In the present experiment a pentavalent arsenic compound, sodium arsenate was administered to mice to raise a model organism with arsenicosis following intraperitoneal administration of sodium arsenate at the dosages of 25 mg kg⁻¹ body weight and 30 mg kg⁻¹ body weight for a period of 45 days. Upon this model organism of arsenocosis ameliorative potentiality of a selenium compound, sodium selenite is evaluated. After thorough investigation on the pathological conditions developed in the vital organ testis due to arsenic toxicity and the following conclusion can be drawn. Arsenic exposure is detrimental to the biological system. It causes damage to the testis at tissue level, cellular level which are ultimately reflected in physiological disruption. Significant reductions in the number and diameter of seminiferous tubules as well as in the number of tubules containing healthy germ cells indicate the arsenic induced gonadal pathology and oligospermia. After selenium treatment, the ameliorative groups showed all the features of normozoospermia with respect to structure, count and motility. Serum testosterone concentrations, GSH and GST levels were also decreased in all successive treated groups and these were refurbished by the treatment with sodium selenite. Apoptosis was detected by chromatin condensation assay, ROS generation and FACs due to sodium arsenate treatment while it was refurbished by sodium selenite. Our fruitful observations support the notion that sodium arsenate impairs male reproductive function by inducing oxidative stress and apoptosis, and these experimental results suggest that selenium supplementation through oral route improved sodium arsenate induced toxicity on testis.