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Toxaemia secondary to pyloric foreign body obstruction in two African lion (*Panthera leo*) cubs

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ABSTRACT

A case of toxaemia secondary to pyloric foreign body obstruction in two four-month-old African lion cubs were presented in this article. The lion cubs were presented to the school of veterinary medicine with a complaint of weight loss and stunted growth despite having a normal appetite and seizures. Definitive diagnosis was made based on gross pathology after attempting various symptomatic treatments. This article therefore is meant to discourage the use of blankets as bedding in holding enclosures for warmth and comfort post-weaning in captive lion cubs and indeed wild cats in general as they tend to eat bedding that has been soiled with food.

1. Introduction

Pyloric obstruction or gastric outlet obstruction (GOO) represents a clinical and pathophysiological consequence of any disease process which produces mechanical impediment to gastric emptying [1]. Intrinsic or extrinsic obstruction of the pyloric channel or duodenum is the usual pathophysiology of GOO and the mechanism of obstruction depends upon the underlying aetiology [1]. Several causes have been implicated in the occurrence of GOO, including idiopathic hypertrophic pyloric stenosis [2], foreign body [3], neoplasia [2], gastric polyps, gastric volvulus [2] among others. Depending on the primary cause of the obstruction, the site can undergo tissue damage resulting in perforation, endotoxemia, and hypovolemic shock [3]. The onset of symptoms varies depending upon the aetiology of the obstruction. The most common clinical features of GOO include nausea and vomit, epigastric pain, early satiety, abdominal distension and weight loss [1]. The main hazards of obstruction in any segment of the gastrointestinal tract are toxaemia and septicaemia owing to the migration of toxins and bacteria through ischaemic bowel wall or via a perforation [4]. Bacterial toxins can cause a variety of effects which include among others, seizures [5], haemolysis, and platelet depletion [6]. Pyloric foreign body obstruction has been documented in domestic Felidae and Canidae, but not in captive wild Felidae and Canidae hence necessitating this report.

2. Case report

The cubs were born in a captive lion pride of one adult male, two adult females from a private wildlife estate in Chisamba, Zambia. The two females had 5 cubs between them. Two cubs died and only 3 remained which were subsequently weaned at the age of 4 weeks and recruited into a training programme for lion safari walks. These cubs were fed daily on a cocktail of milk products (milk, cream, human infant replacement milk formula), multivitamin, cod liver oil, crushed tablets of calcium carbonate, egg york and gelatine. In addition, the cubs were fed with poultry meat starting at 100 g at 4 weeks and increased with progression of time. After 3 weeks into training, the two cubs were seen to have lost weight and their growth was not in tandem relative to the other cubs despite having a normal appetite. They were later observed to suffer epileptic seizures that were progressive over a month. The seizures were characterized by circling, urination, defecation, recumbency, tonic spasm of

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skeletal muscles, limb paddling (galloping) and a few seconds of unconsciousness at the terminal phase of the seizure. Following the seizures, the cubs would appear weak and disoriented for nearly an hour before returning to normal. These seizures would initially last for 2 min but progressed to 6 min over a few weeks. The frequency increased from once in 2 weeks–3 times in a week over a period of 8 weeks. During this period an infrequent nonbilious emesis was observed.

Initially the seizures were treated symptomatically with a bolus of 10 mg (1.0 mg/kg) diazepam *i.v.* to effect. Once the seizures subsided, a patent air way was maintained and homoeostasis was conserved with fluid therapy. The body temperature was retained by intermittently covering the cub with a dripping wet towel until its temperature subsided to a range of 38–39.5 °C. Cranial and spinal radiograph were done coupled with a basic neurologic examination which was performed to evaluate a deficit of neural functions that may explain structural damage to the brain and account for the seizures. However this did not yield substantial indication to include or rule out structural brain damage. A tentative diagnosis of idiopathic seizures was made. The cub was then treated twice daily with 5 mg phenobarbital, 75 mg alprazolam, 20 mg thiamine, and 25 mg medrol.

Despite this treatment for over a period of 2 weeks the seizures increased in intensity and frequency. In addition, the cubs had black stool (melena). A second tentative diagnosis was speculated as hepatic encephalopathy. A treatment regime of 12 mg lactulose, 15 mg rifaximin twice daily plus enema was employed. The cub died before abdominal radiographs were taken.

Figure 1. Piece of blanket which caused partial obstruction of the pylorus.

Figure 2. Stomach filled up with pieces of blanket causing impaction.

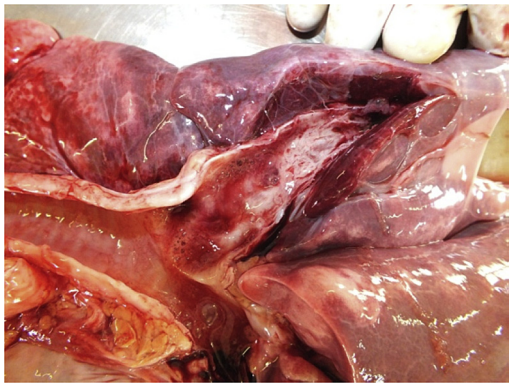

Figure 3. Congested lungs with froth in the bronchi.

Post-mortem findings revealed pyloric obstruction with a piece of a blanket (Figure 1). About three quarters of the stomach was filled up with these pieces of blackest (Figure 2). This blanket was consistent with the one used for bedding when the cubs were 4 weeks old. Pulmonary oedema, froth in the trachea and bronchi, with generalised congestion and echymotic haemorrhages of visceral organs (Figure 3). A definitive diagnosis of toxaemia secondary to pyloric foreign body obstruction was therefore made based on post-mortem examination.

3. Discussion

The cubs died from several complications. Continuous seizures coupled with toxins were causing pyrexia and fever secondary to endotoxaemia. The use of wet towels helped to prolong life of the cubs, but this could only be done at the clinic as it was not possible for the owner to continuously watch the episodes of seizure all the time. Blankets were soiled with food which gave them a scent and a good taste hence being eaten by the cubs which were still learning to eat on their own post-weaning.

Duodenal gastric impeded by foreign body (blanket) caused the cubs to lose weight because the food ingested was trapped in the stomach and thus prevented the maximum nutritional absorption and assimilation by the different segments of the gastrointestinal tract. This was also coupled with a syndrome of nonbilious vomiting. Thus gastric obstruction and emesis explained the loss of weight in cubs as compared to the cub that was not affected.

Gastric obstruction caused an exponential growth of microbials which include bacteria. These bacteria tended to produce toxins and cytotoxins that caused seizures [7,8]. The cranial and spinal radiographs ruled out structural damage to the central nervous system and thus the most probable cause of the seizures was endotoxins. This was also consistent with the gross findings at post-mortem which revealed congested lungs with echymotic haemorrhages due to alveolar capillary damage. The congestion and haemorrhages were also evident in all other visceral organs. It is, however, important to consider endotoxaemic shock as a differential diagnosis, owing to the similar cause.

In this particular case a portal systemic shunt was implicated and thus its treatment did not work. This emphasize that the management of such cases need to be thoroughly worked from blood work to diagnostic imaging. Abdominal radiographs should have been taken the first time when cubs came for treatment. This would have lead to an emergency surgery and saved the life of cubs. We therefore recommend that abdominal

scans and radiography should be taken in cases of seizures in lion as this might indicate obstruction of the gastrointestinal tract similar to this case. Furthermore, the authors recommend discouragement of the use of blankets as bedding in holding enclosures for warmth and comfort post-weaning in captive lion cubs and indeed wild cats in general as they tend to eat bedding that has been soiled with food.

Conflict of interest statement

We declare that we have no conflict of interest.

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