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CASE REPORT
Second order Horner's syndrome in a cat

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This case report describes the clinical and magnetic resonance imaging (MRI) findings of a 3.5-year-old, male neutered, domestic shorthair cat with second order Horner's syndrome as the only clinical abnormality. The neuroanatomical pathway of the sympathetic innervation to the eye, differential diagnoses for Horner's syndrome in cats, and the interpretation of pharmacological testing are reviewed. The unusual MRI findings and the value of fat-suppressed MRI sequences are discussed.

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A 3.5-year-old, male neutered, domestic shorthair cat was referred to the Neurology/Neurosurgery Service at the Animal Health Trust with right-sided miosis, third eye-lid protrusion, upper eye-lid ptosis, and enophthalmos (Horner's syndrome) of about 15 days duration. The cat lived mostly outdoors and was often involved in cat fights. The owner had noticed the eye abnormality after returning home from a short holiday. The referring veterinarian reported that apart of the right-sided Horner's syndrome, general physical (including otoscopic) and neurological examinations were unremarkable. Pre-referral investigations included haematology, serum biochemistry, thyroxine levels (T_4), feline immunodeficiency virus and feline leukaemia virus testing, thoracic radiographs and pharmacological testing with topical 1% phenylephrine hydrochloride. No abnormalities were identified on blood tests and imaging of the thorax. Pharmacological testing with 1% phenylephrine hydrochloride (performed about 6 days after onset) revealed no changes after 40 min.

When the cat was assessed at the Animal Health Trust the clinical findings were consistent with the ones reported by the referring veterinarian. The neuroanatomic localisation was to the sympathetic innervation to the right eye. Based on the absence of other neurological deficits and the results of pharmacological testing performed by the referring veterinarian second order Horner's syndrome was considered most likely. Differential diagnoses included: focal trauma, infection/inflammation, and neoplasia. Clinical examination of the cervical region did not reveal any abnormalities. Magnetic resonance imaging (MRI) of the head, cervical and cranial thoracic spine was performed to investigate further the cause of the Horner's syndrome. Transverse MR images of the cervical region showed an area of abnormal signal within the right jugular region at the level of caudal C2 and cranial C3 vertebrae (Figs 1–3). The abnormal signal extended from the subcutaneous tissue around the external jugular vein, along the sternocephalicus muscle fascia, to the common carotid artery, vagosympathetic trunk, and internal jugular vein. The abnormal tissue was isointense on T1-weighted MR images, moderately hyperintense on T2-weighted MR images and contrast enhanced after intravenous injection of gadolinium (Omniscan, Nycomed). The abnormal tissue extended along the fascial plane without invading adjacent structures or causing significant mass effect. These changes are suggestive of a soft tissue infection and inflammation, and less likely infiltrative neoplasia. Similar signal changes may be seen on T2-weighted MR images following venepuncture but contrast enhancement is not usually as extensive and no venepuncture was performed at that site in this cat. The rest of the MRI study did not reveal any abnormalities.

After clipping the hair of the right jugular region it was possible to palpate a small thickening of the

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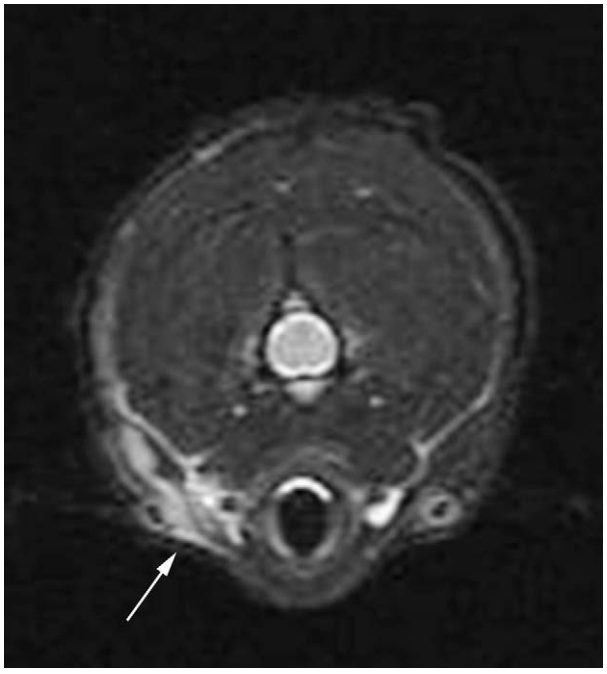

Fig 1. Transverse T2-weighted (with fat saturation) MR image at the level of caudal C2 vertebra. The tissue surrounding the right external and internal jugular veins, the common carotid artery, and the vagosympathetic trunk is increased in signal and is moderately hyperintense (arrow).

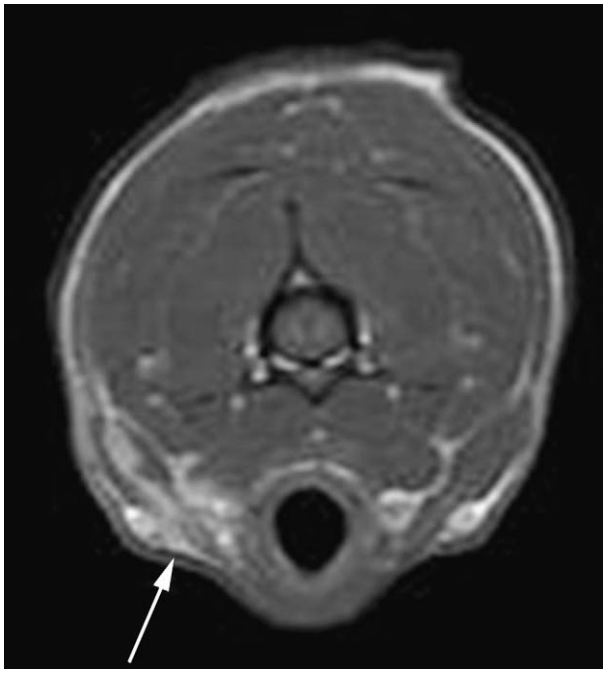

Fig 2. Transverse T1-weighted (post-contrast) MR image at the same level as Fig 1. There is moderate contrast enhancement of the tissue surrounding the right external and internal jugular veins, the common carotid artery, and the vagosympathetic trunk (arrow).

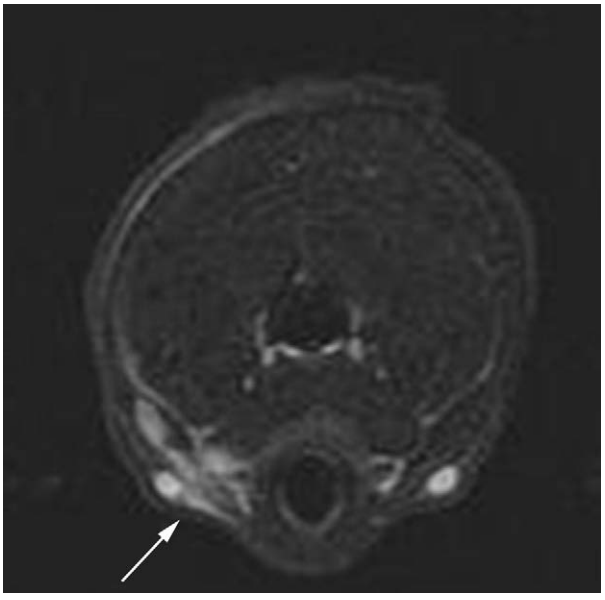

Fig 3. Transverse digital subtraction image at same level as Figs 1 and 2. The area of abnormal contrast enhancement is clearly seen (arrow). The subtraction image shows the areas of contrast enhancement more clearly than the standard post-contrast T1-weighted image.

subcutaneous tissue, but no cutaneous abnormality was seen. Due to the small size of the lesion and its proximity to neurovascular structures, it was decided to treat the cat with amoxicillin and clavulanic acid (Synulox; Pfizer) for 2 weeks and meloxicam (Metacam; Boehringer Ingelheim) for 1 week based on the probable diagnosis of a focal infection and inflammation secondary to a penetrating injury. In case of lack of improvement, the plan was to perform an ultrasound-guided fine needle aspirate or a surgical biopsy. The cat improved after 3 days of treatment and was completely normal after 10 days of treatment.

The sympathetic innervation to the eye originates in the hypothalamus and rostral midbrain (first order neuron), travels down the cervical spinal cord through the tectotegmental spinal tracts to synapse on the second order neuron cell bodies located in the intermediate grey column of the first three thoracic spinal cord segments. The second order neuron axons leave the spinal cord within the T1, T2 and T3 ventral nerve roots and subsequently separate from them to form the ramus communicans which joins the thoracic sympathetic trunk inside the thorax ventrolateral to the vertebral column. The sympathetic trunk courses cranially in close apposition to the descending vagus nerve, together forming the vagosympathetic trunk within the carotid sheath. The sympathetic axons course rostrally through the caudal cervical ganglion, synapsing in the cranial cervical ganglion, adjacent to the tympanic bulla. From here, third order sympathetic axons pass through the tympano-occipital fissure with the internal carotid artery, pass between the tympanic bulla and the petrosal bone into the middle ear cavity, enter the

cranial cavity, pass close to the cavernous sinus, leave the cranial cavity through the orbital fissure in close approximation with the ophthalmic branch of the trigeminal nerve and innervate the iris dilator muscle and the periorbital and eye-lid smooth muscles.[1–3] A lesion anywhere along this pathway can cause Horner's syndrome. The most commonly reported causes of Horner's syndrome in cats include trauma, infection/inflammation, neoplasia and idiopathic.[4–12] First order Horner's syndrome is invariably associated with additional neurological deficits such as ataxia, paresis/plegia, postural reaction deficits (brain stem or spinal cord lesion), altered mental status and involvement of other cranial nerves (brain stem lesion). Horner's syndrome can occur as the only clinical abnormality when either second or third order neurons are affected.

Pharmacological testing, based on the principle of denervation hypersensitivity, can be used to determine more precisely the site of the lesion in patients affected by Horner's syndrome, however, it should be used only as a guide as results vary according to the time elapsed since the lesion occurred and the completeness of the lesion.[13] Dilation of the pupil within 20 min after instillation of 1% phenylephrine hydrochloride is suggestive of a third order neuron lesion, while dilation of the pupil after 30–45 min suggests a second order neuron lesion, and dilation of the pupil after 60–90 min suggests a first order neuron lesion or no sympathetic denervation of the eye.[3,13] In this case, the referring veterinarian reported no changes 40 min after instillation of 1% phenylephrine hydrochloride (performed about 6 days after onset). Based on this finding, a third order neuron lesion was unlikely. The absence of other neurological deficits made a first order neuron lesion very unlikely. Therefore, we suspected a second order Horner's syndrome which was confirmed on MRI.

The use of fat suppression MRI sequences is helpful when evaluating pathology affecting fat or muscles. On fast-spin-echo T1-weighted, and T2-weighted sequences fat is hyperintense. As most pathology results in an increase in signal intensity on T2-weighted images it may be difficult to recognise increased signal in tissue that is normally hyperintense. The same problem occurs when trying to assess contrast enhancement on T1-weighted images in tissue that is already hyperintense. A variety of fat-saturation/suppressing sequences may be used, the choice depending on a range of factors including type of scanner and area being imaged. Fat suppression/saturation sequences are valuable in screening for pathology of soft tissues. Areas of increased water content (eg, inflammation) continue to appear hyperintense but are easier to visualise as the fat is rendered hypointense resulting in increased contrast between areas of pathology and muscles and fat. The use of digital subtraction post-processing (where the pre-contrast T1-weighted image is subtracted from the post-contrast T1-weighted images) or using fat-saturation techniques also makes detection of abnormal contrast enhancement easier.

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