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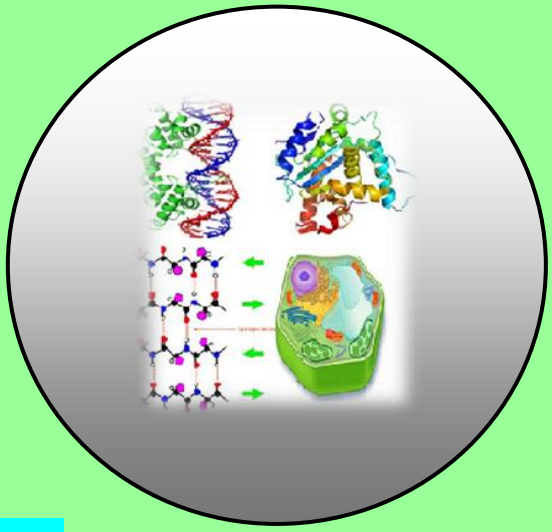

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RESEARCH PAPER

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Incidence of Enterotoxaemia in Goat at Government Goat Development Farm, Sylhet, Bangladesh

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ABSTRACT

A retrospective study of clinical records in the government goat development farm, Sylhet was carried out to investigate the clinical signs, postmortem lesions and the occurrence of naturally occurring enterotoxaemia in Black Bengal goats over the period of five and half years (January, 2010 – May, 2015).Younger animals (near to adults) (57.37%) were found more prone to the disease and it was likely to be more prevalent during Rainy season (62.30%) followed by at winter (21.31%) and summer (16.39%). Diarrhoea (90.16%), dullness and depression (80.32%), anorexia and kid's crying (73.77%), abdominal pain (65.57%) were recorded as major clinical signs whereas haemorrhagic enteritis (100%), Blood stained contents in intestine, serous fluid in peritoneal cavity and lung edema (95.08%), congestion and ulceration in mucosa and serosa (90.16%), distended rumen (80.32%) etc. were most common post mortem lesions found. A few cases showed haemorrhagic lesions on heart (40.98%), and/or liver/spleen (32.78%), brain edema (40.98%). Intestinal mucosa was subjected to Gram's staining to identify the causal agents. Based on the morphological characteristics, the causal agent was identified as Clostridium perfringens.
Keywords: Enterotoxaemia, Incidence, Black Bengal Goat, Clostridium and Sylhet.

INTRODUCTION

The goat called the "poor man's cows" is the second most important livestock in Bangladesh which provides 23% milk & 27% meat production in the country amounting to 450 thousands metric tones milk 105 thousand metric tones meat respectively (Kamaruddin, 2003). Although goat rearing is easy, less expensive, less laborious and highly profitable business. It is being seriously hampered due to variable disease problems. Enterotoxaemia

caused by *Clostridium perfringes* D is a disease of great economical and sanitary importance for sheep and goat farming worldwide (Niilo, 1980 and Kriek *et al.*, 1994), and is probably the most important cause of sudden death in goats of different ages (Blackwell and Butler, 1992). Most developing countries in the world such as Bangladesh require improvement to the productivity of their livestock sector or increased productivity of small ruminants, these conditions is most important in domesticated animals in the country, is restricted by various factors. Infectious bacterial disease is one of the most important factors that hinder the growth and productivity of small animal industry. Among the bacterial diseases, enterotoxaemia causes significant loss to sheep and goat industry. Caprine enterotoxaemia occurs worldwide in goat of any ages over 2 weeks (Baxendell, 1988) including Bangladesh (Shamimumzzaman, 1999). In Bangladesh, some confound reports are available from Rahman *et al.* (1968), Dewan *et al*.(1984) , Rahman (1997), Ehsan (2000) and Islam (2001) who mainly worked on diarrheic diseases but detailed reports are not available for caprine enterotoxaemia which constitutes a great threat to the successful goat production and its industry.

C. perfringes produces disease in sheep and goat, most of which are generally called enterotoxaemias. This microorganism is classified into five types (A, B, C, D and E) according to the production of four major toxins, namely alpha, beta, epsilon and iota (Niilo, 1980; Hatheway, 1990; Vaikosen and Ikhatua, 2005). *C. perfringens* can be a normal inhabitant of the intestine of most animal species (Niilo, 1980) including human (Johnson and Gerding, 1997) but when the intestine is altered by sudden changes in diet or other factors, *C. perfringens* proliferates in large number and produces several potent toxins (Uzal, 2004). History, clinical signs and gross postmortem findings are useful tools in establishing a presumptive diagnosis of enterotoxaemia by *C. perfringens* in sheep and goats (Uzal, 2004), although no definitive diagnosis of the disease can be made without laboratory confirmation.

In this study, an attempt has been made to boost up the present knowledge regarding the disease by compiling the clinical and postmortem signs of naturally occurring goat enterotoxaemia together with the occurrence in relation to age, season and causal agents and their economic impact.

MATERIAL AND METHODS

Collection of history, clinical signs and postmortem lesions

Sixty one goats died from January 2010 to December 2015 with signs associated with enterotoxaemia in government goat development farm, Sylhet, Bangladesh made the materials for the study. The history and clinical signs showed by the goats before death were recorded carefully from the register book of government goat development farm, Sylhet. Dead animals were subjected to immediate through postmortem study in the Department of Parasitology, Sylhet Agricultural University and Pathology Laboratory, Shaheed Suhrawardy Medical College, (SSMC), Dhaka as par the standard procedure and lesions found in each organ were recorded with care in the register book.

Sample collection and preservation

Intestinal mucosa from different parts of intestine of dead animals were collected in sterile disposable falcon tube and immediately stored on ice in an air tight container and preserved at -20°C until use.

Identification of the causal agents

Gram's staining of the samples was carried out following the procedure described by Soulsby (2000) and Rahman (1995).

RESULTS AND DISCUSSION

The goats died with signs of enterotoxaemia were reared under semi intensive management system where they were maintained on concentrate maxture twice daily, green grass (mainly Napier) twice daily and water ad-libitum. Molasses were mixed to green grasses and given to the goat once daily. Among 61 goats died, 8 were reported to have an accidental access to a large amount of grain before the clinical signs appeared/death of the animals. This findings support the previous findings by Blackwell and Butler(1992), Uzal and Kelly (1996) and Lewis (2000) who also reported most cases of enterotoxaemia to occur soon (a few hours to few days) after sudden changes in diet, usually to diets rich in highly fermentable carbohydrates. In the present study, it was noticed that goat enterotoxaemia is likely to occur in younger goats (near to adults) (57.37%) were found more prone to the disease though enterotoxaemia is more susceptible to goat kids (Islam *et al.*, 2008). This findings support the previous findings of Baxendell (1988) and Uzal *et al.*, (1994). This might happen as younger animals posses lower immunity. In addition males are usually stronger and eventually more vigorous than female. So they can have more access to feed and consequently get affected. It is also found that high prevalence (62.30%) of the disease was recorded in Rainy season which is also concordance with Seifert (1992) who reported it to occur mainly at the beginning of the rainy season but disagreed with Uzal *et al.* (1994) and Islam (2001). In this study there was found 21.31% cases which was less then rainy season but more than summer (16.39%). Most of the outbreaks have been reported to occur in dairy goat under intensive or semi intensive condition, although some cases have been reported in angora under extensive grazing condition (Uzal *et al.*, 1994)). Individual sporadic outbreaks of enterotoxaemia with high morbidity have also been observed (Smith and Sherman, 1994).

Sudden death (19.67%) occurs only minutes after a kid's shows sign of central nervous system alteration. Prior to death enterotoxaemia suspected goats showed clinical signs of anorexia and (73.77%), dullness and depression (80.32%), diarrhea (90.16%), mixed with blood and microfibrinous clots, nervous sign with convulsion and head thrown straight over back (42.62%), distended abdomen (13.11%) and death with 30 hours. In most cases the affected animals showed signs of acute abdominal pain (65.57%) accompanied by violent bellowing i.e. kid's crying (73.77%). Figure 1 shows the intensity of clinical findings of goats died of suspected enterotoxaemia.

The clinical signs showed by enterotoxaemic goats resembled to those were reported by Baxendell (1988), Blackwell and Butler (1992), Parhi *et al* (1993), Smith and Sherman (1994) Uzal *et al* (1994), Uzal and Kelly (1996), Phukan *et al* (1997), and Uzal and Kelly (1998). Three forms of caprine enterotoxaemia have been described; per acute, acute and chronic. The per acute form typically affects young growing kids and, as in this case, causes death within hours. The rapid clinical course may or may not be associated with clinical sign, which may include severe pain, vocalization, recumbency, fever, fibrino hemorrhagic diarrhea and death.

Acute enterotoxaemia has less rapid clinical course and signs are less severe than those observed with the per acute form. However, if left untreated acute enterotoxaemia usually culminates in death with clinical signs like diarrhea, abdominal pain and discomfort and severe shock with cold extremities and convulsion. The diarrhea in this form may initially be yellow- green and pasty but rapidly become watery and mucoid with shred of bowl mucosa and blood. The chronic from has been recognized in adult goats and is associated with chronic intermittent diarrhea often containing blood and mucous, anorexia, wet loss and decreased milk production. Thus the cases in this study were considered as of peracute and acute natures. All the cases in this study terminated into dead within 30 hours of appearances of clinical signs.

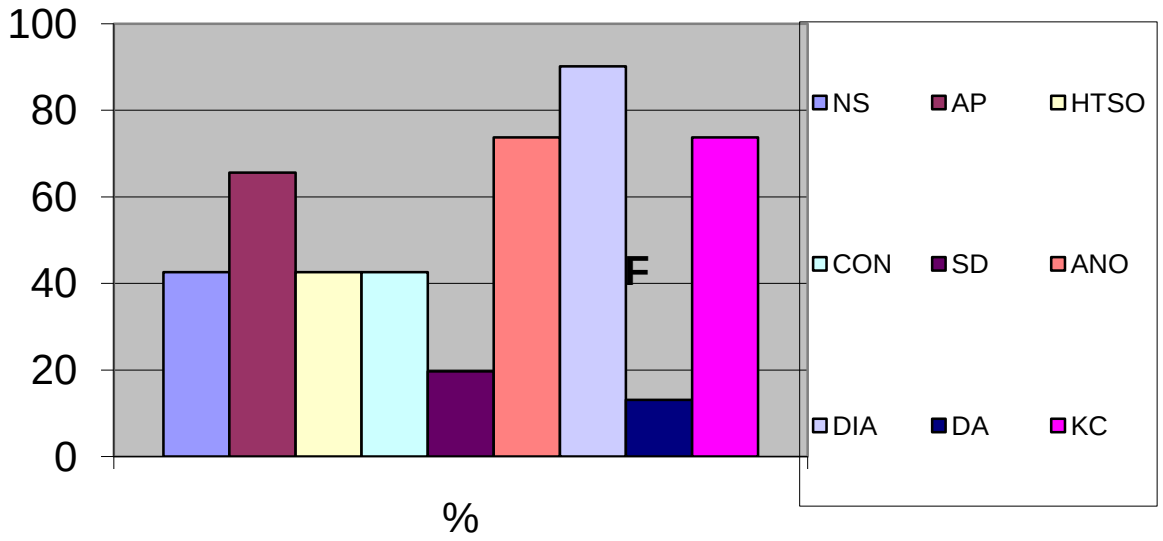

Figure 1. Clinical signs associated with goat enterotoxaemia.

NS= Nervous sign, AP= Abdominal pain, HTSO= Head thrown straight over back, CON= Convulsion, SD= Sudden death, ANO= Anorexia, DIA= Diarrhoea, DA= Distended abdomen, KC= Kid's crying

The most striking postmortem findings consisted of haemorrhagic and necrotizing enteritis (100%) together with adherent whitish pseudomembranes. The wall of the colon was thickened and a variable degree of edema was observed in the serosa of the colon and adjacent mesentry. The mesenteric lymph nodes were edematous, enlarged and congested. The most consistent gross lesion were found in the small and large intestine (95.08%), which were filled with watery content, blood and fibrin clots while gelatinous fluid was found in the abdominal cavity and peritoneal sac (95.08%). The ruminal mucous membrane was moderately congested (90.16%), as was the mucosa of the abomasums, where numerous small, rounded ulcers were evident. The rumen was also distended (80.32%) with gas and studded with feed particles.

Another predominant change was recorded in lungs (95.08%). The lungs were red, wet and heavy and slightly collapsed. Severe edema of the airways and interstitium of the lungs, together with abandoned froth in the trachea and bronchi were recorded.

Petechial hemorrhages of the endocardium and sub endocardial haemorrgahe around the mitral valve were also recorded in some cases (40.98%).

There was perivascular edema of the brain (40.98%), hemorrhagic lesion on the surface of liver and spleen were also noted in some cases (32.78%). Figure 2 describes the occurrence of the common postmortem lesions found in this study.

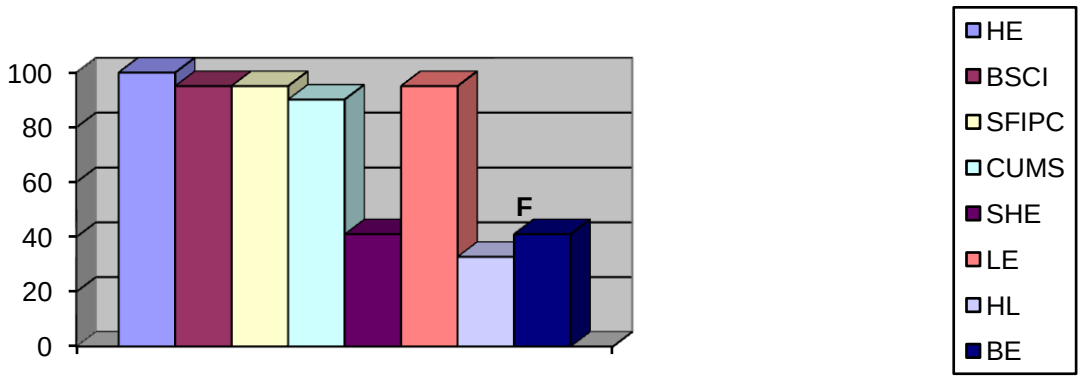

Figure 2. Major postmortem lesions found in enterotoxaemic goats.
HE= Hemorrhagic enteritis, BSCI= Blood stained contents in intestine, SFIPC= Serous fluid in peritoneal cavity, Congestion and ulcer in mucosa and serosa, SHE=Sub endo/epicardial hemorrhage, LE= Lung edema, HL= Hemorrhage in liver, BE= Brain edema

Postmortem lesions recorded in goats died in naturally occurring enterotoxaemia in this study also in accordance with the reports of earlier workers such as Papoff (1984), Baxendell (1988), Nillo (1988), Blackwell and Butler (1992), Timothy *et al* (1992), Barker *et al* (1993), Phukan *et al* (1997) and Prabhakar *et al* (1999). But no gross changes were observed in the kidneys of any of the goats. Thus the so called pulpy kidney lesion was absent which is in disagreement with Phukan *et al* (1997), Uzal and Kelly (1998) and Shamimuzzaman (1999) but showed similarity with the findings of Timothy *et al* (1992) who also reported pulpy kidney as not an important signs for enterotoxaemia in goats. This pulpy kidney lesion in earlier workers' findings might be due to advance autolysis of renal parenchyma or due to delay in postmortem observation.

Gram's staining of the specimens prepared from impression smears from different parts of the intestine revealed predominantly numerous short, thick, grams positive rods with blunt ends arranged in single, pair and in groups. In a smears prepared from infected tissues, the distinct capsule were observed surrounding the bacilli, which were arranged in single, pair and groups. Mixed type of bacteria but predominantly gram positive rods were also observed in the grams stained smears prepared from intestinal contents or faeces and the organism were surrounded by clear spaces that indicated the presence of capsules.

In this present experiment *C. perfringens* has been isolated and identified from both fecal samples and different parts of intestine of goats suspected to have died of enterotoxaemia. Enterotoxaemia in goats was reported in, Algeria, Argentina, Australia, Bangladesh, Canada, China, Great Britain, India, Iran, New Zealand, South Africa, Spain, Srilanka, Switzerland and in the United States. But no concise report is available regarding goat enterotoxaemia in Bangladesh although Shamimuzzaman (1999) attempted to isolate and identify *C. perfringens* from enterotoxaemic goats while reports on cattle enterotoxaemia in Bangladesh are available from Dewan *et al*. (1984) , Rahman (1997), Ehsan (2000) and Islam (2001).

The economic importance of the enterotoxaemia is due to the clinical disease and mortality in goats. An attempt was made to analyze economic losses due to only mortality caused by outbreaks of enterotoxaemia in Government goat development farm, Sylhet (Table 1).

Table 1. Economic losses caused by outbreaks (mortality) due to goat enterotoxaemia.

Year	Number of dead animal		Managemental Cost (MC) (feed, treatment others etc.) Tk.	Market Price (MP) Tk.	Loss (MP- MC) Tk.
	Adult	kids			
2010	6	2	16,000	20,000	16000
2011	9	1	18000	28,000	18000
2012	9	0	19,800	27,000	19,800
2013	11	0	22,500	38,000	22,000
2014	10	2	30,000	38,000	30,000
2015	10	1	33,000	36,000	33,000

It may be assumed that outbreaks of enterotoxaemia in government goat development farm, Sylhet is an important devastating disease which can accounting for severe economic losses in goat industry.

CONCLUSION

Conclusively, to the best of our knowledge present study is the first endeavor to study this deadly disease on goat of Government goat development farm, Sylhet. It shows clear pictures of goat enterotoxaemia in relation to clinical signs, postmortem lesions and the causal agents of occurrence. Although this study was carried out in a certain area, the knowledge must help to develop further strategy to help prevent and control the deadly disease of goat. But further study to identify the serotype and toxin type of the causal agent should be carried out throughout the country to get a comprehensible picture of the prevailing type of the organism so that more actions like vaccine or toxoid preparation could be performed.

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REFERENCES

Ardehali, M., Moosawi, M. and Pilehchian, R. 1996. Isolation of toxaegenic strains of Clostridium perfringes from the soil of farms in Iran. *Archiv Institute Razi* 44-45:95-100.

Barker, I.K., Van, Dreumel, A.A. and Palmer, N. 1993. The alimentary system in jubb KVF, Kenedy PC, *palmer N. Pathology domesticanimals* Vol. 2, 4 eds.

Baxendell, S.A. 1988. The diagnosis of the diseases of goats. The University of Sydney post graduate foundation in *Vrt Sci Sydney*.

Benaouda, K. 1985. Isolation and typing of Clostridium perfringes strains Algeria. *Maghreb Vet* 2(8):27-29.

Blackwell, T.E., Butler, D.G. and Prescott, J. 1991. Differences in signs and lesions in sheep and goats with enterotoxaemia induced byintra dudenal infusion of Clostridium perfringes type D.

Blackwell, T.E. and Butler, D.G. 1992. Clinical signs, treatment and postmortem lesions in dairy goats with enterotoxaemia.

Chakrabarti, K., Dutta, B.M. and Sharma, A.K. 1980. Enterotoxaemia in goats due to clostridium perfringes type, D. *Ind Vet J.*, 57: 195-197.

Dewen, M.L., Bari, A.S.M. and Rahman, M.M. 1984. Pathological investigation on an epidemic diarrheal disease in cattle of Bangladesh. *Bangl Vet J* 20: 5-9.

Dray, T. 2004. Clostridium perfringes type D type E α and β toxins associated with Enterotoxaemia in a 5 week old goat.

Ehsan, M.A. 2000. Study on clostridial enterotoxaemia in of Mymensingh. M.S (in Medicine). Thesis Bangladesh Agriculture University.

Guss, S.B. 1975. Dairy goat hard health problems. *Jam Vet Med Assoc,* 167 (12): 1076-1079.

Hatheway, C.L. 1990. Toxigenic clostridia. *Clin Microbial Rev* 3:66-98.

Hendersone, T.G. 1984. The detection of Clostridium perfringes type D enterotoxin in the intestinal contents of animals by counter immuno–electrophoresis. *New Zealand JSCI* 27:423-426.

Islam, K.S. 2001. Final report of the project. Control of clostridia enterotoxaemia of ruminant with experimentally prepared vaccine /Toxoid. BAURES, Mymensingh.

Johnson, S. and Gerding, D.N. 1997. The clostridia molecular biology and pathogenesis. San Diego, CA Academic press 117-140.

Kamaruddin, K.M. 2003. Goat farming as a means of poverty alleviation. Proceeding of the BSVER symposium, Goat farming in Bangladesh; problems and prospect. BAU. *Bangladesh society for veterinary education and Research*. BSVER 25; 26-35.

Lewis, C.J. 2000. Clostridial diseases in martin WB, Aitken ID, ed. Diseases of sheep. Oxford: Blackwell science.

Munoz, M. A., lvorez and M., Lanza, I. 1996. Role of enteric pathogens in the etiology of neonatal diarrhea's in lambs and goat kids in Spain. *Epizool Infect,* 117: 203-211.

Niilo, L. 1980. Clostridium perfringes in animal disease: a review of current knowledge. *Can Vet J.,* 21: 141-148.

Nillo, L. 1988. Hemolytic patterns for presumptive identification of Clostridium perfringes type C. *vet ref.,* 122:281.

Oxer, D.T. 1956. Enterotoxaemia in goats, *Aust Vet J*., 32:62-66.

Papoff, M.R. 1984. Bacteriological examination in enterotoxaemia of sheep and lamb Vet ref., 114: 324.

Parhi, N.K., Kar, B.C. and Rao, A.T. 1993. Enterotoxaemia in Orissa goats.

Phukan, A., Dutta, G.N. and Daube, G. 1997. Characterization of clostridium isolates from goats.

Prabhakar, T.G., Dorairajan, N. and Swaminathan, T.R. 1999. Report of caprine enterotoxaemia Tamilnaru Nadu. *Indian Vet J.,* 76: 81-83.

Rahman, A., Mia, A.S. and Ali, K.M. 1968. A preliminary report on an outbreak of haemorrhagic gastroenterities in cattle of unknown etiology. *Pak J Vet Sci.,* 2: 38-40.

Rahman, M.M. 1995. Bacteriology. Lumah Sakib Prokasoni Bangldesh Agricultural University Campus, Mymensingh.

Rahman, M.S. 1997. Investigation on an outbreak of enterotoxaemia in cattle of Mymensingh.MS (in Medicine). Thesis Bangladesh Agricultural University.

Rotz, A., C. Arbnozl, Waldvogel, A. and Von, R.A. 1984. Clostridium perfringes type D enterotoxaemia of goats in Switzerland. *Schweizer Archive Tierheilkunde,* 126: (7): 359-364.

Seifert, S.H. 1992. Trophentierhygiene, (Gustav Fischer Verlag, *Jena and Stuttgart*. 60-263.

Shamimumzzaman, M. 1999. Characterization of Clostridium perfringes isolated from goat and its antibiotic sensitivity. M.S. (in Microbiology). Thesis Bangladesh Agricultural University, Mymensingh.

Shanks, P.L. 1949. Enterotoxaemia in goats. *Vet Ref*., 61:262-264.

Smith, M.C. and Sherman, D.M. 1994. Goat medicine. Baltimore: Lippincott, Williams and Wilkins: 298-302.

Timothy, E., Blackwell, T.E. and Butler, D.G. 1992. Clinical signs, treatment and post mortem lesion in dairy goats with enterotoxaemia. (1979-1982). *Vet med Asso,* 200: 214-217.

Uzal, F.A. and Kelly, W.R., 1996. Enterotoxaemia in goats; A Review. *Vet Res Comm*. 29 (2) 481-492.

Uzal, F.A. and Kelly, W.R. 1998. Experimental C. perfringens Type D enterotoxaemia in goats.

Uzal, F.A., Pasini, M.I. and Olaechea, F.V. 1994. An outbreak of enterotoxaemia caused by clostridium perrfriengens type D in goats in Patagonia. *Vet Rec*., 135 (12); 79-80.

Uzal, F.A. 2004. Diagnosis of Clostridium perfringes intestinal infection in sheep and goats. *Anearobe* 10: 135-143.

Vaikosen, E.S. and Ikhatua, U.J. 2005. Detection of high level of enterotoxin of Clostridium perfringes type C and D in small ruminant in Nigeria. *Small Ruminant RS* 58: 287-290.

Van Tonder, E.A. 1975. Notes on some disease problem in angora goats in South Africa, *Vet Medical Rev.* 1-2: 109-138.

Wanasinghe, D.D. 1973. AN outbreak of enterotoxaemia due to Clostridium perfringes type D in goats. *Ceylon Vet J*. 21:62-65.

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