Effects of tissue trace minerals status and histopathological changes in chronic arsenicosis in goats

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Abstract

The study was carried out on goats intoxicated with sodium arsenite @ 2 mg /kg body weight for six months. Results indicate that elevated levels of copper and zinc in tissues were found with respect to increase arsenic concentration. Pathological changes were observed in liver, kidney and brain.

Keywords: Arsenic, Chronic toxicity, Tissue, Histopatholgy, Goat.

Introduction

Micronutrients interact with toxic metals at primary site of action, absorption, transport of toxic metals in the body; binding to target proteins; metabolism, sequestration and excretion of toxic metals and finally, in secondary mechanisms of toxicity (*Peraza et al.*, 1998). Arsenic, as soluble arsenate or arsenite, is well absorbed (80%) in animals exposed by oral route. Absorption appears to occur by passive diffusion distribution occurs throughout the body (ATSDR, 2000). The present study was therefore undertaken to evaluate the trace mineral status in biological samples and histopathological changes in chronic arsenicosis in goats.

Materials and methods

Twelve goats of 2 to 3 years of age (30 ± 4.5 kg body weight) were randomly divided into 2 groups having six animals in each. Animals of group I were treated as healthy control where as group II received the chronic toxic dose of sodium arsenite i.e. @ 2 mg/ Kg body weight orally daily for the period of six months. Blood and tissue samples (liver, kidney, brain, and blood) were collected after the completion of experimental trial.

Biological samples were digested in bomb calorimeter at 100°C. Digestion was made by adding perchloric, sulphuric and nitric acid (1.0 ml HClO4, 1.5 ml H2SO4 and 4.0 ml HNO3) and heated at (110-120°C) until a clear solution was obtained. Dilution to known concentration was finally made with triple distilled water. The digested samples were then analyzed for total arsenic, copper, and zinc at the laboratory using Atomic Absorption Spectrometry (ASS) equipped with hydrous generation. Rigorous quality control procedures were followed throughout the analysis. Tissues were collected from group II animals in 10% buffered formalin and processed through conventional histological procedure. Results of concentration of arsenic, copper and zinc in tissues were presented as Mean ± Standard error. The data were statistically analysed and compared by analysis of variance (ANOVA) at 5% level of significance using standard statistical methods (Snedecor and Cochran, 1994).

Results and Discussion

The level of arsenic, copper and zinc was observed in the present study during pre and post treatment in experimental groups is furnished in the table. It was found that tissues like blood, brain, liver and kidney has elevated arsenic concentration but significant ($P< 0.05$) increase arsenic was observed in kidney and liver tissues. Maximum arsenic was found in liver as this is main organ, which helps in the metabolism of arsenic (Vahter and Marafanate, 1987). Non-significant decrease copper level was observed in blood kidney and brain, but significant ($P< 0.05$) decrease was found in the liver. Uthus, 2001 also observed the copper deprivation on high dietary arsenic. Experimental animals exposed to arsenic have increased hepatic and renal zinc concentrations, when the intake of zinc is marginal. Non-significant increase zinc level was observed in the blood and

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Table-1. Tissue arsenic copper and zinc level (ppm) before and after treatment in goats with chronic arsenic toxicity.

Samples	Control healthy (Group I)			Intoxicated (Group II)		
	Arsenic	Copper	Zinc	Arsenic	Copper	Zinc
Blood	0.03±0.26	1.02±0.33	10.25±0.67	6.7±0.38	0.62±0.36	12.26±0.54
Kidney	0.08±0.54	3.26±0.32	18.45±1.22	13.72±0.43*	2.69±0.47	32.16±2.41*
Liver	1.26±0.62	46.11±0.76	24.21±0.15	21.3±0.44*	34.27±0.44*	38.46±0.28*
Brain	0.01±0.22	14.33±0.66	14.28±0.21	4.5±0.31	12.47±0.27	16.47±0.27

* Significantly different (P< 0.05) as compare to the normal animals.

brain but significant increase values were found in liver and kidney of group II animals. *Milton et al*. (2004) suggested that zinc has been linked to decreased arsenic toxicity. Zinc protects animals against arsenic toxicity might be due to induction of metallothionein synthesis in animals, which reduces the toxicity of arsenic (*Kreppel et al.*, 1994). *Falnoga et al.,* 2000 reported the binding of arsenic with metallothionein protein. These suggest that the essential elements may contribute to the protection of man and animal from the effects of heavy metal exposure, while their deficiency may increase toxicity. Histopathological examination revealed vascular congestion, massive haemorrhages and degenerative changes in liver parenchyma. Fatty degeneration of hepatic cells leads to coagulative necrosis which corroborates the findings of Gorden and Lough, (1972). Kupffer cells present in the liver became swollen. There was brownish pigment hemosiderin in the cytoplasm of hepatocytes. In kidney there was coagulative necrosis and increased cellularity in the glomerulus possibly due to increase in the number of mesenchymal cells in corroborating the finding with *Biswas et al.,* (2000). In nervous tissue there was swelling of the endothelial cells of the capillaries along with central chromatolysis in the neuron was observed. Proliferation of gliall cells was also prominent finding in chronic arsenic toxicosis.

Conclusion

From the present study it can be concluded that the body burden altered the micronutrient concentration and also causes tissue damage.

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