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General Medicine

A STUDY ON RED CELL DISTRIBUTION WIDTH AS A PREDICTOR FOR VENTILATORY SUPPORT AND MORTALITY IN PATIENTS WITH ORGANOPHOSPHATE POISONING

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ABSTRACT **Background:** Organophosphorous poisoning is one of the leading medico toxic emergency in india. **Aims And Objectives:** The aim of this study is to investigate the relation between red cell distribution width and final outcome and also to prove it as an outcome predictor for ventilator support in individuals with OP compound poisoning **Methodolgy:** This is a cross sectional study of 50 patients with OP poisoning admitted at government hospital medical ward, Kurnool.The severity of poisoning,important biochemical,clinical parameters were measured and the requirement for ventilator support were studied accordingly. **Results:** 72% of individuals fall in the age group of 20 to 40 years with male to female ratio 1.5:1. A total of 64% are from rural areas with most of them were agriculture labourers.60% of patients belonged to low socioeconomic status. Profenofos was consumed by 38% and monocrotophos was consumed by 28%.Most common symptom found was excessive salivation(78%) followed by vomiting(52%).Most common sign found was miosis in 94% and bradycardia in 62%.Respiratory failure was most common cause of death in this study.Alcoholism was found in 38% individuals.22% individuals had thrombocytopenia. Raised RDW was found 40%.Percentage of individuals ventilated among raised RDW was 65% while 45% individuals were dead among raised RDW and percentage of deaths among ventilated individuals with raised RDW was 69.23%.Raised RDW had a significant correlation with poor prognosis and risk for ventilation. **Conclusion:** was 65% while 45% individuals were dead among raised RDW and percentage of deaths among ventilated individuals with raised RDW was 69.23%.Raised RDW had a significant correlation with poor prognosis and risk for ventilation.

KEYWORDS : organophosphate poisoning, red cell distribution width ,ventilatory support, mortality

INTRODUCTION:

For greater than 50 years, organophosphates have been utilised as pesticides all over the world. Because of their toxicity, their use has reduced in the preceding 10 to 20 years. Organophosphates are used for glaucoma, myasthenia gravis, and Alzheimer's disease therapeutically for reversing neuromuscular inhibition (eg., neostigmine, edrophonium, pyridostigmine, echothiophate, tacrine, and donepezil).

3,000,000 men and women are exposed internationally every year, with up to 300,0002,3 deaths. Most commonly via exposure to agricultural pesticides. Consumption of contaminated food and wearing contaminated clothes also different causes for poisoning. Chlorpyrifos is present in wide range of famous roach and ant sprays. Chlorpyrifos now banned by US, Environmental Protection Agency (EPA) in 2001 for domestic use 4 . Several organophosphorus nerve poisons have been created in Germany during the 1940s, such as tabun [GA], sarin [GB], and soman [GD], however they have been no way utilised for army purposes5 . Prevention, detection, and remedy of organophosphorus ("nerve") agent-related casualties is alarming the globe .High Red Cell Distribution Width (RDW) readings have been linked to a terrible prognosis in people struggling from acute myocardial infarction, congestive coronary heart failure, stroke, and sepsis1,2,6. Elevated RDW is related with acute inflammation and accelerated oxidative stress7,8, and it is a easy and cheaper diagnostic. We want less expensive and easily measurable biomarkers in a resource-constrained country like India. We hope to see if RDW can be used as a prognostic marker for Organophosphorus toxicity in this study. Leading cause of death in OPC poisoning is Respiratory failure. Respiratory Failure due to acute 2 cholinesterase crisis is the most frequent complication of OPC poisoning which leads to death.

Early attention of poisoning and instantaneous ventilator support may also enhance survival. It is consequently necessary that scientific methods and standards to predict the need for ventilatory aid to be recognized at preliminary examination. Serum cholinesterase level is decreased after OPC poisoning. Peradenya OP compound scale is easy scale used to pick out the severity of OPC poisoning. In this study, the medical situation of ventilated sufferers due to OPC poisoning used to be taken and tried to link the correlation between more than a few elements to predict the mortality and morbidity due to the OPC poisoning. This study is undertaken to analyze the function of red cell distribution width (RDW) as a biochemical parameters in predicting the severity of OPC poisoning amongst the ventilated sufferers and its outcome.

AIMS AND OBJECTIVES:

1. To investigate the relation between red cell distribution width and final outcome in patients with Organophosphate poisoning.
2. To analyze the correlation between the biochemical parameters and mortality among Organophosphate poisoning patients.
3. To analyze validity of red cell distribution width and prognosis in Organophosphate poisoning.
4. To prove relationship between red cell distribution width as a prognostic marker for mechanical ventilation among poisoned individuals.

PERIOD OF STUDY: 1.5 Years

NUMBER OF CASES: 50

DESIGN OF THE STUDY: crossectional study

Inclusion Criteria:

All ingestional and inhalational op compound poisoning Patients between age 13 to 60 years with initial clinical assessment

Exclusion Criteria:

1. The subtype of organophosphate poison and their fatal outcomes were not studied separately
2. Levels of folic acid ,Vitamin b12 and serum iron that might influence RDW were not included in this study.
3. Recent hemorrhage, chronic liver disease, prior chemotherapy were excluded.

METHOD OF COLLECTION OF DATA:

All patients had undergone thorough clinical examination ,vital signs

like pulse rate ,blood pressure,temperature,respiratory rate,conscious levels by GCS and systemic examination were done.Diagnosis of OP poisoning was done bedside by using POP scale.

Investigations For Diagnosing op Compound Poisoning: Serum Cholinesterase Estimation was done.

RESULTS:
A total of 50 cases of organophoshate poisoning were studied.

RED CELL DISTRIBUTION WIDTH
Table 1 : Red Cell Distribution Width

RDW	NO OF PATIENTS	%
RAISED	20	40%
NORMAL	30	60%
	50	100%

40% of poisoning cases had raised values of RDW. 60% of remaining individuals had normal RDW (Red cell Distribution Width).In the present study RDW values were taken after >1 day of admission to hospital.

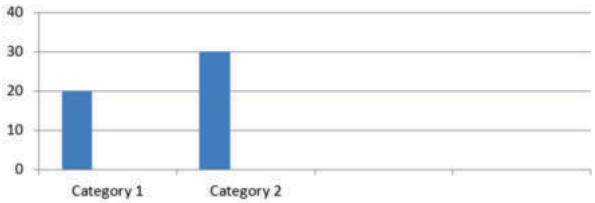

Figure 1: Ventilated Patients
category 1: ventilated category 2:non ventilated

Table 2 :Deaths And Survived During Study:

DEATHS	13	26%
ALIVE	37	74%
TOTAL	50	100%

Out of 50 individuals of this study 20 persons were ventilated. This study included all sort of patients irrespective of their clinical status at presentation to emergency department.

Ventilated Among Raised Rdw
Table 3 : Ventilated Among Raised Rdw Individuals

NO OF PATIENTS(n=20)	%
13	65%

Deaths Among Raised Rdw Individuals
Table 4 : Number of Deaths Among Raised Rdw Individuals

NO OF PATIENTS(n=20)	%
9	45%

Deaths Among Ventilated Individuals With Rasied Rdw
Table 5 : Number of Deaths Among Ventilated Individuals With Rasied Rdw

NO OF PATIENTS(n=13)	%
9	69.23%

DISCUSSION:
In growing nations like India, organophosphate insecticides (OPIs) are regularly employed in horticulture and agriculture. Poisoning with them is pretty familiar due to their easy availability. Suicide with the consumption of OPI is a massive public health issue, with an estimated 200,000 deaths per year worldwide. These pesticides are amongst the hazardous pesticides in India, poisoning humans, commonly farmers. Organophosphates and carbamates, regardless of their chemical differences, have comparable medical toxicity and require comparable care in overdosing.

Types of organophopshate compounds

INSECTICIDES	NERVE GASES	ANTIHELMINTHIC AGENTS	HERBICIDES
Malathion	saman	trichorfon	Tribufos
Parathion	sarin		Merphos
Diazinion	tabun		
Fenthion	Vx		
Dichlorvos			
Chlorpyrifos			
Ethion			

ROUTES OF EXPOSURE:
1. cutaneos
2. inhalational
3. ingestional
4. injection

MECHANISM OF ACTION:
Op compunds contain carbon and phosphorous acid derivatives. These agents are well absorbed through skin, lungs, GI tract and Irreversibly bind to serine –OH group at the active site of acetylcholinesterase (AChE) & establish covalent bond (phosphorylation).AGING refers toloss of alkyl group + strengthening of covalent bond.Phosphorylated AChE is very stable.Inhibition of AChE enzyme activity.No hydrolysis of Ach to choline & acetic acid leading to Accumulation of Ach in the synapse and NMJ and Overstimulation of cholinergic receptors.

Clinical Features:
Acute toxicity from Ops derive from excessive stimulation of muscarinic & nicotinic cholinergic receptors by Ach in the central & autonomic nervous systems & at skeletal neuromuscular junctions.

- Toxic features usually obvious in 30min to 3 hours or can be delayed in some cases depending upon the rate & amount of systemic absorption.
- Toxicity is produced by rapid absorption of the compound through the GIT, respiratory tracts & skin.
- Majority of patients give H/o intentional or accidental ingestion of op compounds.
- Clinical symptoms & signs depend on the specific agent , quantity & route of entry.

1. CARDIAC MANIFESTATIONS:
*Most common cardiac manifestations following poisoning are hypotension & bradycardia. Rarely pt can present with tachycardia & hypertension due to predominant nicotinic receptor stimulation.

*ECG MANIFESTATION : prolonged Q-Tc intervals , elevation of ST segment , inverted T waves & prolonged PR interval.

*RHYTHM abnormalities includes : sinus bradycardia , ventricular extra systoles, ventricular tachycardia & fibrillation.

***3 phases of cardiac toxicity:**
- Phase I : A brief period of increased sympathetic activity.
- PHASE II : A prolonged period of parasympathetic activity including AV node blockade.
- PHASE III : Q-T prolongation f/b torsade de pointes , ventricular tachycardia & ventricular fibrillation .

2. RESPIRATORY MANIFESTATIONS:
* In case of acute poisoning –bronchorrhoea, rhinorrhoea, bronchospasm & laryngeal spasm ---- d/t op compound action on muscarinic receptors.
* The airway integrity may be compromised by excessive secretions.
* The nicotinic effects leads to weakness of oropharyngeal muscles.
* This increases likelihood of both airway obstruction & gastric contents aspiration.
* Finally, central neurological depression leads to respiratory arrest.

3. NEUROLOGICAL MANIFESTATIONS :
*Three different types of paralysis are recognized based on time of occurrence & their differing pathophysiology :
- Type I paralysis / acute paralysis
- Type II paralysis / intermediate syndrome
- Type III paralysis / organophosphate – induced delayed polyneuropathy.

Type I paralysis / Acute paralysis
* Seen during the initial cholinergic phase.
* Due to persistent depolarisation of muscuranic & nicotinic receptors at the NMJ.
* c/f: muscle fasciculation, cramps, twitching & weakness.
* During this stage pt may require ventilatory support d/t weakness

of respiratory muscles leading to respiratory depression & arrest. Type II paralysis / Intermediate syndrome

* It is a distinct clinical entity which occurs 24 to 96hrs after ingestion of op compound.
* Approx. 10 – 40 % of patients treated for acute poisoning develop this illness. Onset is rapid with progression of muscle weakness from:

- Ocular muscles
- Neck muscle (patient can't raise the head from the pillow).
- Proximal limbs.
- Respiratory muscles (intercostals& diaphragm).

Clinical manifestations of IMS occur within 24 to 96 hrs & affect conscious patients without fasciculation or other cholinergic signs.
C/F :

- Marked weakness of neck flexion & varying degree of proximal limb muscle weakness, manifesting as weakness of shoulder abduction & hip flexion .
- Respiratory insufficiency is common & frequently draws medical attention .
- Other possible manifestations –involvement of muscles innervated by motor cranial nerves &decreased DTR.
- Sensory impairment is not a clinical manifestation.

*Proposed mechanism of IMS are :

1. Downregulation or desensitization of postsynaptic Ach receptors.
2. Prolonged Ach esterase inhibition.
3. Failure of postsynaptic Ach release.
4. Oxidative stress related myopathy.
5. Muscle necrosis.

Type III / Organophosphate –induced delayed polyneuropathy (OPIDN)

* It is a sensory – motor distal axonopathy that usually occurs after ingestion of large doses of an OP compound.
* Neuropathy presents as weakness & ataxia following a latent period of 2-4 wks.
* Initial stimulation causes excitatory fasciculations which then progresses to an inhibitory paralysis.
* Cardinal symptoms are distal weakness of hands & feet.
* This is often preceeded by calf pain, & in some cases paresthesias of distal part of limbs.
* Delayed CNS signs include tremor , anxiety & coma.

The following are the standards linked to a bad prognosis:

1. The longer it takes to begin therapy, the greater the fatality rate.
2. Mortality is inversely related to level of pseudo cholinesterase. The lower the level of pseudo cholinesterase, the greater the hazard of death.
3. Higher mortality rates observed in individual with poor GCS score at admission. The lower the GCS score, the greater the chance of death.
4. Early remedy with an appropriate dosage of atropine may also enhance survival chances.
5. Higher the red cell distribution width greater the risk for ventilation and more increase in mortality

CONCLUSION:

Ventilator requirement was more for patients with high RDW values. Mortality in the present study was 26% .Red cell distribution width an easy and cheaper method of predicting mortality amongst poisoned individuals and various others like Changwoo kang et al66. and Dundar et al68. showed significant association between red cell distribution and prognosis. Mahmaud et al.53 with a cut off RDW of 14.4% showed significant association among individuals with OP poisoning as RDW is one of the prognostic indicator for ventilator support. Present study also signifies the same with comparatively low sample size and less cutoff value of RDW i.e; >14%.

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