

Effects of Lead Toxicity on Spermatogenesis in the Testes of Albino Rats

Dr. Saffia Shaukat, Dr. Shabana Ali, Dr. Fatima Riaz

ABSTRACT

Objective: The objective of this study was to observe changes in spermatogenesis in the testes of albino rats exposed to lead acetate.

Study Design: An experimental animals study

Place of Study and Duration: This study was carried out in the Department of Anatomy, Islamic International Medical College Rawalpindi and at National Institute of Health Islamabad from January to April, 2009.

Materials and Methods: Male adult rats were exposed to lead acetate with intraperitoneal dose of 4mg/Kg body weight for 5 days a week for 6 weeks. The animals in group A were used as control. The animals of groups B were treated with lead acetate with specified dose for 6 weeks. After 6 weeks the animals of subgroup B were sacrificed. The results of these two groups were then compared.

Results: After six weeks, it was observed that the number of spermatogenic cells had decreased in the test groups as compared with control group ($p < 0.05$).

Conclusion: Lead is toxic for cells of spermatogenic series, injurious to health and plays a significant role in reducing male fertility.

Key words: *Spermatogenesis, Lead toxicity, Rat fertility*

Introduction

Lead is a bluish-grey metal present in the earth's crust. It is one of the most ancient and commonest environmental pollutants, which has been reported to have caused damage in multiple body systems. It can be a cause of severe health problems such as high blood pressure, renal damage, nervous system breakdown, and anomalies in reproductive system (Meyer, 2008).¹ Pakistan is a developing country. People here are specially exposed to lead pollution through three main sources i.e., air, soil, and water (Rajendra, 1997).²

Various researches carried out in Pakistan, have stated that the majority of population has blood lead levels much above the internationally acceptable limits. Another survey conducted in lead factory workers in Pakistan showed blood lead levels hovering

around 60 $\mu\text{gms/dl}$ (Khan, 1994)³. Lead is found in almost all agricultural food. Lead present in air falls onto crops or soil which is absorbed by plants, thus entering the food chain and finally consumed by humans gaining entry into human blood (George, 1993).⁴ Following are the major sources of lead:-

- Insecticide/Pesticide sprays (Tollestrup, 1995).⁵
- Lead can enter the water supply from lead solder in plumbing, lead service connections or lead pipes in home. Lead is resistant to corrosion and hence old lead used as drains in the baths are still in service (Lanphear, 2005).⁶
- Lead based indoor and outdoor paints (Lanphear, 1998).⁷

Materials and Methods

The study was carried out in the Department of Anatomy, Islamic International Medical College Rawalpindi and National Institute

Correspondence:
Dr. Saffia Shaukat,
Assistant Professor
Anatomy Department,
IIMCT Rawalpindi

of Health, Islamabad. It was an analytical trial. The duration of study was 6 weeks. Eight weeks old healthy male albino rats of Sprague Dawley strain weighing 200 ± 10 gm. were selected for the study. A total of 30 adult male albino rats of Sprague Dawley strain were used in this study. These animals were randomly divided into two groups; one Control (A) and one Experimental group (B). The animals in group A (control) were given normal diet ad libitum. The animals in group B were treated with intraperitoneal lead acetate in the dosage of 4 mg / Kg body weight / day, 5 days a week for a period of 6 weeks. These animals were sacrificed at the end of six weeks. All animals were kept in the animal house under standard conditions at a room temperature (18°C to 26°C) for six weeks. They were maintained on 12 hours light and dark cycle. The rats were fed ad libitum. Day 0 was considered to be the starting day of experiment. Pure lead acetate, taken from Pharmaceutical Laboratory in Islamic International Dental College (IIDC), was used. It was purchased from the Merck Company (Lot No. 107372). The temperature was maintained between 18°C to 26°C. The animals were sacrificed by an overdose of ether anesthesia. Cotton soaked in ether was placed into the jar. The animal to be sacrificed was lifted by the tail and dropped into the jar. After sacrifice, the animal was taken out of the jar and placed on the dissecting board. The scrotal sac was then opened with the help of forceps and scissors. After fixation, histological sections were made and staining was done by Haematoxylin and Eosin.

Results

Data was entered in a data base using Statistical Package for Social Sciences (SPSS) for window version 13. The results were analyzed and considered significant with P value less than 0.05 ($P < 0.05$) applying t test. Only three cross sections of seminiferous tubules (randomly selected in different fields), were observed. Total numbers of germ cells in the selected tubules were counted.

Histological Examination: The following observations were made: Number of spermatogenic cells/cross section of seminiferous tubule at 40x.

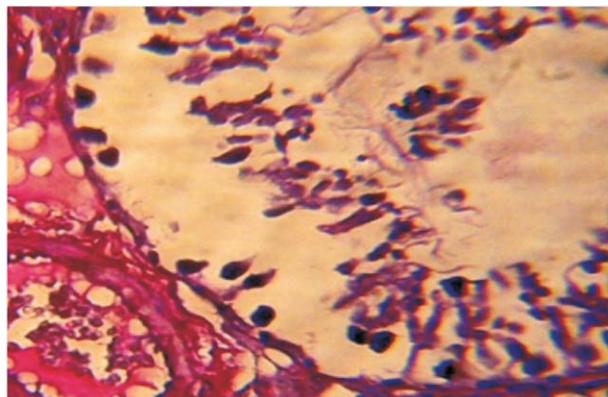


Fig.1: Photomicrograph-Section of testis of experimental group (Group B) animal number 15, showing reduced germ cell count and reduced height of germinal epithelium. PAS stain. x 420.

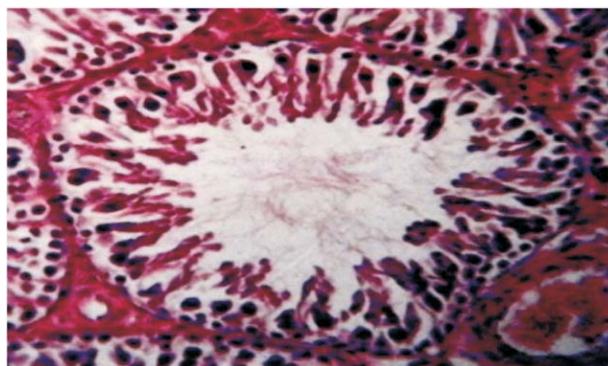


Fig. 2: Photomicrograph; Section of testis showing seminiferous tubule with normal germ cell count in group A, Animal Number 13, PAS Stain, x 400

Germ Cell Count / Cross Section Of Seminiferous Tubule)

The average germ cell count in group A was 304.53 cells/cross section of seminiferous tubule (SD + 28.46), in group B it was 116.91 cells/unit (SD + 32.66).

The germ cell count was significantly higher in group A as compared to groups B ($p < 0.001$). (Fig No. 1). The germ cell count was significantly lower in group B as compared to groups A (Fig. 1) ($p < 0.001$).

Discussion

The present study was conducted to evaluate whether or not, lead has toxic effects in the rat's testes. For this purpose, albino rats of Sprague Dawley strain were selected. The animals were randomly divided into a control A and experimental group B. The animals of experimental group (B) were treated with intraperitoneal lead acetate in the dosage of 4 mg / Kg body weight / day, 5 days a week for a period of 6 weeks. After 6 weeks, the animals of group B were sacrificed to see the toxic effects of lead in their testes. In lead toxic testes the seminiferous tubules decreased in size and had a wavy outline. The germ cell count was significantly reduced in group B, as compared to the control group A, which indicated the intensity of harm caused by lead on the cells of spermatogenic series. No effect on Sertoli cells was observed.

The population of Leydig cells had become dispersed and they were sparingly seen in clumps. These findings are similar to those of Chowdhury et al, Saxena et al and Hilderbrand et al.8, 9, 10. All these scientists described variety of toxic changes induced by lead in the testes depending upon dose

and duration of the treatment which are in accordance with present study. At the end it is concluded that lead is injurious to the cells of spermatogenic series and the interstitial cells of Leydig, while the Sertoli cells are spared.

Chapin (2002) described in his study that CDC defines blood lead levels exceeding 10 micrograms/dl as elevated in children¹¹. This may lead to cognitive and behavioral developmental changes. The researcher reported that lead induced reproductive abnormalities may occur in male rats at a dose as low as 10 µg/dl¹¹. Manlay (1995) administered lead acetate to rats in the dose of 8mg/kg body weight, 5 days a week for 35 days¹².

During past three decades, the decline in male reproductive health and fertility has been linked with environmental toxicants and xenobiotics. (Sikka and Wang, 2008)¹³.

My present study validated the previous studies?, results and further added a new dimension to the existing literature by investigating lead-induced toxicity in the testes of Sprague Dawley rats.

References

1. Meyer PA, Brown MJ, Falk H. Global approach to reducing lead exposure and poisoning. *Mutation Research E Pub* 2008;50:166-75.
2. Ramlogan A. Environment and human health: a threat to all. *Environmental Management and Health*, 1997; 8: 51-66.
3. Khan DA, Malik 1A, Saleem M, Hashmi R, Bashir R. Screening for chronic lead poisoning in lead factory workers. *J.PMA*1994;239-41.
4. George A. Gellert GA. Wagner, Roberta M. Maxwell, Douglas M. FASTER I. Lead Poisoning Among Low-Income Children in Orange County, California. *JAMA*1993;69-71.
5. Tollestrup K, Baling Jr., Allard J. Mortality in a cohort of orchard workers exposed to lead

- arsenate pesticide spray. Archives of Environmental health;1995;221-9.
6. LanphearBP. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environ. Health Percept 2005;113:894-99.
 7. Lanphear BP. Matte TD, Rongers J. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. Environment research 1998;51-68.
 8. Eyden BP, Maisin JR and Mattelin G. Long term effect of dietary lead acetate on survival, body weight and seminal cytology in mice. Bull Environ Contam Toxicol 1978;19:266-72.
 9. Saxena DK, Srivastara RS, Lal B and Chndra SV. The effects of lead exposure on the testis of growing rats. Exp Pathol 1987;31: 240-52.

