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

Dhiaa J. Hamzah* Mohammed A. Abo- Ktifa** Saif S. Rasheed*
Bushra H. Faris*

* Department of Pathology and Poultry Diseases, College of Veterinary Medicine, University of Kufa, Kufa, Iraq.

** Department of physiology and pharmacology, College of Veterinary Medicine, University of Kufa, Kufa, Iraq.

(Received 12October 2014 ,Accepted 21 December 2014)
Keywords; Methomyl**,** toxicity, pigeons.

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, 3months old and about 500g average body weight pigeons were used in this study. The pigeons were equally divided in to tow 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, vacculation 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 vacculation and hemorrhage, congestion, edema, congestion and microgilliosis. (Figure 13, 14, 17, 20 & 22)

The histopathological result on spinal cord is vacculation. (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 vacculation and hemorrhage, congestion, edema, congestion and microgilliosis and the histopathological result on spinal cord is vacculation 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

Figure

Figure 3.

Figure 4. Seve and

Figure 5. Dys

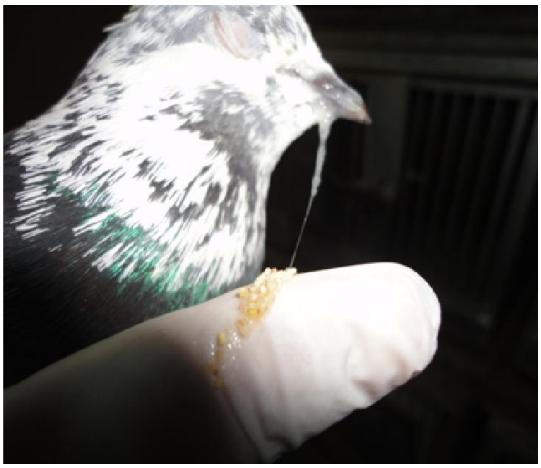


Figure 6.

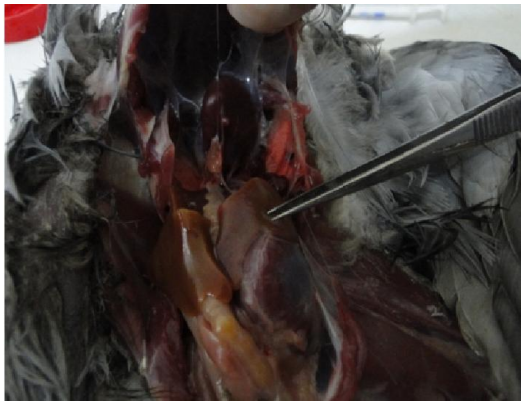


Figure 7. Liver

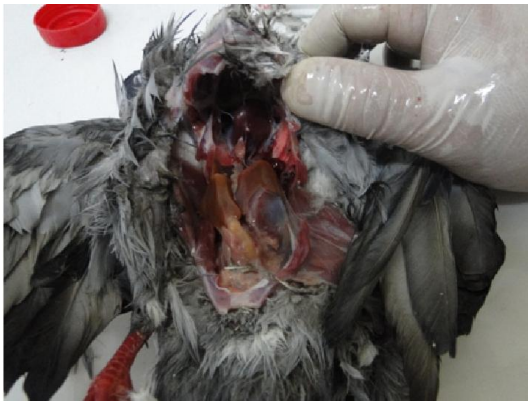


Figure : nd pale

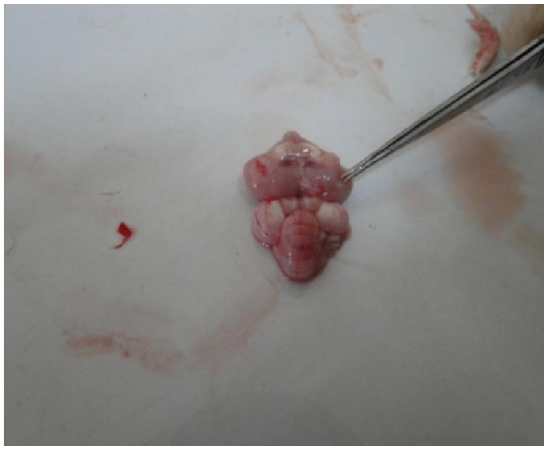
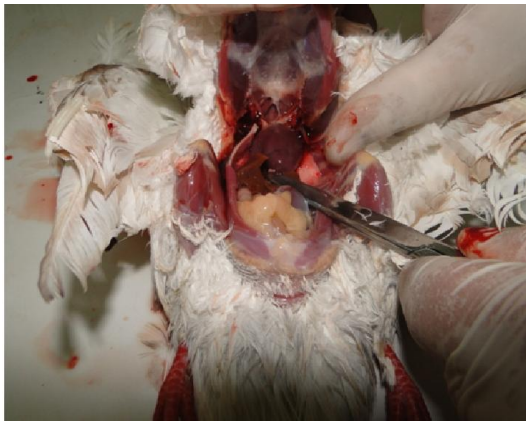


Figure 9. B



Figu

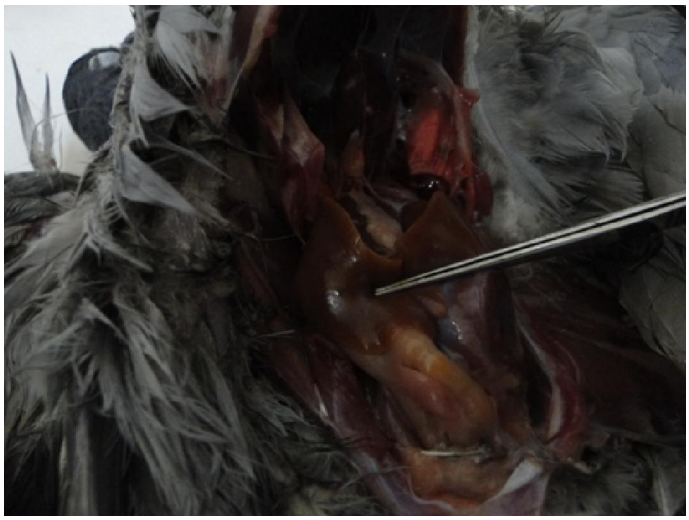


Figure 11. Li

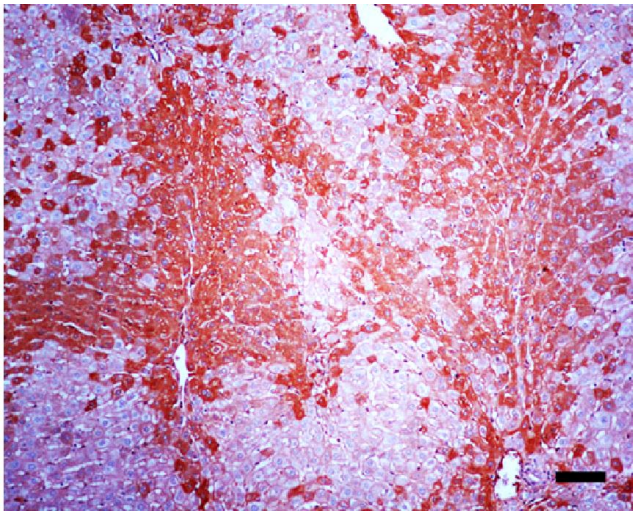


Figure
s
al section
10X

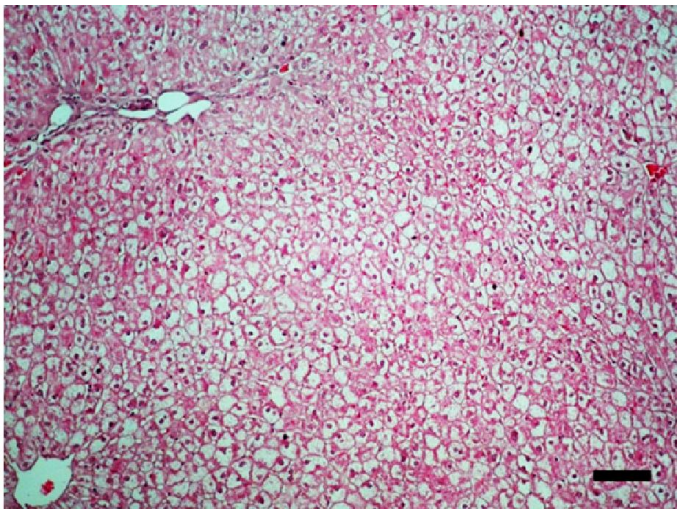


Figure 13 . Liver histopath
liver fatty degeneration an
as ring sha
ed
ed

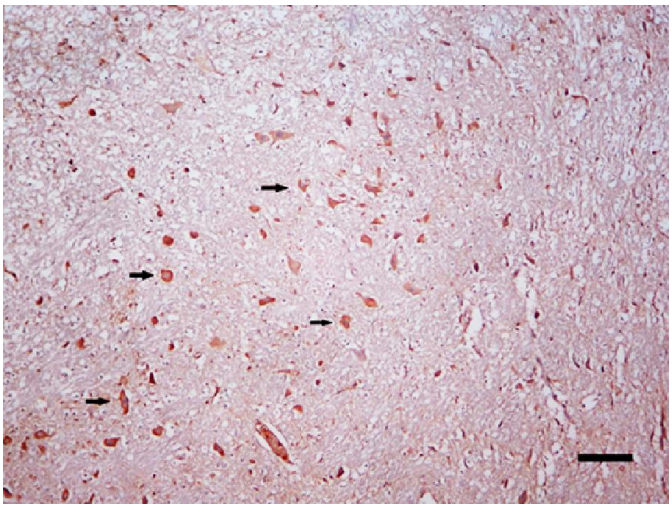


Figure
:
cal section
10X

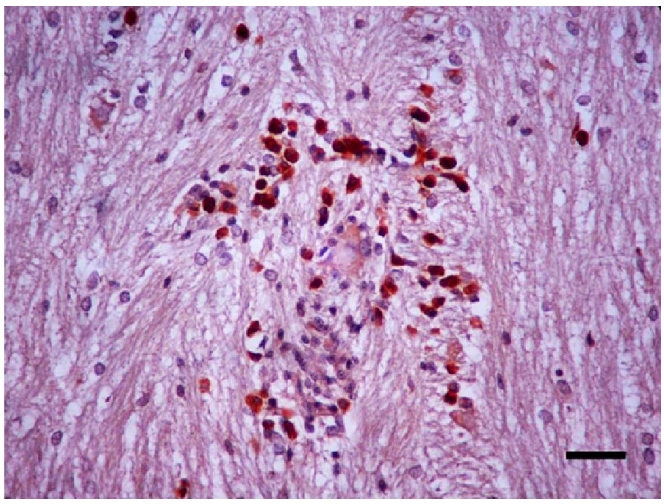
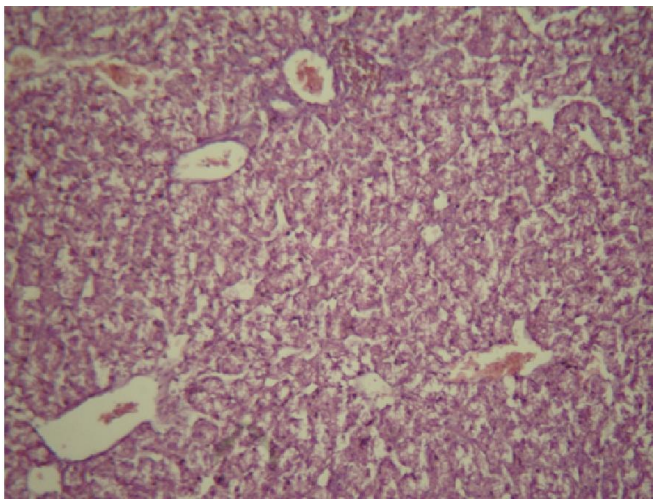


Figure 15 . Brain histo n
brain vacculation and hemc d



Figur gical section
showe ngestion.10X

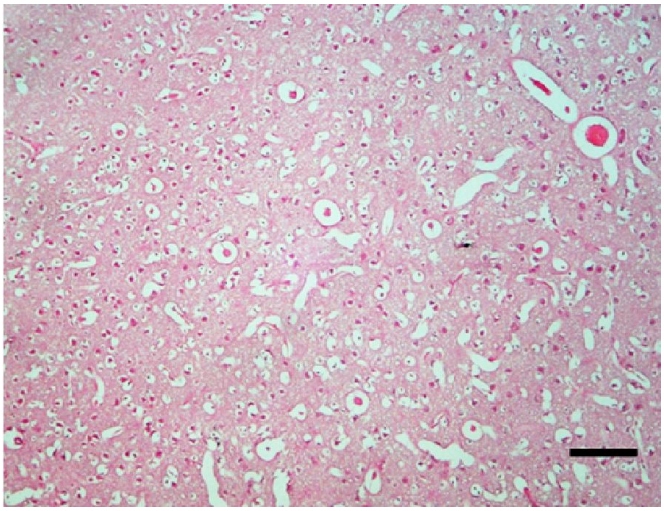


Figure 17. Brain histopathol
showed brain odem

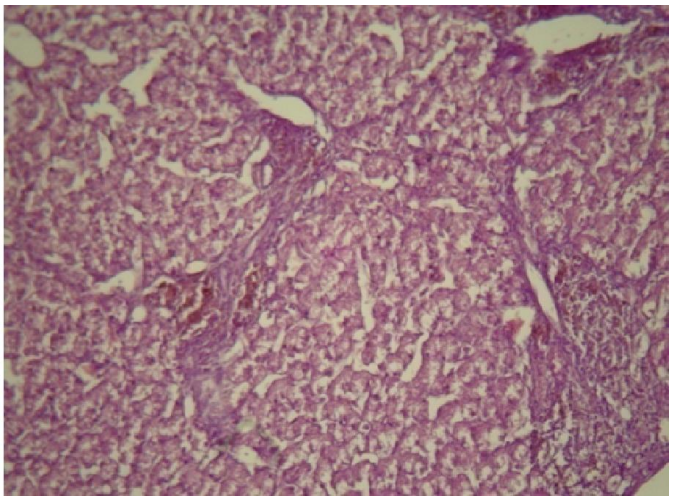


Figure gical section
showe .T formation
10X

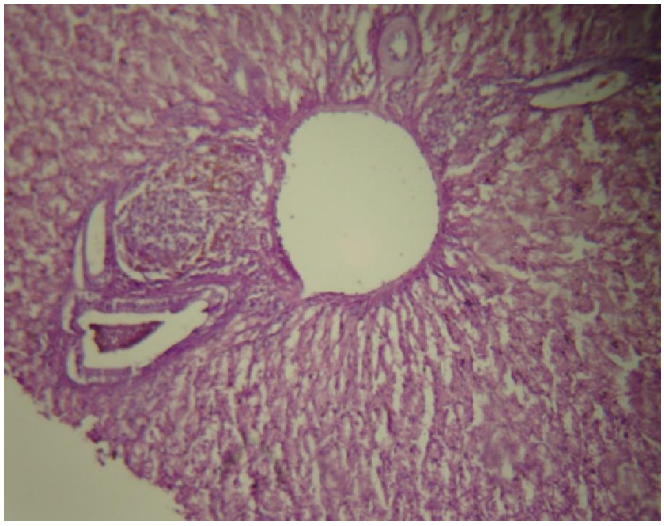
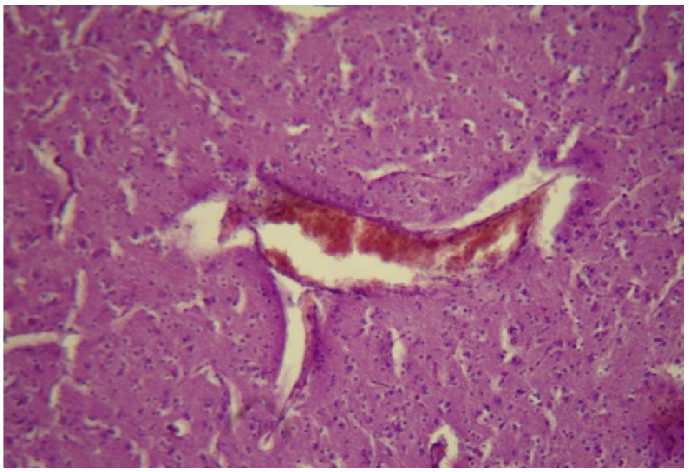


Figure 19 .Liver histopatho ed
liver hemorrhage and bloc n
thickness



Fi
showe

ological section
icrogiliosis.10X

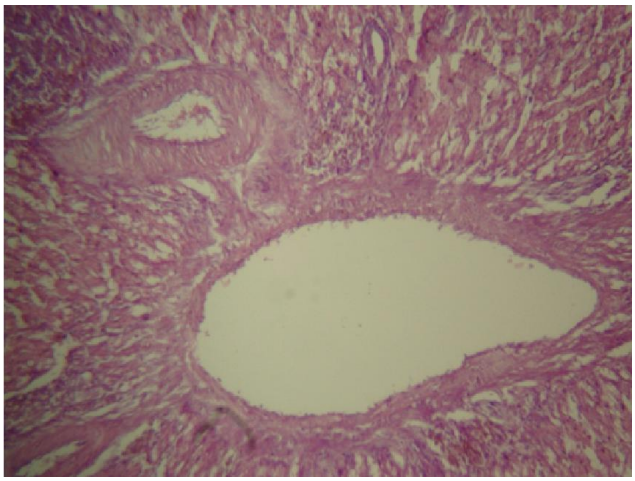


Figure 21. Liver histop
showed liver blood ve
thickness

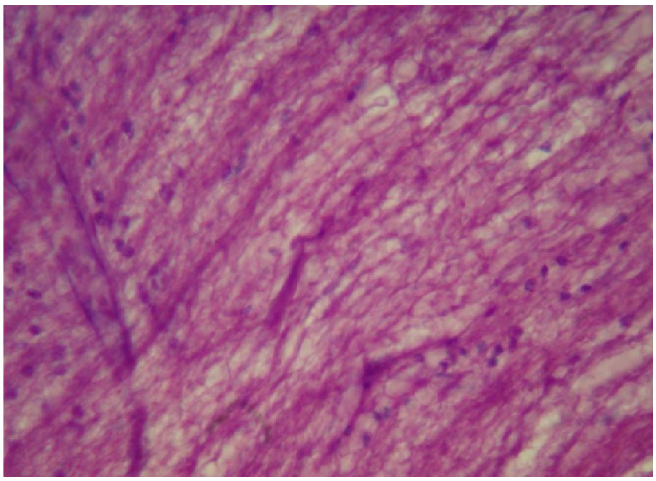


Figure
showed b

cal section
ogiliosis.10X

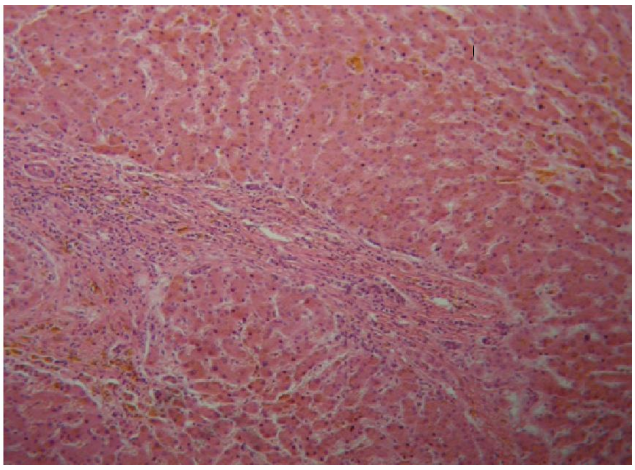


Figure 24. Liver histopath
showed liver pigmentary sir
formation with accumulatic
billerobin).

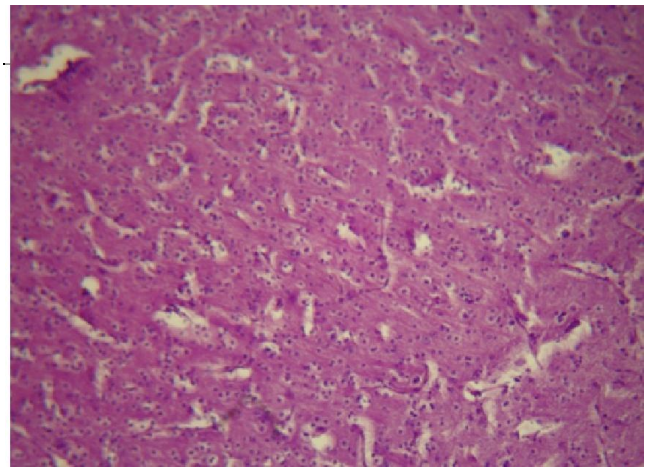


Figure23 . Spi
sh
al section
ition. 10X

دراسة التأثير السمي المرضي لمبيد الميثوميل على الحمام الصخري

ضياء جابر حمزة* ، محمد عبد الغفار ابو كطيفة** ، سيف ستار رشيد*

بشرى حمزه فارس

* فرع الأمراض وأمرض الدواجن، كلية الطب البيطري،جامعة الكوفة ،النجف العراق.

** فرع الفسلجة والادوية ،كلية الطب البيطري ،جامعة الكوفة ،النجف العراق.

الخلاصة

أجريت هذه الدراسة في البيت الحيواني في كلية الطب البيطري في جامعة الكوفة. و عملت هذه الدراسة لمعرفة تأثير احد مبيدات الفوسفورية العضوية (الميثوميل) وأحداث الاعتلال العصبي بعد فترة طويلة من التجريع وبجرعة منخفضة في الحمام البري . وقد أظهرت التجربة أعراض هذا المرض، وتقييم الآعراض بالفحص النسيجي لأنسجة الجهاز العصبي .وتتكون التجربة من ٢٠ حمامة بعمر٣ أشهر ، ٥٠٠ غم وزن الجسم . قسم الحمام إلى مجموعتين متساويتين تم حساب الجرعة على أساس دراسة تجريبية بجرعة ٠.٠٢ مل وتقل الجرعة تدريجيا وصولا الى جرعة سامة غير قاتلة. كانت الجرعة ٠.٠١ مل و تجرع يوميا لمجموعة واحدة عن طريق الفم بجرعة مقدارها ٠.٠١ مل من الميثوميل؛ لمدة ٢١ يوما .أما المجموعة الأخرى كانت مجموعة السيطرة تركت بدون تجريع. المجموعة المعرضة للتجريع بمبيدات (الميثوميل) عانت من أعراض المسكرينية و النيكوتينة.

REFERENCES

1-Gupta R.C., Milatovic S., Dettbarn W.D., Aschner M. and Milatoric D. (2007): Toxicol App and Pharmacol, 219(2-3), 97-105.

2- Andersen H. R., Vinggaard A. M., Rasmussen T. H., Gjermandsen I. M. and Bonefeld-Jorgensen E.C. (2002): Toxicol. Appl. Pharmacol, 179, 1-12.

3-Fukuto T. R. (1990): Mechanism of action of organophosphorus and carbamate insecticides. Environ Health Perspect JUL; 87:245-54.

4-El-Fakharany II, Massoud AH, Derbalah AS and Saad Allah MS. (2011): Toxicological effects of methomyl and remediation technologies of its residues in an aquatic system. J Environ Chem Ecotoxicol 3(13):332–339.

5- Luna, L. G. (1968): Manual of histological staining methods of the armed forces institute of pathology 3 rd. ed. McGraw-Hill book Comp. New York.

6-Karalliedde L. and Senanayake N, (1989): Organophosphorous insecticide poisoning.Br J Anesthesia; 63:736-50.

7-Pillay VV. (2003): Comprehensive medical toxicology, Paras publishing, 1st ed.:2-52.

8-Reddy K. S. N. (2001): The essentials of Forensic Medicine and Toxicology 20th ed.:403-421.

9-Knight Bernard. (1996): Forensic Pathology; 553-581.

10-Dalal J. S. and Gorea R.K. (1998): Poisoning trends- A postmortem study; J. Indian Acad Foresic Med.; 20(2):27-31.

11- Kapoor, V.; Katoch, R.C.; Sharma, M.; and Asrani, R.K. (2004): Pathogenicity test of P. multocida in mice A: 1. Indian J. Anim. Sci. 74:495-496.

12- Cunningham, J. G. (2002): Textbook of veterinary physiology. 3rd Ed. Sanders, U.S.A. 575p.

13-Becher, B.; Fedorowicz, V. and Antel, J. P. (1996): Regulation of CD14 expression on human adult central nervous system-derived migroglia. J. Neurosci. Res. 45: 375-381.