AFRICAN JOURNAL OF CLINICAL & EXPERIMENTAL MICROBIOLOGY JAN. 2003 ISSN1595-689 Vol 4 No 1
AJCEM/2002097/2301

THE EFFECT OF *SCHISTOSOMA MANSONI* CERCARIA INFECTION AND TREATMENT WITH NIRIDAZOLE ON TESTICULAR HISTOLOGY OF THE MICE.

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The testicular histology of mice infected with *Schistosoma mansoni* (*S.mansoni*) cercaria and treated with Niridazole was examined. The results reveal that infection of mice with *Schistosoma mansoni* cercariae resulted in distortion of the testicular cyto-architecture including disruption of spermatogenesis as shown by the absence of spermatozoa in the lumen of the sominiferous tubule and distruction of the inter-tubular connective tissue of the infected mice. These changes were reversed to normalcy following two-course treatment of the infected mice with Niridazole after five weeks.

INTRODUCTION

Schistosomiasis of the testis is still rare in many countries. When present, it may simulate cancer resulting in misdiagnosis and invariably leading to orchidectomy, an unfortunate event in the young patient (1). In Africa schistosomiasis of the testis may arise as a result of complication of infestation of the lower urinary tract by *Schistosoma haematobium*. However, a single case of infection of the testis by *Schistosoma mansoni* specie has been reported (2). This shows that both Schistosoma haematobium and *Schistosoma mansoni* are capable of affecting the testis, although the mechanism by which this occurs is not clearly understood. It is presumed that in the case of *Schistosoma haematobium* it may be due to retrograde passage of the egg from the urinary bladder into the vas deferens to the testis. The maturation of the Schistosoma might occur in the spermatic venous plexus and the deposited egg is carried distally into the smaller vessels of the testis (3).

Although early observations on the pathology of schistosomiasis have been reported (4), only a few reports of testicular schistosomiasis were made in the last sixty years (5,6,7). The pathological changes in this organ following infection are still incompletely described. Ihekwaba reporting a case of testicular schistosomiasis described the pathological changes to include the presence of calcified Schistosoma ova, scattered granulomata and fibrous tissue replacement of testicular substance. His report was silent on the state of the seminiferous tubules and spermatogenesis. It is the need to evaluate the effect of testicular schistosomiasis on spermatogenesis and testicular histology that necessitated this study, especially in this era of unexplained male factor infertility, with a view of understanding the mechanisms by which these occur, and hoping to introduce appropriate treatment module.

MATERIALS AND METHODS

i. Source of Parasite Strain

Schistosoma mansoni cercaria was obtained from naturally infected snails *(Biomphalaria pfeifferi)* collected from a stream in Jos, Plateau state of Nigeria where schistosomiasis is endemic. Snails were induced to shed cercaria in beaker containing distilled water by light illumination for one and half hours in the laboratory. The cercaria contained in the distilled water was transferred into a clean beaker and the cercarial density was determined by counting the number of cercaria in 0.5mls or 0.1ml of water.

ii. **Source of Mice**

Male adult white mice were bought from the animal house of the University of Jos. They were fed with Pfizer mouse cubes and water *ad libitum* and the mice were maintained in cages.

iii. **Infection of Mice**

The mice were stimulated to defecate and urinate in warm water (25°C) for twenty minutes in a bucket after which they were individually transferred into glass jars which had perforated covers in which about 20mls of distilled water had been placed. In order to infect the mice, between 180-200 cercaria were then transferred into each jar and the mice were allowed to paddle in the water for one and half hours as described by Moore(8).

iv. **Treatment of Mice with Niridazole**

The infected mice were given 250mls per kg body weight of Niridazole orally daily for five days as one course. After seven days' rest the second course of treatment were repeated. After the first course, 10 mice each from the control and infected groups were sacrificed and the testes obtained and fixed in Buioin's fluid for processing for histology. Five weeks after administering the second course ten mice were sacrificed in each group and the testes also obtained for histology.

v. **Histology**

the testes were processed for slide preparation and stained with heamatoxylin and eosin examined with the light microscope according to the method of Drury (9).

RESULTS

The control group showed normal testicular cyto-architecture. The seminiferous tubules showed spermatogonial cells at various stages of development and numerous spermatozoa in their lumen. The interstitial cells and inter tubular connective tissues were intact (Figure 1). The testes of the infected mice with *Schistosoma mansoni* cercaria showed distortion of testicular cyto architecture. The seminiferous tubule revealed disorganized spermatognial cells and empty seminiferous tubules suggestive of poor spermatogenic activity or disruption in the process of spermatogenesis (Figure 2). There is also destruction of the inter-tubular connective tissue including interstitial cells. This histological picture did not reverse after one course of treatment with Niridazole.

Figure 1: Cross Section of testes of uninfected mice. This shows normal testicular cyto-architecture, testicular cells at various stages of development and numerous spermatozoa in their lumen.

Figure 2: Section of testes of mice infected with cercaria of S. mansoni. There is distortion of testicular cyto-architecture. Seminiferous tubules reveal disorganized spermatozoal cells and they are devoid of spermatozoa in their lumen.

Five weeks after treatment of the mice with the second course of Niridazole, the testicular histology showed evidence of return to normalcy. The seminiferous tubules revealed the presence of numerous spermatozoa in the lumen (Figure 3). There is also regeneration of the inter-tubular connecting tissue including the interstitial cells of Leydig. This experimental result reveals that infection of mice with *Schistosoma mansoni* cercaria can result in testicular damage including cessation of spermatogenesis and hence may be a rare cause of male infertility. This damage is however, reversible after five weeks of two courses of treatment with Niridazole.

Figure 3: Section of infected mice testes 5 weeks after completing treatment with Niridazole. Numerous spermatozoa are seen in the lumen of the seminiferous tubules.

DISCUSSION

Our work revealed that infection of mice with *Schistosoma mansoni* induces distortion of the testicular cyto architecture and disruption of spermatogenesis as evidenced by absence of spermatozoa in the lumen of the seminiferous tubules in the s. mansoni cercaria treated group. This agrees with the finding of extensive replacement of testicular tissue with fibrous tissue (1). However, we did not find any granulomata or calcified schistosoma ova in the testicular tissue. Our finding of poor or scanty spermatozoa in the lumen of the seminiferous tubules is suggestive of disruption in the process of spermatogenesis. In highly endemic area of schistosomiasis such as ours it may be cause of infertility in the male. The mode of spread and mechanisms by which *Schistosoma mansoni* induces these histological change are uncertain. This may be because the incidence of schistosomiasis induced orchitis in the human is rare (2), and therefore, grossly understudied.

Several mechanisms and modes of spread have been proposed for schistosoma heamatobium infection, causing testicular pathology. These include retrograde passage of the eggs along the vas deferens to the testes as well as their maturation in the spermatic venous plexus and subsequently carried distally into smaller vessels of the testes (3). In our study no schistosoma ova was seen in the testicular tissue. Given the close relationship between the rectum and the base of the urinary bladder, seminal vesicles, prostrate gland and the vas deferens in the male, as well as the rich lymphatic anastamoses around the base of the bladder and rectum; and also the fact that rectal venous plexus which surround the rectum communicates anteriorly with the vesical plexus in the male (10), it is possible for *Schistosoma mansoni* ova whose route to the exterior is the rectum, to enter the vesical plexus and hence the testicular vessels where it may induce immunological response which might result in testicular histo-architectural distortion and disruption of spermatogenesis without the ova being deposited in the testicular tissue.

The finding that these changes were reversible five weeks after treatment with a second course of Niridazole suggest the valuable use of Niridazole in testicular orchitis due to schistosomiasis with or without infertility. It further suggests the need

for a careful investigation of *Schistosoma mansoni* in cases of unexplained male infertility with a view of better management.

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