

Suspected Bacillary Haemoglobinuria in a Calf: A Case Report

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Abstract | The present case reports the clinical management of a suspected bacillary haemoglobinuria in a Friesian calf. A 3-weeks-old Friesian cross female calf weighing 30kg was presented with a primary complaint of inappetance, haematuria and diarrhoea. Clinical evaluation revealed that the calf was pyrexia (40.8°C), having dyspnoea, pale mucous membrane with capillary refill time of > 2 seconds, bilateral prescapular and prefemoral lymph nodes enlargement and sign of abdominal pain during palpation. Furthermore, there was haematuria and greyish diarrhoea. The differential diagnoses at this point were babesiosis, anaplasmosis, trypanosomosis and bacillary haemoglobinuria. The blood result revealed an anaemia and leukocytosis with marked hyperbilirubinaemia. In addition, blood parasite screening showed negative results. Thus, the present case was diagnosed as suspected bacillary haemoglobinuria. Treatment was instituted with antihistamine 1mL/50kg administered intramuscularly 30 minutes prior to blood transfusion. 600mL of blood was collected from a donor cattle and transfused intravenously. Oxytetracycline (20mg/kg), was administered intramuscularly SID on day one and day four of hospitalization to treat bacillary haemoglobinuria. Flunixin meglumine (2.2mg/kg), was given intramuscularly SID for four days as anti-inflammatory, anti-pyrexia and analgesic. Percobsang (1ml/10kg), was also administered intramuscularly SID for four days as iron supplement. Bacillary haemoglobinuria is a severe and fatal disease; however in this case, prompt and effective treatments were able to prevent unnecessary death and losses to the farmer.

Keywords | Bacillary haemoglobinuria, *Clostridium haemolyticum*, Clinical management, Jaundice, Haematuria

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INTRODUCTION

Bacillary haemoglobinuria is caused by anaerobic spore forming *Clostridium haemolyticum* which is a soil borne organism that may be found in the gastrointestinal tract as normal flora (Kahn and Line, 2005). This disease is a highly fatal clostridial disease where the liver is the principal site of infection (Hussein et al., 2012). The morbidity rate of bacillary haemoglobinuria range from 0.25% to 12% and mortality from 80% to 100% (Uzal et al., 2016).

The percentage of morbidity and mortality depend both on the soil characteristics and the presence of liver flukes in endemic area. On the other hand, infections, liver biopsies, telangiectasia, metabolic disturbances, and reduced hepatic blood circulation associated with pressure of the pregnant uterus against the liver may be the factors for the germination of *Clostridium haemolyticum* spores in sporadic area such as Malaysia (Uzal et al., 2016). Haematuria, depression, fever, abdominal pain, dyspnea, dysentery and anaemia are the few clinical signs that will be observed in

infected cattle (Kahn and Line, 2005). Besides, cattle may be found dead without premonitory signs during bacillary haemoglobinuria outbreaks (Takagi et al., 2009). The most prominent gross lesions during post mortem examination is the ischemic infarct of the liver characterised by slightly elevated, lighter in colour than the surrounding tissue, and outlined by a bluish red zone of congestion. In addition, the kidneys are also dark, friable and studded with petechial haemorrhages, while the bladder contains purplish red urine (Kahn and Line, 2005). This case reports the clinical management of suspected bacillary haemoglobinuria in a Friesian calf where rapid diagnosis with right the treatment was able to treat the disease and prevent death.

HISTORY

A 3-weeks-old Friesian cross female calf weighing 30kg was presented with primary complaint of inappetance, haematuria and diarrhoea. The calf was managed intensively while the vaccination and deworming status of the farm were up-to-date.

PHYSICAL EXAMINATION

Physical examination revealed that the calf was pyrexia (40.8°C) with sign of laboured breathing. The mucous membrane was pale with capillary refill time of more than two seconds. Furthermore, the bilateral prescapular and prefemoral lymph nodes were enlarged with presence of tick infestations with sign of abdominal pain during palpation. There was haematuria and greyish diarrhoea during the physical examination. The differential diagnoses at this point of time were babesiosis, anaplasmosis, trypanosomiasis and bacillary haemoglobinuria.

DIAGNOSTIC WORK-UP

Blood result revealed that the calf was having marked haemolytic anaemia with erythrocytes ($2.48 \times 10^{12}/L$), haemoglobin (36.7 g/L) and packed cell volume (0.12 L/L). The white blood cells were also elevated with neutrophilia ($9.38 \times 10^9/L$), lymphocytosis ($8.36 \times 10^9/L$), and monocytosis ($1.84 \times 10^9/L$) which was suggestive of a bacterial infection and inflammation. There was also marked hyperbilirubinaemia (113.1 $\mu\text{mol}/L$) due to present of free bilirubin and hypoglobulinaemia (26.7 g/L). All other parameters were within the normal range. In addition, faecal sample was also collected for Modified McMaster Technique where the result revealed coccidian oocyst at 4750 o.p.g which was considered not significant. Besides, blood was collected through venipuncture for screening of blood parasite. Nonetheless, negative results were obtained from direct wet mount, haematocrit centrifugation technique and Giemsa-stained thin blood film. Based on the laboratory

findings, blood parasites such as babesia, anaplasma and trypanosomes were ruled out. Thus, based on the clinical signs and blood parameters, the present case was strongly suggestive of a suspected bacillary haemoglobinuria.

TREATMENT AND PROGRESSION

The therapeutic plan for this case was to treat for bacillary haemoglobinuria along with blood transfusion to increase the PCV level. On day one of hospitalization, antihistamine 1mL/50kg was administered intramuscularly 30 minutes prior to blood transfusion. With a desired PCV of 20% to be achieved, 600mL of blood was collected from donor cattle with PCV of 24%. Blood transfusion was then administered intravenously at the rate of 1 drop/second for the first 15 minutes to monitor for adverse reaction (Yagi and Holowaychuk, 2016). The infusion rate was then increased to 2 drop/second throughout the whole blood transfusion cycle. Furthermore, oxytetracycline (20mg/kg), was administered intramuscularly SID on day one and day four hospitalization to treat bacillary haemoglobinuria. Flunixin meglumine (2.2mg/kg), was administered intramuscularly SID for four days as anti-inflammatory, anti-pyrexia and analgesic. Fercobsang (1ml/10kg), was administered intramuscularly SID for four days as iron supplement. On day two of hospitalization, a quick PCV test was performed where the PCV increased from 12% to 20%. On day five of hospitalization, the calf was discharged after showing sign of improvement where the mucous membrane was pink with no sign of haematuria.

DISCUSSION

Clostridium haemolyticum organism can survive for long period in contaminated soil where ingestion of this organism leads to the disease development where latent spores will enter into the gastrointestinal tract. The spores will then penetrate the lining of the intestines spreading into the blood circulation. As a result, *Clostridium haemolyticum* spores will then lodge in the liver due to the high affinity of the organism towards the organ. The anaerobic condition of the liver will allow the spores to germinate and undergo multiplication causing the release of β toxin which lead to intravascular haemolysis and haemoglobinuria. This condition worsen with presence of liver fluke larvae that migrate around the liver worsen with the presence of inflammation and necrosis which is favourable for spore germination and growth of hypoxic-producing vegetative cells (Hussein et al., 2012). Infected cattle will show sudden onset of severe depression, fever, abdominal pain, dyspnea, dysentery, and anaemia. The most prominent sign is the typical portwine-colour urine, which foams freely when voided or on agitation. However, cattle may be found dead without premonitory signs (Takagi et al., 2009).

The routine method for diagnosis of bacillary haemoglobinuria from live cattle involves microbiological isolation of the organism *Clostridium haemolyticum* in blood samples (Shinozuka et al., 2011). Nonetheless, the organism is difficult to culture, thus the presence of typical liver infarcts is sufficient for a presumptive diagnosis during post mortem examination (Khan and Line, 2005). Isolation of *Clostridium haemolyticum* is time consuming and very tedious as it requires strict anaerobic conditions and may be contaminated by other anaerobic clostridia from the soil which grow faster than the pathogenic strain (Sasaki et al., 2001). As a result, rapid and accurate diagnosis have been developed by demonstrating the organism in the liver tissue by a fluorescent antibody or immunohistochemical test or by demonstrating the toxin in the fluid in the peritoneal cavity or in a saline extract of the infarct. However, these techniques must be interpreted with caution as *Clostridium haemolyticum* may present naturally in the gastrointestinal tract (Hussein et al., 2012). In the present study, the calf was diagnosed with bacillary haemoglobinuria after excluding blood parasites infestation. The presence of clinical signs such as haematuria, pyrexia, and laboured breathing showed the toxemic state in the affected calf due to vascular damage and intravascular haemolysis (Radostits et al., 2007). According to Smith (1990), bacillary haemoglobinuria infection leads to 30 to 50% of red blood cells destruction as was observed in this case where the calf had severe anaemia and blood transfusion was performed on day one of hospitalization.

Early treatment with penicillin or broad-spectrum antibiotics is essential for clinical cases of bacillary haemoglobinuria, while whole blood transfusions and fluid therapy are also helpful (Kahn and Line, 2005). Procaine penicillin G is usually the antibiotic treatment of choice, it but may be more valuable in preventing the disease in unvaccinated animals at risk during an outbreak, before they are vaccinated. Although prompt treatment with high doses of specific antibiotics and antitoxin serum are recommended, there is usually not enough time to initiate treatment with most animals dying within few hours of disease onset (Takagi et al., 2009). Nevertheless, the calf in the present case was administered with broad spectrum oxytetracycline promptly (20mg/kg) and recovered completely with the help of antibiotic therapy together with blood transfusion. Besides, the calf may be in the early stage of infection which prompt treatments was able to prevent further vascular damage and intravascular haemolysis.

As for prevention, the disease is associated with liver flukes, thus reducing or preventing liver flukes infestation is considered very effective in reducing the incidence of bacillary haemoglobinuria cases (Kahn and Line, 2005). This is achieved by treating the animals with fasciolocides and manages the animals intensively. There are currently

no effective methods to achieve this under extensive conditions (Uzal et al., 2016). Cattle that are in contact with animals from areas where this disease is endemic should be vaccinated, as the latter may be carriers (Smith and Jasmin, 1956). Nonetheless, there are no vaccines against *Clostridium haemolyticum* in Malaysia although vaccinations against this organism are practiced worldwide (Hjerpe 1990; Takagi et al., 2009). Thus, control of fascioliasis or liver fluke in Malaysia farm is the main factor to prevent the occurrence of bacillary haemoglobinuria that leads to unnecessary economic losses.

CONCLUSION

It can be concluded that bacillary haemoglobinuria is a fatal disease associated with clinical signs and hemato-biochemical alterations that are helpful for a rapid preliminary diagnosis of the disease. Failure of diagnosis will eventually lead to an acute disease and death due to circulatory failure resulting from severe intravascular haemolysis. Therefore, prompt therapeutic management as was done in this case was paramount in avoiding economic losses due to the disease.

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CONFLICT OF INTEREST

There exists no conflict of interest.

AUTHORS' CONTRIBUTION

All authors contributed equally and approved the final manuscript.

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