

Case Report





Clinico-pathological findings of septicaemic colibacillosis in a calf

Abstract

Colibacillosis is one of the major cause of mortality among young calves due to severe diarrhoea associated with calf scour and septicaemia. A two weeks old male Friesian cross calf weighing about 50 kg was presented to the large animal clinic of the University Veterinary Hospital with complains of yellowish-watery diarrhoea for the past two days, lethergy and recumbency. The calf was dull and depressed, diarrhoeic, dehydrated with bilateral sunken and congested eyes. Three other calves had recently died of recurring similar symptoms in the same herd. Antibiotic and fluid therapy with analgesic as adjunct treatment plan, but the calf died while being treated. The blood biochemical analysis revealed hyperkalaemia, hypocalcaemia, slightly elevated urea, creatinine and aspartate aminotransferase (AST). Necropsy findings were congestion and haemorrhage in the abomasum, jejunum and colon with sloughing off of the mucosa. The liver was pale, kidneys and heart were congested and haemorrhagic. Bacteriological examination revealed that *E. Coli* was consistently isolated in biopsy samples of the visceral organs. Improved sanitation and provision of adequate colostrum to new-born calves have forestalled the occurrence of the disease in the herd.

Keywords: E. Coli, colibacillosis, septicaemia, enteritis, clinical, necropsy, calf

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Faez Firdaus Jesse Abdullah, ^{1,6} Muhammad AbubakarSadiq, ^{2,3} Yusuf Abba, ^{2,4} Eric Lim Teik Chung, ¹ Lawan Adamu, ⁵ Asinamai Athlamai Bitrus, ¹ Idris Umar Hambali, ^{2,3} Mohammed Azmi Mohammed Lila, ¹ Abdul Wahid Haron, ^{1,6} Abdul Aziz Saharee, ¹ Annas Sallah, ¹ Abraham Gabriel

¹Department of Veterinary Clinical Studies, Universiti Putra Malavsia. Asia

²Department of Veterinary Pathology and Microbiology, Universiti Putra Malaysia, Asia

³Department of Veterinary Public Health and Preventive Medicine, University of Maiduguri, Nigeria

⁴Department of Veterinary Pathology, University of Maiduguri, Nigeria

⁵Department of Veterinary Medicine, University of Maiduguri, Nigeria

⁶Research Centre for Ruminant Diseases, Universiti Putra Malaysia, Asia

Correspondence: Yusuf Abba, Department of Veterinary Pathology and Microbiology, Faculty of Veterinary Medicine, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia, Tel +601 116 689 774, Email yabbavet@gmail.com

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Introduction

Colibacillosis, an infection with Escherichia coli (E. coli) is a major cause of mortality among young calves. 1 E. Coli is one of the common resident microbiota of the intestinal tract of calves; however there are certain strains of this organism that are pathogenic.² These pathogenic strains of E. Coli are the cause of severe diarrhoea, dehydration, fever, fatigue, malaise and depression that results in economic losses in both dairy and beef calves production.3,4 Transmission is most often through oral-faecal route by ingestion of contaminated feed and water, however infection through umbilical vein and nasopharyngeal were believed to occur in certain serotypes of E. Coli.4 Diagnosis of colibacillosis is based on history and clinical findings, enzyme linked immunosorbent assay (ELISA) and DNA gene probes specific for genes encoding enterotoxin.4 The definitive etiological diagnosis of colibacillosis depends on the isolation and characterization of the E. Coli. The treatment of colibacillosis requires vigorous antibiotic treatment together with intravenous fluid (IVF) and non-steroidal anti-inflammatory agents (NSAID).6 Parenteral antimicrobial such as oxytetracycline and sulfachlorpyridazine were reported to be effective in treatment of enteric colibacillosis.⁷ However most pathogenic E. Coli have demonstrated resistance to ampicillin, hence amoxicillin with clavulanic acid is likely to be the antibiotic of choice, together with rehydration and NSAIDs.8 Strict sanitary measures to ensure clean environment, provision of adequate colostrum and vaccination of calves have been shown to be an effective way in the prevention and control of bovine Colibacillosis.4 In this case report highlights

the common clinical and necropsy findings in a clinical case of septicaemiccoli bacillosis that may assist in diagnosis of the disease and prompt action taken to avoid losses.

Case history

A two weeks old male Friesian cross calf weighing about 50kg was presented to the large animal clinic of the University Veterinary Hospital, Universiti Putra Malaysia, with complains of yellowishwatery diarrhoea for two days and recumbency. The animal was previously administered Bitamax 2.5mL in the farm with subsequent cessation of diarrhoea but reoccurred a day later. The animal was lethargic and refused to feed and was kept in an isolation pen. The farmer reported that three other calves had previously died of similar symptoms in the herd.

Physical examination

Physical examination revealed that the calf had a body condition score of 2/5, was dull and depressed, had bilateral sunken and congested eyes, fecessoiled perineal region and watery faecal material with evident haematochezia. There were evidence of gingivitis and petechial haemorrhages on the tongue. The rectal temperature of the calf was 37.8°C, pulse rate 92/minute, respiratory rate was 48/minute and the mucous membranes were pale and dry, with capillary refill time (CRT) of 4seconds, the skin tent was 4seconds. There was ruminal stenosis evidenced by lack of rumen motility during auscultation and cardiac arrhythmia with heart auscultation.



Laboratory investigation

Swab sample from the gum was obtained using sterile swab and sent to the virology laboratory for viral isolation. Blood was aseptically collected and sent to the haematology laboratory for complete blood count and blood biochemistry analysis. Virological investigation revealed no viral growth detected and haematological analysis showed low leukocytes (WBC) count of 0.268X10⁹/L which is indicative of panleukopaenia. Blood chemistry revealed slight hyperkalaemia, hypocalcaemia with sodium (Na⁺) and chloride (Cl⁻) at high normal level. There are slightly elevated levels of urea (14.1mmol/L) and creatinine (215µmol/L), suggestive of renal function compromise. There was also an elevated blood level of aspartate aminotransferase (AST)(245.5 U/L), which is suggestive of liver damage.

Tentative diagnosis

Tentative diagnosis colibacillosis was made based on the history and clinical findings.

Treatment and case management

The initial treatment administered intramuscularly (IM) antibiotic pen-strept (1mL/25kg) which is a combination (penicillin and streptomycin) (Pen-strep) for five days. Flunixin meglumine, 1.1mL was administered IM(1.1mg/kg body weight) for two days. Dehydration was corrected by administering lactated Ringers (LR) solution and 20% glucose saline (G20) intravenously (IV). The calf was constantly monitored during the treatment. Unfortunately the calf died on the following night of hospitalization. The carcass was sent to the necropsy for post-mortem examination.

Post-mortem examination

Post-mortem examination revealed that the animal had rough hair coat, serous atrophy of fat in the ventral abdomen. There were petechiations on the tongue and inflammation of gingiva. Severe, generalized pulmonary congestion, with severe pulmonary oedema and frothy exudates in the trachea were observed (Figure 1). The abomasum was severely congested and haemorrhagic (Figure 2), segmental congestion and haemorrhages were observed in the jejunum and colon (Figure 3). Sloughing off of the intestinal mucosa was also observed (Figure 4). The liver was enlarged, pale and the gall bladder was distended (Figure 5). There was renal congestion with extravasations (Figure 6), generalized congestion of the heart, all of which are lesions suggestive of septicaemia. Histopathology revealed numerous infiltration of lymphocytes and macrophages in the gastrointestinal tract, severe necrosis of the small intestinal mucosa. There was also infiltration of lymphocytes around the central vein and portal triads of the liver. Biopsy samples of kidney, liver, lung and intestine were collected and sent for bacteriological examination. The result revealed that E. Coli was consistently isolated in all the tissues specimens submitted, that conclude this is acolibacillosis.

Definitive diagnosis

Based on the clinical involvement and laboratory findings, a definitive diagnosis of colibacillosis was made.

Herd management and control efforts

All apparently healthy calves were transferred to a new clean pen where general herd sanitary condition was improved by washing and disinfection of feeding and watering troughs. Herd water supply was improved to provide clean water and early feeding of calves with colostrum was advised. Subsequent fortnightly visits showed that no new case was recorded, indicating that the outbreak was resolved.

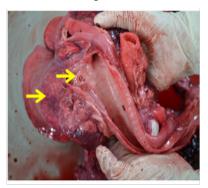


Figure I Congested lung with frothy exudates in the trachea (arrows).



Figure 2 Haemorrhagic inflammation of the abomasum (Abomasitis) (arrows).

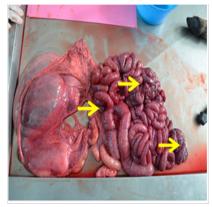


Figure 3 Severely congested and haemorrhagic jejunum and colon (arrows).



Figure 4 Necrotic enteritis with sloughing of the intestinal mucosa (arrows).

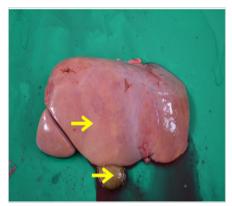


Figure 5 Pale liver with distended gall bladder (arrows).

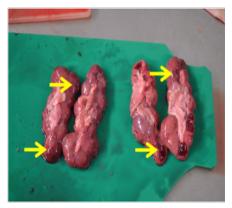


Figure 6 Longitudinal section of the kidneys showing congestion and haemorrhage (arrows).

Discussion

Calf mortality is one of the major economic threats that affect both dairy and beef cattle production in the livestock industry.1 Colibacillosis, a collection of diseases caused by E. Coli is known to be one of the greatest cause of mortality in calves. 4 This organism is the most important bacterial cause of diarrhoea in calves during the first few weeks of life. 9,10 Two pathotypes of diarrhoeagenic E. Coli were recognized to be most common, enteropathogenic E. Coli (EPEC) and a subset of the Shiga toxin-producing E. Coli (STEC) known as enterohemorrhagic E. Coli (EHEC).11 While septicaemia is caused by certain invasive serotypes of enterotoxigenic E. Coli(ETEC) that possess virulence properties which enables them to cross the mucous membranes of intestines, nasopharynx, crypts of tonsils or umbilical veins. 4 They also have the ability toresist the hosts' defence mechanisms following invasion and produce bacteraemia and septicaemia.5,12 In this case a two weeks old male Friesian cross calf initially showed typical sign of enteritis with yellowish-watery diarrhoea for two days and recumbency and the signs are consistent as described by Radostits, Gay.4 The stereotyped clinical manifestations of enteric Colibacillosis including diarrhoea and dehydration, weakness accompanied by loss of condition; systemic reaction as well as abdominal distention were observed in this case. The death of the calf in this case might be caused by the hypovolaemic shock from severe dehydration due to diarrhoea consistent with Gruenberg, 10 who reported that loss of >12% of body weight in fluid with subsequent hypovolemic shock and death may occur within 12-24hour. Postmortem examinations have shown lesions characteristic of enteritis

as well as septicaemia, which suggest systemic involvement. It has been observed that *E. Coli* strains that cause septicaemia are believed to produce endotoxins, which results in toxic shock and rapid death particularly in calves that are agammaglobulinemic due to lack of early colostrum feeding.⁴ Septicaemic colibacillosis often occurs as an acute disease and usually results in the rapid death of the calf due to *E. Coli* bacteraemia.¹³

In this case, necropsy lesion observed were consistent with Shesh, Joshi¹³ and Radostits, Gay⁴ who reported that calves that died of septicaemic colibacillosis will show areas of congestion, haemorrhages and presence of white necrotic foci on the lung, abomasum, jejunum, colon with sloughing of the mucosa of the intestines and involvement of the liver and kidneys. In this case changes in blood chemistry is in accord with the reports by Roy & Fernandes. 14 The therapeutic management of this case were in accord with approach recommended by Constable,7 Berchtold & Constable6 and Radostits, Gay4 where antimicrobials and intravenous fluid and electrolytes replacement are recommended in calves with colibacillosis. Radostits, Gav⁴ recommended control measures against colibacillosis such as clean environment to reduce degree of exposure of the new-born to the infectious agents, provision of adequate colostrum and optimum animal management and vaccination of the dam or the new-born calves. In this case, measures on sanitation and cleanliness of the farm was adapted and subsequently no outbreak was reported following the implementation of good sanitary practice.

Conclusion

In conclusion proper practice of biosecurity and sanitation in farm will prevent or reduce the incidence of clinical case of colibacillosis.

Acknowledgements

None.

Conflict of interest

Author declares that there is no conflict of interest.

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