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Vagus indigestion in bovines: A review in historical perspective

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Abstract
In 1940, Hoflund, in his experimental study, produced chronic indigestion in cattle simulating functional stomach disorders after selective vagotomy. Hoflund concluded that injury to the vagus nerve was the main etiological factor for the production of chronic indigestion and hence coined the term 'vagal indigestion.' After the name of the scientist, vagal indigestion is also referred to as Hoflund syndrome. The causes of vagus indigestion are numerous but currently, reticular adhesions or vagal nerve injury due to traumatic reticuloperitonitis is considered to be the most common cause. The typical clinical symptoms are progressive abdominal distension resulting into papple shaped abdomen, dehydration and scanty faeces. The common biochemical alteration is metabolic alkalosis with hypokalemia and hypochloremia. In this review article, the authors have summed up the historical and current perspectives of vagus indigestion in cattle and buffaloes.

Keywords: Vagus indigestion, bovine, late pregnancy, treatment

Introduction
Throughout the world, the diseases of the digestive tract in ruminants especially cattle and buffaloes constitute a major clinical problem. It is often difficult to decide whether a gastrointestinal disease requires medicinal or surgical treatment or both. One of such conditions is vagal indigestion. Vagal indigestion comprises of a group of gastrointestinal disorders of cattle and buffaloes resulting from mechanical or functional obstruction of the forestmach and/or abomasum outflow [1-5]. There is accumulation of ingesta in the forestomach or abomasum, reduced appetite or anorexia, scant faeces, decreased milk production, acid base abnormalities, dehydration, weight loss, and eventually weakness and recumbancy. Prognosis is usually unpredictable and often considered poor except in late pregnancy indigestion [6]. For many years, vagus indigestion was considered to be caused by vagal nerve injury because Hoflund [7] was able to stimulate clinical signs of the disease in experimental animals after selective vagotomy. Hence vagal indigestion was/is also known as Hoflund's syndrome. In fact any case of chronic indigestion with clinical signs similar to of traumatic reticuloperitonitis and not responding to routine treatment was described as vagal indigestion. Leek [8, 9] did extensive work on the patho-physiology of forestomach and abomasum diseases and came to conclusion that reappraisal of the so called 'vagal indigestion' was necessary. Leek concluded that a clinical lesion must destroy more than 50% of vagal innervations to the forestomach and abomasum to produce signs observed in clinical cases, and this is an unlikely situation.

Later vagal indigestion was described as two distinct entities i.e. functional disorders of the forestomach and abomasum [10-14]. In forestomach disorders or cranial functional disorders, there appears to be achalasia of the reticulo-omasal orifice resulting into delayed or cessation of passage food across the omasum [14]. Hence the term omasal transport failure (OTF) is also used [15, 16]. In the abomasal disorder or caudal functional disorder, there may be either achalasia of the pylorus (leading to abomasal distension or atony) [14] or the primary abomasal impaction leading to its atony [17]. Another name for caudal functional disorder is functional pyloric stenosis or secondary abomasal impaction. In some cases both cranial and caudal disorders may be present together, depending on duration of illness. Prolonged illness may turn a cranial disorder into a caudal functional disorder due to intestinal ileus in advanced gastrointestinal cases. It is important for the clinicians to remember that, there is no real obstruction of the reticulo-omasal orifice or pylorus but rather achalasia exists thus stimulating the effects of obstruction or stenosis.

Based on the clinico-pathological observations of 42 cows with complications of traumatic reticuloperitonitis including vagus indigestion, it is now proposed that the disturbances in the flow of ingesta are associated with particle-separation in the reticulo-rumen caused by mechanical inhibition of reticular motility associated with extensive adhesions of the reticulum [14]. Currently this disease is considered as a syndrome which includes functional disorders of rumeno-reticulum, omasum and abomasum.

Incidence

The prevalence of vagus indigestion has not been documented by a well-designed epidemiological study. The incidence of pyloric obstruction associated with impaction of the abomasum was recorded in cattle by Chinayya [18] and later on pyloric stenosis was reported by Arthur [18]. Subsequently, impairment in the outflow of forestomach and abomasal contents was documented by many workers [10-12, 15, 20-25]. However, the information about this disorder in buffaloes is scanty. The work done in this species is that of Behl *et al* [13] and Hussain *et al* [6]. Hussain *et al* [6] reported that out of 268 bovines (173 buffaloes and 95 cattle), suffering from gastrointestinal disorders, 15 animals (8 buffaloes and 7cows) had late pregnancy indigestion.

Etiology

The stomach of ruminants is innervated by vagus and splanchic nerves, however, it is the former which is essential for the cyclic movements. At the level of diaphragm, the ratio of sensory to motor fibres in the vagus nerve is 9:1, and therefore, vagus essentially has a sensory role [26]. Both left and right vagus nerve divides in the thorax into a dorsal and a ventral branch. Then two dorsal branches combine to form a dorsal trunk and two ventral branches form a ventral trunk. Dorsal trunk innervates the medial and caudal part of the reticulum, rumen, omasum and abomasum. The ventral trunk innervates the cranial part of the reticulum, omasum and abomasums, but not the rumen [27].

In his experimental study on selective vagotomy, Hoflund [7] concluded that injury to the vagus nerve is the main aetiological factor for the production of chronic indigestion and hence coined the term 'vagal indigestion'. After the name of the scientist, 'vagal indigestion' is also referred to as Hoflund syndrome. Hoflund classified the disturbances of stomach into four types:

1. Functional stenosis between the reticulum and omasum with atony of the rumen and reticulum
2. Functional stenosis between the reticulum and omasum with normal or hyperactive ruminal and reticular activity.
3. Permanent functional stenosis of pylorus with atony or retained activity of the reticulum
4. Incomplete pyloric stenosis, includes late pregnancy indigestion

In 1953, Clarke [28] corroborated the findings of Hoflund. However, after at that time of Hoflund and until many years, many clinicians believed that involvement of the vagus nerve was not the sole factor in such cases. In one study on digestion disorders, Neal and Edwards [1] observed that out of 52 cases, only nine were possibly due to vagus involvement. They were of the opinion that injury or involvement of the vagus nerve was only one of the several factors responsible for functional disorders of the stomach of ruminants.

Dysfunction of the low thresh hold reticular tension receptors (in medial surface of reticulum) produced as a sequel to the traumatic reticulitis or actinobacillosis affecting the reticular wall is considered to be the possible cause of the vagus indigestion [1, 14, 28]. A study on vagotomy concluded that for production of the reticulo-rumen hypomotility, the lesion must destroy more than 50% of the vagus innervations [28]. The cases where there is no involvement of the vagus nerve, the fibrosis and induration of the medial wall of the reticulum occurs (region with maximum density of tension receptors). Therefore, Leek [28] hypothesised that induration of this area would abolish normal tension receptor drive to the gastric centres thus accounting for those cases of hypomotility which are not accompanied by significant vagal nerve damage. Currently the main cause of vagus indigestion is divided into two main sequels of traumatic reticuloperitonitis (TRP), vagal nerve injury and reticular adhesions [14]. Indigestion of late pregnancy, considered a type of vagus indigestion (type IV), is a less well defined syndrome of partial pyloric obstruction or generalized ileus, in which the rumen and abomasum are grossly distended, but the cause is uncertain [6]. In such cases, the fetus may physically impede pyloric outflow or proximal intestinal motility. A vagal indigestion–like syndrome may be a postsurgical complication of abomasal volvulus [29].

In addition to vagal nerve damage, a number of mechanical causes have been implicated as cause functional disorders. These mechanical causes can be intraluminal or extra luminal. The intraluminal causes include ingestion of materials like sand gravel, hair balls, cloth and foreign bodies, coarse food material, presence of neoplasia such as lymphosarcoma or fibropapilloma adjacent to stomach, fibromyxoma of cardia [14-15, 22, 30-34].

Extra luminal mechanical lesions, which can result in defects in the stomach motility in cattle clinically resembling Hoflund's type II and IV, can be wide spread reticular adhesions or ruptured diaphragm. Peritoneal adhesions, adhesions between fundus of the abomasums and the ventral sac of rumen can result in abomasal impaction [21-22]. Liver abscess can also cause omasal transport failure [35].

However, functional disorders of the forestomach and abomasum may exist under diverse conditions and causative factors may sometimes be located away from the gastrointestinal tract or even may not be related to the gastrointestinal tract [10-13]. Diseases such as pneumonia, nephritis, metritis, viral infections and unlocated inflammatory processes in the body have been incriminated as cause of functional stomach disorders. [10-11]. This may be explained by the fact that gastric centre in medulla is influenced by nervous activity in other parts of the nervous system [8], and, fever and/or pain at any site in the body can influence the motility of the stomach [14, 36].

So, in conclusion the impairment in the transport of ingesta across the reticulo-omasal orifice and or the pylorus can be either neurogenic or mechanical in origin.

Clinical Signs

Over the years, both cranial and caudal functional disorders have been reported to exhibit the signs of abdominal distension, tympany, anorexia, dehydration, scanty faeces, depression, muscular weakness, apthay and indifference to normal stimuli, sunken eyes, dry muzzle, and cold extremities [14, 22, 31, 33, 37-38].

The most important clinical sign is progressive abdominal distension. Usually when an animal becomes anorectic, the abdomen size decreases. However, if an animal retains normal

fullness or abdomen becomes distended then functional disorders of the stomach should be kept in differential diagnosis list. The accumulation of gas and fluid in the rumeno-reticulum is the major cause for the distension. The abdominal distension usually involves whole left side of abdomen and right ventral quadrant of abdomen, giving 'apple shaped' appearance to left side and 'pear shaped' appearance to the right side when viewed from behind. Both the shapes when considered jointly form a 'papple shape' [1, 14, 21, 31, 36, 39, 40] (Fig. 1a & b). Although papple shaped abdomen is considered to be a characteristic in cattle, in buffaloes it may be observed only in few cases [6] and even in cases where the primary diagnosis is not vagal indigestion [41].

The faecal output is decreased gradually with an increase in undigested particles [1, 13-14] (Fig. 2) and sometimes there may be complete cessation of faeces [6, 12, 14, 40]. The consistency of faeces is usually pasty [14]. Melena may be noticed in some cases due to ulceration of the abomasum [42]. In all cases weight loss occurs but may go unnoticed by the owner because abdominal enlargement masks the fact that animal's muscle mass is shrinking [14, 26 42]. In dairy cattle, there is progressive decrease in milk production.

Rectal temperature is usually normal but subnormal temperature has also been recorded [13, 15, 23, 32, 40]. The respiration remains unaffected in most of the cases but may be increased due to severe abdominal distension. Bradycardia has been observed as a classic sign of vagus indigestion in cattle but is not always present [1, 14, 16, 40, 42]. Vagotonic bradycardia can be differentiated from other forms of bradycardia by means of atropine test. The increased pulse rate may occur in pyloric obstruction [7, 23, 32]. In buffaloes, heart rate is usually in the normal range in clinical cases of functional stomach disorders [43].

Rumeno-reticular motility may be normal, decreased or increased [40]. Initially the motility is normal or increased due to activation of low thresh hold tension receptors (LTHTR) present in reticulum. These receptors are excitatory for rumino-reticular motility and are activated by mild to moderate abdominal distension observed in initial stages of vagus indigestion [14]. But as rumeno-reticular distension increases, the motility is reduced in frequency and amplitude due to stimulation of high thresh hold tension receptors (HTHTR). These HTHTR are inhibitory for rumeno-reticular moltility [14]. When distension becomes more severe, movements cease, probably due to over distension of the ruminal wall resulting in higher degree of HTHTR activation. During the stage of rumeno-reticular motility, the contractions are usually ineffective to propel the ingesta beyond the first two chambers of stomach, so the contents of the rumen-reticulum become homogenous or mushy (Fig. 3) in consistency due to continuous and prolonged churning [14, 16]. The differention of hypermotile and hypomitle/amotile rumen stage is observed only in cases of OTF and usually not in functional pyloric stenosis.

Haematological Alterations

The changes in packed cell volume (PCV) and haemoglobin are not consistent. Changes in these parameters usually depend on the chronicity of the disease and the degree of dehydration that may develop. Due to more acute nature and higher degree of dehydration in functional pyloric stenosis, the PCV usually increases [12]. In buffaloes packed cell volume may remain within the normal range even though the clinical signs of dehydration are present [6]. In omasal transport failure, the PCV may remain within the normal range or may slightly increase [15, 35]. Increase in the total white blood cell count with an increased percentage of either band cells or polymophonuclear cells or both is observed, especially in cases which occur as a sequel to TRP [10, 11, 16, 35]. However, normal range of leukocytic count has also been reported in acute pyloric functional stenosis in cattle [12] and in buffaloes having cranial or caudal functional disorder [44]. Most of the animals show hyperproteinemia and hyperfibrinogenaemia [12, 35]. However normal values of total plasma proteins in such case have also been reported [11].

Biochemical Alterations

The metabolic changes are more severe functional pyloric stenosis as compared to omasal transport failure., due to well known syndrome of abomasal reflux. As a result of abomasal reflux, the abomasal contents get refluxed back into reticulo-rumen and causes dehydration, hypochloremic hypokalemic metabolic alkalosis, azotemia and significant increase in ruminal fluid chloride concentration [2, 6, 11-13]. The plasma sodium concentration after functional pyloric stenosis or after ligation of pylorus has been reported to be unaffected [11-12]. Reduction in the sodium levels of ruminal fluid has been reported in cattle after pyloric functional stenosis but not after ligation of the pylorus [11]. The reflux of the abomasal contents into forestomachs results in marked decrease in the buffering capacity of the rumen fluid although rumen pH may or may not change [11].

In omasal transport failure, the ingesta accumulate in the rumeno-reticulum, as it fails to pass through the reticulo-omasal orifice. But the abomasal fluid can pass on to the duodenum, therefore, no metabolic alkalosis can be expected. Significant changes in the plasma bicarbonate concentration have not been observed in cattle with OTF [10, 12]. However reduction in the sodium concentration in the rumen fluid has been reported [10]. As the abomasal reflux is absent in the OTF, significant changes in the chloride and potassium concentration in the plasma and rumen fluid do not occur. Blood urea nitrogen and creatinine, and pH and the buffering capacity rumen fluid shows non-significant variation [10]. But it is important to consider that even in advanced cases of OTF, especially the chronic cases where ileus sets in, abomasal reflux can occur to some degree.

In chronic non-specific cases of functional gastrointestinal disorders, the reported biochemical alterations are deranged liver and kidney function tests, increased mean level rumen chloride, and decreased mean levels of calcium, phosphorus, potassium and chloride [44].

Diagnosis

The history and general clinical picture are important, yet these give nonspecific indication of the disorder. The diagnosis of the functional pyloric stenosis can be made when an abomasal reflux syndrome is indicated by increased rumen fluid chloride concentration, hypochloremic, hypokalemic metabolic alkalosis along with clinical signs especially papple shape of the abdomen and reduced defecation [1, 10-13,31]. In addition laprorumenotomy would show distension of the abomasum and rumen [12]. The degree of hypochloremia, hypokalemia, metabolic alkalosis and azotemia can be used to differentiate OTF and pyloric functional disorder [40]. It is considered that the detection of rumen content chlorine levels, might be helpful, especially in the diagnosis of functional gastric stenosis, and that the glutaraldehyde test might be useful

in detecting back functional stenosis cases due to TRP [45].

The diagnosis of late pregnancy indigestion (LPI) is made on the basis of clinical examination, presence of abomasal reflux, and spontaneous recovery after induced or natural parturition [6]. In some animals that die during the course of LPI, diagnosis is established after clinical examination and necropsy examination. However it is important to mention that before predicting the diagnosis other causes of gastrointestinal dysfunction have to be ruled out by physical examination and other ancillary tests.

Differential Diagnosis

The functional disorders of ruminant stomach have to be differentiated from conditions such as ruminal tympany, displaced abomasum, diaphragmatic hernia, intestinal obstruction, paralytic ileus, hydroallantois, ascites and ruptured bladder. There are other causes of abomasal influx like abomasal dilatation and torsion, abomasal displacement, hepatic abscess, lymphosarcoma of intestine, these need to be over-ruled on the basis of history, clinical signs, rectal temperature, laboaratory investigations and/or laparotomy findings [13]. Diaphragmatic hernia in buffaloes may also produce a similar picture but can be easily ruled out on radiographic examination or laparotomy. Ascites, rupture bladder, hydroallantois can be overruled by rectal examination, as in functional disorders of stomach, the distension will be confined to the rumen only. Ileus of large bowl like caecal dilatation can be diagnosed by rectal examination and on the basis of pings in right paralumbar fossa. However, various forms of ileus in the duodenum and proximal jejunum are more difficult to diagnose, as these may not be detected on rectal examination. Duodenum or proximal jejunum obstruction due to phytobazoars and incarceration of the duodenum results in clinical signs similar to pyloric functional disorder, and abomasal reflux also occurs. Bile acid determination in rumen fluid may be used to differentiate pyloric disorder from disease of proximal duodenum. Increased bile acid concentration in rumen fluid is observed in duodenal obstruction, provide the blockade is located caudal to papilla duodenum. However, rumen fluid of some animals with pyloric disorder may also show high concentration of bile acids [12].

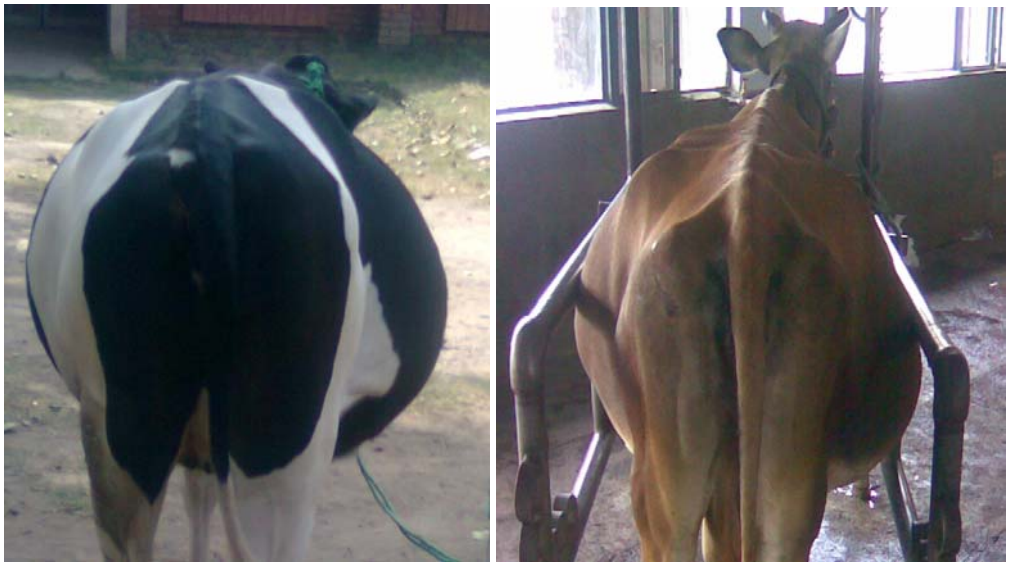
Treatment

Earlier, the treatment adopted in cases of functional disorders of the stomach included evacuation of rumen contents through a stomach tube or rumenotomy. This treatment used to be ineffective and very few cases used to recover [1]. Afterwards, treatment was changed to left paralumbar exploratory laprorumenotomy to palpate the reticular adhesions or abscess. This was followed by drainage of abscess, if any, and administration of fluids, broad spectrum antibiotics, calcium, analgesics and ruminal transfaunation [16]. Now-a-days, treatment is mainly aimed at correction of hypokalemic hypochloremic metabolic alkalosis and control of inflammatory and infectious processes. Any intra-abdominal abscess etc, of course, needs drainage. Corrections of electrolyte disturbances require large amounts of solution containing chloride and potassium. A course of antibiotics is added to check infection and unlocated inflammatory process [6, 10, 12]. Therapy with sodium chloride and potassium chloride solutions normalises the chloride and potassium deficit and acid-base disorders almost completely within 24 hours. Rumenotomy and evacuation of contents may not be necessary [12]. The low serum chloride and potassium concentrations cause increasing functional disorder from which animal cannot recover without external assistance, therefore administration of sodium chloride and potassium chloride and glucose is important. To correct electrolyte imbalances, large quantities of the fluid, rich in chloride ion are not only essential to correct hypochloraemia, but also to correct hypokalemia [46]. Hypokalemic alkalosis does not get corrected unless sufficient chloride ions are infused into the body [47]. In buffaloes, administration of 2.7% saline solution, in order to reduce the volume of solution to be infused, have given encouraging results [13, 48]. In conclusion, biochemical changes should be taken into consideration while dealing with gastrointestinal disorders in cattle and buffaloes [44].

In a recent study [6], treatment of 12 cases of LPI is reported. Treatment included intravenous administration of 10 liters of normal saline for 3-5 days, one-dose calcium therapy (450ml of MIFEX, Novartis India Limited, India), 200ml of liquid POTKLOR (containing 3 g of potassium chloride for 3 days), and liver tonic (8-10ml of injection Livadex, Virbac Animal Health, India) and 100 g charcoal (as antibloat agent) on daily basis. Ampicillin (22mg/Kg body weight) and enrofloxacin (7.5mg/Kg BW) twice daily were administered to the animals with inflammatory leukogram, for 5 days. This study concluded that LPI cases could be managed medically, till parturition, in order to reduce the losses due to calf mortality, which may occur by induction of parturition. The prognosis of the disease was reported to be good and recurrence rate was very low.

Necropsy Findings

Post-mortem examination of the clinical cases usually reveals distension of the rumen with liquid or semi-liquid contents along with reticular adhesions especially in sequels of TRP. In cases of omasal transport failure, the peritonitis, abscess and adhesions of the reticulum with diaphragmhave been recorded as the possible causes [1,16,49]. The vagal nerve damage at post mortem examination is often very difficult to demonstrate because of other lesions in that region [1]. The necropsy findings of LPI include distended rumen and reticulum with frothy contents, compression of diaphragm and abomasum cranially, and collapsed intestines [6].



(A) (B)

Fig 1a, b: Different degrees of Papple shaped abdomen in cattle suffering from functional gastrointestinal disorders

Fig 2: Undigested feed particle in the faeces of a cow suffering from omasal transport failure due to traumatic reticuloperitonitis

Fig 3: Pasty rumen contents recovered during laparo-rumenotomy from a buffalo suffering from diaphragmatic hernia. These types of contents give mushy consistency to rumen

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