Post anaesthetic myelopathy in the horse: a case report

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ABSTRACT: The first case of post anaesthetic myelopathy in a horse is described. A two year old 530 kg Shire stallion underwent surgical removal of a granuloma in the ventral sternal region under general inhalation anaesthesia in dorsal recumbency. Total duration of the operation was 85 min. The anaesthesia was uneventful except for profuse sweating and arterial hypertension observed during the whole period. During recovery the horse was not able to stand, and flaccid paralysis of hind limbs, absence of reaction to an induced pain stimulus on the hind limbs and no patellar or anal reflex was recorded; in addition, tail tonus was weak. Panniculus reflex was absent distally from the 17th intercostal space. Head, neck and front limb movement was not affected. The horse did not respond to treatment by intravenous administration of dexamethasone, hypertonic or isotonic saline. The status deteriorated and the horse was euthanised 4 h after the end of anaesthesia. The main pathological findings were haemorrhage, oedema and malacia of L5–L6 spinal cord segments and *cauda equina*. Histological examination of the spinal cord revealed haemorrhage and areas of necrosis predominantly in the grey matter of L5 and L6 segments. Impairment of spinal cord perfusion due to haemodynamic changes associated with dorsal recumbency and general anaesthesia is presumed. Predisposition factors could include young age, dorsal recumbency and high weight.

Keywords: general anaesthesia; horse; myelomalacia

Equine post anaesthetic myelopathy is a rare complication of equine general anaesthesia. There have been 30 cases reported to date (Schatzmann et al. 1979; Blakemore et al. 1984; Zink 1985; Brearley et al. 1986; Yovich et al. 1986; Lerche et al. 1993; Lam et al. 1995; Raidal et al. 1997; Joubert et al. 2005; van Loon et al. 2010; Ragle et al. 2011). The syndrome has been described mostly in young horses anaesthetised for elective procedures in dorsal recumbency. The main clinical signs include an inability to stand after general anaesthesia, paralysis of the hind limbs and absence of panniculus reflex from the middle to the caudal thorax distally. Prognosis is fatal. *Post mortem* examination findings include variable degrees of oedema, haemorrhage and necrosis at different levels of the spinal cord with grey matter most often affected. Definitive causes of equine post anaesthetic myelopathy have not yet been identified. Vascular pathogenesis, failure of oxygenation of the spinal cord and immaturity or individual variation of the spinal cord microcirculation in young horses have been proposed as possible reasons (Schatzman et al. 1979; Blakemore et

al. 1984; Zink 1985; Brearley et al. 1986; Yovich et al. 1986; Lerche et al. 1993; Lam et al. 1995; Raidal et al. 1997; Joubert et al. 2005).

This article describes a case of equine post anaesthetic myelopathy.

Case description

A two year old 530 kg Shire stallion was admitted in 2010 for surgical removal of granuloma with fistula in the ventral sternal region. Clinical examination of the horse did not reveal any abnormalities in general health status. The horse was fasted overnight. A 14 G catheter (Vasovet; BBraun, Germany) was introduced into the right jugular vein and 1.1 mg/kg of flunixin meglumine (Meflosyl; Pfizer, Spain) was administered intravenously. The horse was sedated with 1.1 mg/kg of xylazine (Xylapan 2%; Vetoquinol Biovet, Gorzow Wlkp, Poland) intravenously and anaesthesia was induced with 0.02 mg/kg of diazepam (Apaurin; Krka, Novo Mesto, Slovenia) and 2.2 mg/kg of

ketamine (Narketan 10%; Vetoquinol SA, Lure Cedex, France) intravenously. When the horse became recumbent it was intubated orotracheally with an endotracheal tube (Smiths Medical Pm Inc, Waukesha, USA) of suitable diameter, moved into the operation theatre and placed into dorsal recumbency.

The endotracheal tube was connected to a large animal circle system (Stephan GmbH; Gackenbach, Germany). Anaesthesia was maintained with isoflurane (Aerrane; Baxter SA, Lessines, Belgium) with oxygen and a mixture of oxygen/air. Intermittent positive pressure ventilation in a control pressure setup with a peak inspiratory pressure 22–26 cm H_2O was used. The end tidal concentration of isoflurane ranged between 1.3% and 1.6% and end tidal concentration of CO_2 between 5.3 kPa and 5.7 kPa. Respiratory rate was six breaths per minute. An arterial catheter (Surflo 22 G; Terumo, Leuwen, Belgium) was placed into a facial artery for arterial blood pressure measurement and blood sample withdrawal. The pressure transducer was zeroed at the level of the right atrium. EKG (electrocardiography) and SpO_2 (saturation of arterial blood from pulse oxymetry) were monitored continuously throughout the anaesthesia. Supportive therapy consisted of crystalloid administration (Infusio Ringeri Mediekos; In Mediec, Luhacovice, Czech Republic) at a rate of 10 ml/kg/hour throughout the anaesthesia.

Blood gas analysis of arterial blood was performed immediately after catheter introduction using Rapidlab 855 (Rapidlab 855; Bayer, Germany). The recorded PaO_2 value was 48.47 kPa, the $PaCO_2$ was 6.7 kPa, the arterial pH was 7.399 and the PaO_2/FiO_2 ratio was 409; therefore, oxygen was switched to a mixture with air (FiO_2 decreased from 92 to 58). Total duration of anaesthesia was 85 min. The horse sweated profusely throughout the duration of anaesthesia and arterial hypertension was recorded with highest values over the last 20 min of anaesthesia. Systolic and diastolic blood pressure values (mean ± SD) throughout the anaesthesia and in the last 20 min were 115 ± 6 mmHg to 79 ± 6 mmHg and 121 ± 4 mmHg to 86 ± 4 mmHg, respectively. No haemodynamic support medication was used. Nystagmus was observed on two occasions during anaesthesia (on 60th and 80th minute) and bolus of ketamin was administered (0.2 mg/kg *i.v.*).

The granuloma was completely removed and the skin was sutured. Penicillin (22 000 UI/kg, Norocillin; Norbrook Laboratories Limited, Newry, Ireland) and tetanus antitoxin (Clotean; Bioveta Ivanovice na Hane, Czech Republic) were administered intramuscularly. After completion of the surgery the horse was placed into a padded recovery stall in the left lateral position. The endotracheal tube was removed after the swallowing reflex had returned, 9 min after the end of anaesthesia, and sedation was administered (0.15 mg/kg of xylazin *i.v.*). During recovery nasal oxygen was administered at a rate of 15 l/min. The horse was quiet during recovery, and first attempt to stand was recorded 25 min after the end of anaesthesia. The horse was able to lift the head and move with the front limbs without any hind limb movement. This status persisted for 2 h after the anaesthesia had been terminated. The horse was not able to move from lateral recumbency to sternal and no movement of hind limbs was observed. 0.025 mg/kg of acepromazine (Vanastress; Vana, Austria) was administered intramuscularly due to suspected myopathy. Clinical and neurological examination revealed soft and non-painful muscles, a heart rate of 40/min, rectal temperature of 37.5 °C and a respiratory rate of 12/min. The horse was calm, although it occasionally responded to unpleasant handling on the head or trunk with movement of the front limbs and lifting of the neck and head. Pupillary light reflex, palpebral reflex, sensitivity in the ears and nostrils were all present. No spontaneous nystagmus or strabismus on the right side of the head was found. Anal reflex was absent and tail tone was very weak. Flaccid hind limb paralysis was found. Patellar reflex was absent (right hind limb), while triceps reflex was present (right front limb). Perception of induced pain was absent on the hind limbs. Panniculus reflex was not present distally from the 17th intercostal space.

A blood sample for biochemistry was taken and levels of creatine kinase 734.4 IU/l (12.24 µkat/l) and aspartate aminotransferase 638.4 IU/l (10.64 µkat/l) were determined.

Treatment consisted of intravenous administration of dexamethasone (0.1 mg/kg, Dexadreson; Intervet International B.V., Netherlands), hypertonic saline (4 ml/kg) and isotonic saline (20 ml/kg). The loss of panniculus reflex spread cranially up to the 14th intercostal space and breathing became shallower suggesting respiratory muscle involvement. A diagnosis of post anaesthetic myelopathy was suspected and the horse was euthanised 4 h after the end of anaesthesia with the owner's agreement.

Necropsy revealed haemorrhage and oedema of the spinal cord at the L5–L6 level and *cauda equina*.

Other findings included oedema of the large colon and *caecum* with prominent pale 1–2 mm nodules, enlarged mesenteric lymph nodes and hyperaemic parenchymatous organs (kidneys, liver, spleen). Histological examination of the spinal cord revealed haemorrhage and areas of necrosis predominantly in the grey matter of L5 and L6 segments. Small areas of necrosis were also localised in the white matter and *cauda equina*. The submucosa of the large colon was oedematous with eosinophilic and neutrophilic infiltrate and hyperplasia of lymphatic follicles was noted. The kidneys and liver were hyperaemic. A diagnosis of myelomalacia of L5–L6 spinal cord segments and *cauda equina* was made.

DISCUSSION AND CONCLUSIONS

The case described here is the first report of equine myelopathy after general anaesthesia detected in the Czech Republic. To date 30 cases of post anaesthetic myelopathy have been described, with the first case reported in 1979 (Schatzmann et al. 1979; Blakemore et al. 1984; Zink 1985; Brearley et al. 1986; Yovich et al. 1986; Lerche et al. 1993; Lam et al. 1995; Raidal et al. 1997; Joubert et al. 2005; van Loon et al. 2010; Ragle et al. 2011). Most commonly the affected horses were young, up to 24 months old, males and heavily muscled breeds. Two horses in the group were nine and 10 years old. The age distribution of the group was six months to 10 years. Median weight was 381 kg (range 214–650 kg). Horses were admitted mostly for elective surgery (orthopaedic or soft tissue surgery) and, according to the preoperative examination, were considered to be healthy (Ragle et al. 2011). Lam et al. (1995) described this disorder in a seven month old Connemara pony presented as an emergency with herniation of abdominal content following castration. Although there are some exceptions in age and weight distribution in the group of affected horses, it appears that immature heavily muscled or large breed horses are prone to the development of post anaesthetic myelopathy. No breed predisposition has been described, although in an earlier report six Friesians and seven draught horses were found from a total of 30 cases (Ragle et al. 2011). The horse described here was also young and well-muscled and was considered healthy except for a granuloma in the ventral sternal region.

All but one horse were positioned in dorsal recumbency and underwent surgery with inhalation anaesthesia (Ragle et al. 2011). The one exception was a two year old Thoroughbred colt which underwent castration in right lateral recumbency with total intravenous anaesthesia (Raidal et al. 1997). According to the number of anaesthetics used in all the cases, there seems to be no influence of anaesthetic protocol related to this disorder.

Median anaesthetic time was 90 min (range 25 to 205 min) (Ragle et al. 2011). In our case anaesthesia duration was 85 min. The results of the study of Ragle et al. (2011) where both short-term and long-term anaesthesias were employed, together with our own data suggest that the occurrence of this condition is not dependent on the length of anaesthesia but on other factors.

The most common clinical signs include an inability to stand during recovery, flaccid hind limb paralysis, absence of patellar reflex and absence of a reaction to an induced pain stimulus on the hind limbs. The panniculus reflex is usually decreased or absent distal to the lesion. Perineal and anal reflex and tail tonus may be absent and the horse may be incontinent. Neurological deficits may be progressive. The function of the neck and front limbs is usually normal (Blakemore et al. 1984; Zink 1985; Yovich et al. 1986; Lerche et al. 1993; Lam et al. 1995; Raidal et al. 1997; Joubert et al. 2005; van Loon et al. 2010; Ragle et al. 2011). In a few cases the horses were able to stand following anaesthesia but collapsed later. Zink (1985) described a horse which stood with assistance a day after the surgery but subsequently returned to the lateral position. The horse described by Brearley et al. (1986) was able to stand following umbilical herniorrhaphy but showed signs of colic and weakness of hind limbs and was recumbent on the next day. The horse was not able to stand after the second surgery which was performed to rule out intestinal damage. The horse described by Raidal et al. (1997) stood uneventfully but collapsed after 5 h with flaccid paralysis of both hind limbs. The horses described by Lam et al. (1995) and van Loon et al. (2010) underwent two surgeries; the first recovery was uneventful but the horses were not able to stand after the second surgery. Other less common clinical signs include profuse sweating, arterial hypertension and tachycardia during anaesthesia which was not related to surgical manipulation (Zink 1985; Brearley et al. 1986; Lerche et al. 1993). Yovich et al. (1986) described a disturbance of spontaneous respiration during recovery, suggesting paralysis of intercostal muscles.

Differential diagnoses include femoral nerve paralysis, peroneal nerve paralysis, generalised myopa-

thy, hind limb adductor myopathy, hyperkalaemic periodic paralysis, traumatic fracture or luxation (Grandy et al. 1987; Dodman et al. 1988; Dyson et al. 1988; Lindsay et al. 1989; Trim 1997). Clinical signs in our case were typical for the described condition. In addition, profuse sweating and arterial hypertension without tachycardia was observed throughout the anaesthesia which did not correlate with surgical manipulation. Neurological signs progressed during recovery, loss of panniculus reflex spread cranially and spontaneous breathing became shallower. Myopathy was ruled out, muscles were soft and non-painful and serum concentrations of creatine kinase and aspartate aminotransferase were only slightly increased which could be related rather to positioning of the horse than to general anaesthesia. In addition, arterial blood pressure was maintained at the increased levels throughout the anaesthesia. Incidence of post anaesthetic myopathy is associated with hypotension during general anaesthesia (Grandy et al. 1987). Peripheral nerve paralysis was ruled out too because in this condition withdrawal reflex, anal and tail tone and skin sensation are usually present (Trim 1997).

Reported treatments have not been successful and have consisted of corticosteroids, nonsteroidal antiinflamatory drugs, calcium, dimethyl sulfoxide, sedatives, vitamin E and intravenous fluid administration, padding and rolling (Zink 1985; Lerche et al. 1993; Lam et al. 1995; Raidal et al. 1997; Joubert et al. 2005; Ragle et al. 2011). Horses were either euthanised or died between 2 h and 192 h after the end of anaesthesia (Ragle et al. 2011). Our case was euthanised 4 h after the end of anaesthesia due to the deterioration of the status and poor prognosis. Treatment did not elicit any improvement in clinical signs.

Pathological findings in all reported cases include haemorrhage, oedema and malacia of the grey matter of the spinal cord at different levels. Thoracic, thoracolumbar, lumbar, lumbosacral to sacral spinal cord have been described to be affected. Histologically, congestion, haemorrhage, necrosis and ischaemic neuronal damage have been confirmed (Schatzmann et al. 1979; Blakemore et al. 1984; Zink 1985; Brearley et al. 1986; Yovich et al. 1986; Lerche et al. 1993; Lam et al. 1995; Raidal et al. 1997; Joubert et al. 2005). In our patient the grey matter of the spinal cord was affected by haemorrhage and necrosis but small areas were found in the white matter too. Malacic changes were also found in the *cauda equina*.

The ethiopathogenesis of equine post anaesthetic myelopathy is not fully understood. The changes observed in the affected spinal cord are most consistent with compromised oxygenation. However, the mechanisms leading to this impaired oxygenation are unknown (Ragle et al. 2011). Most authors have proposed a vascular pathogenesis due to haemodynamic changes during general anaesthesia and dorsal recumbency. During general inhalation anaesthesia a decrease in cardiac output and peripheral vascular resistance is most commonly observed (Steffey and Howland 1978; Steffey and Howland 1980). Schatzmann et al. (1979) has suggested that in dorsal recumbency there is a pooling of blood in the spinal area and venous congestion in the paravertebral and venous sinus systems due to *vena cava* compression. This may, together with decreased cardiac output and blood pressure result in hypoxic damage to the vessels supplying the spinal cord. Moreover, it has been postulated that the vasculature of the spinal cord may be not fully developed in young horses and that some individual variation of the spinal cord microcirculation could play a role.

Blakemore et al. (1984) suggested that according to the localisation of pathologically changed parts of the spinal cord in their case, i.e. that part which drains into the *posterior vena cava*, the cause could be the failure of perfusion by dorsal and ventral spinal arteries and obstruction at the capillary or post capillary venous level. Failure of spinal cord perfusion could occur due to hypotension and increased pressure on the *posterior vena cava* in dorsal recumbency as a result of pressure of abdominal viscera.

A vascular pathogenesis is also supported by the findings of Raidal et al. (1997). Extensive haemorrhage and oedema occurred in the dependent side of the spinal cord in the horse in lateral position suggesting that haemodynamic disturbances and compromised venous drainage could play a primary role.

Changes in the spinal cord perfusion could be more profound in heavy horses. Abdominal viscera may cause extensive venous compression which may lead to stagnant hypoxia (Brearley et al. 1986).

Ischaemic damage of the spinal cord and vessels usually leads to necrosis of neuroectodermal cells (malacia) and haemorrhage (Lerche et al. 1993; Ragle et al. 2011). Experimental ischaemia of the spinal cord was reported to cause myelomalacia in rabbits (DeGirolami and Zivin 1982).

The grey matter of the spinal cord is affected more often than the white matter, which could be due to its

higher susceptibility to hypoxia than white matter. The grey matter has a higher metabolic rate and concentration of blood vessels and contains neuronal cell bodies (Zink 1985; Yovich et al. 1986).

Additionally, ischaemia of the spinal cord could be caused by arterial vasoconstriction due to increased catecholamine levels in a few of the described cases. Increased systemic catecholamine levels may induce vasoconstriction of intramedullary vessels of the spinal cord (Osterholm 1974). Zink (1985) observed profuse sweating during general anaesthesia which may be indicative of a prolonged period of epinephrine release from the adrenal medulla. This could, in addition to the influence of dorsal recumbency and general anaesthesia, affect spinal cord blood flow. Systemic arterial blood pressure was not measured in this case. A period of arterial hypertension and tachycardia was recorded during anaesthesia in the cases of Brearley et al. (1986) and Lerche et al. (1993). Haemorrhagic lesions in the grey matter were associated with arterial hypertension as a consequence of spinal cord impact injury in cats (Griffiths et al. 1978).

It is possible that, in agreement with other authors, myelomalacia and haemorrhage in our case was the result of impaired spinal cord perfusion. Arterial hypertension was recorded throughout the period of anaesthesia with the highest values during the last 20 min. Hence haemodynamic support by inotropic drug administration was not performed. A possible cause of arterial hypertension could be adrenergic stimulation and increased catecholamine levels which might cause systemic vasoconstriction, including spinal cord vessels, with decreased spinal cord perfusion and ischaemia as a consequence. Additionally, the influence of decreased cardiac output could contribute to decreased spinal cord perfusion. The possibility of an increased adrenergic state is supported by the profuse sweating seen throughout the period of anaesthesia. To confirm this hypothesis systemic vascular resistance and cardiac output should be measured. We do not believe that surgical manipulation had an influence on the increased blood pressure because the end tidal concentration of isoflurane was kept at a higher level, heart rate and respiratory rate were low, palpebral reflex was reduced and nystagmus was not present except on two occasions at the end of anaesthesia. Ketamine was administered on these occasions and therefore it is possible that this period of highest blood pressure could have been additionally caused by the sympathomimetic effect of ketamine. Arterial hypertension could moreover have caused a haemorrhage in the spinal cord in our case. However, the reason for an increase in the adrenergic state in our patient is unknown. Additionally congestion of the spinal cord vessels due to *vena cava* compression could contribute to haemorrhage and ischaemia of the spinal cord. *Vena cava* compression occurs in dorsal recumbency due to the pressure of abdominal content and it can therefore explain why this condition occurs mostly in horses in dorsal recumbency. It is possible that in heavy horses there are more profound haemodynamic changes and more pronounced pressure on the *vena cava* due to a larger amount of abdominal content.

On the other hand, it is known that clamping of the thoracic aorta and the subsequent distal ischaemia results in sympathoadrenal activation and an increase in catecholamine levels (Normann et al. 1983). Spinal cord impact injury leads to arterial hypertension (Griffiths et al. 1978). Therefore, it is possible that primary spinal cord ischaemia induced catecholamine release and the arterial hypertension seen in our case. A possible explanation could be, similarly as for *vena cava* compression, increased pressure on the abdominal part of the aorta. Therefore, it is unclear if the spinal cord ischaemia was the cause or a consequence of the increased adrenergic state.

Lerche et al. (1993) suggested that starvation prior to general anaesthesia should be longer than 12 h in large young horses, in order to attempt to decrease the degree of *vena cava* compression by the weight of abdominal viscera. A position slightly oblique to the dorsal was suggested by Blakemore et al. (1984). Despite these recommendations this condition still occurred in horses in oblique or lateral positions suggesting that changes in positioning do not completely eliminate the risk (Raidal et al. 1997; Ragle et al. 2011).

Post anaesthetic myelopathy is a rare but fatal complication of general anaesthesia in the horse. A few predisposition factors have been identified and include a young age, greater weight and dorsal recumbency. Arterial hypertension during general anaesthesia without any obvious cause should be taken into account as a possible indication of this condition. The ethiopathogenesis of the condition has not yet been elucidated but clinical signs are most consistent with impaired spinal cord perfusion. Unfortunately, at present, it is difficult to determine possible preventative measures.

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