VAGAL INDIGESTION IN A BUFFALO DUE TO OBSTRUCTION OF CARDIA BY A CLOTH

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ABSTRACT

This short communication describes the clinical signs, haemato-biochemical alterations and successful management of cloth induced vagal indigestion in a water buffalo.

Keywords: buffalo, Bubalus bubalis, cloth, vagal indigestion

INTRODUCTION

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 is 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 various causes of vagal indigestion reported by various scientists include foreign bodies (Braun et al., 1990a), advanced pregnancy (Van Metre et al., 1995), phytobezoar (Nayak and Suresh Babu, 1996), fibropapilloma (Gordon, 1997), liver hemangioma (Curtis and De Groot, 1967), reticular abscess (Fubini et al., 1989), right volvulus of abomasums, diffuse peritonitis (Rebhun et al., 1988), abomasal impaction (Radostitis et al., 2007), liver abscess, omasal impaction, pericarditis and idiopathic causes (El-Sabaie et al., 1997). The clinical symptoms in cattle and buffaloes are anorexia, dehydration, depression, muscular weakness and indifference to normal stimuli, dropping of ears, sunken eyes, dry muzzle, distended abdomen and cold extremities (Behl et al., 1997; Radostits et al., 2007).

HISTORY AND CLINICAL OBSERVATIONS

An adult 8-year-old buffalo was admitted to the Teaching Veterinary Clinics of Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, Punjab, with a history of abdominal distension, passage of scanty faeces, gradual reduction in milk yield and normal water intake from last 6 days. Clinical examination revealed severe abdominal distension in the form of “papple shape” (Figure 1). The rumen was atonic and fluid splashing sounds were audible on ballotment of the left flank. Pain test (pinching of wither) evidenced a negative response to pain (in the form of ventroflexion of spine). On per rectal examination scanty pasty

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faeces were present in the rectum and the dorsal sac of the rumen was filled with fluids and gas giving it a mushy consistency. There was no evidence of intestinal obstruction or abomasal displacement. The rumen was L shaped. The respiration rate (36/minutes) and heart rate (91/minutes) were elevated but rectal temperature (101.2°F) was within normal reference limits. The eyes were sunken and skin elasticity was lost indicating signs of dehydration. Lateral radiograph of reticular area revealed no apparent abnormality. Abdominal ultrasonography revealed no abnormality except absence of reticular motility.

LABORATORY FINDINGS

Haematological analysis revealed relative neutrophelia (total leukocyte count, 11350/μl, neutrophils, 70% and lymphocytes, 30%) with mild left shift, increased PCV (48%). There were no significant changes in BUN, creatinine, sodium, calcium and serum proteins. Blood acid-base gas analysis revealed hypochloremic hypokalaemic metabolic alkalosis (Table 1). Rumen fluid analysis revealed normal pH (~7) and increased chloride concentration (38 mEq/l), indicating abomasal reflux.

DISCUSSION

The case was diagnosed as vagal indigestion especially from papple shaped abdomen, hypochloremic hypokalaemic metabolic alkalosis and abomasal reflux. Vagus indigestion was characterized clinically by gradual distension of the left paralumbar fossa and bilateral distension of the ventral aspects of the abdomen due to accumulation of ingesta in the rumen, decreased appetite, scant faces, acid-base imbalance and dehydration, as reported earlier (Rebhun et al., 1988; Radostits et al., 2007). As per Behl et al., (1997), papple shape of the abdomen in buffaloes may be observed only in few cases but in the present case it was prominent. Fluid splashing sound on ballottement of the left flank indicated the presence of massive fluids in the upper sac of the rumen. Failure of fluids to move beyond the abomasum into the small intestine for absorption could be the possible cause for varying degrees of dehydration (Radostits et al., 2007).

The haematological alterations were
Figure 1. (A & B): Cloth recovered on rumenotomy.
similar to those previously reported (Behl et al., 1997). The altered liver function may be ascribable to chronic anorexia (Kaneko et al., 1997). The continuous secretion of potassium, hydrogen and chloride ions into abomasum could have resulted in alkalosis, hypokalemia and hypochloremia (Taguchi, 1995). Although alkalosis is mostly reported in pyloric stenosis or posterior functional disorders but inhibition of gastrointestinal motility due to any reason can lead to abomasal reflux and leads to development of metabolic alkalosis (Kuiper and Breukink, 1986; Radostits et al., 2007). The complex patho-physiology of functional disorders results in hypovolemia, disturbed electrolyte balance and consequent changes in acid base balance (Garry, 2002). The increased lactate and BUN concentration may be attributed to hypervolemia dehydration (Kaneko et al., 1997; Allen and Holm, 2008).

REFERENCES


