Case Report

Right-sided laryngeal hemiplegia and Horner’s syndrome in a horse

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Summary

This study describes right laryngeal hemiplegia (LH) and right-sided Horner’s syndrome (HS) in a horse. The average temperature of the face was 3.5°C higher on the right compared with the left side, as determined by thermographic imaging. The syndrome occurred following an episode of right mid-cervical cellulitis due to inadvertent perijugular deposition of gentamicin.

Introduction

Horner’s syndrome (HS) consists of a group of clinical signs resulting from interruption of the sympathetic innervation to the head (Smith and Mayhew 1977; Sweeney and Sweeney 1984; Ghafir et al. 1996; Fishman et al. 2003; Hahn 2003, 2006). This innervation originates in sympathetic neurons located in the caudal hypothalamus, ventrolateral medulla, ventral mid-brain and pons. Descending fibres from the brain stem travel through the cervical spinal cord and leave the lateral funiculus to synapse on preganglionic neurons in the intermediolateral horn (grey matter) of the cranial thoracic segment (T1–T3). These preganglionic neurons emerge from the spinal cord through the ventral roots in the T1–T3 spinal nerves and extend cranially in the vagosympathetic trunk as a cervical paravertebral or cervical sympathetic chain adjacent to the carotid artery (Fig 1). These neurons then synapse at the level of the atlas on post ganglionic neurons in the cranial cervical ganglion which is in the wall of the guttural pouch and intimately related to the internal carotid artery, glossopharyngeal nerve and vagus nerve (Smith and Mayhew 1977; Ghafir et al. 1996; Murray et al. 1997; Hahn 2003, 2006; Furr and Reed 2008). In horses, the structures of clinical importance innervated by post ganglionic fibres are the blood vessels and sweat glands of the head and cranial neck and the iris dilator muscle, smooth retrobulbar muscles and certain palpebral muscles (Hahn 2003).

The clinical signs of HS depend on the site, duration and the severity of the lesion and on the affected species. Ophthalmic changes, such as enophthalmos, third eyelid protrusion, ptosis and miosis are predominant in dogs and cats (Kern et al. 1989). Smith and Mayhew (1977) demonstrated experimentally that cattle with HS also present with hyperthermia and an engorgement of the vessels of the pinnae and a decreased number and size of droplets of sweat on the muzzle on the affected side. In contrast, goats and sheep develop transient facial and auricular hyperthermia and palpebral ptosis as the most obvious signs of HS.

Horner’s syndrome in horses is characterised by upper palpebral ptosis, hyperthermia and unilateral sudoreisis of the face and variable regions of the neck and trunk, whereas enophthalmos, third eyelid protrusion and miosis are less common signs. In contrast, facial blood vessel dilation and hyperaemia of the nasal mucosal and conjunctival membranes are described (Smith and Mayhew 1977; Firth 1978; Mayhew 1980, 1989, 2009; Purohit et al. 1980; Sweeney and Sweeney 1984; Green et al. 1992; Knottenbelt and Pascoe 1994; Bacon et al. 1996; Fishman et al. 2003; Hahn 2003, 2006). Inspiratory noises due to blood vessel engorgement in the nasal cavity (Firth 1978; Purohit et al. 1980; Green et al. 1992; Ghafir et al. 1996) and dermatitis (Green et al. 1992) resulting from chronic sweating have also been observed in association with HS. Such affected animals may not perform as well as expected, particularly when there is a reduction in upper airway airflow (Green et al. 1992).
The most common causes of HS in horses are trauma, inflammation adjacent to the cervical vagosympathetic trunk (Smith and Mayhew 1977; Firth 1978; Green et al. 1992; Firshman et al. 2003), inappropriate administration of medications into the region of the jugular vein (Smith and Mayhew 1977; Sweeney and Sweeney 1984; Green et al. 1992), thoracic neoplasia (Firth 1978; Milne 1986; Bacon et al. 1996; Murray et al. 1997), guttural pouch disease (Cook 1968; Smith and Mayhew 1977), periorbital masses and trauma of the basisphenoid region (Furr and Reed 2008).

A diagnosis of HS is based on the clinical history and physical and neurological examinations. Thermography can confirm the presence of unilateral hyperthermia by measuring temperature using infrared emission, which is closely related to vascular activity and cutaneous metabolism (Purohit et al. 1980; Ghafir et al. 1996). Radiography, endoscopy and ultrasonography should be performed to assist in lesion localisation (Firth 1978; Green et al. 1992; Bacon et al. 1996; Firshman et al. 2003).
The case reported here clearly demonstrates the clinical signs of HS in the horse and prompts discussion of complementary examinations in this syndrome.

**Case history**

A 6.5-year-old female Quarter Horse was seen at the Veterinary Hospital of the Julio de Mesquita Filho Veterinary Medicine and Animal Science University, Botucatu Campus (São Paulo, Brazil). The horse was referred for evaluation of right upper palpebral ptosis that evolved over 4 months. The ptosis began after jugular phlebitis and severe cellulitis of the right cervical region due to inadvertant perivascular administration of gentamicin.

**Clinical findings and diagnosis**

The patient’s respiratory rate, heart rate and rectal temperature were within the normal range. Sweating was observed on the right side at the base of the ear and on the face and the cranial third of the neck to the level of C2/C3 [Fig 2a]. Right upper palpebral ptosis was observed along with discrete enophthalmos and third eyelid protrusion [Fig 2b,c]. No asymmetry in the colour of mucous membranes of the head was observed and no abnormal breathing sounds heard at rest. Anisocoria was not evident even when the patient was evaluated in low ambient light. The temperature of the right side of the face felt warmer than the left side.

The haemogram and biochemical determinations yielded results that were within the normal range for the species (Orsini and Divers 2008). Cervical (C1–C7) radiography and ultrasound examination of the right jugular vein revealed no abnormalities. Endoscopy revealed right laryngeal hemiplegia [Fig 2d,e] and normal guttural pouch anatomy. Thermography1 measured a higher temperature on the right side of the face compared to the left side. The average difference between the

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1. Thermography
temperature measured at 8 points on each side of the face was 3.5°C (Fig 2f). It was also possible to observe that regions with greatest sweating presented lower temperatures at the affected side (Fig 2g).

Discussion

The clinical signs exhibited by the horse in this report confirmed the diagnoses of right-sided HS (Smith and Mayhew 1977; Purohit et al. 1980; Sweeney and Sweeney 1984; Mayhew 1989, 2009; Green et al. 1992; Bacon et al. 1996; Fishman et al. 2003; Hahn 2003, 2006) and right-sided laryngeal hemiplegia (LH) (Anderson 2007). Upper palpebral ptosis has been reported to be the most frequently observed clinical sign in animals suffering from HS, regardless of the species (Smith and Mayhew 1977; Firth 1978; Sweeney and Sweeney 1984; Green et al. 1992). This sign was observed by the owner and the referring veterinary surgeon and led to the request for a neurological examination of the horse. Palpebral ptosis occurs as a result of decreased tone in Muller’s superior tarsal muscle occurring as a result of a deficiency in smooth palpebral muscle sympathetic innervation (Ghafir et al. 1996; Hahn 2003, 2006). Cranial VII paresis can result in mild ptosis in horses due to paresis of the levator anguli oculi muscle but this was not observed in the horse described here which showed no ear or lip paralysis or dry keratitis as is usually observed in such cases (Mayhew 2009). The enophthalmos observed in this study was not an evident clinical sign, but it was possible to observe partial third eyelid protrusion. According to Firth (1978) and Hahn (2003, 2006), ptosis can hinder the evaluation of the presence or absence of enophthalmos that occurs due to decreased retrobulbar muscle tone. In horses, enophthalmos may be present to a degree such that the palpebral and bulbar conjunctivae are visible on the medial part of the lower lid because the eyeball is no longer in perfect apposition with the lower lid (Firth 1978). Miosis can occur due to a loss of sympathetic control of the pupillae dilator muscle. This clinical sign is not always evident in horses with HS (Firth 1978; Green et al. 1992; Fishman et al. 2003), which was the case in this study (Kern et al. 1989). In contrast, this sign is frequently reported in dogs with HS.

During the evaluation, excessive unilateral sudoresis (i.e. hyperhidrosis) on the face, at the base of the ears and over the atlas and axis was observed. In horses with HS, the most evident clinical sign is unilateral sudoresis (Smith and Mayhew 1977; Sweeney and Sweeney 1984; Mayhew 1989; Green et al. 1992; Hahn 2003, 2006). This sign is not observed in other species, including man, dogs, cats and sheep and the cause of its occurrence is not yet fully understood (Hahn 2003, 2006). It has been suggested that the sudomotor pathway involves action of the sympathetic nervous system on the cutaneous vascular supply and local chemical transmission probably of catecholamines. It is suggested that sympathetic denervation results in loss of cutaneous vasoconstrictor tone, thus vasodilatation and increased blood flow taking more noradrenaline to the sweat glands promoting sudoresis. There may also be a component of upregulation of adrenoreceptors on sweat glands resulting in denervation hypersensitivity, however no direct adrenergic nerve supply to equine sweat glands appears to have been recognised (Jenkinson et al. 2006). The presence of sudoresis helps to locate the lesion because it indicates the denervated area of skin. Horses with post ganglionic lesions (cranial cervical ganglion or distal usually exhibit sudoresis as far as the level of the atlas (Bacon et al. 1996) and sudoresis has been observed caudally to this area (C2–C3) in horses with preganglionic (mid-cervical) lesions (Mayhew 1989).

Usually, results from thermography are not influenced by a horse’s coat, providing quite reliable face and neck temperatures (Ghafir et al. 1996). Purohit et al. (1980) and Ghafir et al. (1996) reported that temperature differences can be verified and measured using thermography. Purohit et al. (1980) observed that, physiologically, the eyes and nostrils are the hottest areas of the face and that after surgically-induced vagosympathectomy, an increase of 1–2.5°C occurs on the affected side. In this study, we used thermography to measure an average difference of 3.5°C between the right and left sides of the face, confirming the clinical finding. In addition, these temperature difference values were similar to those described previously by Ghafir et al. (1996). This difference in temperature results from an increase in blood flow and vasodilation on the affected side (Smith and Mayhew 1977) and it is also interesting to note that the regions with the greatest sweating presented lower temperatures at the affected side. This is in agreement with previous observations by I. Mayhew (personal communication) suggesting this is due to the latent heat of evaporation reducing surface temperature where there is more moisture.

Endoscopy confirmed the presence of right LH. This was also believed to be due to the previous cervical cellulitis damaging not only the sympathetic trunk but also the recurrent laryngeal nerve that lays against the trachea. Such association of LH with inadvertent injection of irritant substances around the jugular vein has been previously documented (Green et al. 1992; Anderson 2007). Horner’s syndrome in horses in conjunction with laryngeal hemiplegia has been previously described by Firth (1978), Purohit et al. (1980), Skarda et al. (1986), Simoens et al. (1990), Green et al. (1992) and Ghafir et al. (1996). Endoscopy ruled out guttural pouch abnormalities, which was an important evaluation because the cranial cervical ganglion is intimate with the vagus nerve (that gives rise to the recurrent laryngeal nerve) in the wall of the guttural pouch (Furr and Reed 2008).

Because the sympathetic pathway is long, HS may be caused by damage at many different sites along its pathway. It is important to note that mesencephalon and brain stem lesions were unlikely in this case because no abnormalities were noted in the gait, sensorium or other cranial nerve functions. Complementary examinations did not determine the exact location of the lesion; however,
the location of excessive sweating, the finding of right laryngeal hemiplegia and history of perivascular drug administration in the right cervical region suggested that the lesion occurred in the preganglionic fibres of the neck between the thoracic inlet and gullet pouch.

Horner’s syndrome after i.v. drug administration has been previously reported with similar signs to those observed in the present case. Sweeney and Sweeney (1984) reported transient HS after jugular i.v. administration of xylazine, without evidence of perivascular drug administration. The signs disappeared 14 h after onset. A more persistent syndrome that lasted approximately 5 months was described in two horses by Green et al. (1992).

In this instance, the animals received an i.v. injection of vitamin E and selenium and swelling was reported in the cervical region compatible with a local tissue reaction due to inadvertent perivascular deposition of some of the drugs as is reported in the present case. It should be noted that in the two cases described by Green et al. (1992), laryngeal hemiplegia also occurred, which may have led to a future loss of athletic ability in these animals.

The present study describes a horse with right-sided HS and right-sided LH that resulted from perijugular drug administration. This administration possibly led to the damage caused to the preganglionic fibres in the cervical sympathetic trunk within the carotid sheath and to the recurrent laryngeal nerve adjacent to the trachea. This case confirms that thermography can be successfully used to observe increased temperature on the affected side of the face in HS.

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Manufacturer’s address

1Infra Cam, Flir Systems Inc., Boston, MA, USA.

Conflicts of interest

The authors declared no conflicts of interest.

References


