Research Article

Study of serum liver enzymes, Amylase and blood glucose level in acute organophosphorus poisoning

Pritee G Pendkar[1*], K S Ghorpade[2], G S Manoorkar[3], Amol Shinde[4]

[1]PG, [4]Assistant Professor, Department of Biochemistry, Dr S C Government Medical College, Nanded, Maharashtra, INDIA.
[2]Professor & HOD, Department of Biochemistry, Government Medical College, Akola, Maharashtra, INDIA.
[3]Associate Professor, Department of Biochemistry, Grant Medical College, Mumbai, Maharashtra, INDIA.
Email: priteependkar081@gmail.com

Abstract

Introduction: Acute Organophosphorus poisoning is widespread in developing countries like India specifically in rural area and its frequency is increasing. Acute organophosphate poisoning may cause the life-threatening events resulted in different organ failure. **Aim and Objective:** 1. To study level of serum liver enzyme and amylase and their correlation with severity of organo phosphorus poisoning 2. To estimate blood glucose level and its association with complication of organophosphorus poisoning during their hospital stay. **Material and Method:** Study was done in Dr S. C. Govt. Medical College and Hospital, Nanded. Ninety patients with acute organophosphate poisoning admitted to casualty were included in this study. Patients above 18 yr age were included and those patients with mixed poisoning, alcohol consumption and history of diabetes were excluded. Detailed history, clinical examination was done and clinical severity was graded according to WHO classification. In these patients serum ALP, AST, ALT, Amylase and random blood glucose level was done at day admission. **Result:** Statistically significant difference was obtained for AST, ALT, ALP and Amylase determined between the grades. Glucose conc. was found to be highest in grade 3 and higher in grade 2 compared to grade 1 ($p < 0.001$). Hyperglycemia observed in 36 % of patients out of which 73 % patients developed complication and 50% required ventilator. **Conclusion:** The marked increase of liver enzyme activity and serum amylase were appears to be associated with clinical severity in patients with acute organophosphorus poisoning. Hyperglycemia observed in patients of grade 2 and 3 and these are the patients who were associated with complications.

Keywords: acute organophosphorus.

[*]**Address for Correspondence:**
Dr. Pritee G Pendkar, Post Graduate Student, Department of Biochemistry, Dr S C Government Medical College, Nanded, Maharashtra, INDIA.
Email: priteependkar081@gmail.com

Received Date: 01/06/2015 Revised Date: 10/06/2015 Accepted Date: 12/06/2015

Access this article online	
Quick Response Code:	Website: www.statperson.com
	DOI: 14 June 2015

INTRODUCTION

Organophosphorus compounds share a major proportion in the commonly used pesticides. As per World Health Organization (WHO), three million cases of pesticide poisoning occur every year and most of them occur in Asia and at least 50% due to organophosphorus poisoning.[1] OP poisoning causes adverse effects on tissues and organ function[2,3,4,5] and they may lead to hyperglycemia. Constant increase in the number of cases of organophosphorus poisoning demands a detailed study with respect to various systems. Hence, this study was conducted to investigate the relationships between the initial emergency laboratory parameters and degree of organophosphate intoxication admitted to casualty.

AIM AND OBJECTIVE

- To estimate level of serum liver enzyme, amylase and their correlation with severity of organophosphorus poisoning.
- To estimate blood glucose level and its association with complication of organophosphorus poisoning during their hospital stay.

How to site this article: Pritee G Pendkar, K S Ghorpade, G S Manoorkar, Amol Shinde. Study of serum liver enzymes, Amylase and blood glucose level in acute organophosphorus poisoning. *International Journal of Recent Trends in Science and Technology* June 2015; 15(2): 342-344 http://www.statperson.com (accessed 15 June 2015).

MATERIAL AND METHODS

Study was conducted in Dr S C Govt. Medical College and Hospital, Nanded. The study was approved by local ethical committee and after obtaining informed written consent detailed history, examination and investigations were done. We had taken 90 patients with acute organophosphorus poisoning admitted to casualty after their diagnosis.

Inclusion Criteria

Patients were selected irrespective of sex but cases above 18 yrs were included in study. Patients with history of consumption of organophosphorus compound presenting within 24 hrs were included in study.

Exclusion Criteria

Patients with age less than 18 yr, patients with history DM, patients already treated at other centers and referred to our center for further management and patients who had consumed alcohol, other poisoning drug, mixed poisoning were excluded from study. Clinical severity was graded according to WHO classification.

WHO classification for severity

Severity	Sign/symptoms
Grade I	Anorexia, headache, dizziness, weakness, anxiety, tremors of tongue and eyelids, miosis, impairment of vision.
Grade II	Nausea, salivation, lacrimation, abdominal cramps, vomiting, sweating, slow pulse, muscular tremors.
Grade III	Diarrhea, pinpoint pupils and non reactive pupils, respiratory difficulty, pulmonary edema, cyanosis, loss of sphincter control, convulsion, heart block, coma.

All patients underwent following investigation

1. Serum ALT by IFCC method without PLP.
2. Serum AST by IFCC method without PLP.
3. Serum ALP by AMP method.
4. Serum Amylase by CNPG method.
5. Admission blood glucose level by GOD – POD method.

All these test are performed on XL – 640 chemistry auto analyzer and these patients were followed up during their hospital stay.

Statistics

Statistical analysis is carried out by one way ANOVA test and chi square test.

RESULT

Table 1: Laboratory values on the day of admission in organophosphorus poisoning

Parameter	grade I (n = 58)	grade II (n = 24)	grade III (n= 8)	p value
AST U/L	26.68 ± 12.5	36.46 ± 21.4	48 ± 34.7	< 0.05
ALT U/L	16.34 ± 10.27	32.46 ± 19.3	44 ± 32.7	< 0.05
ALP U/L	72.23 ± 11.4	94.3 ± 16.5	124.7±22.4	< 0.05
AMY U/L	112.6 ± 92.8	265.3 ± 252.2	520 ± 302.1	< 0.05
GLU mg/dl	164 .5 ± 56.2	224.6 ± 90.1	264.1±123.7	< 0.05

In present study, 32 (36 %) patients were hyperglycemic on day of admission and 58 patients were normoglycemic. *BSL>200 mg% is taken as hyperglycemia.* In present study, 36 (40%) patients were developed complication. Respiratory failure was most common complication (i.e. 30 %) and other complications were convulsion*, ARDS,* pneumonia, cardiac arrest, acute pancreatitis.

Table 2: Association between BSL on day of admission and complication

Blood sugar	Complication present	Complication absent	p value
Hyperglycemic	23 (72%)	9 (28%)	p < 0.05
Normoglycemic	13 (22%)	45 (78%)	

χ^2 = 21 and odds ratio= 9

Table 3: Association of Hyperglycemia and need of ventilator

Blood glucose	Ventilator		p value
	Yes	No	
Hyperglycemia	16 (50 %)	16 (50 %)	p < 0.05
Normoglycemia	13 (22%)	45 (78%)	

χ^2 = 9.2 and odds ratio= 3.5

DISCUSSION

In our study, level of all hepatic enzymes was found to be increased in all grades of OPP and increased values are present in association with disease severity[1]. Clark *et al* [7] showed that hepatic enzymes can increased following the acute OPP. In our study, we found higher glucose levels in patients of grade I but much more in grade II and III compared to reference range. Yurumez Y *et al*[4] and Kerem M *et al*[5] had demonstrated that abnormal liver function tests, hepatic necrosis, liver necrosis of mid-zonal type and fatty change can occur in OPP. In the present study, serum amylase was considerably high in patient grade II and III and acute pancreatitis was appeared in three patients of these grades. Singh *et al*[8] also found hyperamylasemia and acute pancreatitis in patients with OPP.[8] Matsumiya N *et al*[9] and Li T Nagayama N *et al*[16] stated that Elevated serum amylase activity in absence of clinical pancreatitis could be attributed to acute hypoxemia. Dressel *et al* showed that O P compounds cause an increase in intraductal pressure and exocrine pancreas flow rate which results in extravasation of fluid.[15] O P compound inhibit cholinesterase allowing accumulation of acetylcholine at cholinergic sites resulting in continuous stimulation of cholinergic fibers and leading to marked increase in catecholamine and cortisol.[7,10,11] In addition, excessive adrenergic influences on metabolism causes glycogenolysis leading to hyperglycemia[7]. In present study 32 patients were hyperglycemic. These finding correlate with study of Sangur *et al*[13] and Shobha *et al*[12]. In this study, it was observed that admission

hyperglycemia above 200mg% was associated with complication in 73% patients as compared to 22 % patients in normoglycemia. This is in accordance with the study of Goel *et al*,[12] Sangur *et al*.[13] In addition hyperglycemia also showed a significant association with need of ventilatory support ($p<0.05$). 50% of patient with hyperglycemia were found to needed ventilatory support as compared to 22% with normoglycemia. The above results indicate hyperglycemia >200 mg% is good marker for predicting morbidity and also assessing need for ventilatory support.

CONCLUSION

- The marked increase of liver enzyme activity and serum amylase were appears to be associated with clinical severity in patients with acute organophosphorus poisoning.
- Hyperglycemia observed in patients of grade II and III and these are the patients who were associated with complication.
- Hence this study suggests that hyperglycemia >200 mg % is good marker for predicting morbidity and also assessing need for ventilatory support. In this regards further research is needed.

REFERENCES

1. Singh S. organophosphorus poisoning: an evidence based approach. MJAFI 2004; 60; 2-4.
2. Hsiao C-T, Yang C-C, Deng J-F, Bullard MJ, Liaw S-J. (1996) Acute pancreatitis following organophosphate intoxication. Clin Toxicol. 34 (3): 343-47.
3. Agostini M, Bianchin A. (2003) Acute renal failure from organophosphate poisoning: a case of success with haemofiltration.Hum Exp Toxicol.22: 165-67.
4. Yurumez Y, Ikizceli I, Sozuer EM, Soyuer I, Yavuz Y, Avsarogulları L, and Durukan P. (2007) Effect of interleukin-10 on tissue damage caused by organophosphate poisoning. Basic Clin Pharmacol Toxicol. 100: 323-27.
5. Kerem M, Bedirli N, Gürbüz N, Ekinci O, Bedirli A, Akkaya T, Sakrak O, Pasaoglu H. (2007) Effects of acute fenthion toxicity on liver and kidney function and histology in rats. Turk J Med Sci. 37 (5): 281-88.
6. Singh S. Blood glucose changes following anticholinesterase insectisides poisoning. JAPI 200; 48; 1145 – 46.
7. Clark RF. (2002) Insecticides: Organic phosphorus compounds and carbamates. In: Goldfrank's Toxicologic Emergencies, 7 edn, The McGray-Hill companies, Inc. Chapter 88, page: 1346-1357. 8. Singh S, Bhardwaj U, Verma S, Bhalla A, Gill K. (2007) Hyperamylasemia and acute pancreatitis following anticholinesterase poisoning. Hum Exp Toxicol. 26 (6): 467-71.
8. Singh S, Bhardwaj U, Verma S, Bhalla A, Gill K. (2007) Hyperamylasemia and acute pancreatitis following anticholinesterase poisoning. Hum Exp Toxicol. 26 (6): 467-71.
9. Matsumiya N , Tanaka M, Iwani M N, Kondo T, elevated amylase related to development of respiratory failure in OPP, Human and Exp. Toxicology 1996 ; 15 : 250-253.
10. Schrickel JW, Lewalter T, Lüderitz B, Nickenig G, Klehr HU,Rabe C. (2009) Recovery from ultra-high dose organophosphate poisoning after "in-the-field" antidote treatment: potential lessons for civil defence. J Emerg Med. 37 (3): 279-82.
11. Ikizceli I, Yurumez Y, Avsaroğulları L, Kucuk C, Sozuer EM, Soyuer I, Yavuz Y, Muhtaroglu S. (2005) Effect of interleukin-10 on pancreatic damage caused by organophosphate poisoning. Regulatory Toxicol Pharmacol. 42: 260-64.
12. Goel A, Joseph S, Dutta T K, organophosphate poisoning: predicting need for ventilatory support JAPI 1998: 46(9): 786-790. 13. Sungur M, Guven M. Intensive care management of organophosphate insectiside poisoning, crit.care 2001; 5(4); 211-5.
13. Shobha T R, Prakash O, Glycosuria in organophosphate and carbamate poisoning. JAPI 2000; 48(12).
14. Dressel T D, Goodale R L, Arneson M A, Borner I W, pancreatitis as complication of anticholinesterase insectiside intoxication, Ann Surg 1979; 189: 199-204.
15. Li T Nagayama N, Kawade Y, Ohtuska Y, Machida K , Haga T, Hyperamylasemia in acute excerbation in patient with chronic respiratory failure. Nihon kyoba skikkan Gakkai Zasshi 1989, Apri: 27 (4):488-96.

Source of Support: None Declared
Conflict of Interest: None Declared