

International Journal of Veterinary Medicine and Animal Health Vol. 10 (1), pp. 001-009, January, 2019. Available online at www.internationalscholarsjournals.org © International Scholars Journals

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Full Length Research Paper

Posterior paresis in pregnant gilts experimentally infected with *Trypanosoma brucei*

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Accepted 24 October, 2018

The effect of *Trypanosoma brucei* infection on reproductive efficiency in gilts (n=12) was conducted. The gilts were bought as piglets aged eight weeks from piggeries in Samaru village in Zaria, Nigeria. On attaining puberty, the gilts were divided into experimental and control groups, each group containing six animals. All the gilts were subsequently bred by fertile boars and they were confirmed pregnant. The pregnant gilts were then inoculated with about 1.8×10^6 trypanosomes via the anterior vena cava. The infected gilts developed clinical trypanosomosis after a pre-patent period of 2 to 3 days. The clinical signs observed were intermittent fever, short and moist cough, moist rales, mucopurulent ocular discharges, hyperaemia of the skin, reduced feed intake, loss of body condition, recumbency, uncoordinated movements, posterior paresis, loss of pregnancy and death. Severe degeneration of the fibers of the hamstring muscles was observed along with elevated levels of serum potassium, aspartate amino transferase and creatine kinase.

Key words: Trypanosomosis, infection, pregnant gilts.

INTRODUCTION

The physiology of animals alters when they are infected with trypanosomosis (Biryomumaisho et al., 2003). This is usually due to the wide range of biochemical changes that take place in the infected animals (Katunguka– Rwakishaya, 1996). Hematological aberrations do occur in animals infected with trypanosomes (Anosa and Isoun, 1980; Singla and Juyal, 2000). The severity of the hematological and biochemical changes that take place in infected animals is determined by the strain of the infecting trypanosome and the host (Anosa, 1983a, b). The evaluation of blood indices and parameters is required to enable the health status of animals to be determined (Coles, 1986). This gives an indication of the degree of degenerative changes that have occurred to host tissues as well as the severity of the infection (Otesile et al., 1991).

Pigs like other domestic livestock are infected with several species of trypanosomes. The trypanosomes that cause infections in pigs are *Trypanosoma simiae*, *Trypanosoma brucei Trypanosoma congolense and Trypanosoma suis* (Losos, 1986; Sekoni, 1994; Seifert 1996). Infections in pigs by *T. brucei* have been reported to cause nervous signs (Otesile, 1992; Onah and Ozuokwu 1991), uncoordinated movements and posterior paresis characterized by wobbling of the hind-legs (Mumah, 1996; Allam, 2004). The posterior paresis interfered with mating efficiency of gilts (Allam et al., 2006). This paper intends to report the cause of the posterior paresis that was noticed in pregnant gilts infected with *T. brucei* along with other pathogenic effects of this parasitic infection.

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MATERIALS AND METHODS

Experimental animals

Twelve cross breed piglets aged eight weeks were bought from piggeries in Samaru village in Sabon gari Zaria Local Government Area of Kaduna State, Nigeria. They were housed in clean fly proof pens in the Faculty of Veterinary Medicine, Ahmadu Bello University Zaria, Nigeria. Base line hematological data of the piglets were obtained on arrival and they were screened for endo and ecto parasites. The piglets were subsequently ear notched for identification and treated for nematodes and ectoparasites with vermectin (lvomec®), at 200 μ g/kg body weight sub-cutaneously. The piglets were fed compounded diet of 18% crude protein (composition: maize 36.8%, soya bean 5%, ground nuts 23.5%, rice bran 30%, beniseed 2%, bone meal 2%, premix 0.2%; table salt 0.5%) and water was provided *ad libitum*.

Strain of T. brucei used

The *T. brucei* used in this present study was obtained from the Nigerian Institute of Trypanosomiasis Research Vom, Nigeria. It was originally isolated from a pure natural infection in cattle in Federe, Kaduna State Nigeria. The parasite was inoculated into 2 mice and transported to the Faculty of Veterinary Medicine, Ahmadu Bello University Zaria, Nigeria.

Inoculation of animals

When the gilts were 7 months old, they were randomly divided into two groups of 6 experimental and 6 controls. About 2 ml of the infected rat blood containing approximately 1.8×10^6 *T. brucei* organisms were inoculated into each animal in the experimental group via the anterior vena cava while the ones in the control group were left intact. Following inoculation, all the gilts were clinically examined, weighed and their blood samples collected daily using EDTA as anticoagulant. The blood was examined for levels of parasitaemia, packed cell volume (PCV) and serum biochemical parameters. The values obtained were recorded daily along with their weights and rectal temperatures. This continued until all the infected animals were positive for trypanosomes. Subsequently, all the gilts were clinically examined weighed and their blood collected weekly to determine PCV levels, parasitaemia, and serum biochemical parameters until the experiment was terminated.

Gross pathological examination

Post mortem examination was carried out on the infected gilts that died during the study and also on the remaining ones that were sacrificed at the end of the experiment.

Histo-pathological examination

The samples of the heart, brain and hamstring muscles were obtained from all the infected pigs. They were put into appropriately labeled bijou bottles and fixed in 10% formalin. The tissues were dehydrated and processed in tissue wax embedded in paraffin and were subsequently cut in to 5 μ m thickness on slides and stained with hematoxyline and eosin. The slides were eventually examined using a light microscope.

Statistical tests used

Data obtained from the study were analyzed using student t test.

Values of p<0.05 were considered to be statistically significant (Chatfield, 1983).

RESULTS

Clinical observations

All the pigs developed clinical trypanosomosis after a pre-patent period of 2 to 3 days. There was a steady increase in the levels of parasitaemia of the infected gilts which was followed by fluctuations. Peak parasitemia was recorded between days 7 and 35 post infection (pi) (Figure 1).

The rectal temperatures of all the infected gilts increased during the course of the infection. This was also followed by fluctuations. The highest mean temperature attained by the infected gilts was 40.3° C. During this period, the temperatures of the animals in the control group were within the normal range. The difference in the ranges of temperature between infected and the control animals was significant (p<0.05) (Figure 2).

There was a gradual decrease in the PCV of the infected gilts. This was first noticed on day 14 pi in one of the pigs. The lowest PCV value recorded during the experiment was 17%, the mean PCV values of the infected animals were significantly different from those of the control (p<0.05) (Figure 3).

The infected animals from day 28 pi were observed to be gaining less weight than the ones in the control group. The infected animals were gaining an average of 0.71 kg every two weeks whereas the controls were gaining an average of 1.5 kg during the same period. By day 42 pi, the infected animals started losing weight while the control gained weight steadily (Figure 4). The difference in the weights between the infected and the control groups was found to be statistically significant (p<0.05).

Other clinical signs observed in the infected gilts were pale mucus membranes, short and moist cough, moist rales, mucopurulent ocular discharges, hyperemia of the skin, reduced feed intake, recumbency, uncoordinated movements, posterior paresis, loss of pregnancy and death.

During the study, the mean aspartate amino transferase (AST) levels of the infected pigs increased significantly from day 35 pi and its level remained elevated until the end of the study. The highest mean level of 60.6 I. U/L was recorded on day 91 pi (Figure 5).

The mean serum levels of creatine kinase (CK) increased progressively from day 21 pi till the end of the experiment in the infected animals. The highest mean level recorded for this enzyme was 856.7 l. U/L on day 42 pi. The values of CK were significantly different from those of the control animals (p < 0.05) (Figure 6).

The mean serum values of potassium in the infected gilts increased progressively during the study. This

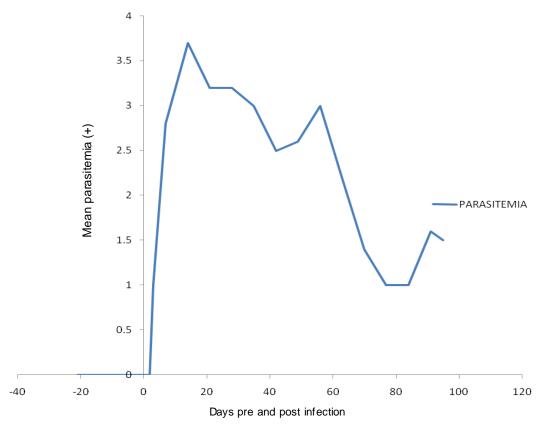


Figure 1. Mean parasitemia of *T. brucei* Infected gilts.

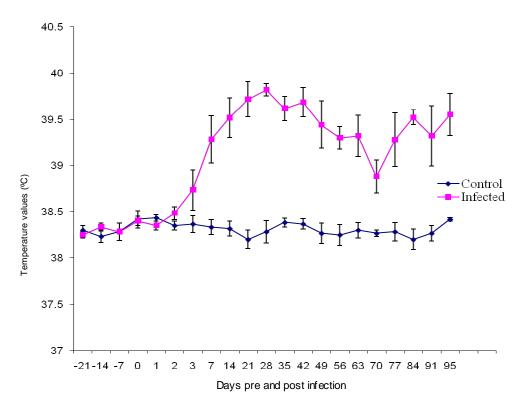


Figure 2. Mean temperature changes of *T. brucei* Infected and control gilts.

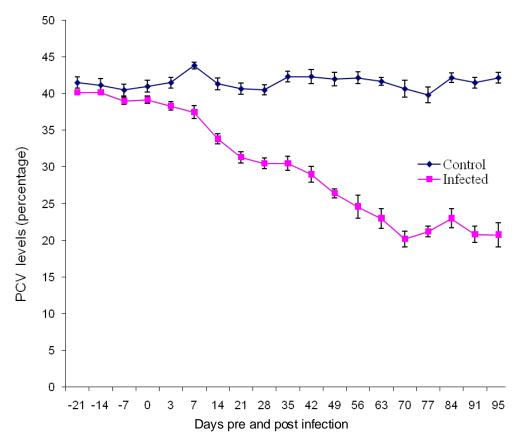


Figure 3. Mean packed cell volume of *T. brucei* Infected and control gilts.

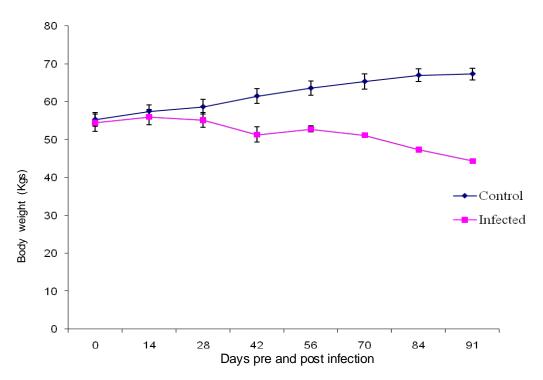


Figure 4. Mean body weight changes of *T. brucei* Infected and control gilts.

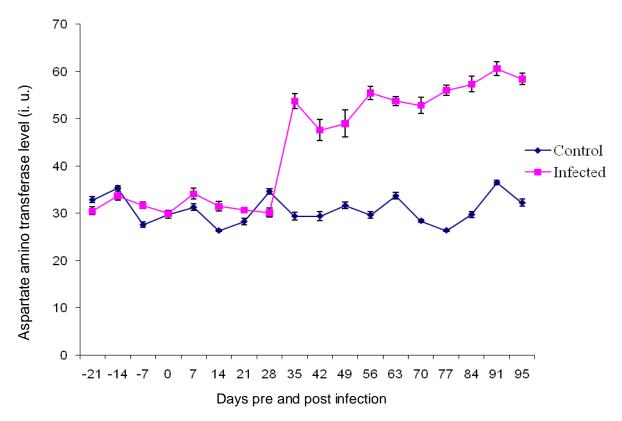
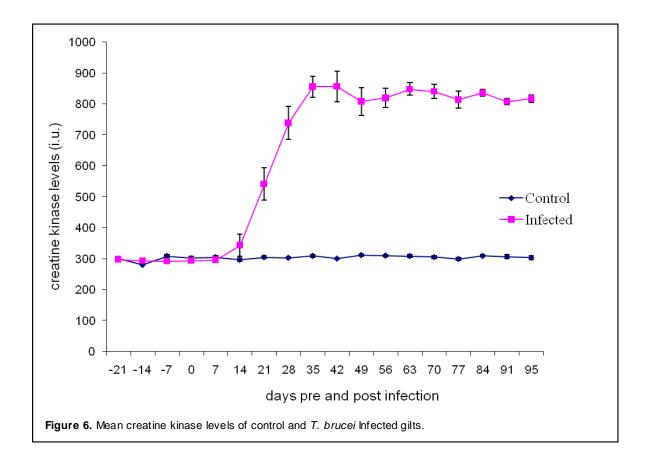
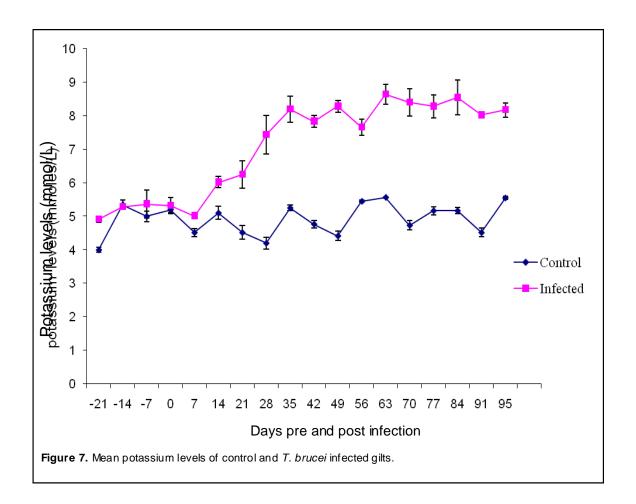


Figure 5. Mean aspartate amino transferase levels of control and T.brucei infected gilts.





increase started on day 14 pi. The highest mean level recorded was 8.6 mmole/ L on day 63 pi. These levels were significantly different from those of the control animals (p < 0.05) (Figure 7).

Histopathological lesions

Severe inflammatory lesions were observed in the heart of the infected gilts (Figure 8). In some cases, necrosis of the myocardium with massive mononuclear cellular infiltration was observed. The blood vessels in the cerebrum were congested (Figure 9). The fibers of the hamstrings muscle were degenerated. The perimysial tissue of the muscles had widened and had become edematous (Figure 10).

DISCUSSION

The increase in AST levels noticed in this study agrees with the results obtained during an infection in sheep by *T. brucei* (Taiwo et al., 2003), *T. vivax* infection of cattle and sheep (Gray, 1963), *T. congolense* infection of goats (Adah et al., 1992), and also in dogs infected with *T. brucei* (Omotainse et al., 1994). However, it contradicts

observations were made by Taiwo et al. (2003) during an infected of sheep with *T. congolense*.

The increase in the levels of CK agrees with results obtained in a *T. cruzi* infection in mice (Cano et al., 2000), but disagrees with results obtained by (Lunkins, 1992; Chaudhary and Iqbal 2000), who observed no change in CK values in animals with trypanosomosis.

The causes of elevation of AST levels in the serum of animals are necrosis of the liver, skeletal muscles and kidneys, whereas CK is increased in skeletal muscle disease, myocardial injury or necrosis and cerebral cortical necrosis (Lording and Friend, 1991).

Conclusion

In this study, the increase in AST and CK levels in the infected gilts could only have been due to the degeneration of skeletal muscle fibers that was observed histopathologically. Even though histopathological lesions were also observed in the heart and brain of the infected animals, they were not that severe as to cause the increase in the levels of AST and CK as was noticed in this experiment.

The hyperkalemia that was noticed is suggestive of massive leak ages of this electrolyte from the skeletal

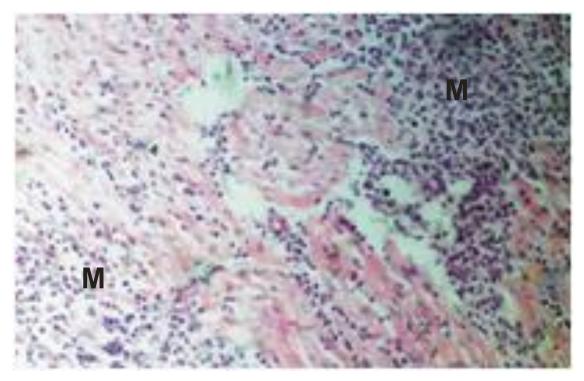


Figure 8. Photomicrograph of the heart of a *T. brucei* infected gilt. Note the mononuclear cellular infiltration (M). H and E X 303.

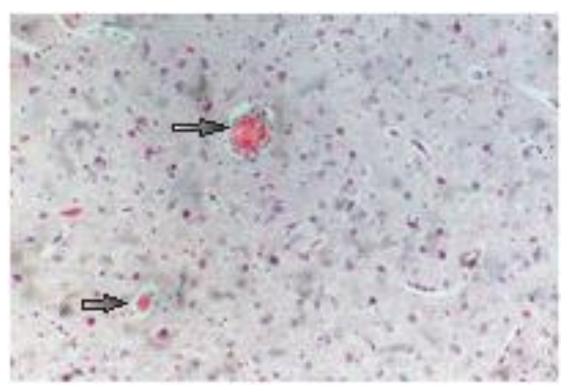


Figure 9. Photomicrograph of the brain of a gilt infected with *T. brucei*. Note the congestion of the cerebral blood vessels (arrow s). H and E X 303.

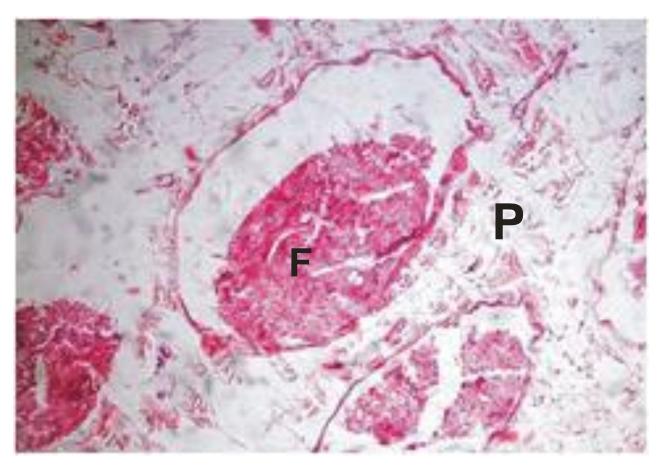


Figure 10. Photomicrograph of the transverse section of the Hamstrings muscle of a gilt infected with *T. brucei*. Note the degeneration of muscle fibres in the fascicule (F) with widened and edematous perimysial tissue (P). H and E X147.

muscles that were damaged during this infection. The low levels of this electrolyte in the skeletal muscles could have caused the decrease in neuromuscular excitability of the muscles.

The pathology in the hampstring muscles could have caused the posterior paresis and the wobbling that was observed in the infected gilts. However, whether this abnormality could be reversed in treated cases or how the trypanosomes could have caused this problem was not within the scope of this study. Further studies in these areas are therefore recommended.

REFERENCES

- Adah MJ, Otesile EB and Joshua RA (1992). Changes in level of transaminases in goats experimentally infected with *T. congolense*. Rev d' Elev. Me'd Vet. Pays Trop., 45 (3-4): 284-286.
- Allam L (2004). The effect of *Trypanosoma brucei* infection on reproduction in female pigs. MSc Thesis, Ahmadu Bello University Zaria.
- Allam L, Ogw u D, Agbede RIS and Sackey AKB (2006). Clinical and mating problems in gilts experimentally infected with *Trypanosoma brucei*. J. Trop. Bios., 6: 28-31.
- Anosa V0 (1983b). Mammalian blood cells in health and in

trypanosomiasis. Trop. Vet., 1: I77-199.

- Anosa V0 (1983a). Diseases produced by *Trypanosoma vivax* in ruminants, horses and rodents. Zent fur Vet B, 30 : 717-741.
- Anosa VO, Isoun TT (1980). Haematological studies on *Trypanosoma vivax* infection of goats and intact and splenectomised sheep. J. Comp. Path., 90: 155-168.
- Biryomumaisho S, Katunguka-Rw akishaya E, Rubaire-Akiiki CM (2003). Serum biochemical changes in experimental *Trypanosoma congolencse* and *Tryapanosoma brucei* infection in small east African goats. Vet. Arhiv., 73 (3): 167-180.
- Cano RC, Hliba E, Rubilo ER (2000). Creatine kinase and lactate dehydrogenase levels as potential indicators of *Trypanosoma cruzi* infectivity and histotropism in experimental Chagas' disease. Parasitol. Res. 86:244-252.
- Chaudhary ZI, Iqbal J (2000): Incidence, biochemical and haematological alterations induced by natural trypanosomosis in racing dromedary camels. Acta Trop., 77, 209-213.
- Coles EH (1986). Veterinary Clinical Pathology. Edition Published by W.B. Sounders Company Philadelphia, London, Toronto, Mexico City Rio de Janeiro, Sydney, Tokyo Hong Kong, 4: 1-486.
- Gray AR (1963). Serum transaminase levels in cattle and sheep infected with *Trypanosoma vivax*. Exp. Parasitol., 14: 374-381.
- Katunguka-Rw akishaya E (1996). The prevalence of trypanosomosis in small ruminants and pigs in a sleeping sickness endemic area of Buikw e country Mukono district, Uganda. Rev d' Elev. Me'd Vet. Pays Trop., 49: 56-58.
- Lording PM, Friend SCE (1991). Data analyasis guide. Interpretation of laboratory results. Austral. Vet. Pract., 21 (4): 186-195.

- Losos GJ (1986). Infectious Tropical Diseases of Domestic Animals. Longman Scientific and Technical, published in Association with the International Development Research Centre, Canada, pp. 182-318.
- Luckins AG (1992): Protozoal diseases of camels. Proceedings of the First International Camel Conference Dubai, United Arab Emirate, pp. 23-27.
- Mumah ET (1996). The Pathogenicity of *Trypanosoma brucei brucei* of Cattle origin in experimentally infected piglets. MSc thesis, Ahmadu Bello University Zaria.
- Omotainse SO, Ánosa VO, Falaye C (1994). Clinical and biochemical changes in experimental *Trypanosoma brucei* infection of dogs. Isrl J. Vet. Med., 49: 36-39.
- Onah DN, Uzoukw u M (1991). Porcine cerebral *Trypanosoma brucei* brucei trypanosomiasis. Trop. Anim. Health. Prod., 23 (1): 39-44.
- Otesile EB, Akpavie SO, Fagbemi BO, Ogunremi AO (1991). Pathogenicity of *Trypanosoma brucei brucei* in experimentally infected pigs. Rev d' Elev. Me'd Vet. Pays Trop., 44 (3): 279-282.
- Otesile EB, Fagbemi BO, Makinde MO, Akinboade OA (1992). The response of pigs experimentally infected with *T. brucei* to Isomethamedium® chloride therapy in relation to nutrition. Vet-Quart, 14 (3): 88-91.

- Seifert SHH (1996). Vector born disease: Trypanosomosis. In: *Tropical Animal Health*. Edited by Kluver Academic Publishers, 152-169.
- Sekoni VO (1994). Reproductive disorders caused by animal trypanosomosis. A Review Therio. 42: 557-570.
- Singla LD, Juyal PD (2000). Blood cellular responses in *Trypanosoma evansi* infected and levamisole treated bovine calves. J. Vet. Parasitol., 14: 137-139, 2000
- Taiwo VO, Olaniyi MO, Ogunsanmi AO (2003). Comparative plasma biochemical changes and susceptibility of erythrocytes to in vitro peroxidation during experimental *Trypanosoma congolense* and *Trypanosoma brucei* infections in sheep. Isrl J. Vet. Med., 58 (4): 1-10.