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A REVIEW ON ORGAN PHOSPHORUS POISONING

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ABSTRACT

Organ phosphorus (OP) poisoning is a generally encountered major clinical problem in numerous countries of the world, more so in the developing countries. The authors present a case of suicidal OP poisoning with an overview of operation of the acute intoxication. The early recognition of symptoms and prompt treatment helps in reducing the morbidity and mortality Organ phosphorus (OP) insecticide suicidal poisoning is a major public-health concern across the maturity of the pastoral Asia, including the pastoral India. It's also of great significance to the clinicians due the high morbidity and mortality associated with its ingestion(1- 4) Early recognition of the poisonous pattern and its prompt operation is the key for saving the case's life.

Keywords*: insecticide, morbidity, mortality*

1. INTRODUCTION

The authors present a case of contended suicidal OP poisoning with an overview of operation of acute intoxication. Organophosphorus composites are form the large groups of chemicals that used over the once 60 times for guarding crops, beast, mortal health and as warfare agents. Organophosphates(OPs) are chemical substances firstly produced by the response of alcohols and phosphoric acid. In the 1930s, organophosphates were used as germicides, but the German service developed these substances as neurotoxins in World War II. They serve as cholinesterase impediments, thereby affecting neuromuscular transmission. On the base of structural characteristics they're divided into at least 13 types similar as phosphates, phosphonates, phosphonates, phosphonothioates, phosphonothioates, phosphonothioates, phosphorotrithioates, phosphoramidothioates(Gupta, 2006). OPs are the most extensively used fungicides worldwide and their metabolites are wide across different populations(Aprea, 2000; Barr etal., 2004; Curl etal., 2003). The adverse short- term goods of exposure to these chemicals have been studied substantially in the nervous system, which is the main target(Gupta etal., 2001), but there's a growing concern about their possible poisonous goods fornon-target apkins and(long- term) habitual goods that haven't been studied in similar detail. The maturity of people are continually exposed to low OP attention, and long- term epidemiologic studies reveal relation to advanced threat of cancer development(Brown etal., 1990; Waddell etal., 2001). Organophosphates effect on insects and mammals primarily by phosphorylation of the acetylcholinesterase enzyme(pang) at whim-whams consummations. thus, a loss in available pang happed and the affected organ becomes over stimulated by the redundant acetylcholine(pang, the impulse- transmitting substance) in the whim-whams ending. Some critical proportion of the towel enzyme mass must be inactivated by phosphorylation before symptoms and signs of poisoning come overload. At sufficient lozenge, loss of enzyme function allows accumulation of Ach peripherally at cholinergic neuroeffector junctions(muscarinic goods), cadaverous whim-whams- muscle junctions, and autonomic ganglia(nicotinic goods), as well as centrally(Routt and Roberts, 1999). Certain reports were published in the last 30 times clarified that Fungicides especially Organophosphates group was responsible for admission millions of people to hospitals with accidental poisoning, in addition to suicidal cases. Meanwhile, it was estimated that about 25 million agrarian workers suffering from poisoning every time in the third world countries(Alavanja, 2009). therefore, this work aimed to make a small memorial about the pitfalls of organophosphate fungicides. Organophosphorus composites are available as dust, grains, or liquids, and some products are needed to adulterated with water before use(1). The deaths due to organophosphorus poisoning are adding in pastoral areas of India. The organophosphorus composites are impediments of acetylcholinesterase(pang) enzymes which are responsible for the hydrolysis of acetylcholine(Ach) to choline and acetic acid. Due to the inhibition of acetylcholinesterase enzyme acetylcholine gets accumulated leading to a continued simulation of original receptors, causes palsy of whim-whams or muscle. Palsy is observed foremost in organophosphate(OP) poisoning latterly causes a complete arrestment of whim-whams or muscle exertion. piecemeal from(pang) inhibition, OP also inhibits carboxylic ester hydrolases similar as chymotrypsin, butyl cholinesterase, tube hepatic carboxylesterases, and other proteases. OP absorbed through any route similar as inhalational, across gastrointestinal(GI), transdermal, and gastro urinary mucosa. The symptoms of OP poisoning generally begin within a many twinkles to a many hours after consumption. Symptoms include expectoration, lacrimation, urination, diarrhea, GI torture, and emesis. habitual symptoms include muscle cramps, weakness, gait diseases, anxiety, tachycardia, and blurred vision may be present for several months after treatment(2). Atropine is considered the most dependable and common cure in OP poisoning. Atropine is a competitive antagonist of Ach at the muscarinic postsynaptic membrane. Competitive enmity leads to inhibition of the muscarinic exertion of the body. Overdosing of atropine leads to bronchospasm, bradycardia, eye inflammation,non-responsive, and dilated pupils. Coma with circulatory system collapses and respiratory depression, flushed skin, and distraction(3). The goods of atropine due to its overdosing treat using benzodiazepines similar as diazepam, and lorazepam(4). Physostigmine can also be given as an cure by slow intravenous infusion fleetly reduces the distraction, coma caused by large boluses of atropine. Artificial respiration or mechanical ventilation with oxygen is necessary for oppressively

affected cases(5). Organophosphorus(OP) composites are used as fungicides, dressings, and chemical warfare agents in the form of whim-whams feasts.1 Acute poisoning by these agents is a major global clinical problem, with thousands of deaths being every time.2 utmost of the OP fungicide poisoning and posterior deaths do in developing countries following a deliberate tone ingestion particularly in youthful, productive age group, as largely poisonous fungicides are readily available at the moments of stress. Poisoning has been a common cause of medical admissions and deaths in Nepalesehospitals.4- 11 Thirty- one percent of all suicidal deaths in the country in 1999- 2000 were due to poisoning.12 Sanitarium- grounded studies from five major hospitals across the country in 1999- 2000 showed OP composites were the most common form of poisoning comprising 52 of total cases.13 colorful insulated sanitarium- grounded studies also easily demonstrate that OP composites enthrall the topmost burden of poisoning related morbidity and mortality in Nepal.4 Encyclopedically, organophosphorus(OP) fungicide poisoning is a serious occupational hazard account for further than 80 of fungicide - affiliated hospitalization.(1)

India being an husbandry - grounded country, OP fungicide remains the main agent for crop protection and pest control. It's thus likely to have adverse goods on growers who are accidentally over exposed while handling these fungicides. still, because of low cost and easy vacuity, it has also come an agent of choice for tone poisoning.(2) Fungicide tone - poisoning is responsible in killing roughly people worldwide every time and substantially from pastoral background. In developing countries the mortality can be as high as 70.(). crucial to survival lies in early opinion followed by rapid-fire decontamination and definitive remedy which purely lies under the expert sphere of exigency drug. In this composition, we report the retrospective analysis of cases with severe OP germicide poisoning in a tertiary care sanitarium in Northern India. Anticholinesterase are fluently available and considerably used as agrarian and ménage germicides. accidental as well as suicidal and sanguine poisoning is common

Organophosphorus compounds are chemical agents in wide-spread use throughout the world, mainly in agriculture. They are also used as nerve agents in chemical warfare (e.g. Sarin gas), and as therapeutic agents, such as ecothiopate used in the treatment of glaucoma. They comprise the ester, amide or thiol derivatives of phosphoric acid and are most commonly used as pesticides in commercial agriculture, field sprays and as household chemical There are no rules and regulations governing the purchase of these products, and they are therefore readily available “over the counter”, despite them being a major cause of morbidity and mortality. Exposure to organophosphates in an attempt to commit suicide is a key problem, particularly in the developing countries, and is a more common cause of poisoning than the chronic exposure experienced by farmers or sprayers in contact with pesticides.

CLASSIFICATION:

There are more than a hundred organophosphorus compounds in common use.

- **Insecticide –** Malathion, parathion, diazinon, fenthion, dichlorvos, chlorpyrifos, Ethicon
- **Newer gases–** Soman, sarin, tabun, VX
- **Ophthalmic agents –** Echothiophate, isoflurophate
- **Antihelmintics –** Trichlorfon
- **Herbicide–** Tribufos(DEF), morphs
- **Artificial chemical (plasticizer) –** Tricresyl phosphate

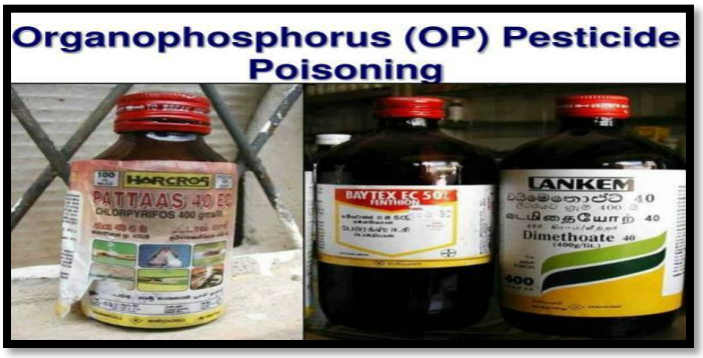

Fig.1 examples of . Organophosphorus poisoning

EPIDEMIOLOGY:

OP poisoning results either from suicidal or accidental poisoning or from occupational exposure similar as husbandry. A large proportion(68) of cases presented to the ICU following an acute self-murder attempt. Ulmost cases are youthful(lower than 30 times) with. Occupational and accidental poisoning do less generally in India.(26) Acute OP poisoning is one of the common and serious medical extremities • Asia 3 million cases/ time of acute fungicide poisoning 3 lakh deaths per time(CFR = 10- 15) • 99 cases are seen in developing world 91.86% of cases were suicidal and remaining cases were accidental According to WHO report 2002, about 849,000 people die globally from self-harm yearly. In India pesticide poisoning is a major problem in the agricultural group. In a study conducted in Christian Medical College and Hospital, Vellore, OP poisoning accounts for 12% of ICU admissions and 29% of total poisoning admissions 3

INDIAN DATA:

Poisoning is 4th MC cause of deaths annually • OP poisoning is the MC poisoning • Pattern of poisoning is region- dependant • OP poisoning is more common in south India • North India- aluminium phosphide india The most common OP to be consumed was Methyl parathion(27), followed by Chlorpyrifos(22) The least common emulsion was Phorate(4)(Figure 1).

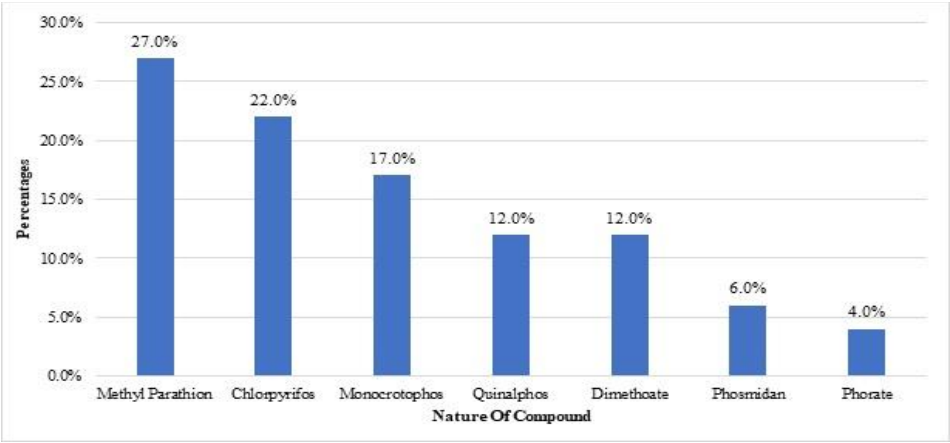

Fig no 2 graphical representation of op poisoning deaths in india

OP POISONING IS COMMON:

India is an agrarian country. Routinely used in farming CAUSES OF HIGH MORTALITY

- MORTALITY High toxicity of available compounds
- Time gap in transporting patients
- Paucity of Health care personnel
- Lack of training facilities
- Lack of antidotes

ABSORPTION ROUTE:

- Cutaneous
- Ingestion (Accidental Or Suicidal)
- Inhalation
- Injection

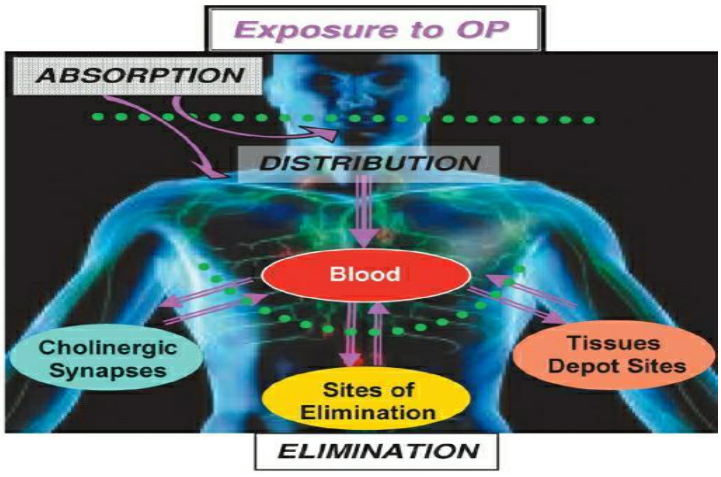

Fig.no.3 ingestion of op poisoning

PHARMACOKINETICS:

Most organophosphates are highly lipid soluble compounds and are well absorbed from intact skin, oral mucous membranes, conjunctiva and the gastrointestinal and respiratory tracts. o They are rapidly redistributed to all body tissues. o The highest concentrations are found in the liver and kidneys. This high lipid solubility means that they easily cross the blood/brain barrier and therefore produce potent effects on the CNS.Metabolism occurs principally by oxidation in the liver with conjugation and esterase hydrolysis producing a half-life of minutes - hours. The oxidative metabolites of malathion and parathion (malaoxon and paraoxon) are active forms and are subsequently hydrolyzed into inactive metabolites. Elimination of organophosphorus compounds and its metabolites occur mainly via urine, bile and faeces

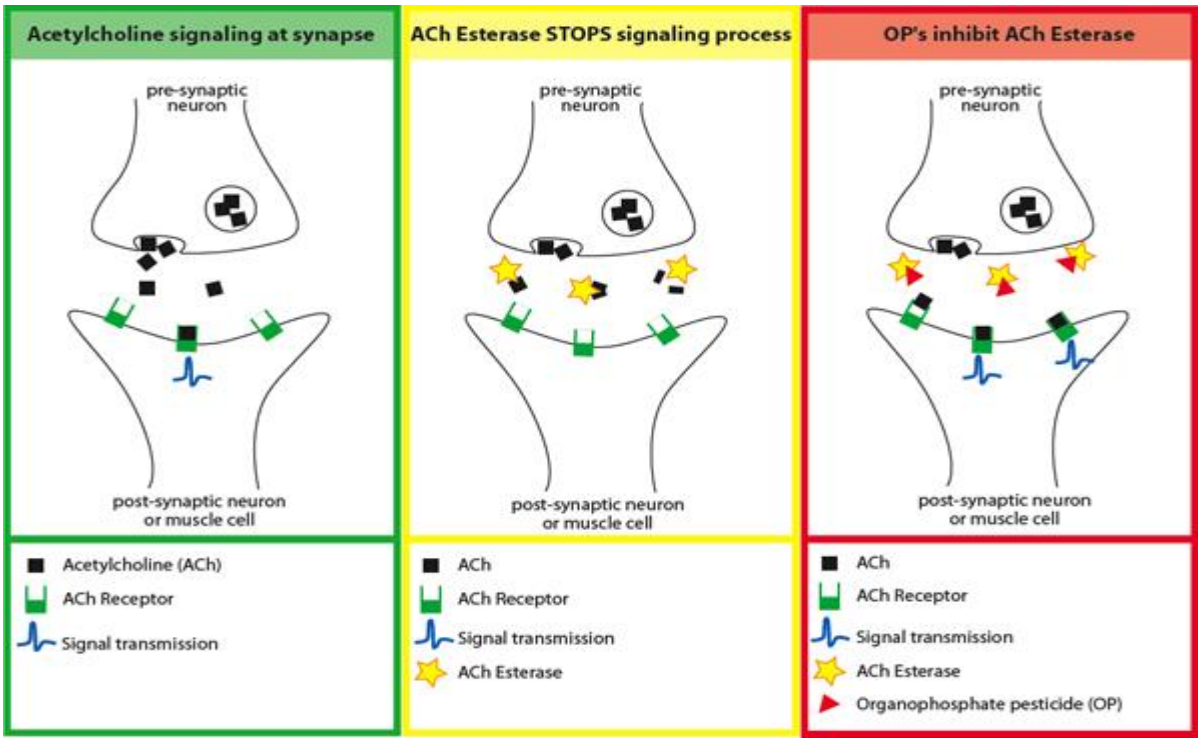

Fig.no.4 mechanism of op poisoning

MECHANISM OF TOXICITY:

The poisonous mechanism of OP composites is grounded on the unrecoverable inhibition of acetylcholinesterase due to phosphorylation of the active point of the enzyme. This leads to accumulation of acetylcholine and posterior over-activation of cholinergic receptors at the neuromuscular junctions and in the autonomic and central nervous systems. The rate and degree of pang inhibition differs according to the structure of the OP composites and the nature of their metabolite. In general, pure thion composites aren't significant impediments in their original form and need metabolic activation(oxidation) in vivo to Oxon form. For illustration, parathion has to be metabolized to paraxon in the body so as to laboriously inhibit pang. The poisonous medium of OP fungicides differs from that of carbamates which inhibit the same enzyme reversibly and are occasionally useful as drugs(neostigmine, pyridostigmine) as well as germicides(carbaryl). After the original inhibition and conformation of pang- OP complex two farther responses are possible(1) robotic reactivation of the enzyme may do at a slow pace, important slower than the enzyme inhibition and taking hours to days to do. The rate of this regenerative process solely depends on the type of OP emulsion robotic reactivation half life of0.7 hours for dimethyl and 31 hours for diethyl composites. In general, pang- dimethyl OP complex spontaneously extinguish in lower than one day whereas pang- diethyl OP complex may take several days and reinhibition of the recently actuated enzyme can do significantly in similar situation. The robotic reactivation can be whisked by adding nucleophilic reagents like oximes, liberating more active enzymes. These agents thereby act as an cure in OP poisoning.19(2) With time, the enzyme- OP complex loses one alkyl group making it no longer responsive to reactivating agents. This progressive time dependent process's known as geriatric. The rate of geriatric depends on colorful factors like pH, temperature, and type of OP emulsion; dimethyl OPs have geriatric partial life of3.7 hours whereas it's 33 hours for diethyl OPs The slower the robotic reactivation, the lesser the volume of inactive pang available for ageing. Oximes, by catalyzing the of active pang from enzyme- OP complex, reduce the volume of inactive pang available for ageing. Since ageing occurs more fleetly with dimethyl OPs, oximes are hypothetically useful before 12 hours in similar poisoning. still, in diethyl OP intoxication they may be useful for numerous days.

SIGNS AND SYMPTOMS OF OP POISONING:

Clinical features of acute poisoning do within 24 h of ingestion of the OP emulsion. These can be astronomically classified as follows.(21)

Muscarinic effects: muscarinic effect developed because they act on m- receptor and affect the m receptor

- Cardiovascular Bradycardia, hypotension.
- Respiratory Rhinorrhea, bronchorrhea, bronchos- pasm, cough.
- Gastrointestinal Increased expectoration, nausea and
- Abdominal pain, diarrhea, fecal incontinence.
- Genitourinary Urinary incontinence.
- optical Blurred vision, increased lacrimation, miosis.
- Dermatologic inordinate sweating.

Nicotinic effects: nicotinic effect is developed because they act on Nm receptor and affect the Nm receptors.

- Cardiovascular Tachycardia, hypotension.
- Musculoskeletal Weakness, fasciculation's, cramps,
- Palsy

Central effect:

- Headache
- Confusion
- Storms
- Coma and death due to respiratory failure substantially

Intermediate syndrome:

The intermediate syndrome is a condition of muscular weakness and palsy that occurs 1- 4 days after the resolution of acute cholinergic toxidrome due to organophosphate exposure. Numerous cases aren't diagnosed until significant respiratory insufficiency has passed.

COPIND (chronic op induced Neuro- psychiatric disorder) :

Habitual low- cure exposure to OP composites 40 hours/ week, 9 months/ time No cholinergic symptoms Non responsive to levodopa Tube cholinesterase situations are normal

DIAGNOSIS :

The diagnosis of OP poisoning is made primarily based on the history of exposure and a combination of signs and symptoms developed, including the typical odour of the insecticide. Some patients may present with nicotinic effects of tachycardia and mydriasis rather than the anticipated bradycardia and miosis. Some poison centers can identify the compound from the stomach contents.

WHAT TO DO?

Call AIIMS poison's cell(NPIC)- National Poisons Information Cell- Toll free no. 1800116117-Other 011- 26589391- Open 24 X 7, 365 days a time

IDENTIFICATION OF POISON:

- History by case/ attendant
- Clinical donation.
- By showing photos.
- WHO colour codes on bottle

POISON IDENTIFICATION WHO COLOUR CODE ON BOTTEL:

- **Red colour marker:** Extremely poisonous e.g Monocrotophos, zinc phosphide, ethyl mercury acetate, and others.
- **Yellow colour marker:** largely poisonous e.g Endosulfan, carbaryl, quinalphos, and others.
- **Blue colour marker:** relatively poisonous e.g Malathion, thiram, glyphosate, and others.
- **Green colour marker:** Slightly poisonous e.g Mancozeb, oxyfluorfen, mosquito repellant canvases and liquids, and utmost other ménage germicides.
- **Identification of poison**: Signs of cholinergic excess or developing intermediate syndrome Disparity between history and clinical presentation, follow, clinical judgment. After identification classify as Organophosphorus and non-Organophosphorus.

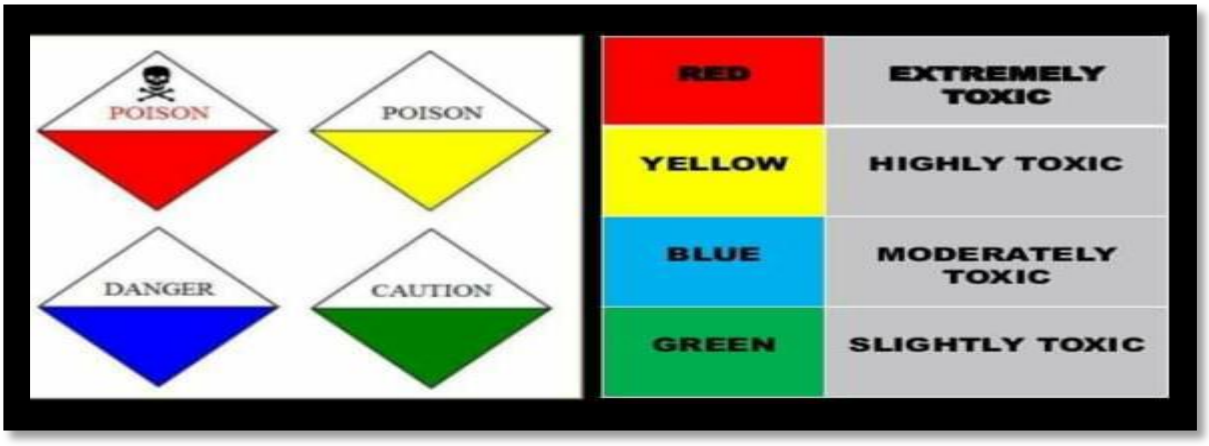

Fig no.5 poison identification colour on bottle

ORGANOPHOSPHOSPHATE MANAGEMENT:

Immediate, Protocol Assess and record 15- point Glasgow Coma Scale. Palpitation rate, BP and auscultate Case left side position- head lower than the bases. Oxygen, Intubate if respiratory torture. Start atropine reduce bronchorrhea.0.9 normal saline, Aim SBP> 80 mm Hg & urine affair> 30 ml/ h

CHOLINESTERASE ESTIMATION:

True and pseudocholinesterase situations can be estimated. The situations are markedly reduced in OP poisoning. Although true cholinesterase situations relate with inflexibility at donation, pseudocholinesterase situations do not. periodical estimations don't relate with clinical enhancement, and hence these tests are to be used primarily to confirm the opinion. Other causes of low- serum cholinesterase include parenchymal liver complaint, congestive cardiac failure with hepatomegaly, metastatic melanoma, malnutrition, dermatomyositis, and acute infection A 25 or lesser depression in red- blood- cell cholinesterase position is taken as the stylish index of op poisoning(22)

WHEN NOT SURE ABOUT THE POISON ?

Then first we can perform atropine test. After atropine test we can find out op poisoning toxin or not toxin of op poisoning.

- **Atropine test**: inject 0.6- 1 mg IV atropine. Then If palpitation rate goes up by 25 per nanosecond or skin flushing develops case has mild or no toxin for OP's.

TREATMENT:

Treatment should be initiated incontinently on clinical grounds without staying for blood examinations.

Fig.no.6 management of op poisoning

GENERAL TREATMENT:

- **Decontamination:**

Decontamination of the skin It's veritably important and it should be done veritably completely. Remove defiled cloths, cough, skin with cleaner and water. This will help farther immersion through the skin. Health- care labor force should wear defensive apparel and spectacles.

- **Gastric decontamination:**

It's should be done by forced emesis if the case is completely awake or through a gastric lavage. All cases should also admit0.5 – 1 g/ kg actuated watercolor every 4 Sodium sulfate or sorbitol can be used as a cathartic.

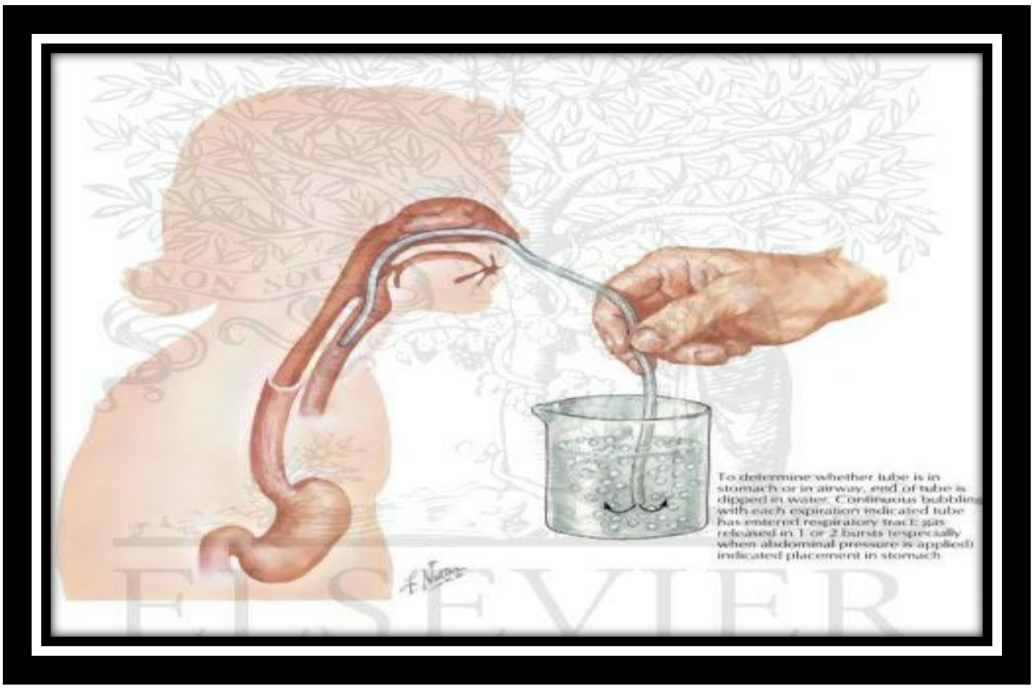

Fig.no.7 gastric lavage in op poisoning

A recent study has shown that the addition of serotonin adipinate, a medicine that enhances the propulsive function of the gastrointestinal tract, redounded in a shortening of the toxicogenic phase and a3.5-fold reduction of mortality.(23) Airway and Respiration The airway should be secured and acceptable oxygenation should be assured. Atropine can precipitate ventricular arrhythmia in hypoxic cases. On the other hand, early use of atropine will reduce respiratory concealment, ameliorate muscle weakness, and thereby ameliorate oxygenation. Careful observation of the respiratory status is needed as these cases are prone to respiratory failure during the acute phase and intermediate pattern. The important parameters to be covered on a regular base are

a) Symptoms of optical muscle involvement (e.g.; presbyopia),

b) Neck muscle weakness,

c) Respiratory rate,

d) Tidal volume or vital capacity,

e) Single breath count, and

f) Arterial blood gas estimation or palpitation oximetry.

g) Respiratory infections should be anticipated and treated. Theophylline is contraindicated in the operation of OP poisoning.(23)

- **Cardiac Monitoring:**

Wide- ranging cardiac instantiations do, and careful hemodynamic and electrocardiographic monitoring should be accepted. Hypoxemia and electrolyte abnormalities contribute to cardiac complications. Some of these may return atropine. Ventricular tachycardia with a prolonged QTc is stylish treated with electrical pacing.(24) Supraventricular tachycardia can be treated with an IV gel cap of short- acting â- blockers.

SPECIFIC ANTIDOTE IN OP POISONING:

- **Atropine:**

Atropine has been use in OP poisoning for decades and will remain so in the future. It acts competitively at the supplemental and central muscarinic receptors and antagonizes the parasympathetic effects of redundant pang at these spots. It reverses life hanging features from poisoning; detention or atropine can affect in death from central respiratory depression, bronchospasm, inordinate Broncho stashing, severe bradycardia, and hypotension. There are wide variations in recommended boluses and rules of atropine remedy in different corridor of the world. also, duration of atropine treatment and titration of the cure isn't clear. Current guidelines recommend the use of gel cap boluses to attain target end- points, followed by setting up an infusion to maintain these end- points.24 Target end- points for Atropine remedy Heart rate> 80/ min, Dilated pupils, Dry axillae, Systolic blood pressure> 80 mm Hg Clear casket with absence of wheeze We use an original gelcap of 3- 5 ampoules of atropine(each ampoule containing0.6 mg) with posterior boluses doubled every 5 twinkles until atropinization is achieved.39 When the case achieves utmost of(at least 4 out of 5) the target end- points for atropine remedy i.e., ‘ completely atropinized ’, an intravenous infusion is set up to maintain the remedial goods of atropine. While there are different approaches of atropine infusion, we use 20 of original atropinizing cure per hour for first 48 hours and gradationally taper over 5-10 days, continuously the acceptability of remedy. There's a tendency to give redundant atropine, which can be dangerous. Atropine toxin can affect in agitation, confusion, hyperthermia, and severe tachycardia that can precipitate ischemic events in cases with underpinning coronary roadway complaint. So, close observation and cure adaptation is essential to avoid the features of both under and over atropinization. Some centers use another anticholinergic agent glycopyrrolinium platitude along with atropine in order to limit the central stimulation produced by atropine, because the former doesn't cross blood brain hedge.

- **Oximes:**

Oximes work by reactivating acetylcholinesterase that has been bound to the OP patch. Pralidoxime is the most constantly used oxime worldwide; Oximes can be largely effective in restoring cadaverous muscle strength and perfecting diaphragmatic weakness where atropine has nearly no effect. Clinical opinions of oxime remedy in OP poisoning are divided, indeed in cases of massive mortal intoxication.40 issues following oxime remedy depend on colorful factors like the type of bane ingested, the bane cargo, time ceased between OP ingestion and institution of remedy, and the duration and lozenge of the oxime remedy. In some cases oximes may prove ineffective for several reasons shy cure leading to sub-therapeutic blood situations, early termination of oxime remedy, and nonstop reinhibition of the reactivated pang from fungicide persisting in the body.The remedial window for oximes is limited by the time taken for ‘ geriatric ’ of the enzyme- OP complex, because progressed ’ enzyme can no longer be reactivated by oximes(see medium of toxin, over). still, others propose dragged conservation of an applicable oxime attention irrespective of the type of ingested OP. Some advise oxime remedy for the treatment of intermediate pattern. colorful lozenge rules have been recommended from intermittent oxime administration to nonstop infusion following a lading cure. While there's no clear agreement on the cure and duration of oxime remedy, lately the WHO recommended Pralidoxime cure of 30 mg/ kg gel cap iv followed by nonstop infusion of 8 mg/ kg/ hour until clinical enhancement. Dizziness, headache, blurred vision, and presbyopia, are common side goods of oxime remedy. Rapid administration may lead to tachycardia, laryngospasm, muscle spasm, and flash neuromuscular leaguer. delicate vacuity and cost factor are other downsides for routine use of oximes in clinical practice.

- **Benzodiazepines**

Diazepam and other benzodiazepines are extensively used for the treatment of OP convinced seizures and restlessness and agitation consequent to either bane itself or effect of atropine remedy. also diazepam, due to its central respiratory depressant action, is also believed to attenuate OP- convinced respiratory depression which generally follows over stimulation of the CNS respiratory centers.

- **Pregnancy**

Pregnant cases who have ingested OP germicides during the alternate or third trimester of gestation have been treated successfully with atropine and Pralidoxime and latterly delivered healthy babe with no significant abnormalities. still, fetal torture is a possible complication of both of the poisoning as well as its treatment

NEWER FORMS OF CURATIVES IN OP POISONING:

One small unbridled study from Iran concluded that the infusion of sodium bicarbonate significantly reduced total sanitarium stay, total atropine demand, and the need for ferocious care remedy; mortality rate was also low in the treatment Group.

1. **Magnesium Sulphate**

- MgSO4 (4g) i/ v in first day after admission, drop hospitalization period and mortality
- It blocks Calcium channels and reduce acetylcholine release from presynaptic outstations.
- Also reduces CNS over stimulation from NMDA receptor activation.

2. Clonidine

- Centrally acting α2- adrenergic receptor agonists.
- Reduces acetylcholine conflation and release from presynaptic outstations
- Beast studies, shown benefit in combination with atropine. Effect in humans unknown.

ADVANCED NEUROPROTECTIVE MEDICINES:

- Ketamine Noncompetitive NMDAR antagonist, within 1 hour of whim-whams gas agent convinced seizures along with Midazolam/ diazepam.
- Tezampanel Glutamate receptor antagonist specific for kainate subtype Rc, useful in Soman(whim-whams gas) convinced seizures and neuropathy.
- Galyclidine Another antiglutamatergic emulsion set up salutary in confluence to standard remedy in whim-whams gas poisoning.

Intermediate syndrome pattern management: generally presents 12 to 96 hours after exposure:

- Early signs action temblors and pharyngeal weakness(delicate deglutition, pooling of concealment).
- Latterly incapability to flex neck, DTR's lost, cranial neuropathies, proximal muscle weakness and respiratory muscle palsy.
- Not all bear intubation and ventilation, but cases with temblors and pharyngeal weakness, at increased threat.

TREATMENT:

Ventilator support if respiratory muscle palsy. Ventilatory Support Indicated in torpor/ coma, Hypoxemia(PaO2< 60 mmHg) and profound muscle weakness. Predictors for need of mechanical ventilation

- Delay in the inauguration specific treatment.
- Low position of sensorium at admission
- Pinpoint pupils and generalized fasciculation's.
- Presence of storms
- Presence of respiratory failure at admission.
- High original atropine demand for atropinization.

Alkalinization:

- IV infusion of Soda. Bicarbonate produce moderate Alkalinization(pH7.45 to7.55) in OP poisoning.
- Infusion of advanced tablets(5mEq/ Kg) in 60 twinkles followed by 5- 6 mEq/ kg/ day was shown useful.(In tykes)
- More salutary in whim-whams agent poisoning.
- Cochrane review Insufficient substantiation to establish use of NaHCO in mortal OP Poisoning.

Enteral Feeding:

- Early enteral feeds associated with bettered issues in critically ill because it prevents enterohepatic rotation.
- Early nutritive supplementation in OP poisoning assumes significance as these cases may bear prolonged Ventilatory support.

Fresh Frozen parts:

- FFP contains important factors like clotting factors, proteins, enzymes.
- It's hypothecated that butyrylcholinesterase present in FFP sequester free bane in blood and remove it from rotation.
- Two trials, both unfavourable to FFP intervention

2. CONCLUSION

Reduce or exclude home and conterminous area use of organophosphate fungicides, especially if pregnant women or youthful children are in the home. Insure proper storehouse and labeling of fungicides. Do not store largely poisonous fungicides, especially agrarian fungicides, in homes. The use of defensive outfit by fungicide applicators as well as other threat populations should be encouraged. Individualities who are constantly exposed to low boluses of organophosphates should periodically estimate their health status to enable them to seek professional help and comforting. We describe what has been determined to date, bandy exiting gaps in our knowledge and make suggestion on necessary studies in future we can avoid the contact of organophosphorus. stay healthy and stay safe make your future.

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