly, a central nervous system disorder was suspected, and MRI was conducted to differentiate from intracranial disease. In MRI, the mass was well-demarcated, amorphous to round-shape, and was located at the left lateroventral region of the medulla oblongata, extending as a tubular lesion from the medial and caudal region of the left tympanic cavity to the jugular foramen and tympano-occipital fissure, which are the pathways for cranial nerves IX, X, and XI. The mass appeared hypointense to isointense on T1-weighted images (T1W), isointense to hyperintense on T2-weighted images (T2W), and uniformly hyperintense on fluid-attenuated inversion recovery (FLAIR) sequences, with strong peripheral rim enhancement. On retrospective CT evaluation, severe atrophy of the sternocephalic, cleidocephalic, and trapezius muscles ipsilateral to the tumor was also observed. These clinical signs and imaging findings are consistent with those typically observed in peripheral nerve sheath tumors originating from cranial nerves IX, X, and XI.

***Discussion*:** The presumptive diagnosis of a peripheral nerve sheath tumor originating from cranial nerves IX, X, and XI was supported by four key findings: characteristic morphology and location of the mass, its signal intensity on MRI, neurogenic muscle atrophy, and clinical signs consistent with jugular foramen syndrome. The tubular shape reflected the endoneurial longitudinal spread of peripheral nerve sheath tumors, with smooth widening of the jugular foramen and extension through the tympano-occipital fissure along the expected anatomical pathway of these nerves. On MRI, the lesion was isointense to hypointense on T1W, hyperintense on T2W and FLAIR, and showed peripheral rim enhancement, suggestive of a malignant peripheral nerve sheath tumor. Marked neurogenic atrophy of cervical and shoulder muscles innervated by XI cranial nerve were more readily appreciated on CT than MRI, because field of view on MRI is limited. Clinically, chronic vomiting was presumed to be associated with dysfunction of cranial nerves IX and X, while muscle atrophy strongly suggested cranial nerve XI involvement. These findings are consistent with jugular foramen syndrome, in which lesions around the jugular foramen cause concurrent deficits in cranial nerves IX, X, and XI. In this dog, head tilt and abnormal gait were likely due to both direct neurologic injury and secondary neurogenic muscle atrophy. Although histopathological confirmation and laryngeal examination were not performed, the combination of imaging findings and compatible neurological signs strongly supported a presumptive diagnosis of peripheral nerve sheath tumors. When histological confirmation is not feasible, combined MRI and CT are valuable for assessing anatomical relationships and imaging features to support the presumption of the tumor's origin.

Keywords: peripheral nerve sheath tumor, PNST, jugular foramen syndrome, JFS, CT, MRI.

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INTRODUCTION

Peripheral nerve sheath tumors (PNSTs) are neoplasms arising from Schwann cells, perineurial cells, or intraneural fibroblasts [12]. Among the cranial nerves (CNs), PNSTs are most commonly reported in CN V (trigeminal nerve), although they have also been rarely documented in CNs IX (glossopharyngeal nerve), X (vagus nerve), XI (accessory nerve), and XII (hypoglossal nerve) [5,6,12]. When PNSTs originate from CNs IX, X, and XI, they often affect all three nerves simultaneously, resulting in mixed neurological deficits [5,7,9], because these three nerves share a common anatomical pathway associated with the jugular foramen [1,3,7]. Functionally, they innervate the pharynx, larynx, esophagus, and several muscles of the neck and shoulder [1,12]. Therefore, a tumor around the jugular foramen may lead to concurrent dysfunction of these structures [5,7,9]. Lesions around the jugular foramen that affect the combined function of CNs IX, X, and XI may result in complex clinical signs referred to as 'jugular foramen syndrome', or 'Vernet syndrome' in human medicine [2,9-11].

In cases of PNST, magnetic resonance imaging (MRI) is useful in identifying the lesion and assessing nerve involvement [2,11]. Nevertheless, in some cases, only nerve thickening without a distinct mass formation may be observed, requiring careful interpretation [11]. Computed tomography (CT) can complement MRI by providing additional diagnostic clues [7,9].

This report describes a rare case of a PNST originating from CNs IX, X, and XI, diagnosed through a combination of clinical signs and imaging findings from CT and MRI.

CASE

A 5-year-old intact male Spitz dog was referred for CT by a local clinic due to mild elevation in liver enzymes (ALT: 102 IU/L; reference interval [RI]: 17-78, ALP: 267 IU/L; RI: 47-254), increased serum ammonia (157 μmol/L; RI: 0-9), and a suspected portosystemic shunt. At the time of presentation, the patient showed chronic vomiting, a left head tilt, and abnormal gait. However, the serum ammonia was within the reference interval (6 μmol/L; RI: 0-9), and the bile acid level was only mildly elevated (26 μmol/L; RI: 0-25). No other significant abnormalities were noted on blood analysis. In gait evaluation, the dog exhibited a subtle left head tilt and tended to lean to the leftward while walking, resembling vestibular syndrome. Mentation was normal, and no other remarkable findings were detected on neurological examination. Radiographs and abdominal ultrasound revealed no significant findings.

Multiphase CT[1] [Aquilion Start - 1.0-mm slice thickness, 120 kVp, 150 mAs, 512 x 512 matrix, and 0.75 s/rotation] was performed. Although multiple collateral vessels were identified between the spleen and the renal vein, the diameter of the portal vein appeared to be within normal limits. When interpreted in conjunction with the clinical signs and bloodwork results, the findings were considered unlikely to be related to the patient's current clinical signs. Accordingly, a central nervous system disorder was suspected, and MRI was conducted to assess for intracranial disease. MRI[2] examinations [SIGNA™ Creator] of the head were performed using a 1.5 Tesla unit. The following sequences were obtained: T2-weighted images (T2W), fluid-attenuated inversion recovery (FLAIR) and pre-and post-contrast [intravenous injection of 0.1 mmol/kg of gadoterate meglumine] T1-weighted images (T1W). The MRI revealed a well-demarcated, amorphous to round shape, approximately 10.4 x 12.3 x 10.6mm (width x height x length) mass located at the left lateroventral region of the medulla oblongata (Figure 1). The lesion appeared hyperintense on T2W and FLAIR sequences (Figure 1 A & B), hypointense on T1W images (Figure 1C), and showed overall moderate enhancement, with more pronounced peripheral enhancement (Figure 1D). Notably, the mass extended caudally and medially through the jugular foramen and tympano-occipital fissure, forming a tubular-shaped lesion. The lesion extended extracranially into the caudomedial aspect of the tympanic cavity, located between the longus capitis and digastric muscles, and dorsal to the external carotid artery. The extracranial portion of the mass exhibited a well-demarcated, oval shape, measuring approximately 9.50 x 12.4 x 14.0 mm (width x height x length) and displayed similar enhancement characteristics to the intracranial portion of the mass (Figure 1 E & F). Given the lesion's anatomical location and imaging characteristics, a PNST originating from CNs IX, X, and XI was strongly suspected.

Based on these MRI findings, the CT images were re-evaluated retrospectively. The mass showed similar morphology to that observed on MRI. The intracranial portion appeared well-defined with an amorphous to oval shape, iso-attenuated to adjacent

brain parenchyma (39.86-59 Hounsfield Unit[s] [HU]), and mildly enhanced after contrast administration (59.43-89.14 HU). The extracranial portion appeared well-defined, oval-shaped, and homogeneous, with slight contrast enhancement (43 HU pre-contrast, 53 HU post-contrast) and peripheral rim enhancement up to 128 HU (Figure 2A). In addition, smooth widening of the left jugular foramen, relative to the contralateral side, and marked lysis of the tympanic part of the left temporal bone were evident (Figure 2 B). Also, severe unilateral muscle atrophy was noted on the left side, ipsilateral to the location of the mass. The left thyroarytenoid and cricoarytenoid muscles, components of the vocal fold apparatus, were atrophic, resulting in marked asymmetry. Further muscle atrophy was observed in the left sternocephalic muscle, cleidocephalic muscle (both cervical and mastoid parts), and trapezius muscle (Figure 2 C-F). These findings strongly support the involvement of adjacent cranial nerves, either through direct invasion or compression by the mass.

In summary, based on the identification of a mass at the level of the jugular foramen on MRI; the observation of unilateral muscle atrophy on the same side as the mass on CT; and the presence of chronic vomiting, head tilt, and abnormal gait, the lesion was diagnosed as a PNST originating from CNs IX, X, and XI.

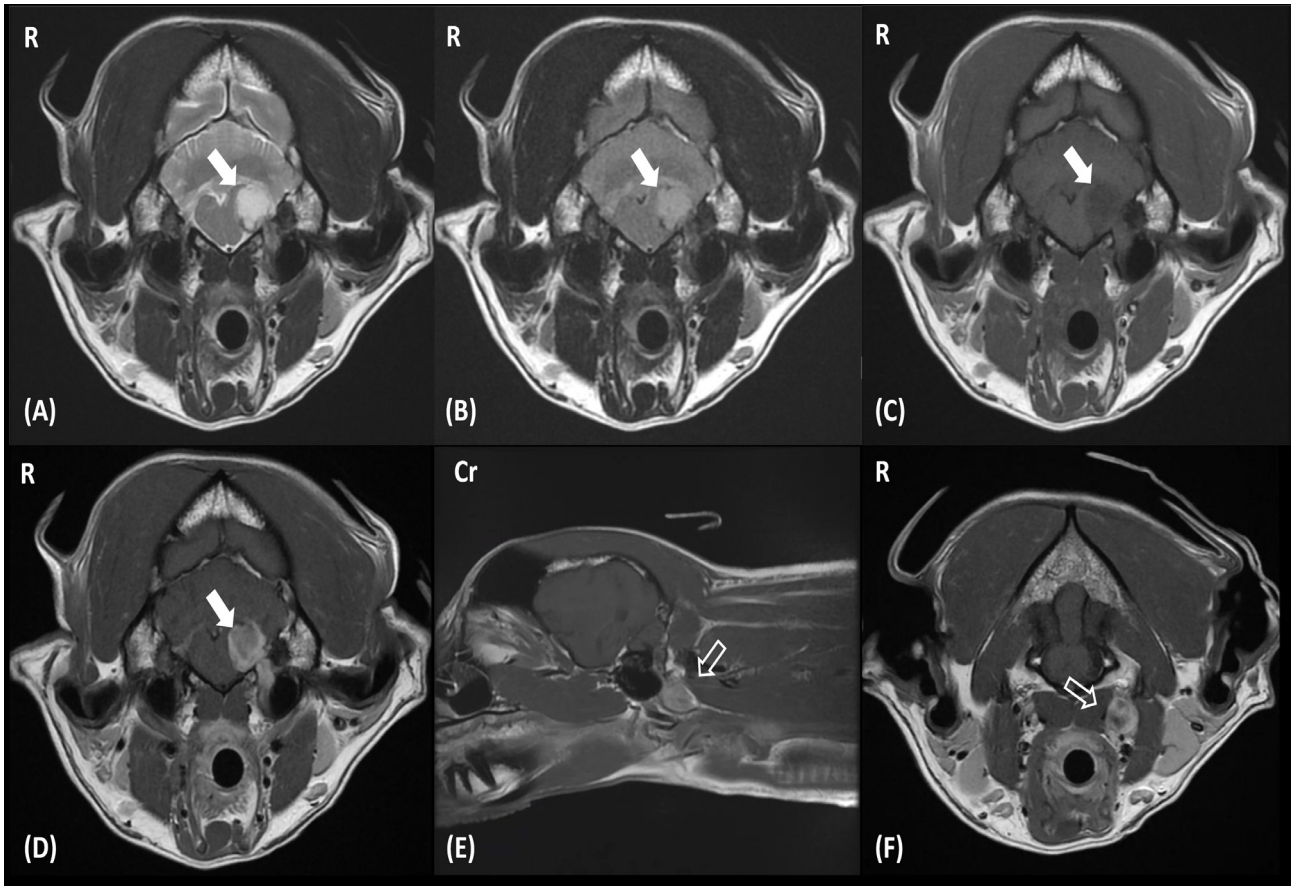

Figure 1. Magnetic resonance imaging of the intracranial and extracranial lesion. Left side corresponds to the right side of the patient. A well-demarcated mass (white arrows) was located at the left lateroventral region of the medulla oblongata and extended caudally and medially through the jugular foramen and tympano-occipital fissure, forming a tubular-shaped lesion. The lesion appeared hyperintense on T2-weighted (A) and FLAIR (B) images, hypointense on T1-weighted images (C), and showed overall moderate enhancement with more pronounced peripheral enhancement on post-contrast T1-weighted images (D-F). The extracranial portion of the mass (open white arrows) exhibited a well-demarcated, oval shape and displayed similar enhancement characteristics to the intracranial portion of the mass (E & F).

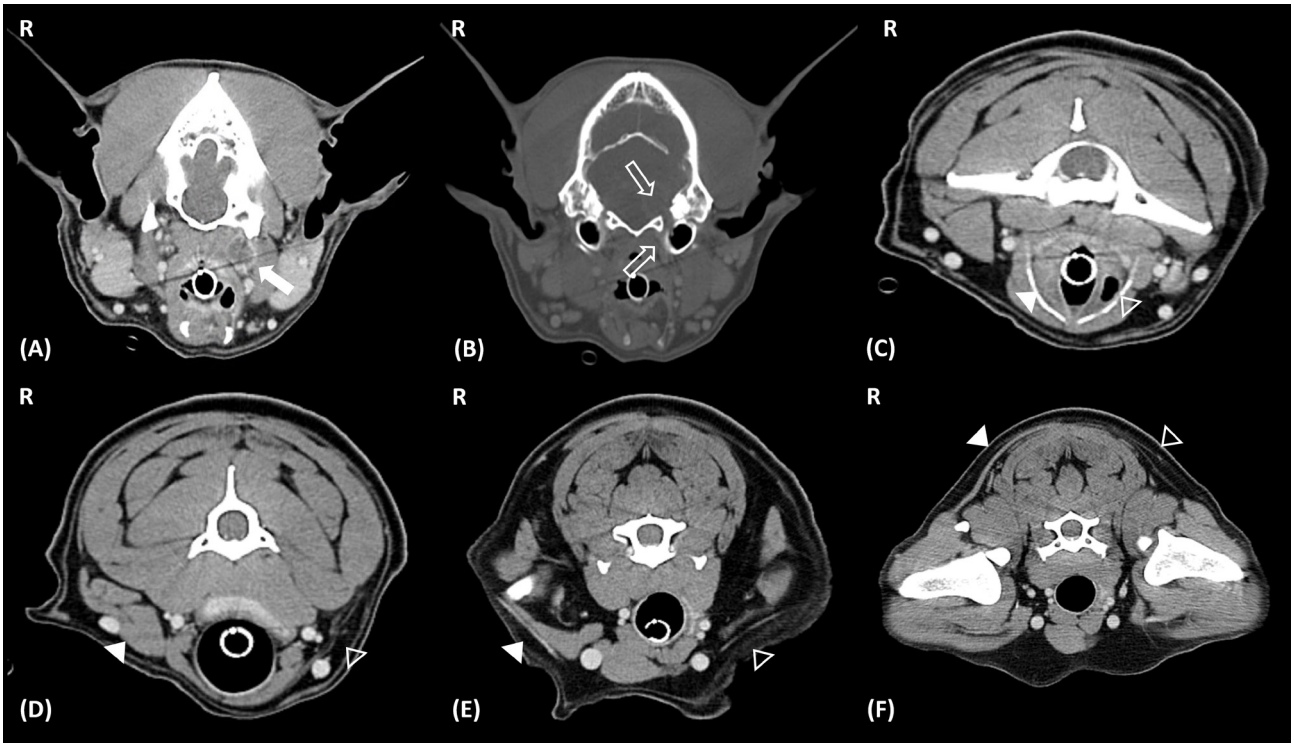

Figure 2. Computed tomography (CT) images of the skull and muscle atrophy. Left side corresponds to the right side of the patient. A- Transverse post-contrast CT image (soft tissue window). The extracranial lesion was well-defined, relatively oval-shaped, and slightly enhanced (white arrow). A rim-enhancing pattern (up to 128 HU) was also observed. B- Transverse post-contrast CT image (bone window). It demonstrates smooth widening of the left jugular foramen and marked osteolysis of the tympanic part of the left temporal bone (open white arrows) compared to the contralateral side. C- Vocal fold (Thyroarytenoid muscle, Cricoarytenoid muscle). D- Sternocephalic muscle. E- Cleidocephalic muscle (mastoid part). F- Trapezius muscle. Severe unilateral muscle atrophy is evident on the left side, ipsilateral to the location of the mass. [Normal muscles: white arrowheads, atrophy of muscles: open white arrowheads].

DISCUSSION

In this case, the presumptive diagnosis of a PNST originating from CNs IX, X, and XI was supported by 4 key findings: characteristic morphology and location of the mass, its signal intensity on MRI, neurogenic muscle atrophy, and the clinical signs consistent with jugular foramen syndrome.

PNSTs may appear either round or tubular in shape, with the tubular form being more commonly observed due to its origin [9,11]. This tubular shape reflects the characteristic endoneurial longitudinal spread of PNSTs, where the tumor extends along the affected nerve. This pattern is a distinctive feature from non-neurogenic tumors, which more often exhibit a transperineurial growth pattern [9,11]. In addition, CNs IX, X, and XI originate from the medulla oblongata (CN XI also arises from the motor neurons of the cervical spinal cord segments, typically from C1 to C7), and exit the cranial cavity together through the jugular foramen and tympano-occipital fissure [1,3]. Thus, when a mass arises intracranially around the jugular foramen along these nerves, widening of the foramen may be observed. According to previous reports, this change results from pressure-induced bony change caused by the expanding mass [7,9]. In this case, smooth widening of the jugular foramen was observed, with the lesion extending in a tubular form through the jugular foramen and tympano-occipital fissure, following the expected anatomical pathway of these three cranial nerves.

On MRI, the signal intensity of PNSTs typically appear hyperintense on T2W and FLAIR images, and isointense to hypointense on T1W [11]. On post-contrast T1W, the enhancement pattern varies according to tumor grade: low-grade PNSTs tend to show homogeneous enhancement, whereas high-grade PNSTs more often exhibit heterogeneous or peripheral rim enhancement [11]. These imaging characteristics are not only useful in supporting the diagnosis of PNST, but may also provide valuable insights into the tumor's biological behavior and grade. Based on these MRI characteristics, the lesion in this case was

considered highly suggestive of a malignant PNST, as it demonstrated typical features of PNST along with peripheral rim enhancement.

Each cranial nerve performs specific and distinct functions. CN IX innervates the pharynx to facilitate swallowing (in conjunction with CN X), supplies parasympathetic fibers to certain salivary glands, and conveys taste sensation from the caudal 3rd of the tongue [1]. Its function can be evaluated clinically by assessing the gag reflex and monitoring signs of dysphagia [14]. CN X innervates the pharynx, larynx, and esophagus, and is likewise tested via the gag reflex [1,14]. CN XI provides motor innervation to several cervical muscles, including the sternocephalic, cleidocephalic, trapezius, and omotransverse muscle [1]. When this nerve is infiltrated or compressed by a mass such as PNSTs, significant neurogenic atrophy of these muscles may be observed [2,7,9]. Neurogenic muscle atrophy, as in this case, is more readily appreciated on CT compared to MRI. This is due to the limited field of view and resolution constraints of MRI when attempting to image from head to shoulder regions simultaneously. Therefore, if a mass is identified near the jugular foramen on MRI, additional CT examination to assess the innervated cervical and shoulder musculature for signs of atrophy can provide valuable diagnostic support. Conversely, in cases where only CT has been performed and no obvious intracranial lesion is detected, the presence of unilateral cervical or shoulder muscle atrophy should raise suspicion for cranial nerve pathology as well as a primary musculoskeletal disorder.

Jugular foramen syndrome (JFS) refers to the simultaneous dysfunction of CNs IX, X, and XI due to a lesion around the jugular foramen, through which these nerves course together [4,13]. In humans, JFS presents with clinical signs such as dysphonia (CN X), dysphagia (CNs IX and X), loss of taste and sensation in the posterior one third of the tongue (CN IX), and weakness of the shoulder muscles (CN XI) [4,9]. Similar to humans, affected dogs may show upper respiratory tract noise and dysphagia, in addition to coughing, retching, gagging, vomiting, hypersalivation, lip smacking, and ipsilateral shoulder drop [4,7,9]. In this case, chronic vomiting was a prominent clinical sign and is presumed to be associated with dysfunction of CNs IX and X. In addition, marked muscle atrophy observed on imaging strongly suggests involvement of CN XI. The patient's clinical signs, including head tilt and abnormal gait, are likely the result of both direct neurologic injury caused by the intracranial lesion and secondary neurogenic muscle atrophy affecting postural and cervical muscles.

In this case, a presumptive diagnosis of PNST was considered highly reasonable based on the overall findings; however, several limitations remain. First, histopathological confirmation of the lesion was not obtained due to the anatomical location of the mass and the owner's decision, which precluded surgical intervention or biopsy. As a result, the diagnosis of PNST involving CN IX, X, and XI remains presumptive, based on imaging characteristics and associated clinical signs. Second, laryngeal examination was not performed, which limited the assessment of potential dysfunction of cranial nerves IX and X, such as laryngeal paralysis or dysphagia [8,14]. Despite these limitations, the lesion's imaging features combined with compatible neurological signs strongly support a presumptive diagnosis of a cranial nerve PNST. Complementary MRI and CT imaging are valuable tools for anatomical localization and characterization in cases where histological confirmation is not feasible.

In conclusion, although histopathological examination is essential for determining the origin and malignancy of a tumor, a presumptive diagnosis of PNST originating from CNs IX, X, and XI can be made when imaging findings are interpreted in conjunction with secondary muscle atrophy and associated clinical signs. As in this case, if histopathological confirmation is not feasible due to the tumor's anatomical location or the owner's decision, combined use of MRI and CT can play a critical role in assessing the lesion's anatomical relationships and imaging characteristics, thereby supporting the presumption of its origin.

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REFERENCES

1 **Bordoni B., Reed R.R., Tadi P. & Varacello M. 2023.** Neuroanatomy, cranial nerve 11 (accessory). In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing [cited 2025 Jul 24]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK507722/.

2 **Cooper-Khan R.S., Frankovich A.N., Thompson C.A., Thomovsky S.A. & Lewis M.J. 2024.** Clinical findings and outcome in 30 dogs with presumptive or confirmed nerve sheath tumors. *Veterinary Sciences*. 11(5): 192. DOI:10.3390/vetsci11050192.

3 **Couturier L., Degueurce C., Ruel Y., Dennis R. & Begon D. 2005.** Anatomical study of cranial nerve emergence and skull foramina in the dog using magnetic resonance imaging and computed tomography. *Veterinary Radiology & Ultrasound*. 46(5): 375-383. DOI:10.1111/j.1740-8261.2005.00069.x.

4 **Das J.M. & Al Khalili Y. 2019.** Jugular foramen syndrome (Vernet). In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing [cited 2025 Jul 24]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK549871/.

5 **Giraud L., Lezmi S, Gomes E. & Cauzinille L. 2020.** A rare case of malignant vagus nerve sheath tumor presenting with multiple cranial nerve dysfunction in a dog. *Canadian Veterinary Journal*. 61(10): 1049-1054.

6 **Hidari K., Nakamoto Y., Sakurai K., Sakurai Y., Nibe K. & Nakamoto M. 2022.** Presumptive subcutaneous malignant peripheral nerve sheath tumor with intracranial invasion and osteolysis in the posterior fossa of a dog: A case report. *Frontiers in Veterinary Science*. 9: 977099. DOI:10.3389/fvets.2022.977099.

7 **Lluesma B., Whitley N.T. & Hughes J.R. 2024.** Computed tomographic features of canine intracranial and jugular foraminal masses involving the combined glossopharyngeal, vagus, and accessory nerve roots. *Veterinary Radiology & Ultrasound*. 65(3): 308-316. DOI:10.1111/vru.13359.

8 **Lux C.N., Archer T.M. & Lunsford K.V. 2012.** Gastroesophageal reflux and laryngeal dysfunction in a dog. *Journal of the American Veterinary Medical Association*. 240(9): 1100-1103. DOI:10.2460/javma.240.9.1100.

9 **Madden M., Liatis T., Llanos C., Dancer S., Alvarez P., Tayler S., Hardas A. & De Decker S. 2025.** Jugular foramen syndrome: Concurrent neurological deficits, advanced imaging findings, underlying diagnoses, and outcomes in 14 dogs (2016–2024). *Journal of Veterinary Internal Medicine*. 39: e70088. DOI:10.1111/jvim.70088.

10 **Monteiro F., Oliveira P., Peneda J. & Condé A. 2019.** Vernet syndrome: intracranial extension of a slow-growing mass. *BMJ Case Reports*. 12: e228039. DOI:10.1136/bcr-2018-228039.

11 **Morabito S., Specchi S., Di Donato P., Pollard D., Dennis R., De Risio L., Bacon N.J., Potamopoulou M., Rupp S., Corlazzoli D., Ribeiro J., Cozzi F., Jurina K., Cappello R., Mercuriali E., Beckmann K., Flegel T., Menchetti M., König F., Matiasek K. & Rosati M. 2023.** Relationship between magnetic resonance imaging findings and histological grade in spinal peripheral nerve sheath tumors in dogs. *Journal of Veterinary Internal Medicine*. 37(6): 2278-2290. DOI: 10.1111/jvim.16839

12 **Rossmeisl J.H. & Pancotto T.E. 2020.** Tumors of the nervous system. In: Vail D.M., Thamm D.H. & Liptak J.M. (Eds). *Withrow and MacEwen's Small Animal Clinical Oncology*. 6th edn. St. Louis: Elsevier, pp.653-685.

13 **Ruaux C., Champion C.P., Pemberton S. & Munday J.S. 2020.** Vernet's syndrome (jugular foramen syndrome) secondary to osteoma of the tympanic bulla in a young male dog. *Veterinary Record Case Reports*. 8: e001122. DOI: 10.1136/vetreccr-2020-001122.

14 **Johnson L.R. 2016.** Laryngeal structure and function in dogs with cough. *Journal of the American Veterinary Medical Association*. 249(2): 195-201. DOI:10.2460/javma.249.2.195.