Research Article

PATHOLOGICAL CHANGES OF DIMETHOATE IN MALE REPRODUCTIVE SYSTEM OF ADULT RABBITS

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Abstract
In present study, we investigated the pathological lesions in adult male rabbits resulting from chronic exposure of dimethoate, the animals were divided into four groups each group consist from six male rabbit, the G2,G3 and G4 consider as treated groups and received dimethoate orally (10, 25 and 50 mg /kg of BW ) respectively for two months while the G1 consider as control group and was given water only, after two months, all animals were scarified and post mortem were done, testis and epididymis were taken and preserved in 10 % formaldehyde. The results showed that the main pathological lesions in testis and epididymis of male rabbits are dose concerning which included mild to moderate to severe degeneration in germinal layer of somniferous tubules of testis especially in G2 and G3 and suppression of spermatogenesis while in group G4 there were sever testicular destruction and damage which characterized microscopically neurosis in germinal layers of seminiferous tubules of testis with sloughing and atrophy of epithelial lining of it, and complete suppression of spermatogenesis, while the main the histopathological lesions in epididymal tubules in most treated groups there were empty and not contain spermatozoa and complete loss of Spermatogenesis. It concluded that dimethoate caused severe histopathological lesions in male reproductive system of male rabbits especially in testis and epididymis, these results were severe necrosis and degenerations in germinal layers of seminiferous tubules with complete loss of spermatogenesis and complete absence of spermatozoa in epididymal tubules.

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1. Introduction
A pesticide defines as any substance that is purposed to repel or destroy a pest (EPA, 2008). Pesticides are a great and diverse group of chemicals (Kamel and Hoppin, 2004) which used to kill and eradicate insects, rodents, fungi and weeds (Mnif et al., 2011). Also, pesticides are responsible for various disorders and diseases included cancer, neurobehavior disorders, retrograde reproductive outcomes, peripheral neuropathies, reduced immune functions and allergic sensitization reactions, especially of the skin (WHO, 1990). Dimethoate (DM) is consider as one of One of the most widely used group of insecticides in the world is organophosphate (OP) compounds with different uses on field, agricultural crops and

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ornamentals (Hayes and Laws, 1990) and it is also applied indoor to control houseflies (Meister, 1992). The inclusive use of Dimethoate may cause a health hazard to most animals and humans on account of its persistence in crops and soil (WHO, 1996). Results from previous experiments on the influences of dimethoate on reproductive and endocrine function offered that it could affect on serum concentration of reproductive system and metabolic hormones (Rawlings et al., 1998). Dimethoate at 28 mg/kg of body weight was accompanied with a significant decreased in sperm count, also mobility and viability with significantly increased in the percentage of the morphologically abnormal spermatozoa contrast with the controls (Abdallah et al., 2010).

Dimethoate was caused developmental toxicity also reproductive failures in organisms after repeated exposures, developmental toxicity of dimethoate includes decreased number of implantations and live fetuses, happening of resorptions and decreased fetal body weights (Farag et al., 2006). Reproductive toxicity of dimethoate on adult rodents in both sexes are demonstrated, the irregularity of estrous cycle in addition to altered level of serum gonadotrophins were reported in females (Kaur and Dhanju, 2005), while failure of fertility, repressed libido, semen quality deterioration, changed testosterone levels and testicular degeneration are reports available in males (Farag et al., 2005; Sayim, 2007; Ngoula et al., 2007). The aim of the current study to investigate the pathological lesions associated with Dimethoate intoxication on reproductive organs and fertility in adults male rabbits.

2. Materials and Methods

Animals and Experimental design

Twenty - four local malerabbits about 6 months old, weight ranged from 1500 – 2000 g which were obtained from local market, all animals were housed in the animal house in cages in a room and exposed to 12 hours light /12 hours dark in 22 -25 °C. The animals were randomly distribute into four groups any group consist of 6 animals. Group (G1) consider as the control group which received water only. Group 2 (G2) was exposed to Dimethoate (10 mg/kg of body weight (BW) per day) (American Cyanamid Company, 1984) for two months. Group 3 (G3) was received Dimethoate (25 mg/kg of (BW) per day) for two months. Group 4 (G4) which given (50 mg/kg of body weight (BW) per day) for two months. All remediation were given by oral gavage.

Pathological study

At the termination of experiment, the animals were scarified and postmortem was done for each animals. Specimens such as testis and epididymis were taken; the tissues were preserved in 10 % formaldehyde directly after removal. Next 48 hours of the fixation, the processing was routinely achieved with a set of rising alcohol concentrations, tissue section was embedded in paraffin blocks, which sectioned by microtome at 5 µm for each tissue .the tissues were stained with hematoxylin and eosin stain (H and E stain) and the histopathological lesions were observed by light microscope (Luna, 1968).

3. Results and Discussion

Different pathological changes were seen in different groups due to Dimethoate toxicity in rabbits but more severe lesion were seen in group four (G4): In case of control untreated animals (G1) there were no significant microscopic lesions (Fig.1: A). In case of treated group (G2, G3 and G4). Grossly, the more sever pathological lesions in testicular tissue which appear atrophied. While in light microscopic appearance, the main histopathological lesions of the treated rabbits showed that Dimethoate caused dose - related in testicular damage which is characterized by mild hydropic degeneration in the germinal layers which lining seminiferous tubules and sloughing of their cell lining, and by partial suppression of spermatogenesis, while the epididymis it appear microscopically, there is empty and not contain spermatozoa and al loss of spermatogenesis in case of G2. While in the G3 the main microscopic changes, it more sever in testicular damage than G2, there is moderate hydropic degeneration in the germinal layer of lining of seminiferous tubules and sloughing and atrophy of their cell lining, also there is suppression of spermatogenesis, also epididymal tubules were also empty and not
contain spermatozoa and complete loss of spermatogenesis (Fig - 1B). In G4 the histopathological lesions is more severe and extensive in all testicular tissue than other groups (G2, G3), there is severe necrosis in the germinal layers of lining of seminiferous tubules, and complete suppression of spermatogenesis with no spermatogonia (Fig - 1C and Fig - 2A), while the epididymal tubules were also empty and not contain spermatozoa and complete loss of spermatogenesis (Fig - 2B and C). These results are acceptance with that finding by Ferah (2007) who reported same histopathological lesions of the treated rats included that Dimethoate which caused dose-related testicular damage which characterized by mild to moderate to severe seminiferous tubule degeneration as sloughing, atrophy of epithelial lining of it, germ cell degeneration and necrosis and by partial to complete arrest of spermatogenesis. In the current study the microscopic lesions of tissue structure of testes of rabbits fed Dimethoate (10, 25 and 50 mg/kg of BW for two months) show span of Sertoli cells damage with suppression and impairment of spermatogenesis, these results are consistent with Chakroun et al. (2002) who obtained identical results, but in testis of rats which exposed to nickel.

A significant decrease in the relative size and weight in testis of rabbits, our results are agree with Joshi and Bansal (2012), the decrease in testicular size and weight may be occur due to a depression in the tubule size, spermatogenic arrest and inhibition in biosynthesis of steroid in Leydig cells, which consider as a site of steroid biosynthesis (Sujatha et al., 2001; Sanchez Pena et al., 2004).

In our study, there were decreased in testicular and epididymal size and weight, this results are coordinated with results of (Afifi et al., 1991; Huanh et al., 2006; Joshi et al., 2007; Choudhary et al., 2008) who showed in adult rodents due to direct incur to dimethoate and other organophosphorus pesticides also to the pesticides which having antiandrogenic potential (Gray et al., 1999; Turner et al., 2002). In the present study, the effect of dimethoate to the spermatogenic also steroidogenic compartment of the testis was showed in its histopathology, decreased seminiferous tubule diameter, damage of germ cells, complete absent of sperms in lumens, and decreased in Leydig cells size and number were observed in doses of 10, 25 and 50 mg/kg of BW, complete arrest of spermatogenesis was distinct in the 25 and 50 mg/kg exposed group where numbers of germ cell layers were significantly decreased with complete absence of spermatozoa in many tubules, similar histopathological lesions in the adult rodent testes when direct incur to sub-lethal doses of dimethoate and another organophosphates have been showed by Farag et al. (2007), the significant atrophy of epididymal tubules and changes in histoarchitecture of the principal cells of epididymis was also indicative of the negative impact of dimethoate on male reproductive system, when adult rodent epididymis incur to dieldrin, dichloro diphenyl trichloroethane and lindane exhibited similar histopathological finding who approved by Dalsenter et al. (1997); Ben Rhouma et al. (2001); Hallegue et al. (2003).

4. Conclusion

In conclusion, the current study showed that the Dimethoate is caused different pathological lesions in male genital system of adult rabbits especially testis and epididymis which appeared microscopically sever necrosis and generations in the germinal layer of lining of seminiferous tubules, and complete suppression of spermatogenesis with vaculation of spermatogonia, and the epididymal tubules appeared empty and not contain spermatozoa and complete loss of spermatogenesis.
Figure - 1: Testis: A: Normal structure of testis, showed somniferous tubules contain normal A: spermatozoa, B: Spermatogenesis (X 40 H & E). B; Testis of rabbits which exposed to 25 mg/ kg of BW per day from dimethoate appeared A: Suppression of spermatogenesis, B: Reduced numbers of spermatozoa in lumen (X 20 H & E). C; Testis of rabbits which exposed to 50 mg/ kg of BW per day from dimethoate showed A: Sever suppression of spermatogenesis, B; Complete reduction numbers of spermatozoa in lumen (X 40 H & E).

Figure – 2: Testis of rabbits (A) which exposed to 50 mg/kg of BW per day from dimethoate showed complete destruction of testicular tissue and A: Sever suppression of spermatogenesis, B; Complete reduction numbers of spermatozoa in lumen, C; Decrease in the numbers and vaculation of spermatogonia (X 40 H & E). Epididymis of rabbits (B) which exposed to 50 mg/kg of BW per day from dimethoate showed complete loss of spermatogenesis (A) (X 20 H & E); Epididymis of rabbits (C) which exposed to 50 mg/kg of BW per day from dimethoate showed majority of tubules were empty and no spermatozoa (A) (X 40 H & E).

5. References


