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Case report

SECONDARY TYPE 1 ABOMASAL ULCER IN CATTLE AND BUFFALO: A NECROPSY BASED CASE CONTROL STUDY

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Summary

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This prospective study describes the occurrence, morphology and localisation of type 1 abomasal ulcers (AU1) in various diseases of buffaloes and cattle. The carcasses were examined to confirm the cause of death. The abomasa were examined for AU1 and their characteristics. The AU1 were categorised into four subtypes, 1a, 1b, 1c and 1d, as per standard procedure. Traumatic reticuloperitonitis/pericarditis, reticular diaphragmatic hernia, intestinal obstruction, peritonitis, bronchopneumonia and theileriosis were the common causes for AU1. The overall prevalence of AU1 was 62.9%, which did not differ significantly with species and age. The prevalence of acute ulcers (1a and 1b) was significantly higher than that of chronic ulcers (1c and 1d). Most AU1 were located in the caudal third of abomasal body on parietal surface along the greater curvature. Most of the 1a ulcers were located in the pylorus, while 1b, 1c and 1d were located in the abomasal body. The overall prevalence of AU1 was lower ($P<0.05$) in the fundus than in other anatomical regions of the abomasum. Type 1b ulcers were more numerous than other subtypes. It was concluded that AU1 may be an important cause of slow recovery/poor prognosis under clinical situations and hence, the therapy protocol for such cases should include treatment for probable gastrointestinal bleeding.

Key words: buffalo, cattle, necropsy, prevalence, type 1 abomasal ulcer

The abomasal ulcers develop due to destructive action of gastric juice on abomasal mucosa, as a result of altered balance between protective and destructive processes. The abomasal ulcers in cattle and buffaloes may occur as mildly or heavily bleeding ulcers or perforating ulcers resulting into anaemia, peritonitis and death (Smith *et al*., 1986). Depending upon the degree of penetration into the abomasal wall, the abomasal ulcers are divided into four types (Smith *et al.,* 1983). Type 1 ulcers are shallow defects which reach deeper than the lamina muscularis of the

mucosa, type 2 ulcer is a deeper defect of the wall of the abomasum with bleeding from an artery, type 3 are perforating ulcers with localised peritonitis, and type 4 are perforating ulcers with ongoing acute diffuse peritonitis (Constable *et al.,* 2017). The type 1 ulcers (AU1) can be both acute and chronic lesions and are classified into four sub-types (Braun *et al.,* 1991). Subtype 1a are erosions with minor defects of the mucosa, sub-type 1b are sharply defined ulcers with punched-out appearance and local mucosal bleeding, subtype 1c are crater-like ulcers with superficial coating of fibrin or inflammatory products, and subtype 1d: ulcers whose crests converge towards the centre of the ulceration or holes through the spiral folds of abomasal mucosa (Braun *et al.,* 1991; Hussain *et al.,* 2019).

In recent studies, the prevalence of AU1 at the time of slaughter has been reported to be 49–84% in cattle (Hund *et al.,* 2016; Munch *et al.,* 2019) and 56–66% in buffaloes (Tajik *et al.,* 2013; Hussain *et al.,* 2019). Munch *et al.* (2019) examined the abomasa of 1,629 Danish Holstein cows and found an AU1 prevalence of 84% with subtype 1a and 1c mainly observed in the pyloric area. In a prevalence study of AU1 in apparently healthy buffaloes, Hussain *et al.* (2019) examined (both macroscopically and histopathologically) 134 randomly collected abomasa and reported an overall AU1 prevalence of 66.4%. The prevalence of different subtype of AU1 was 13.43%, 26.12% 21.64% and 5.22% for subtype 1a, 1b, 1c and 1d, respectively. The prevalence of AU1 is reported to be similar for various age groups of buffaloes (Tajik *et al.,* 2013; Hussain *et al.,* 2019) but in cattle, the prevalence of subtype 1d seems to increase with increase in parity and that of subtype 1a seems to decrease with increase in parity (Munch *et al.,* 2019).

Although the exact etiology of primary abomasal ulcers is unclear, stress, abomasal hyperacidity, mechanical abrasions are considered as possible causes (Constable *et al.,* 2017). In addition to a primary disease, abomasal ulceration can also occur secondary to other diseases like abomasal lymphoma and bronchopneumonia (Kureljusic *et al.,* 2013; Constable *et al.,* 2017). The perforating ulcers have been linked to abomasal displacement in cattle (Constable *et al.,* 2017) but the relationship of AU1 with other non-infectious diseases has not been established, particularly for buffaloes. However, it is recommended that ulcers of the abomasum should be distinguished from secondary ulcers which accompany rinderpest, malignant catarrhal fever, mucosal disease, actinomycosis and tuberculosis (Brown *et al.,* 2007). The reflux of bile acids into the abomasum in abomasal displacement is strongly associated with AU1 (Constable *et al.,* 2017). The main cause of bile acid reflux is a phenomenon called abomasal reflux. In bovines, abomasal reflux mainly occurs in abomasal and intestinal diseases (Constable *et al.,* 2017). In addition, abomasal reflux has also been reported in other gastrointestinal diseases like traumatic pericarditis, reticular diaphragmatic hernia (RDH), peritonitis, late pregnancy indigestion (Hussain *et al.,* 2014; 2018; 2021; 2022b). So there are chances that AU1 can occur secondary to other diseases. Further, there is evidence that melena is almost a pathognomonic sign of abomasal bleeding and melena has been reported in many cases of RDH, intestinal obstruction, peritonitis. Also, AU1 are described as the most common changes in the abomasum of slaughtered cattle (Tehrani *et al.,* 2012; Munch *et al.,* 2019) and

buffaloes (Tajik *et al.,* 2013; Hussain *et al.,* 2019). So, there is need is confirm the occurrence of AU1 in various diseases of cattle and buffaloes. It is also important to find more details about the number, morphology and location of AU1 in buffaloes and cattle. Therefore, the aim of this study was to describe the occurrence, morphology and localisation of AU1 in various diseases of buffaloes and cattle.

This was a prospective case control study on 140 bovines (76 buffaloes and 64 cattle) dead due to various causes and with a confirmed diagnosis based on necropsy findings. The cattle and buffaloes were all greater than 1 year of age and had been admitted to the post-mortem Examination Unit, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana from February 2013 to July 2014. The cases with primary abomasal ulcers and those with bleeding or perforated ulcers were excluded. There were 132 female and 8 male carcasses. The carcasses were examined to identify or confirm the cause of the death. On the basis of age the animals were divided in to three group viz. group 1 (up to 3 years), group 2 (>3–6 years) and group 3 (>6 years).

The abomasa were separated from surrounding viscera and severed from the rest of the gastrointestinal tract, incised along the lesser curvature. The abomasal contents were removed and rinsed away with water. The abomasum was thoroughly examined for the presence of AU1, if any. The AU1 were registered according to anatomical site on a sketch diagram representing all the anatomical parts, and both the parietal and visceral surfaces of the abomasum. The AU1 were also recorded photographically. The number, size and shape of all AU1 were recorded. Pathological classification of AU1 into four subtypes, 1a, 1, 1c and 1d, was performed using their gross characteristics according to Braun *et al.* (1991) with some modifications (Hussain *et al.,* 2019). The macroscopic findings were confirmed by histopathological examination.

The representative samples from all the gross lesions were collected for histopathological examination. These collected tissue samples were stored in 10% neutral buffered formalin and then underwent routine processing and wax embedding. Sections (4–5 μm thick) were stained with haematoxylin and eosin for microscopic examination.

The categorical data were presented as frequency and/or percentage. The χ^2 square test was used for comparisons between binary outcome and binary explanatory variable. The prevalence was compared between cattle and buffaloes, three age groups and between the anatomical parts of the abomasum.

The 140 cases of cattle and buffaloes were divided into 20 disease conditions on the basis of necropsy findings (Table 1). The animals categorised as fracture had fracture of various limb bones and have been euthanised due to poor prognosis. The animals with dystocia had been either euthanised or had died during the correction of dystocia. The overall prevalence of AU1 was 62.85% (88/140) (Table 1). AU1 were observed in 74.6% (50/67) cases of gastrointestinal disorders, 66.7% (6/9) cases of pulmonary disorders, 54.2% (13/24) cases of dystocia, 32% (8/25) cases of fracture, and all cases of theileriosis.

Among various AU1, the prevalence of type 1a (32.14%) and type 1b (25%) was significantly ($P<0.01$) higher than that of type 1c (3.57%) and 1d (2.14%) ulcers (Table 2). The prevalence of type 1a and 1b; and 1c and 1d did not differ significantly. The overall prevalence of AU1 did

Table 1. Prevalence of type 1 abomasal ulcers in various diseases of buffaloes and cattle

Disease condition (n)	Subtype of AU1				Number of abomasa with AU1	Percentage of abomasa with AU1
	1a	1b	1c	1d		
Gastrointestinal diseases						
Rumen impaction (3)	2	0	0	0	2	66.7
Omasal impaction (3)	1	1	0	0	2	66.7
TRP (33)	12	10	2	0	24	72.7
RDH (8)	0	4	0	1	5	62.5
Reticular abscess (1)	0	0	0	0	0	0
Peritonitis (6)	3	2	0	0	5	83.3
Intestinal obstruction (8)	3	3	1	0	7	87.5
Caecal dilatation (2)	1	0	1	0	2	100
Abomasal impaction(1)	1	0	0	0	1	100
Lactic acidosis (2)	2	0	0	0	2	100
Pulmonary diseases						
Bronchopneumonia (4)	3	1	0	0	4	100
Pleurisy (3)	1	0	0	0	1	33.3
Hydatosis (2)	1	0	0	0	1	50
Dystocia (24)	9	2	1	1	13	54.2
Fracture (25)	4	3	0	1	8	32.0
Theileriosis (8)	0	8	0	0	8	100
Meningitis (4)	1	1	0	0	2	50.0
Nasal tumour (1)	1	0	0	0	1	100
Haemoglobinuria (1)	0	0	0	0	0	0
Obstructive urolithiasis (1)	0	0	0	0	0	0
Total (140)	45	35	5	3	88	62.9

not differ significantly between cattle (62.5%, 40/64) and buffaloes (63.2%, 48/76) as did the odds of AU1 between cattle and buffaloes. Among 88 ulcer cases, three were male animals and the others female. This was similar to general population of cattle and buffaloes presented for necropsy. Majority of ulcer affected cases were in the age group 2 (n=44) and group 3 (n=28). The prevalence and odds of AU1 did not differ significantly between the three age groups.

Table 3 depicts the distribution according to subtype and anatomic location of AU1 in the abomasa. The prevalence of type 1a ulcers was significantly ($P<0.05$) higher in the pyloric region (31.1%) than in the fundus and body regions. Seventy four percent of type 1b ulcers were observed in the body region of the abomasum or throughout the abomasal mucosa (Table 3). The prevalence of type 1b ulcers was significantly ($P<0.05$) higher in the body region (37.1%) compared to fundus and pyloric regions. The overall prevalence of AU1 was significantly ($P<0.05$) lower in fundus than other anatomical regions of the abomasum. All theileriosis associated ulcers were grouped under type 1b and were typically observed throughout the abomasal mucosa.

The odds of AU1 for the anatomical parts of abomasum are depicted in Table 4. Considering the abomasal body as standard, the odds ratios for abomasal body with respect to pylorus and fundus were 1.39 and 11.93 i.e. the body region had 1.39 and 11.93 times higher odds of having AU1 than pylorus and fundus, respectively. The pylorus had 8.57 times greater odds of having AU1 than fundus.

On over all basis, the ulcers were observed over both the parietal and visceral surfaces of the abomasum, they were more commonly observed in the caudal third of the parietal surface of the abomasal body along the greater curvature (Fig. 1 and 2). Among the 35 abomasa with type 1b ulcers, additionally five abomasa had healed type 1b ulcers, two had type 1a ulcers and one had type 1c ulcers. Additionally, type 1a ulcers were observed in two abomasa with type 1c and type 1d ulcers.

The number of ulcers per abomasum is presented in Table 5. Among 88 abomasa, majority (42%) had 1 to 5 ulcers and 37.5% abomasa had more than 20 ulcers. Type 1b ulcers were observed in large numbers than other subtype of ulcers. Majority of the type 1a ulcers were round and <0.5 cm in diameter. Type 1b ulcers were typically round (0.4–4 cm in diameter) except one elongated ulcer of 10 cm. The size of type 1c and 1d ulcers was less than 2 cm. The statistically significant differences of frequency of various AU1 subtypes are presented in Table 5.

To our best knowledge this is the first study to document the prevalence of secondary AU1 in cattle and buffaloes. The study recorded a high prevalence of sec-

Table 2. Distribution of AU1 subtypes in 140 buffaloes and cattle

Ulcer type	Number	Percent of examined abomasa (n=140)	Percent of abomasa with AU1(n=88)
1a	45	32.14[a]	51.14
1b	35	25.0[a]	39.77
1c	5	3.57[b]	5.68
1d	3	2.14[b]	3.41
Total	88	66.85	100.00

The values with different superscripts differ significantly (P<0.01).

Table 3. Topographic distribution of type 1 abomasal ulcers in 88 abomasa

Ulcer type	Number	Fundus	Body	Pylorus	Body and pylorus	Body and fundus	Throughout abomasum
1a	45 (100)	1(2.2)[b]	2(4.4)[b]	14(31.1)[a]	12 (26.7)[a]	4 (8.8)[b]	12 (26.7)[a]
1b	35 (100)	1 (2.8)[b]	13 (37.1)[a]	1 (2.8)[b]	6 (17.1)[a]	1 (2.8)[b]	13 (37.1)[a]
1c	5 (100)	0 (0.0)	3 (60)	0 (0.0)	1 (20)	1 (20)	0 (0.0)
1d	3 (100)	0 (0.0)	2 (66.6)	0 (0.0)	1 (33.3)	0 (0.0)	0 (0.0)
Total	88 (100)	2 (2.3)[b]	20 (22.7)[a]	15 (17.1)[a]	20 (22.7)[a]	6 (6.8)[b]	25 (28.4)[a]

Numbers in parenthesis indicate row percentages. Within a row the values with different superscripts differ significantly (P<0.05).

Table 4. Odds of AU1 for different anatomical parts of abomasum

Location of ulcer	AU1 positive	AU1 negative	Total	Odds
Body	20 (47.01)[a]	120 (52.98)	140 (100)	20/120=0.167
Pylorus	15 (6.7)[a]	125 (93.3)	140 (100)	15/125=0.12
Fundus	2 (12.69)[b]	138 (87.31)	140 (100)	2/138=0.014

The numbers in parenthesis represent row percentages. The values with different superscripts differ significantly (P<0.05).

ondary AU1 in various bovine diseases. We believe that AU1 may be a frequently occurring event in clinical cases of buffaloes and cattle. The AU1 were observed in some unexpected disease conditions like

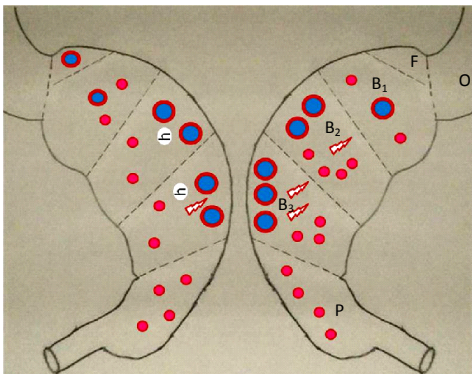

Fig. 1. Common location of subtype 1a, 1c and 1d ulcers plotted on a sketch diagram. Red circles represent subtype 1a ulcers, circles with blue filling – subtype 1c ulcers and the irregular shapes with white filling – subtype 1d ulcer. Circles with letter "h" represent healed ulcers. Left side of diagram represents interior view of the visceral surface of the abomasum and the right side is interior view of the abomasal parietal surface. O: omasum; F: fundus of the abomasum; B1: one- third of the body of the abomasum towards the fundus; B2: middle third of the abomasal body; B3: one-third of the body of the abomasum towards the pylorus; P: pylorus of the abomasum.

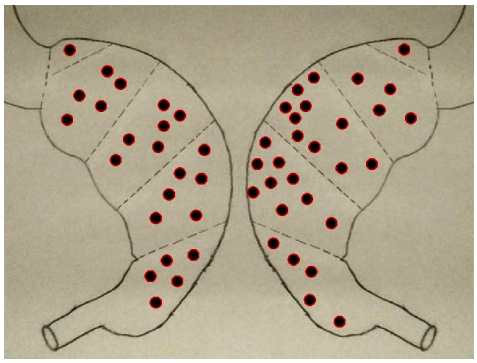

Fig. 2. Common location of subtype 1b ulcers plotted on a sketch diagram (circles with black filling and red outline).

fracture and dystocia. Due to higher prevalence, it is affirmed that AU1 may be an important cause of slow recovery or poor prognosis of these disorders under clinical situations. Although AU1 are clinically difficult to diagnose, they can be important cause of low production. The clinical signs of AU1 are non-specific and faecal occult blood test (FOBT) seems to be the only reliable method for the diagnosis of such ulcers (Munch *et al.,* 2020). In a study on bovines, the FOBT was positive in 0.83% of the healthy cattle and buffaloes (Hussain *et al.,* 2015a). However, under clinical situation the FOBT may be positive in a higher percentage of cases due to greater and varied types of stresses in disease conditions. So, we sug-

Table 5. Frequency of various type 1 abomasal ulcers per abomasum

Ulcer type	N	Number of ulcers per abomasum			
		1–5 ulcers	6–10 ulcers	11–20 ulcers	>20 ulcers
1a	45 (100)	16 (35.6)	8 (17.7)	7 (15.5)	14 (31.1)
1b	35 (100)	14 (40)[a]	2 (5.7)[b]	1 (2.8)[b]	18 (51.4)[a]
1c	5 (100)	4 (80)	0 (0.0)	0(0.0)	1 (20)
1d	3 (100)	3 (100)	0 (0.0)	0 (0.0)	0 (0.0)
Total	88 (100)	37 (42.0)[a]	10 (11.4)[b]	8 (9.1)[b]	33(37.5)[a]

Numbers in parenthesis indicate row percentages. Within a row the values with different superscripts differ significantly ($P<0.05$).

gest that FOBT should be routinely employed in clinical practice to diagnose secondary AU1, and the treatment protocol for such cases should also include treatment for the probable gastrointestinal hemorrhage. Further, the use of drugs to reduce gastric acid production and secretion would be desirable.

Currently we have no evidence to assert when the ulcers could have developed and there could be different mechanisms for different disease conditions. The human gastric ulcer syndrome resembles closely abomasal ulcer disease so a similar etiology and pathogenesis is suspected (Smith *et al.,* 1983). In human medicine, the increased gastrin concentration is believed to be an important factor for the increased gastric acidity leading to ulcers in the stomach and duodenum and even refluxes esophagitis (Roy *et al.,* 2001, Orlando *et al.,* 2007). Similar to gastric ulcers in humans, increased gastrin concentration has been documented in AU1 in buffaloes (Hussain *et al.,* 2022a). In present study, disturbances in the food intake could have resulted in increase in gastric acidity and then AU1. Gastric ulcers are commonly associated with stress and metabolic disturbances. The stress in animals may be related to the environment, nutrition, hyperacidity, lactic acidosis, and coarse ration (Cebra *et al.,* 2003, Mostaghni *et al.,* 2008). We suggest that the metabolic disturbances and hyperacidity due to anorexia could have predisposed the animals to AU1 in this study. However there could be other specific causes related to each disease conditions due different pathogenesis of different diseases.

One of the theories for ulcer formation is the 'abrasion theory', which claims that abrasive agents like course feedstuffs and abomasal trichobezoars could physically abrade the abomasal mucosa, initiating an ulcerogenic process (Bus *et al.,* 2019). In the area of study, the feeding of coarse feed (wheat straw) is a common practice and such practice could be a probable cause of AU1. However, in one study, young 5-day-old calves have died of perforating ulcers (Jelinski *et al.,* 1996). This early development of ulcers undermines the significance of course feedstuffs as necessary factors in the pathogenesis of abomasal ulcer, as it is unlikely that such young calves would be exposed to abrasive agents.

In gastrointestinal disorders, abomasal reflux could be the cause for bile acid reflux and then AU1. The abomasal reflux has been established in many gastrointestinal disorders like abomasal diseases, intestinal obstruction, caecal dilatation, peritonitis, traumatic pericarditis, RDH (Hussain *et al.,* 2015b; 2018; 2021; 2022b; Constable *et al.,* 2017; Braun *et al.,* 2019). Similar to presented findings AU1 have been confirmed in traumatic pericarditis (Hussain *et al.,* 2018), peritonitis (Hussain *et al.,* 2018) and omasal impaction (Imran *et al.,* 2011) and melena (suggestive of AU1) has been reported in RDH (Hussain *et al.,* 2021). In pulmonary diseases the development of AU1 could be attributed to hypoxia. Hypoxia decreases the resistance of the gastric mucosa and hypercapnia increases the secretion of hydrochloric acid. Theileriosis is well established to cause punched out abomasal ulcers (subtype 1b) and lactic acidosis is reported to cause abomasal erosions (subtype 1a) due to corrosive effect of lactic acid (Constable *et al.,* 2017). AU1 was observed in 58.3% and 32% cases of dystocia and fracture cases which is a new interesting finding and addition to the current knowledge on these disorders.

In this study, 62.9% prevalence of secondary AU1 was found out. The AU1 have been reported with a prevalence of up to 84% in dairy cattle (Munch *et al.,* 2019), 66% in fattening bulls (Hund *et al.,* 2016), up to 77% in veal calves (Brscic *et al.,* 2011), and 56% to 66.4% in adult buffaloes (Tajik *et al.,* 2013; Hussain *et al.,* 2019). The prevalence of AU1 found in this study could be high compared to population of cattle and buffaloes at farms. The bovines included in this study were affected by various disease conditions resulting into death or euthanasia of the animals. The similar prevalence of AU1 between age groups was in agreement with literature data that suggested no significant difference between either sex and between the different age groups (Tajik *et al.,* 2013; Hussain *et al.,* 2019). In contrast to our results, Munch *et al.,* (2019) reported that the prevalence of subtype 1d ulcers increased with increase in parity and that of subtype 1a decreased with increase in parity. However, whether this trend in prevalence with increase in parity is statistically significant or not has not been explained.

There is no consensus about the description of anatomical divisions of abomasum among various studies. Braun *et al.,* (1991) divided the abomasum into fundus and pylorus, without providing a detailed explanation for how fundus and pylorus were distinguished anatomically. Another study (Mesaric, 2005) gave no description of ulcers as per the anatomical location. Although Hund *et al.,* (2016) used a distinct anatomical division of the abomasum, they also did not give a description of how they did so. Munch *et al.* (2019) divided the abomasum into three zones: the zone 1 described by the authors corresponds to our fundus and body and our pylorus correspond to their description of zones 2 and 3.

The occurrence of AU1 subtypes in cattle and buffaloes is reported to differ among various studies. This study documented a significantly higher prevalence of acute (1a and 1b) than chronic lesions (1c and 1d). The higher percentage of subtype 1a and 1b ulcers was in agreement with an earlier slaughterhouse study (Hussain *et al.,* 2019). However, Braun *et al.* (1991) have observed almost equal percentages of type 1a, type 1b and type 1c ulcers in cattle. In another study (Munch *et al.,* 2019), subtype 1c consti-

tuted the highest prevalence (34%) followed by 1a (25%) and 1b (21%).

The significantly higher prevalence of type 1a ulcers in pyloric region and type 1b ulcers in the body region of abomasum was suggestive of different pathogenesis of AU1 in the pyloric and body regions of the abomasum. On one hand it is difficult to comment about the possible different mechanisms for ulcer development in different regions of the abomasum as 58% of ulcers were observed in more than one region of the abomasum. On the other hand, the predilection cannot be ruled out because type 1a ulcers were not accompanied by any other subtype of AU1 while rest of the ulcers had additional lesions of other subtype of ulcers. As type 1a ulcers were solitary and not accompanied by other type of ulcers, it may be affirmed that the pathogenesis of type 1a ulcers may be different from that of other subtype of AU1. The available literature on AU1 in cattle and buffaloes also suggests that each subtype of AU1 has a predilection for a particular site in the abomasal mucosa (Braun *et al.,* 1991; Hussain *et al.,* 2019). Braun *et al.,* (1991) observed that 75% and 71% of subtype 1a and 1c ulcers were in the pyloric region while 71% of 1b and 69% of 1d subtype ulcers were in fundic region. Our results revealed that subtype 1a ulcers had a relatively high affinity for abomasal pylorus whereas subtype 1b ulcers had a higher affinity for the abomasal body. We found that >50% of subtype 1a and 1b were observed in more than one anatomical parts of abomasum (Table 3). The multiple AU1 were more frequent than the solitary ulcers, which was in agreement with previous reports (Tajik *et al.,* 2013; Hussain *et al.,* 2019). Krusalgesia *et al.* (2013) also reported that abomasal ulcers did not occur individually.

The AU1 differed in size, location, morphology and degree of penetration. Majority of the AU1 were located in the caudal third of the abomasal body on parietal surface along the greater curvature of the abomasum while subtype 1a ulcers were mainly located in the pyloric region. Majority of the AU1 were round and <0.5 cm in diameter. Theileriosis-associated ulcers (subtype 1b) were typically observed throughout the abomasal mucosa. It is difficult to estimate the effect of the acute or chronic subtype of AU1 on the production and welfare of the animals but it seems more likely that acute lesions will cause more pain than the chronic ones. Therefore due to acute nature and higher number per abomasum, the acute subtype ulcers (1a and 1b) could have greater effect on the pain perception and hence production of the animals as compared with the animals with more chronic lesions (1c and 1d).

A high prevalence of AU1 of buffaloes and cattle was observed in clinical cases. The diseases with higher percentage of animals having AU1 were traumatic reticuloperitonitis/pericarditis, RDH, intestinal obstruction, peritonitis, bronchopneumonia and theileriosis. Subtype 1a and 1b were observed more frequently ($P<0.01$) than subtype 1c and 1d ulcers. The common location for AU1 was the parietal surface of the abomasal body. The impact on production and reproduction was not evaluated in this study, but the lesions would still be painful to the animal. These ulcers may be an important cause of slow recovery in clinical cases, and hence decreased production and economic losses depending upon the number and type of ulcers in the abomasum. The economic effect of AU1 in bovidae has not been investigated so far and may be taken into consideration in future studies.

The use of drugs to reduce production and secretion of gastric acid would be desirable.

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