Hypocalcaemia in a six-month-old hand-reared female giraffe (Giraffa camelopardalis)

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SUMMARY

Radiological examination and surgical biopsy were required of a swelling in the cervical region in a healthy 200 kg, six-month-old hand-reared female giraffe (Giraffa camelopardalis). Induction was with intramuscular administration of medetomidine, butorphanol and ketamine and maintained with 1.5 per cent isoflurane-in-oxygen on a circle anaesthetic machine. Induction and maintenance were uneventful but recovery delayed and characterised by hindlimb weakness, opistotonus and torticollis of the head and neck. Atipamezole and naltrexone were administered to facilitate recovery but with minimal clinical improvement. Venous blood gas analysis indicated moderate metabolic acidosis, hypochloraemia, increased anion gap and marked hypocalcaemia (0.64 mmol/l). Intravenous administration of 60 ml calcium borogluconate resulted in a rapid improvement in muscle tone and the ability to stand. Hypocalcaemia was diagnosed in a juvenile giraffe after anaesthesia characterised by delayed recovery. Intravenous administration of calcium borogluconate resulted in rapid recovery of muscle strength and ambulance.

BACKGROUND

Hypocalcaemia has not been reported previously in anaesthetised giraffe. As it resulted in the inability of the giraffe to stand, it may have a profound effect on anaesthesia morbidity and mortality. In this instance, it was a clinically healthy animal apart from the hardened mass in the cervical region. Giraffe in an artificial environment such as nature parks or zoos may sometimes require anaesthesia for clinical procedures. Delayed recoveries in immobilised or anaesthetised wild animals are not unusual events and it is necessary that practitioners be made aware of such aetiology.

CASE PRESENTATION

A hand-reared giraffe calf was presented for anaesthesia that negated the use of more rapid-acting techniques using potent drugs such as etorphine or thiafentanyl. Rapid immobilisation is essential in free-ranging giraffe to prevent problems such as muscle exertion, capture myopathy or injury when when getting lost in the bush before they become recumbent (Aprea and others 2013). In the recumbent animal regurgitation and aspiration of rumen contents should be prevented by supporting the head above the level of the rumen. Tympany of the rumen may enhance regurgitation and limit ventilation. For recovery from immobilisation, a rapid regain of consciousness and muscle coordination is required to minimise risk of injury (Benbow and Lyon 2001, Kock and Burroughs 2012). Reversal with naltrexone or diprenorphine results in rapid recovery (Bush 2003, Citino and Bush 2007). The use of a ketamine and medetomidine combination for immobilisation was also reported (Bush and others 2001, Flach and others 2002).

Anaesthesia was required in a six-month-old hand-reared female giraffe (Giraffa camelopardalis) with an estimated bodyweight of 200 kg. This was to enable radiological examination of a suspected neck injury associated with a hardened mass (size 80×40×40 mm) over cervical vertebrae C3–C4. It was fasted from solid food and water from 15.00 the previous day but allowed to suckle the acidified milk replacer formula from a bottle as usual until 08.00 on the morning of the examination. Transport to the Veterinary Academic Hospital at Onderstepoort required approximately two hours travel time in a horse box.

On arrival, premedication was administered intramuscularly with the giraffe in the horse box with 2 ml medetomidine (Domitor 1 mg/ml, Zoetis Sandton) and 0.2 ml butophanol (Torbugesic 10 mg/ml, Fort Dodge Animal Health, Iowa). Fifteen minutes were allowed for drug onset that resulted in moderate signs of sedation and ataxia. The giraffe was led with a rope around its neck to a padded horse induction box. As jugular venous cannulation was unsuccessful in the horse box, induction was administered intramuscularly with 6 ml ketamine (Anaket-V 100 mg/ml, Bayer AH, Isando). Induction time was approximately 16 minutes until recumbent. The giraffe was manually stabilised during this period. For tracheal intub-ation a stomach tube was first placed in the trachea with the aid of a veterinary laryngoscope (blade 300 mm) and then the tracheal tube (25 mm cuffed silicon tube) rail-roaded over the stomach tube. Anaesthesia was initially maintained with 3 per cent isoflurane (Isofor, Safeline, Weltevreden Park) in oxygen that was reduced to 1.5 per cent after 15 minutes with a circle anaesthetic machine (Foal ventilator, JD Medical). Ventilation was assisted with the ventilator set at 15 breaths/minute. A jugular venous catheter (14 G Jelco) was placed and 1 l Lactated Ringers was administered during examination. Physiological parameters were monitored with a patient monitor (Advisor Vital Signs Monitor, Smiths Medical). Radiography was per-formed of the cervical vertebrae possibly involved in the injury and a biopsy taken from the mass. Median and range physiological parameters during anaesthesia were heart rate 40 (40, 40) bpm, mean arterial blood pressure 130 mmHg (135, 167), end-tidal carbon dioxide partial pressure 37 mmHg (35, 37), peripheral haemoglobin saturation 92 per cent (90, 96) and respiration rate 30 (15, 30) breaths/minute. Duration of anaesthesia was 70 minutes.

For recovery the ventilator and isoflurane were turned off, and the giraffe was allowed to breathe oxygen spontaneously from the breathing circuit for 10 minutes. Ten milligrams atipamezole (Antisedan 5 mg/ml, Zoetis, Sandton) was administered intramuscularly to reverse the medetomidine. Return of the palpebral reflex was observed and tracheal extubation was per-formed with the onset of high mandibular tone. The giraffe was manually restrained until strong purposeful movements to right itself occurred before it was allowed to attain sternal recumbency. However, it was unable to stand and it remained in sternal recumbency. Mild torticollis and 'star gazing' was observed with a tendency to fall backwards when it attempted to stand. Recovery was delayed and multiple unsuccessful attempts to stand were made. Hindlimb weakness was pronounced. With manual assistance to stand, only the front limbs supported its weight but the hindlimbs remained flexed and sus-pended in air with the rest of the body. A second dose of 20 mg atipamezole was administered intramuscularly but with no improvement. Fifty milligrams naltrexone (Trexonil 50 mg/ml, Wildlife Pharmaceuticals SA, Karino) was administered intra-muscularly. Only minimal improvement was observed after this. Rectal temperature was 39°C.

A venous blood gas sample was collected during recovery. The results were pH 7.196, pCO_2 55.6 mmHg, pO_2 36.6 mmHg, HCO_3^{-1} std 17.8 mmol/l, BE −7.2 mmol/l, Na^+ 133 mmol/l, K^+ 2.79 mmol/l,

Ca^{2+} 0.64 mmol/l and Cl^{-} 89 mmol/l. AnGap 25.9 mmol/l and haematocrit 32 per cent. A diagnosis of hypo-calcaemia was made and 60 ml calcium borogluconate (Lionel's Veterinary Supplies) was administered intravenous as a slow bolus over five minutes. General demeanour improved and swallowing reflexes appeared within five minutes after treatment, and the giraffe was able to stand within 10 minutes thereafter. An additional 40 ml calcium borogluconate was administered subcutaneously. From turning off the isoflurane to be able to stand, the duration of recovery was 170 minutes. A further 15 minutes were required before the giraffe was sufficiently stable to be loaded in the horse box and transported home. Full recovery of the giraffe was reported by the owner on the following day.

INVESTIGATIONS

Venous blood gas and electrolyte analysis.

DIFFERENTIAL DIAGNOSIS

Incomplete reversal of anaesthetics.

TREATMENT

- Atimapezole
- Naltrexone
- Calcium borogluconate.

OUTCOME AND FOLLOW-UP

Intravenous calcium borogluconate administration resulted in a dramatic recovery in the ability to stand, ambulate and to return home by horse box.

Full recovery of the giraffe was reported by the owner on the following day.

Histological examination of the biopsy material only revealed fibrous tissue.

DISCUSSION

Hand-rearing the giraffe calf facilitated handling and negated the use of a potent drug such as etorphine for immobilisation. First sedating it with medetomidine and butorphanol reduced induction time after intramuscular ketamine administration. The intravenous induction route would be preferred if a venous cannula could be placed in the conscious giraffe. Inhalation anaesthesia negated the use of intravenous drugs for maintenance of anaesthesia and the risk of prolonged recovery. Fasting was considered appropriate as no gas accumulation was observed in the rumen and the saliva that flowed from the mouth remained clear. Hand recovery was regarded as appropriate to limit possible injury as result of its long extremities that may increase risk of injury. Recovery was considered delayed as the return of muscle strength was not as expected. Reversal of the medetomidine was performed at the end of anaesthesia as medetomidine is a potent sedative and muscle relaxant, and residual effects from the drug will cotribute to a delay in recovery into the standing position. Failed attempts to stand resulted in the administration of naltrexone to counter any possible sedation from butorphanol. The clinical impression of the giraffe was that it

deteriorated after the first few attempts to stand and that was a cause of concern. The continued hindlimb muscle weakness coupled with torticollis and opistotonus of the neck (star gazing) prompted an electrolyte and blood gas analysis to search for possible metabolic causes of the delayed recovery. The venous blood gas values compared favourably with arterial blood gas values reported previously (Bush and others 2001, Flach and others 2002) in anaesthetised giraffe. However, abnormal electrolyte and acid–base values were observed: a severe hypocalcaemia, metabolic acidosis, hypochloraemia and an increased anion gap. Calcium borogluconate was immediately administered intravenously as hypocalcaemia can be associated with the muscle weakness (Brozos and others 2011). The recovery was spectacular as the giraffe was able to stand within 10 minutes after intravenous calcium administration. Muscle exertion during recovery may have resulted in a metabolic acidosis as result of lactic acid production. Hypocalcaemia as a complication of anaesthesia was not previously reported in game animals such as the giraffe or any other species. Milk was only withheld from 08.00 in the morning, and therefore unlikely to be the cause of the hypocalcaemia. Clinical observation of the giraffe in the horse box on arrival did not raise any suspicion of muscle weakness and the presence of hypocalcaemia during the preoperative period is considered unlikely. The nearly instantaneous response to the administration of calcium is a convincing indication that hypocalcaemia was involved in the muscle weakness associated with the inability of the giraffe to stand, and probably induced during the period of immobilisation. In retrospect a complete blood count and blood gas analysis should have been performed after induction and may have provided a better understanding if the hypocalcaemia was induced during induction. The initial intention with the anaesthesia was a radiological examination that will require a short anaesthetic period; however, it extended much longer when a surgical biopsy of the mass was obtained. The question that needs to be answered is whether the hypocalcaemia was an isolated incident possibly associated with the age of the animal or does it occur more often in giraffe only or including other ungulates, but was not noticed before?

Contributors GFS: primary anaesthetist, wrote the manuscript; RB: assist anaesthesia, review manuscript; JO: referral veterinarian, review manuscript.

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