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Vagal indigestion in cattle: a retrospective study

Indigestão vagal em bovinos: estudo retrospectivo

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Highlights

Type II vagal indigestion was the most prevalent.
Traumatic reticuloperitonitis was the main disease associated with vagal indigestion.
Hypomotility, abdominal distension, and bloat were the most common clinical signs.

Abstract

This study aimed to carry out a retrospective study of vagal indigestion cases diagnosed in cattle admitted at the Garanhuns Bovine Clinic, Federal Rural University of Pernambuco campus. This syndrome, caused by dysfunctions of the vagus nerve and characterized by motility disorders of the pre-stomachs and abomasum, represented 5.5% (70/1279) of digestive cases diagnosed in a period of 10 years and had an unfavorable prognosis in 78.3% of cases. Type II vagal indigestion was the most prevalent, accounting for 40% of cases, followed by type I (24.3%) and types III and IV, which accounted for 18.6% and 10.0% of cases, respectively. Vagal indigestion in 67.1% (47/70) of cases occurred as a result of other illnesses, such as traumatic reticuloperitonitis (27.7%), lung diseases (12.8%), gastric impaction (10.6%), abomasal ulcer (10.6%), lymphosarcoma (6.4%), and liver abscesses (6.4%). Motor changes in the gastrointestinal tract, such as hypomotility, abdominal distension, and bloat, as well as their consequences, were the most frequent clinical signs. Laboratory, ultrasonographic, and anatomopathological alterations mainly originate from the primary illnesses present in each case. The approach of this disease is essential due to its clinical and economic importance for livestock to expand the knowledge of its etiopathogenesis, thus contributing to a more accurate diagnosis by veterinarians working in the field of internal medicine for cattle.

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Key words: Functional stenosis. Vagus nerve. Hoflund syndrome. Digestive system. Recurrent bloat. Traumatic indigestion.

Resumo

Objetivou-se realizar um estudo retrospectivo dos casos de indigestão vagal diagnosticados em bovinos atendidos na Clínica de Bovinos de Garanhuns, campus da Universidade Federal Rural de Pernambuco. Essa síndrome, causada por disfunções do nervo vago e caracterizada por transtornos de motilidade dos pré-estômagos e abomaso, representou 5,5% (70/1279) dos casos digestivos diagnosticados num período de 10 anos e apresentou prognóstico desfavorável em 78,3% dos casos. A indigestão vagal tipo II foi a mais prevalente, representando 40% dos casos, seguida do tipo I (24,3%) e dos tipos III e IV, que corresponderam a 18,6% e 10,0%, respectivamente. Em 67,1% (47/70) dos casos, a indigestão vagal ocorreu em consequência de outras enfermidades, tais como reticuloperitonite traumática (27,7%), doenças pulmonares (12,8%), compactação gástrica (10,6%), úlcera de abomaso (10,6%), linfossarcoma (6,4%) e abscessos hepáticos (6,4%). As alterações motoras do trato gastrointestinal, tais como hipomotilidade, distensão abdominal e timpania, assim como suas consequências foram os sinais clínicos mais frequentes. As alterações laboratoriais, de imagem e anatomopatológicas são oriundas principalmente das enfermidades primárias presentes em cada caso. Devido sua importância clínica e econômica para a bovinocultura, é primordial a abordagem dessa enfermidade, visando ampliar o conhecimento da sua etiopatogenia contribuindo dessa forma para um diagnóstico mais preciso pelos médicos veterinários atuantes na área de medicina interna de bovinos.

Palavras-chave: Estenose funcional. Nervo vago. Síndrome de Hoflund. Sistema digestivo. timpanismo recidivantes. Indigestões traumáticas.

Introduction

Vagal indigestion or Hoflund syndrome is characterized by changes in the motility pattern of the pre-stomachs and abomasum and occurs due to vagus nerve dysfunction in adult dairy cows and less frequently in beef cattle, buffaloes, calves and small ruminants (Dirksen, 1981; Hussain et al., 2014a; Lacasta et al., 2013; Motta et al., 2017; Reis et al., 2016; Soares et al., 2017). This syndrome is mainly associated with inflammatory processes, such as adhesions resulting from traumatic reticuloperitonitis and perforated abomasal ulcers. But other diseases, traumas, or compression can also cause damage to the thoracic or abdominal vagal trunks, and the course and clinical manifestations of cases are determined by the location and extent of the damage caused to this nerve (Dirksen et al., 2005; Garry & McConnel, 2015).

The syndrome is classified according to the location of the functional disorder in the gastric compartments and comprises four types: type I or failure of eructation, with abdominal distension caused by the accumulation of free gas in the rumen; type II or failure of omasal transport resulting from compromised ingesta flow from the reticulum to the abomasum through the omasal orifice, also classified as anterior functional stenosis; type III or failure of pyloric flow, which results in distension of the abomasum and omasum

due to accumulation of ingesta, also classified as posterior functional stenosis; and type IV or impediment to ingestion flow due to external compression caused by the pregnant uterus in an advanced stage of gestation (Borges & Moscardini, 2007; Perkins, 2017; Ribeiro et al., 2020; Smith et al., 1992). Furthermore, the occurrence of this disease in miniature cattle breeds is classified as idiopathic vagal indigestion (Rizzo et al., 2015).

Considering the clinical importance of this syndrome for cattle farming and the scarcity of studies conducted in Brazil that relate the etiopathogenesis to clinical, laboratory, ultrasonographic, and anatomopathological findings, this study aimed to carry out the clinical-epidemiological, anatomopathological, and laboratory characterization of vagal indigestion in cattle through a retrospective study of cases diagnosed in a period of 10 years in a hospital routine.

Materials and Methods

A retrospective study of clinical cases of vagal indigestion diagnosed in cattle at the Garanhuns Bovine Clinic, Federal Rural University of Pernambuco campus (CBG-UFRPE), was carried out over a period of ten years (January 2008 to December 2017).

Initially, screening was carried out in the registration books, where a total of 70 cases diagnosed during the study period were found. The clinical records were then consulted for data collection. The diagnosis establishment in all animals was performed through clinical examination (Dirksen, 1993) and complementary examination, when necessary, according to the institution's protocol. The classification adopted by the institution for this syndrome is based on the location of the functional disorder in the gastric compartments, as follows: type I or failure of eructation; type II or failure of omasal transport; type III or failure of pyloric flow; and type IV or partial obstruction of the gastric compartment associated with advanced gestation (Smith et al., 1992).

Blood count, total plasma protein, and plasma fibrinogen were performed in 64 animals from the collection of a blood sample in a tube with EDTA anticoagulant (10%) through jugular venipuncture (Harvey, 2012). Ruminal fluid samples were analyzed in 57 cattle (Dirksen, 1993) and the concentration of chloride (Cloretos Liquiform, Labtest) was measured in 31 of these samples. The ultrasound examination was performed in 27 animals, according to Braun (2009a), using a Mode B device (Z6 Vet, Mindray Bio-Medical Electronics Co. Ltd., Shenzhen China) and a convex transducer with frequencies of 5.0 MHz (Z6 Vet). Laparotomy was performed in 13 animals for diagnostic and/or therapeutic purposes. However, it is noteworthy that the use of this invasive technique for diagnostic purposes has been replaced by imaging examination (ultrasound). All cattle that died naturally or were euthanized (Luna & Teixeira, 2007) during hospitalization were submitted to anatomopathological examination (n=54).

The data collected from clinical records were tabulated in an electronic spreadsheet (Microsoft Office Excel 2016) and analyzed using descriptive statistics in the Minitab 18 software (Petrie & Watson, 2013).

Results and Discussion

The 70 cases of vagal indigestion diagnosed in cattle represented 1.4% (70/5063) of the total number of animals of this species admitted in the studied period and 5.5% (70/1279) of the digestive disorders diagnosed at CBG-UFRPE.

Type II vagal indigestion was the most prevalent, representing 40% (28/70) of cases, followed by type I with 24.3% (17/70), type III with 18.6% (13/70), and type IV with 10.0% (7/70). Furthermore, 7.1% (5/70) were classified as idiopathic since it was diagnosed in animals of the Santa Rosália breed (miniature cattle).

Vagal indigestion occurred as a result of other diseases in 67.1% (47/70) of cases, with traumatic reticuloperitonitis being the most frequent with 27.7% of cases (13/47), followed by pulmonary diseases with 12.8% (6/47), gastric impaction (abomasal and forestomach impaction) with 10.6% (5/47), abomasal ulcer with 10.6% (5/47), lymphosarcoma with 6.4 (3/47), and liver abscesses with 6.4% (3/47) of cases. Other diseases such as diaphragmatic hernia, frothy bloat, right abomasum displacement with volvulus, and cardia papilloma were also associated with the occurrence of vagal indigestion although less frequently.

The main changes reported by the owners in the anamnesis were apathy, decreased appetite and milk production, lack of rumination, and chronic bloat. The age of affected animals ranged from four months to eight years, with higher occurrence in animals older than 24 months, which were mostly crossbred dairy cows, raised in a semi-intensive regime. In addition, this disease was more prevalent during the dry period of the year (September to March) (Table 1).

Table 1
Epidemiological data of cattle diagnosed with vagal indigestion and admitted at the Garanhuns Bovine Clinic (CBG-UFRPE) from January 2008 to December 2017

s.d	n	Category	Number of animals	
			Absolute value	Relative value (%)
Sex	70	Female	61	87.1
		Male	9	12.9
Breed	66	Crossbred	45	68.2
		Holstein	13	19.6
		Miniature cattle	4	6.1
		Others	4	6.1
Age	53	< 2 years	13	24.5
		> 2 years	40	75.5
Raising system	60	Intensive	8	13.3
		Semi-intensive	43	71.7
		Extensive	9	15.0
Season	70	Dry	48	68.6
		Rainy	22	31.4

Table 2
Clinical findings of cattle diagnosed with vagal indigestion and admitted at the Garanhuns Bovine Clinic (CBG-UFRPE) from January 2008 to December 2017

Characteristic	n	Clinical findings	Number of animals	
			Absolute value	Relative value (%)
Posture	69	Stading	66	95.7
		Recumbency	3	4.3
Appetite	68	Present	35	51.5
		Absent	16	23.5
		Decreased	17	25.0
Temperature (°C)	64	Physiological (38.0-39.0)	33	51.6
		Hypothermia (< 37.5)	4	6.2
		Fever (> 39.0)	27	42.2
Dehydration	68	Absent	10	14.7
		Slight (6%)	15	22.1
		Moderate (8%)	29	42.6
		Severe (10%)	14	20.6
Heart rate	67	Physiological (60-80)	31	46.3
		Bradycardia (<60)	18	26.9
		Tachycardia (>80)	18	26.9
Respiratory rate	68	Physiological (24-36)	32	47.1
		Bradypnea (<24)	21	30.9
		Tachypnea (>36)	15	22.1
Ruminal motility	68	Physiological (2 - 3/min)	9	13.2
		Hypomotility	40	58.8
		Hypermotility	11	16.2
		Atonic	8	11.8
Ruminal bloat	68	Absent	22	32.4
		Present	46	67.6
Ruminal stratification	67	Defined	16	23.9
		Undefined	51	76.1
Abdomen shape	68	Physiological	19	27.9
		Bilaterally distended	22	32.4
		Unilaterally distended	6	8.8
		Apple-pear	21	30.9
Feces	53	Present	31	58.5
		Scanty	18	34.0
		Absent	4	7.5
Feces digestibility	30	Well digested	8	26.7
		Poorly digested	12	40.0
		Overly digested	10	33.3

Motor changes in the gastrointestinal tract, such as hypomotility or hypermotility with reduced range of motion, bloat, undefined rumen stratifications, increased abdominal tension with unilateral or bilateral distension, and/or apple-pear abdominal contour stood out among the most frequent clinical findings (Table 2) Moreover, the transrectal palpation showed that the rumen of these animals was predominantly distended and had a firm consistency in some cases and extended to the right antimere, assuming an L shape (Figure 1). Poorly digested feces with large particles or overly digested was another frequently recorded change (Figure 2).

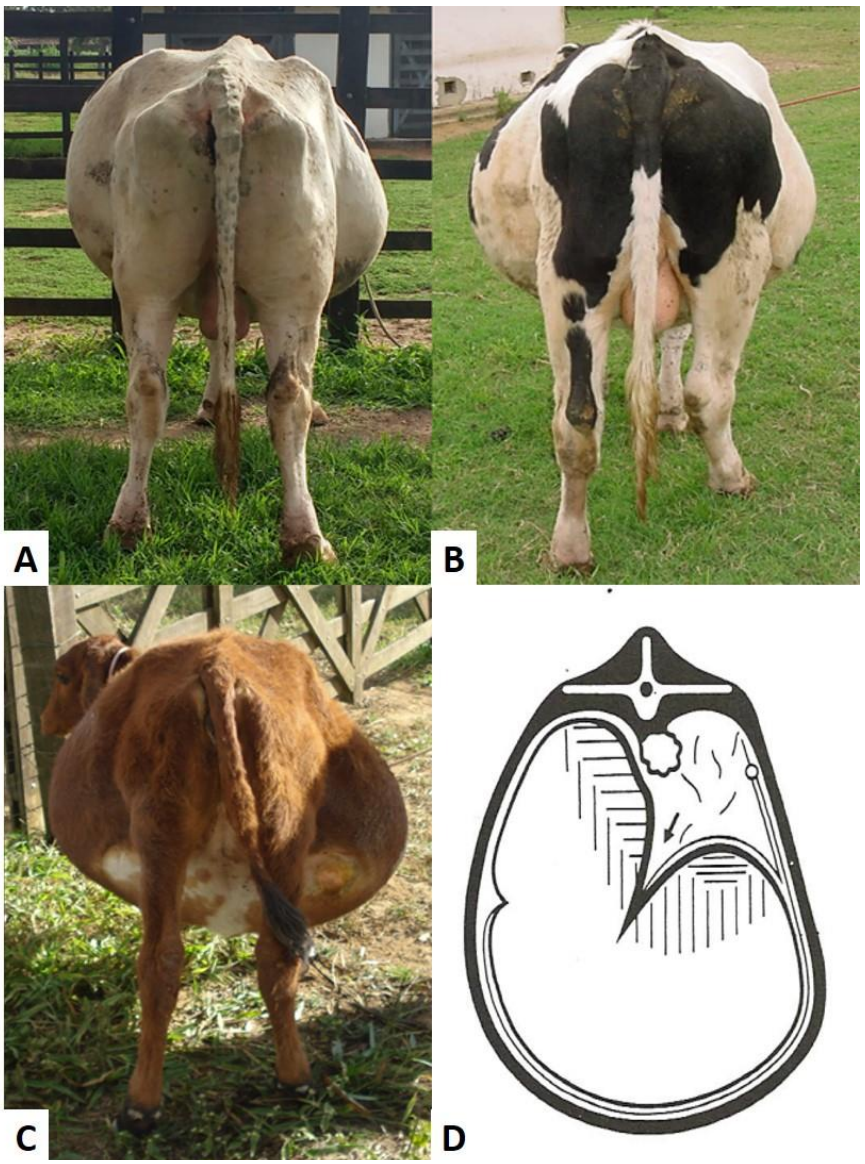

Figure 1. A, B, and C - Cattle with vagal indigestion diagnosed at Garanhuns Bovine Clinic (CBG-UFRPE), showing bilateral abdominal distension and apple-pear abdominal contour. D - Schematic representation of the main finding found through rectal palpation in cattle with vagal indigestion. Source: Dirksen, (1993).

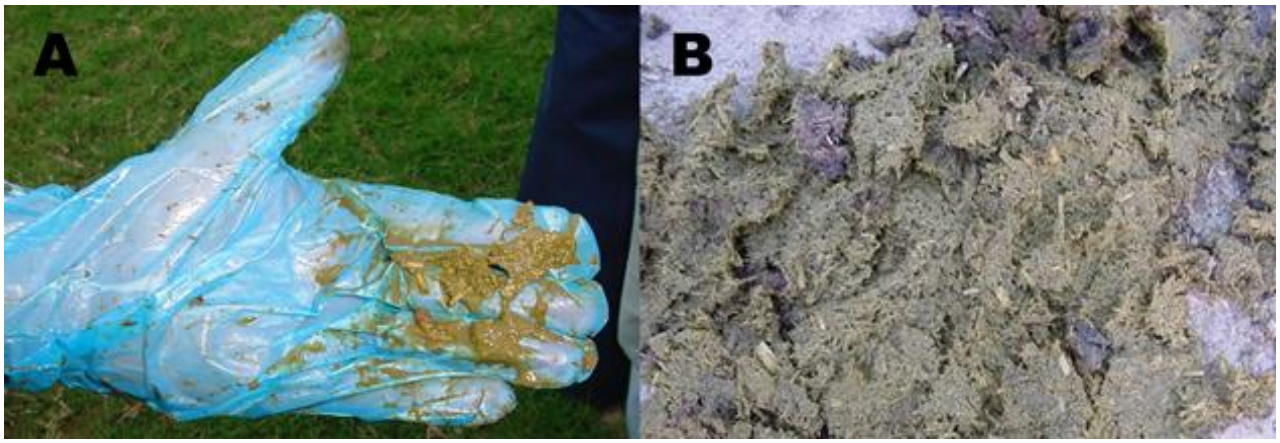

Figure 2. Feces characteristics of cattle with vagal indigestion. A - Glove after rectal palpation, with scanty, poorly digested feces and presence of mucus. B - Dry feces, poorly digested and with large particles. Garanhuns Bovine Clinic (CBG-UFRPE).

Regarding the clinical outcome, the prognosis was unfavorable in 78.3% (54/70) of the cases, in which the animals died naturally or were euthanized with authorization from the owner or sent to sanitary slaughter. In the other cases (21.7%), the animals were discharged from the clinic after performing a therapeutic approach.

The hematological findings are described in Table 3. The main changes were seen in the white blood cell count, in which the median of total leukocytes was above normal and more than 75% of the animals presented neutrophilia. The analysis of the ruminal fluid showed a change mainly in the percentage of live infusoria, which was lower than 50% in more than 75% (44/57) of the analyses, the methylene blue reduction test (MBRT), which did not occur or occurred after the expected time in more than 60% of the samples, and the concentration of chloride, was high (> 30 mEq/L) in 83.9% of the 31 animals in which the measurement was performed.

Table 3
Median and interquartile range of hematological parameters of cattle with vagal indigestion (n=64) admitted at the Garanhuns Bovine Clinic (CBG-UFRPE) from January 2008 to December 2017

Parameter	Q1a	Median	Q3b	Reference valuec
Red cells (x 10^6 µL)	6.3	6.9	7.7	5.0-10.0
Hematocrit (%)	28.0	31.0	35.0	24-46
Hemoglobin (g dL^{-1})	9.2	10.1	11.4	8.0-15.0
MCV[a] (*f*L)	40.8	44.2	50.0	40-60
MCHC[b] (%)	31.6	32.8	34.2	30-36
Total leukocytes (µL)	11050.0	14925.0	20912.5	4000-12000
Lymphocytes (µL)	5025.0	6424.5	8296.8	2500-7500
Monocytes (µL)	56.0	213.0	337.5	25-840
Eosinophils (µL)	0.0	0.0	217.0	0-2400
Segmented neutrophil (µL)	5151.0	7599.0	10544.0	600-4000
Rod cells (µL)	0.0	0.0	221.0	0-120
Total plasma protein (g dL^{-1})	7.0	8.0	8.3	7.0-8.5
Plasma fibrinogen (mg dL^{-1})	400.0	700.0	800.0	300-700

[a]First quartile; [b]Third quartile; [c]Jain (1993).

The ultrasonographic and anatomopathological examination showed changes originated mainly from the primary diseases present in each case. However, some changes are commonly observed in vagal indigestion, such as increased reticular contractions (four to seven biphasic contractions in three minutes), increased echogenicity of the ruminal content seen on ultrasound (Figure 3), intense dilatation of pre-stomachs and/or abomasum, and ingesta with foamy consistency at necropsy (Figure 4).

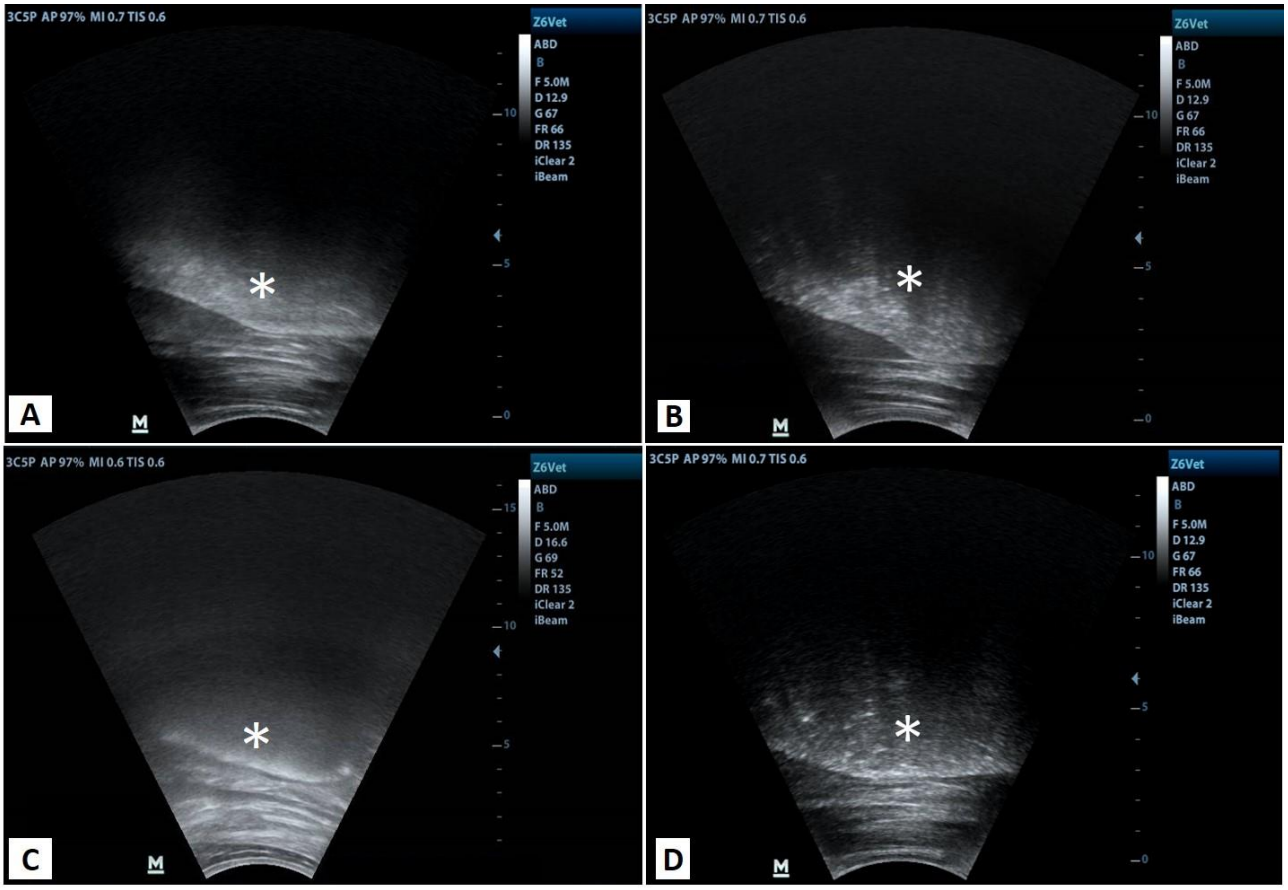

Figure 3. Abdominal ultrasound in cattle. A and C - Ruminal content in cattle without change of reticular motility (asterisk). B and D - Ruminal content showing increased echogenicity (asterisk) in cattle with increased reticular motility. Garanhuns Bovine Clinic (CBG-UFRPE).

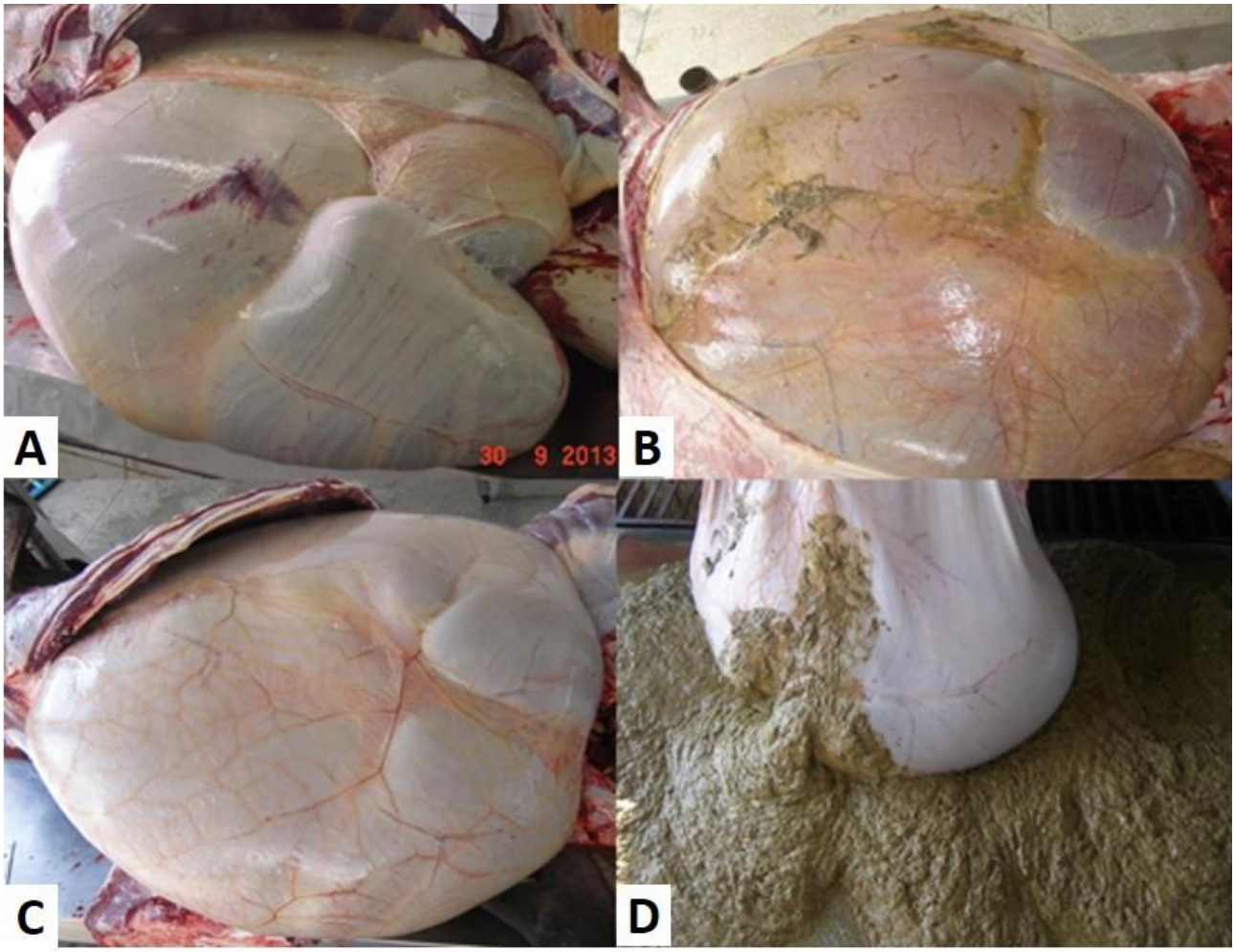

Figure 4. Necroscopic examination of cattle with vagal indigestion. A, B, and C - Pre-stomachs distended due to digesta accumulation. D - Loss of ruminal content stratification, with a predominance of content with foamy consistency. Garanhuns Bovine Clinic (CBG-UFRPE).

The frequency of occurrence of vagal indigestion in cattle found in the present study shows that this clinical condition stands out among the digestive diseases that affect these animals, corroborating with Marques et al. (2018) and Sharma et al. (2015).

Although case reports are present in the literature (Motta et al., 2017; Reis et al., 2016; Romão et al., 2012), a retrospective study that describes a series of cases (n=70) with clinical, epidemiological, laboratory, and ultrasonographic data is still scarce. A single study with similar characteristics, published in the early 1980s by Dirksen (1981) in Germany, was found in the literature.

Animals admitted at CBG-UFRPE often come from municipalities that make up the largest dairy region in the state of Pernambuco, Brazil. Thus, the higher frequency of this disease, found in crossbred dairy cows raised in a semi-intensive system is related to the characteristics of the livestock sector in the

region. Vagal indigestion can also affect beef cattle, buffaloes, goats, and sheep (Hussain et al., 2014a; Lacasta et al., 2013; Reis et al., 2016; Soares et al., 2017). Moreover, the occurrence of this disease has no predilection for sex, being recorded in males and females in a similar proportion (Sharma et al., 2015). Although the occurrence of vagal indigestion was more frequent in animals older than 24 months, it has also been recorded in younger animals (Motta et al., 2017; Reis et al., 2016). It suggests that age is not a variable that influences the occurrence of this disease, as observed for sex.

The occurrence of vagal indigestion in Santa Rosália cattle (miniature cattle) was also reported in the states of São Paulo and Pernambuco. The occurrence of this syndrome in this breed is probably due to genetic and/or anatomical factors, such as the chondrodystrophic conformation, in which the small abdominal cavity compared to the expansion of gastric compartments can favor the vagus nerve compression (Rizzo et al., 2015).

The occurrence of vagal indigestion as a result of other primary illnesses has also been reported by other authors (Fubini et al., 1985; Rehage et al., 1995; Gordon, 1997; Motta et al., 2017). The lesions frequently observed in cases of type II vagal indigestion (peritonitis and/or adhesions) corroborate with those described by Constable et al. (2017), who stated that complications of traumatic reticuloperitonitis, mainly reticular adhesions, are the most common causes of vagal indigestion. Other less frequent causes associated with failure of omasal transport were the formation of liver abscesses, frothy bloat, and diaphragmatic hernia. The aboral ingesta flow from the omasum to the abomasum can be compromised in these situations, leading to the clinical condition of vagal indigestion, as described by other authors (Costa et al., 2013; Fubini et al., 1985; Soares et al., 2017).

Type I vagal indigestion, the second most prevalent type in this study, was mainly associated with pulmonary diseases such as pleuritis and pneumonia and less frequently related to enzootic bovine leukosis and cardia papilloma. In these cases, the syndrome derives from compression and/or inflammatory involvement of the thoracic vagal trunks, interfering with the eructation and vagal control of the gastric function, causing chronic ruminal bloat (Perkins, 2017).

Abomasal affections such as ulcer, impaction, and right displacement with torsion were the main primary illnesses related to type III vagal indigestion. In these cases, the impairment in the ingesta flow through the pylorus results in food accumulation inside the abomasum and a clinical condition of vagal indigestion. According to Sattler et al. (2000), a gastric dysfunction similar to vagal indigestion occurred in approximately 13% to 17% of cattle diagnosed with RAD or abomasal volvulus and treated surgically. For these authors, motor dysfunction in this condition can be types II, III, or IV, with structural damage to the abomasal wall, peritonitis, and vagus nerve damage being the main causes of clinical signs compatible with the syndrome.

Animals with type IV vagal indigestion, less frequently diagnosed, were at the final third of gestation. Functional pyloric stenosis can occur partially and reversibly during the advanced stage of gestation due to

compression of the proximal portions of the small intestine caused by the pregnant uterus (Hussain et al., 2014b).

The motor dysfunction of the gastrointestinal tract found in the present study, characterized by hypomotility, bloat, and marked abdominal distension, has also been described by other authors (Dirksen, 1981; Hussain et al., 2014a; Romão et al., 2012). Abdominal distension occurs mainly due to gas and fluid accumulation in the rumen-reticulum and/or due to abomasal impaction. According to Hussain et al. (2017), this is the most important clinical change in the disease. The authors emphasized that failures in the eructation process and impairments in transporting the ingesta through the reticulo-omasal orifice and pylorus may have a neurogenic and/or mechanical origin.

The changes observed in gastrointestinal motility can be initially attributed to inflammatory processes resulting from primary conditions commonly associated with vagal indigestion, which may involve the vagus nerve (Foster, 2017). The pain and fever generated in these processes are considered important inhibitory factors, influencing the gastric center responsible for controlling the motility of pre-stomachs (Rehage et al., 1995). Also, the accentuated distension of the pre-stomachs greatly contributes to the motor dysfunctions in this disease since the stimulation of low-threshold tension receptors, present in the rumen wall, drastically reduces the intensity of rumino-reticular contractions, increasing their frequency, with a subsequent decrease in contractions with the progressive increase in distension, which can lead to atony (Foster, 2017; Hussain et al., 2017; Rehage et al., 1995). According to Rehage et al. (1995), extensive reticular adhesions are another common cause of changes in gastrointestinal motility, often found in cases of traumatic reticuloperitonitis.

Ruminal hypermotility was not a very consistent motor change, which was mainly associated with cases of type II vagal indigestion. Similarly, Rehage et al. (1995) also reported a low occurrence of hypermotility in cows with traumatic reticuloperitonitis and clinical condition of vagal indigestion.

The loss of ruminal stratification found in more than 75% of animals is a clinical change often described in cases of vagal indigestion (Reis et al., 2016; Rizzo et al., 2015; Soares et al., 2017). According to Borges and Moscardine (2007) and Constable et al. (2017), changes in the reticulo-ruminal contraction pattern lead to blurring of ruminal strata, making the content more homogeneous and with a viscous to a foamy consistency.

In addition to the motor dysfunction of the gastrointestinal tract, dehydration was a very consistent clinical change in vagal indigestion, which may occur in different degrees. This condition can be attributed to inappetence and/or decrease in fluid intake commonly presented by animals, as well as the interruption of the ingesta flow to the intestine, which is considered the greatest site of fluid absorption. Other studies have also pointed to dehydration as a clinical change commonly present in this syndrome (Hussain et al., 2014a; Motta et al., 2017; Reis et al., 2016; Romão et al., 2012).

Although bradycardia is a clinical change usually attributed to vagal lesions (Dirksen, 1981), less than 30% of the animals in the present study showed this alteration, which reinforces the assertion

that this change is not present in all cases and, consequently, the possibility of vagus nerve damage should not be ruled out in the absence of bradycardia.

Leukocytosis due to neutrophilia found in the hematological examination is not specific for the disease, being essentially related to the primary causes of the vagal lesion. This type of hematological response was also evidenced by Fubini et al. (1985) and Hussain et al. (2014b), who considered it compatible with the inflammatory process resulting from the peritonitis present in these cases.

Changes found in the rumen fluid analysis characterize the impairment of the ruminal microbiota, which are mainly due to the change in the pattern of contraction of the reticulo-rumen. On the other hand, the increase in concentration of chloride often observed in cases of type III and IV vagal indigestion can be explained by the reflux of abomasal content into the rumen due to a failure in the pyloric flow. These changes corroborate what has been reported by other authors (Foster, 2017; Hussain et al., 2014a; Lacasta et al., 2013).

In addition to alterations in the primary disease, the change in the reticular contraction pattern, such as hypermotility, was the most prevalent finding in the ultrasound examination. This change was also observed by Braun et al. (2009b), who reported an exacerbated pattern of reticular contractions in cattle with vagal indigestion. Therefore, reticular hypermotility can be an indicator of the presence of a vagal lesion although a normal or even reduced motility pattern predominates in many cases, especially in the presence of peritonitis.

The anatomopathological findings observed in the pre-stomachs and abomasum may be justified by the impairment of the ingesta flow, content retention, and distension of these organs as a result of lesions caused by primary diseases, which compromised the proper nerve function. Also, the ingesta can be liquid or foamy, with large food particles due to changes in the pattern of ruminal contractions (Borges & Moscardine, 2007; Constable et al., 2017).

Conclusion

There is a clinical and economic relevance of the disease for the livestock sector, as the severity and chronicity of clinical signs contribute to the unfavorable outcome of the disease. Type II is the clinical condition commonly diagnosed and often associated with the inflammatory process and adhesions resulting from traumatic reticuloperitonitis. The disease is characterized by motor dysfunction of the gastrointestinal tract associated with dehydration and marked abdominal distension resulting from the retention of digesta and gas. Although laboratory, ultrasonographic, and anatomopathological changes do not have specificity for the syndrome, the use of these complementary examinations substantially contributes to higher diagnostic precision and better prognostic assessment of the disease.

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Conflicts of interest

The authors declare that there are no conflicts of interest regarding this study.

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