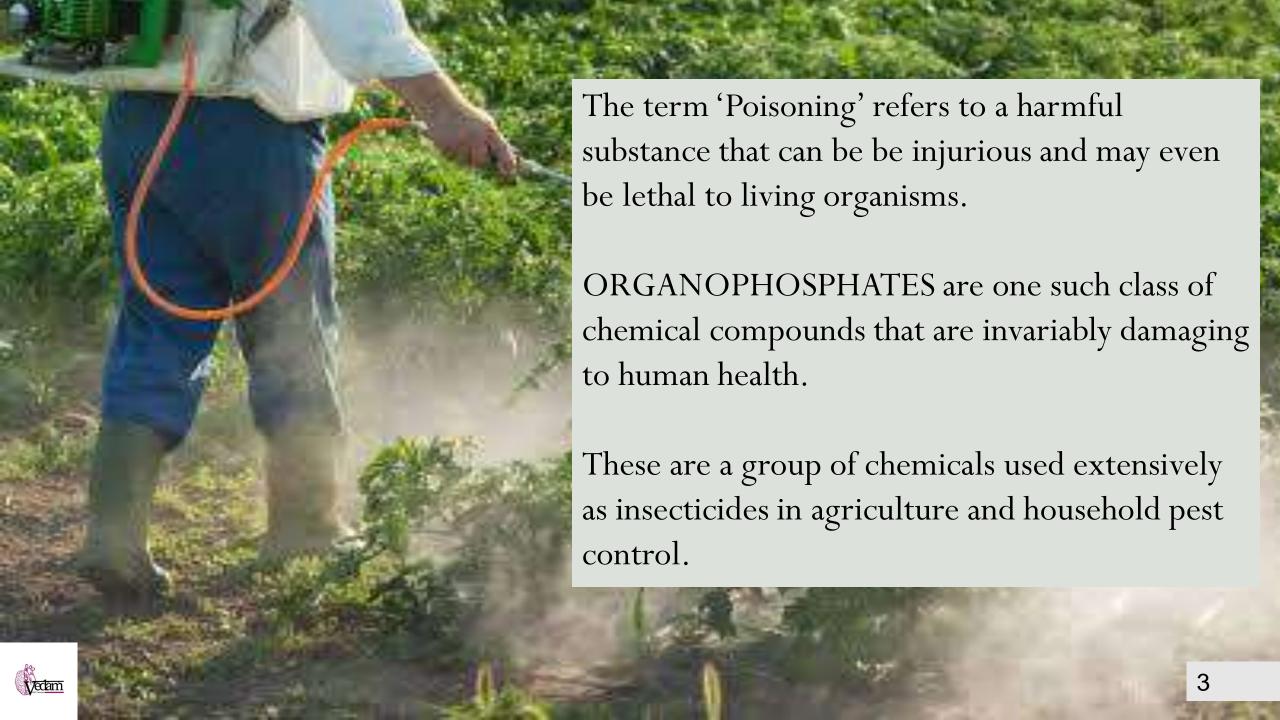
CARDIOVASCULAR EFFECTS OF ORGANOPHOSPHATE POISONING

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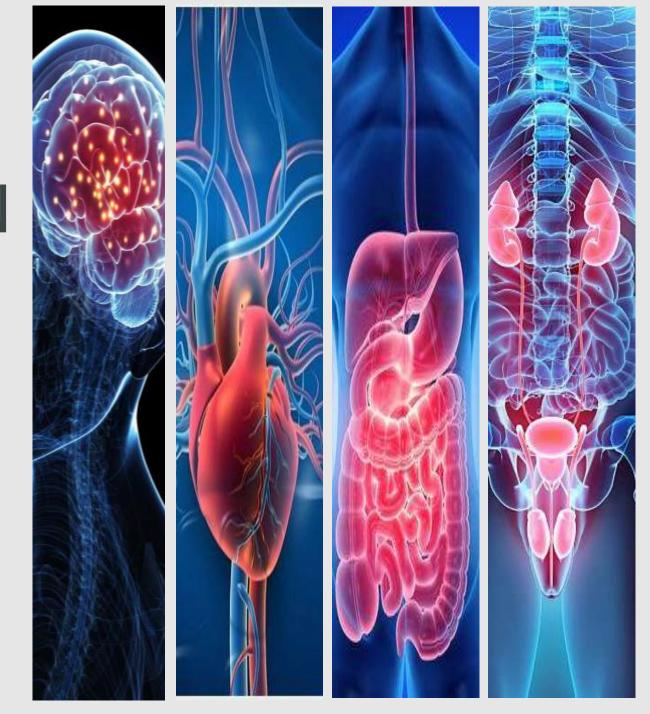
CASE DETAILS

External Examination

- A 69-year old male, with a history of chronic alcohol use, measuring 160 cm in height and weighing 55 kg was found
 deceased.
- Body was cold and stiff all over, conjunctiva of both the eyes were congested.
- All other external natural body orifices were intact and unremarkable.
- There was no evidence of any external injury present over the body.



INTERNAL EXAMINATION





BRAIN

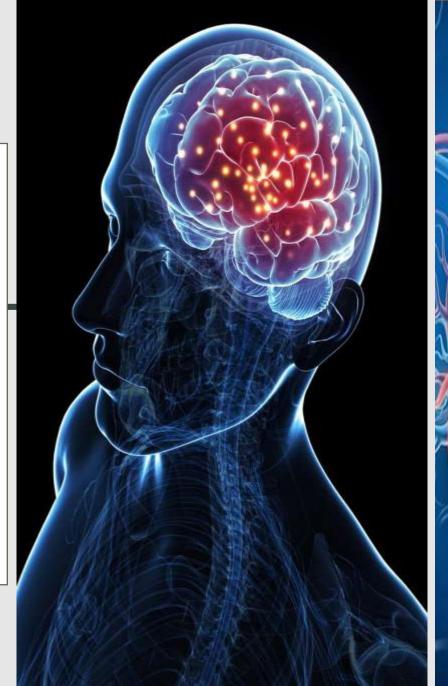
Skull: Intact and unremarkable

Dura: Intact and tense

Brain: Soft, congested and oedematous.

There was coning of the orbital part of the frontal lobe, uncal part of the hippocampal gurus bilaterally.

Brain was unremarkable on cut section











THORAX

Larynx and trachea: Airway lumen contained scanty greenish mucoid fluid.

Lungs: Both lungs were soft, congested and oedematous.

Heart: Weighed 296 g. Diffuse
hyperaemia with associated epicardial
hemorrhages present in posterior aspect
of left ventricle and lower half of
anterior wall. LAD artery showed 30%
luminal narrowing up to a point 1.5 cm
from its origin.

Large vessels: Aorta: Multiple fatty streaks and atheromatous plaques present in aortic intima.







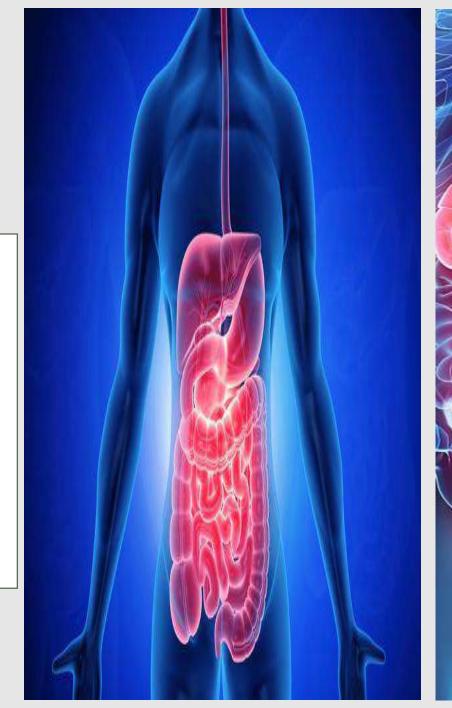


GASTRO INTESTINAL TRACT

Mouth, trachea, oesophagus: Oesophageal lumen contained scanty greenish mucoid fluid. Stomach: Contained 250 ml of greenish coloured mucoid fluid with greenish black coloured lump with charcoal smell.

Small intestine: Proximal part contained contents same as stomach.

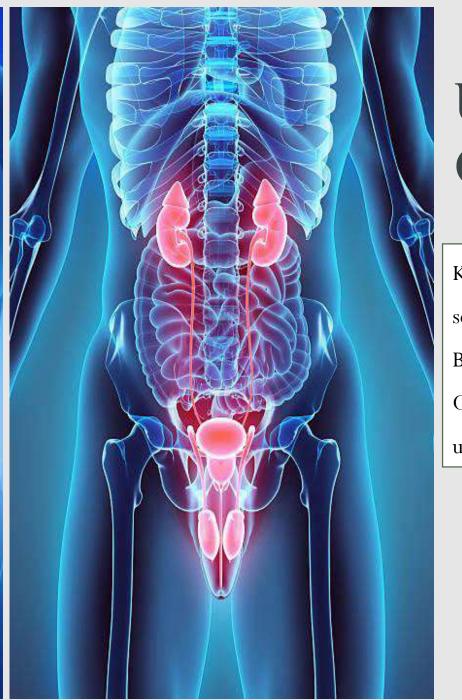
Large intestine: Intact and unremarkable.











GENITO-URINARY ORGANS

Kidney: Both found congested on cut

section.

Bladder : Empty

Organs of generation: Intact and

unremarkable



Histopathology reports reveals the following:

LUNG: Diffuse alveolar damage

LIVER: Submassive hepatocyte necrosis/ acute fulminant

hepatitis

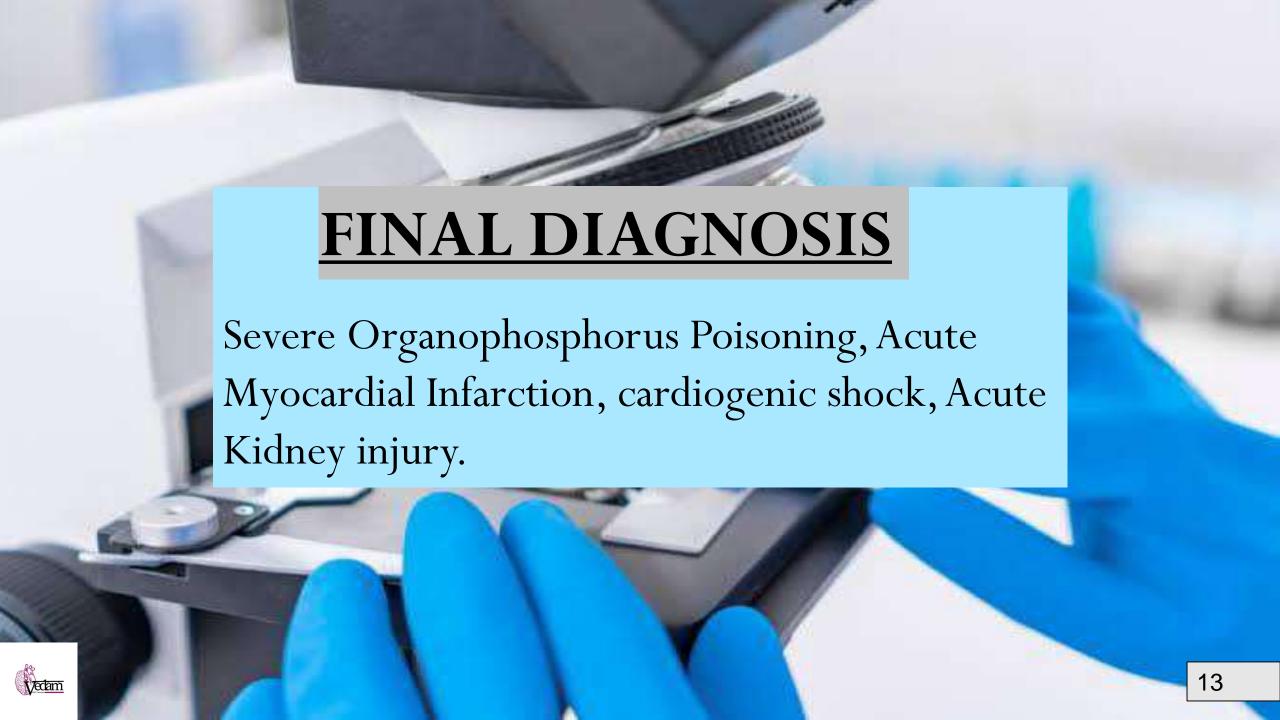
KIDNEY: Acute venous congestion

HEART: Left ventricular hyper trophy, atheromatous plaques in

coronaries and aorta. Petechial haemorrhage in pericardium.

PANCREAS: No specific pathology





FINAL OPINION

Based on hospital records, autopsy findings, histopathology report is deceased died due to complications secondary to **consumption of organophosphate poisoning** .



DISCUSSION

Organophosphates encompass a diverse group of chemical compounds with applications in pesticides and herbicides, as well as nerve agents in chemical warfare.



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These compounds are a class of compounds with general structure O=P(OR)3, a central phosphate molecule with alkyl or aromatic substituents.



Parathion

Chlorpyrifos

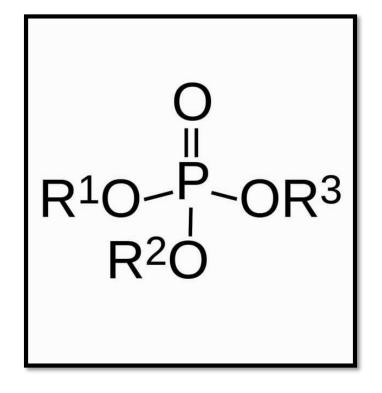
Diazinon

Dichlorvos

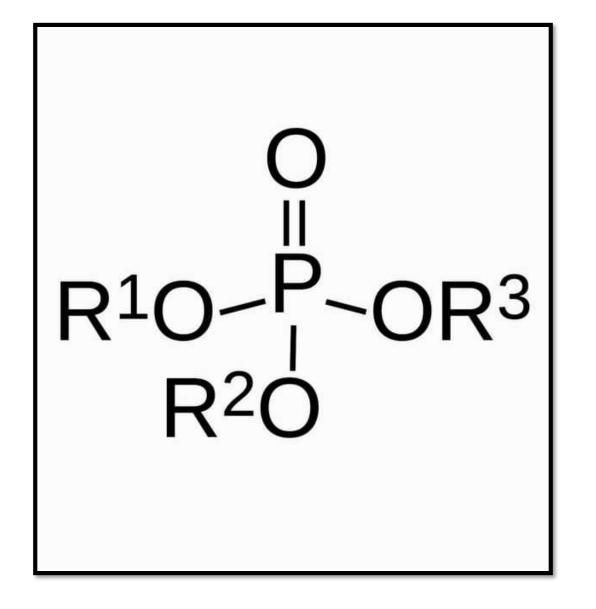
Phosmet

Fenitrothion

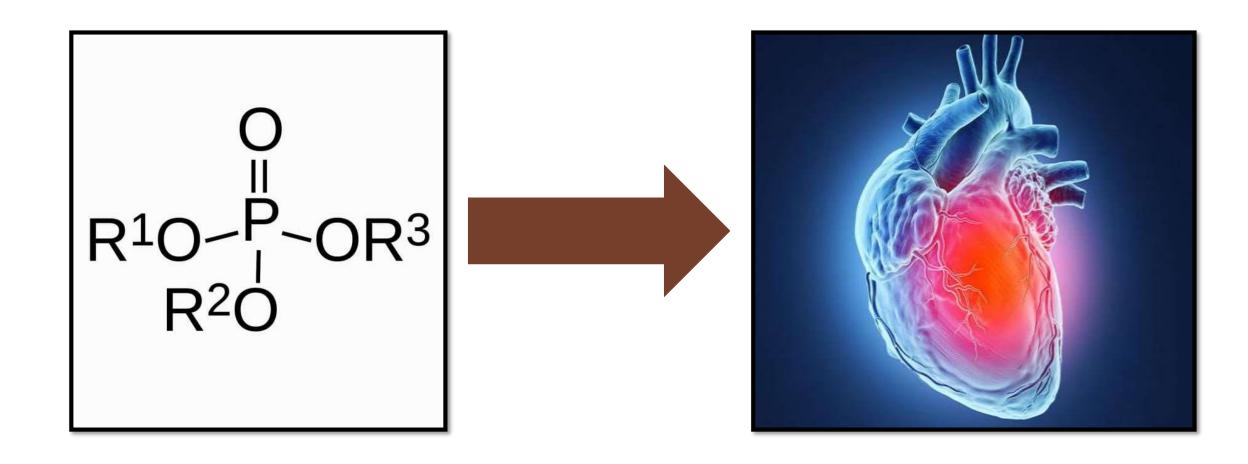
Malathion















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Organophosphates act by <u>irreversibly inhibiting</u> the cholinesterase enzyme, leading to accumulation of acetylcholine at neuromuscular junctions.



Organophosphates poison mainly by phosphorylation of acetylcholinesterase enzyme (AChE) at nerve endings. This results in deficiency of available AChE so organ become overstimulated by excess acetylcholine.

The **accumulation of acetylcholine** at neuromuscular junctions affects both the peripheral and central nervous systems. This results in symptoms associated with nicotinic and muscarinic receptor activation.

At skeletal muscle junctions, excess ACh may be excitatory (cause muscle twitching) but may also weaken or paralyze the cell by depolarizing the end plate. Impairment of the diaphragm and thoracic skeletal muscles can cause respiratory paralysis. In the CNS, high ACh concentrations cause sensory and behavioral disturbances, incoordination, depressed motor function and respiratory depression.



Additionally, the toxic effects of organophosphates may provoke excessive sympathetic stimulation



followed by parasympathetic activity



culminating in QT interval prolongation



According to case research done by Department of Medicine, Jawahar Lal Nehru Medical College, DMIMS, Sawangi, Wardha, Maharastra
Published on National Library of Medicine

Pesticides release increased the amount of catecholamines and other vasoactive amines (histamines and neutral proteases) that penetrate the collagen matrix of plaque causing erosions and rupture which can lead to myocardial injury. These inflammatory mediators can cause coronary thrombosis, as well as spasm leading to myocardial infarction.



According to case research done by Department of Medicine, Jawahar Lal Nehru Medical College, DMIMS, Sawangi, Wardha, Maharastra
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Pesticide exposure can also trigger the release of inflammatory substances, potentially causing coronary thrombosis and spasms, both significant contributors to myocardial infarction.

In this case, the patient exhibited left ventricular hypertrophy, epicardial hemorrhages

30% luminal narrowing of the left anterior descending artery.

Multiple fatty streaks and atheromatous plaques were also observed.





CONCIUSION

- Although the exact pathophysiological mechanisms linking organophosphate poisoning to cardiac complications are <u>not fully understood</u>, this case highlights the need for further research in this area, making it a compelling subject for study.
- Rare cases like these are an indication that severe complications like myocardial infarction is a fatal symptom of Organophosphate poisoning, especially in developing countries like India.
- Therefore, it is essential for the clinicians to assess cardiac findings in organophosphate poisoning, as it may lead to myocardial infarction which is fatal.





THANK YOU!

