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Case Report

Computed tomographic and magnetic resonance imaging of a coup contrecoup traumatic brain injury in a horse

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Summary

A 3-year-old Thoroughbred mare found recumbent in the field was referred for further assessment with suspicion of a skull fracture. Neurological examination identified compulsive tight circling to the left, and hypermetria in all four limbs. The mare was obtunded, with a mild head tilt to the right, absent menace response of the right eye and decreased facial sensation on the right. Standing computed tomographic examination revealed a subtle depression fracture of the dorsal calvarium and moderate intra-axial midline shift consistent with a traumatic brain injury (TBI). Despite supportive treatment, the mare deteriorated and was subjected to euthanasia. Post-mortem high field magnetic resonance imaging revealed findings consistent with a small cerebral contusion adjacent to the fracture site, and moderate to severe ipsilateral cerebral oedema within the caudal cerebrum and rostral brainstem, consistent with a coup contrecoup TBI. Brainstem lesions indicate a poor prognosis and support the decision for euthanasia. This is the first report of the imaging findings of a coup contrecoup TBI in a horse.

Case details and history

A 3-year-old Thoroughbred mare was found recumbent in the field, with abrasions to the face, and blood in each nostril. A hairless area of skin, associated soft tissue swelling and crepitus were present on the left side of the forehead, between the left eye and forelock, raising suspicion of a skull fracture.

Clinical findings

On presentation to the Royal Veterinary College Equine Hospital, the vital parameters were within normal limits and there was dried blood in each nostril; however, bilateral wounds on the dorsal aspect of the carpi were acquired during transportation. Neurological evaluation at admission and the following day identified intermittent compulsive tight circling to the left and the mare reacted to minor stimuli in an exaggerated and unexpected manner. Minimal cerebellar ataxia manifested as hypermetria when the mare walked forward, and occurred in all four limbs, more pronounced in the thoracic limbs, and proprioceptive deficits were noted in all four limbs. The mentation was obtunded and a mild, intermittent head tilt to the right was evident. Cranial nerve examination identified absence of a menace response in the right eye, and funduscopic examination was bilaterally unremarkable. Bilateral palpebral reflexes, eye position, pupillary size and pupillary light reflexes were normal. Facial sensation, or response to stimulation, was decreased on the right side of the nasal septum, and in the right nostril. No tongue tone or movement was detected bilaterally when the tongue was pulled out of the mouth. The neuro-localisation was considered multifocal including left sided forebrain, cerebellum and caudal brainstem.

Serum biochemistry identified increased CK and AST activities (4783 U/L and 633 U/L, respectively) consistent with trauma and recumbency, and mildly decreased albumin concentrations (27.6 g/L). The packed cell volume was 34%, total protein concentration 57 g/L and lactate concentration <0.7 mmol/L.

The mare was administered 2 L intravenous hypertonic saline, 8 mg/kg intravenous oxytetracycline hydrochloride (Engemycin 10%)[1] and 1.1 mg/kg intravenous flunixin meglumine (Finadyne 50 mg/mL)[1]. The carpal wounds were superficial, and were cleaned and bandaged. The mare received a further 10 L of intravenous isotonic saline overnight. The following day the mare was slightly more responsive with mildly improved tongue tone. However, she remained obtunded and circled to the left. A further 2 L intravenous hypertonic saline and 0.1 mg/kg intramuscular morphine sulphate (30 mg/mL)[2] were administered. Her appetite improved slightly following the morphine administration, yet subsequently reduced again.

Imaging diagnosis and outcome

The horse underwent standing computed tomographic (CT) imaging of the head using a 16-slice multi-detector CT scanner (GE Lightspeed Pro 16)[3] using 120 kV, 200 mAs, 1.25 mm slice thickness with an inter-slice interval of 1.25 mm. Images were reconstructed using both a bone and soft tissue algorithm in a 512 × 512 matrix, and analysed by a European College of Veterinary Diagnostic Imaging board-certified radiologist (R.E.M.).

In the left parietal bone, 1.3 cm lateral to midline, extending rostrally into the left frontal bone, there was a moderately well defined, linear, transverse discontinuation in the bone. It coursed 1.2 cm in a caudomedial to rostrolateral direction, and 0.9 cm in a ventromedial to dorsolateral direction (through both the internal and external surface of the bones), and was between 1–2 mm wide (**Fig 1**). The rostral aspect entered the left frontal paranasal sinus, where there was adjacent mild soft tissue thickening. The dorsal aspect of the cranium in this area was flattened and mildly

ventrally displaced into the cranial cavity, resulting in an incongruity (step-defect) on the external surface of the bone. This was consistent with a subtle depression fracture of the left calvaria (**Fig 1**). There was concurrent mild thickening of the overlying soft tissues, with multifocal subcutaneous areas of gas attenuation overlying the left temporal muscle. Moderate midline shift towards the right side was present within the caudal half of the forebrain including midline shift at the level of the hyperattenuating supracollicular fluid accumulation (70 HU), with depression of the dorsal aspect of the left brainstem (**Fig 2**). The left side of the caudal forebrain had mildly lower attenuation (19 HU) compared to the right side (36 HU). Reduced attenuation indicates reduced density of the cerebral parenchyma, which can be caused by increased water content due to conditions such as vasogenic oedema, necrosis, abscessation, neoplastic or metastatic processes (Lanksch 1982). The combination of midline shift and decreased attenuation of the cerebral parenchyma likely indicated moderate cerebral oedema.

Conservative treatment, including the aforementioned antimicrobial and anti-inflammatory treatment, continued for 4 days during which the mare's condition waxed and waned. However, a lasting improvement in the clinical signs was not achieved. The mare remained obtunded with a marginal nutritional intake and continued circling. Due to the lack of progress despite intensive treatment, welfare considerations and the poor long-term prognosis to full recovery, the decision was made to euthanise the mare.

Post-mortem magnetic resonance imaging

Within 24 h of euthanasia, the cranial cervical spine was disarticulated and magnetic resonance imaging (MRI) of the head was performed with the head in dorsal recumbency, centred on the brain, using a 1.5-Tesla superconducting magnet (GE Sigma Echospeed System)[4]. Images were obtained in two planes (transverse and sagittal) and sequences including T2-weighted (T2W) fast spin echo (FSE), T2W fast field echo (FFE) and T2W fluid attenuating inversion recovery (FLAIR) were acquired. There was moderate to severe increased T2W FSE signal intensity within the white and grey matter, centred on the left occipital and temporal

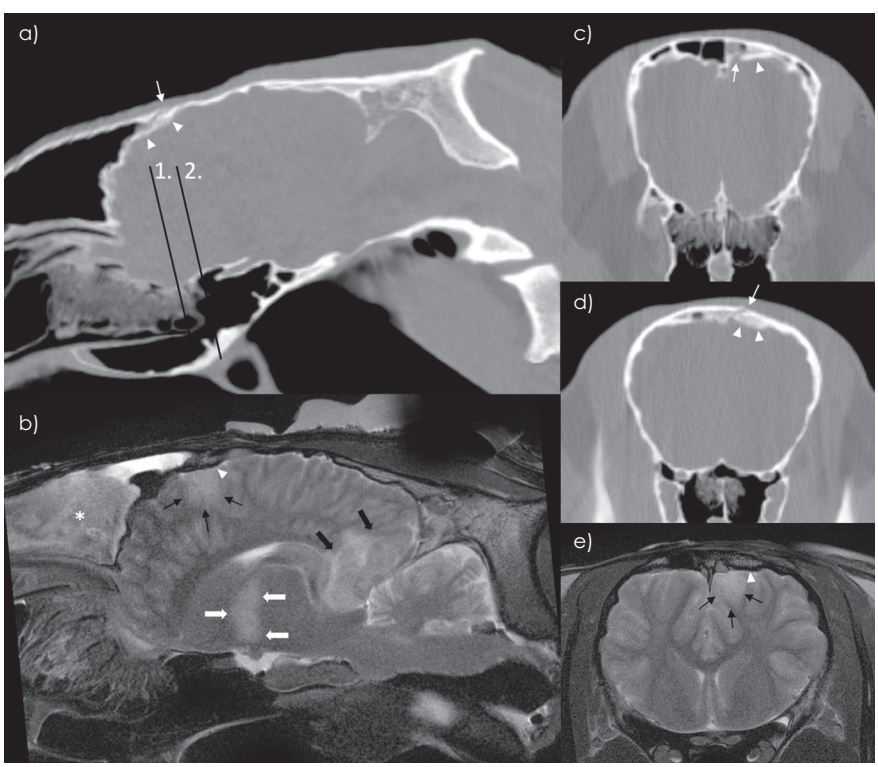

Fig 1: a) Sagittal CT image reconstruction on bone window (Window Level 800HU, Window Width 2800HU), 1.3 cm left lateral to midline, at level of the fracture. The black lines 1 and 2 depict the level of the transverse images in c and d, respectively. b) Sagittal T2W FSE magnetic resonance (MR) image at the same level as (a). The moderate T2-weighted (W) fast spin-echo (FSE) hyperintensity rostral to the cranial cavity represents fluid within the left frontal paranasal sinus (asterisk). c) Transverse CT image at the rostral aspect of the fracture line (level with line 1 in image a), showing communication of the fracture line (white arrow) with both the left frontal paranasal sinus and the cranial cavity. d) Transverse CT image at the caudal aspect of the fracture line (level with line 2 in image a). e) Transverse T2W FSE MR image at the caudal aspect of the fracture line (level with line 2 in image a). The white arrow indicates the fracture line (both internal and external aspects). The white arrowheads represent the ventral deviation of the parietal and frontal bone into the cranial cavity. There is a T2W FSE hyperintense intra-axial lesion (black arrows) within the parietal lobe at the fracture site (coup injury) consistent with oedema. Similar lesions were also visible within the left thalamus (white block arrows) and left occipital lobe (black block arrows). (a and b) are displayed with the rostral aspect to the left of the image. All transverse images are displayed with the left side of the patient on the right side of the image.

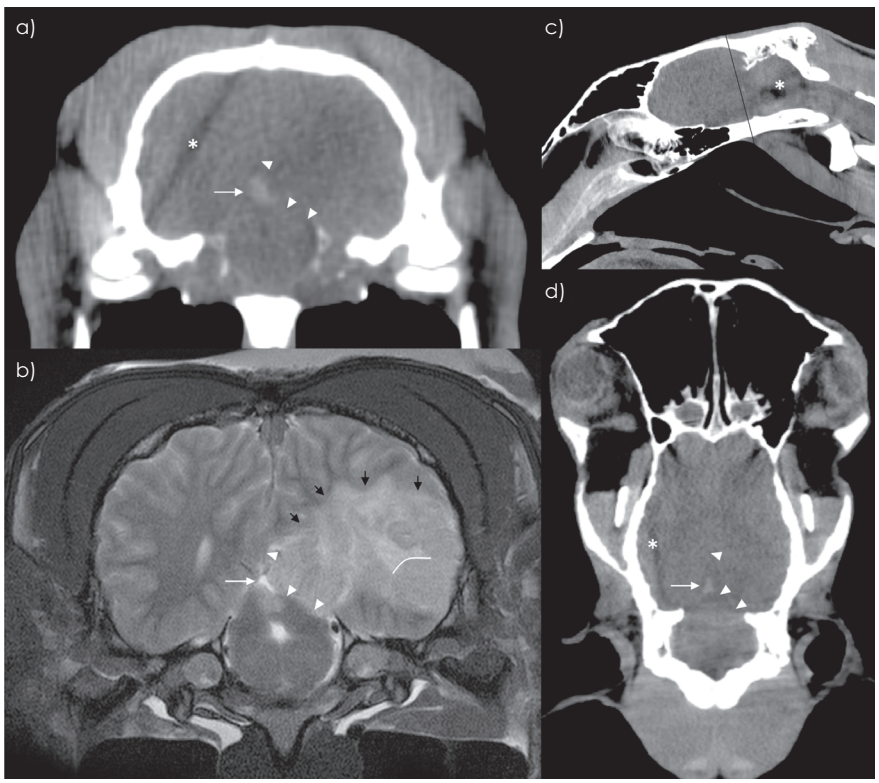

Fig 2: a) Transverse CT image at the level of the midbrain (black line in image c). b) Transverse T2W FSE magnetic resonance image at the same level as A (black line in image c). c) Sagittal CT reconstruction, aligned on midline. d) Dorsal CT image at the level of the supracollicular fluid. In image (b), the moderate T2W FSE hyperintensity within the occipital lobe is outlined by the black arrowheads. The curved white line represents the transition zone between the hyperintense white matter (likely vasogenic oedema) and slightly less hyperintense grey matter (likely cytotoxic oedema). The white arrowheads indicate the mass effect, causing right midline shift and compression of the dorsal midbrain. The white arrow indicates the right shift of the supracollicular fluid which is hyperattenuating on the CT and on T2W FSE and FFE/GRE sequences which may indicate chronic haemorrhage. a, b and d are displayed with the left side of the patient on the right side of the image. In (c) the rostral aspect is to the left side of the image, and in (d) the rostral aspect is to the top of the image. All CT images are displayed using a soft tissue filter (Window Level 45HU, Window Width 350HU). The white asterisks indicate beam hardening artefacts.

lobes, extending to the piriform and parietal lobes. This was consistent with moderate diffuse cerebral vasogenic and cytotoxic oedema and/or necrosis, causing a mass effect with subsequent moderate midline shift towards the right side (**Figs 2** and **3**). The left aspect of the thalamus, dorsal colliculi and tegmentum of the midbrain were compressed by this mass effect, and the cerebellum was compressed towards the caudal aspect of the skull (**Fig 3**). Ill-defined moderate increased T2W FSE signal intensity was also present within the left thalamus, caudate (tail) and lentiform nuclei. There was a focal area of T2W FSE hyperintensity when compared to normal grey matter, with a centre of T2 FFE GRE hypointensity near the left internal capsule, consistent with a magnetic susceptibility artefact due to haemorrhage (**Fig 3**). There was loss of a smooth continuous bone margin at the previously described fracture site, with mild ventral deviation of the parietal bone. The adjacent meninges were ill-defined and there was a small volume of extradural material, of T2W FSE iso-intensity to grey matter, at the fracture site. Within the grey matter of the left parietal lobe, adjacent to the fracture site, there was a small area of mild to moderate T2W FSE signal intensity, consistent with mild oedema (**Fig 1**). A moderate volume of fluid was present within the left frontal paranasal sinus, which had either been produced ante-mortem subsequent to the CT examination, or post-mortem (**Fig 1**).

Post-mortem histopathology

Post-mortem examination confirmed the presence of a fracture on the left side of the calvarium, as well as damage to the surrounding tissues. Within the left occipital cortex, there was marked, focally extensive neuronal necrosis and gemistocytosis with axonal degeneration and vacuolation. Severe bilateral, subacute cavitation and necrosis with Gitter cells and axonal degeneration were present within the dorsal colliculi. The cerebellar peduncles had marked bilateral axonal degeneration and vacuolation, and there was segmental Purkinje cell loss within the cerebellum. Mild, multifocal oedema and haemorrhage of the meninges, and mild, multifocal, perineural haemorrhage of the right optic nerve were also present; however, these were per-acute and most likely agonal changes.

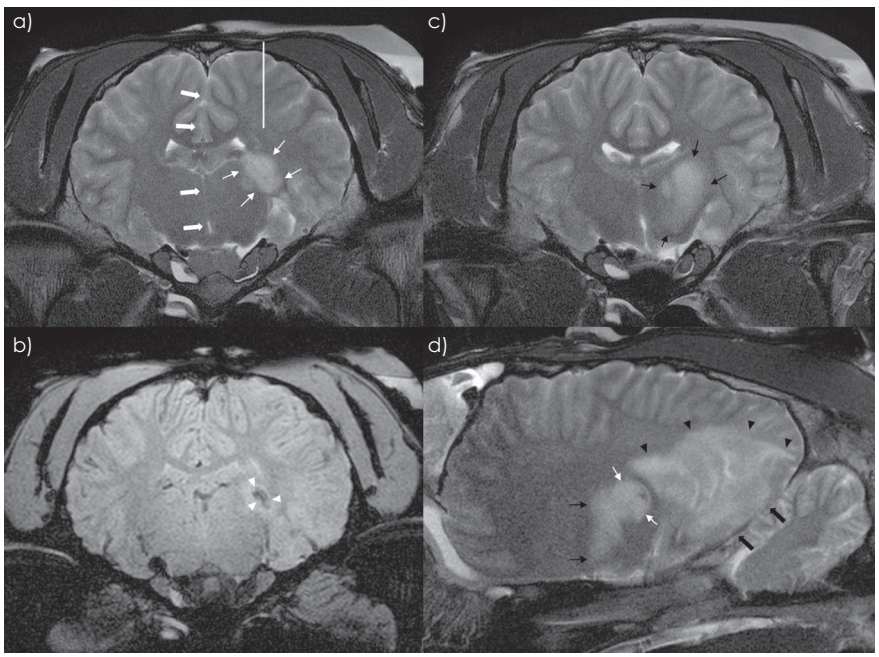

Fig 3: a) Transverse T2W FSE magnetic resonance (MR) image at the level of the thalamus. b) Transverse T2W FFE MR image at the level of the thalamus. c) Transverse T2W FSE MR image one slice rostral to (a) and (b). d) Sagittal T2W FSE MR image at the level of the white line in (a). There is moderate T2W FSE hyperintensity at the left internal capsule (white arrows) with a core of T2W FFE hypointensity (white arrowheads), representing a magnetic susceptibility artefact due to haemorrhage. Moderate T2W FSE hyperintensity representing oedema is also present within the left thalamus (black arrows) and left occipital lobe (black arrowheads), causing a mass effect and subsequent right midline shift (white block arrows) and mildly compressing the rostral aspect of the cerebellum (black block arrow).

Discussion

The clinical examination, CT, MRI and histopathology findings are consistent with a traumatic brain injury (TBI) mainly affecting the left forebrain, cerebellum and brainstem. The point of impact is represented by the subtle depression fracture located at the left dorsal aspect of the calvarium. Adjacent to this, there is a mild cerebral contusion, termed the coup injury. The moderate to severe oedema, haemorrhage and necrosis within the left occipital lobe and adjacent areas, were likely due to a contrecoup injury. The CT and MR imaging of a coup contrecoup TBI has not previously been described in the horse. A coup contrecoup TBI indicates that the traumatic forces have been dissipated throughout the brain, and when the contrecoup injury is greater than the coup injury, a poor prognosis is associated in men (Banga *et al*. 2017). There are several theories to explain circumstances in which the contrecoup injury is more severe than the coup injury. The positive pressure theory believes when the head is accelerated forward prior to impact, the brain lags the skull (Lindenberg 1993; Drew and Drew 2004). On impact, the compressive forces are transmitted through the brain causing compression of the brain against the lagging or opposite surface of the skull, resulting in a larger contrecoup lesion at that location. The negative pressure theory dictates that when the forward motion of the skull is stopped on impact, the brain continues to travel forward, and the negative pressure created at the contrecoup site is responsible for the larger region of injury (Russell 1932). A third theory suggests that initial movement of the brain, on impact, will be in a direction away from the location of impact, resulting in initial impact of the brain and skull at the contrecoup location (Drew and Drew 2004). There are further rotational sheer stress (Holbourn 1943) and angular acceleration theories; however, due to the multifactorial nature of these injuries, no one theory has been found to entirely explain the contrecoup injury. Horses commonly suffer from high velocity, blunt force impacts through kick wounds or colliding with stationary or moving objects (Scrivani 2013). On presentation of a subtle fracture or wound to the cranium, with concurrent neurological defects, a contrecoup injury should be suspected. Some authors define a contrecoup brain injury by using a location greater than 90 degrees from the site of the coup impact (Cepeda *et al*. 2016). This implies that many contrecoup lesions are not directly opposite the side of the coup impact (on the contralateral side) but can be caudal in the ipsilateral side, if the coup lesion is rostral. No bone lesions, muscle changes or skin lesions were found associated with the left caudal aspect of the head. Therefore, a direct injury to cause that degree of brain damage would not have been possible due to a lack of changes on CT and MR images that correlate with an injury at that level. Additionally, the lesion in the caudal part of the brain (contrecoup injury) in this patient is extensive, involving the left occipital lobe and left caudal part of the thalamus, caused by compression on the midbrain. This pathology collectively belongs to the contrecoup injury. Often, the severity of the injury at the contrecoup site is greater than that occurring at the coup site. Indeed, there are several reports of patients with contrecoup injuries in the absence of coup injuries (Asha'Ari *et al*. 2011).

The dorsal colliculi are well protected in the centre of the brain. The severe cavitation, necrosis and axonal

degeneration present within the dorsal colliculi were bilaterally symmetrical, likely the result of raised intracranial pressure. Axonal degeneration has been observed in cases of equine TBI, due to stretching of neurons as the brain bounces back and forth (Brooks *et al*. 2014). There was no significant damage to the optic or trigeminal nerves, so the neurological deficits were associated to a left forebrain dysfunction. In the horse, up to 80% of the retinal ganglion cell axons cross over to the contralateral side of the brain at the level of the optic chiasm (Cummings and De Lahunta 1969; Herron *et al*. 1978). Moderate injury was present with the left thalamus, which contains the lateral geniculate nucleus, left internal capsule and the left occipital lobe (visual cortex). These are vital elements of the visual pathway in producing conscious visual perception (Beltran *et al*. 2012), and injury to these areas explain the absent menace response on the contralateral eye. As found in dogs, vestibular signs could be associated with paramedian thalamic lesions, whereas vestibular ataxia, circling and absent menace response can be caused by extensive dorsal thalamic lesions (Gonçalves *et al*. 2011). Therefore, the possibility of the vestibular signs due to these thalamic lesions was highly likely in this case. Although this has been reported in dogs and men, it is likely these vestibular signs caused by thalamic lesions could be present in horses. The reduced tongue tone improved, which indicated bilateral hypoglossal nucleus dysfunction or bilateral hypoglossal neuropraxia due to concussion of the caudal part of the brainstem or injury to the hypoglossal nerves. This was assumed a transient dysfunction due to improvement of the clinical signs rather than hypoglossal nerve transection. The tight circling to the left side and right sided head tilt can be caused by thalamic lesions as previously reported in dogs (Gonçalves *et al*. 2011).

The midline shift subsequent to severe oedema of the caudal forebrain was visible on the CT examination. In human medicine, CT is often the first-line imaging modality used to investigate acute TBI and certain characteristics such haemorrhage, fractures and signs of raised intracranial pressure are primarily evaluated (Maas *et al*. 2005). Assessing these characteristics on the CT images in this case, allowed diagnosis of a traumatic brain injury. However, MRI was required to assess severity, and this modality also allows a more detailed assessment (De Zani *et al*. 2013). MRI has high sensitivity for detection of non-haemorrhagic contusions and brainstem lesions. Some of the MRI findings in this case represented diffuse axonal injuries as seen in histopathology. Research in human medicine has indicated that specialised MR sequences, such as susceptibility-weighted imaging, helps further identify diffuse axonal injury (Bansal *et al*. 2018); however, there is not any clear association with the presence of diffuse axonal injury after TBI and long-term outcome (Humble *et al*. 2018). The supracollicular fluid accumulation was hyperattenuating on both the CT and T2W FSE and FFE/GRE MRI sequences, which may indicate chronic haemorrhage. Bilateral brainstem injuries have been associated with a very poor prognosis and were found to have a significant negative correlation with the modified Glasgow coma scale in dogs with TBI (Beltran *et al*. 2014). Therefore, the decision to euthanise the horse in this case was supported. Diffusion weighted imaging and apparent diffusion coefficient maps can help further characterise cerebral oedema as vasogenic or cytotoxic, for example (Wykes and Vindlacheruvu 2015). Whilst there were imaging characteristics indicating cerebral oedema; necrosis was detected on post-mortem examination. Cerebral oedema may have been present pre-mortem which later progressed to necrosis. The small volume of intracranial, extradural material at the fracture site may represent a small-volume extradural haematoma; however, this was not found at post-mortem. It is also important to note that clinical signs do not always fully correlate with structural changes in histopathology. After a TBI, some of the clinical signs may be due to a concussion rather than a contusion and therefore those clinical signs do not correlate with physical damage to the brain.

To conclude, in cases of subtle skull fractures found in horses with severe neurological signs, a coup and contrecoup lesion should be suspected. Moreover, it is important to consider that sometimes the contrecoup lesion can be more severe and clinically significant than the coup lesion. Computed tomographic assessment might be sufficient to indicate the presence or absence of some lesions consistent with TBI. However, MR imaging can provide further information on the severity and location of the lesions.

Authors' declaration of interests

No conflict of interests have been declared.

Ethical animal research

This study was ethically reviewed by the Clinical Research Ethical Review Board (CRERB) of the Royal Veterinary College, UK, and ethical approval has been granted for this study.

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Authorship

R. Morgan contributed to study design, study execution, data analysis and interpretation, and preparation of the manuscript. B. Dunkel contributed to study execution and preparation of the manuscript. E. Beltran contributed to data analysis and interpretation, and preparation of the manuscript. S. Spiro contributed to study execution. All authors gave their final approval of the manuscript.

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