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REPRODUCTIVE ORGAN DAMAGE OF DOMESTIC RUMINANTS IN AFRICAN ANIMAL TRYPANOSOMIASIS: A REVIEW

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ABSTRACT

Animal African trypanosomiasis affects both production and reproduction in domestic ruminants. The reproductive damages caused by trypanosomes have direct or indirect link to some specific organs like endocrine and pituitary gland which secrets follicle stimulating hormone (FSH), interstitial-cell stimulating hormone (ICSH) and growth hormone (GH). These hormones play an important role in the spermatogenic cycle in males and oestrus cycle in females. However, the pathogenesis of these damages have not been clearly elucidated. We believe that this aspect deserves closer study especially in livestock where reproductive performance is the pillar upon which productivity is built. It is hoped that this review which is centred on the chronological report of works that have been carried out on some reproductive organs damage in domestic ruminants infected with or affected by trypanosomes will add more light to the pathophysiology of the affected organs.

Keywords: Trypanosomiasis, Pituitary gland, Endocrine, Ruminant, Spermatogenesis, Oestrus cycle.

INTRODUCTION

African trypanosomiasis (AT) is a disease peculiar to sub-Saharan Africa which affects both human (sleeping sickness) and animals (*Nagana* or *Sammoré* in cattle). The disease affects virtually every organ where it causes anaemia at the first stage of infection and death on the long run (Rodriguez *et al*., 2012). Trypanosome is transmitted cyclically through biting of tsetse fly (*Glossina* spp.) and remains one of the major infections limiting the animal growth and livestock industry in Africa (OIE, 2013). *Trypanosoma brucei brucei*, *T. congolense*, and *T. vivax* are the major pathogenic trypanosomes peculiar to animals Desquesnes, (2004); even though there exist other causative agents like *T. suis*, *T. equiperdum* and *T. evansi* that affects such domestic animals like pigs, horses and camels, respectively. AT is characterised by anaemia, anorexia, depression, increased respiratory and heart rates, nasal discharge, subcutaneous oedema, intermittent fever, enlargement of lymph nodes, central nervous system and reproductive disorders (Bentivoglio *et al*., 1994; Desquesnes, 2004). Considering trypanosome effect on reproductive disorders in male ruminants, reports have shown that it can cause severe testicular degeneration, scrotal inflammation, penile protrusion, prepucial inflammation, epididymitis, abnormal spermatogenesis and deterioration of semen characteristics (Sekoni, *et al*., 1990; Adamu *et al*., 2007; Victor *et al*., 2012).

In female ruminants, Faye *et al*. (2004) and Silva *et al.* (2013) documented that the disease causes irregular oestrus cycle, cystic degeneration of the ovary, follicular cyst, decreased conception rate, abortion, low birth weight and neonatal death. In view of these, an estimated 50 million heads of cattle are at risk, and in a situation where the disease is tolerable, up to 50% cases of mortality and morbidity are recorded in animals and consume a sum of US$600 million to US$1.2 billion annually in Africa (FAO, 1994; Maudlin, 2006). This threat has been noticed to hinder food security globally (Samdi *et al*., 2010).

These reproductive disorders have been recognised in animal African trypanosomiasis since the early part of 19th century (Ikede *et al*., 1988). It is our strong believe that this aspect deserves closer look especially in livestock where reproductive performance is the pillar of upon which productivity is built. It is hoped that this review which is centred on the chronological report of works that has been carried out on some reproductive

organs damage in domestic ruminants infected with African trypanosomiasis will add more light to the pathophysiology of these organs.

ENDOCRINE SYSTEM

The endocrine system plays a central role in the regulation of most body functions such as growth, differentiation, reproduction, maintenance of the internal environment, and adaptation to changes in the external environment. Its involvement in reproduction has direct link to hypothalamus and anterior pituitary (Senger, 2005). The gonadotropin releasing hormone (GnRH) produced from the hypothalamus induces release of gonadotropin, follicle stimulating hormone (FSH) and luteinising hormone (LH) from the anterior pituitary. FSH and LH are required for spermatogenesis and sperm maturation, while development of male secondary characteristics and libido depends on the testosterone (Steinberger and Duckett, 1967; (Khisk, 2008). *T. congolense* and *T. vivax* are known to be intravascular and non-tissue invasive which is capable of affecting endocrine system, and the mechanism of these endocrine lesions has been postulated on the ability of the trypanosomes to localize in the organs thereby causing severe damage (Raheem, 2014).

PITUITARY GLAND

Pituitary hormones (gonadotropins) play a major role in the reproductive process. In particular, follicle stimulating hormone (FSH), interstitial-cell stimulating hormone (ICSH) and growth hormone (GH) play an important part in the spermatogenic cycle in males and oestrus cycle in females. Consequently, absence of pituitary gonadotropins leads to a block in sperm maturation in rats (Clermont and Morgentaler, 1955) or to marked testicular degeneration and abortion in man and animals (Apted, 1970; Ikede and Losos, 1975; Raheem, 2014). The pituitary gland, which is connected to the central nervous system (CNS) through the hypothalamus, is one of the endocrine organs affected by trypanosomiasis. Ikede (1979) reported focal coagulative necrosis and interstitial mononuclear infiltration in pituitaries of sheep experimentally infected with *T. brucei*, these lesions were associated with extra-vascular localization of trypanosomes in the pituitary gland. Studies on infected domestic and laboratory animals have provided more evidence of specific damage to the pituitary gland (Ikede *et al*., 1988). Extensive mononuclear inflammation of the gland has been described in cattle, sheep and goats experimentally infected with *T. brucei* (Losos and Ikede, 1970, 1972; Ikede *et al.*, 1977; Moulton and Sollod, 1976; Morrison *et al.*, 1981). Abebe *et al.* (1993) reported that the vessels of the microvasculature in the pituitary glands of infected animals with *T. Congolense* were highly distended with trypanosomes, erythrocytes, macrophages, cellular debris, pituitary cell secretory granules, and microvasculature dilation in skeletal muscle, myocardium, and brain. These effects directly reduce the availability of necessary hormones required during mating; thus hindering normal reproductive processes in ruminants. Extensive dilation of the pituitary microvasculature could lead to pooling of blood in the capillary beds, affecting the efficiency of circulation through the capillaries and in turn possibly causing the impairment of normal nutrient and metabolite exchange, leading to cellular apoptosis (Losos and Ikede, 1972; Wellde *et al*., 1989; Nyeko *et al*., 1990). Reincke *et al.* (1998) reported that animal African trypanosomiasis caused local inflammation of the pituitary and the gonads, associated with increase in the level of cytokines such as tissue necrotic factor α (TNF- α) and interleukins 6 (IL-6). Studies on hormonal assay under trypanosomiasis infection revealed decline in the level of Luteinsing hormone and testosterone in ruminants (Waindi *et al.*, 1986; Boly *et al.*, 1994; Mutayoba *et al.*, 1994).

FEMALE REPRODUCTIVE ORGANS

Ovary is the primary female reproductive organ and has two important functions: producing the female reproductive cell (the egg or ovum) and producing the hormones estrogen and progesterone. The secondary sex organs are a series of tubes that receive semen, transport sperm to the egg so it can be fertilized, nourish the fertilized egg (embryo), and allow the calf to be birthed. These organs include the vagina, cervix, uterus, uterine horns, and oviducts (also called Fallopian tubes), which each has a funnel-shaped opening called the infundibulum. The released egg is caught by the infundibulum and moves into the oviduct, where fertilization occurs if viable sperm are present. The egg remains capable of fertilization for only a few hours. Thus, it is very important that fertile sperm be present near the time of ovulation. The egg moves through the oviduct and into the uterus within the next three to four days. Following the trend of embryological development, any alteration may terminate the process and thereby causes infertility in ruminants.

Histopathological examination of *Trypanosoma brucei* infected West African dwarf does by Leigh *et al*. (2014) revealed sub-acute necrotising adenohypophysitis characterized by widespread necrosis and disruption of the architecture of the adenohypophysis. Multiple foci of inflammatory cells which were mostly lymphocytes and a few neutrophils were also observed in the pituitary gland. Also, the hypothalamus of the infected does showed widespread congestion of blood vessels with severe perivascular lymphocytic cuffs meanwhile uterus showed acute severe endometritis characterized by marked extensive necrosis of the epithelium with varying degrees of necrosis and distortion of the tubular glands in the endometrial stroma. Acute placentitis characterized by marked vascular congestion and

multifocal lymphocytic aggregates was also observed in infected pregnant does. The lesion observed in the uterine body, a severe necrotising endometritis, is capable of reducing its potential to carry out its physiological functions which include the production of prostaglandin $PGF_{2\alpha}$ (i.e. luteolysin) and nidation (Leigh and Fayemi, 2013). *T. vivax* causes lesions in the placenta of Ewe (Silva *et al.,* 2013). These lesions are capable of interfering with normal exchanges between foetus and dam, leading to foetal hypoxia and death *in utero* or the initiation of early dehiscence of the placenta and subsequent abortion (Dubey *et al.,* 2006). *T. brucei* possesses the ability to traverse the placenta causing placentitis and also infection *in utero* which may be linked to cord atrophy due to impaired nutritional and gaseous exchanges between the dams (Marcato *et al.,* 1991; Rocha *et al.,* 2004). Adenowo *et al*. (2005) observed inflammatory responses of hypothalamus and adenohypophysis when examining histological changes in *T. vivax* infected sheep. Leigh and Fayemi (2013) reported reduction in the concentrations of follicle stimulating hormone during oestrus, and luteinizing hormone during pro-oestrus and oestrus, as well as oestradiol during the follicular phase in *T. brucei* infected West African Dwarf does.

MALE REPRODUCTIVE ORGANS

Trypanosome infection has been reported to cause reproductive dysfunction both in male and female ruminants. In males, it has been documented that it causes decline in libido, and changes in semen characteristics and testicular pathology are among the reported abnormalities observed in trypanosome infection.

Libido is a critical aspect of male sexual function because it indicates the ability of the male to detect and subsequently service the female animal on oestrus (Farin *et al.,* 1989). Sekoni *et al*. (2004) established a reduction in libido in bull, due to trypanosomosis infection. The same was also reported for West African dwarf buck experimentally challenged with *Trypanosoma congolense* (Raheem *et al.,* 2009). The mechanism of causing decline in libido has been premised on decline in plasma testosterone (Adamu *et al*., 2006) which has been reported in several studies. Testosterone plays an important role in optimal functioning of the testis and initiation of sex drive (Senger, 2005). Testosterone concentration is an indicator of the level of libido expressed by the bull (Nix *et al.,* 1998). Therefore, when its testicular production is impaired, the clinical manifestation is reduced libido.

Inflammatory changes in the genital organs are usually mild or absent but there is progressive and marked testicular degeneration that can lead to atrophy and aspermia (Isoun and Anosa, 1974; Isoun *et al*., 1975; Anosa and Isoun, 1980; Kaaya and Oduor-Okelo, 1980; Masake, 1980; Anosa, 1983). The cause of the testicular lesion is believed to be due to the effect of prolonged fever, thrombosis of spermatic blood vessels, and the general wasting of body organs (Anosa and Isoun, 1980; Anosa, 1983). In male animals infected with the *brucei* group, the lesions are a combination of scrotal dermatitis, orchitis and peri-orchitis. Soon after infection the parasites localize in the scrotal skin and hydrocoel fluid and also invade the tunica vaginalis, testis, epididymis and spermatic cord, provoking a non-purulent granulomatous inflammation in sheep (Ikede, 1979).

The severity of testicular and epididymal lesions is reflected in poor quality of semen and the high percentage of abnormal spermatozoa present in the ejaculate of bulls (Isoun *et a*l., 1975; Grundler, 1985) and rams (Isoun and Anosa, 1974; Anosa and Isoun, 1980; Akpavie *et al*., 1987) experimentally infected with *T. vivax. T. congolense* or *T. brucei*. It has also been shown in rabbits and sheep that successful treatment with trypanocides will lead gradually to abnormal spermatogenesis over a period of several months if the original lesions have not been complicated by secondary bacterial infections (Ikede and Akpavie, 1982; Akpavie *et al.*, 1987).

Sekoni *et al*. (1990) revealed the scrotal oedema and testicular degeneration characterized by degenerated spermatogenic cells in bull infected with *T. congolense*. Scrotal oedema can also occur as a result of inflammation caused by the parasite (Rakha *et al*., 2006). Okubanjo *et al*. (2014) reported reduction in semen concentration and increase in percentage sperm abnormalities of Yankasa rams infected with *T. congolense*. Low semen concentrate was also observed in rams infected with *T. brucei* and *T. vivax* (Sekoni, 1992) which may be attributed to localization of trypanosomes in the scrotal skin, provoking non-purulent inflammation that leads to degeneration of the seminiferous and spermatozoa abnormalities.

CONCLUSION

In Trypanosomiasis infection, the animal becomes less productive because of the damages done to the reproductive organs by the invading parasites. These damages are either directly to the organs or to the micro-vascular structures of the organs. The pathogenesis of these damages has not been clearly elucidated. We believe that this aspect deserves closer study especially in livestock where reproductive performance is the cornerstone of productivity.

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