Case Report

A clinical case of bovine trypanosomosis in an endemic farm in Malaysia

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ABSTRACT

Objective: This case report describes the management of a clinical case of trypanosomosis in an adult Friesian Sahiwal cow.

Materials and methods: An adult cow aging 3 years was presented with a complaint of wound infection, weakness and inappetence. Physical examination was carried out and samples were collected for laboratory investigations.

Results: The clinical history revealed generalised enlargements of the pre-scapular and pre-femoral lymph nodes, pale mucous membrane and weight loss. Laboratory investigation showed that the cow had normocytic normochromic anemia with hyperproteinemia. Thin blood smear examination revealed the presence of *Trypanosoma evansi*. Treatment was instituted with Diminazene aceturate dosed at 3.5 mg/kg bwt through intramuscular (IM) route for 3 days, 20 mL of Fercobsang for 3 days, IM, Flunixin meglumine dosed at 1.1 mg/kg bwt, IM, and Oxytetracycline dosed at 20 mg/kg bwt, IM once. The wounds were cleaned daily for one week. Examination of the blood film after therapy showed no parasite.

Conclusion: The findings of this case report demonstrate the importance of an effective treatment regimen in managing bovine trypanosomosis in an endemic farm.

KEYWORDS

Bovine trypanosomosis, Clinical management, Endemic, *Trypanosoma evansi*

INTRODUCTION

Bovine trypanosomosis poses a serious threat to the development of livestock and agricultural production of many tsetse-infected zones (Cherenet et al., 2004; Abdoulmoumini et al., 2015). Trypanosomosis is a fatal and debilitating disease of various domestic livestock and wild animals. The disease is caused by a protozoan parasite of the trypanosomes species cyclically and mechanically transmitted by tsetse fly and other biting flies. The disease is one of the major problems of the livestock industry in many tropical countries in Africa and Asia (Zecharias and Zeryehun, 2012). There are principally three major causative agents of the disease transmitted by different species of tsetse flies which includes; Trypanosoma congolense, T. vivax, T. brucei and occasionally T. evansi (Chau et al., 2016).

The impact of bovine trypanosomosis is not restricted to livestock production alone, but extends to changes in land use and exploitation of natural resources use, access to available and cultivable land, restriction of opportunities for diversification of agricultural production (Swallow, 2000). Furthermore, the disease reduces the efficiency of bulls and discourages the use of drought animals in crop production (Omotainse et al., 2004; Leta et al., 2016).

The distribution of the causative organism is mostly restricted to the African continent and Asia. However, T. vivax has been observed to cross the Atlantic moving upwards towards South America through mechanical transmission by biting flies (Hide et al., 1996; Naessens, 2006). In addition T. evansi have also been observed to have a wide host range causing infection in livestock such as buffalo and camel in some parts of Asia (Njiru et al., 2004; Naessens, 2006). A major clinical feature consistent with trypanosomosis in livestock is Fever and anaemia. However, there is a generalized leukopenia, weight loss, hypertrophy of the liver and spleen. In chronic cases the infected animals becomes lethargic, emaciated due to inappetence and finally die due to congestive heart failure (Morrison et al., 1983). This case describes the management of a clinical case of trypanosomosis in an endemic cattle farm.

CASE HISTORY

An adult female Friesian Sahiwal dairy cow aging 3 years was presented with wounds at multiple joints and inappetence. The cow was managed semi-intensively. The owner stated that multiple wounds at the rump region may be due to birds pecking. The body condition score (BCS) was 2 out of 5 (Figure 1).

During physical examination, the cow was dull but responsive. The vital signs were normal. Mucous membrane was pale with capillary refill time (CRT) of more than 2 sec (Figure 2). There was presence of circumscribed wounds at left hock joint, left and right knee joint and ventral thoracic region (Figure 1). There were enlargement of bilateral prescapular, prefemoral and axillary lymph nodes approximately 6 cm x 10 cm, 5 cm x 8 cm, 4 cm x 4 cm respectively (Figure 3a and b). Blood was taken from jugular vein for blood parasites detection, complete blood count and serum biochemistry analysis. The differential diagnosis for this case at this point in time was highly suggestive of blood parasite infection. White blood cell parameters were normal. There was normochromic, normocytic moderate anemia with hyperbilirubinemia with low conjugated bilirubin indicative of hemolytic anemia. This is consistent with blood parasitism. There was hyperproteinemia due to hyperglobulinenia. This may be due to host immune response towards the blood parasitism. Hypoalbuminemia could be due to reduced albumin production by the liver during blood parasitism. It could be also caused by low feed intake due to inappetence for several days.

Figure 1: General appearance of cattle. BCS was 2/5. Presence of circumscribed wound at bilateral knee joints.

Figure 2: Pale mucous membrane with CRT of more than 2 sec.
owing the first injection. 20 mL patient, MCHC=Mean Corpuscular Hemoglobin. 2013; Figure 4); The final diagnosis was. The animal responded well towards the rane surface active within one week of treatment. Blood was treatment as the appetite recovered and become more after treatment Follow up visitation: The cow was revisited one week to reduce the pain from the wounds. Flunixin mg/kg bwt was bacterial infection from the wound while Flunixin mg/kg bwt was dosed at 20 mg/kg bwt given intramuscularly once to treat joints. On the same time, Oxytetracycline dosed at 20 mg/kg bwt was given intramuscularly once to treat bacterial infection from the wound while Flunixin meglumine was given intramuscularly dosed at 1.1 mg/kg bwt once to reduce the pain from the wounds. Anti-parasite therapy started on the day of first visit with Diminazene aceturate dosed at 3.5 mg/kg bwt through intramuscular (IM) route. It was given every 2 days for another 2 doses following the first injection. 20 mL Fercobsang was given intramuscularly once as iron supplement to treat anemia caused by trypanosomosis. Wound cleaning were performed at wounds at multiple joints. On the same time, Oxytetracycline dosed at 20 mg/kg bwt was given intramuscularly once to treat bacterial infection from the wound while Flunixin meglumine was given intramuscularly dosed at 1.1 mg/kg bwt once to reduce the pain from the wounds. Follow up visitation: The cow was revisited one week after treatment. The animal responded well towards the treatment as the appetite recovered and become more active within one week of treatment. Blood was resampled to check for trypanosomes and there was none. The prognosis of this case was good as we diagnosed the trypanosomosis during early phase, treated it aggressively and the animal responded well towards the treatment.

**DISCUSSION**

There are various factors which contribute to higher chance of reinfection and outbreak in a farm that is endemic with trypanosomosis (Radostits et al., 2006; Auty et al., 2015). Trypanosomes developed a well-known escape mechanism known as antigenic variation by which they successively exhibit various main membrane surface glycoproteins: the variant surface glycoprotein (VSG) whenever host immune system eliminating them (Masumu et al., 2012; Desquesnes et al., 2013; Holmes, 2013). In endemic farms, this mechanism enables some of the trypanosomes to survive host immune response and re-emerge once the host immunity is weakened causing reinfection and outbreak. The risk of outbreak is increased when there is presence of wildlife surrounding the farm which is another potential reservoir for

**Table 1(a). Complete blood count parameters of the patient during first visit.**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Unit</th>
<th>Results</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythrocytes</td>
<td>×10¹²/L</td>
<td>4.71</td>
<td>5 – 10</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>g/L</td>
<td>65.3</td>
<td>80 – 150</td>
</tr>
<tr>
<td>Packed cell volume</td>
<td>L/L</td>
<td>0.21</td>
<td>0.35 – 0.55</td>
</tr>
<tr>
<td>MCV</td>
<td>fL</td>
<td>45</td>
<td>40 – 60</td>
</tr>
<tr>
<td>MCHC</td>
<td>g/L</td>
<td>311</td>
<td>300 – 360</td>
</tr>
<tr>
<td>Band neutrophils</td>
<td>×10⁹/L</td>
<td>0.09</td>
<td>&lt; 0.2</td>
</tr>
<tr>
<td>Segmented neutrophils</td>
<td>×10⁹/L</td>
<td>3.51</td>
<td>0.6 – 4.0</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>×10⁹/L</td>
<td>4.77</td>
<td>2.5 – 7.5</td>
</tr>
<tr>
<td>Monocytes</td>
<td>×10⁹/L</td>
<td>0.45</td>
<td>0.05 – 2.4</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>×10⁹/L</td>
<td>0.81</td>
<td>0.1 – 1.3</td>
</tr>
<tr>
<td>Basophils</td>
<td>×10⁹/L</td>
<td>0.09</td>
<td>Rare</td>
</tr>
<tr>
<td>Thrombocytes</td>
<td>×10⁹/L</td>
<td>328</td>
<td>100 – 800</td>
</tr>
<tr>
<td>Plasma protein</td>
<td>g/L</td>
<td>80</td>
<td>60-80</td>
</tr>
<tr>
<td>Icterus index</td>
<td>Unit</td>
<td>10</td>
<td>&lt; 15</td>
</tr>
</tbody>
</table>

MCHV=Mean corpuscular volume, MCHC=Mean Corpuscular Hemoglobin Concentration

**Table 1(b). Serum biochemistry profiles of patient during first visit.**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Unit</th>
<th>Results</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total bilirubin</td>
<td>mmol/L</td>
<td>54.3</td>
<td>1.7 – 27.2</td>
</tr>
<tr>
<td>Conjugated bilirubin</td>
<td>mmol/L</td>
<td>0.8</td>
<td>&lt;10.2</td>
</tr>
<tr>
<td>Unconjugated bilirubin</td>
<td>mmol/L</td>
<td>53.5</td>
<td>&lt;10.2</td>
</tr>
<tr>
<td>y-GT</td>
<td>U/L</td>
<td>19</td>
<td>&lt;25</td>
</tr>
<tr>
<td>AST</td>
<td>U/L</td>
<td>56.2</td>
<td>50 – 100</td>
</tr>
<tr>
<td>Total serum protein</td>
<td>g/L</td>
<td>111.7</td>
<td>55 – 75</td>
</tr>
<tr>
<td>Albumin</td>
<td>g/L</td>
<td>19.2</td>
<td>25 – 40</td>
</tr>
<tr>
<td>Globulin</td>
<td>g/L</td>
<td>92.5</td>
<td>27 – 45</td>
</tr>
<tr>
<td>A/G ratio</td>
<td>Unit</td>
<td>0.2</td>
<td>0.8 – 1.2</td>
</tr>
</tbody>
</table>

AST=Aspartate aminotransferase, y-GT=Glutamyl transferase
trypanosomes (Mulatu et al., 2016). Tabanid flies (Tabanus stratus and T. reducens), stable flies (Stomoxys calcitrans), buffalo flies (Haematobia spp.) or hematophagous flies are known as the primary transmitter of the disease (Manuel et al., 1998; Birhanu et al., 2015). Hot and humid tropical climate in Malaysia favours the breeding of these flies all year round. High vectors challenges in endemic farm will increase the risk of outbreak or re-infection as it is the most effective route of transmission of the disease (Manuel et al., 1998; Radostitis et al., 2006; Masumu et al., 2012).

The golden diagnostic test is direct parasitological detection using microscopic examination of blood smear. However, the sensitivity of blood smear is limited as less than 50% of infected animal can be identified with blood smear (Sivajothi et al., 2016). In acute trypanosomosis, it is always associated with high parasitemia which increase the detection rate via blood smear. The detection window decrease as the disease progress into more chronic form due to low parasitemia (Desquesnes et al., 2013).

Most domestic and many wild animals are susceptible to infection with T. evansi. However, the pathogenicity of the causative agent on the host animal varies according to the host specie, virulence of the trypanosomes and other stress factors as well as the local epizootological conditions (Brun et al., 1998). In most domestic and wild animals, the clinical manifestation of Surra includes fever and anaemia, emaciation, oedema, weight loss and hypertrophy of the lymph nodes and spleen. This clinical manifestation was consistent with the clinical signs of the disease reported in this case, which was characterized by a normocytic normochromic anemia.

Various attempts made to control the spread of bovine trypanosomoses was hampered by the problem of geographical scale and in most cases the biology of the organism itself. Since it has been established that the organism undergo antigenic variation in order to evade the action of the host immune response. In addition, resistance development to most trypanocidal drugs is on the increase and there seems to be dim hope of producing a conventional vaccines against trypanosomes (Brun et al., 1998; Keating et al., 2015). Furthermore, a particular specie of trypanosae can have a number of different serodemes or strains all having the capacity to cause infection (Barry and Turner, 1991). These listed factors and other myriads problems associated with the tsetse fly control program have restricted the rearing of cattle and other ruminant animals to areas where there is less tsetse. But, deficient in abundant feed and water. Exception to this rule is however, West Africa where there are a number of breeds of cattle such as West African Shorthorn, N’dama cattle and some small ruminant breeds who have over the time developed resistance to trypanosomes (Terefe et al., 2015). Such breeds of animals can survive in tsetse endemic zone even though due to overstocking of livestock, undernutrition sets in and cause immune compromise (Holmes, 2013).

Trypanosomosis can only be treated medically either by chemotherapy or chemoprophylaxis. Diminazene aceturate (at 3.5-7.0 mg/kg bwt, IM) is the first line of treatment in trypanosomosis. The recommended dosage is 3.5-7.0 mg/kg bwt, 2 doses with 5 days interval which can totally eliminate the trypanosomes (Radostitis et al., 2006). However, Diminazene acetate provide shorter protection period, which is about 2 weeks at the highest dosage (at 7.0 mg/kg bwt). Therefore, it is good to treat individual cases in a non-endemic farm (Uilenberg, 1998). Isometamidium (at 0.25-1.0 mg/kg bwt) provides longer protection period, which is up to 4 months at 1.0 mg/kg bwt. Therefore, it is more suitable for the purpose of chemoprophylaxis to treat infected herd in an endemic farm (Vreysen et al., 2013). In this case, Isometamidium was not available, hence, 3 doses of 3.5 mg/kg bwt Diminazene acetate were administered within 5 days (2 days interval each) to establish high plasma concentration and longer plasma half-life in the animal.

CONCLUSION

This case report describes a case of bovine trypanosomosis caused by T. evansi. Individual cases of trypanosomosis can be treated with Diminazene aceturate. In this case, 3 doses of Diminazene aceturate led to a clinical cure. The control of trypanosomosis in endemic farms may involve control of vectors, prophylactic treatment and good husbandry of animals at risk. Total elimination of trypanosomas is unpractical, but there are control strategies to achieve “tolerable” level where cost-effective production of animal is possible.

CONFLICT OF INTEREST

None of the authors have any conflict of interest.

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