TRAUMATIC INJURY TO THE BRAIN IN A SPORT HORSE

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Abstract
Brain injury after impact to the head is due to both immediate mechanical effects and delayed responses of neural tissues. This case report describes the posttraumatic evolution and treatment in a nine years old stallion for sport, which landed on its poll area during a riding competition. The clinicopathologic examination revealed: dilatation of the pupils, absence of the pupillary light reflexes, head tilt, ataxia with tendency to fall at walk and trot, unsteadiness on foot, circling, moderate neutrophilia and mild lymphopenia. After trauma, the horse received a single dose of dexamethasone (0.05mg/kg, IM) but result was uncertain. Ten days later, it was established a treatment with ketoprofen (2.2mg/kg IV) and vitamins B1 and B6 (10 ml IM). After five days of treatment, the horse was showing ataxia and right eye vision deficitis. After another ten days, it was tried a combination of ketoprofen (five days) and vitamins B1 and B6 (five days) with a deproteinized hemoderivative of calf blood product, Actovegin®, (1600mg/day, IV, 14 days), with good results. The horse started an easy training technique preparing for the next riding competitions.
Keywords*: ataxia, blindness, brain injury, circling, deproteinized hemoderivative of calf blood*

Introduction

Traumatic brain injury results from impact to the head, although acceleration-deceleration forces accompanying vigorous ‘‘whiplash’’ head movements also have potential to damage the brain (Southwood and Wilkins, 2015). Traumatic injuries to nervous system of the horse are a relatively frequent occurrence and affect the central nervous system as well as the peripheral nerves. An older study from Europe reported central nervous system (CNS) trauma to account for 22% of neurologic disorders (Feige et al., 2000), which is similar to an Australia report in which CNS trauma accounted for 24% of neurological case (Tyler et al., 1993). Feige et al. (2000) reported a diagnosis of traumatic brain injury (TBI) in 5 out of 22 (23%) horses that were presented for traumatic neurologic disease, whereas 17 of the 22 (77%) were diagnosed with (spinal cord injury) SCI. Tyler et al.(1993) reported 47 cases of TBI and cranial nerve disease in 107 (44%) horses examined, whereas 60 horses in this group had SCI (56%). The damaging effects of head trauma usually are focused on the portions of the brain adjacent to (coup) and opposite (contrecoup) the site of impact. Additionally, the brain is subjected to other forces after trauma, such as rotational and shock wave forces. (Furr and Reed, 2015)

Clinical syndromes as a result of traumatic injury to the CNS are: abnormal level of consciousness, abnormal behavior, cranial nerve deficits, vestibular disease, tetra· and paraparesis or paraplegia, and cauda equina syndrome. Treatment regimens for CNS injury are directed toward reducing inflammation and swelling, halting secondary injury mechanisms, and promoting regenerative and plasticity mechanisms to improve functional recovery. (Furr and Reed, 2015).The most accurate prognosis is based on repeated detailed neurological examinations with assessment of the rate of progression or resolution and responses to specific therapeutic measures.

The potential of novel treatments as Actovegin, was introduced as a potential multimodal therapy for the management of the neurological diseases in humans. A number of beneficial effects of Actovegin have been demonstrated (Stelmakh et al., 2016). The most important of them include enhanced cellular glucose uptake (Buchmayer et al., 2011, Machicao et al., 2012), improvement of oxygen utilization and energy metabolism (Sondergard et al., 2016), neuroprotective effects (Elmlinger et al., 2011), reduction of oxidative stress and apoptosis (Dieckmann et al., 2012), accelerated wound healing and improvement of blood microcirculation. This case report describes the posttraumatic evolution and treatment in a nine years old stallion for sport, which landed on its poll area during a riding competition.

Material and methods

A 9 year old stallion, Hungarian Sport Horse breed, 400kg bodyweight, had an accident during a riding competition, falling over an obstacle, landed on its poll area. Immediately after the accident, the horse was standing up presented circling to the right side. A few minutes later, the horse received a single dose of dexamethasone (0.05mg/kg, IM) but result was uncertain. 10 days later, the horse was brought to the clinic for examinations. The evaluation has begun by a general observation of the horse, including its attitude and alertness, head and body position, position of the limbs, and symmetry of muscle development, continuing with cranial nerves and spinal cord examination. Blood chemistry, hematology and X-ray of the skull base and cervical spine were done.

Results and discussions

The purpose of the examination was to develop both a differential diagnosis list and to determine the neuroanatomic localisation of any suspected abnormalities. Horse suffering from neurological damage as a result of direct trauma may consequently be very difficult to assess accurately and considerable skill must be employed to definitively identify the site and extent of the lesion.

At 10 days after the accident, the horse presented apathy with the neck slightly deviated to the right and pain at the poll area. Consciousness and mental alertness is mediated by the ascending reticular activating system of the central nervous system (CNS) and the cerebral hemispheres. Adopting abnormal postures are clear signs of cerebral disease.

Examination of cranial nervs

The menace reflex was absent, which could indicate defective vision, paralysis of the eyelids or serious depresion of consciousness. The appropriate response was to blink the eye and move the head away. It should be noted, however, that cerebellar disease can result in a loss of the menace response in a visual horse.

Pupillary reflex revealed a lack of pupillary contraction of both eyes. In normalcy, a bright light directed into one eye should result in constriction of both the ipsilateral eye as well as the contralateral eye (consensual response). The pathway for this response involves the optic nerve and chiasma, then through the optic tracts in the mid• brain to the oculomotor nuclei. The nerve tracts for the pupillary light response are within the brainstem and are not affected by lesions of the visual cortex. A widely dilated pupil in a visual eye suggests oculomotor nerve damage—there will be no direct or consensual light response (Furr and Reed, 2015). In some cases, trauma to the poll or frontal area may cause bilateral blindness with mydriatic unresponsive pupils. The prognosis from recovery of vision is poor (Lorenz et al., 2011).

Nistagmus test is referred to as a "normal vestibular" nystagmus, and its presence suggests an intact vestibular system, as well as normal function of cranial nerve (CN) 3 (Oculomotor N.), 4 (Trochlear N.), and 6 (Abducens N.). Spontaneous or positional nystagmus is always abnormal. Intact sympathetic innervation to the eye is evaluated by observation for Horner's syndrome. (Furr and Reed, 2015). Physiologic nystagmus was normal to the left side, but absent to the right side in our horse. Central vestibular disease results in a nystagmus, which varies with different head positions ("positional nystagmus"), while peripheral vestibular disease is nonpositional.

The head was examined for facial symmetry, reflecting function of CN 7 (Facial N.), and facial sensation, which is mediated by CN 5 (Trigeminal N.). We noticed a slight lingual protrusion, sluggish mastication, hypotonic tongue and normal swallowing. Swallowing is mediated by input from both the glossopharyngeal nerve (CN 9) and the vagus (CN 10). Tongue tone is dependent upon the function of the hypoglossal nerve (CN 12) and can be tested by grasping the tongue and applying gentle traction. Inability to resist or withdraw the tongue suggests hypoglossal nerve damage.

Examination of the neck

The neck was examined with particular attention paid to symmetry, abnormal sweating patterns, presence or absence of masses or deviations from normal anatomy presence or absence of pain on manipulation and flexibility. Our horse had the neck slightly deviated to the right. Abnormalities of the neck generally suggest a lesion within the bony cervical spinal column or skull. Cervical reflexes were examined for cervical spinal cord disease. The cervico-auricular reflex (tapping the skin between the jugular groove and the crest at the level of C2) and local cervical reflex between C3 and C6 (taping the skin in the area), resulting in local muscle contraction, revealed no abnormalities.

Examination of the body

Symmetry, strength and presence or absence of muscle mass changes were evaluated. The panniculus reflex performed by running a pen down the length of the horse bilaterally from the neck to the tail of the midline, will indicate if skin sensation and control of muscles underlying the skin is intact. The cutaneous trunk reflex, perianal skin reactions, tail carriage and anal tone were normal. Abnormalities of tail strength and anal reflex may be seen in horses with botulism or inflammation of the cauda equina. Elevated tail carriage is commonly seen in horses with equine lower motor neuron disease (Furr and Reed, 2015).

Limb placement tests in horses have a low sensitivity for the detection of abnormalities of the CNS, and these tests should be coupled with careful observation of the foot placement during dynamic maneuvers. Abnormal limb positions, particularly a base-wide stance in the front limbs, may also be associated with vestibular disease (Furr and Reed, 2015). Our horse presented paresis (dragging hind limbs, repeated tripping), ataxia (unsteadiness, stumbled limbs), dysmetria (hypermetria right front limb), tendency to fall at walk and trot and a base-wide position of hind limbs. Gait was evaluated as an assessment of brainstem, spinal cord and peripheral nerves function.

Radiography is the most practical diagostic imaging technique to use initialy in traumatic injury to the head. We analyzed skull base and cervical spine at our horse, but no lesions were observed. Radiographic interpretation of the base of the skull is made difficult by the complex three-dimensional anatomy of the equine skull and radiographic superimposition of structures. Absence of an obvious fracture line radiographically does not preclude a diagnosis of basilar skull fracture (Lorenz et al., 2011).

Hematological examinations (total and differential leukocyte count, red blood cells count, hemoglobin concentration, hematocrit, mean corpuscular hemoglobin concentration and platelet count) were carried out revealing moderate neutrophilia and mild lymphopenia. No **biochemical** parameters were pathological modified.

Diagnosis. Our horse had cerebellar syndrome and probably lesions of brainstem with a reserved prognosis regarding the future riding competitions.

Prognosis depends primarily on severity of primary injury and on the neuroanatomic location and extent of CNS damage. Recovery of function in the short term can be helpful in determination of long-term prognosis. (Southwood and Wilkins, 2015). Some horses are able to return to their intenden use despite persistent neurologic deficitis (Feary et al., 2007). When injury causes either loss of function, timely treatment is extremely important (Ragle, 1993).

Treatment regimens for CNS injury was directed toward reducing inflammation and swelling, halting secondary injury mechanisms, and promoting regenerative and plasticity mechanisms to improve functional recovery

Ten days days after the accident, it was established a treatment with ketoprofen (2.2mg/kg IV) and vitamins B1 and B6 (10 ml IM). After five days of treatment, the horse was showing ataxia and right eye vision deficits. After another ten days, it was tried a combination of ketoprofen (five days) and vitamins B1 and B6 (five days) with a deproteinized hemoderivative of calf blood product, Actovegin®, (1600mg/day, IV, 14 days), with good results: reduced ataxia and mild right

eye vision deficits.

Actovegin is an ultrafiltrate of calf blood, composed of more than 200 biological substances. Actovegin's main constituents are lowmolecular weight substances, including amino acids, biogenic amines and polyamines, sphingolipids, hexoses, eicosanoids, lactate, succinate, choline, vitamins, adenosine monophosphate (AMP) and inositol phospho-oligosaccharides (IPOs). Only small amounts of acylcarnitines, phospholipids, free fatty acids, and oxysterols have been detected; prostaglandins, oxidized polyunsaturated fatty acids, and bile acids are present in even smaller amounts. In a recently published experimental study, Elmlinger and al., (2011) found that Actovegin increased the number of neurons and excitatory synapses. They also observed that the drug exhibited potent anti-apoptotic and anti-oxidative effects. More recent studies suggested that Actovegin has neuroprotective effects on neurons by increasing neuron and synaptic numbers (Skoog et al., 2012, Stelmakn et al., 2016). Our horse returned to easy training technique preparing for the next riding competitions.

Conclusions

1. After 14 days of Actovegin treatment, the horse with cerebellar syndrome and probably lesions of brainstem, remained with reduced ataxia and mild right eye vision deficits, with reserved prognosis regarding the future riding competitions.
2. These data, coupled with positive results from this study case, served as a foundation for the design of a new trial investigating the efficacy and effects of Actovegin in equine head trauma.

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