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دراسة الأعراض المكملية والآفات التشريحية والتغيرات الباثولوجية الناجمة عن تأثير مبيد القوافع (البايالوسيد) على أسماك بلطي النيل

علم نفاذ، عادل شحاته، ثابت عبد النعيم، عبدالعزيز شعبان

يهدف هذا البحث إلى دراسة الأعراض المكملية والآفات التشريحية والتغيرات الباثولوجية الناجمة عن كل من التسم الحاد تحت المزمن على أسماك بلطي النيل بمادة الباليوسيد المستخدمة بجمهرية مصر العربية كمبيد للقوافع.

اجريت هذه الدراسة على ثمانية من أسماك بلطي النيل (تسار كل واحدة ما بين 100-150 جرام)، وقد تراقبة هذه الأسماك على العيادة العملية لمدة أسبوعين قبل بدء التجربة. وقد قسمت تلك الأسماك إلى مجروعتين احدهما أجرى عليه التسمم الحاد والأخرى للتسمم تحت المزمن.

وقد ظهرت الأعراض المكملية في التسم الحاد، فقد اشتملت على ظهور بعض الحركات العصبية مع احتقان شديد بالأوعية الدموية للعين والخلايا مع فتح الفم باستمرار الاستلقاء على جانبها قبل نفوقها مباشرة، هذا ولم تظهر أي علامات الأكليمنية على أسماك البلطي في حالة التسم تحت المزمن.

وبعد انتهاء التشرحيه، وجد احتقان عام بجميع الأعضاء الداخلية (الطيب، الكبد، الدماغ، والكبد) وذالك في التسم الحاد. بينما اقتصرت الآفات التشريحية في التسم تحت المزمن على احتقان الخلايا مع اضطرار في الكبد.

وقد أثبتت الدراسات الهستوباثولوجية للتسم الحاد عن وجود احتقان شديد باوعية الخلايا الدموية مجتمعة في الأوعية الدموية، بما في حالة التسم تحت المزمن فافتحت الدراسة الباثولوجية عن عدم وجود تغيرات في الأعضاء الداخلية.

وقد استخلص الباحثون من هذه الدراسة مدى خطورة استخدام البايالوسيد كمبيد للقوافع خاصة في الجرعات عالية التركيز (التسم الحاد) لما له من اضرار بالغة بالاقتصاد القومي.
CLINICAL SIGNS, POST-MORTEM FINDINGS AND
HISTOPATHOLOGICAL CHANGES IN TILAPIA NICOTICA FISH
INTOXICATED WITH BAYLUSCIDE

(With 6 Figures)

By

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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 allow 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 scientific 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 before experimental testing. Teteramine fish feed was twice daily ad-libidum and withheld 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 carried out by subjecting 40 fish to 1/2 LC (0.16 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


The clinical manifestation 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 μ thickness were prepared as usual 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, continuous 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 coagulative necrosis in the mucosa could be noticed (Fig. 2). At 12, 24, 48 hours, the parenchymatous 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 7th days, hydropic degeneration of the parenchymatous cells were observed in liver, kidney and heart.

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

2- **Subchronic toxicity:**

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

REFERENCES

United States Department of Interior fish and wildlife service (1964): Procedure for evaluation of acute toxicity of pesticides to fish and wildlife.

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

Fig. (2): Intestinal villi showed degeneration and coagulative necrosis.
(H.E. stain 12.5 X 25).
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).
Fig. (5): Liver showed moderate fatty infiltration of the hepatic cells. (H&E stain 12.5 X 25).

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