HORSES AND OTHER EQUIDS

Delayed recovery from general anaesthesia associated with pre-existing neurological disease in a horse

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SUMMARY

A seven-year-old gelding presented for surgical management of right-sided olecranon bursitis. Preanaesthetic examination revealed a right-sided head tilt which had been present since birth. General anaesthesia and surgery went smoothly without incident. Recovery was prolonged and the horse made no attempt to stand despite regaining motor function consistent with recovery from general anaesthesia. Increasingly loud auditory stimuli and mild physical stimuli yielded no attempt to move into sternal recumbency or stand. After over two hours in the recovery box an equine neurologist suggested a diagnosis of vestibular disease and proposed that without a horizon the horse would not be able to orientate itself, therefore advising that the horse be shown the horizon. The outside recovery box door was opened allowing the horse to see the horizon, on which the horse made an immediate coordinated attempt to stand, standing successfully on its first attempt.

BACKGROUND

This case report describes a previously unreported recovery complication following general anaesthesia in a horse with pre-existing neurological disease. Although a head tilt is a rare finding in preoperative clinical examinations in equine patients, it is possible as in this case, that one may be noted as a concurrent finding. This report highlights the potential for pre-existing neurological clinical signs to impact anaesthetic recovery and the need for clinicians to disseminate this information to owners. The use of a multidisciplinary approach to problem solving in this case of prolonged recovery resulted in a successful outcome.

CASE PRESENTATION

A seven-year, nine-month-old Thoroughbred gelding weighing 558 kg presented to The Dick Vet Equine Hospital, University of Edinburgh, for investigation and treatment of right-sided olecranon bursitis. Ultrasound examination revealed a large capsulated structure with multiple compartments filled with anechoic fluid. There was a history of multiple unsuccessful attempts at conservative management; consequently the owner presented the horse for elective surgical excision under general anaesthesia. It was noted on admission that the horse had a persistent right-sided head tilt which the owner reported had been present since the horse was a foal; the horse was in full work with no ataxia or any other neurological signs reported. Neurological examination was carried out by a senior equine medicine clinician prior to general anaesthesia; menace reflex, pupillary light reflex and all cranial nerve reflexes were reported to be normal. A pre-general anaesthetic clinical examination was carried out, the horse was hyper-responsive and head-shy but amenable to handling and was similar when approached on both the right and the left sides. Heart rate was 36 beats per minute with a regular rhythm and no heart murmur was auscultated on either the left or right side. Respiratory rate was 10 breaths per minute with a regular breathing pattern, and auscultation of the left and right hemithorax was within normal limits. Mucous membranes were pink and moist, and capillary refill time was less than two seconds. Rectal temperature had been taken earlier in the day and was 37.7°C; it was elected not to repeat this to avoid causing unnecessary stress to the horse.

Acepromazine (30 mcg/kg; Calmivet, Vetquinoil, France) was given by intramuscular injection and 20 minutes after administration a 13 cm, 14 Ga, polyurethane jugular venous catheter (Milacath extended use, Mila International) was placed percutaneously, aseptically in the left jugular vein. The horse was moved to the induction box 40 minutes after the administration of acepromazine and sedated with 80 mcg/kg romifidine (Sedivet, Boehringer Ingelheim, Berkshire, UK) by intravenous injection via the jugular catheter. Sedation was deemed adequate with a wide-based stance, low head carriage, no response to environmental noise and minimal ataxia after five minutes. Anaesthesia was induced with 2.2 mg/kg ketamine (Aneastamine, Animalcare, York, UK) and 50 mcg/kg diazepam (Diazepam injection, Hameln Pharmaceuticals, Gloucester, UK) by intravenous injection via the jugular catheter. Assisted induction against a wall was smooth. The horse’s trachea was intubated with a 28 mm internal diameter silicone endotracheal tube with no difficulties. The horse was winched onto the table, placed into left lateral recumbency and connected to a circle breathing system (Tafonius, Vetronic Services, Abbotskerswell, UK) which had been prefilled with oxygen and 5 per cent sevoflurane (Sevoflo, Zoetis, Leatherhead, UK). Anaesthesia was maintained with sevoflurane in oxygen with end tidal anaesthetic agent values of 1.9 per cent to 2.2 per cent and oxygen flows of 2–4 litres per minute. Monitoring included invasive arterial blood pressure, electrocardiography (ECG),...
with a tidal volume of 7000 mL, peak inspiratory pressure of 19 cm H\textsubscript{2}O and respiratory frequency of 8 breaths per minute although the frequency was reduced 10 minutes before the end of surgery to encourage earlier return to spontaneous ventilation. \textit{PaO\textsubscript{2}} at 25 minutes was 438 mmHg and at 90 minutes was 158 mmHg. Hartmann’s solution (Vetivex 11, Dechra, Shrewsbury, UK) was administered at approximately 10 mL/kg/h. Morphine 60 mg, (Morphine Sulfate Injectable, Martindale Pharmaceuticals, Romford, UK) was given by slow intravenous injection 25 minutes after induction, 1.1 mg/kg of flunixin meglumine (Flunixin Injection, Norbrook, Newry, UK) was given by slow intravenous injection 30 minutes after induction and 6.6 mg/kg gentamicin (Genta-Equine, Dechra, Shrewsbury, UK) was administered by slow intravenous injection 45 minutes after induction. Total general anaesthesia time was 95 minutes, at 90 minutes 20 mcg/kg romifidine was given by intravenous injection and shortly before the horse was disconnected from the breathing system 5 mL of 0.1 per cent phenylephrine (Phenylephrine Minims, Bausch and Lomb, Kingston On Thames, UK) was sprayed intranasally into each nostril as is routine practice at the present institution.

Following completion of surgery, the horse was moved to the recovery box and placed in left lateral recumbency with the affected limb uppermost. Return to spontaneous ventilation was noted shortly after disconnection from the circle breathing system. The horse’s trachea was extubated shortly after return of spontaneous ventilation; bilateral nasal airflow was present and the tidal volume was judged, subjectively, to be adequate. Supplemental oxygen was provided at 15 litres/minute intranasally via oxygen tubing. After 20 minutes horizontal nystagmus was noted but the horse made no attempt to move into sternal recumbency or move its head. Nystagmus was noted intermittently until shortly before the horse stood. After 30 minutes it was noted that the horse was moving its ears in response to auditory stimuli but it still made no attempt to move. Around 60 minutes after entering the recovery box there had been no attempt at movement. Increasingly loud auditory stimuli which included loud human voices and gentle banging on the recovery box door elicited no response other than ear movement. Due to the complete lack of movement the horse was examined for signs of myopathy; the latisimus dorsi and gluteal muscles were not firm, tense, painful and hot and therefore myopathy was deemed unlikely. The horse had a normal menace response and did not appear to be obtunded. Mild physical stimuli which consisted of gentle consistent pressure on the gluteal region yielded normal movement in all four legs but no attempt to stand. Approximately 90 minutes after entering the recovery box atipamezole (Antisedan, Zoetis, Leatherhead, UK) was administered at a dose of 100 mcg/kg by intramuscular injection to antagonise any potential residual effects of the romifidine; no effects were observed after atipamezole administration. Shortly afterwards attempts were made to lift the horse into sternal recumbency which were unsuccessful. The horse seemed to push against personnel and attempt to lie back down when attempts were made at moving this way and at this point the horse appeared to be fully conscious. Finally, after a total of 120 minutes, an attempt to place a sling to assist the horse with standing was made which was unsuccessful as the horse started to kick out backwards and panic. At this point an equine neurologist was contacted for advice. Based on the history of a head tilt and the behaviour in recovery the neurologist suggested a tentative diagnosis of vestibular disease and proposed that without a horizon the horse would not be able to orientate itself. She suggested opening the outside recovery box door to show the horse the horizon. The recovery box door was opened wide so the horse could see the horizon and immediately the horse made a successful attempt to stand and stood on the first attempt without evidence of ataxia or weakness. After recovery the previously noted mild head tilt was greatly exaggerated. Over a period of several hours the head tilt gradually reduced until it was similar to pre-general anaesthesia.

**INVESTIGATIONS**

The neurologist based her suggestion of vestibular disease as a likely diagnosis in this case on the reported preoperative clinical signs and the behaviour exhibited by the horse in recovery.

**DIFFERENTIAL DIAGNOSIS**

Differential diagnoses for prolonged recoveries were considered in this case. Most potential causes of the prolonged time to standing were ruled out by performing a thorough clinical examination. Post anaesthetic myopathy was deemed unlikely due to the normal motor function noted in all four limbs as well as no evidence of hot firm muscles on palpation. Neuropathy was also considered unlikely due to the normal motor function noted in all four limbs. Hypothermia was removed as a differential diagnosis by taking a rectal temperature which was within normal limits. Hypoxaemia was ruled out due to the fact that the horse was not hypoxaemic on movement into the recovery box; respiratory rate and apparent tidal volume were normal and oxygen was supplemented in the recovery box. Prolonged action of the \(\alpha\)-2 agonist administered for recovery was discussed hence administration of atipamezole, but again this was deemed to be unlikely in this case due to the clinical picture and the time that had elapsed following \(\alpha\)-2 agonist administration. Finally exacerbation of the pre-existing neurological disease was hypothesised to be the cause of the unusual presentation which is when advice was sought from an equine neurologist. Essentially all other possibilities had been ruled out at this point. Following advice from the neurologist the horse stood immediately.

**OUTCOME AND FOLLOW-UP**

Four months after the general anaesthetic a full neurological examination was performed and no neurological abnormalities were detected other than the right-sided head tilt. The right-sided head tilt was noted to be similar in severity to pre-general anaesthesia. It was noted that the horse had a forelimb lameness but this was not a neurological lameness. It was not possible to perform a blindfold test due to the temperament of the horse.

**DISCUSSION**

To the authors’ knowledge no reports exist of general anaesthesia in a horse with a pre-existing head tilt. It is known anecdotally that general anaesthesia of canine patients with vestibular disease can lead to a deterioration in clinical signs during and after the recovery period, however vestibular signs are not...
commonly seen in horses therefore there is limited experience of recovery from general anaesthesia of these patients. In this case no specific underlying cause of the vestibular disease was found although further neurological workup was limited due to financial reasons.

The vestibular system is the primary system responsible for balance, orientation relative to gravitational field and maintenance of the position of the eyes, neck trunk and limbs relative to the head position. Clinical signs associated with vestibular disease include imbalance, vestibular ataxia, abnormal posture, head tilt, strabismus and nystagmus. Vestibular disease is relatively rare in horses in comparison to other domestic veterinary species although there are multiple possible aetiologies. Most published cases are related to temporohyoid osteoarthropathy (THO) which has been reported in horses of different breeds and ages. Other causes of vestibular signs in horses include otitis media, temporal bone fractures, neoplasia, external trauma, lightning strike and idiopathic self-limiting vestibular signs. As no cause for the head tilt was found and it had been present from birth it is unclear what the aetiology behind the clinical signs in this horse were. Alongside the vestibular system vision is also a significant contributor to balance. Since the vestibular system was impaired in this case it was reasonable to assume that optimising visual input by allowing a view of the horizon would give the horse the confidence to stand.

Recovery is an important and high-risk component of equine anaesthesia with multiple studies investigating equine anaesthetic mortality noting a high incidence of complications during recovery from general anaesthesia. To the authors’ knowledge there is no existing definition of prolonged recovery following equine anaesthesia and there are no studies looking specifically at causes of prolonged recovery. In this case the duration to standing was considerably longer than is typical at the present institution where most horses stand within 60 minutes. A recent case report about equine anaesthetic recovery suggested that hypoxaemia, residual drug effects and hypothermia may have contributed to a delayed recovery in a nine-year-old Boulonnais gelding. In the present case these factors were all considered but were deemed unlikely. Oxygenation was good immediately before moving into the recovery box and oxygen was supplemented nasally at 15 litres/minute, however a pulse oximeter was not used at this point. A pulse oximeter would have given us information on the percentage saturation of haemoglobin with oxygen which would have helped to rule out hypoxaemia. Temperature was within normal limits when taken using a rectal thermometer and residual drugs were not deemed to be a contributing factor due to the horse appearing fully conscious. Atipamezole, an α2-antagonist, was administered in this case as the only drug deemed likely to be contributing to delayed recovery was romifidine which had been administered for recovery. A study looking at atipamezole administration following detomidine sedation in horses was shown to shorten recovery from sedation without completely antagonising the effects, hence it was administered. Studies have looked at factors affecting recovery quality and found that shorter duration of anaesthesia, lower American Society of Anesthesiologists status, less invasive surgery, longer recovery times, lower body mass and younger age were all associated with better quality recovery. Other commonly encountered morbidities or causes of mortality in the recovery period include postanaesthetic myopathy, neuropathies, respiratory obstruction, pulmonary oedema and spinal cord malacia. Postanaesthetic myopathy was considered as a possibility in this case, however the horse was not distressed, did not exhibit signs of pain and the muscles were not palpably swollen, hard or hot, which are signs typically seen with postanaesthetic myopathy, therefore it was effectively ruled out as a possibility.

Sevoflurane was used despite not being licensed for use in equine patients due to the improved quality of recoveries seen with sevoflurane when compared with isoflurane. The use of sevoflurane for maintenance of anaesthesia is standard practice in the present institution.

In conclusion, this case highlights a cause of delayed recovery in a horse with pre-existing neurological disease. The fact that the horse stood as soon as the outside door was opened to allow visualisation of a horizon helps to rule out any other cause of delayed recovery. The worsening of the head tilt, immediate response to opening of the door and the fact that the horse actively pushed against any attempts at moving it into sternal recumbency, while appearing conscious, confirms exacerbation of vestibular disease and loss of orientation in relation to gravity as the likely cause of the horse’s failure to make any attempt to stand. In the future, allowing any horse with suspected underlying vestibular syndrome to visualise the horizon during recovery from anaesthesia if recovery is delayed, will be considered. This case highlights the importance of discussion and careful planning when dealing with a horse that has pre-existing neurological signs and requires general anaesthesia.

Learning points
► Recovery from anaesthesia can be affected by pre-existing neurological disease.
► Caution should be applied when anaesthetising a horse with pre-existing neurological disease and owners should be made aware of the potential for increased risk during the recovery period.
► Multidisciplinary approach to problem solving in difficult recoveries can be beneficial.
► General anaesthesia may lead to a transient deterioration in clinical signs associated with vestibular disease.

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