Research Paper

Volume : 2 | Issue : 7 | July 2013 • ISSN No 2277 - 8179

Serum biochemical changes induced by imidacloprid

Veterinary

KEYWORDS : imidacloprid, serum biochemistry, vitamin C

S. Soujanya

Department of Veterinary Pathology, College of Veterinary Science, Rajendranagar, Hyderabad-500 030 (AP), INDIA

ABSTRACT

Effect of oral administration of imidacloprid on serum biochemical parameters was studied in male rats and the protective role of vitamin C was assessed against imidacloprid-induced toxicity. Forty eight male rats were divided into four groups of 12 animals each. Group 1 served as control, while groups 2 and 4 were administered with imidacloprid (80 mg/kg b.wt/day) by oral gavage for 28 days. In addition to imidacloprid, group 4 also received vitamin C @ 10 mg/kg. Group 3 was maintained as vitamin C control. At the end of 14th day, six rats from each group and the remaining at the end of 28th day were sacrificed. Blood samples were collected before sacrifice for sero-biochemical analysis. The biochemical assays showed a significant (P < 0.05) increase in ALT, AST, serum creatinine and decrease in total protein in group 2. Co-administration of vitamin C brought moderate protection in all the above parameters.

INTRODUCTION

Imidacloprid, a neonicotinoid insecticide, is extensively used in agriculture for control of the sucking insects and coleopteran *beetles (Cox, 2001). It is also used as flea control agent on dogs and cats (Hutchinson et al., 2001). It is one of the fastest sold insecticide across the world because of its high selective toxicity in insects and apparent safety in humans. It acts on nervous system by blocking post synaptic acetylcholine receptors (Tomizawa et al., 2005). T*wo fatal intoxication cases have been reported recently (Proenca *et al., 2005)*.

Vitamin C plays an important role in protection against insecticide-induced toxicity as an antioxidant agent and prevents the effect of free radicals on vital cells (Abd-El-Ghaney, 2002). Present study was conducted to evaluate the serum biochemical alterations induced by imidacloprid in male rats and protective role of vitamin C against imidacloprid-induced toxicity was also assessed.

MATERIALS AND METHODS

Chemicals

Imidacloprid was procured from GSP crop science Pvt. Ltd., Gujarat and Vitamin C was obtained from Abbott Health Care Pvt. Ltd., Bhiwandi.

Animals

Forty eight male *Sprague dawley* rats weighing 200-250 g were procured from National Institute of Nutrition (NIN), Hyderabad. The experiment was conducted as per CPCSEA guidelines and approved by the Institutional Animal Ethics Committee (Approval No. I / 3 / 2012).The rats were housed in polypropylene cages at lab animal house, College of Veterinary Science, Hyderabad and were maintained in controlled environment (Temperature 20-22^{0}C). they were provided *ad libitum* with standard pellet diet (procured from NIN) and water throughout the experimental period.

Experimental design

Following an acclimatization period of one week, the animals were divided into four groups consisting of 12 in each. Group 1 served as control, group 2 was treated with imidacloprid at the rate of 80 mg/kg b.wt, group 3 was treated with vitamin C at the rate of 10 mg/kg b.wt and group 4 was treated with both imidacloprid and vitamin C. These drugs were administered by oral gavage every day consequently for 28 days. At the end of 14th day, six rats from each group and remaining at the end of 28th day were sacrificed by cervical dislocation.

Serumbiochemistry

A day before sacrifice, 2 ml of blood was collected into a clot promoting vaccutainers and allowed to clot for 3 to 4 hours, later centrifuged at 20k rpm for 10 minutes, serum was separated into eppendorf tubes and stored at -20^{0}C. The stored samples were subjected to serum biochemical analysis {Alanine amino transferase (ALT), Aspartate amino transferase (AST) as per International Federation of Clinical Chemistry (IFCC) method (Shaw *et al., 1983), serum creatinine by modified Jaffes reaction as per alkaline picrate techniques (*Burtis and Ashwood, 1999) and serum total protein as per the standard Biuret procedure (Benjamin, 2001)} using semi-automatic biochemical analyzer by employing standard biochemical kits

Statistical analysis

The data were subjected to statistical analysis by applying one way ANOVA. Differences between means were tested using Duncan´s multiple comparison test and significance was set at P < 0.05 (Snedecor and Cochran, 1994).

RESULTS

The biochemical assays showed a significant (P < 0.05) increase in serum creatinine, ALT and AST, and decrease in total protein on day 14, and a similar trend was observed on day 28 in group 2 (Table 1).

DISCUSSION

The increased levels of ALT and AST could be due to degeneration and necrosis of hepatocytes, which attributes an increased permeability of cell membrane that results in release of transaminases into the blood stream. These results are in accordance with (Bhardwaj *et al., 2010, Kammon et al., 2010 and Mohany et al., 2012).* Increase in serum creatinine levels might be due to failure of glomerular filtration process induced by imidacloprid toxicity to kidney. Decreased total protein levels were in accordance with observations of (Siddiqui *et al., 2007)* and it may be attributed to imidacloprid induced hepato toxicity which was also evident from elevated serum ALT and AST activities.

Vitamin C plays primary role in neutralizing free radicals, it can work both inside and outside the cells to combat free radical damage. The free radicals will seek out an electron to regain their stability, vitamin C is an excellent source of electrons so it can donate electrons to free radicals such as hydroxyl and superoxide radicals and quench their reactivity (Bindhumol *et al.,* 2003). In the present study, supplementation of vitamin C brought moderate protection in all the above parameters.

CONCLUSION

Present study revealed that exposure to imidacloprid (80 mg/kg) in male rats resulted in alterations in serum biochemical parameters. However, vitamin C supplementation along with imidacloprid to rats, manifested significant protective effects.

Table 1: Effect of oral administration of imidacloprid on serum biochemical parameters in male Rats

	Group 1		Group 2		Group 3		Group 4	
	Day 14	Day 28	Day 14	Day 28	Day 14	Day 28	Day 14	Day 28
Total Protein (g/dL)	6.71 ± 0.03^{a}	6.60 ± 0.01^{a}	5.82 ± 0.03^{c}	5.24 ± 0.03^{c}	6.66 ± 0.03^{a}	6.42 ± 0.03^{a}	6.28 ± 0.02^{b}	6.02 ± 0.10^{b}
Serum Creatinine (mg/dL)	1.36 ± 0.01^{c}	1.44 ± 0.01^{c}	2.40 ± 0.05^{a}	2.81 ± 0.03^{a}	1.40 ± 0.02^{c}	1.49 ± 0.01^{c}	1.91 ± 0.04^{b}	2.25 ± 0.07^{b}
ALT activity (IU/L)	30.65 ± 0.12^{c}	30.24 ± 0.08^{c}	61.51 ± 0.28^{a}	65.2 ± 0.38^{a}	30.98 ± 0.25^{c}	30.65 ± 0.29^{c}	53.51 ± 0.31^{b}	55.91 ± 0.36^{b}
AST activity (IU/L)	66.96 ± 0.19^{c}	67.38 ± 0.21^{c}	101.81 ± 0.27^{a}	121.53 ± 0.23^{a}	67.31 ± 0.35^{c}	67.66 ± 0.40^{c}	80.41 ± 0.32^{b}	91.1 ± 0.25^{b}

Values are mean ± SE (n=6), One way ANOVA, Means with different superscripts differ significantly (P<0.05)

REFERENCE

1. Abd-El-Ghaney A. (2002) Study the effect of imidacloprid insecticide on some physiological parameters in Japanese quail. Thesis for M.Sc. Faculty of science, Al-Azhar University. | 2. Benjamin M. M. (2001). Outline of veterinary clinical pathology, 3rd Edition, Colorado Stat University, U.S.A. | 3. Bhardwaj S., Srivastava M. K., Upasana K. and Srivastava L. P. (2010). A 90 days oral toxicity of imidacloprid in female rats morphological, biochemical& histopathological evaluation. Food and chemical toxicology, 48(5), p. 1185-1190. | 4. Bindhumol V., Chitra K. C. and Mathur P. P. (2003). A induces reactive oxygen species generation in the liver of male rats. Toxicology, 188, p. 117-124. | 5. Burtis C. A. and Ashwood E. R. (1999). Text book of clinical chemistry Ed by Teitz, N. W. 2nd edition PA WB saundres, p.1535. | 6. Cox C. (2001). Imidacloprid- Expert overview. Germany. Bayer; | 7. Hutchinson M. J., Jacobs D. E. and Mencke N. (2001). Establishment of the cat flea (Ctenocephalides felis) on the ferret (Mustela putorius furo) and its control with imidacloprid. Med. Veterinary Entomology, 15(2), p. 212-214. | 8. Kammon A. M., Brar R. S., Banga H. S. and Sodhi S. (2010). Pato biochemical studies on hepatotoxicity and nephrotoxicity on exposure to imidacloprid in layer chickens. Veterinarski arhiv, 80(5), p. 663-672. | 9. Mohany M., Badr G., Refaat I., Garraud O. and El-feki M. (2012). Thymoquinone ameliorates immunological and histological changes induced by exposure to imidacloprid insecticide. Toxicology Science, 37(1), p. 1-11. | 10. Proenca P., Teixeira H., Castanheira F., Pinheiro J. and Monsanto P.V. (2005).Two fatal intoxication cases with imidacloprid. Forensic Sci. Int., 153, p. 75–80 | 11. Shaw L. M., Stromme J. A., London J. L. and Theodorsen L. (1983). International Federation of Clinical Chemistry, Scientific Committee, Analytical Section. IFCC methods for measurement of catalytic conc. of enzymes. Journal of Clinical Chemistry & Biochemistry 2(10), p. 633-646. | 12. Siddiqui A., Choudhary M., Goriya H. V., Bhavsar S. K. and Thaker A. M. (2007). Evaluation of immunotoxic effect of short-term administration of quinalphos and imidacloprid in white leghorn cockerels. Toxicology international, 14 (1), p. 15-19. | 13. Snedecor G. W. and Cochran G. (1994). Statistical methods, 8th edition, IOWA State University Press, Amer, IOWA, USA. | 14. Tomizawa M., Lee D. L. and Casida J. E. (2005) Neonicotinoid insecticide toxicology, mechanisms of selective action. Annuals of Revised Pharmacology & Toxicology, 45, p. 247-268.