

Trigeminal nerve destruction due to atypical malignant chemodectoma metastasis in a dog

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Introduction

Tumours of chemoreceptor organs (chemodectomas) are rare neoplasms primarily arising from the aortic and carotid bodies. Malignant chemodectomas often metastasize into the lungs, bronchial and mediastinal lymph nodes, liver, pancreas, and kidney. Multisector computed tomography (CT) and MRI are both considered gold standards in the imaging of head and neck chemodectomas in human medicine. Most common canine head and neck paragangliomas (HNPGs) are benign. Here we report an unusual case of canine malignant chemodectoma with a trigeminal nerve-associated clinical manifestation.

Material and methods

A 13-year-old female, intact Doberman admitted to the Companion Animal Clinic, AUTH, with keratoconjunctivitis sicca, left-sided Horner syndrome, and trigeminal nerve neuropathy (left temporal and masticatory muscle atrophy and facial paralysis) with loss of left eye sympathetic innervations (postsynaptic nerve fibers) (Fig.1). After further biochemical and diagnostic imaging investigation (MRI), the dog was euthanized due to persistent dysphagia and debilitation and submitted for necropsy, with the suspicion of trigeminal nerve tumour (proposed as nerve sheath tumor) and left tympanic bullae fluid accumulation (Fig.2). Tissue samples collected at necropsy, were stained with HE or Synaptophysin-, NSE (Neuron Specific Enolase)-, and S-100-specific immunohistochemistry (IHC).

Results

Grossly, two unilateral firm, encapsulated, smooth-surfaced, coarsely multilobulated, ovoid masses with largest diameters of 5.8 and 1.5 cm were found in the inferior dorsolateral cervical area. Suspected bone metastatic lesions, probably starting from the bone surrounding the distal part of the trigeminal nerve, were seen in the cranial cavity surface of the temporal bones (Fig.3). Also, aspiration pneumonia was noticed.

Both primary neoplastic and metastatic lesions showed histopathological and immunohistochemical features typical for chemodectoma. Round to polygonal neoplastic cells were separated by variable amounts of fibrovascular stroma into lobules, nests or packets. Capsule and vascular invasion were evident in both cervical masses (Fig.4). The tumor cells showed extensive and strong S-100-positivity. To a lesser extent tumor cells multifocally had weak to moderate NSE and Synaptophysin immunopositivity and were universally negative for Chromogranin A (Fig.5).

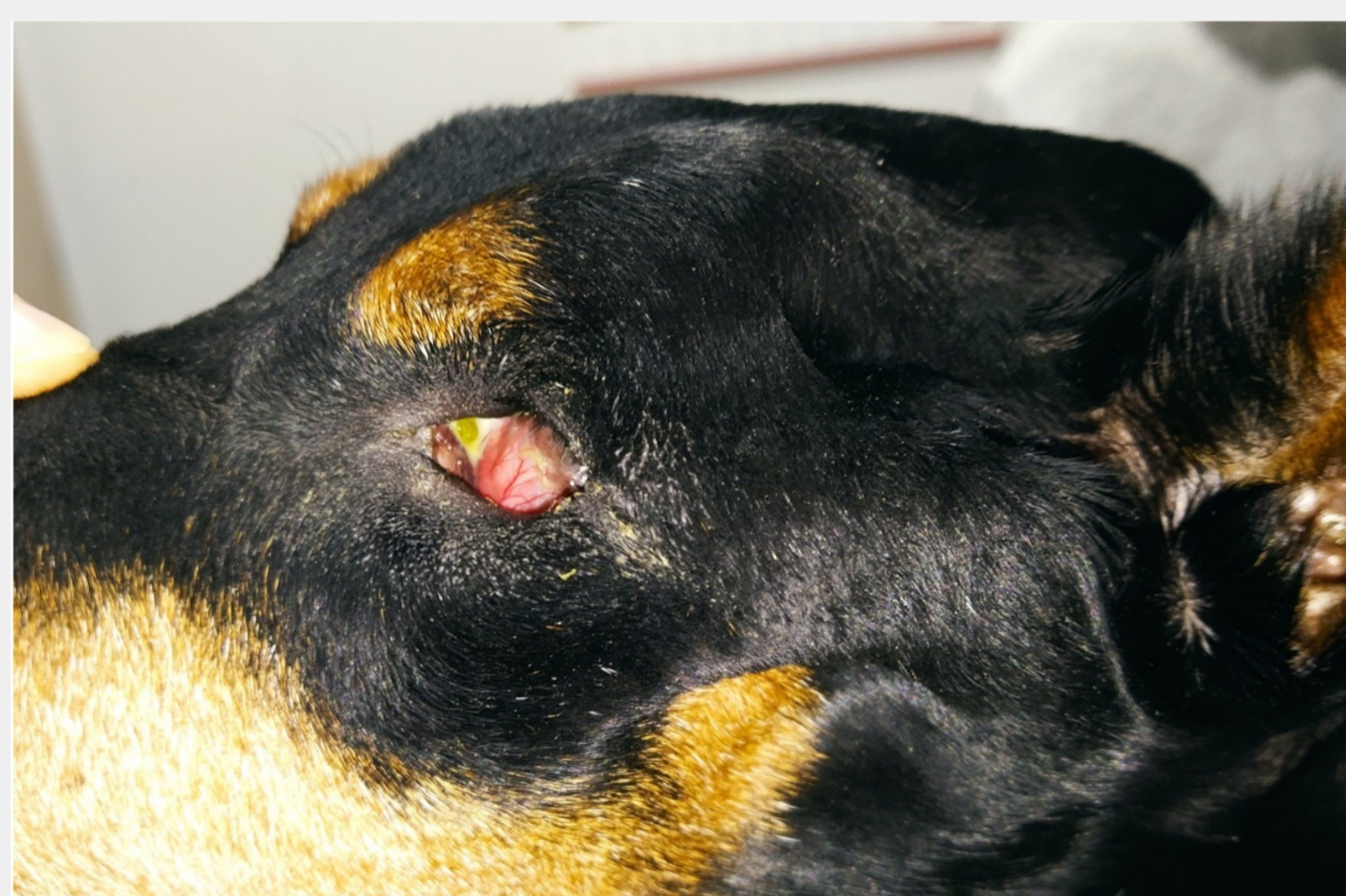


Fig.1: Temporal and masticatory atrophy, Horner's syndrome and Keratoconjunctivitis sicca in a 13-year-old female, intact Doberman.

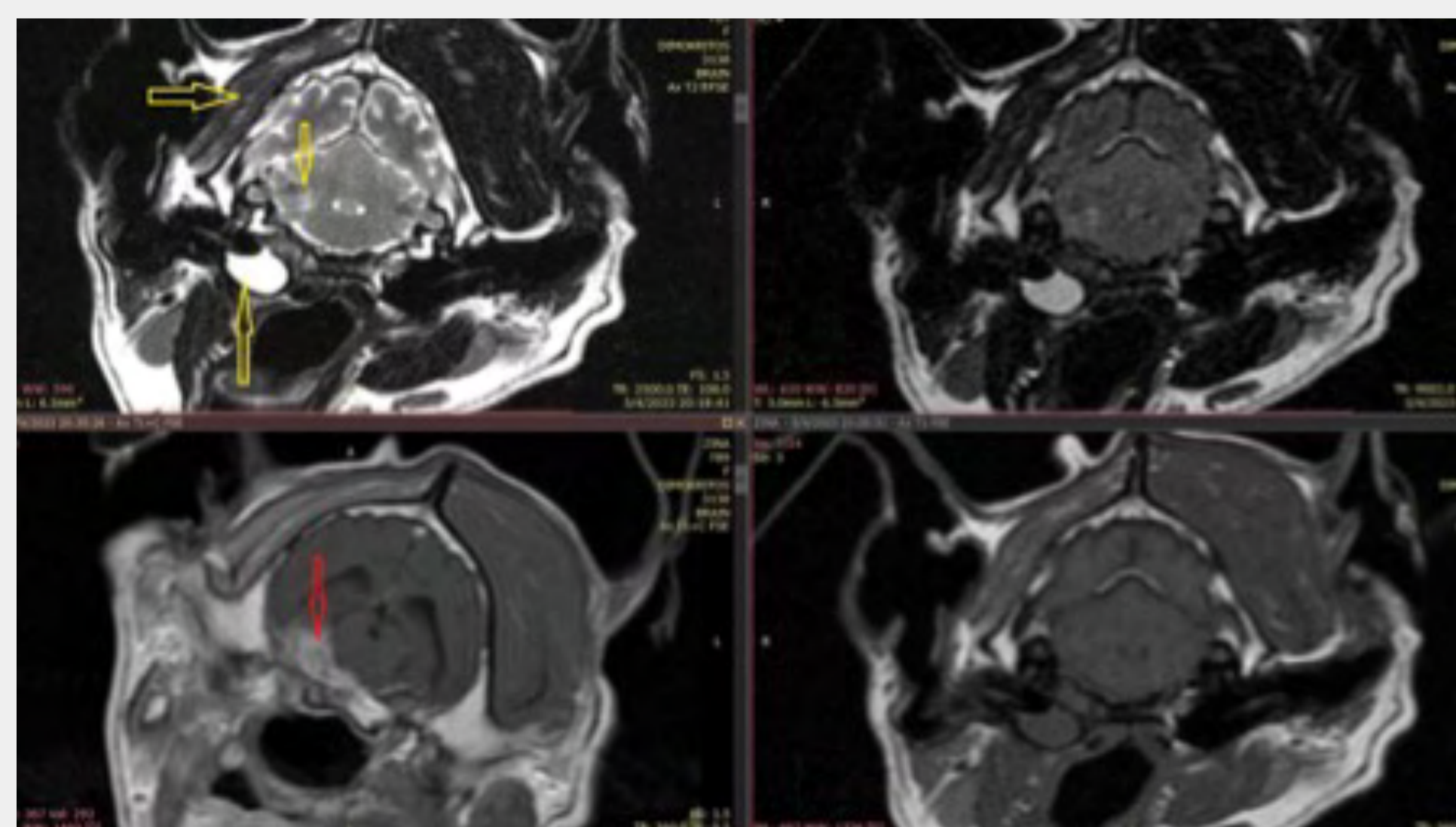


Fig.2: MRI of the head revealed temporal (horizontal yellow arrow), masticatory and pterygoid muscle atrophy, a trigeminal nerve mass (9mm- vertical yellow arrow-downward), which was strongly enhanced after contrast medium administration and a tympanic bulla fluid accumulation (vertical yellow arrow-upward).

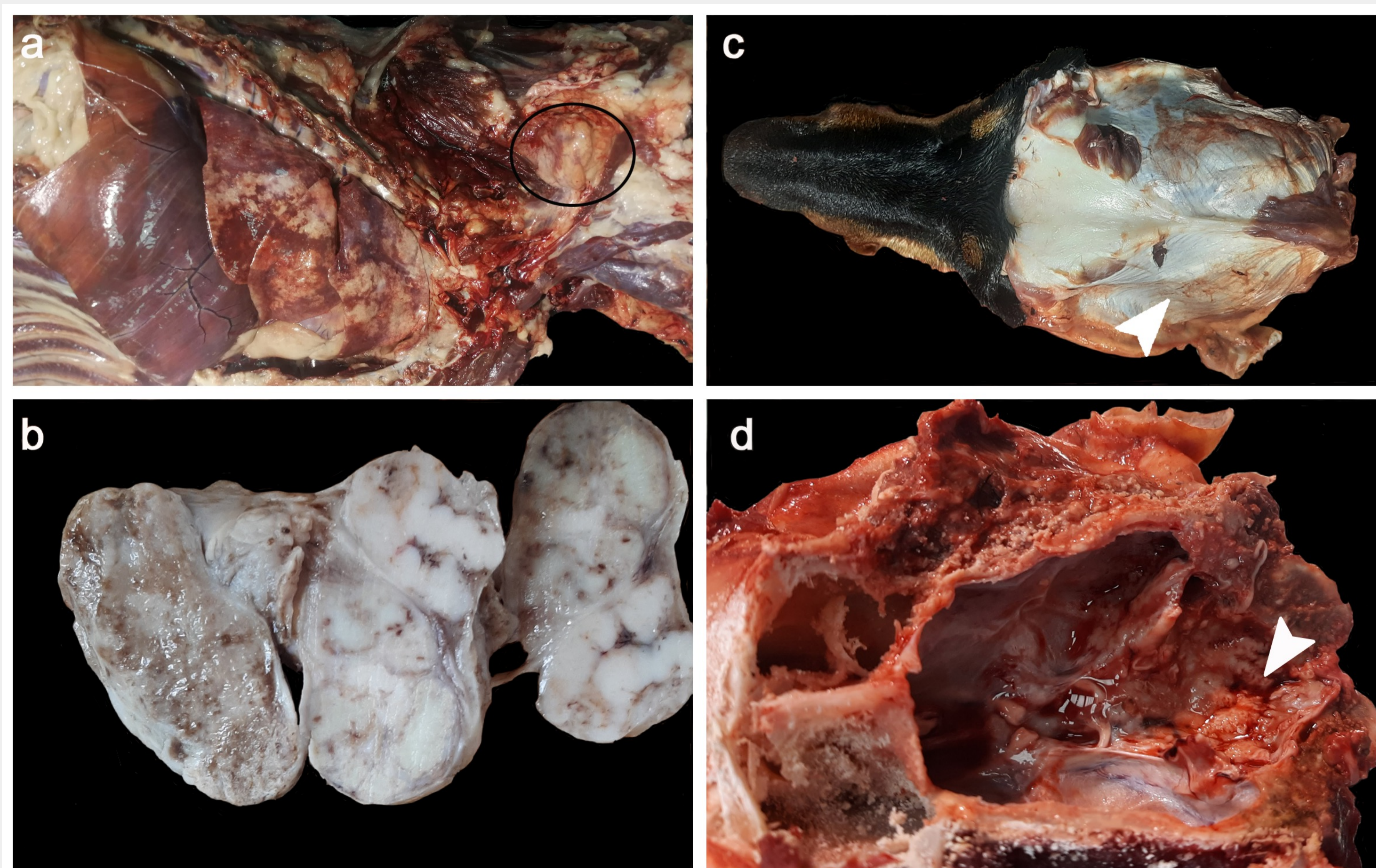


Fig.3: At necropsy the larger of the two masses found (in circle) measuring 5.8x2.5 cm located superficially on the subcutis of the inferior dorsolateral cervical area. The mass appears slightly distant from its presumptive location near the lower part of extrathoracic trachea due to upper right limb abduction b) Sectioning of the formalin fixed tumors revealed an off-white to gray, multilobulated cut surface with multifocal areas of necrosis. c) Severe left temporal muscle atrophy (arrow) d) The periosteal dura matter of inner skull has a roinely rugged surface (arrowhead) in the apex of the petrous temporal bone in the posterior medial part of the middle cranial fossa (trigeminal nerve entrance).

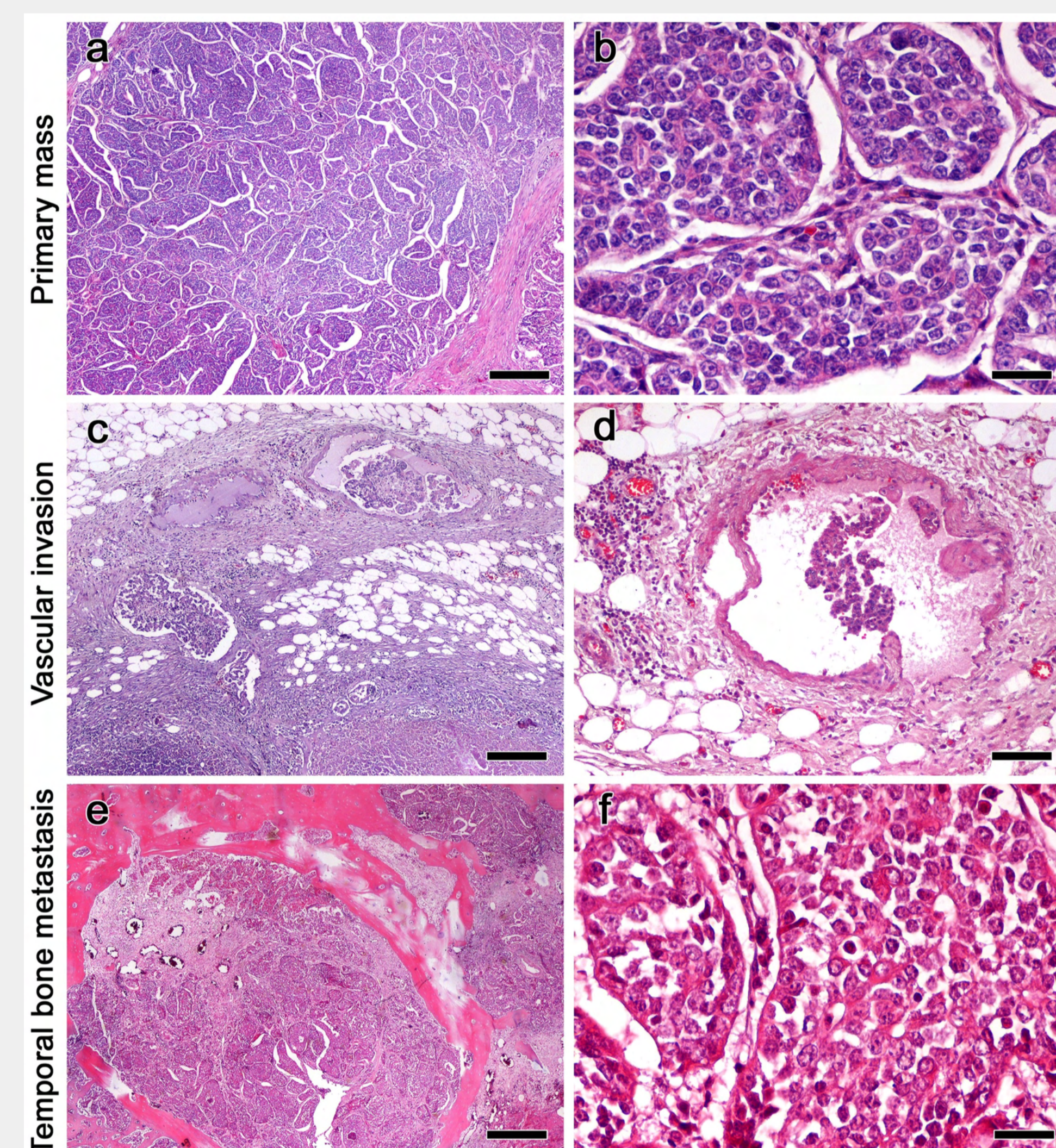


Fig.4: Histopathology of the primary tumors. a, b) Polygonal cells organized in nests and packets with fine fibrovascular stroma. c, d) Neoplastic emboli in the lumen of capsule and invasive front vessels. e, f) metastasis in the temporal bone with similar histomorphology as the primary tumor. H&E, Bar= 200 µm (a,c,d,e), 50µm (b,f)

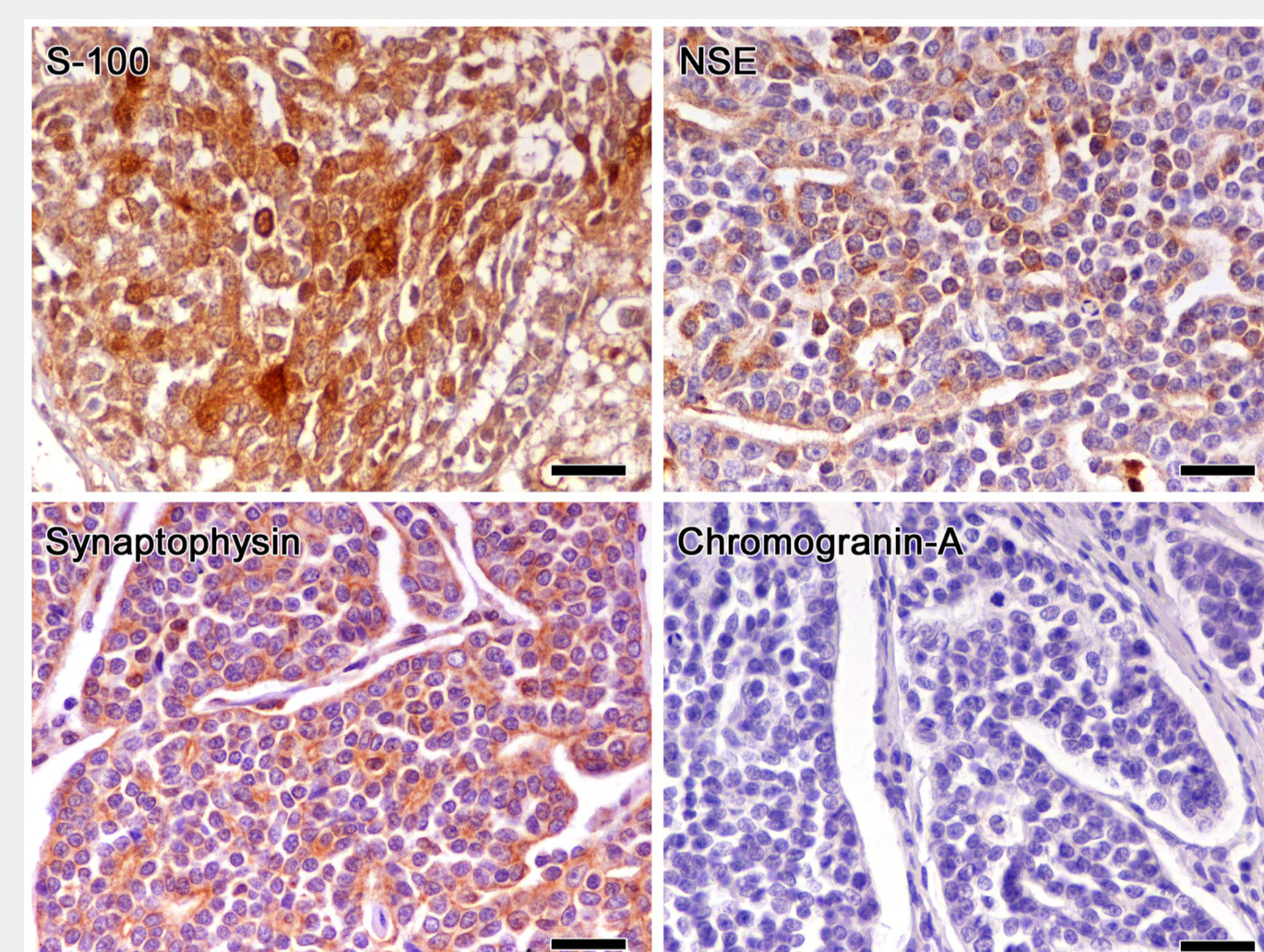


Fig.5: Immunohistochemical staining proves neuroendocrine origin by staining the neoplastic cells positive for: S-100 (cytoplasmic and nuclear staining), NSE (multifocal regions of moderate signal density of cytoplasmic and nuclear staining) and Synaptophysin (focal regions of moderate signal density of cytoplasmic staining). Chromogranin staining was negative. Bar=50µm

Conclusions

- ✓ Chemodectoma arising from the innominate artery below the right subclavian artery metastasized probably via the internal carotid artery into the petrous bone resulting in trigeminal nerve destruction, veli palatini muscle dysfunction, and Horner syndrome in this dog.
- ✓ Innominate artery chemodectoma showed classical neuroendocrine tumor histomorphology, ample S-100, scarce NSE and Synaptophysin and absent Chromogranin A expression.
- ✓ MRI proved particularly helpful in detecting chemodectoma metastasis.
- ✓ Innominate artery chemoreceptor tissue is a rare anatomical site of chemodectoma occurrence. The metastatic behavior of chemodectoma was also unusual, with metastasis occurring in the skull, while classical metastatic target organs such as lungs, liver, pancreas and kidneys remained unaffected.
- ✓ Tympanic bulla accumulation may be explained by the dysfunction of the veli palatini muscle, which is innervated by the maxillary branch of the trigeminal nerve.

