Haematobiochemical profiles of affected cattle at arsenic prone zone in Haringhata block of Nadia District of West Bengal in India

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Citation

Abstract

Haematology and biochemical analysis of arsenic affected cattle revealed lower level of Haemoglobin ($8.300^b \pm 0.221$ g/dl), total erythrocytic count ($5.477^b \pm 0.096$ X10^6/µl), total leucocytic count ($6.025^b \pm 0.086$ X10^6/µl), total serum protein ($6.230^b \pm 0.006$ g/dl) and increased levels of blood glucose ($50.631^b \pm 0.673$ mg/dl), Aspertate aminotransaminase /AST($33.771^b \pm 0.577$ IU/l, Alanine aminotransaminase/ALT($7.566b \pm 0.108$ IU/l), blood urea nitrogen/BUN ($23.995^b \pm 0.736$ mg/dl) and Creatinine ($1.020^b \pm 0.031$ mg/dl) than healthy control cattle (Hb-$10.800^a \pm 0.327$ g/dl, TEC-$6.764^a \pm 0.133$ X10^6/µl, TLC-$6.375^a \pm 0.106$ X10^3/µl, TSP-$6.741^a \pm 0.107$ g/dl, BGL-$47.029^a \pm 0.772$ mg/dl, AST-$28.095^a \pm 0.631$ IU/l, ALT-$6.478^a \pm 0.178$ IU/l, BUN-$15.793^a \pm 1.023$ mg/dl and Creatinine -$0.816^a \pm 0.034$ mg/dl respectively).

INTRODUCTION

Arsenic is one of the most toxic elements that can be found. Despite their toxic effect, inorganic arsenic bonds occur on earth naturally in small amounts. Animals are exposed to arsenic through food, water and air. Exposure may also occur through skin contact with soil or water that contains arsenic. Food is usually the largest source except in areas where drinking water is naturally contaminated with arsenic. The effects depend on the chemical form of the arsenic, the nature of the surrounding environment and their own particular biological sensitivity.Arsenic is of great environmental concern due to extensive contamination of groundwater in the Bengal delta basin with this toxin, thereby causing carcinogenic toxicity to millions of people as well as animals. Nonaghata area of Haringhata block of Nadia District of West Bengal in India is highly arsenic affected zone. The present study was undertaken to record the clinical, haematological and biochemical changes in arsenic affected cattle and to find out parameter that is affected primarily.

MATERIALS AND METHODS

A total number of thirty clinical cases suspected to be suffering from arsenic toxicity with the clinical signs including depression, prostration, weight loss, weakness, dehydration, anaemia, anorexia, diarrhoea with blood, ruminal stasis, lethargy, dermatosis, reddish urine, dry dull rough, epilated hair coat, anoestrus were screened by haemato-biochemical examinations and kept as experimental (Gr.II).Ten healthy cattle from non affected zone were kept as a healthy control group (Gr. I). Blood samples were analysed for haemoglobin (Hb), total erythrocytic count (TEC), total leucocytic count (TLC) (Schalm et al. 1986). Total serum protein (TSP) (Kollar, 1984), Blood glucose level (BGL) (Hultman, 1959), serum aspertate amino transaminase (AST) and serum alanine aminotransaminase(ALT) (Reitman and Frankel, 1957),blood urea nitrogen(BUN) (Marsh ,1965) and serum creatinine (Toro and Ackermann (1975) using standard reagent kits. The statistical analysis of the data was done as in SPSS (version 10.0) following general linear model. All the data obtained were analysed in SPSS (version 10.0) following general linear model. The means were compared using Independent t tests. Probability of $P < 0.01$ and $P<0.05$ were described as highly significant (at 1% level) and significant (at 5% level) respectively.

RESULTS AND DISCUSSION

The mean values of Haemoglobin (Hb) percentage of Gr. II and Gr.I were 8.300 ± 0.221 g/dl and 10.800 ± 0.327 g/dl respectively. Statistical analysis revealed significance difference ($P<0.01$) of the Hb between two groups. Low haemoglobin percentage in animals of Gr. II was indicative

of anaemia and the findings was corroborated with the reports of Goodman and Gilman (1990) and Biswas et al.(1998) who also recorded the decreased level of Hb in experimentally produced animals. The low level of haemoglobin in Gr. II might be attributed to interference the activity of enzymes for sulphur metabolism as it acted as analogues of the sulphur containing amino acids required for protein synthesis. The anaemia or detoriation of the level of Hb was due to interference of metabolism and suppression of bone marrow as a residue of toxicant. TEC of Gr. I and Gr. II were 6.764 ± 0.133 (x10^6) /µl and 5.477 ± 0.096 (x10^6) / µl respectively. The values of TEC in Gr. II dropped significantly (P<0.01) compared to Gr. I (table.1) what was corroborated with the reports of Fusari and Ubaldi (2000) who recorded the decreased level of TEC in arsenic toxicated cows.The reports of the present study was corroborated with the report of Biswas et al. (1998) in experimentally produced animals who also recorded decreased level of TEC which was suggestive of suppression of bone marrow. The mean values of Total Leucocytic Count (TLC) of Gr. I and Gr. II were found to be 6.375 ± 0.106 (x10^3)/ µl and 6.025 ± 0.086 (x10^3)/ µl respectively.The values of TLC of Gr. II decreased significantly (P<0.05) in comparison to Gr. I (table.1). The report was simulated with the reports of Ianchev (2001).The dropped values of TLC might be ascribed to suppression of granulopoietic action of bone marrow as a result of excessive intake of arsenic through drinking water and plants. TSP levels were 6.741 ± 0.107 g/dl & 6.230 ± 0.066 g/dl for Gr. I and Gr. II respectively. The levels of TSP of animals of Gr. II decreased significantly (P<.01) in comparison to Gr.I. The dropped level of TSP was also observed by Pandey and Misra (1985), Sarkar and Misra (1991) in anaemic animals. Depletion of TSP level suggests that the cattle tried to compensate the decrease level of blood glucose level by the process of catebolising protein & concomitant gluconeogenesis. Decrease level of TSP was due to extensive damage to capillaries causing increased permeability and exudation of serum into tissue spaces. The mean values of glucose was significantly higher (P<0.01) in Gr. II (50.631 ± 0.673 mg/dl) than Gr. I (47.029 ± 0.772 mg/dl).The analytical results of the analysis indicated the increase level of blood glucose significantly (P<0.01).Ghosh et al. (1993) and Biswas et al.(1998) also recorded the increase level of blood glucose in experimentally produced arsenic toxicity in goats and in natural cases of selenium toxicity in buffaloes. The augmented level of blood glucose in spite of inappetance might be attributed to stress factors.

The increased release of glucocorticoids secretion by the adrenal cortex, receiving the stimulation from anterior pituitary and there by it caused gluconeogenesis through rapid mobilisation of amino acid and glucose needed by the different tissues of the body and decreased peripheral utilisation of glucose by the cells or increased glycogenolysis. The values of AST of Gr. I and Gr. II were 28.095 ± 0.631 IU/l & 33.771 ± 0.577 IU/l respectively. The value of AST increased significantly (P<0.01) than the animals of Gr.I suggesting the possibility of alteration in the cell metabolism of liver as a result of toxic effects of arsenic and leaking out into the blood from the damaged tissues. The increased value of AST was stimulating with the observation of Biswas et al. (2000) in experimentally produced arsenic toxicity in goats. The mean values of ALT in serum were 6.478 ± 0.178 IU/L & 7.566 ± 0.108 IU/ L respectively for Gr. I and Gr. II.The values of ALT in serum increased significantly (P<0.01) in comparison to Gr.I.The findings of the present study was consistent with the result of Ghosh et al. (1993), who recorded the increase level of it in chronic arsenic toxicity. The significant lowered level of ALT might be attributed to inhibition of enzymes synthesis and damage of excretory & secretary tissues as result of necrosis of the cell. Alteration of cell metabolism increases the enzyme activity & necrosis of the cell decreases the enzyme activity. The levels of BUN were 15.793 ± 1.023 mg/dl & 23.995 ± 0.736 mg/dl for Gr. I and Gr. II respectively. The BUN level of animals of Gr. II increased significantly (P<0.01)) in comparison to Gr.I.The rise of urea might be of the indicative failure of kidney to remove metabolic products. The increased level of BUN might be due to increased reabsorption of urea from renal tubules as a result of failure of the selective reabsorption property of kidney tubules. The creatinine level of Gr. I and Gr. II were 0.816 ± 0.034 mg/dl and 1.020 ± 0.031 mg/dl respectively.The value of creatinine level of Gr.II was significantly higher (P<0.01) than the control animal (Gr. I). Increased level of creatinine obviously indicated the sign of renal failure.

Figure 1

Table 1: Mean ± S.E. of the values of certain haematological changes of control (Gr.I) and affected (Gr.II) animals.

Parameters	Healthy control (Gr.I)	Affected (Gr.II)
Haemoglobin(g/dl)	10.800ᵃ± 0.327	8.300ᵇ± 0.221
Total erythrocytic count(TEC) (X10⁶/μl)	6.764ᵃ± 0.133	5.477ᵇ± 0.096
Total leucocytic count(TLC) (X10³/μl)	6.375ᵃ±0.106	6.025ᵇ±0.086
Total serum protein (TSP) (g/dl)	6.741ᵃ± 0.107	6.230ᵇ± 0.006
Blood glucose level (BGL) (mg/dl)	47.029ᵃ±0.772	50.631ᵇ±0.673
Serum aspertate amino transaminase(AST) (IU/l)	28.095ᵃ±0.631	33.771ᵇ±0.577
Alanine amino transaminase (ALT) (IU/l)	6.478ᵃ±0.178	7.566ᵇ±0.108
Blood Urea Nitrogen (BUN) (mg/dl)	15.793ᵃ±1.023	23.995ᵇ±0.736
Creatinine (mg/dl)	0.816ᵃ±0.034	1.020ᵇ±0.031

Superscript (a, b) denotes there is significance difference exist between two mean.

Significant value (P<0.01) indicates highly significant at 1% level.

Significant value (P<0.05) indicates significant at 5% level.

CONCLUSIONS

From the above results it can be concluded that the arsenic toxicity results in the significant haemoconcentration and ALT, AST, BUN and Creatinine estimation are most sensitive indicator assessing the liver and kidney damage.

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