Laura Rosewell RVN DipAVN (Small Animal) MBVNA

Laura began nursing in 2007, qualifying in 2010. Following two years as a surgical nurse at an orthopaedic referral hospital, Laura is now Clinical Nurse Manager at Moor Cottage Veterinary Hospital in Berkshire. She has recently completed her DipAVN and is currently studying for a BSc(Hons) degree at Harper Adams University.

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Nursing management of the aspiration pneumonia patient: A case study

Laura Rosewell RVN DipAVN(Small Animal) MBVNA
Moor Cottage Veterinary Hospital, London Road, Binfield, Berkshire, RG42 4AA

ABSTRACT: Patients with aspiration pneumonia often require intensive nursing care. These cases can be challenging to nurse but utilise the extensive knowledge of experienced veterinary nurses and allow us to demonstrate our skills. The requirement for frequent monitoring, oxygen therapy, physiotherapy, management of hydration and nutritional management, in addition to the provision of environmental enrichment and other day-to-day care, make these cases extremely rewarding. This case study focuses on the inpatient care given to a patient with myasthenia gravis, megoesophagus and aspiration pneumonia. The care requirements are discussed and the level of care received by the patient is evaluated.

History

Signalment and presentation

Buddy, a nine-year-old, neutered male West Highland White Terrier, weighing 9.5 kg was presented with shivering, regurgitation, coughing and inappetence. He had been diagnosed with megoesophagus four days earlier and was treated with intravenous fluid therapy (IVFT), ranitidine, sucralfate and supportive care for three days.

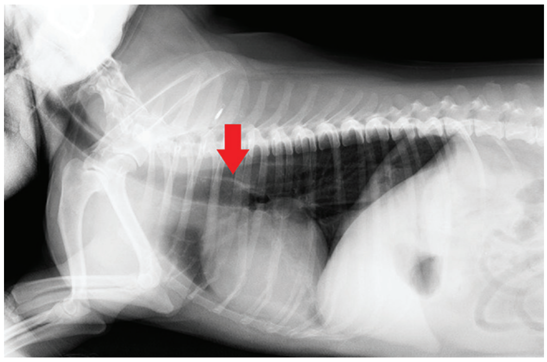

Figure 1. Lateral view. © Moor Cottage Veterinary Hospital 2014. All Rights Reserved

Patient assessment

On presentation, Buddy was dyspnoeic, with cyanotic mucous membranes (MMs), a respiratory rate (RR) of 88 breaths/minute, increased respiratory effort (RE) and a heart rate (HR) of 144 beats/minute. On chest auscultation, harsh crackling sounds were heard over his lung fields.

Buddy was placed in an oxygen cage before further assessment was carried out, to avoid stress exacerbating his dyspnoea. Once Buddy had settled, conscious thoracic radiographs were taken. These revealed consolidated lung fields and a definite megoesophagus (**Figures 1 and 2**).

Blood was obtained for biochemical and haematological analysis. This revealed hypokalaemia, monocytosis and neutrophilia with left shift, indicating infection.

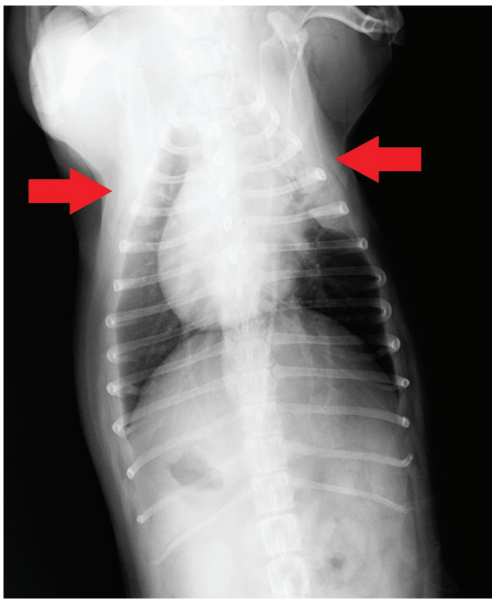

Figure 2. Dorso-ventral view. © Moor Cottage Veterinary Hospital 2014. All Rights Reserved

Because Buddy developed muscle weakness and exhibited reluctance to stand two days following admission, separated serum was sent to an external laboratory for acetylcholine receptor (AChR) antibody testing. This revealed that Buddy had developed myasthenia gravis (MG).

Myasthenia gravis

MG is a failure of the neuromuscular junction, resulting in skeletal-muscle weakness. The condition can be acquired or congenital, and commonly affects several terrier breeds (Garosi, 2013). A bimodal age distribution has been reported in dogs, with dogs of 2–3 and 9–10 years commonly affected (Taylor, 2005). Acquired MG is an immune-mediated disease caused by the production of acetylcholine receptor (AChR) antibodies, which bind to AChR sites and reduce the number of functional receptors (Platt & Shelton, 2010). This causes a reduction in neuromuscular transmission.

Acquired MG may be diagnosed by determining the presence of circulating AChR antibodies. It is treated by administration of anticholinesterase drugs and supportive care, and, occasionally, immunosuppressive drugs (Taylor, 2005).

Up to 90% of dogs with MG have concurrent megoesophagus, due to the large amount of skeletal muscle in their oesophagus (Taylor, 2005). With this condition, the weakened oesophageal muscles cause oesophageal dilatation, resulting in regurgitation (Willard, 2005).

Aspiration pneumonia (AP) commonly occurs due to inhalation of regurgitated stomach contents. The aspirated content reaches the alveoli, causing inflammation leading to impaired lung defence and allowing bacterial colonisation to occur. Associated clinical signs include coughing, dyspnoea, tachypnoea, cyanotic mucous membranes (MMs) and harsh lung sounds. AP is treated with intravenous antibiotics, oxygen supplementation, nebulisation and coupage (Waddell & King, 2010).

Treatment plan

The veterinary surgeon initiated the following treatment plan:

- dexamethasone (Dexadreson, MSD) – 1 ml subcutaneously (SC) as a one-off dose
- frusemide (Dimazon, MSD) – 0.8 ml intravenously (IV), (with a further 0.4 ml being given nine hours later following an episode of cyanosis)
- buprenorphine (Buprecare, Animalcare) – 0.4 ml IV, twice daily (BID)
- cefuroxime (Zinacef, Glaxosmithkline) – 3 ml IV, three times daily (TID)
- ranitidine (Zantac, Glaxosmithkline) – 1.3 ml IV, BID
- metronidazole (Metronidazole, Baxter) – 9 ml IV, BID
- sucralfate (Antepsin, Chugai) – 5 ml orally (PO), four times daily (QID)
- supportive care – including IVFT, oxygen supplementation, humidification BID and percussion QID
- pyridostigmine (Mestinon, Valeant) – 1/6th tablet PO, BID (initiated two days following admission)

Nursing care

Patient monitoring

Buddy was initially monitored hourly for changes in position, respiratory effort, respiratory rate and MM colour. Frequency of coughing and expulsion of secretions were also monitored hourly.

Hyperthermia was detected (39.8°C) four hours after admission, so the environment was cooled and temperature monitoring carried out at 2–3 hourly intervals until normothermia was achieved. Temperature and Heart Rate (HR) were then monitored BID.

Oxygen supplementation

Initial oxygen supplementation took place in a paediatric incubator. These allow high inspired oxygen concentrations (80–90%) to be reached (Aldridge & O'Dwyer, 2013), but are generally only suitable for small patients. Buddy was too large to move freely inside the incubator, and after one hour became stressed. He was therefore moved to a kennel and oxygen delivered via an improvised Crowe collar (**Figure 3**). Crowe collars consist of an Elizabethan collar partially covered with cling film. They provide an inspired oxygen concentration of around 60% and are well tolerated by most patients (Aldridge & O'Dwyer, 2013).

Disadvantages associated with Crowe collars include hyperthermia and drying of MMs. For these reasons temperature monitoring and lubrication of the eyes using sterile carbomer lubricating gel (Viscotears, Alcon) are recommended (Cave, 2006).

Buddy's temperature was monitored at least twice daily; however hyperthermia was detected four hours after admission, and hourly temperature checks might have detected this sooner. Eye lubrication was not performed, but would have been beneficial, as Buddy received oxygen therapy for 48 hours.

Figure 3. Crowe collar and humidification chamber. Oxygen enters the humidification chamber before entering the Crowe collar via a breathing circuit and endotracheal tube. © Laura Rosewell 2014. All Rights Reserved

Lee (2010) suggests blood-gas analysis should be utilised to assess adequacy of ventilation and the requirement for on-going oxygen therapy. This was not done, due to a lack of necessary equipment. Pulse oximetry would have provided an easy, non-invasive measurement of oxygenation.

Humidification

Long-term oxygen therapy can result in irritation and dessication of MMs, decreased muco-ciliary clearance and increased respiratory infection risk. It is therefore recommended that, if oxygen therapy lasts for longer than one hour, it should be humidified (Aldridge & O'Dwyer, 2013). This is achieved by passing the oxygen through a purpose-built chamber containing sterile water. In Buddy's case, oxygen was continuously humidified throughout treatment. The incubator's chamber was filled twice daily, and an improvised device was built into the Crowe collar and circuit (**Figure 3**).

Physiotherapy

Respiratory physiotherapy aids the expulsion of secretions and aspirated contents, and should be performed from the point of diagnosis in patients with AP. A combination of nebulisation, percussion and vibration is recommended. *Nebulisation* is the reduction of water to steam, and should be performed prior to percussion; this helps to break up secretions (Lee, 2010). During *vibration*, the physiotherapist places their hand over the chest wall and creates a fine shaking action on expiration. This helps to loosen secretions (Sharp, 2010). *Percussion*, or coupage, is the 'clapping' of the physiotherapist's hands against walls of the thorax. This has a similar effect to vibration and should be performed for around ten minutes, three to four times daily (Hotston Moore, 2006).

Percussion was performed at least four times a day depending on patient condition. Nebulisation was not performed, due to a lack of necessary equipment, but placing a bowl of boiling water in front of the kennel (out of reach of the patient) could have had a similar effect due to the steam released into the kennel. Vibration was not performed due to a lack of staff training in this area, but may also have been beneficial.

As Buddy's muscle weakness became more generalised, he became weak and unable to stand. He was maintained in sternal recumbency to aid his respiration. Supported standing and passive range of motion exercises were implemented QID, to prevent muscle atrophy and joint stiffness (Sharp, 2010). Once Buddy was able to walk, short lead exercise was introduced. This encourages deep breathing in addition to maintaining mobility (Lee, 2010).

Nutrition and hydration

Food and water were withheld for 24 hours, until Buddy's condition had stabilised. Small balls of Hills i/d and cooked chicken were then offered from a height, as feeding in this manner reduces aspiration risk (Lee, 2010). Buddy would not eat and a three-day period of inappetence followed.

Patients with a reduced food intake for longer than 48 hours are recognised as anorexic, and nutritional support is indicated (Yam & Cave, 2003). No force-feeding or syringe feeding was carried out in this case, as these carry a high risk of aspiration.

Placement of a percutaneous endoscopic gastrotomy (PEG) tube would have provided a convenient method of feeding that bypassed the oesophagus (Yam & Cave, 2003). However anaesthesia is required for placement and this would not have been suitable in this case.

Parenteral nutrition might have met some of the nutritional needs of the patient, however the complication rate is high and specialist equipment is needed (Watson & Chan, 2010).

Fortunately, Buddy began eating spontaneously four days after admission. Due to the risks associated with PEG tube placement, parenteral nutrition and syringe feeding, options for nutritional support were limited.

Hartmann's solution was administered intravenously at maintenance rate (2 ml/kg/hour) from the onset of treatment. Potassium chloride supplementation was initiated three days following admission, to avoid dehydration and fluid losses associated with pneumonia (Lee, 2010). Serum electrolyte levels were monitored daily until they returned to normal.

Water was introduced one day following admission, using a raised feeder to avoid aspiration (**Figure 4**). Buddy was closely monitored whilst eating and drinking, and, following this, maintained in an upright position for five minutes, to reduce the risk of aspiration (**Figure 5**).

Figure 4. Buddy was fed using a raised feeder for food and water to minimise the risk of aspiration pneumonia. © Laura Rosewell 2014. All Rights Reserved

Figure 5. Buddy was kept in an upright position for five minutes after eating or drinking to minimise the risk of aspiration pneumonia. © Laura Rosewell 2014. All Rights Reserved

Conclusion

This case highlighted the need for further training in the nursing care required for patients of this type. Physiotherapy; nebulisation and vibration were not carried out by any of the nurses due to a lack of necessary equipment and training in this area. Completion of a nursing-care plan would have helped standardise the care Buddy received.

Buddy was monitored regularly throughout hospitalisation. Monitoring arterial blood gases was not possible, but pulse oximetry might have been of use whilst oxygen was administered.

Further nutritional support could have benefitted this patient, but with anaesthetic risks associated with PEG tube placement and parenteral nutrition carrying a high complication rate, treatment options were limited.

After two weeks of intensive nursing, Buddy was discharged and his supportive care continued at home. He made a full recovery and his myasthenia gravis went into remission four months after being discharged.

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