Pathological Changes in the Genitourinary Tracts of Rabbit Bucks Experimentally Infected with *Trypanosoma brucei brucei*

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Abstract

*Samples for histological studies were taken from the genitalia of eight domesticated adult rabbit bucks (four infected with *T. brucei brucei* and four uninfected control), the rabbits were sacrificed and necropsied 13 days post infection and 29 days post infection. Infection with *T. brucei brucei* caused various grades of lesions in the male reproductive organs, especially the testes and epididymides. Histopathology of the testis and epididymis of the infected rabbit bucks showed empty seminiferous tubules with cessation of spermatogenesis as no viable spermatozoa was seen. There was massive infiltration of inflammatory cells (neutrophils with lymphocytes and other mononuclear cells) in the testis and epididymis indicative of orchitis and epididymitis. This study has shown that trypanosome infection causes either serious infertility or even sterility in rabbit bucks.*

Key words: Rabbit Bucks, *Trypanosoma brucei brucei* Infection, Parasitaemia, Testis and Epididymis


Introduction

The rabbit (*Oryctolagus cuniculus*) possess a number of features that might be of advantage in a small holder subsistence–type integrated farming in developing countries. Rabbit (*Oryctolagus cuniculus*) production could be of advantage in alleviating animal protein deficiency in affected countries such as Nigeria (Abdulmalik, 1994; Hassan and Owolabi, 1996; Ajala and Balogun, 2004).

Trypanosomosis is a group of protozoan infections of both man and animals caused by trypanosomes. The most common pathogenic species of trypanosomes in animals have been identified to consist of *Trypanosoma brucei, T. vivax, T. congolense, T. simiae and T. evansi* (Sekoni, 1994; Pepin and Meda, 2001; Kamuanga, 2003; Bawa et al., 2005; Courtin et al., 2008).
Trypanosomes cause reproductive impairment by their ability to cause pathological changes in the endocrine glands and gonads (Ikede, 1979; Sekoni et al., 1990; Sekoni, 1992; 1994). Other reproductive pathologies associated with trypanosomosis in both man and animals include irregular oestrus, sterility, uterine infections and abortions in the female (Reincke et al., 1998). It has also been reported that testicular degeneration and aspermatogenesis and other sperm abnormalities could occur in the male (Akpavie et al., 1986; Sekoni et al., 1990; Sekoni, 1993; Omer et al., 1998; Al-Qarawi et al., 2004). Some studies have shown that following treatment of trypanosomosis affected animals, regeneration of seminiferous epithelium could occur at a rate depended on the severity of the initial lesions (Ikede and Akpavie, 1982; Sekoni, 1990; Al-Qarawi et al., 2004; Kaufmann, 2005).

The degenerative changes caused by trypanosomosis in the spermatogenic epithelium and the interstitial cells of Leydig may disrupt steroidogenesis and cause reduced libido in infected animals (Sekoni et al., 1988). Previous report in rabbits observed increased scrotal diameters, scrotal inflammations, scab formation with alopecia as well as peri orchitis, epididymitis, severe testicular degenerations and abnormal spermatogenesis in male camels, sheep, goats and cattle (Sekoni et al., 1991; Sekoni., 1992; Omer et al., 1998; Al-Qarawi et al., 2004) and preputial haemorrhages have been reported (Ikede and Akpavie, 1982) in rabbits infected with \textit{T. brucei}.

**Objective**

The objective of this work was to determine the pathological changes of the genitourinary tracts of rabbit bucks experimentally infected with \textit{Trypanosoma brucei brucei}

**Materials and Methods**

**Acquisition of Experimental Animals**

Twenty (20) domesticated adult Dutch cross rabbit bucks weighing an average of 2.0 ± 0.8 kg were acquired from a rabbitry within Zaria metropolis of Kaduna State.

**Management of Experimental Animals**

The rabbit bucks were kept in individual fly proof cages and given access to growers mash and water was provided \textit{ad libitum}. The rabbit bucks were allowed to acclimatize for 14 days. They were dewormed with Levamisole HCl (0.1mg/kg) and treated for other diseases such as \textit{Eimeria staedia} with Embazine © forte containing sulphaquinoxaline B.P 9.4 g, diaveridine B.P.V. 0.98 g and vitamin k 0.053 g at a dose of 1g/5kg before the commencement of the experiment. The rabbit bucks were randomly assigned into two groups; control and infected groups consisting of ten rabbit bucks respectively.

All institutional and national guidelines for the care and use of laboratory animals were strictly observed.
Infection procedures

Stabilates of *T. brucei brucei* were acquired from the Department of Parasitology and Entomology of the Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, who also sourced it from National Institute for Trypanosomosis Research, Kaduna state. Before infecting the rabbits, the trypanosomes were maintained by serial syringe passages in white rats, and periodically checked for the viability of the parasite. Blood was obtained from the passaged rats by tail bleeding into normal saline and the parasitaemia adjusted to $1 \times 10^6$ trypanosomes per milliliter (ml) by the method of Herbert and Lumsden, 1976. Each rabbit in Group B was inoculated intraperitoneally with 1ml of saline diluted blood containing $1 \times 10^6$ trypanosomes *T. brucei brucei*, while Group A rabbits served as uninfected control.

Physical Examination

The general body condition of the infected rabbit bucks especially of the reproductive organs (testis and epididymis) was conducted and observations recorded. Scrotal diameters of the rabbit bucks were done with the use of the measuring tape, and results recorded.

Histopathological Examination

A total of eight rabbits comprising two rabbits from each of the groups were sacrificed 13 days post infection and 29 days post infection. The sacrificed rabbits were necropsied immediately and samples from the testis and epididymis were immersed in 10 % formalin dehydrated in graded concentration of absolute alcohol and xylene and embedded in paraffin. Thin sections (5 µ) mounted on a clean glass slide were stained routinely for histopathological examination by light microscopy using haematoxylin and eosin (H & E) standard protocols.

Results and Discussion

Gross pathological lesions observed were scrotal dermatitis, orchitis and balanoposthitis 13 days post infection, increased scrotal diameters, scrotal necrosis and excessive thickening of the scrotal corneum (SC) of the left side 29 days post infection. Also there was inflammation and haemorrhages at 29 days post infection as illustrated on plates I and II.

The effects of trypanosomosis on the testis and epididymis of rabbit bucks are illustrated on plate III.
Gross pathology

Plate I: Photographs of the genitourinary tract and penile region. Note: A, Normal scrotal skin (arrow) in the normal control buck (NSS), B, Scrotal dermatitis/orchitis (arrow) and balanoposthitis (BP) at 13 days post infection with *T. brucei brucei*, C, Increased scrotal diameters (L & R, arrow), scrotal necrosis and excessive thickening of the scrotal corneum (SC) of the left side 29 days post infection and D, balanoposthitis (BP) at 29 days post infection with *T. brucei brucei*. 

**NSS**

**BP**

**L**

**R**

**SC**

**BP**
Plate II: Photograph of the testes and epididymis of a buck showing inflammation and haemorrhages (arrow) at 29 days post infection with *T. brucei brucei*.

**Histopathology**

Plate III: Photomicrographs of the epididymis (A & B) HE, ×200, testis (C) of a *T. brucei brucei* infected and testis (D) of non-infected rabbit bucks HE, ×400. Note. A, neutrophils with mononuclear infiltrations around the empty epididymal lumen (arrow), B, areas of necrosis (Arrow), HE, ×400 C, necrotized seminiferous tubules (arrow) in the *T. brucei brucei* infected bucks, HE, ×400 and D, the intact seminiferous tubules with normal spermatogenic activity in the uninfected controls (H and E stains X 400).
There were genital lesions within 13 - 29 days post infection such as alopecia, scrotal dermatitis/orchitis, balanoposthitis, increased scrotal diameters, scrotal necrosis, excessive thickening of the scrotal corneum, periorchitis, epididymitis, severe testicular degeneration and haemorrhage which agrees with reports by Isoun and Anosa, 1974 and Anosa and Isoun, 1980.

Histological sections showed neutrophils with mononuclear infiltrations around empty existing lumen during the early stage of the infection and as the infection progressed, and necrotized seminiferous tubules with lymphocytic infiltrations at the later stage of the infection.

There are direct and indirect detrimental effects of trypanosomosis on reproduction in male and female animals. It is well established that most tissues and organs are damaged during the course of infection (Morrison et al., 1981).

The reproductive system which is controlled by a well co-ordinated and efficient neuro-endocrine system (Nalbandov, 1976), has been reported in literature to be affected in both natural and experimental trypanosome infections (Apted; Isoun and Anosa, 1974, Ikede et al., 1988). The hormones involved in reproduction originate in three principal structures which are the hypothalamus, the pituitary and the gonads. Any detrimental effect on any of the organs would result in detrimental effects on reproduction. It has been well established that severe degenerative changes of the interstitial cells of leydig within the testes takes place, and this cells are responsible for the production of testosterone, which is the hormone responsible for libido, anabolic effects and secondary male characteristics (Sekoni, 1991). Therefore, poor libido or lack of libido in all male species with trypanosomosis is a possibility, which can be supported by the observations of Mulligan et al. (1970) who reported that chronic T. congolense infections in cattle may lead to loss of libido, interference with reproduction and delayed puberty in calves. Waindi et al., (1986) found low levels of plasma testosterone in goats infected with T. congolense.

Hoogenboezem and Swanepoel, (2001), reported that testicular degeneration might be due to nutritional deficiencies and management related factors. Degenerative changes in the seminiferous tubules, testicular degeneration, low semen output, low spermatozoa concentration, high percentage of dead spermatozoa and spermatozoa abnormalities have also been reported to be caused by heat stress (Kumi-Diaka et al.,1981, Sekoni et al.,1991, Mamabolo, 1999, Hoogenboezem and Swanepoel, 2001).

Some pathogenic effects on the male reproductive systems have been reported. The reduced libido, increased scrotal diameters, scrotal inflammations, alopecia, periorchitis, severe testicular degenerations, balanitis, abnormal spermatogenesis in the infected rabbit bucks agrees with the findings of trypanosoma infected animals as reported by Ikede and Akpavie, 1982; Sekoni et al., 1991; Sekoni 1992, which may be influenced by the effect of trypanosomes on the testis affecting the leydig cell steroidogenesis (Sekoni et
through the indirect effects of pyrexia and the accompanying waves of increasing parasitaemia during infection and increased scrotal temperatures (Mutayoba et al., 1995b).

Conclusion

This study has indicated that *T. brucei brucei* is pathogenic to rabbit bucks. It reveals that *T. brucei brucei* may be a threat to the reproductive performance of rabbit bucks and could be detrimental to the efforts to increase animal protein, and the socio-economic wellbeing of the tsetse endemic areas in the country where rabbit farming is important.

The study also showed a need of considering trypanosomosis as a differential diagnosis when previously productive rabbit bucks suddenly have a low libido and develop serious reproductive anomalies especially in tsetse endemic areas.

References