



A Clinical Case of Severe Enteric Colibacillosis in a Doe and its Pathological Findings: A Case Report

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Abstract | This case reports the clinical management of enteric colibacillosis in a doe due to *E. coli* infection. An adult Jamnapari doe weighing 50kg was presented with a primary complaint of inappetance and weakness. The doe was weak, and on sternal recumbency when presented to the University Veterinary Hospital, Universiti Putra Malaysia. Physical examination findings showed pyrexia at 41.0°C, 5% dehydration, capillary refill time of more than 2 seconds, presence of light brown pasty fecal staining around the perineum region and well-formed feces covered with orange mucoid discharge. Bacteriology isolation and identification revealed *E. coli* from the fecal samples. Therefore, based on the physical examination findings and laboratory result, the case was diagnosed as enteric colibacillosis. The doe was treated promptly and vigorously with fluids, antibiotic, non-steroidal anti-inflammatory drug, and supplement for enteric colibacillosis. However, the doe was euthanized after 4 days of post hospitalization due to poor prognosis. Gross post mortem findings include presence of transparent thinning of the small and large intestinal wall indicative of necrotizing intestinal layer; and petechial haemorrhages in the mucosa of both the small and large intestines. Histopathological findings in the small and large intestine showed infiltrations of inflammatory cells in the submucosa, loss of architecture in the Crypt of Lieberkuhn and necrosis of the Brunner's glands. Both gross and microscopic pathological findings were suggestive of bacterial enteritis. Prompt diagnosis of cases of bacterial enteritis is paramount in order to avoid economic losses associated with the disease.

Keywords | Enteric colibacillosis, doe, clinical management, gross pathology, histopathology

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INTRODUCTION

Escherichia coli is a gram negative, rod, facultative anaerobic bacteria that is commonly found in the intestine of humans and animals (Wells et al., 1991; Besser et al., 1997; Fairbrother and Nadeau, 2006). Most *E. coli* are harmless and are usually normal flora in the intestine.

However, some *E. coli* strains such as shiga toxin-producing *E. coli*, enterotoxigenic *E. coli*, enteropathogenic *E. coli*, enteroaggregative *E. coli*, enteroinvasive *E. coli* and diffusely adherents *E. coli* are pathogenic and cause diarrhea in the host (Wani et al., 2013). These pathogenic strains can be transmitted through contaminated water, food, direct or indirect contact with infected animals or a person (Kahn

and Line, 2005; Fairbrother and Nadeau, 2006). Enteric colibacillosis is a common disease in young animals caused by colonization of enterotoxigenic strain of *E. coli* in the small intestine (Kahn and Line, 2005). Proper hygiene with good husbandry practice in the farm is the key to control and prevent *E. coli* disease.

Enteritis is an inflammation of the intestinal mucosa resulting in clinical signs of diarrhea, dysentery, abdominal pain, dehydration as well as electrolyte loss and imbalance. In severe case, mucus might be present in the feces while in worst case there might be shreds or sheets of exfoliated mucosa (Blood et al., 2011). The condition depends on the cause, severity and location. According to Ghanem et al. (2012), there are a few predisposing factors causing enteritis. Firstly, newly born kids with deficiency of immunoglobulins are more susceptible than the adults. Second, multiple stresses such as transportation, deprivation of food and water or weaning may cause immunosuppression to the animal thus leading to enteritis. Next, prolonged use of antibacterial agents through oral route may alter the intestinal microflora and permit the development of resistance by the organisms. In brief, all factors causing enteritis lower the immunity of affected animals causing an increase in the pathogenicity of the causative agent (Ghanem et al., 2012). This case report describes the clinical management of severe clinical enteritis in a doe and its pathological findings.



Figure 1: Doe with head tilted to the left flank

CASE REPORT

HISTORY

An adult Jamnapari doe weighing 50kg with body condition score of 2 out of 5 was presented to the University Veterinary Hospital, Universiti Putra Malaysia with a primary complaint of inappetence and weakness. During presentation, the doe was weak and in sternal recumbency with the head tilted towards the left flank (Figure 1). The

doe had a history of kidding two days prior to presentation. Vaccination and deworming were not practiced in the semi-intensive farm.

PHYSICAL EXAMINATION

Physical examination findings were pyrexia (41.0°C), 5% dehydration, capillary refill time of more than 2 seconds, presence of light brown pasty fecal staining around the perineum region and well-formed feces covered with orange mucoid discharge (Figure 2 and 3).



Figure 2: Light brown pasty fecal staining around the perineum region



Figure 3: Well-formed feces covered with orange mucoid discharge

The differential diagnoses were coccidiosis, parasitic gastro-enteritis (PGE) and bacterial enteritis.

LABORATORY INVESTIGATION AND RESULTS

Haematology findings revealed a normochromic normocytic anaemia with packed cell volume of 0.20L/L, which may be due to the previous parturition associated haemorrhage; and leukopaenia ($1.95 \times 10^9/L$), which was suggestive of immunosuppression. The serum biochemistry showed hypoalbuminaemia (20.1g/L), which may be due to malnutrition and protein losing enteropathies. Besides that, fecal sample was collected for parasitology and bacteriology identification. For parasitology identification, Modified McMaster Fecal Egg Count Technique was done and the result was negative for strongyle and coccidian ova. However, for bacteriology identification and isolation, the fecal sample was positive for *E. coli*. Thus, the final diagnosis for this case was enteric colibacillosis.

TREATMENT

The doe was treated promptly and vigorously for enteric colibacillosis. The dehydration was corrected by intravenous infusion of Lactated Ringer's solution administered for three days. Oxytetracycline 20mg/kg was injected intramuscularly once as broad-spectrum antibiotic to treat *E. coli* infection. Flunixin meglumine 2.2mg/kg was administered intravenously TID for three days as analgesic, anti-inflammatory and anti-pyrexia. 10mL of Kaolin-Pectin was given orally BID for two days to solidify the loose feces. 5mL of Biodyl was also administered intramuscularly once as vitamin supplement. Furthermore, physiotherapy was also performed twice daily to improve blood circulation and muscle functions.

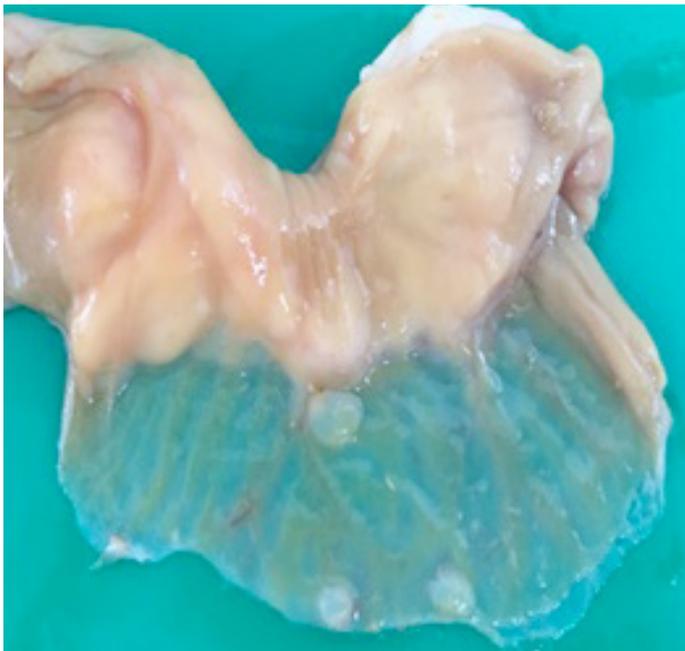


Figure 4: Transparent thinning of the intestinal wall indicative of necrotizing enteritis

PROGRESSION

The condition of the doe was not improving with persistent head tilt and frequent vocalization due to pain. Hence, the doe was euthanized using Xylazine 2% and Pentobarbital 200mg/kg, intravenously on Day 4 of hospitalization. The carcass was then sent for necropsy examination.



Figure 5: Petechial haemorrhage on the mucosal layer of the intestine

PATHOLOGY FINDINGS

Gross pathology findings showed presence of transparent thinning of the small and large intestinal wall indicative of necrotizing enteritis (Figure 4), and petechial haemorrhages in the mucosa of both small and large intestines (Figure 5). Histopathological examination of the small and large intestines showed infiltration of inflammatory cells in the submucosal layer, loss of architecture in Crypt of Lieberkuhn and necrosis of the Brunner's gland (Figure 6).

FINAL DIAGNOSIS

Based on the physical examination, diagnostic work-ups, post mortem and histopathological findings, the final diagnosis made was *E. coli* associated enteritis.

DISCUSSION

Bacteria, viral, protozoa and fungal are few virulent agents that may cause enteritis in small ruminants such as sheep and goat (Kahn and Line, 2005). In this case, the isolated bacteria were *E. coli* which caused enteric colibacillosis in an adult doe. However, colibacillosis is a major cause of diarrhea in kids. There are four main type of *E. coli* that can potentially be associated with diarrhea in goat kids; these are enterotoxigenic *E. coli* (ETEC), enteropathogenic *E. coli* (EPEC), enterohemorrhagic *E. coli* (EHEC) and

necrotogenic *E. coli* (NTEC). Nonetheless, enteropathogenic *E. coli* (EPEC), which also known as adhering and effacing *E. coli* (AEEC) is usually the causative agent for severe diarrhea in both kids and adult sheep and goat (Wani et al., 2013).

antibiotic of choice, but the doe did not response well to the treatment. According to Bogaard et al. (2000) and Wani et al. (2013), the wide usage of antibiotics had caused the development of antimicrobial resistance in many types of pathogenic *E. coli* and this may be the reason for poor response in the doe. Abu Daud et al. (2014) had stated that, polymerase chain reaction can be used in detection of antibiotic resistance in some *E. coli* strains, but this was not performed in this case.

CONCLUSION

Enteric colibacillosis may occur in animals of any age due to *E. coli* infection where common clinical signs such as diarrhea, dehydration, weight loss, and abdominal pain will be observed. The disease can be prevented with proper farm hygiene and can be control by early diagnosis and prompt treatment to prevent great economic losses in the ruminant industry.

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CONFLICT OF INTERESTS

There exists no conflict of interest.

AUTHORS' CONTRIBUTION

All authors contributed equally to the work and are responsible for the data presented herein.

REFERENCES

- Abu Daud NHB, Htin NN, Abba Y, Paan FH, Kyaw T, Khaing AT, Jesse FFA, Mohammed K, Adamu L, Tijjani A (2014). An outbreak of colibacillosis in a broiler farm. *J. Vet. Adv.* 4(7): 648-653.
- Besser TE, Hancock DD, Pritchett LC, McRae EM, Rice DH, Tarr PI (1997). Duration of detection of fecal excretion of *Escherichia coli* O157: H7 in cattle. *Concise Comm.* 175: 726-729.
- Blood DC, Studdert VP, Gay CC (2011). *Saunders Comprehensive Veterinary Dictionary, 4th Edition*, Saunders, London.
- Bogaard AEVD, Stobberingh EE (2000). Epidemiology of resistance to antibiotics link between animals and humans. *Int. J. Antimicrob. Agents.* 14: 327-335. [http://dx.doi.org/10.1016/S0924-8579\(00\)00145-X](http://dx.doi.org/10.1016/S0924-8579(00)00145-X)
- Fairbrother JM, Nadeau E (2006). *Escherichia coli*: On-farm contamination of animals. *Rev. Sci. Tech. Off. Int. Epiz.* 25(2): 555-569. <http://dx.doi.org/10.20506/rst.25.2.1682>

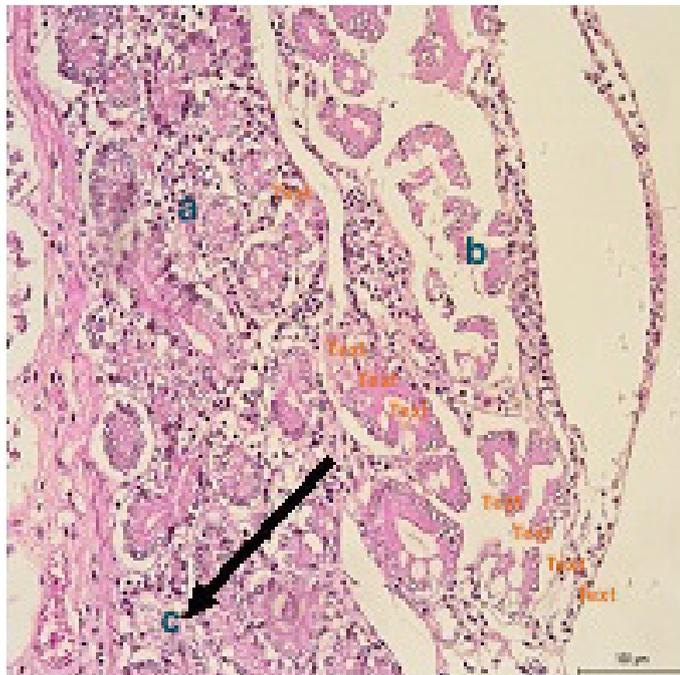


Figure 6: a) Infiltrations of inflammatory cells in the submucosa layer; b) loss of architecture in Crypt of Lieberkuhn; c) necrosis of the Brunners' gland in the small and large intestine

There are several common clinical signs that can be seen in the animal having enteritis; these include diarrhea, dehydration, weight loss, systemic reaction, acid-base and electrolyte imbalance as well as abdominal pain which were observed in this case (Blood et al., 2011). In the present case, if euthanasia was not done, the cause of death in the doe may be due to circulatory failure because of the severe dehydration. Inflammation and necrosis of the intestinal wall, efficiency of fluid absorption and excretion in the intestine will be reduced significantly. *E. coli* produces enterotoxins which are virulent factor that adhere to the enterocytes through the use of frimbriae and cause severe infection and inflammation in the submucosal layer of the intestine (Kaper et al., 2004). As observed in this case, severely dehydrated animals will have electrolyte imbalances and metabolic acidosis causing hyperventilation and muscle weakness due to inadequate bicarbonate and sodium concentrations in the blood circulation (Ghanem et al., 2012).

The primary therapeutic plan for animals with enteric colibacillosis is usually vigorous antimicrobial therapy to reduce morbidity and mortality (Wani et al., 2013). In the present case, Oxytetracycline 20mg/kg was used as the an-

- Ghanem MM, El-Fkhrany SF, Abd El-Raof YM, El-Attar HM (2012). Clinical and haematochemical Evaluation of Diarrheic Neonatal Buffalo calves (*Bubalus bubalis*) with Reference to Antioxidant Changes. *Benha Vet. Med. J.* 23(2): 275-288.
- Kahn CM, Line S (2005). *The Merck Veterinary Manual*, 9th Edition, MERCK & CO., INC, USA.
- Kaper JB, Nataro JP, Mobley HLT (2004). Pathogenic *Escherichia coli*. *Nat. rev. Microbiol.* 2: 123-140. <http://dx.doi.org/10.1038/nrmicro818>
- Wani SA, Hussain I, Beg SA, Rather MA, Kabli ZA, Mir MA, Nishikawa Y (2013). Diarrhoeagenic *Escherichia coli* and salmonellae in calves and lambs in Kashmir: absence, prevalence and antibiogram. *Rev. Sci. Tech. Off. Int. Epiz.* 32(3): 833-840. <http://dx.doi.org/10.20506/rst.32.2.2213>
- Wells JG, Shipman LD, Greene KD, Sowers EG, Green JH, Cameron DN, Downes FP, Martin ML, Griffin PM, Ostroff SM, Potter ME, Tauxe RV, Wachsmuth IK (1991). Isolation of *Escherichia coli* serotype O157: H7 and other shiga-like-toxin-producing *E. coli* from dairy cattle. *J. Clin. Microbiol.* 29(5): 985-989.