TOXOPATHOLOGICAL STUDY OF METHAMYL EFFECT ON THE ROCK PIGEONS (CULUMBA LIVIA GADDI)

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ABSTRACT

This Study was conducted in animal house in Faculty of veterinary medicine of university of kufa. This study was designated to assess the neuropathy of Organophosphorus pesticide (Methomyl) after long–term administration of low dose in Rock pigeons. The Clinical signs, gross lesions and histopathological assessment of nervous tissue were described. A total of 20 pigeons, 3 months old and about 500g average body weight pigeons were used in this study. The pigeons were equally divided into two groups. The dose was calculated based on study at close pilot at 0.02 ml and gradually lose the dose down to the toxic non-lethal dose. The toxic dose was 0.01 ml. One group was daily administered via oral gavages with 0.01 ml of methomyl; for 21 days where as the other group were leave control without administration. Organophosphorus pesticide treated groups exhibited both muscarinic and nicotinic signs of toxicity.

INTRODUCTION

Methomyl is a water-soluble crystalline solid that gives off a sulfurous odor. Highly toxic, it is classified as a carbamate insecticide that is designated as being a restricted use Pesticide (RUP) by the U.S. Environmental Protection Agency (EPA). [1]

Since the late 1960s, the substance has been used as a pesticide on commercial fruit and vegetable crops as well as stored products. Its application as an insecticide is highly effective against a wide variety of pests, particularly those that are resistant to
organophosphorus pesticides; also it is a carbamate pesticide that induces acute cholinergic poisoning in birds by reversible inhibiting acetylcholinesterase activity. [2]

In the Netherlands, methomyl is not legally registered for use as an active substance in pesticides. However, intoxications with non-registered pesticides have been reported before. The goal of this review, therefore, is to summarize clinical and post-mortem aspects that should be considered in forensic investigations of suspected methomyl intoxications. Ingestion and other exposures to the methomyl can cause various symptoms. The type and severity of symptoms varies depending on the amount of chemical involved and the nature of the exposure. The chemical may be absorbed through the skin. [3]

Poisoning by methomyl is readily absorbed through the skin and by inhalation of fine particles. Its mechanism of action is inhibit cholinesterase, an enzyme produced in the liver that regulates nervous system functioning. The telltale symptoms of toxicity by this route are uncontrolled muscle movements, spasms, convulsions, etc. The insecticide is also absorbed through the intestinal tract. In fact, ingestion of this substance equates to a fast-acting poison in both humans and animals. However, if the ingested dose is not too high and action is taken quickly, poisoning may be counterchecked by one or more injections of atropine. Metabolism of methomyl occurs by hydrolysis before it degrades into the byproducts carbon dioxide and acetonitrile. [3] & [4]

The poisoning by methomyl can causes a serious health and economic especially great on farm animals, especially birds as poultry, pigeons, ducks and geese plus fish to lakes, Therefore prepared this study to find out
1. Gross and histopathological changes resulting from the impact of effect of methomyl.
2. Knowledge of the clinical signs seen on the animals given the poisoning material.

MATERIAL AND METHODS

A 20 pigeons, 3 months old with average 500g body weight pigeons have been used in this study. The pigeons were equally divided in to 2 groups. One group administered via oral gavage which 0.01g of methomyl dissolved in 10 ml distal water, where the second group is control. Administration was continued for 21 days
and observed clinical manifestations. The pigeons has been killed and subjected for post-mortem examination of Brian, spinal cord, sciatic nerve and liver.

Postmortem examinations were done for all animals. The macroscopic appearance was recorded to detect any abnormal gross change in the internal organs, including location, color, size, shape, consistency and appearance of cut section.

Specimens were taken from internal organs include (liver, kidney, lymph node, heart blood, spleen and lung) the tissues were kept in 10% formaldehyde solution immediately after removal. After 72 hrs. Of the fixation the specimens were washed with tap water and then processing was routinely done with a set of upgrading alcohols concentration from 50% to absolute 100% for two hrs. in each concentration to remove water from the tissue, then clearance was done by xylol, then the specimens were infiltrated with semi-liquid paraffin was at 58c in two stages, then blocks of specimens were made with paraffin wax and sectioned by rotary microtome at 5 um for all tissue. All tissues were stained with Hematoxylin and eosin and the histopathological changes were observed under light microscope. [5]

RESULTS AND DISCUSSION

The clinician results appeared on pigeon is excess salivation ,termer, wing dropping, eye dilator ,enlargement of blood vesicles of eye, gasping, dyspnea , congestion of eye, severe diarrhea, incoordination, and nervous signs.(Figure 1,2,3,4,5,6 &7)

The grossly results on liver enlargement, pail, adhesions, congestion. And results on brain are congestion, adhesions. (Figure 8,9,10&11)

The histopathological results on liver fatty degeneration and hepatocyte appeared ring shape, congestion of sinusoid, vacuolation and congestion, hemorrhage and blood vesicles increase in thickness, fibrosis (fibrous C.T formation with hemorrhage ), pigmentory cirrhosis (fibrous C.T formation with accumulation of bile pigment bilirubin.(Figure 12 ,13, 15,16 ,18 ,19 ,21 &24)

The histopathological results on brain are vacuolation and hemorrhage, congestion, edema, congestion and microgilliosis. (Figure 13, 14, 17, 20 & 22)

The histopathological result on spinal cord is vacuolation. (Figure 23) Out of total cases presented with vomiting, diarrhea, excessive salivation, blood vessels congestion of eye and Pupil expansion with nervous signs such as wing paralysis,
convulsions and respiratory distress at end stage. While on postmortem examination, cases revealed froth and cyanosis at fingernails. All cases of poisoning were presented with above symptoms in more or less same proportions. These signs and symptoms are in agreement with authors [6], [7], [8]. Also external examination finding by [9] shows that cyanosis at fingernails and lips. Froth from mouth and nose was observed in cases. On gross examination, cases had enlarged liver while shown reduced size, congested and pale on cut section.

As shown in histopathological section, the cases affecting liver poisoning constitute and cause fatty changes, congestion, centrilobular necrosis and Sinusoidal dilatation in affected birds. Methomyl group affected liver in birds showed all these lesion and agreement with [10].

The liver showed congestion, blood vessels congestion of hepatic tissue occur because the effect of IL-1 and TNF-α which product by macrophages and it's direct effect on the endothelial cell line of blood vessels in hepatic tissue [11]. Pre vascular cuffing and multifocal fibrosis as a results of methomyle poisoning that play a role in infiltration of leukocytes around central vein also causes inflammation and inflammatory reaction in bile duct in liver, Other lesion which occur during the days of poisoning such as fibrosis related to injury that caused by TNF-α and TL-1 [12].

The histopathological result on brain is vaculization and hemorrhage, congestion, edema, congestion and microgilliosis and the histopathological result on spinal cord is vaculization becused the best-known effect of the organophosphate compounds is inhibition of acetylcholinesterase enzyme (AChE), which causes the accumulation of acetylcholine (ACh) in the body. The inhibition of cholinesterase activity leads to accumulation of ACh at synapses, causing overstimulation and disruption of neurotransmission in both central and peripheral nervous systems. Finally, acute organophosphate poisoning may result in serious life-threatening conditions, such as initial acute cholinergic crisis, and sometimes intermediate syndrome. For this reason, early recognition of such conditions is very important, especially to institute the appropriate treatment, the mortality rate of organophosphate poisoning is high, fatal issue is often related to delay in diagnosis or improper management. Acute treatment includes rapid administration of atropine, which blocks the muscarinic effects, and that of pralidoxime, which reactivates the AChE inhibited by the organosphosphate. At present, the use of the traditional antidotes, atropine and oximes, has not significantly reduced the morbidity and mortality of organosphosphate poisonings.
despite the great advances in patient monitoring and critical care medicine. The need to develop newer treatment regimens is urgent. Interleukin-10 is a recently characterized cytoprotective agent, and it may be useful as an alternative or adjunctive therapy in organophosphate poisonings, but has been studied little in connection with organophosphate poisonings. [13].
Figure 11. Liver histopathology showing liver fatty degeneration and asring shes.

Figure 13. Liver histopathology showing liver fatty degeneration and asring shes.

Figure 5. Histological section 10X

Figure 17. Histological section 10X
Figure 15. Brain histoc
brain vacuolation and hemo

Figure 16. Tissue section
showed increased 10X

Figure 17. Brain histopathol
showed brain oedema

Figure 18. Tissue section
showed increased 10X
Figure 19. Liver histopathology showed liver hemorrhage and block thickness.

Figure 21. Liver histopathology showed liver blood vessel thickness.

Figure: pathological section congestion, 10X

Figure: pathological section congestion, 10X
دراسة التأثير السمي المرضي لمبيد الميثوميل على الحمام الصخري

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