

Occurrence, clinical signs, postmortem lesions and etiology of enterotoxaemia in Black Bengal goats

K. B. M. Saiful Islam¹, Md. Siddiquir Rahman², Md. Ershaduzzaman³,
M. J. F. A. Taimur³, Hee-Jong Song^{4*}

¹Animal Health Research Division, Bangladesh Livestock Research Institute, Savar, Dhaka-1341, Bangladesh, and Research fellow, Lab. of Microbial Physiology, Graduate School of Agriculture, Hokkaido University, Sapporo-060-8589, Japan. ²Department of Medicine, Bangladesh Agricultural University, Mymensingh, ³Bangladesh Livestock Research Institute, Savar, Dhaka-1341, Bangladesh, ⁴College of Veterinary Medicine, Chonbuk National University, Jeonju 561-756, South Korea

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Abstract

A year round study was carried out to investigate the etiology, clinical signs, postmortem lesions and occurrence of naturally occurring enterotoxaemia in Black Bengal goats. Sixteen goats of different age and sex died in different seasons with sign associated with enterotoxaemia made the materials of this study. Accidental access to large amount of concentrate was noted as one of the predisposing factors although few cases were reported to occur without known diet change. Younger animals (50%) and males (62.50%) were found more prone to the disease and it was likely to be more prevalent during winter (50%) followed by at rainy season (31.25%) and summer (18.75%). Diarrhoea (81.25%), dullness (56.25%), drooping of the ears (50%), anorexia (43.75%) were recorded as major clinical signs whereas enterocolitis (100%), lung edema (87.50%), fluid filled intestines (87.50%), enlarged mesenteric lymph nodes (56.52%) etc. were most common post mortem lesions found. A few cases showed lesions on heart (31.25%), brain (25%) and/or liver/spleen (18.75%) but no lesion was found on kidney. Thus the so called 'pulpy kidney' lesion was absent. Intestinal contents were subjected to conventional bacteriological culture based methods to identify the causal agents. Based on the morphological, cultural and biochemical properties the causal agent was identified as *Clostridium perfringens*. Despite the study was carried out at certain area it showed a clear picture of goat enterotoxaemia in terms of etiology, clinical signs, postmortem lesions and occurrence of goat enterotoxaemia in Bangladesh.

Key words : Enterotoxaemia, Etiology, *C. perfringens*, Clinical signs, Postmortem lesions, Occurrence.

*Corresponding author :

Phone : +82-63-270-2562, Fax : +82-63-270-3780
E-mail : hjsong@chonbuk.ac.kr

Introduction

The goat called the "poor man's cow" is the second most important livestock in Bangladesh which provides 23% milk and 27% meat production in the country amounting to 450 thousands metric tons milk and 105 thousands metric tons meat respectively¹⁾. 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 perfringens*, is one of the most important diseases of small ruminants that causes a great threat to the sheep and goat industry all over the world. Most developing countries in the world such as Bangladesh require improvement to the productivity of their livestock ect or. Increased productivity of small ruminants, these condmost important 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 world wide in goats of any age over two weeks²⁾ including Bangladesh³⁾. In Bangladesh, some confound reports are available from Rahman et al⁴⁾, Dewan et al⁵⁾, Rahman⁶⁾, Ehsan⁷⁾ and Islam⁸⁾ who mainly worked on diarrhoeic diseases of cattle and revealed that *C. perfringens* as a causal agent of diarrhoeal diseases but detailed reports are not available for caprine enterotoxaemia which constitutes a great threat to the successful goat production and its industry.

C. perfringens produces disease in sheep

and goats, most of which are generically called enterotoxemias. 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^{9,10,11)}. *C. perfringens* can be a normal inhabitant of the intestines of most animal species⁹⁾ including humans¹²⁾ but when the intestine is altered by sudden changes in diet or other factors, *C. perfringens* proliferates in large numbers and produces several potent toxins¹³⁾. History, clinical signs and gross post-mortem findings are useful tools in establishing a presumptive diagnosis of enterotoxaemia by *C. perfringens* in sheep and goats¹³⁾, although no definitive diagnosis of the disease can be made without laboratory confirmation.

In this paper, an attempt has been made to boost up the present knowledge regarding the disease by compiling the clinical and post mortem signs of naturally occurring goat enterotoxaemia together with the prevalence in relation to age, sex and season and etiology.

Materials and Methods

Collection of history, clinical signs and Postmortem lesions

Sixteen goats died between November 2004 and November 2005 with signs associated with enterotoxaemia in Bangladesh Livestock Research Institute (BLRI) goat farm made the materials for the study. The history and clinical signs showed by the goats before death were recorded carefully in a preset format. Dead animals were subjected to immediate thorough post mortem study as par the standard procedure and lesions

found in each organ were recorded with care.

Sample collection and preservation

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

Identification of the causal agents

For the identification of causal agent, approximately 1 gm of each intestinal sample was taken in 10 ml of PBS in a screw-capped bottle and mixed vigorously using vortex to homogenize. The suspended large faecal particles were removed by filtering through sterile gauze filter. The filtrate was then left for a few minutes to allow the debris to sediment. A 0.1 ml of the supernatant was inoculated in duplicate on 5% sheep blood agar (Merck, Germany) and nutrient agar (Difco, USA) media and incubated anaerobically for 48 hours at 37°C using CO_2 incubator (Model No. MCO-15 AC, Sanyo Electric Co Ltd Japan). Predominant colonies showing typical double zone of hemolysis from each plate were subcultured on 5% sheep blood agar. Passages were repeated on blood agar plate until the cultures were considered pure. Further, the colonies with double zone of hemolysis were also cultured in cooked meat broth and brain heart infusion broth and incubated anaerobically at 37°C for 6 days and 3 days respectively to observe the nature of growth in broth media. Biochemical tests were performed with the isolated bacteria

according to the procedure described in Bergey's manual¹⁴. Fermentation reaction with basic sugars (sucrose, maltose, lactose, dextrose, and mannitol) were carried out by taking 5 ml of each sugar solution into 5 sterile test tubes and inoculating with the isolated organism. The tests were read after an overnight anaerobic incubation at 37°C . In addition, skimmed milk coagulation test was also performed with the isolated organism by inoculating freshly grown organism into 5 ml of skimmed milk (Difco, USA) in sterile test tube and incubating anaerobically at 37°C for 24 hours. Gram's staining of the samples was carried out following the procedure described by Rahman¹⁵.

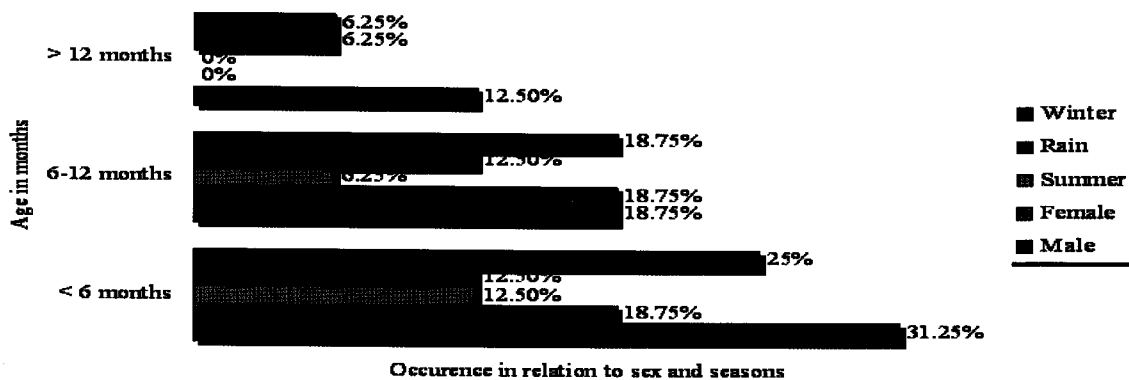
Results and Discussion

The goats died with signs of enterotoxaemia were reared under semi intensive management system where they were maintained on concentrate mixture twice daily, green grass (mainly Nepier and Para) twice daily and water *ad libitum*. Molasses were mixed to green grasses and given to the goat once daily. Among 16 goats died, 5 were reported to have an accidental access to a large amount of grain before the clinical signs appeared/death of animals. This finding support the previous findings by Blackwell and Butler¹⁶, Uzal and Kelly¹⁷ and Lewis¹⁸ who also reported most cases of enterotoxaemia to occur soon (a few hours to a few days) after sudden changes in diet, usually to diets rich in highly fermentable Carbohydrates. Three healthy kids in this study allowed for routine grazing on nearby pasture, returning from pasture and kept in their respective sheds were found dead in the next morning with distended abdomen.

This finding was in accordance with Van Tonder¹⁹⁾ and Uzal et al²⁰⁾ who reported some outbreaks of enterotoxaemia type D to occur in goat sunder extensive grazing systems

without known diet change.

The occurrence of goat enterotoxaemia in relation to age, sex and season of 16 goats died is shown in Fig 1.



*Summer: Feb - May; Rainy: June - Sept; Winter: Oct - Jan

Fig 1. The occurrence of goat enterotoxaemia in relation to age, sex and seasons

In the present study, it was noticed that goat enterotoxaemia is likely to occur in goat kids less than 6 months of age (50%) and male animals are more prone (62.5%) to the disease. This findings support the previous findings of Baxendell²⁾ and Uzal et al²⁰⁾. This might happen as younger animals possess 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 (50%) of the disease was recorded in winter which is also in accordance with Uzal et al²⁰⁾ and Islam⁸⁾ but disagreed the result of Seifert²¹⁾ who reported it to occur mainly at the beginning of rainy season. In our study we found 31.25% cases occurred in rainy season which was less than winter but more than summer. Most of the outbreaks have been reported to occur in dairy goat sunder intensive or semi-intensive condition, although some cases have been reported in angora goats under extensive

grazing condition²⁰⁾. Individual sporadic outbreaks of enterotoxaemia with high morbidity have also been observed²²⁾.

Prior to death enterotoxaemia suspected goats showed clinical signs of anorexia (43.75%), dullness (56.25%), watery diarrhoea (81.25%) mixed with blood and mucofibrinous clots, opisthotonus (31.25%), convulsions (37.5%), retraction of the head (37.5%), tetany (37.5%), drooping of the ears (50%), distended abdomen (18.75%), respiratory distress (37.5%) and death within 30 hours. In some cases, the affected animals showed signs of acute abdominal pain accompanied by violent belching and agonal struggling (12.5%). Fig 2 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 Baxendell²⁾, Blackwell and Butler¹⁶⁾, Parhi et al²³⁾, Smith and Sherman²²⁾, Uzal et al²⁰⁾, Uzal and Kelly¹⁷⁾, Phukan et al²⁴⁾, and Uzal and Kelly²⁵⁾. Three forms of caprine enterotoxaemia have been described;

peracute, acute and chronic^{16, 22, 26)}. The peracute form typically affects young growing kids and, as in this case, causes death within hours²⁶⁾. The rapid clinical course may or may not be associated with clinical signs, which may include severe pain, vocalization, recumbency, fever, fibrinohaemorrhagic diarrhoea and death^{22, 26, 27)}. Acute enterotoxaemia has less rapid clinical course and signs are less severe than those observed with the peracute form²⁶⁾. However, if left untreated acute enterotoxaemia usually culminates in death with clinical signs like diarrhoea, abdominal pain and discomfort and severe shock with cold extremities and convulsion^{16, 22, 26, 27)}. The diarrhoea in this form

may initially be yellow-green and pasty but rapidly become watery and mucoid with shred of bowel mucosa and blood²⁾. The chronic form has been recognized in adult goats^{16, 26)} and is associated with chronic intermittent diarrhoea often containing blood and mucus, anorexia, weight loss and decreased milk production^{16, 22, 26)}. Thus the cases in this study were considered as of peracute and acute natures. All the cases in this study terminated in to dead within 30 hours of the appearance of clinical signs.

The most striking postmortem findings consisted of hemorrhagic and necrotizing enterocolitis (100%) together with adherent

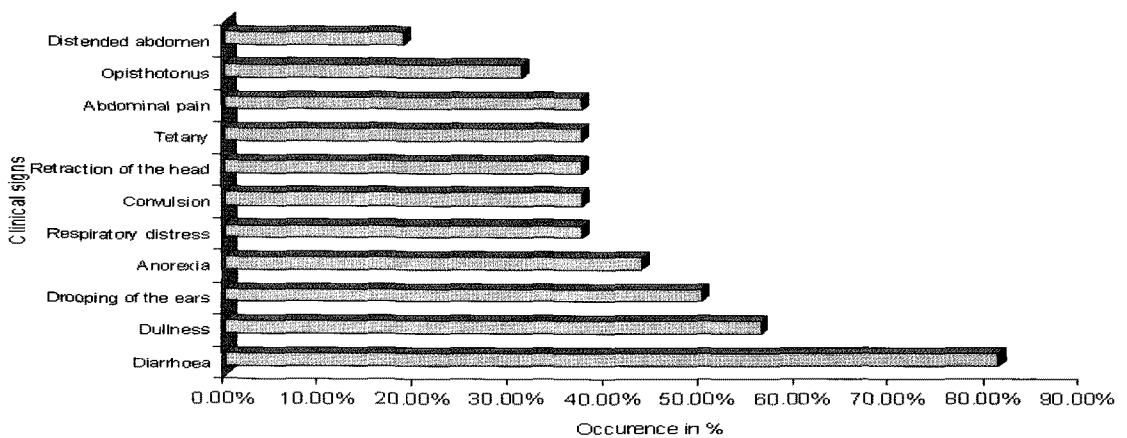
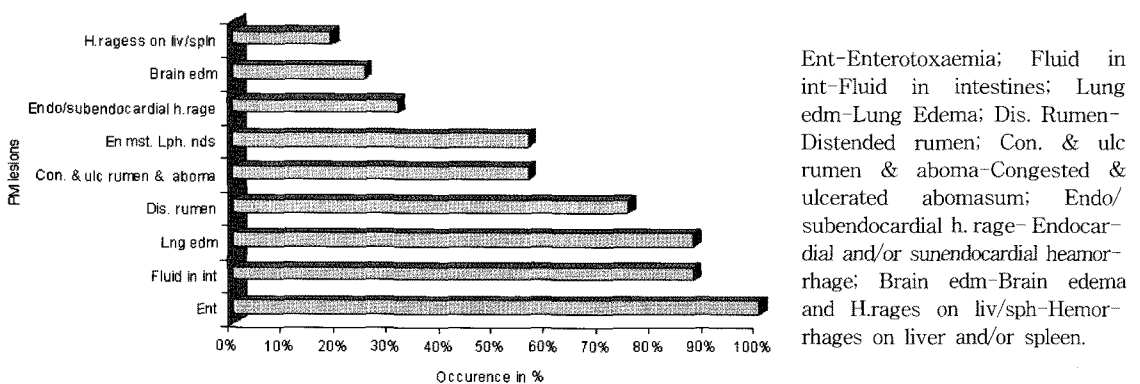


Fig 2. Clinical signs associated with goat enterotoxaemia



Ent-Enterotoxaemia; Fluid in int-Fluid in intestines; Lung edm-Lung Edema; Dis. Rumen-Distended rumen; Con. & ulc rumen & aboma-Congested & ulcerated abomasum; Endo/subendocardial h.rage-Endocardial and/or subendocardial hemorrhage; Brain edm-Brain edema and H.rages on liv/spln-Hemorrhages on liver and/or spleen.

Fig 3. Major post mortem lesions found in enterotoxaemic goats

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 mesentery. The mesenteric lymph nodes were edematous, enlarged and congested (56.52%). The most consistent gross lesions were found in the small and large intestines (87.5%), which were filled with watery contents, blood and fibrin clots while gelatinous fluid was found in the abdominal cavity and peritoneal sac. The ruminal mucus membrane was moderately congested (56.25%), as was the mucosa of the abomasum, where numerous small, rounded ulcers were evident. The rumen was also distended with gas and studded with feed particles.

Another predominant change was recorded in lungs (87.5%). The lungs were red, wet and heavy and slightly collapsed. Severe edema of the airways and interstitium of the lungs, together with abundant froth in the trachea and bronchi were recorded. Petechial hemorrhages of the endocardium and sub endocardial hemorrhages around the mitral valve were also recorded in some cases (31.25%). There was perivascular edema of brain (25%), hemorrhagic lesions on the surface of liver and spleen were also noted in some cases (18.75%). Fig 3 describes the occurrence of the common post mortem lesions found in this study.

Post mortem lesions recorded in goats died in naturally occurring enterotoxaemia in this study also in accordance with the reports of earlier workers such as Papoff²⁸⁾, Baxendell²⁾, Nillo²⁹⁾, Blackwell and Butler¹⁶⁾, Timothy et al³⁰⁾, Barker et al³¹⁾, Phukan et al²⁴⁾ and Prabhakar et al³²⁾. 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 al²⁴⁾, Uzal and Kelly²⁵⁾, Shamimuzzaman³⁾ and Phukan et al³³⁾ but showed similarity with the findings of Timothy et al³⁰⁾ 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, Gram's positive rods with blunt ends arranged in single, pair and in groups. In smears prepared from infected tissues, the distinct capsules were observed surrounding the bacilli, which were arranged in single, pair and groups. Mixed type of bacteria but predominantly Gram's positive rods were also observed in the Gram's stained smears prepared from intestinal contents/faeces and the organisms were surrounded by clear spaces that indicated the presence of capsules.

On sheep blood agar the colonies were circular, entire, smooth, dome shaped, gray to grayish yellow and glistening and were surrounded by a typical zone of hemolysis. Large number of short, thick, singly arranged, rod shaped Gram's positive bacteria were observed under the microscope in the smears prepared from the colonies grown on blood agar. On nutrient agar plate, the colonies were round, 2-5 mm in diameter, low convex, amorphous, opaque with smooth surface and entire edge. In cooked meat broth, the growth of the organism was indicated by pinkish coloration of the broth and huge quantities of gas production. The organism produced sediment and turbidity along with

gas production in brain heart infusion broth. The organism was non motile, indole and urase negative and reduced nitrate to nitrite. The isolated bacteria fermented dextrose, maltose, lactose and sucrose but failed to ferment mannitol. The fermentation reactions were indicated by yellowish discoloration of the medium. In skimmed milk coagulation test, there was formation of stormy clot with gas indicating the presence of *C. perfringens* (Table 1). All these cultural and biochemical characteristics force the organism to be identified as *C. perfringens* as previous workers reported^{3, 6-8, 34-37}.

Based on the characteristics mentioned in biochemical and sugar fermentation test, the organism was identified as *C. perfringens*. Since *C. perfringens* was first described as *Bacillus aerogenes capsulatus* in 1892³⁸, the bacterium has been identified as an anaerobe responsible for a widerange of disease in humans and animals³⁹. *C. perfringens* is

the causative agent of enterotoxaemia in several animal species⁹. Some types of *C. perfringens* (mainly type A) are consistently recovered both from the intestinal tracts of animals and from the environment while others (types B, C, D and E) are less common in the intestinal tracts of animals⁴⁰ and can occasionally be found in the environment in areas where diseases produced by these organisms is enzootic⁹.

In this present experiment, *C. perfringens* has been isolated and identified from both fecal samples and different parts of intestines of goats suspected to have died of enterotoxaemia. Enterotoxaemia in goats was reported in, Algeria⁴¹, Argentina²⁰, Australia⁴², Bangladesh³, Canada¹⁶, China⁴³, Great Britain⁴⁴, India⁴⁵, Iran⁴⁶, Newzeland⁴⁷, South Africa¹⁹, Spain⁴⁸, Srilanka⁴⁹, Switzerland⁵⁰ and in the United States⁵¹. But no concise report is available regarding goat enterotoxaemia in Bangladesh although Shamimuzzaman³ at-

Table 1. Identifying characteristics of the isolated organism

Gram's staining features	Staining	Positive
	Morphology	Rod
Cultural in media	Blood agar	circular, entire, smooth, dome shaped with hemolysis
	Nutrient agar	round, low convex, amorphous, opaque, smooth surfaced with entire edge
Biochemical features	Cooked meat broth	A+G
	Brain heart infusion broth	S+G
	Dextrose	+
	Maltose	+
	Lactose	+
	Sucrose	+
	Manitol	-
	Nitrate reduction test	Nitrate to Nitrite
	Urease	-
	Indole	-
	Skimmed milk coagulation test	A+G
Identified organism	<i>C. perfringens</i>	

tempted to isolate and identify *C. perfringens* from enterotoxaemic goats while reports on cattle enterotoxaemia in Bangladesh are available from Dewan et al⁵⁾, Rahman⁶⁾, Ehsan⁷⁾ and Islam⁸⁾.

Conclusively, to the best of our knowledge present study is the first endeavor to study this deadly disease of goat. It shows clear pictures of goat enterotoxaemia in relation to etiology, clinical signs, post-mortem lesions and 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 toxinotype 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/toxoid preparation could be performed.

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