دراسة الأعراض الاكلينيكية والآفات التشريحية والتغيرات الباثولوجية الناجمة عن تأثير مبيد القواقع (البايلوسيد) على اسماك بلطى النيل

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اجريت هذه الدراسة على ثمانين من أسماك بلطي النيل (تزن كل واحده مابين من أسماك على المعيشه المعمليه لمدة اسبوعين قبيل بدء التجربة وقد قسمت تلك الاسماك الى مجموعتين احدهما اجرى عليه التسميم الحاد والاخرى للتسم تحت المزمين .

وقد ظهرت الاعراض الاكلينيكية في التسمم الحاد ، فقد اشتملت على ظهور بعض الحركات العصبية مع احتقان شديد بالاوعية الدموية للعين والخياشيم مع فتال الفم باستمرار الاستلقاء على جانبها قبل نفوقها مباشرة ، هذا ولم تظهر ايا مال العلامات الاكلينيكية على اسماك البلطي في حالة التسمم تحت المزمن

ويعمل الصفة التشريحية وجد احتقان عام بجميع الاعضاء الداخلية (الطحـــال والكبد والمعده والامعاء وكذلك الخياشيم) وذلك في التسمم الحاد بينما اقتصــرت الافات التشريحية في التسمم تحت المزمن على احتقان الخياشيم مع اصفرار في الكبد

وقد اثبتت الدراسات الهستوباثولوجية للتسمم الحاد عن وجود احتقان شديد باوعية الخياشيم الدموية مع تكسير العديد من الزوائد الخيشوميه لها، وقد لوحيظ ايضا وجود احتقان عام في الأعضاء الداخلية، اما في حالة التسمم تحت المزمين فاوضحت الدراسة الباثولوجية عن عدم وجود تغيرات في الاعضاء الداخلية عيدا احتقان بالخياشيم والكبيد،

وقد استخلص الباحثون من هذه الدراسة مدى خطورة استخدام البابلوسيد كمبيد للقواقع خاصة في الجرعات عالية التركيز (التسمم الحاد) لما له من اضرار بالغــــــة بالاقتصاد القومـــي٠

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CLINICAL SIGNS, POST-MORTEM FINDINS AND HISTOPATHOLOGICAL CHANGES IN TILAPIA NICOTICA FISH INTOXICATED WITH BAYLUSCIDE

(With 6 Figures)

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SUMMARY

In acute toxicity induced by Bayluscide in Tilapia nilotica fish, degenerative and necrotic changes of gills and gut. hepatosis, nephrosis and congestion of the vasculature allover the body were noticed. In short-term toxicity by the same compound (1/10 LC) for six weeks, no mortality were recorded. Microscopically, congestion of the gills, hyperplasia of the covering epithelium of the gut and fatty change of the liver could be found.

INTRODUCTION

The practice of bilharziasis control in Egypt by direct introduction of the moulluscicide "Bayluscide" into rivers, canals and laks for destruction of Schistosomiasis snails, proved to be highly toxic to Tilapia nilotica fish (SHEHATA et al., 1985).

The available studies on the toxic effects of Bayluscide in fish were done in the united states of America by MARKING and HOGAN (1967) and by SHEHATA et al., (1985) in Egypt. Also, no available literatures concerning the histopathological changes induced in fish by Bayluscide were obtained.

The aim of the present study is to investigate the clinical signs, post-mortem finding and histopathological changes in both acute and short-term toxicity of Bayluscide.

MATERIAL and METHODS

Bayluscide was obtained from Bayer, Cairo scientifc office as wettable powder containing 70% active ingredient.

80 Tilapia nilotica fish, weigh from 100-150 gm. each; were used during our toxicological studies. The fish were obtained from El-Ibrahimia canal at Assiut locality and acclimatized to laboratory conditions at least two weeks befor experimental testing. Teteramine fish feed was twice daily ad-libidum and withold three days prior to introduction to bioassay to empty the gut (According to united states Department of interior fish and wildlife services Report, 1964).

Acute toxicity studies of Bayluscide were caried out by subjectin 40 fish to 1/2 LC (0.18 ppm) for one week. Also subchronic toxicity studies were done by subjecting 40 fish to 1/10 LC (0.0366 ppm) daily for six weeks. The LC was previously determined by

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The clinical mainifestation of intoxicated fish in both acute and subchronic toxicity were recorded. Also post-mortem examination was carried out for all tested fish. Tissue samples (gills, brain, liver, spleen, stomach, intestine, tests and ovaries), were taken from living fish, after 12, 24, 48, 72, 96, hours and one week in case of acute toxicity, and after 1, 2, 3, 4, 5. and 6 weeks in case of subchronic toxicity. The fixed tissues were embedded in paraffin wax and sections of 3 - 5 u thickness were prepared assusual and stained with haematoxylin and eosin and Masson trichrom stains. Frozen sections were also carried out from the liver and stained by sudan III stain, a specific stain for fat.

RESULTS

Clinical signs:

The clinical signs recorded in acute toxicity were, nervous movements, contenious opening of gill cover and mouth, fish lies on its side before it die. No clinical signs were observed on subchronic toxicity.

Post-mortem findings:

Congestion of the internal organs (spleen, liver, gut, intestine and brain), also, gills were the only finding recorded in acute toxicity. In subchronic toxicity, congestion of gills and yellow coloration of the liver were observed.

Histopathology:

1- Acute toxicity:

Gills showed severe congestion of vasculature, oedema and varying degree of degeneration. Also moderate number of the gill filaments were destructed. The destruction of the filaments proportionally increased in relation to the duration of exposure, (Fig. 1). Gut and intestine were showing oedema and congestion in the submucosa, also degeneration and sometimes focal areas of coagultive necrosis in the mucosa could be noticed (Fig. 2). At 12, 24, 48 hours, the pareanchymatous organs such as liver, kidney and heart revealed a granular proteinous dystrophy where the cytoplasm of the cells contain acidophilic granules with swelling of the cells (Fig. 3). While at the 3rd, 4th and 7h days, hydropic degeneration of the pareanchymatous cells were observed in liver, kidney and heart.

Spleen, brain, ovaries and tests were showing only congestion with perivascular oedema.

Subchronic toxicity:

During subchronic toxicity, all organs appeared normal, except gills, gut and liver. The gills showed congestion and desquamation of the opithelium of the filaments. The gut shwoed only hyperplasia of the epithelial surface (Fig. 4). Liver at the 3rd week, showed mild to moderate degree of fatty infilteration (Fig. 5). While at the end of the experiment, the liver revealed a severe fatty infilteration where all the hepatic cells appeared ocupied by fat droplets (Fig. 6). Also Haemosidrine pigmentation in the spleen could be observed.

BAYLUSCIDE IN TILAPIA NIOICA FISH

DISCUSSION

In the acute toxicity, the most common lesions in the gills, gut and intestine, were degenerative and local necrotic changes in the mucosa and congestion of the vasculature. We suggest that Bayluscide induce a mild direct histotoxic effect to which the alterative changes recorded in our results could be attributed. Also this suggestion is the main cause of the degenerative changes which observed in liver, kidney and heart.

The hyperplasia of the epithelium of the gut and intestine in the end of subchronic toxicity may be due to the regeneration processes of the necrotic changes, which observed in the begening of the test. The fatty change of liver seems to be due to the interference of Bayluscide with the cellular metabolism. The severe degenerative changes in the liver explained the decrease in the values of GPT, COT and alkaline phosphatase as an index for lowered liver function previously puplished by our Co-authers SHEHATA et al. (1985).

The appearance of haemosidrine pigments in the spleen may be resulted from lysis of the erythrocytes induced by the effect of the Bayluscide. The results of decrease Hb % and RBCs count recorded in subchronic toxicity study (SHEHATA et al., 1985) by the same compound confirming the previous evidence.

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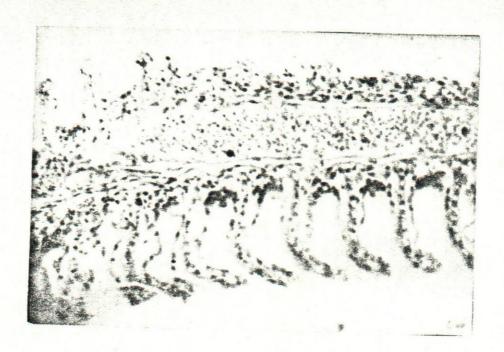


Fig. (1): Gills showed severe congestion, oedema and destruction of some filaments. (H.E. 12.5 \times 25).



Fig. (2): Intestinal villi showed degeneration and coagulative necrosis. (H.E. stain 12.5 \times 25).

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Fig. (3): Hepatic tissue showed granular protein dystrophy. (H.E. Stain 12.5 X 25).

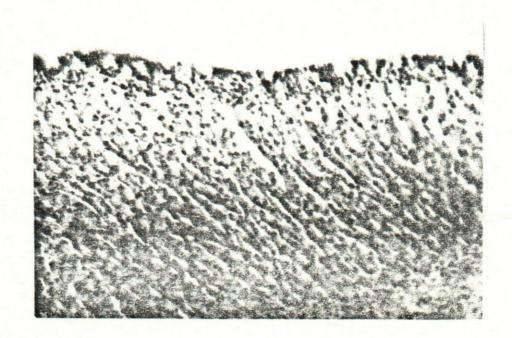


Fig. (4): Gut showed hyperplasia of the covering epithelium. (H.E. Stain 12.5 X 25).

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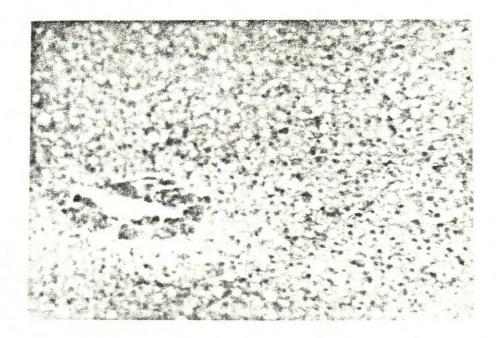


Fig. (5): Liver sowed moderate fatty infilteration of the hepatic cells. (H.E. stain 12.5 \times 25).

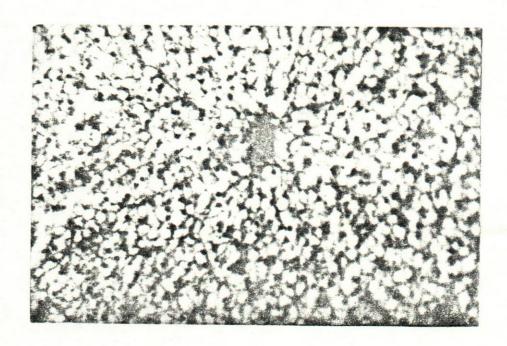


Fig. (6): Severe fatty infilteration of the hepatic cells. (H.E. stain 12.5 X 25).

