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Successful management of meloxicam induced abomasal ulcer type III in a jersey crossbred cow: Case report

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Abstract

A Jersey crossbred cow, aged four years, was brought in with a history of loss of appetite, dark tarry stool, and teeth grinding over the past three days. Prior to this, the cow had received meloxicam to treat fever. During the clinical assessment, the cow's conjunctival mucous membranes were observed to be pink and moist. Rectal examination revealed the presence of dark tarry faeces. Haematological analysis indicated an increased count of neutrophils. Occult blood was detected in the faecal sample. An ultrasound examination of the cow's lower abdomen showed a small amount of anechoic fluid. The diagnosis established was a type III abomasal ulcer caused by non-steroidal anti-inflammatory drug (NSAID) usage. The treatment plan involved administering normal saline, lactated Ringer's solution, pantoprazole, streptopenicillin and oral antacids over a span of three days. The cow's condition improved steadily, and it made a full recovery without any complications.

Keywords: Meloxicam, abomasal ulcer, melena, NSAID, pantoprazole

Introduction

Non-steroidal anti-inflammatory drugs have been valued as effective therapeutic agents offering pain relief, anti-inflammatory effects, and fever reduction. Nonetheless, their utilization has been linked to a notable occurrence of adverse effects, including gastrointestinal bleeding, ulcer formation, and the suppression of platelet aggregation. (Gomes *et al.*, 2008)^[6]. Because of their ability to reduce pain and reduce pyrexia, NSAIDs were frequently used in veterinary medicine. It irritates the mucosa, lowers prostaglandin synthesis and reduces gastric blood flow, all of which increase the risk of abomasal ulcers (Enberg *et al.*, 2006)^[5]. This case study outlines the successful treatment of an abomasal ulcer induced by meloxicam in a Jersey crossbred cow.

Case history and clinical examination

A Jersey crossbred cow, aged four years, was brought in with a history of loss of appetite, dark tarry stool, and bruxism over the past three days. The owner reported that the animal had been treated for pyrexia with meloxicam for a week. On examination, the cattle were dull with normal conjunctival mucous membranes. The animal showed signs of bruxism, slight abdominal discomfort and no ruminal motility. Rectal examination revealed dark tarry coloured faeces (Fig. 1).

Diagnosis

Haematology results showed neutrophilia (60%) and haemoglobin (7.3 g/dL), packed cell volume (25.41%) and red blood cells ($5.21 \times 10^6/\text{mm}^3$). Serum biochemistry revealed hypokalemia (2.78 mEq/L) and hyperglycemia (117 mg/dL). The fecal examination results showed double positive for the Occult blood test. During ultrasonographic examination, the ventral abdominal revealed anechoic fluid suggestive of mild peritonitis (Fig. 2). Based on the history, clinical examination, laboratory examination and ultrasonographic findings, this case was diagnosed as Type III abomasal ulcer induced by Meloxicam.

Therapeutic management

The animal was administered with Streptopenicillin 5 gm intramuscularly, Pantoprazole @ 1 mg/kg BW intravenously with Normal saline and Ringer lactate @ 10 ml/kg BW

intravenously, Tribivet 20 ml intramuscularly and antacid Blucil (Aluminum hydroxide, magnesium hydroxide and simethicone) 400 ml orally. This treatment protocol was continued for three days. Recovery was noticed from the third day of treatment with normal feeding habits and normal colour and consistency of dung (Fig. 3).

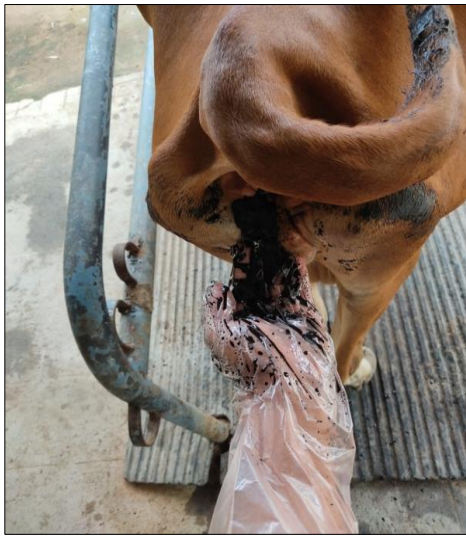


Fig 1: Dark, tarry colour faeces on per rectal examination



Fig 2: Anechoic fluid revealed peritonitis on ultrasonography



Fig 3: Normal colour and consistency of dung after treatment (recovered animal)

Discussion

Abomasal ulcers were a vital cause of improper digestion in ruminants. It mostly occurs in lactating dairy cows during the 1st six weeks of lactation and at various stages of growth like hand fed calves, veal calves, sucking beef calves and mature bulls (Radostits *et al.*, 2007) [10]. Hund and Wittek., 2017 [7]; Whitlock, 1980 [13] classified abomasal ulcer into four types namely Type 1 ulcers (non-perforating lesions), Type 2 (haemorrhagic ulcer with perforation), Type 3 (local peritonitis due to perforation), and Type 4 (generalized peritonitis induced by perforated ulcer). In dairy cows, ulcers were found equally distributed in both pyloric and corpus abomasi but bleeding ulcers were more common in corpus abomasa (Aukema *et al.*, 1974) [1].

Abomasal ulcers in adult cattle could be caused by ruminal acidosis (Braun *et al.*, 1991) [2], postpartum stress, copper deficiency, infections such as *Clostridium perfringens* Type A, *Helicobacter pylori* and *Candidatus helicobacter bovis* as well as trauma caused by abrasive agents like trichobezoars or sand and excessive use of anti-inflammatory drugs (Hund and Wittek, 2017) [7]. Additionally, abomasal hypersecretion of acid and pepsin due to stress-induced corticosteroid elevation was also associated with ulcer development (Braun *et al.*, 1991) [2].

Abomasal ulcers in cattle can lead to perforation of the abomasal wall, causing peritonitis (Tanwar *et al.*, 2009) [12]. Haemorrhagic abomasal ulcers result in tarry to black faeces (melena) and pale mucous membranes. Clinical signs include tachycardia, abdominal pain, tachypnea, disrupted rumen motility and reduced appetite (Breukink, 1976) [3]; Smith *et al.*, 1983 [11]). Elevated serum pepsinogen activity (>5.0 U/L) and increased serum gastrin activity were indicative of ulcers in abomasum (Ok *et al.* 2001[9]; Mesaric, 2005) [8]. Further, the Faecal Occult blood test assists in identifying the origin of gastrointestinal bleeding and indirectly detects the abomasal ulcer (Hund & Wittek, 2017) [7].

Treatment of abomasal ulcers in cattle could be symptomatic, medicinal or surgical reconstruction for perforated ulcers through laparotomy. Medications such as Ranitidine, Cimetidine (H₂ receptor antagonists) and Omeprazole (a potent proton pump inhibitor) were used to inhibit gastric acid secretion. Antacids like aluminium or magnesium hydroxide were drenched to protect mucosal integrity and reduce bleeding. In cases of anaemia, blood transfusion should be performed (Constable *et al.*, 2017) [4].

Conclusion

Prostaglandins protect the abomasum by increasing mucus production and bicarbonate secretion, decreasing gastric acid secretion and regulating mucosal blood flow. Meloxicam reduces abomasal mucosal integrity, resulting in Type III abomasal ulcers by inhibiting mucus and bicarbonate production through the suppression of Prostaglandin E synthesis via COX-1 inhibition. Thus, Pantoprazole is an effective medication in the treatment of acid related disorders.

Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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