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EFFECT OF LIV-52 ON VANADYL SULPHATE

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An Ayurvedic medicine Liv-52 was studied as a prophylactic agent against vanadium induced toxicity in rats by administration of Vanadyl sulphate at a dose of 40 mg/ kg (I.P.) once only. Blood biochemical parameters also showed disturbed RBC, WBC, Hb% after vanadium exposure. Activities of SGOT and SGPT increased significantly. Liv-52 primed rats, however, exhibited comparatively less marked toxic effects.

Key words : Vanadyl sulphate, Liv-52, rats, chelating agent.

INTRODUCTION

Vanadium is distributed extensively in nature. It is a trace element and is present in almost all living organisms including man. Exposure to this metal has become concern for toxicologists as it is used in the hardening of steel, manufacture of pigments, in photography and in insecticides. It is also used as a catalyst in the production of various compounds which has attracted the investigators interest due to their potent effects on biological systems (Donaldson *et al.*, 1985). Inspite of important health risks produced by vanadium, there is relatively little information about the possible chelating agents to be used therapeutically in cases of vanadium poisoning (Mitchell & Floyd, 1954; Jone & Basinger, 1985). In the present study an ayurvedic preparation Liv-52 (Himalaya Drugs Co., Bombay) which is well known to correct liver.

MATERIALS AND METHODS

Adults healthy male rats (160 ± 10gm) of Sprague Dawlet strain were selected from the Departmental animal colony. These animals were maintained under uniform husbandry conditions of light and temperature. They were given standard pelleted diet of Lipton India Ltd., New Delhi (India) (meal content of diet in ppm dry weight Cu 10.0, Mn 55.0, FE 70.0, Zn 45.0, Co 5.0 and drinking water *ad libitum*. A dose of 40 mg/ kg body weight of Vanadyl sulphate was prepared in pyrogen free distilled water. Animals were divided in to 4 groups of 5 rats each and were treated as follows.

- Group-1 : Animals were given distilled water at a dose of 1 ml/rat/day. This group served at control.
- Group-2 : Animals were primed with Liv-52 for 15-days at a dose of 1 ml/day.
- Group-3 : Distilled water primed animals were administered Vanadyl sulphate intra peritonealy once at a dose of 40 mg/kg.
- Group-4 : Animals received Liv-52 as in Group-2 and Vanadyl sulphate as in Group-3.

Biochemical assays : Standard techniques were employed to determine haemoglobin percentage, total erythrocyte and lecucoyte counts were enumerated by using Neubaur's chamber (Wintrobe, 1933). The differential leucocyte count (DLC) was carried out in blood films stained with Leishman's strain. Erythrocyte sedimentation rate (ESR) was determined by using the

methods of Wintrobe & Landsberg (1935). Heparinized blood used for quantifying blood sugars (Asatoor & King, 1969). Serum samples were processed for the estimation of glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) (Reitman & Frankel, 1957).

RESULTS AND DISCUSSION

Haematological vanadium administration evoked severe alteration in blood/ serum biochemical parameters. Blood sugar level, Hb%, hematocrit, neutrophils, monocytes, basophils percentage and RBC counts were significantly decreased after vandium administration (Table I). On the contrary serum GOT and GPT activity, WBC counts, ESR and lymphocytes percentage showed increased values following vanadium exposure. Concomitant supplementation of Liv-52 during vandium exposure restored most of the above biochemical alterations to some extent.

Table I : Vanadium administered haematological blood/serum parameters.

Parameters	Control	Vanadium	Liv-52	Vanadium+Liv-52
Haemoglobin (g/100ml)	14.64±1.2	7.6±0.65*	13.06±0.79	12.93±0.99
Hematocrit (%)	42.0±3.62	0.0±1.8*	37.0±2.93	5.0±2.9**
RBC count (million/cu mm)	8.80±0.7	7.64±0.63	9.5±0.74	8.13±0.78
WBC count (thousand/cu mm)	7800±400	8050±635*	7650±426	7935±496
Lymphocytes (%)	67.55±4.6	83.50±6.8*	66.44±5.6	71.90±6.6
Neutrophils (%)	27.0±1.91	0.9±0.15*	2.8±0.16	2.4±0.14**
Eosinophils (%)	2.5±2.01	0.5±0.9*	2.6±0.19	2.5±0.16**
Monocytes (%)	2.7±0.19	1.9±0.15*	2.8±0.16	2.4±0.14**
Basophils (%)	0.25±0.16	0.03±0.002*	0.26±0.019	0.20±0.17**
ESR (mm/hr)	0.16±0.15	0.50±0.04*	0.19±0.017	0.17±0.01**
SGOT (IU/l)	67.00±5.4	84.73±6.6*	68.10±6.4	70.34±4.9
SGPT (IU/l)	53.00±4.5	63.50±5.4	56.40±4.1	58.27±3.1
Blood sugar (mg/ 100ml)	97.05±7.7	81.05±4.2*	98.81±82	94.28±5.9

* = Significant ar 5% level: ** = Significant at 1% level.

Single administration of Vanadyl sulphate at a dose of 40 mg/kg followed by 3 days of chelation therapy, resulted in decrease in haemoglobin percentage, hematocrit percentage and RBC count. It also increased the number of WBC, lymphocytes and ESR, however, neutrophils, eosinophils, monocyte and basophil percentage decreased. Serum transaminases increased significantly. On the contrary, the level of blood sugar decreased. More recent reports indicate that oral vanadium in all forms metavanadate, orthovanadate or vanadyl sulphate elicited toxicity that ranged from decreased weight gain and increased serum concentration of urea and creatinine to death (Damingo *et al.*, 1991). Atsauko Adachi *et al.* (2000) reported that significant decrease in hemoglobin, hematocrit after vanadium treatment. Prophylactic agents like Liv-52 showed protective action in all the parameters, present findings reveal that with the vanadium administration the erythrocyte count study was decreased significantly but crenated RBC were increased. In the present study with the supportive treatment of Liv-52, the affected hematological parameters were found to restitute towards normal.

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REFERENCES

ASATOO, A.M. & KING, E. 1969. Practical Clinical Biochemistry (Vorley, H. ED.). 4th edn., Gulab Vaziraninn Publication, India for Arnold Heinemann, pp. 86.

DOMINGO, J.L., GOMEZ, M., LLOBET, J.M., CORBELLA, J. & KEEN, C.L. 1991. Improvement of glucose homeostasis by oral vanadyl or vanadate treatment in diabetic rats accompanied by negative side effects. *Pharmacol. & Toxicol.* **68** : 249.

DONALDSON, J., HEMMING, R. & LABELLA, F. 1985. Vanadium exposure enhances lipid-peroxidation in the kidney of rats and mice. *Canad. J. Physiol. Pharmacol.* **63** : 196.

JONES, M.M. & BASINGER, M.A. 1983. Chelate antidotes for sodium vanadate and vanadyl sulfate intoxication in mice. *J. toxicol. Environ. Health.* **12**(4-6) : 749.

MITCHELL, W.G. & FLOYD, E.P. 1954. Ascorbic acid and ethylenediaminetetra acetate (EDTA) as antidotes in experimnetal vanadium poisoning. *Proc. Soc. Exp. Biol. Med.* **85** : 206.

REITMAN, S. & ANA FRANKEL, A.S. 1957. A coloimetric method for the determination of serum glutamic oxalacetic and glutamic pyruvic transaminases. *Am. J. Clin. Pathol.* **28** : 56.

WINTROBE, M.M. 1933. Macroscopic examination of the blood. *Am. J. Med. Sci.* **185** : 58-71.

WINTROBE, M.M. & LANDSBERG, J.W. 1935. A standardised technique for blood sedimentation test. *Am. J. Med. Sci.* **189** : 102.