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Modulatory effects of ascorbic acid on the reproductive pathologic changes caused by experimental *Trypanosoma brucei* infection in Nigerian indigenous adult male *sus scrofa*

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Abstract

African animal trypanosomosis is a hemoparasitic disease caused by the protozoan parasite, trypanosoma. Tsetse flies (Glossina species) and biting flies transmit the parasite. Trypanosomosis infects humans as well as livestock.

The reproductive pathological changes and the effects of ascorbic acid were investigated in Nigerian indigenous boars experimentally infected with *Trypanosoma brucei*. A total of fifteen boars were used for the experiment and assigned into three groups of five animals each. Group I was infected with T. brucei and treated with ascorbic acid at the dosage of 150 mg per kg per os. Group II was infected with T. brucei only. Group III (uninfected control) were administered normal saline.

Acute trypanosomosis was observed following infection. The testes were harvested and the epididymal sperm reserves of all the boars determined. Also, the sperm quality (sperm motility, concentration, and morphology) was determined. Epidydimal sperm parameters were significantly low ($p < 0.05$) but abnormal morphologies were higher in the infected when compared to the control group but gradually improved following treatment with ascorbic acid. The low serum testosterone levels observed from day post infection (pi) gradually improved following treatment. The gross morphological studies of the testes of the boars shows testicular atrophy in the infected groups relative to the control and there was remission in the group treated with ascorbic acid. It was therefore concluded that infection of boars with T. brucei adversely affected testicular morphology and function. Treatment with ascorbic abrogated the reproductive abnormalities caused by the parasite.

Keywords: *T. brucei*; Spermatogenesis; Reproductive hormone; *Sus scrofa*

1. Introduction

The livestock and agricultural industries are Africa's two most important economic sectors. The livestock industry in West Africa is rapidly expanding, with projects aiming at enhancing production systems to meet rising food demand [1].

Pigs and small ruminants are reared for their socioeconomic advantages in sub-Saharan Africa. In addition to providing protein, dairy products, hide and skin for the tannery industries, and organic manure for crop production, they also serve as a source of income [2], [3], [4]. However, several factors like diseases including African Animal Trypanosomiasis

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(AAT) or “nagana” could limit the nutritional and economic value of small ruminants and pigs [5]. AAT is an infectious disease caused by protozoan parasites belonging to the genus Trypanosoma [6].

Nagana, Sura or AAT is arguably the most devastating disease of the domestic and wildlife animal that impacts production in sub-humid and humid regions of Africa [7] and it is caused by varying species of trypanosome including *T. equiperdum, T. simiae, T.uniforme* and *Trypanosoma congolense* and to a lesser extent *Trypanosoma brucei brucei, T. vivax* and *T. evansi,* affecting different species of domestic animals. However, in addition to the aforementioned trypanosomes, *T. simiae, T. savo*, and *T. theileri* have been identified in animals. *T. simiae, T. savo*, have been uncommon in ruminants but are widely recognized for their pathogenicity in domestic and wild pigs. [8].

Surra is an ATT caused by *Trypanosoma evansi* that affects livestock and has cosmopolitan distribution [9].

Trypanosomoses has a significant impact on livestock production, accounting for $4.5 billion in lost productivity. These economic losses are incurred by individuals in some of the world's poorest areas, particularly small-scale subsistence farmers and rural communities in Africa, who experience lower meat and milk yields as well as a decrease in draught animal production [10], impacting sub-Saharan Africa's economic growth of the livestock industries.

Livestock species affected by AAT includes cattle, sheep, dogs, donkeys, goats, camels, and pigs [11], Nevertheless, cattle are reported to be the most impacted domestic animal [12].

The disease is mainly transmitted by the bite of a hematophagous fly of the genus *Glossina* (tsetse fly) [13], which carry and transmit different species of trypanosome within the tsetse belts of Africa [14], mechanically transmission can occur within and outside tsetse fly-infested zones [15] essentially, alternative transmission methods consist of mammals as carriers (e.g., vampire bats for *T. evansi* and marsupials for *T. cruzi*), besides iatrogenic, transcutaneous, and transmembrane routes, in addition to oral, venereal, and intraplacental transmission, horizontal and vertical transmission can occur on occasion. and some species of trypanosome are transmitted sexually [16].

Clinical signs associated with AAT ranges from anaemia, immunosuppression, depression with inability to rise, pyrexia or fever, pale mucous membrane, rapid pulse rate, retarded growth, roughness of hair coats, enlargement of peripheral lymph nodes, low milk production, low meat quality, weight loss and reproductive disorders [17], [18], [19], [20], [21]. *T. vivax* or *T. congolense* infection in rams occasioned scrotal oedema, poor semen quality or the cessation of semen production, degeneration of the testes, loss of libido as well as infertility [22]; [23].

[19] reported that *T. brucei brucei* and *T. evansi* caused severe testicular degeneration with consequent depletion and absence of gonadal spermatid reserves in Yankasa rams.

Rabbits infected with *T. brucei brucei* developed increased scrotal circumference, scrotal inflammation, severe testicular degeneration, aberrant spermatogenesis, aspermatogenesis, and inadequate resolution of the genital organs [24]. The existence of *T. brucei brucei* in the male reproductive tract and the brain, in addition to severe lesions resulting in the degeneration of testes that included the Leydig cells, basement membrane, Sertoli, and germ cells, with concomitant loss of libido have been observed in boars [25]. Animals that survived infection often remained infertile [26].

Infection with *T. brucei* leads to weight loss due to the increased formation of free radicals, including superoxides, reactive oxygen species, and nitrogen species, and low levels of glutathione (GSH) in the host's cells and tissues. Free radicals can cleave sialic acids on erythrocyte membranes, causing anemia and severe organ damage [27]. Excessive free radical production can lead to lipid peroxidation and decreased levels of antioxidants such as vitamin C and ascorbic acid in the bloodstream [28]; [29].

Vitamin C and E have been shown to improve organ damage and anaemia severity in *T. brucei* infected animals [28]; [30]. However, there is dearth of information on the effect of *T. brucei* and ascorbic acid on the reproductive pathology of boars. Thus, this study was aimed at assessing the modulatory effects of ascorbic acid on the reproductive psathologic changes caused by experimental *T. brucei* infection in Nigerian indigenous adult male *Sus scrofa.* It is expected that the results of this study will assist in improving swine breeding and the maintenance of sexually active boars.

2. Materials and methods

2.1. Experimental animals

The experiment was approved by the Research Ethics Committee of the College of Veterinary medicine, Federal University of Agriculture, Makurdi, Benue State, and the study was conducted under protocols in the guidelines established by the "Guide for the Care and Use of Laboratory Animals" [31].

Fifteen (16) indigenous boars aged between 8 and 14 months and weighing 20 to 25kg were purchased from piggeries in Makudi metropolis and on arrival, the animals were housed in fly proof pens at the Teaching and Research Farm of the Department of Animal production, University of Agriculture, Makurdi. The boars were screened for the presence of ecto-, endo- and haemoparasites and thereafter were treated against helminthes and ectoparasites using ivermectin (ivomec® super) at a dose of 0.4mg/k*g* and the animals were ear tagged for identification.

The boars were fed on compounded balanced feed ration and water *ad- libitum* throughout one month of acclimatization during which the animals were subjected to physical examination, determination of body weight, rectal temperature, collection of blood samples for determination of baseline haematological and serum biochemical parameters and testosterone levels.

2.2. Source of trypanosomes

The *Trypanosome brucei* stabilate was obtained from Nigerian Institute for Trypanosomiasis Research (NITR), Vom, Plateau State, Nigeria. The parasites were inoculated into two rats and transported to the Veterinary Teaching Hospital (VTH), University of Agriculture Makurdi. At high parasitaemia, infected blood of the rat was collected and inoculated into a donor pig.

2.3. Inoculation of Donor Pig

T. brucei organisms were first detected in the rats 5 days post inoculation with the organisms. At peak parasitaemia when parasitic load was 1.8 X 10^6 trypanosomes per ml of blood, the rat was put on chloroform anaesthesia. Blood was obtained from the infected rats via the media canthus into an EDTA-containing sample bottle, which was used to inoculate the donor pig intraperitoneally (IP).

Following infection of the donor pig with blood containing 1.8 X 10^6 *T. brucei* organisms, 0.5 ml of blood from the donor pig was each time collected, and tested every day for the appearance of parasites in peripheral circulation. *T. brucei* organisms were first detected in the blood of the donor pig on day 5 post infection.

2.4. Experimental Design

The15 boars were randomly assigned to three groups of 5 animals each

- Group I: Boars were experimentally infected with *T. brucei* and treated with vitamin C at a dosage of 150mg/kg per os.
- Group II: Boars were infected with *T. brucei* only
- Group III: Served as the uninfected control, each boar received 2 mL normal saline.

2.5. Inoculation of experimental animals

When parasitaemia in the donor pig was 0.9 × 10^6 trypanosomes/ml 5 to 7 days post inoculation, blood was collected from the donor pig from the anterior vena cava into a blood sample bottle containing Ethylene Diamine Tetra-acetic Acid (EDTA) and diluted with cold physiological saline. The number of parasites in the diluted blood was determined through the method described by Herbert and Lumsden (1976) and a volume containing approximately 1.8 × 106 in 0.2 mL blood/phosphate buffered saline (PBS) of parasites was injected intraperitoneally into each rat in groups I and II.

2.6. Clinical Examination

Post inoculation of the animals in the infected groups I and II, they were clinically examined and their rectal temperatures and body weights monitored daily.

2.7. Measurement of rectal temperature

Rectal temperature was determined using a digital clinical thermometer.

2.8. Determination of weights of the Boars

Live body weight of all the animals in the groups was taken twice per week and throughout the experimental period with the aid of a portable weighing scale (CAMRY®, Germany) and were recorded in kilogram (kg)

2.9. Determination of level of parasitaemia

Parasitaemia was determined using blood film and buffy coat technique [33]. Parasitaemia levels were estimated using the rapid matching method of [32].

2.10. Orchidectomy

Orchidectomy was carried out using the procedure described by [34].

2.11. Preparation of serum samples for biochemical analysis

Exactly 5ml of blood was similarly collected from each animal into plane tubes without anticoagulant, weekly, and was allowed to stand and clot before being centrifuged at 3000 rpm. The serum was harvested and was stored in a freezer at – 20^{o}c until needed for the biochemical analysis.

2.12. Determination of serum testosterone

Serum testosterone concentration was assayed using DIA source$^{(R)}$ testosterone enzyme linked immunosorbent assay (ELISA) kit (DIA source Immuno Assay SA, lovvain-In-Neuve, Belgium) according to the manufacturer's instruction. Absorbance was measured at 450 nm using an ELISA reader (opys MR, DYNEX)

2.13. Semen Analysis

Semen evaluation was done at necropsy to determine morphological abnormalities of spermatozoa. Parameters examined included mid piece droplets (MPD), incidence of detached head (DH), bent tail (BT) and incidence of coiled tail (CT).

2.14. Sperm motility

Determination of cauda epididymal sperm motility was done using the method described by [35] and [36]. The individual motility was determined by the formula;

Motility

$$\frac{(Indivitdual)\ (\%) = Number\ of\ motile\ sperm \quad x\ 100}{Total\ no.of\ sperm\ (motile + immotile)}$$

2.15. Sperm concentration

Sperm count was determined using an improved Neubauer haemocytometer by the method described by [35]; [37].

2.16. Sperm morphologies

Sperm morphology was determined by examining air-dried slides under oil immersion as described by [35]. The abnormal spermatozoa were counted and the percentage calculated.

2.17. Data Analysis

The data obtained were prepared for analysis using Microsoft office Excel (2010) and expressed as mean ± SD, while analysis of variance (ANOVA) was carried out using statistical package for the social sciences (SPSS) for windows, version 20 (2011). The values of $P < 0.05$ were considered statistically significant.

3. Results and Discussion

3.1. Parasitaemia in Infected Pigs.

After infection of boars on day 0, parasites were first detected in peripheral blood of the boars in group II on day 9 post infection (PI) (+) and those in group I by day 11 PI (+). This was followed by a fluctuating increase in parasitaemia with peak levels of (+++) in group I and (++++) in group II at week 5, after which there was a fluctuating drop in the level of parasitaemia as both infected groups had values of (+) each at week 7, which remained low until termination of the experiment. Generally, the mean parasitaemia was lower in group I that was administered ascorbic acid when compared to that of group II which was untreated.

3.2. Clinical changes in *T. brucei*-infected boars

3.2.1. Variation in body temperature:

On day 0 of infection, the mean rectal temperatures of the experimental animals were in the range of 39 ± 0. 1 °c to 38.9 ± 0.1 °c in groups I, II and III respectively. By week 2, there was a sharp increase in the mean body temperature observed in groups I and II to 38.9 ± 0.9 and 39.6 ± 0.6 °c respectively and was followed by an undulating increase, characterized by peak values of 39.7 ± 0.1 and 39.9 ± 0.3 °c in groups I and II respectively (fig 1) by week 6 after which the temperatures dropped to normal until the end of the experiment. The mean rectal temperatures of the control group varied within the normal range throughout the course of the experiment. However, group II had the highest changes in temperature with a mean value of 39.19 ± 0.1 while group I had a mean value of 39.19 ± 0.1.

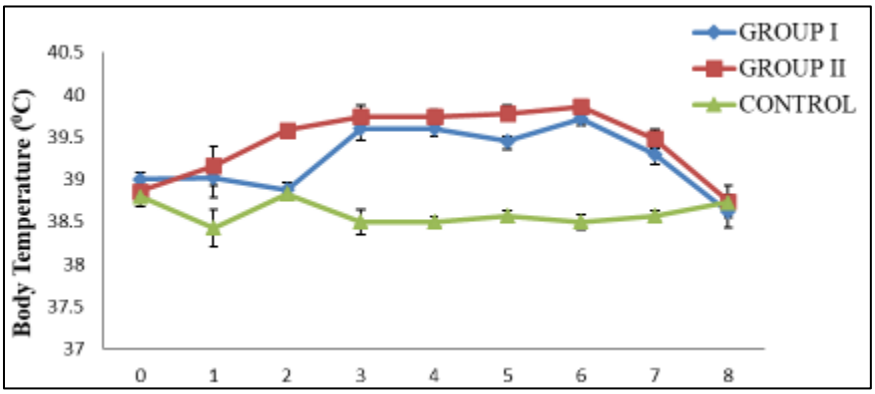


Figure 1 Mean ± SD Mean Body Temperature in the *Trypanosome brucei* Infected Groups I and II and uninfected control Group

3.3. Determination of semen quality

The semen motility values in groups I, II and III (uninfected control) was 60, 20 and 75 % respectively. Semen concentration was 32.8, 8.4 and 42.3 × 10^6 /ml (cells) in *Trypanosome brucei* infected groups I and II and III boars respectively. Detached head values were 43, 44 and 34 % in groups I, II and uninfected control respectively. Coiled tail values were 19, 16 and 17 % in groups I, II and III respectively while mid piece droplets were 38, 40 and 49 % in groups I, II and III respectively. Acrosome Integrity: Intact acrosome value was 65, 38 and 68 % in groups I, II and III respectively while non intact acrosome values were 35, 62 and 32 % in groups I, II and III respectively. On the whole, group II had the highest values of semen morphological abnormalities while group I showed lower value of abnormalities.

Table 1 Semen analysis for morphological abnormalities in Experimental animals

S/N	Motility%	Concentration × 10^6/ml	Morphology %		
			Detached head	Coiled tail	Mid-piece droplet
Group I	60	32.8 cells	43	19	38
Group II	20	8.4 cells	44	16	40
Group III	75	42.3 cells	34	17	49

Key: Group I: Boars infected with *Trypanosoma brucei* and treated with ascorbic acid, group II: Boars infected with *Trypanosoma brucei* only, group III: Served as the uninfected control, each boar received 2 mL normal saline.

Table 2 Variation in acrosome integrity in the experimental Boars

Groups	Intact %	Non-Intact %
Group I	65	35
Group II	38	62
Group III	68	32

Key: Group I: Boars infected with *Trypanosoma brucei* and ascorbic acid. Group II: Boars infected with *Trypanosoma brucei* only. Group III: Served as the uninfected control, each boar received 2 mL normal saline

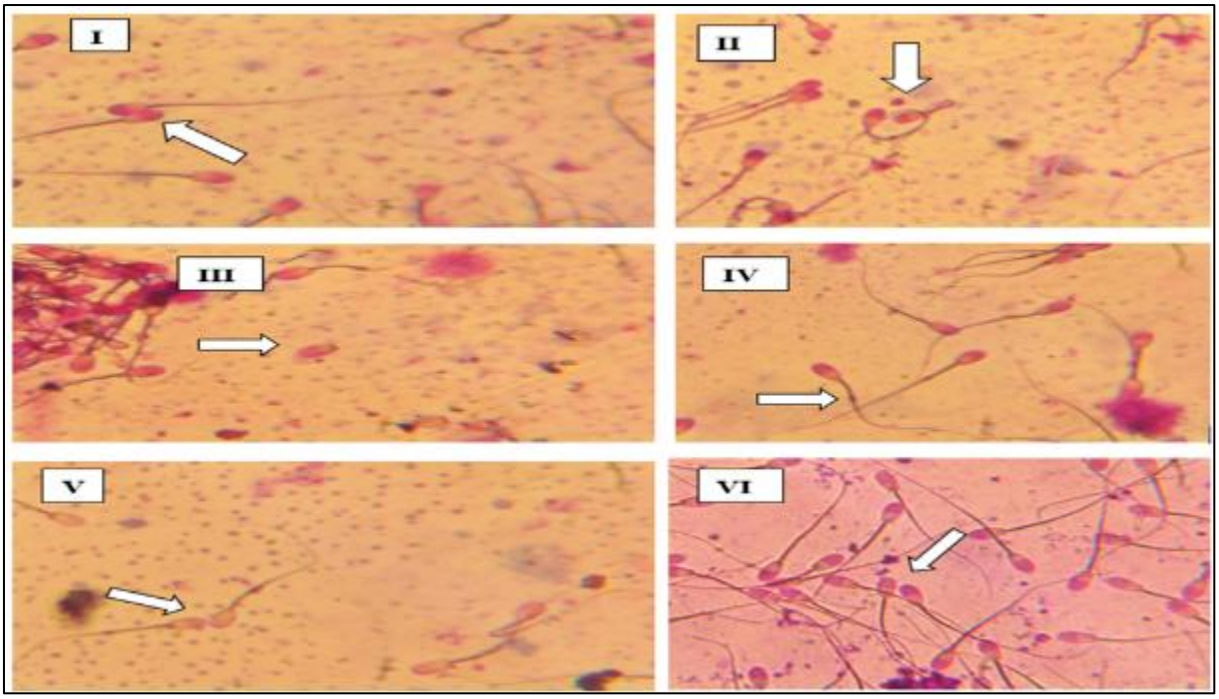
3.4. Variation in Serum Testosterone Levels (ng/dl):

The mean serum testosterone levels in the groups were 671.8 ± 42.08, 364.6 ± 46.78 and 955 ± 49.05 ng/dl in groups I, II and control, respectively. On the whole, group I had a higher mean testosterone value while group II showed the lowest values. However, the control group showed highest mean testosterone level compared to the infected groups.

Table 3 Variation in Serum Testosterone Levels in the *Trypanosome bruce*i Infected Groups I and II and Control Groups.

Experimental Animal Groupings	Serum Testosterone Level (ng/dl)
Group I	671.8±42.08
Group II	364.6±46.78
Group III	955±49.05

Key: Group I: boars infected with *T. brucei* and treated with ascorbic acid, group II: boars infected with *T. brucei* alone, group III: Served as the uninfected control, each boar received 2 mL normal saline

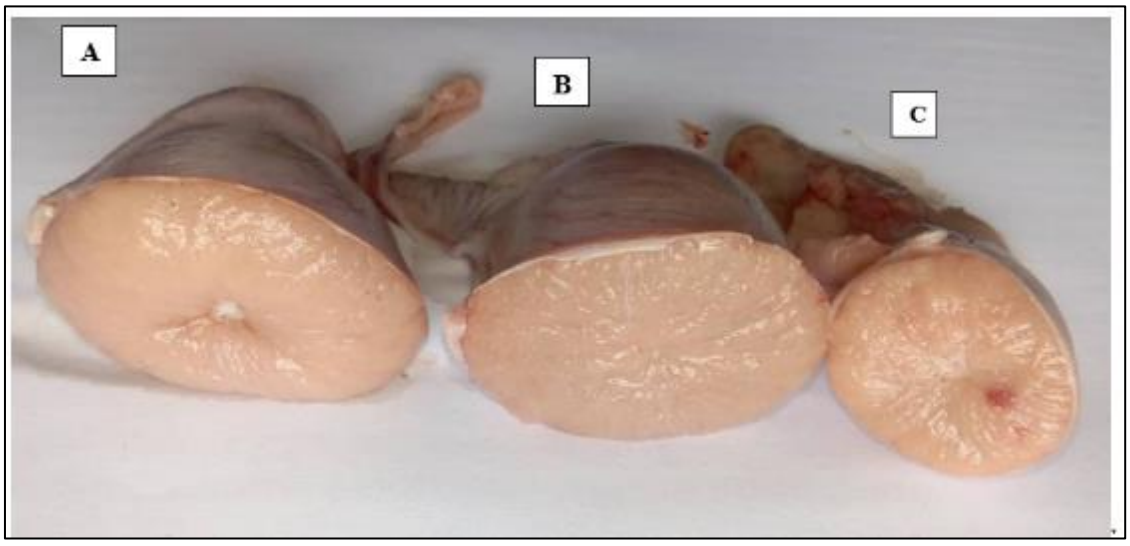


Note: Plate I: Micrograph showing intact acrosome in the semen of a boar from group I, **Plate II**: Micrograph showing coiled tail sperm cell in semen of a boar from group II, **Plate III**: Micrograph showing a boar's detached head sperm cell in group II, **Plate IV**: Micrograph showing a boar's sperm cell with mid piece droplet in group II, **Plate V**: Micrograph showing intact sperm cell in semen of a boar from group III, **Plate VI**: Micrograph Showing normal sperm cell in Semen of a boar from group III

Figure 2 Morphological Characteristics of Boar Spermatozoa Across Experimental Groups

3.5. Gross pathological lesions

Plate VIII. Photograph of testicles showing variation in testicular sizes in groups with group II been markedly degenerated.



Key: Note: Plate A: Shows normal testicular size of Served as the uninfected control, each boarreceived 2 mL normal saline y, B: Shows almost normal testicular size of Boars that was innoculated with *T. brucei* and ascorbic acid, C: Shows atrophied testicle with petechiation from a boar inoculated with *T. brucei* only.

Figure 3 Gross Pathological Comparison of Boar Testes under Different Treatment Regimes

4. Discussion

Trypanosoma brucei infection is associated with significant perturbations of the host internal meliu [38]. But evidence in the recent past has revealed that fortification of body systems with natural antioxidants, such as ascorbic acid, may correct the vitiated homeostasis arising from oxidative stress [39]. The pattern of parasitaemia demonstrated in the *T. brucei* in the infected boars was characterized by undulating parasitemia, intermittent pyrexia. This is in agreement with previous reports in a similar study with sheep infected with T. brucei [40]; *T. vivax* [41] and *T. evansi* [42]. It is also in agreement with the reports of goats infected with *T. congolense* [43] and cattle [44]. The pre-patent periods of *T. brucei* in the present study were found to be shorter in boars in group II inoculated with the parasite only. The shorter pre-patent period observed in the present study agrees with similar findings of [45] but at in variance with the reports of [46]. Howevere, we recorded a longer prepatent period in the group inoculated with the parasite and treated with ascorbic acid relative to the group inoculated *T. brucei* alone. Report has it that undulating parasitemia is believe to be caused by the ability of the parasites to change it antigenic coat during each wave of parasitemia [47], However, in this present study, the longer pre-patent period of the infection with *T. brucei* in the boars treated with ascorbic acid in this study may be due to the anti-oxidant activity of the ascorbic acid which may have conferred an improved immunity to the host against the trypanosomal infection pattern. Ascorbic acid supplement is a potent antioxidant that strengthens body's natural defenses [48].

Reproductive organs are often affected during trypanosomiasis in both male and female animals [49]. Male reproductive organs consist of different compartments including the testes and epididymis. Epididymis and testis are circumscribed by adipose tissue, the epididymal adipose tissue, which is an extension of the gonadal adipose tissue, a major reservoir of *T*. *brucei* parasites [50].

The findings of this study revealed the destructive effects of *T. brucei* infection on the testicular morphology and function. Our research group observed that measuring hormonal level in infected boars revealed a considerable drop in testosterone levels relative to the control group. These results are similar to that of a study by [51]. However, there a significant increase in the hormonal level in the group treated with ascorbic acid.

Similarly, inoculation of boars with *T. brucei* resulted in significant decrease sperm motility, concentration, and increase in the number of abnormal morphologies (detached head, mid-piece and coiled tail) relative to intact sperm cells in the uninfected groups; there was a significant, however co-administration with ascorbic acid enhances sperm motility, count, viability, concentration and ensued decreased sperm abnormalities.

The damaging effects of *T. brucei* on the spermatozoa are in agreement with the findings of [52]. Decrease in these reproductive parameters suggest damages to some reproductive tissues, especial where sperm production occurs, resulting in the degeneration of seminiferous tubules and the disappearance of spermatozoa, spermatids, and spermatocytes. This agrees with earlier reports in boar [53], bulls [54] gazelles [55], and sheep and goat infected with *T. vivax* or *T. congolense* [56]. But these findings were in contrast to that of [57] and [58] following infection of bulls with *T. vivax* and *T. congolense*. However, it was interesting to note that treatment with ascorbic acid ameliorated the deleterious effects induced by the *T. brucei*. Safeguarding organ damage by vitamin C is grounded on its antioxidant properties and Prior researchers have hypothesized that vitamins can lower the risk of certain infections via a variety of mechanisms [59].

5. Conclusion

Based on the findings of this study, we concluded that trypanosomosis-infected boars are infecund and should not be used for breeding or insemination. However, with proper treatment and sexual rest, the swine can recover and are used for insemination and breeding purposes.

Compliance with ethical standards

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Staff of the Clinical Pathology Laboratory of the Veterinary Teaching Hospital (VTH), Livestock Superintendents of the VTH, all of Joseph Sarwuan Tarka University, Makurdi, Benue State, Nigeria.

Disclosure of conflict of interest

The Authors declare no conflict of interest.

Statement of ethical approval

All procedures carried out on animals in this study were approved and carried out in accordance with the standards of the Ethical Committee of the College of Veterinary Medicine, Joseph Sarwuan Tarka University, Makurdi, Benue State, Nigeria.

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