ORIGINAL RESEARCH

A Clinical Study on Organophosphorus Poisoning in a Rural Medical College

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ABSTRACT

Background: Organo Phosphorus Poisoning is very common in India, especially in rural area. It is a common pesticide used in the fields by Farmers. Accidental poisoning is also common with organo phosphorus pesticides while spraying in the fields. The first organophosphate insecticide was created in the mid 1800's. Organo phosphates are used as medications, Insecticide, Nerve gas agents as a weapon in the chemical war. **Aim of the Study:**To know the different clinical features and management of organophosphorus poisoning in a teaching hospital.**Materials and Methods:** This study has been conducted for 6 months from April 2022 to September 2022 in the department of General Medicine in GERMS Medical college, Junagadh.**Results:** We have examined total number of 260 patients. In our study, out of these 260. 118 are females and 142 are males 21 patients were died because of complications.**Conclusion:**Three facets of approach to the symptoms and signs in OP poisoning have been presented. Although all OP compounds are generally considered within a single group entity, it is recognized that di-methyl and diethyl OP poisoning have different outcomes.

Keywords: Organo Phosphorus Poison, Pinpoint Pupils, Mortality, Respiratory Paralysis, Pesticides.

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INTRODUCTION

Organophosphate (OP) poisoning continues to be afrequent reason for admission to hospitals and IntensiveCare Units in developing countries.(1) The traditional
approach to clinical features in acute OP poisoninghas centred on receptor specific effects on muscarinic,nicotinic and central nervous system (CNS) receptors that result in diverse symptoms and signs.(2) Thisconventional classification of clinical features is useful
given that muscarinic effects are reversed by atropinewhilst nicotinic neuromuscular effects are not.(3) Itis also known that drugs that cross the blood-brainbarrier (e.g. atropine) are more likely to reverse CNSsymptoms and signs than drugs that do not crossthe blood-brain barrier.(4) An alternate approach toclinical features may be in terms of the time of onsetof symptoms. In general, following OP exposure,Salivation, Lacrimation, Urination, Defecation, Gastriccramps, Emesis (SLUDGE) symptoms occur acutelywithin minutes to hours. However, some patients develop delayed effects either after an initial period of intense cholinergic symptoms and signs or aftera period of minimal or no clinical features. Furthersymptoms and signs may occur as a continuum, whereinpatients with acute symptoms involving one neuronalsub-system (e.g. neuromuscular weakness) may progressto develop delayed symptoms and signs of otherneuronal sub-systems (e.g. extra-pyramidal). The thirdapproach, an organ specific approach, have focusedon neurologic,respiratoryor cardiovascularеffects of OP. This review was thus undertaken to detaildifferentclassifications of the clinical features of OPpoisoning and discuss mechanisms for the occurrenceof these manifestations. The clinical features were classified as receptorspecific manifestations, based on time of occurrenceand nature of organ system involvement. Mechanismsfor the occurrence of specific manifestations, as wellas the time of symptom onset, were explored frompublished literature. Receptor based manifestations were categorized asnicotinic and muscarinic receptor manifestations.Irreversible binding of OP to acetylcholinesterase inthecholinergic synapses in the CNS and peripheralnervous system

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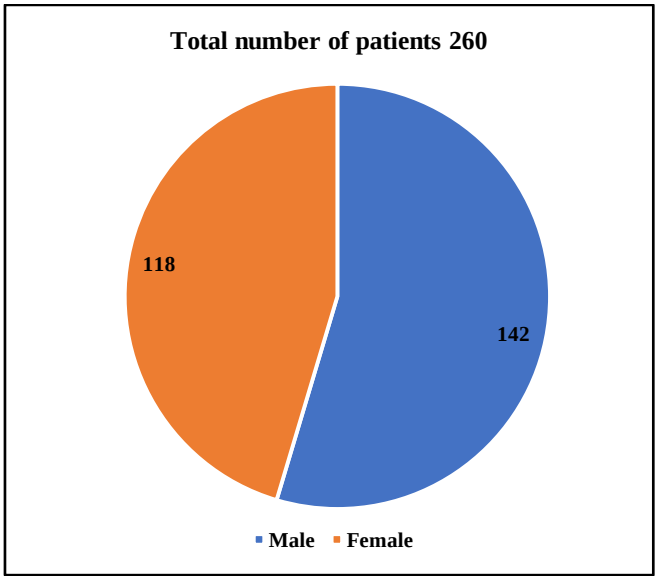
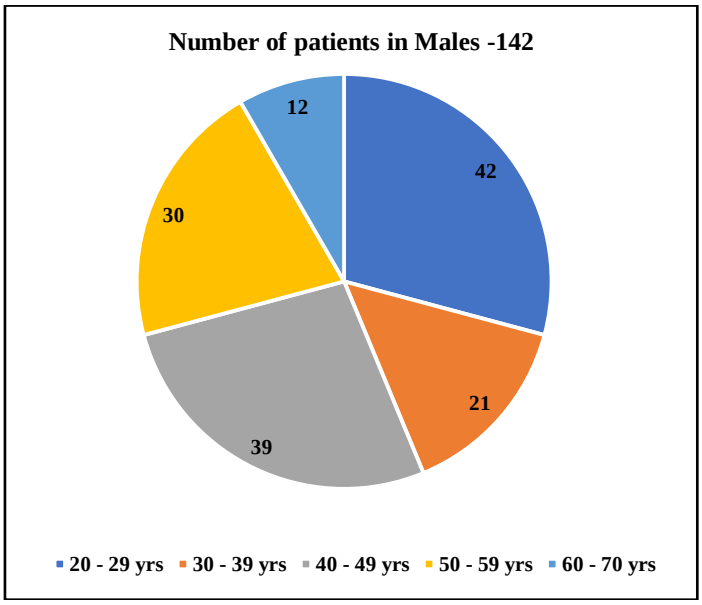
(PNS) results in high concentrationsof acetylcholine in the synaptic clefts that cause initialexcessive stimulation and later, blockade of synaptic transmission. The peripheral muscarinic SLUDGE symptoms are due to actions on the relevant glandswhilst central muscarinic effects result in symptoms suchas confusion, coma and convulsions. Nicotinic effectsare motor and sympathetic (5) and result in fasciculations, muscle weakness, tachycardia and hypertension. Ina retrospective study of OP poisoning, muscarinicsymptoms and signs were the most frequent (84%)followed by CNS (78%) and nicotinic (17%).

MATERIALS AND METHODS

This study has been conducted for 6 months from April 2022 to September 2022 in GERMS Medical College, Junagadh. In the department of General Medicines. In association with emergency medicine department. We have included total no.of 260 patients. In this study out of these 260 Male patients were 142 and 118 were Female patients 21 patients died because of complications and Respiratory paralysis. We have obtained consent by giving the consent forms in their Local Languages. After taking careful history we have examined all the patients in detail and advised investigations. The investigations advised are complete blood picture. Random blood sugar, Blood urea, serum creatinine, serum electrolytes, nerve conducted studies, after collection of data, systematically we have computerized by using MS Office.

RESULTS





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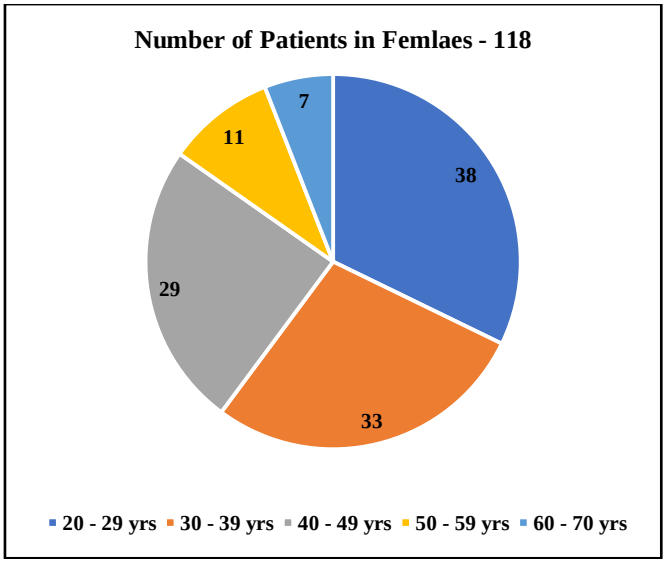

Table 1: Different age groups

Age in Years	Number of patients M(142)	Number of Patients F(118)
20 – 29 yrs	42 (29.5%)	38 (32.6%)
30 – 39 yrs	21 (14.7%)	33 (27.9%)
40 – 49 yrs	39 (27.46%)	29 (24.5%)
50 – 59 yrs	30 (21.12%)	11 (9.32%)
60 – 70 yrs	12 (8.5%)	7 (5.9%)

Table 2: Different Clinical Features

Clinical Features	Number of Patients M(142)	Number of Patients F(118)
Pinpoint Pupil	142(100%)	118 (100%)
Decreased level of consciousness	91(64.6%)	61 (52.7%)
Diarrhea	87(61.5%)	53 (44.3%)
Other symptoms	73 (50.5%)	51 (43.2%)

Table 3: Different Socio-economic groups

Different Income Groups	Number of Patients M(142)	Number of Patients F(118)
Lower income group	95 (72.1%)	78 (68.1%)
Middle income group	29 (19.4%)	24 (20.1%)
High income group	12 (8.5%)	9 (7.62%)

DISCUSSION

We have included total no.of 260 patients out of these 260, Males were 142 (54.2%) and females were 118 (45.8%) (6). The common age group is around 2nd and 3rd decade 29.5% and 14.7% is Males and 32.6% and 27.9% in Females. According to study conducted by Robenshtok E, Luria S, Tashma Zetal shows.33.2% and 20.5% in Males and 28.3% and 29.6% is Females (7). We observed in our study that Middle aged people, especially females were involved nearly 30% and mostly form rural area. This reflects the problems of farmers in agriculture sector in India. The common clinical features noticed in our study are bilateral constricted Pupils (100%) Diarrhea (61.5%) decreased level of consciousness in 69.6%. The study conducted by Singh S, Sharma Netal shows almost similar results; bilateral constricted Pupils (100%) and Diarrhea (71.5%) decreased level of consciousness (59.5%). (8) Pesticide poisoning is more common in rural area and in low socio-economic groups may be because of poverty, unemployment and other social factors. In our study we observed that (72.1%) patientsbelongs to low socio-economic status, in higher income group it is only 8.5% the study conducted by Karki P, Ansari JA etal shows 77.8% and 6.2% respectively. (9)21 patients died due to delayed in transportation and other co-morbid conditions like real failure and respiratory paralysis.

Organophosphate compounds bind irreversibly to acetylcholinesterase in the plasma, red cells andcholinergic synapses in the CNS and the PNS. Reduced red cell or plasma cholinesterase activitysuggests OP exposure. Red cell cholinesterase activity

is better correlated with the severity of exposure thanplasma cholinesterase activity.The central nicotinic receptors are of the neuronalsubtype (N2); this subtype is also present in theadrenal medulla and

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sympathetic and para-sympatheticganglia of the PNS. (10) The peripheral nicotinicreceptors (N1) are present in the neuromuscularjunction. All 5 muscarinic receptor subunits are present in the CNS.Peripheral parasympathetic muscarinic innervationis postganglionic to the heart, exocrine glands andsmooth muscle, while sympathetic postganglionic fibres innervate the sweat glands. Most symptoms and signs in OP poisoning are the resultof excessive muscarinic receptor stimulation. Featuressuch as tachycardia and high blood pressure, which are sometimes observed in acute poisoning and not readilyexplained is postulated to be due to overwhelmingcholinergic effects on the CNS, sympathetic ganglionicsynapses or the adrenal medulla. (11)

The traditional approach offers insight on the possiblesite(s) of action of the OP compound in patients withmuscle weakness. Wadia *et al*. reported that in theso-called Type I paralysis, weakness appeared within24-h and some responded to atropine. In contrast, inType II paralysis, weakness appeared after 24-h withconcomitant atropine being administered in large doses,usually, 30mg or more.(12) Recent electrophysiologicalstudies have suggested possible reasons for thisdifferential effect.Patients with moderate muscle weakness had an initialdecrement-increment pattern on electrophysiologyat high rates of stimulation progressing todecrement-increment patterns at intermediate-andlow-frequency situations. Further progressionwas characterized by decrement-increment andrepetitive fadepatterns.(13). Overstimulation of central receptors may contributeto early death. In addition, focalrespiratory centre seizures result initially in an increasein phrenic nerve output followed by sudden cessationof activity.

The time of occurrence of symptoms and signs dependon the route of exposure, poison load and chemicalnature and solubility characteristics of the compound.Traditionally, symptoms are categorized as acute (minutesto hours) and delayed or late (days to weeks). Thetime of onset and mechanism of delayed manifestationssuch as intermediate syndrome,delayed onset coma (14)and extrapyramidal manifestation are different tothat of late manifestations such as organophosphateinduced delayed polyneuropathy (OPIDP) thattypically occurs after 2-3 weeks and up to 4week postexposure. Thus, we propose that symptomonset is categorized as acute (within 24-h), delayed (24-hto 2-week) and late (beyond 2-week).

The acute symptoms and signs are due to muscarinic,nicotinic and central receptor effects. Muscarinicsymptoms of salivation and bronchorrhea that dominateinitially may cause drowsy patients to drown intheir secretions. Acute muscarinic effects on theheart(bradycardia, hypotension) can be life-threatening.Nicotinic effects of muscle weakness contribute torespiratory distress whilst the acute central effectsof restlessness, agitation, confusion and sometimesconvulsions further compromise airway and breathingand increase aspiration risk and hypoxia. Since manyof these effects are reversed by atropine, early andappropriate medical attention is vital. In developingcountries, where OP poisoning is common, quickaccess to medical care is more problematic than earlyrecognition.

With adequate atropinisation,(15) the acute cholinergicsymptoms abate within a few hours, but somepatients develop delayed effects. Several recentpublications strengthen the case for itsrecognition as a distinct clinical entity.Although acute cholinergicmanifestations typicallyoccur within 24-h of exposure, late onset cholinergicsymptoms andsigns have been observed 40-48 h afterdichlofenthion poisoning.Intermediate syndrome, the best described delayedmanifestation, is characterized by paralysis of proximallimb muscles, neck flexors, motor cranial nerves and respiratory muscles 24-96 h after poisoning, after thecholinergic phase had settled down, with weaknesslasting for up to 18-day. A neuromuscular junctionaldefect has been demonstrated in electromyographystudies. Delayed onset intermediate syndrome hasbeen reported Although intermediate syndrome involves musclegroups, focal weakness has also been reported; inparticular, laryngeal paralysis, either acuteor delayed by 4-14 days presenting as "failedextubating." Laryngeal electromyography was consistentwith bilateral laryngeal paralysis although standardneedle electromyography wasnormal.

CONCLUSION

Three facets of approach to the symptoms and signsin OP poisoning have been presented. Although allOP compounds are generally considered within asingle group entity, it is recognized that di-methyl anddiethyl OP poisoning have different outcomes.Eachindividual compound also has unique characteristics andoutcomes. Other differences such as lipid solubility, biochemical characteristics (oxon-thion), WHO classand nature of solvent used further make each OPcompound unique.

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