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STUDY OF VICTIMS OF ORGNOPHOSPHORUS COMPOUND POISONING IN A TERTIARY CARE CENTER

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ABSTRACT: OBJECTIVES: Indiscriminate use of organophosphorus compounds (OPC) increase the potential threat self-poisoning and death. To make a conclusive diagnosis on academic interest a retrospective record based study was undertaken. **METHODOLOGY:** Post autopsy studies were carried out on 100 victims of OPC poisoning by collecting data from following 3 sources.: Medicolegal autopsy reports, inquest reports and inpatient case sheets. Intention of ingestion of OPC was to commit suicide. Incidence was more in 20 to 30 years of age group, in females and in urban area. **RESULTS:** Diagnosis was confirmed by history of poison exposure, clinical well-defined cholinergic effects, and characteristic autopsy finding. Early and correct identification of the cause of OP poisoning enables specific treatment. **CONCLUSION:** Restrictions on access of very toxic pesticides through national polices and enforcement, public education on storage and safe use, early recognition of poisoning, and appropriate medical management may reduce the incidence of poisoning and death. **KEYWORDS:** OP Compound, antidote

INTRODUCTION: Organophosphorus compounds (OPC) are a diverse group of chemicals used in agricultural, domestic and industrial settings. Occupational exposures occur during manufacturing or use of OPC as insecticides. Intentional exposure for suicidal or homicidal attempt may cause fatal intoxication. Systemic effects appear very quickly within minutes after inhalation and are delayed in onset on oral and percutaneous absorption.

Manifestations of OPC poisoning include muscarinic, nicotinic and central nervous system (CVS) effects of acetylcholine. It presents as excessive salivation, sweating, involuntary defecation and urination, lacrimation, miosis, ocular pain with congestion, diminished vision, cold clammy skin, pallor, bradycardia, hypotension, loss of reflexes, muscle weakness, fatiguability, cheyne-stokes respiration, confusion, slurred speech, convulsions, drowsiness and coma. Sever intoxication (cholinergic crisis) may lead to death if not treated timely with antidotes (atropine and pralidoxime). Suicidal incidence is more because of the easy accessibility of OPC as pesticides.

Thousands of death occurs every year.¹⁻⁴ still, it is necessary to explore the cause of death and extent of manifestations of OPC poisoning correlating clinical and autopsy findings along with laboratory investigations for diagnosis. Better management and also for academic discussion.

Therefore we planned for a retrospective study aiming to evaluate manifestations of OPC poisoning correlating with autopsy findings along with demographic profile of the victims for a conclusive diagnosis and academic interest.

MATERIALS AND METHODS: The study was performed in co-operation with the departments of pharmacology, ophthalmology, forensic and state medicine (FSM) and Medical record section (MRS)

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of BRIMS Teaching Hospital, Bidar. Required permission to conduct this record-based study was obtained from respective departments.

100 victims of OPC poisoning were screened out from the records of mortuary of the FSM department. Post autopsy studies were carried out on those victims by collecting data from the following sources: Medicolegal autopsy reports, inquest reports and in patient case sheets from MRS in case of Hospital admission.

Victim's demographic profile, case history and autopsy reports were recorded in a predesigned proforma. Autopsy surgeon put notes on autopsy report sheet "viscera and contents were collected, preserved in saturated solution of NaCl, sealed and handed over to escorting police personnel for onward transmission to Karnataka State Forensic Science Laboratory (FSL), Gulbarga".

Chemical analysis reports sent by FSL showing nature of OPC when available are also recorded in that proforma. Sign-symptoms observed at the time of hospital admission, clinical finding and medication received during hospitalization were noted in that precoded proforma. All these information were entered in the computer data base, analysed and expressed in standard statistical format and evaluated for a conclusive diagnosis retrospectively.

OBSERVATION: Demographic profile of the victims, as shown in table 1, comprised if age range 20-50 years with highest incidence in 20 to 30 years age group, 42% male and 58% female and 70% urban incidence. Dead after hospitalization was in 50% cases and remainder was brought dead. There was history of consumption poison. Mode of ingestion of OPC was to commit suicide in case of hospitalized victims.

Profile	No. of cases (%)
Age (Years)	
<20	Nil
20-30	61 (61%)
31-40	32 (32%)
41-50	7 (7%)
Sex	
Male	42 (42%)
Female	58 (58%)
Residence	
Urban	74 (74%)
Rural	26 (26%)
Death:	
Hospital Admitted	65 (65%)
Brought dead	35 (35%)
Table 1:Demographic profile of victims of Organophosphorus Compound Poisoning (n=100)	

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Date	Percentage of Cases
Age (Years)	
<1	10.14
>1-3	79.71
>3.4	7.25
>4.5	2.90
Cholinergic manifestations:	
Muscarinic	100.00
Nicotinic	13.04
CNS	98.58
Day of death after exposure:	74 (74%)
1 st	46.38
2 nd	24.63
3 rd	27.54
4 th	1.45

Table 2: Data of Hospitalised Victims of Organophosphorus Compound poisoning (n=50)

Findings	No. of cases (%)
Color	
Yellowish brown	16 (16%)
Bilious	7 (7%)
Greenish blue	15 (15%)
Chocolate	46 (46%)
Reddish	14 (14%)
Violet	2 (2%)
Odor	
Pungent	30 (30%)
Kerosene oil like	47 (47%)
No Untoward smell	23 (23%)
Food	15%

Table 3: Autopsy Findings of Stomach content of victims of Organophosphorus Compound Poisoning (n=100)

Victims were hospitalized with complaint of abdominal cramps, vomiting, diarrhea, chest pain and drowsiness or restlessness. Incidence of time of hospitalization was highest (79.71%) in 1-3 hours-time period Table. Clinical Examination on admission revealed cholinergic sign-symptoms.

Manifestations were muscarinic in 100%, nicotinic in 13.04% and CNS features in 98.58% cases (Table 2). Commencement of death occurs following varied duration of illness ranged from 1 day to 4 days. Mortality was highest (46.38%) on 1st day exposure (Table 2).

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Recommended general supportive measures and dose schedule for antidotes (atropine and pralidoxime)¹⁻⁴ were followed to manage the cases. Brought dead victims were declared dead at Casualty room of BRIMS, Teaching Hospital and other hospitals. They were found lying unconscious at the site of occurrence as mentioned in inquest reports.

Medicolegal Autopsy Reports: External finding –Postmortem staining and frothy discharge from mouth and nostril were present. Sign of venepuncture was found on different sites of hands of all hospital admitted cases. Lips, tongue, ear lobules and finger-nails were cyanosed. Eye finding were hazy cornea and fixed dilated pupil on both sides.

Internal finding – General visceral congestion with pulmonary and cerebral edema were present. Mucus membrane of gastro-intestinal tract was grossly congested except in 7% cases (decomposed). Submucosal and subpleural hemorrhagic spots were found. Stomach contained a varied range of 50-300g% fluids. Characteristics of stomach content are depicted in Table 3. Reports chemical analysis by FSL were still not received.

There was extensive congestion of mucous membrane of vagina with evidence of petechial hemorrhagic spots in vaginal mucosa in 2 cases of poisoning.

DISCUSSION: Pesticides are toxic chemicals by design. Ingestion of pesticides is a common cause of self-poisoning in the developing world⁵. Death due to the poisoning fatalities is a major global clinical problem⁶. The present study was undertaken for academic interest to come into a conclusive diagnosis retrospectively. Victims were reported to consume poison with some specification as insecticides. Characterization of the poisons was attempted on the basis of contained shown or history given by the victim's family members.

Observations of this study were compared with other available studies. Incidence of poisoning was more common among the age group between 20 to 30 years. This correlates to the age distribution reported by other workers.⁷⁻¹⁰ Females outnumbered males in this study. It is comparable with observation of the workers.⁷⁻¹¹

Where males supersede females. In the present study urban incidence was more than the rural. This is in contrast with the observation of other studies^{10,11} where higher incidence was in rural areas. As this medical college is situated in the urban zone, most of the victims got admitted or referred from surrounding area. Moreover, most of the poisoning cases in the rural areas are treated in the rural hospital; only the complicated cases are referred to tertiary level hospital.

Time-interval between consumption and hospital admission is comparable to the findings of Ramesha et al⁸, where, time lapse of more than hours had a significant role in the mortality. In this study all the victims were hospitalized within 5 hours of occurrence and expired following respective duration of illness. Sign-symptoms of cholinergic crisis, noted in this study, are matched with that of literature.^{1-4,12}

Patients had a well-defined cholinergic phase. Presenting features were consistent with organophosphate poisoning. Treatment of the patients was based on minimizing the absorption by induction of vomiting or gastric lavage, and specific pharmacological treatment. Quick hospitalisation and proper therapy could not prevent death; probably large amount of OPC was consumed. Fatal dose of OPC is 1-2mg/kg of body weight⁴ or 15mg-5g.³

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Massive intake of OPC (>1g) results in fulminating poisoning⁴. all the hospitalized victims committed suicide. Case history of brought dead victims revealed suicide almost as cause of death, but subjected to be proved.

Signs of irritant poisonign^{3,4} were found on every tissue on autopsy. Fixed dilated pupil was probably due to terminal phase. Terminal phase anoxia causes dilation of pupil.³ Colors and odor of gastric content confirmed the OPC exposure. Reference shows no smell in red phosphorus and garlicky in yellow phosphorus.⁴

In this study no untoward odor may be due to presence of red phosphorus or may be due to small amount (50g) of gastric content after excessive or induced vomiting or stomach wash. Analytical identification of OPC in gastric content or in the body fluids gives the clue of exposure to OPC²; which was lacking in this study. It might be helpful if cholinesterase level could be measure. Cell and plasma cholinesterase level becomes <30% of normal.

In the present study, diagnosis was confirmed by history of poison exposure, clinically well-defined cholinergic phase, and characteristic autopsy findings. Cause of death was elucidated in all the cases. This study highlights the usefulness of clinical and autopsy finding in the diagnosis of OPC poisoned death.

Indiscriminate use of OPC as insecticides increases the potential for self-poisoning. Restrictions on access of very toxic pesticides through national polices and enforcement, strategies on integrated pest management, public education on toxicity, storage and safe use, early recognition of poisoning, and appropriate medical management may reduce the incidence of poisoning and death.

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