Pakistan Veterinary Journal

ISSN: 0253-8318 (PRINT), 2074-7764 (ONLINE)
Accessible at: www.pvj.com.pk

SHORT COMMUNICATION

Clinico-Pathologic Findings of Enterotoxemia in Chinkara Deer (*Gazella bennettii*) Under Desert Conditions in Pakistan

Riaz Hussain[1]*, M Tariq Javed[2], Fazal Mahmood[2], Tanveer Hussain[3], Haroon Rashid Chaudhry[1], Muhammad Sohaib Aslam[2], Muhammad Tasleem Ghori[1], Abdul Qayyum[1],Wasim Babar[1], Sajid Hameed[1] and Aziz-Ur-Rehman[2]

[1]University College of Veterinary and Animal Sciences, The Islamia University of Bahawalpur; [2]Department of Veterinary Pathology, University of Agriculture, Faisalabad; [3]Department of Forestry and Wildlife, University College of Agriculture and environmental Sciences, The Islamia University of Bahawalpur, Pakistan
*Corresponding author: driazhussain@yahoo.com

ARTICLE HISTORY (12-324)

Received: August 29, 2012
Revised: October 22, 2013
Accepted: November 17, 2013

Key words:
Chinkara deer
Clostridium perfringens
Enterotoxaemia
Pathology

A B S T R A C T

This study was carried out to know clinico-pathologic findings of enterotoxaemia in Chinkara deer (*Gazella bennettii*). At one farm, 10(20%) out of 50 and at other 35(31.8%) out of 110 deer died. Out of 45 animals, peracute deaths were 66.67% whereas acute deaths were 33.33%. Significantly high mortality was recorded in young animals. The case fatality was 100%. Clinical signs including moderate to acute anorexia, opisthotonus, herding in a corner with head down, marked depression, fever, chocolate colored urine and greenish watery diarrhea were observed. Necropsy of dead deer revealed presence of straw colored fluid in peritoneal/abdominal cavity. Hydropericardium, hydrothorax, congested and edematous lungs, swollen and soft kidneys, watery contents in the small intestine, ballooning and hemorrhages of small and large intestine were the consistent lesions in affected animals. Multifocal petechial hemorrhages on myocardium and jejunal mucosa were frequently observed in peracute cases. The histologic changes were hemorrhagic enteritis, pulmonary edema, congestion, proteinaceous fluid in alveoli and perivascular cuffing in lungs. Increased level of glucose was recorded in urine collected from urinary bladder of 17 deer at necropsy. Histopathological sections of kidneys revealed congestion and necrosis along with disruption of renal tubular epithelial cells. The clinical signs, post-mortem lesions and histological findings in present study were consistent with those reported due to *C. perfringens* type D enterotoxaemia.

To Cite This Article: Hussain R, Javed MT, F Mahmood, T Hussain, HR Chaudhry, MS Aslam, MT Ghori, A Qayyum, W Babar, S Hameed and AU Rehman, 2014. Clinico-pathologic findings of enterotoxemia in Chinkara deer (*Gazella bennettii*) under desert conditions in Pakistan. Pak Vet J, 34(3): 400-402.

INTRODUCTION

Clostridium perfringens is gram-positive rod, anaerobic and a spore-forming pathogen of domestic and wild animals. Enterotoxaemia is one of the major endemic diseases of small ruminants in Pakistan. The disease mainly occurs in sheep and goats and rarely in large animals (Khan *et al.*, 2008; Tahir *et al*., 2013). Peracute disease frequently occurs in unvaccinated young and adult sheep and goats (Javed *et al.*, 2009; Nasir *et al*., 2013). Enterotoxaemia is caused by *C. perfringens* type D and has worldwide distribution. Peracute deaths occur due to four major toxins, namely epsilon, alpha, beta and iota produced by *C. perfringens* (Uzal and McClane 2011; Morris *et al.*, 2012). Death results from toxemia following absorption of toxin from the small intestine into circulation and thus reaches the internal organs where it causes damage (Ma *et al.*, 2011). Overeating of lush green fodder and grain, abrupt change in diet and anaerobic environment of small intestine plays vital role in the occurrence of disease (Javed *et al.*, 2009). The spores of *C. perfringens* type D are common in feces and soil. The *C. perfringens* are normal inhabitant of intestine, but are usually present in low numbers. Lethal infections frequently occur due to predisposing factors which permit rapid growth of this organism in gastrointestinal tract (Diab *et al.*, 2011). Peracute disease mostly occurs in young animals when the organisms multiply and produce toxin faster than can be eliminated or neutralized. The enterotoxaemia in sheep due to *C. perfringens* type D is

known as pulpy kidney disease. This infection is characterized by lesions including soft and swollen kidneys, severe haemorrhages in small and large intestine, hydropericardium, hydrothorax, serosal and muscular haemorrhages in heart and colon (Khan *et al.,* 2008; Javed *et al.,* 2009). History, clinical picture, gross lesions at necropsy and histopathological findings are valuable tools in establishing a presumptive diagnosis. Descriptions of gross pathological and microscopic changes in naturally occurring cases of *C. perfringens* type D enterotoxaemia in Chinkara deer *(Gazella bennettii)* are scant. The present study reveals the clinical picture, gross and histopathological findings in 45 Chinkara deer suspected of died due to enterotoxaemia.

MATERIALS AND METHODS

The present study was carried out during an outbreak of enterotoxaemia in Chinkara deer (*Gazella bennettii*) at two farms in district Bahawalpur, Punjab Province. The environmental temperature of that area varied from 41.3 to 42.5°C with 63.1% relative humidity. These farms are at a distance of 3 km from each other. At these farms the deer were not vaccinated against any disease. The lush green fodder Barseem (*Trifolium alexandrinum*) and fresh water was offered to these animals. The goats were also kept with deer at second farm. The goats had the history of diarrhea and enterotoxaemia. The attendants working at one farm also had sheep and goats at their homes.

The clinical signs in sick animals were moderate to acute anorexia, herding in a corner with head down, marked depression, fever, passing of chocolate colored urine and greenish watery diarrhea. The sick animals were treated orally with antibiotics Amoxicillin (10mg/kg BW), Tribersen (15mg/kg BW) and CTC-20 (10mg/kg BW). In addition the vitamin-mineral mixture (1 ml/lit in drinking water) was also given to sick deer. However, the treated animals did not show any improvement in signs and death occurred.

Necropsies were conducted within 1 hour after death and gross changes were observed. Morbid tissues from lungs, liver, kidneys and intestine were collected and fixed in 10% neutral buffered formalin for 15 days. Standard histopathological techniques were used for fixation, dehydration, infiltration and embedding. Four micron thick sections from fixed tissues were cut and stained with hematoxylin and eosin method. The data collected were subjected to Chi square analyses and $P \leq 0.05$ was considered as significance level. Odd ratios and 95% confidence intervals were also determined.

RESULTS AND DISCUSSION

In present study out of 45 animals, peracute death was recorded in 30 (66.7%), while acute death occurred in 15 (33.3%) deer. The peracute and acute cases of death due to this disease in sheep, goats and cattle calf have been reported (Javed *et al.,* 2009). In the present study the mortality rate ranged between 10 (20%) and 35 (33.3%) in two deer flocks. The results indicated a 2 times (OR=2.00) higher mortality in deer flock that had goats as companion animals. Results also showed that the mortality was significantly ($P<0.05$) higher in young animals as compared to old animals (Table 1). Higher mortality in young animals has already been reported in other animal (Javed *et al.,* 2009). The history suggested that the disease has strong correlation with the subtropical conditions. Under similar conditions, peracute and acute deaths have been reported previously in Chinkara deer (Khan *et al.,* 2008), cattle and sheep (Javed *et al.,* 2009).

Table 1: Overall mortality (%) recorded in deer died of enterotoxemia

Sex/age	No. of Animal	Mortality		95% CI	Odd Ratio/ P value
		No.	%		
Sex					
Female	109	27	24.8	17.4 to 33.5	OR = 0.51
Male	46	18	39.1	25.9 to 53.7	[reciprocal = 1.95]
Age groups					
< 1Year	64	25	39.1	29.6 to 49.7	Mantel-Haenszel
1-2 Year	55	12	21.8	13.6 to 33.5	chi-sq P<0.05
3 Years	36	8	22.2	1.2 to 10.1	

Table 2: Overall frequency of gross lesions observed in deer (n=45) died of enterotoxaemia

Gross lesions	Frequency	
	No	%
Hydrothorax	37	82.2
Hydroperitonium	39	86.7
Hydropericardium	36	80.0
Petechial hemorrhages on myocardium	41	91.1
Congested lungs	39	86.7
Pulmonary edema	40	88.8
Swollen and soft kidneys	43	95.6
Hemorrhages on kidneys	42	93.3
Watery contents in intestine	43	95.6
Hemorrhages on jejunal mucosa	39	86.7
Ballooning and hemorrhages of small intestine	41	91.1

In present study no clinical signs were observed in animals died due to peracute infection. However, in acute cases various clinical signs including moderate to acute anorexia, herding in a corner with head down, marked depression, fever, opisthotonus, passing of chocolate colored urine and greenish watery diarrhea were observed among all infected animals. Previously, similar clinical sings in acute cases of enterotoxaemia in deer, cattle, sheep and goats have been reported (Khan *et al.,* 2008; Popoff, 2011; Garcia *et al.,* 2012).

Postmortem changes (Table 2) observed in dead animals included excessive amount of proteinaceous straw colored fluid in epicardium, abdominal and thoracic cavities in peracute and acute cases. In Pakistan similar necropsy findings have been reported in deer (Khan *et al.,* 2008), sheep and goats (Javed *et al.,* 2009). Lungs were congested, edematous and consolidated in all animals showing respiratory signs. Kidneys of affected animals were soft and swollen, darker in color and appeared congested. Similar gross changes in lungs and kidneys of naturally and experimentally induced enterotoxaemia has been reported in deer (Khan *et al.,* 2008), in sheep and goats (Popoff, 2011). Petechial hemorrhages at the base of heart, myocardium and jejunal mucosa were observed during postmortem examination. Ballooning and hemorrhages of small and large intestine and greenish watery material were frequently observed in deer in acute cases. Gross lesions observed in present study are similar to previous cases of enterotoxaemia in cattle, deer and other small ruminants (Khan *et al.,* 2008; Popoff 2011).

Microscopically, lungs exhibited striking changes those were comprised of diffusely packed eosinophilic proteinaceous edema fluid and inflammatory material within alveoli, bronchioles and bronchi. Lung tissue

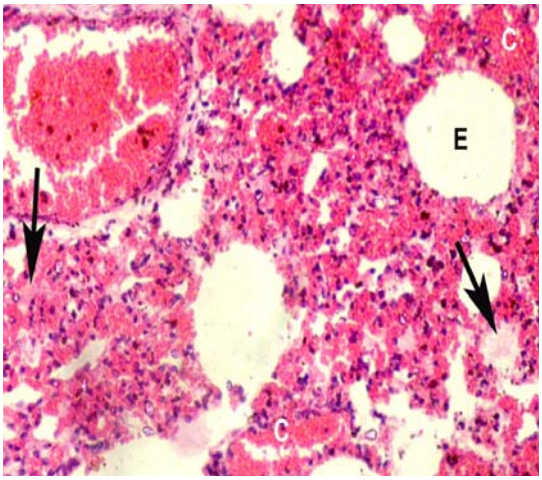

Fig. 1: Photomicrograph of lungs showing emphysema (E), severe congestion (C) and edematous fluid (arrows) in Chinkara Deer died of enterotoxaemia. (H&E, 200X).

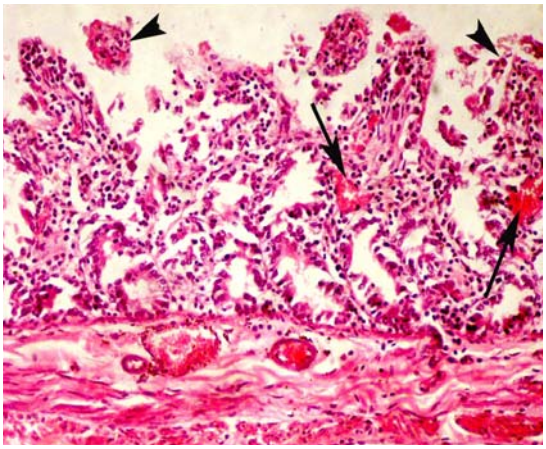

Fig. 2: Photomicrograph of intestine showing congestion (arrows), loss of villus epithelium (arrow heads) and pyknosis in Chinkara Deer died of enterotoxaemia. (H&E, 200X).

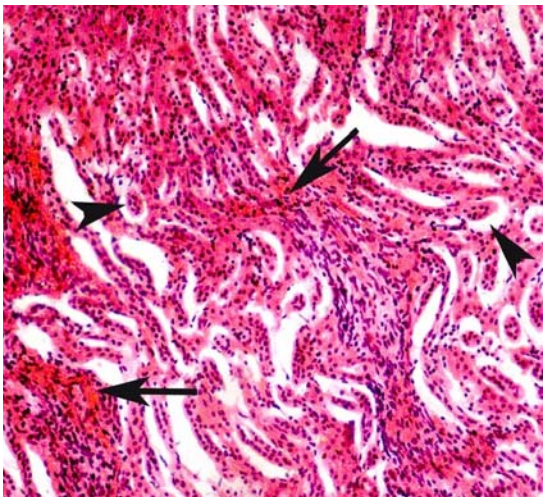

Fig. 3: Photomicrograph of kidney of a deer died of enterotoxaemia showing sloughing of tubular epithelium, severe congestion (arrows) and necrosis of renal tubules (arrow heads). (H&E, 200X).

revealed thick and extensively infiltrated bronchioles by inflammatory cells. There was congestion, extensive infiltration of leukocytes particularly mononuclear cells in lungs (Fig. 1). Histologically, severe congestion and cytoplasmic vacuolation of hepatocytes were observed in liver. The most striking histopathological changes in intestine consisted of congestion, necrosis, hemorrhagic enteritis, dilation of intercellular space and loss of villus epithelium (Fig. 2). Due to enterotoxaemia similar intestinal changes have been reported in deer (Khan *et al.,* 2008), cattle, calves, sheep and goats (Morris *et al.,* 2012; Garcia *et al.,* 2012) and in rabbits (Vidal *et al.,* 2012).The degenerative changes in kidneys were mainly of inflammation of glomeruli, extensive interstitial haemorrhages along with pyknosis and necrosis of renal tubular cells (Fig. 3). Similar histologic lesions in kidneys were described in sheep and goats (Garcia *et al.,* 2012). Increased urinary spaces, atrophy of glomeruli and sloughing of renal epithelium were the consisting changes in kidneys of affected deer. Similar pathological changes in kidneys of different animals due to enterotoxaemia have been reported (Javed *et al.,* 2009). Increased glucose level in urine of dead deer was also recorded. Increased level of glucose in urine is considered an indicator of enterotoxaemia in sheep (Javed *et al.,* 2009). From the findings of this study it can be concluded that in deer the pathogenesis, clinical and histopathological signs of enterotoxaemia are similar to that of sheep, goat and other animal species.

REFERENCES

Diab SS, H Kinde, J Moore, MF Shahriar, J Odani, L Anthenill, G Songer and FA Uzal, 2011. Pathology of Clostridium perfringens type C enterotoxemia in horses. Vet Pathol, 49: 255-263.

Garcia JP, J Beingesser, DJ Fisher, S Sayeed, BA McClane, H Posthaus and FA Uzal, 2012. The effect of Clostridium perfringens type C strain CN3685 and its isogenic beta toxin null mutant in goats. Vet Microbiol, 157: 412-419.

Javed MT, M Irfan, N Mukhtar, S Rahman and R Hussain, 2009. An outbreak of enterotoxaemia at livestock farm during subtropical summer. Acta Trop, 112: 225-227.

Khan A, I Ali, I Hussain and N Ahmad, 2008. *Clostridium perfringens* type D enterotoxaemia in the Chinkara Deer (*Gazella bennettii*). Turk J Vet Anim Sci, 32: 225-228.

Ma M, J Vidal, J Saputo, BA McClane and FA Uzal, 2011. The VirS/VirR two-component system regulates the anaerobic cytotoxicity, intestinal pathogenicity, and enterotoxemic lethality of *Clostridium perfringens* type C isolates CN3685. MBio, 2: e00338-10.

Morris WE, MV Dunleavy, J Diodati, G Berra and ME Fernandez-Miyakawa, 2012. Effects of *Clostridium perfringens* alpha and epsilon toxins in the bovine gut. Anaerobe, 18: 143-147.

Nasir AA, M Younus, MU Rehman, M Latif, A Rashid, R Ahmad and M Abbas, 2013. Molecular detection of *Clostridium perfringens* type D alpha and epsilon toxin genes from various tissues in lambs. Pak Vet J, 33: 492-495.

Popoff MR, 2011. Epsilon toxin: a fascinating pore-forming toxin. FEBS J, 278: 4602-4615.

Tahir MF, MS Mahmood and I Hussain, 2013. Preparation and comparative evaluation of different adjuvanted toxoid vaccines against enterotoxaemia. Pak J Agric Sci, 50: 293-297.

Uzal FA and BA McClane, 2011. Recent progress in understanding the pathogenesis of *Clostridium perfringens* type C infections. Vet Microbiol, 153: 37-43.

Vidal JE, M Ma, J Saputo, J Garcia, FA, Uzal and BA McClane, 2012. Evidence that the Agr-like quorum sensing system regulates the toxin production, cytotoxicity and pathogenicity of *Clostridium perfringens* type C isolate CN3685. Mol Microbiol, 83: 179-194.