ORIGINAL RESEARCH

Plasma Nt-Pro Bnp: Early Marker of Cardiac Toxicity in Patients with Acute Organophosphate Poisoning

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ABSTRACT

Introduction: Organophospate (OP) poisoning is associated with an increasing incidental or suicidal deaths , with wide use of pesticides in the developing world. Hence early recognization ofcardiac toxicity induced by OP is need of the hour. **Aims and objectives**: To assess the incremental benefit of plasma N-terminal pro brain natriuretic peptides (NT- proBNP) as an early marker of cardiac toxicity induced by acute OP poisoning and to compare its beneficial value over the well-assessed cardiac marker, CPK MB and cardiac Troponin I (cTnI)". **Material and methods:** This study consisted of 50 OP poisoning patients. All patients were subjected to full history regarding amount of OP consumed and through physical examination. Estimation of plasma NT pro BNP and Cardiac marker CPK MB and Troponin (TNI) was done by Immuno Fluorence Technique. **Observations and results:** Sinus tachycardia was common in OP poisoned patients. Among cardiac markers, CPK MB and Troponin I was not significantly raised (p value 0.047) in OP poisoned patients with cardiac toxicity .However there was marked raised plasma NT -Pro BNP levels in these patients with p value - <0.0001). **Conclusion:** NT-pro BNP is better cardiac marker than CPK MB and cTnI, for detecting silent earlymyocardial damage that occurs in OP poisoning patients.

INTRODUCTION

Due to the current widespread use of pesticides, Organophosphorus (OP) poisoning remains an important cause of morbidity and mortality in developing countries.[1] Cardiac complications often accompany in two third of cases of OP poisoning,[2] which are potentially preventable if they are recognized early and treated adequately.

AIMS AND OBJECTIVES

To evaluate role of plasma N-terminal pro brain natriuretic peptides (NT-proBNP) as an early marker of cardiac toxicity in patients with acute OP poisoning and to compare it with cardiac marker CPK MB and Troponin I (cTnI)".

MATERIAL AND METHODS

The study was conducted in Jawahar Lal Nehru Hospital Ajmer. This was a cross sectional, prospective study which consisted of 50 OP poisoning patients admitted in casualty ward. Institutional Ethical committee approval and informed consents from all the studied individuals were obtained. The diagnosis of OP poisoning was based on toxic exposure to an OP compound, evidenced by history, the unique impressive clinical presentation, hints of pesticide-like smells from body fluids .The diagnosis of cardiac toxicity was based on presence of hemodynamic instability with regards to pulse, blood pressure and ECG changes. All patients were subjected to detailed history regarding circumstances of exposure, amount of OP consumption, time elapsed between exposure and admission to hospital and any underlying associated chronic disease. Physical examination including vital signs, gastrointestinal, respiratory, neurologic and cardiovascular systems were assessed in detail. Estimation of plasma NT pro BNP and Cardiacmarker CPK MB and Troponin (TnI) was done by Immuno Fluorence Technique. Patients with a history of ishaemic heart disease, trauma , malignancy , chronic malnutrition, liver disease, old age and pregnant females were excluded from the study. Patients on drugs like anticholinergics, bronchodilators, antidepressants and antipsychotics were also excluded.

OBSERVATIONS AND RESULTS

In our study nicotinic manifestations such as

tachycardia and hypertension were the most common cardiac signs on presentation being present in 65% and 60% respectively and 45% of OP poisoned cases presented with sub-normal temperature (<37.2C) . (Table-1) Nt Pro BNP value was significantly raised in OP poisoning patients as compared to CPK-MB and Troponin I (P value<0.0001) (Table-2)

Table 1 Vital parameters of the studied organOphosphorus Poisoned Cases (N=50)

Vital Parameter	% Of Subjects
(A) Pulse	
>100/mt	65
50-100/mt	20
<50/mt	15
(B) Blood Pressure	
<90/60mmHg	28
90/60—120/80mmHg	12
>120/80mmHg	60
(C)Temperature	
>37.4 ° C	55
<37.4 ° C	45

Table 2: Cardiac markers in organophosphorus poisoned patients

Cardiac Marker	Control group	Organophosphorus poisoning group	P value
CPK MB(I/L)	6.5+5	28+3	0.127
Troponin I (ng/ml)	0.09+0.001	0.20+0.003	0.047
NT-ProBNP (pg/ml)	143+64.5	405+45	<0.0001

DISCUSSION

Cardiac complications associated with OP mostly occur during the first few hours or even as the initial presentation (3) Agarwal et al.(4) found that the nicotinic manifestations such as tachycardia and hypertension are the most common cardiac signs on presentation. We also found the same in our study. However, on the other hand, Davis etal (5) documented that hypotension may occur secondary to augmented vagal tone with muscarinic over stimulation of vascular receptors by excessive circulating acetylcholine causing peripheral vasodilatation. As regard axillary temperature, 46% of OP poisoned cases presented with sub-normal temperature (<37.2C). Similar results were previously reported by Moffat etal (6). Some specific mechanisms have been reported to result in cardiac complications in OP poisoning including: (1) sympathetic over-activity at first, followed by prolonged extreme parasympathetic activity, which is responsible for Q-T prolongation followed by polymorphous ventricular tachycardia (Torsades de Pointes) and ventricular fibrillation. Joshi et al. (7) reported a case of OP followed by acute myocardial infarction, which they attributed to cardiac complications of OP. Cha et al. (8) investigated the prevalence of myocardial injury through cardiac biochemical markers such as troponin I (TnI), creatine kinase MB, and B-type natriuretic peptide in severe OP. They found that OP can cause direct myocardial injury during the acute phase, and monitoring of TnI may be needed in severe cases based on the Namba classification. However, more than 80% of severe cases in that studypresented normal sinus rhythm regardless of the TnI level, which weakened the customary use of ECG alone for diagnosing cardiac complications in OP. Results of that study emphasized the importance of monitoring myocardial injury with biochemical markers rather than by ECG changes. Measurement of NT-pro BNP represents an interesting strategy for detecting subclinical cardio toxicity and reflects the hemodynamic status of the heart (9). Jankowski etal (10) and Felker et al.(11) reported that if NT-pro BNP is elevated > 450 pg/ml in patients < 50 years, heart failure is likely. In this study NT-Pro BNPwas significantly higher in all OP poisoned cases.

CONCLUSION

The study concludes, that NT-pro BNP can be used as a early diagnostic tool forsilent myocardial damage that may occur during the course of acute OP poisoning over cTnI and CK-MB for starting early cardio-protective treatment.

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