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CLINICAL AND LABORATORY INVESTIGATIONS ON VAGUS INDIGESTION IN COWS & BUFFALOES

(With 3 Table)

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فحوص اكلينيكية ومعملية على عسر الهضم الناشئ عن وخز العصب الحائر في الأبقار والجاموس

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تم تشخيص عسر الهضم الناشئ عن وخز العصب الحائر في سبع بقرات وخمس جاموسات وقد لوحظ أن هناك نوعين من الضيق الناتج عن هذه الظاهرة ـ الأول وهو الناتج عن ضيق الفتحة البابية بين المنفحة والاثنى عشر وقد تم تشخيصه في حالتين من الأبقار والثاني والناشئ عن ضيق الفتحة بين الشبكية واللفائفي وذلك في خمس بقرات وخمس جاموسات ـ وقد لوحظ وجود فقر الدم ونقص مستوى الحديد في كلا النوعين · بينما كان نقص الكلوريد والبوتاسيوم وزيادة مستوى اليوريا في الدم من أهم مميزات النوع الأول · كما أنه تم الإستيضاح الجراحي لهذه الظاهرة في ستة بقرات وأربع جاموسات ·

SUMMARY

Vagus indigestion was diagnosed in seven cows and five buffaloes. Two types of stenosis were distinguished. Pyloric stenosis in two cows and reticulo-omasal stenosis in five cows and five buffaloes, were recorded. Differentiation between the two forms was explained. Anaemia and decreased serum iron were reported in the two forms of vagus indigestion, while hypochloremia, hypokalemia and uraemia were characteristics of pyloric stenosis. Surgical exploration was conducted on six cows and one buffaloe.

INTRODUCTION

The clinical entity known as vagus indigestion was first recognized in cattle by HOFLUND (1940) who sectioned different branches of the vagus nerve and described the resulting symptoms. The author stated that spontaneous cases of indigestion were

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the result of injuries to the vagus nerve caused by various inflammatory processes.

REBHUN (1980) and GARRY (1990) reported that, cattle with vagus indigestion have a functional outflow problems from the forestomach. They added that, when the outflow problem is centered at the pyloric area, the abomasum as well as the rest of the forestomach would enlarge and become impacted, while enlargement and distention of the rumen and reticulum with a relatively unaffected omasum and abomasum will result if the outflow problem is due to empty of the rumen and reticulum.

LEEK (1969) and WHITLOCK and WINGLIELD (1980) attributed the basic problem in cases of vagus indigestion to a trauma or inflammation which damage the vagus nerve leading to a functional outflow problem. The authors added that traumatic reticuloperitonitis is the most common cause of vagus indigestion, meanwhile peritonitis due to perforating abomasal ulcers is another possible cause. KUIPER and BREUKINK (1986) mentioned that, in rare cases severe thoracic inflammation involving mediastinal area can damage the vagus nerve enough to cause vagus indigestion. On the other hand, NEAL and EDWARDS (1968) and FUBINI, et al. (1985) recorded that the vagal neuritis and injuries of the vagus nerve play a minor role in the development of vagus indigestion and suggested that mechanical obstruction may be the cause.

REBHUN (1980); FUBINI, et al. (1985) and KUIPER and BREUKINK (1986) studied the haematological and serum biochemical alterations in vagal indigestion in cattle. They revealed that hypochloremia, hypokalemia and anaemia were detected in cattle with chronic vagus indigestion.

MATERIAL and METHODS

Seven friesian cows and five buffaloes were presented to the clinical of the Faculty of the Vet. Med., Suez Canal University, from October 1990 to December 1991. The affected animals have a history of anorexia, abdominal distention and intermittent bloat. The abdominal distension was chronic and progressive over few weeks or even months. Medicinal therpay for tympany and indigestion was tried before admission. Compelete clinical examination was performed according to ROSENBERGER (1979). Atropine sulphate test was applied by injecting 30 mg atropine sulphate (s/c) and monitoring the heart rate every five minutes. 15% and more increase of heart rate after fifeteen minutes, indicate positive response in cases of vagus indigestion (DIRKSEN, 1983).

Blood and serum samples were obtained from each animal. Whole blood samples were used for determination of haemoglobin concentration (Hb); total erythrocytic count (T.E.C), haematocrit (P.C.V) and total leucocytic count (T.L.C), after (JAIN,

1986). Colorimetric determinations of serum total protein, albumin, urea, blood urea nitrogen (B.U.N) chloride, potassium and serum iron levels by using test kits*. Serum globulin was calculated by substracting serum albumin concentration from serum total protein value (JAIN, 1986).

A left flank exploratory laparotomy and ruminotomy (Andress technique) were performed in six cow and one buffaloes, after JENNINGS (1984).

RESULTS

Clinical examination:

A summary of the results of the clinical examination is shown in table (1).

On examination, the affected animals had a distention of left paralumbar fossa except in cases No. 10 & 11. Abdominal distention with similar distention of the right lower quadrant was obvious in all affected animals. The rate and strength of the ruminal contractions were decreased in all affected animals, except in cases No. 5, 10 & 11 in which ruminal stasis with splashing sounds were recorded. By rectal examination, the dorsal ruminal sac was distended with gases in all affected animals except in cases No. 10 & 11 where fluid or frothy ingesta were detected, while the ventral ruminal sac was found enlarged and protruded to the right lower abdomen and palpated as a firm sac.

Bradycardia ($\leq$ 45/minute) was recorded in four of the affected cows and four of the affected buffaloes. Tachycardia ($\geq$ 82/minute) was reported in cases No. 10 & 11, while normal heart rate was noticed in cases No. 5 & 6. Atropin sulphate test response was positive in buffaloes 1, 8, 9 & 12 and cows 2, 3, 4 & 7. In these cases the heart rate was increased to 15% or more after fifteen minutes of the injection. Cases No. 10 & 11 showed elevated respiration ($\leq$ 65/minute) and high body temperature (39.5-40.5°C).

Pale mucous membranes, anorexia and emaciation were evident in affected animals. Scanty, pasty faeces was noticed in affected animals.

Surgical exploration:

A left flank exploratory laboratomy and ruminotomy was perfromed. During ruminotomy, digesta were removed and complete exploration was conducted.

* Supplied by Bio-Merieux and Boehringer.

Firm adhesion was detected between the diaphragm and the reticulum in cases No. 2, 3 and 8. Moreover, small pieces of wire were found perforating the reticular wall.

Large abcesses were found between the reticulum and omasum in cases No. 4 & 6. In these cases the palpation of omasal canal revealed widly opened and easily distendible omasal orifice.

In cases No. 10 & 11, fibrous ingesta was found in the reticulum, omasum and abomasum. Intraruminal palpation of the abomasum revealed elongated, distended firm sac containing hard fibrous ingesta; the reticular contents were than completely removed. Small sharp serrated non perforating metal objects were detected on the reticular floor. Reticulo-omasal orifice was widely dilated and atonised.

Laboratory Findings:

The results of haematological investigation and serum biochemical analysis were shown in tables (2 & 3).

DISCUSSION

The history of incurable abdominal distention with bloat, and clinical examination findings confirmed by surgical exploration and laboratory investigations, were all consistent of vagus indigestion syndrome.

Anterior functional stenosis (Reticulo-omasal stenosis) was diagnosed in 5 cows and 5 buffaloes. Posterior functional stenosis (pyloric stenosis) was evidenced in two cows.

In the two types of functional stenosis, a history of anorexia, abdominal distention, firm ruminal contents and pasty, scanty faeces, was reported. In pyloric stenosis, there were no free gas accumulation in the dorsal ruminal sac. Splashy, frothy ingesta were detected with depressed left paralumber fossa. The balloted rumen revealed fluid ingesta. In reticulo-omasal stenosis, there was gas accumulation which distend the dorsal ruminal sac, and rumen contents are palpated as firm mass. KUIPER and BREUKINK (1986) stated that in vagal indigestion, obstruction of ingesta flow results in accumulation of saliva and abomasal secretions in the forestomach which explains the fluid or splashy consistency when the rumen is balloted.

Bradycardia was noticed in reticulo-omasal stenosis in five cows and four buffaloes, while tachycardia was found in 2 cows with pyloric stenosis and one buffaloe with reticulo-omasal stenosis. GARRY (1990) attributed bradycardia to a reflex vagotonia to the heart and the tachycardia to the fluid and electrolyte imbalances. REBHUN

(1980) stated that bradycardia was observed as a classic sign of vagal indigestion in cattle. The auther added that, in severely advanced cases, a normal or elevated heart rates were noticed.

Surgical exploration indicated that the primary cause of the vagus indigestion was traumatic injury to the venteral branch of vagus nerve. NEAL and EDWARDS (1968) stated that adhesions involving the medial surface of the reticulum are most likely to be related to vagus indigestion. Furthermore. HABEL (1956) reported that the location of the traumatic lesions in vagus indigestion correlated well with the anatomic path of the venteral branch of the vagus nerve. WHITLOCK and WINGFIELD (1980) stated that the widely dilated omasal canal, bradycardia and irrigular ruminal contractions were indicative for vagal nerve dysfunction. On the other hand, LEEK (1969), KUIPER & BREUKINK (1986) and GARRY (1990) explained the vagus indigestion syndrome on the basis of dysfunction of the low threshold reticular tension receptors. They added that, traumatic reticuloperitonitis with pain, adhesion and induration as well as severe distention of the forestomach or abomasum, will abolish the normal tension receptor activity leading to an ineffective motor response to motor impulses rather than to vagus nerve damage.

Ancillary diagnostic aids including haematological picture and serum biochemical analysis (Table 2 & 3) revealed a decrease in the values of the T.E.C. (3.45-5.9x 10^6/ul); Hb. concentration (6.6-10.5 gm%); P.C.V. (21-29%) and serum iron level (39.8-110.8 ug%). Leucocytosis (13-15x10^3/ul) was noticed in some affected animals while leucopenea (4.0-5.2x10^3/ul) was reported in other individuals. These previous findings were indicative of inflammatory processes and infection. BEISEL (1976) and JAIN (1986) attributed the anaemia of inflammation to the sequestration of iron into bone marrow macrophages and hepatocytes, thus become unavailable for utilization in haemoglobin synthesis and resulting in inhibition of erythropiosis.

Regarding serum biochemical analysis, it was noticed that in cases suffering from pyloric stenosis, a hypochloramia, hypokalamia, uraemia and hyperglobulinaemia were found while in cases of reticulo-omasal stenosis, there were no abnormal alterations in the values of these respective parameters. WHITLOCK and WINGFIELD (1980); KUIPER and BREUKINK (1986) stated that hypochloramia may arise due to abomasal reflux from an abomasal impaction or stasis of the forestomach, while hypokalaemia results from anorexia and decreased intake of forage rich in potassium. GARRY (1990) attributed the elevated serum urea & blood urea nitrogen (B.U.N) to the dehydration and hypovolaemia in cases of pyloric stenosis. Elevated serum globulin in all affected animals might be a reflection of chronic antigenic stimulation and hypergamaglobulinaemia, as postulated by WHITLOCK and WINGFIELD (1980) and GARRY (1990).

It could be concluded that vagus indigestion was reported in cows and buffaloes suffering from incurable progresive abdominal distention. It is worthly to mention that

two forms of vagus indigestion were identified, reticulo-omasal stenosis and pyloric stenosis. Free gas accumulation in dorsal ruminal sac and firm ruminal contents were characteristics of reticulo-omasal stenosis while depressed left paralumber fossa and frothy to splashy ruminal contents was found in pyloric stenosis. Hypochloremia, hypokalaemia and uraemia were the most significant biochemical alterations in pyloric stenosis.

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Table (1) : Summary of clinical findings of affected animals.

Case No.	Species	Age/ years	Rectal Temp. (°C)	Heart rate /minute	Ruminal mortility	Atropin test response	Respiration /minute
1	Buffalo	4	38.6	45 (B)	hypomotility	+	23
2	Cow	6	39.0	42 (B)	hypomotility	+	21
3	Cow	6	38.7	40 (B)	hypomotility	+	28
4	Cow	5	39.4	42 (B)	hypomotility	+	30
5	Buffalo	7	39.6	68	stasis	-	67
6	Cow	5	38.5	58	hypomotility	-	32
7	Cow	4	37.5	45 (B)	hypomotility	+	28
8	Buffalo	8	38.0	40 (B)	hypomotility	+	27
9	Buffalo	6	38.6	42 (B)	hypomotility	+	22
10	Cow	5	39.8	95	stasis	-	70
11	Cow	7	40.5	82	stasis	-	68
12	Buffalo	2	39.2	45 (B)	hypomotility	+	14

B = Bradycardia.
+ = Positive (≥ 15% incease of heat rate)
- = Negative (No change or slight increase of heat rate, ≤ 5%)
hypomotility 1-2/2 m.

Table (2) : Haematological picture of affected animals.

Case No.	Species	T.E.C. X 10^6/ µl	Hb. gm %	P.C.V %	T.L.C. X 10^3 /µl
1	Buffalo	5.4	9.2	25	4.8*
2	Cow	5.0	10.0	23	4.0*
3	Cow	4.5	8.3	29	4.0**
4	Cow	4.8	8.5	23	13.6*
5	Buffalo	5.9	8.5	24	4.0**
6	Cow	3.45	6.6	22	14.2**
7	Cow	4.6	7.6	23	13.6
8	Buffalo	3.9	6.5	25	10.2**
9	Buffalo	4.8	7.8	22	13.0**
10	Cow	4.9	8.0	21	15.0**
11	Cow	5.7	10.5	26	14.3*
12	Buffalo	4.8	9.0	22	5.2

* Leukopnea ** Leucocytosis

Table (3) : Serum biochemical analysis of affected animals.

Case No.	Species	T.P. gm%	Album gm%	Globuin gm%	Cl m.eq./L	K m.eq./L	Urea mg%	B.U.N mg%	Iron µg%
1	Buffalo	8.6	3.9	4.7	100.0	3.5	25.1	11.4	110.0
2	Cow	7.3	2.4	4.9	96	4.2	30.2	14.5	100.0
3	Cow	9.7	2.9	5.8	102.3	4.0	26.8	12.7	95.0
4	Cow	9.6	4.2	5.4	95	3.8	28.6	12.4	110.8
5	Buffalo	10.5	4.1	6.4	85	2.9	50.3	22.3	39.8
6	Cow	9.4	4.6	4.8	97	4.2	45.6	21.0	85.2
7	Cow	8.9	4.4	4.5	92	5.1	49.2	23.7	96.4
8	Buffalo	9.2	4.5	4.7	98	5.3	62.3	26.6	90.0
9	Buffalo	8.5	3.5	5.0	103.6	5.2	78.8	35.4	82.0
10	Cow	8.2	2.1	5.9	58.8	2.4	158.3	62.7	58.0
11	Cow	10.7	4.4	6.3	62.7	2.2	198.2	75.3	72.0
12	Buffalo	8.4	3.8	5.0	89.3	3.8	75.8	32.8	92.0