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Therapeutic management of non-perforating abomasal ulcer in cattle

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ABSTRACT

This study focussed on the therapeutic management of non-perforating abomasal ulcers in cattle. A total of 442 cattle having gastrointestinal disorders were screened based on the presence of melena and positive faecal occult blood tests (FOBT). Among these cases, only 15 were identified as non-perforating abomasal ulcers and further confirmation was done by serum pepsinogen. Out of this, 12 cases were selected for the study and cattle were divided into two groups, Group II received Inj. Ranitidine @ 3 mg/kg b.wt intramuscular BID for 5 days and Group III cattle received Inj. Pantoprazole @ 1 mg/kg b.wt intravenous, SID for 5 days. The treatment evaluation by clinical improvement, haematology, serum biochemistry, serum electrolytes, rumen chloride, serum pepsinogen, serum NEFA and blood gas changes were performed on day 0 of pre-treatment and day 5 post-treatment in both groups. Pantoprazole @ 1 mg/kg b.wt SID IV for 5 days showed early recovery as compared to ranitidine against non-perforating abomasal ulcer in cattle.

Keywords: Abomasal ulcer, Cattle, FOBT, Melena, Pantoprazole, Ranitidine, Serum pepsinogen

Abomasal ulcer is one of the important gastrointestinal disorders in cattle which is commonly noticed in 1-6 weeks of lactation, hand-fed calves and mature bulls (Constable *et al*. 2017). The clinical signs of abomasal ulcers are non-specific and it becomes a great task to diagnose it (especially type 1 and 2 non-perforated ulcer), even by large animal practitioners (Braun *et al*. 2019). Early diagnosis of abomasal ulcer is important to determine the mode of treatment protocol, because untreated or delayed treatment of non-perforating ulcers could lead to perforation and endanger the life of the animal (Smith *et al*. 1986). The causes of abomasal ulcer are multifactorial which includes changes in feeding pattern, trauma, stress, concurrent disease, copper deficiency, drugs (steroid and NSAIDs) and certain microorganisms (Hund and Wittek 2017). Abomasal ulcer have a strong correlation with high concentrate feeding like providing starch rich diet and boiled rice, especially during abrupt changes in feeding from high roughage to high concentrate (Yasaswini *et al*. 2021). Type 1 abomasal ulcer is an important cause of indigestion in cattle which is usually not diagnosed until slaughter because of its non-specific signs (Braun *et al*. 1991). The clinical signs of type 2 ulcer were melena, pale mucous membrane, tachycardia and tachypnea (Braun *et al*. 2019). Non-perforating abomasal ulcer (type 1) can be diagnosed by faecal occult blood test (Braun *et al*. 2020), where type 2 ulcers are usually diagnosed based on melena, low haematocrit, hypoproteinemia and hypokalemia (Braun *et al*. 2019). Further, the estimation of serum pepsinogen is a simple test to diagnose sub-clinical abomasal ulcer (Mesaric 2005). Serum pepsinogen activities were higher in cow with abomasal ulcer (Hussain *et al.* 2023). The available therapeutic options for non-perforating abomasal ulcer (type 1 and type 2) in cattle are limited, especially with oral antacids. There are only a few reported cases of using parenteral antacids (H_2 blockers) for non-perforating abomasal ulcers in cattle (Tharwat and Ahmed 2012). But, ranitidine has transient effect on abomasal *p*H and multiple daily administrations are required. Proton pump inhibitors (PPIs) are considered to be an alternative to H_2 receptor antagonists and other anti-secretory drugs for treatment of ulcers which are caused by NSAIDs and other types of ulcers and esophagitis (Shin and Sachs 2008) in animals, but there are very limited study in cattle. Considering the aforementioned points, the present study aimed to evaluate the efficacy of ranitidine and pantoprazole against non-perforating abomasal ulcer in cattle.

MATERIALS AND METHODS

In the present study, 442 cattle with various gastrointestinal disorders were presented to Large Animal Medicine Unit, Veterinary Clinical Complex (VCC), Veterinary College and Research Institute, Orathanadu, Thanjavur from September 2022 to October 2023. Six

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apparently healthy cattle were randomly selected to obtain normal data for the parameters under study and considered as group I (Control). Among 442 cases, only 15 cattle were diagnosed with non-perforating abomasal ulcers based on positive FOBT and melena which was further confirmed by elevated serum pepsinogen level. Out of this, only 12 cases were selected for treatment trail and animals were equally distributed as 3 positive melena and 3 FOBT in each treatment groups. The treatment groups: Ranitidine (Group II) and Pantoprazole (Group III), consisted of six animals in each group. Group II received Inj. Ranitidine at the dosage of 3 mg/kg b.wt intramuscular, BID for 5 days. Group III cattle received Inj. Pantoprazole at the dosage of 1 mg/kg b.wt IV SID for 5 days. Treatment trail was conducted for a minimum period of 5 days and parameters were evaluated on the day of presentation (0 day) and 5th day post-treatment. To assess the treatment efficacy, the following parameters were studied, i.e. clinical improvement, haematology, serum biochemistry, electrolytes, serum pepsinogen and NEFA were performed on day 0 and day 5. Fluid therapy and vitamins B complex were given as supportive treatment for both the treatment groups.

Clinical evaluation: The following clinical parameters were recorded before and after treatment of both groups (Table 1): General activity: Dullness / active, Melena: Present / absent, Faecal Occult Blood Test: Positive / Negative, Appetite: Normal / Reduced / Improved, Bruxism: Present / Absent and Dung: Voided normally / reduced / not voided.

Faecal occult blood test (FOBT): Faecal samples were tested for faecal occult blood by commercially available HEMOSPOT® test cards (Crest Biosystems, Goa, India) based on the standard Guaiac method.

Rumen chloride estimation: Rumen fluid (5 mL) was collected by using 18-gauge, 1.5 inch needle inserted in left paralumbar fossa and the fluid was centrifuged at 3500 rpm for 10 min, and the sediment was discarded (Nordlund and Garrett 1994). The supernatant was used for estimation of rumen chloride by using All Care AC 9801 electrolyte analyzer with commercially available kits with standard operating procedure.

Laboratory analysis: About 10 mL blood was collected from jugular vein in EDTA, heparin and without anticoagulant coated vial for haematological studies, blood gas analysis and serum biochemical and electrolyte analysis. Blood gas analysis was done by CG4 cartridge in a handheld blood analyzer (i-STAT 1®). Serum biochemical parameters were performed by using selectra PRO XS® automated biochemical analyzer. Serum electrolytes were estimated by All Care AC 9801 electrolyte analyzer.

Serum pepsinogen: The samples were pooled and serum pepsinogen was estimated by ELISA method as per the manufacturer's instruction and by using Bovine Pepsinogen II (PGII) ELISA Kit (Shanghai Coon Koon Biotech Co., Ltd, China).

Serum NEFA: NEFA was analyzed using bovine NEFA ELISA Kit as per the manufacturer's protocol (Fine test®, Wuhan Fine Biotech Co, Ltd, China).

Ultrasonographic examination: All the selected animals were subjected to trans-abdominal ultrasound examination for the evaluation of abomasum according to Braun *et al.* (1997). Trans-abdominal ultrasound examination was performed in standing animals without any sedation. Colour Doppler ultrasound [My Lab one Vet (Esaote) with 2.5 to 5 MHz convex probe] was used for this study. Hair was clipped off using electronic hair clipper and Isopropyl alcohol (70%) and ultrasound gel was applied on the region before ultrasound examination.

Statistical analysis: One-way analysis of variance (ANOVA) and post-hoc tukey's test were used for multiple comparisons between the treatment (day 0 and day 5) and control groups of cattle. A value of $p<0.05$ was considered as significant in all statistical analysis.

Table 1. Clinical responses of non-perforating abomasal ulcer in cattle (before and after treatment)

Parameter	Group II Ranitidine (n=6)				Group III Pantoprazole (n=6)			
	Before (day 0)		After (day 5)		Before (day 0)		After (day 5)	
General activity	Dullness	6 (100.0%)	Dullness	1 (16.7%)	Dullness	6 (100.0%)	Dullness	1 (16.7%)
	Active		Active	5 (83.3%)	Active		Active	5 (83.3%)
Melena	Present	3 (50.0%)	Absent	2 (33.3%)	Present	3 (50.0%)	Absent	3 (50.0%)
			Present	1 (16.7%)				
FOBT	Positive	3 (50.0%)	Negative	2 (33.3%)	Positive	3 (50.0%)	Negative	3 (50.0%)
			Positive	1 (16.7%)				
Appetite	Normal	1 (16.7%)	Normal	1 (16.7%)	Normal	1 (16.7%)	Normal	-
	Reduced	5 (83.3%)	Reduced	-	Reduced	5 (83.3%)	Reduced	1 (16.7%)
			Improved	5 (83.3%)			Improved	5 (83.3%)
Bruxism	Present	3 (50.0%)	Present	1 (16.7%)	Present	4 (66.7%)	Present	1 (16.7%)
	Absent	3 (50.0%)	Absent	5 (83.3%)	Absent	2 (33.3%)	Absent	5 (83.3%)
Dung	Normal	2 (33.3%)	Normal	5 (83.3%)	Normal	1 (16.7%)	Normal	5 (83.3%)
	Reduced	3 (50.0%)	Reduced	1 (16.7%)	Reduced	4 (66.7%)	Reduced	1 (16.7%)
	Not voided	1 (16.7%)	Not voided	-	Not voided	1 (16.7%)	Not voided	-

FOBT, Faecal occult blood test.

Table 2. Serum electrolytes, rumen chloride, pepsinogen and NEFA of non-perforating abomasal ulcer in cattle (before and after treatment)

Parameter	Unit	Group I Control (n=6)	Group II Ranitidine (n=6)		Group III Pantoprazole (n=6)		p-value
			Before (day 0)	After (day 5)	Before (day 0)	After (day 5)	
Ca+	mg/dL	11.40±0.36^{c}	7.62±0.55^{a}	9.70±0.72^{bc}	8.33±0.23^{ab}	9.75±0.28^{bc}	0.000**
P+	mg/dL	4.78±0.18	4.823±0.79	4.02±0.3	4.06±0.11	4.27±0.5	0.568^{ns}
Mg+	mg/dL	2.89±0.13^{ab}	3.25±0.06^{b}	3.32±0.15^{b}	2.60±0.2^{a}	2.75±0.21^{ab}	0.012*
K+	mmol/L	4.69±0.14^{c}	3.57±0.09^{ab}	3.87±0.11^{b}	3.39±0.12^{a}	4.01±0.08^{b}	0.000**
Sodium	mmol/L	135.68±1.21	131.31±2.43	131.15±2.58	136.02±2.78	131.85±2.92	0.437^{ns}
Chloride	mmol/L	101.22±2.6^{ab}	91.67±1.59^{a}	98.95±2.23^{ab}	92.17±2.85^{ab}	101.67±2.12^{b}	0.008*
Rumen chloride	mmol/L	24.48±1.28^{a}	58.68±5.16^{b}	31.38±2.68^{a}	63.00±3.21^{b}	36.00±1.37^{a}	0.004*
Pepsinogen	U/L	2.08±0.44^{a}	8.45±0.99^{b}	3.03±0.45^{a}	9.33±1.95^{b}	2.45±0.13^{a}	0.00**
NEFA	mmol/L	0.35±0.04^{a}	0.67±0.04^{b}	0.58±0.03^{b}	0.63±0.05^{b}	0.57±0.04^{b}	0.00**

Means bearing same superscript do not differ significantly otherwise significant between groups; NS, Not significant (p≥0.05); *, Significant at 5% level (p≤0.05); **, Significant at 1% level (p≤0.01). Ca+, Calcium; P+, Phosphorous; K+, Potassium; Mg+, Magnesium; NEFA, Non esterified Fatty Acids.

RESULTS AND DISCUSSION

According to previous studies, the diagnosis of non-perforating abomasal ulcer was done mostly by post-mortem and at the time of slaughter. But in the present study, the presence of non-perforating abomasal ulcer (type 1 and 2 ulcer) was diagnosed based on positive faecal occult test and presence of melena, further which were confirmed by elevated serum pepsinogen levels. These findings were supported by Morgado *et al*. (2014) who reported that the estimation of serum pepsinogen was a valuable marker for detecting abomasal ulcers in cattle. Further, Hussain *et al.* (2023) also reported that increases in serum pesinogen and positive FOBT were considered as very good indicator for the diagnosis of abomasal ulcer in cattle and buffaloes.

In clinical response of pantoprazole treatment (group III), all the animals showed significant improvement in general activity, appetite, absence of bruxism and normal dung voiding as observed on day 5, wherein in ranitidine treatment (group II), improvement was noticed in 5 out of 6 animals with presence of melena (16.7%) and positive FOBT (16.7%) (Table 1) which clearly showed that the treatment with pantoprazole showed better efficacy than ranitidine against non-perforating abomasal ulcers in cattle (Supplementary Fig. 1 to 4). Sasikala *et al.* (2021) also reported that pantoprazole @ 1 mg/kg IV for 5 days in Jersey crossbred bull showed improvement on day 4 of post-treatment. The parenteral administration of antacids could reduce the acid production and promote ulcer healing that reduces the occurrence of abomasal bleeding (Ahmed *et al.* 2005, Morgado *et al.* 2014). This could be the reason for clinical improvements that were observed in the present study.

A decrease in haemoglobin, packed cell volume (PCV) and red blood cell (RBC) levels was observed in both groups with abomasal ulcers on day 0 (groups II and III). Hussain *et al*. (2017) recorded low haemoglobin in type 1 and type 2 abomasal ulcers. The presence of anemia in the present study could be due to damage and inflammation of the abomasum which leads to impaired vitamin B_{12} synthesis (Engelking 2015). Post-treatment improvement in the haematology was observed (Supplementary Table 1).

Tharwat and Ahmed (2012) reported that the occurrence of hypoproteinemia and hypoalbuminemia in abomasal ulcer could be due to poor nutrition and abomasal bleeding (Table 3). The observed elevation of total protein in ranitidine treatment could be attributed to the preventive action of H_2 blockers against histamine effects,

Table 3. Serum biochemical parameters of non-perforating abomasal ulcer in cattle (before and after treatment)

Parameter	Unit	Group I Control (n=6)	Group II Ranitidine (n=6)		Group III Pantoprazole (n=6)		p-value
			Before (day 0)	After (day 5)	Before (day 0)	After (day 5)	
BUN	mg/dL	21.58±1.03	36.67±6.94	28.00±2.96	35.00±6.12	29.00±2.63	0.165^{ns}
Creatinine	mg/dL	1.16±0.13	1.31±0.15	1.32±0.27	1.12±0.08	1.06±0.09	0.703^{ns}
Glucose	mg/dL	52.00±6.63	65.17±1.85	67.17±2.89	61.17±5.67	64.17±5.13	0.217^{ns}
Total protein	g/dL	6.39±0.18^{b}	5.43±0.31^{a}	6.15±0.17^{ab}	5.61±0.15^{ab}	6.23±0.11^{ab}	0.007*
Albumin	g/dL	3.00±0.12	2.78±.0.12	3.13±0.1	2.63±0.17	2.80±0.17	0.118^{ns}
Globulin	g/dL	3.39±0.11^{b}	2.65±0.25^{a}	3.02±0.14^{ab}	2.98±0.16^{ab}	3.42±0.15^{b}	0.019*
A:G ratio	-	0.89±0.04	1.09±0.09	1.05±0.06	0.91±0.1	0.84±0.08	0.139^{ns}
AST	U/L	87.98±5.08	117.33±8.25	100.92±13.2	96.18±20.68	95.72±18.31	0.680^{ns}

Means bearing same superscript do not differ significantly otherwise significant between groups. ^{NS}, Not significant (p≥0.05); *, Significant at 5% level (p≤0.05); **, Significant at 1% level (p≤0.01); BUN, Blood Urea Nitrogen; AST, Aspartate aminotransferase.

Table 4. Overall findings of abomasal ulcer treatment in cattle

Parameter	Group II Ranitidine		Group III Pantoprazole	
	Before (n=6)	After (n=6)	Before (n=6)	After (n=6)
Recovery		Recovered 4/6		Recovered 6/6
Melena	3/3	Recovered 2/3 4/6	3/3	Recovered 3/3 6/6
FOBT	3/3	Recovered 2/3	3/3	Recovered 3/3
Haematology				
Hb	Low	Increased (+)	Low	Increased (++)
PCV	Low	Increased (++)	Low	Increased (++)
Serum biochemistry				
Protein	Hypoproteinemia	Significantly increased (+)	Hypoproteinemia	Significantly increased (++)
Globulin	Hypoglobulinemia	Significantly increased (+)	Hypoglobulinemia	Significantly increased (++)
Electrolytes				
Calcium	Hypocalcemia	Highly significant increase (+)	Hypocalcemia	Highly significant increase (+)
Potassium	Hypokalemia	Highly significant increase (+)	Hypokalemia	Highly significant increase (++)
Serum Cl^-	Significantly increased	Significant decreased (++)	Significantly increased	Significantly decreased (+)
Rumen Cl^-	Significantly increased	Highly significant decrease (+)	Significantly increased	Highly significant decrease (++)
Serum Pepsinogen	Highly significant increase	Highly significant decrease (+)	Significantly increased	Highly significant decrease (++)
Serum NEFA		No change		No change
Blood gas analysis		No change		No change

such as suppressing the immune response to antigens, T cell cytotoxic activity, immunoglobulin synthesis, and lymphokine production (Hosseinifard *et al.* 2013).

Low serum calcium and potassium were recorded in all the abomasal ulcer animals (Table 2). Hypokalemia was seen due to shift of potassium from extracellular space to intracellular space as result of metabolic alkalosis due to continuous HCl and potassium secretion into the abomasum (Yasaswini *et al*. 2021). Hypocalcemia might be due to inappetence and reduced absorption from the gut and puerperal hypocalcemia (Hussain *et al*. 2016). Increase in rumen chloride (Table 2) was due to ulcers in the pyloric region that causes pyloric spasms and resulting abomasal reflux which increases chloride levels in the rumen (Braun *et al*. 1991). Post-treatment decrease in rumen chloride might be due to reduction in pyloric sphincter constriction that prevents the back flow of abomasal content into the rumen (Tharwat and Ahmed 2012).

Elevated serum pepsinogen level was due to leakage of pepsinogen from the damaged abomasal mucosa (Hajimohammadi *et al.* 2017). So, serum pepsinogen had been identified as a valuable marker for detecting abomasal ulcers (Morgado *et al*. 2014, Hussain *et al.* 2023). Reduction in serum pepsinogen level was noticed in both treatment groups. But pantoprazole treatment showed better effect than ranitidine treatment against the non-perforating abomasal ulcers in cattle (Table 1). Braun *et al*. (1997), Braun (2019) and Saravanan *et al.* (2019) stated that in type 1 and type 2 ulcers, abomasum could not be imaged through ultrasound. Further, it is opined that ultrasonography is not a valuable tool in diagnosis of abomasal ulcer, especially type 1 and 2. Similarly, in this study also, there were no ulceration or erosion which could be detected during ultrasound examination. Further, abomasum was located in its normal position (Right ventral midline), homogenous echogenic content with abomasums of both the treatment groups.

Increased NEFA was considered to be an important profile to assess negative energy balance and for predicting risk factor in the occurrence of abomasal displacement in cattle (Constable *et al*. 2017). In the present study, there was no significant difference in the non-perforating abomasal ulcer groups of NEFA values. It could be due to the very mild inflammatory changes in the abomasum and it requires further study to reason out the pathophysiology. Similarly, there was no significant difference in the Blood gas levels between the groups (Supplementary Table 2), which shows no systemic involvement in non-perforating abomasal ulcer in cattle.

Based on the observation and parameters in the present study, pantoprazole group (III) showed better improvement than ranitidine treatment (II) against the non-perforating abomasal ulcers in cattle (Table 4). Clark *et al.* (2009) and Himaja *et al.* (2022) stated that pantoprazole directly blocks the action of H^+/K^+ATPase pump and it blocks the HCl production, the stimulatory actions of histamine, gastrin and acetylcholine were inhibited. Where ranitidine inhibits the stimulatory effects of histamine on gastric acid secretion, but HCl secretion through the acetylcholine and gastrin would be present. Shin and Sachs (2008) stated that the H^+/K^+ATPase was the last stage of the gastric acid secretion and proton pump inhibitors (PPIs) were widely accepted as more effective than H_2 receptor antagonists in suppressing gastric acid secretion (Malfara *et al.* 2005).

This proves that PPIs are evaluated as superior alternative to H_2 receptor antagonists against non-perforating abomasal ulcer in cattle.

The present study has documented that Inj. Pantoprazole @ 1 mg/kg b.wt SID IV 5 days showed better therapeutic efficacy than ranitidine treatment against non-perforating abomasal ulcer in cattle.

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