

Effects of Vanadyl Sulphate on Spermatogenesis in Male Rats

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Abstract

Background: It is known that vanadyl compounds are capable of alleviating hyperglycemia in streptozotocin induced diabetic rats.

Objective: To examine the effect of vanadyl sulphate (VS) on spermatogenesis of male rats.

Methods: Male rats (n=10) were administered 32 mg/kg/day of VS orally and 50 mEq/l NaCl (as drinking water) for one month. Meanwhile, 11 male rats, the control group, received vehicle only; 50 mEq/l NaCl as drinking water. At the end of the study, blood testosterone level as well as spermatogram of rats in both groups were determined. The animals were sacrificed and their testes and epididymes were then studied under light microscope.

Results: In VS treated group, blood testosterone level, and sperm count were significantly decreased by 51% (normal = 2.83 ± 0.7 ng/ml, $p < 0.001$), and 80% (normal = 565×10^6 /ml, $p < 0.05$), respectively, as compared to the control group. However, sperm motility, shape, and histology of testes and epididymides were not different from those of the control group.

Conclusion: Vanadyl sulphate has detrimental effects on spermatogenesis.

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Keywords • Vanadyl sulphate • spermatogenesis • testosterone • diabetes mellitus.

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Previous studies have shown that the blood glucose level of streptozotocin induced diabetic rats can be normalized by oral administration of vanadyl sulphate (VS).¹⁻⁴ Since the drug might have some clinical implications, we conducted several trials to determine its possible side effects on different organs. In this work we present the effect of VS on spermatogenesis.

Twenty-one male Charles River rats, weighing 250-350 g were divided into two groups. The first group (n=10) received 32 mg/kg/day VS orally as drinking solution for one month. The control group (n=11) received only vehicle, 50 mEq/l NaCl as drinking water. Blood samples were taken from the tip of their tails, to measure testosterone level. Using a Hemacytometer lame, the count, motility and shape of spermatozoa which has been taken from the vas

deferens of rats under anesthesia, were determined. The rats then were sacrificed and their testes and epididymes were examined under light microscope.

Mean±SD plasma testosterone level was 1.37 ± 0.7 ng/ml in VS treated rats. This value was significantly ($p<0.001$) lower than that measured in the control group (2.83 ± 0.7 ng/ml). The mean sperm count of 115×10^6 /ml in VS treated rats was also significantly ($p<0.05$) less than that of the control group (565×10^6 /ml). No statistically significant difference was observed between the two groups considering the sperm motility and shape. None of the histological sections of testes or epididymes of VS - treated rats showed any significant change when compared to those of control rats.

The results of our study showed a reduction in blood testosterone level and sperm count of 51%, and 80%, respectively. Unlike what was observed in some reports⁵, regarding intraperitoneal administration of vanadium and induced testicular damage, we could not observe any histological change in either testes or epididymis tissues. This discrepancy might be due to the route of administrations of the drug. In conclusion, we believe that oral administration of VS has detrimental effects on spermatogenesis at the dose given.

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