RECURRENT YERSINIOSIS IN CULTURED OREOCHROMIS NILOTICUS ASSOCIATED WITH IMPROPER MANAGEMENT

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ABSTRACT

Yersiniosis caused by Yersinia ruckeri was studied in Oreochromis niloticus fish farm in Egypt. Recurrences of the disease condition occurred in summer season and correlated with improper management represented by poor water quality and continuous farming operations. Symptomatic and asymptomatic infections were recorded in naturally and experimentally infected fish where the principal signs of the disease were congestion of the head region, bases of the fins and internal organs. Naturally, the percents of recovery of Y.ruckeri from clinically infected, asymptomatic dead and apparently healthy fish as well as from their corresponding pond water samples reached 88 %, 76 %, 56 % and 100 %, 80 %, 40 % respectively. Experimentally, cumulative morbidity and mortality percents of intraperitoneally infected fish groups with Y.ruckeri at 22 ± 1°C and at 30 – 1°C reached 60%, 100% and 25%, 55% respectively; however, these percents reached 35%, 95% and 10%, 40% in fish groups kept in immersion with Y.ruckeri at the same temperatures respectively. Histopathological changes were mainly inflammatory and degenerative in nature. The prevention and principals of disease control were also discussed.

INTRODUCTION

Enteric redmouth (ERM) is an acute or chronic infection of fish caused by Yersinia ruckeri, a Gram-negative enteric bacterium generally widespread in freshwater environments **(Noga, 2000)**. Yersinia ruckeri is present in a carrier state in many species of fish and remains undetected until stress mediated dominantly by poor water quality occurs **(Woo and Bruno 1999)**. Enteric redmouth is considered primarily a disease of channel catfish Ictalurus punctatus **(Lewis, 1981)** and salmonids **(Busch, 1982)**, however, infection was also recorded in eel Anguilla anguilla **(Fuhrmann et al., 1984)**, common carp Cyprinus carpio **(Fuhrmann et al., 1984)** , gold-

fish Carassius auratus **(Mc Ardle and Dooley-Martin 1985)**, sole Solea solea **(Michel et al., 1986)**, bighead carp Aristichthys nobilis **(Xu et al., 1991)** and silver carp Hypophthalmichthys molitrix **(Xu et al., 1991)** .

The early terminology used to describe the disease , such as redmouth , redthroat or bacterial septicaemia was derived from the gross clinical signs **(Ross et al. 1966 and Rucker 1966)** . however, standardization as enteric redmouth was made by the Fish Health Section of the American Fisheries Society in 1975 and this, with yersiniosis , remained the common names .

Enteric redmouth disease has been identified in most areas of Europe , Canada , USA , Australia and Africa where freshwater fish are cultured **(Mc Daniel 1971 , Wobeser 1973 , Green & Austin 1982, Bragg & Henton , 1986 and Noga 2000)** . The principal signs of ERM are inflammation in the head area especially the mouth, haemorrhages in the intestine and congestion of the bases of the fins **(Fuhrmann et al., 1984)**. Clinical conditions similar to ERM caused by different Gram-negative rods: Aeromonas hydrophila and Pseudomonas flourescens have been recorded **(Roberts, 2001)**, however, the signs of classical redmonth disease are usually produced by Y. ruckeri, a member of the family Enterobacteriaeeae **(Ewing et al., 1978)** .

Enteric redmouth disease may be controlled to acceptable levels in fish farms by good husbandry, particularly high water-quality and antibiotic treatment. Even so, it presents a constant threat that erodes the profitability of the farms on which it is endemic **(Woo and Bruno, 1999)**.

This work characterizes the occurrence of recurrent yersiniosis in summer season in an Oreochromis niloticus fish farm in El-Sharkia province in Egypt. Clinical, bacteriological and histopathological examinations of naturally and experimentally diseased fish as well as the role played by water quality and improper management associated with such infection are studied. The principals of disease prevention and control are also discussed.

MATERIALS AND METHODS

Fish

*** Naturally affected fish Samples**

A total number of 50 O. niloticus of body weight ranging between 60 to 200 g represented by clinically infected (living and freshly dead), asymptomatically dead and apparently healthy fish were collected from earthen ponds of a commercial fish farm in El-Sharkia province in Egypt suffering from low mortality and a septicaemic disease condition .Collected fish were examined clinically and bacteriologically as described by **Post (1987) and Austin & Austin (1999)**.

*** Fish for experimental infection**

Apparently healthy O. niloticus of an average body weight 90 ± 10 g were collected from a commercial disease free fish farm and divided into six equal groups of 20 fish each in glass aquaria supplied with aerated and dechlorinated freshwater. Water temperature in all aquaria was thermostatically adjusted at 22 ± 1°C. The fish were fed a commercial ration and left for ten days for acclimation.

Pond water samples

Water samples were collected from affected as well as apparently healthy fish ponds and subjected to physico-chemical analysis for the following parameters : Temperature , pH , unionized ammonia , nitrite and dissolved oxygen , using the standard methods **(Dass, 1989)** non-ionized.

Bacteriological examination

- Collected fish samples were bacteriologically examined **(Balows et al., 1992)**. Samples taken from internal organs of fish (liver- kidneys - spleen and intestine) were cultured on tryptic soy agar (TSA - Gibco) and 10% sheep blood agar (BA - Oxoid) plates. The plates were incubated aerobically at 25°C for 48 h.
- Pond water samples taken under aseptic precautions were also bacteriologically examined **(Balows et al., 1992)**.
- Bacterial isolate was identified by colonial morphology, microscopic appearance and physiochemical characteristics **(Balows et al., 1992 and Cowan & Steel, 1993)**, using API 20E system for identification (Bio Mericux - France).

Antimicrobial susceptibility

Antimicrobial sensitivity patterns of the recovered isolate were determined by the disc diffusion method **(Koneman et al., 1997)** using Muller - Hinton agar (Oxoid). The following antimicrobial discs **(Bio Merieux, France)** were tested:

ampicillin , chloramphenicol , enrofloxacin , gentamicin , kanamycin , nalidixic acid , neomycin , novobiocin , sulphonamide , oxolinic acid and oxytetracyline.

Experimental design

Fish in the first experimental group were intraperitoneally (I/P) injected with a dose of 0.2 ml of Y.ruckeri cell suspension per fish (LD_{50} $1.3x10^7$ colony forming units, CFUs, ml^{-1} according to **Reed and Meunch, 1938**). Infectivity in the second experimental group was carried out **(Bullock et al. 1971)** by removing fish from their aquarium and immersing in 2-Liter cell suspension of Y.ruckeri ($1.3x10^7$ colony forming units, CFUs ml^{-1}) for 5 minutes and then returning

fish to their home aquaria . Fish in the third experimental group were treated with PBS and left as a control group. The three fish groups were continuously kept at 22 ± 1°C. The fourth, the fifth and the sixth fish groups were similarly treated as mentioned in the first three groups and subjected to rise in water temperature from 22 ± 1°C to 30 – 1°C **(Bullock et al. 1971)** . All fish groups were clinically examined throughout six weeks where internal organs (liver, kidneys, spleen and intestine) of representative fish samples were collected and subjected to bacteriological examination.

Histopathologieal examination

Tissue specimens taken from muscles and internal organs (liver, kidneys, spleen and intestine) of natutally and experimentally infected fish were preserved in 10% formalin solution, embedded in paraffin, sectioned at 5µm , stained with hematoxylin and eosin (H&E), and examined microscopically **(Roberts, 2001)** .

RESULTS AND DISCUSSION

Examination of clinically infected, asympromatic dead and apparently healthy fish revealed that symptomatic infection occurred in large sized fish (more than 150 g) , while smaller fish were generally died asymptomatically. The dominant expressed signs of clinically infected fish were dark skin colouration, deep seated haemorrhages of the mouth and congestion of the head tissues especially the operculum (Fig.1 and 2). Congestion of the bases of the fins and vent region were frequently encountered. However, variable degrees of septicaemic pictures affecting internal organs (liver - kidneys - spleen - intestine) as well as abdominal musculature were internally seen. Meanwhile, asymptomatic dead fish appeared externally normal; however, internally there were mild degrees of septicaemia affecting internal organs. On the other hand, apparently healthy fish appeared normal both externally and internally. The recorded disease condition represented a classical form of ERM **(Noga, 2000 and Roberts, 2001)** and confirmed the results of **Stevenson et al., (1993)** who succeeded to recover Y.ruckeri from some diseased and asymptomatic fish species. The disese condition may be confused with other Gram-negative haemorrhagic septicaemias but the virtual confinement of external lesions in the present study to the head of affected fish is a clinical feature of some significance, supporting the findings of **Ewing et al. (1978).**

Recovered bacterial colonies on TSA were yellowish white, smooth, round and rose up to 1 - 1.5 mm (Fig. 3). The bacteria were Gram-negative rods, motile and cytochrome oxidase - negative. Based on cultural and phenotypic characteristics (Table 1), the bacterial isolate was identified as Y.ruckeri **(Austin & Austin, 1989 and Inglis et al. 1993).**

The recovered Y.ruckeri isolates were sensitive to oxytetracycline, enrofloxacin, neomycin, oxolinic acid, sulphonamide and chloramphenicol and resistance to nalidixic acid, ampicillin, gentamicin, kanamycin and novobiocin. The results were supporting the findings of **Martinsen et al. (1992) and Inglis et al. (1995).**

Yersinia ruckeri was successfully recovered from internal organs of examined fish and pond water samples. The percents of recovery of Y.ruckeri from clinically diseased , asymptomatic died and apparently healthy fish as well as their corresponding pond water samples reached 88, 76, 56% and 100, 80, 40% respectively (Table 2). The recovery of Y.ruckeri from the intestine of asymptomatic died and apparently healthy fish may suggest the presence of sub-clinical and / or carrier state of infection , supporting the results of **Stevenson et al. (1993)** who succeeded to recover Y.ruckeri from some diseased and asymptomatic fish species. However, **Busch and Lingg (1975)** demonstrated those 45 days postinfection, 25% of a rainbow trout population carried Y.ruckeri asymptomatically in the lower intestine and maintained the pathogen indefinitely. Thus the variations in the prevalence of Y.ruckeri in examined fish and pond water samples of the present study could be explained on the bases of cyclical shedding of Y.ruckeri from asymptomatic carrier fish **(Bruno & Munro, 1989)** .

Concerning water quality, data in Table 3 revealed an increase in the values of water temperature, ammonia and nitrite and decrease in the value of dissolved oxygen in comparison to the optimum standards **(Brown and Gratzek 1980).** The recorded water temperature range of 33.6 - 35.2 in summer season could be considered high enough to predispose apparently healthy and/ or carrier fish to clinical infection, supporting the reports of **Bullock and Snieszko (1975)** who regarding the reflection of poor water quality as a prime predisposing cause on the occurrence of fish yersiniosis . However, the expression of the disease condition may be correlated with the degree to which fish is stressed. Additional striking management factor in the present study was the continuation of farming operations without periods of dryness and disinfection of ponds as essential regular farm activities among production cycles. Furthermore, in spite of the capability of Y. ruckeri to survive in freshwater for at least 4 months **(Thorsen et al. 1992),** the formal usage of fertilization processes routinely carried out in earthen ponds of the examined fish farm may synergistically extend the survival of Y.ruckeri in ponds'sediments **(Romalde et al.1994)** leading to the spontaneous existence, dissemination and transmission of the pathogen.

Regarding experimental infection, data in Table 4 showed that cumulative morbidity and mortality percents reached 60%, 100% and 25%, 55% in fish groups I/P infected with Y.ruckeri at 22 ± 1°C and at 30 ± 1°C respectively . However, the percents of morbidity and mortality in fish groups kept in contact with Y.ruckeri at 22 ± 1°C and at 30 ± 1°C reached 35%, 95% and 10%,

40% respectively. The cumulative morbidity and mortality percents were higher in I/P infected fish groups as a result of crossing most body barriers by such route of infection **(Roberts, 2001)**. These results confirmed those recorded by **Bullock et al. (1971)** and reflected the low virulence profile of the inoculated Y.ruckeri strain however, the mechanisms responsible for virulence are unknown **(Noga 2000)**.

Clinically, most moribund and died fish at 30 ± 1°C showed sluggish movement , dark skin colouration, congestion of the head region and the bases of the fins (Fig. 4 & 5) . Internally, there were congestion of visceral organs, slight enlargement of the liver and spleen and enteritis especially in I/P infected fish group. On the other hand, cases of subclinical and asymptomatic infection were frequently encountered at 22 ± 1°C rather than at 30 ± 1°C, confirming the direct influence and the critical stressful role played by water temperature in the expression of disease course of Y.ruckeri infection and supporting the findings of **Ross et al. (1966)** who reported that conditions predisposing fish populations to clinical infection with Y.ruckeri are related primarily to stress. The expressed clinical signs and lesions subsided from the majority of experimentally infected fish at 22 ± 1°C by the end of the third week post infection with the dominance of subclinical and asymptomatic types of infection . Meanwhile, at 30 ± 1°C signs and lesions became progressively milder by the end of experimental period with some cases of subclinical and asymptomatic infection. However, Y. ruckeri was successfully recovered from internal organs (Liver, spleen, kidneys and intestine) of the majority of clinical, subclinical and asymptomatic infected fish both at 22 ± 1°C and at 30 ± 1°C through out the experimental period, suggesting the development of carrier states among infected fish groups and confirming our results recorded in natural infection. In this regard, **Stevenson et al. (1993)** reported the occurrence of about monthly shedding of Y.ruckeri from some experimentally infected fish populations depending upon environmental conditions. However, **Woo and Bruno (1999)** reported that where isolations of Y.ruckeri have been made from fish without the presence of clinical signs, it is probable that such fish would succumb to infection if sufficient husbandry stress were applied.

Histopathological examination of clinically infected fish in natural and experimental conditions were nearly similar where inflammatory and degenerative responses occurred in virtually all body tissues especially abdominal musculature , liver, spleen (Fig. 6), kidneys (Fig. 7) and intestines with the presence of necrotic foci heavily infiltrated with leucocytes in hematopoietic tissues . On the other hand, subclinical infected fish and asymptomatic carriers showed milder histological lesions (Fig. 8) or even none in most examined cases. These results confirmed those recorded by **Noga (2000) and Roberts (2001)**.

The possibility of using antibiotics in controlling fish yersiniosis is a matter of concern re-

garding the documented potential environmental and human health hazards as well as the possible development and spread of antibiotic resistant strains **(Kerry et.al., 1995 and Capone et.al., 1996)**. However, prevention of yersiniosis could be achieved by avoiding those husbandry conditions which promote infection, reduction of handling and treatment of water **(Lilt-ved et al., 1995)**. On the other hand, failure to remove the husbandry conditions which influence the occurrence and severity of the disease results in regular recurrences requiring further antibiotic treatments **(Hunter et al., 1980)**.

In conclusion, recurrences of the disease condition correlated with improper management represented in poor water quality and continuous farming operations. Thus, maintenance of good husbandry practices and proper and efficient treatment of disease condition are essential toward the efficient prevention and control means of fish yersiniosis.

Table 1: Growth and Biochemical characteristics of recovered *Y.ruckeri* isolates.

Character	Reaction
Colonial morphology on TSA media	Yellowish white, round
Haemolysis on 10% sheep blood agar	-
Growth on TSA + 2 % NaCl	+
Gram Stain	-
Motility	+
β-galactosidase (ONPG)	+
Arginine dihydrolase (ADH)	-
Lysine decarboxylase (LDC)	-
Ornithine decarboxylase (ODC)	-
Tryptophane deaminase (TDA)	-
Gelatinase (GEL)	+
Cytochrome oxidase (OX)	-
Urease (URE)	-
Hydrogen sulphide (H2S)	-
Indole (IND)	-
Acetoin (VP)	+
Citrate (CIT)	-
Glucose (GLU)	+
Mannitol (MAN)	-
Inositol (INO)	-
Sorbitol (SOR)	-
Rhamnose (RHA)	-
Sucrose (SAC)	-
Melibiose (MEL)	-
Amygdalin (AMY)	+
Arabinose (ARA)	-

+: Positive
- : Negative
TSA: Tryptic Soy Agar

Table (2): Prevalence of recovery of *Y.ruckeri* from examined fish and pond water samples

Sample / Nature of infection	*O.niloticus* Fish			Pond Water		
	No. of samples	positive samples	%	No. of samples	positive samples	%
Clinically diseased	25	22	88	5	5	100
Asymptomatic freshly dead	25	19	76	5	4	80
Apparently Healthy	25	14	56	5	2	40

Table (3): Quality of affected pond water parameters

Water Quality Parameter	Range *
Temperatue (°C)	33.6 – 35.2
pH	6.9 - 7.1
Dissolved Oxygen (ppm)	1.5 – 2.6
Ammonia-NH_3 (ppm)	0.24 - 0.29
Nitrite-NO_2 (ppm)	0.13 - 0.22

*** Range of 20 collected samples**

Table (4): Morbidity and mortality rates of *O.niloticus* experimentally infected with *Y.ruckeri*

Fish group	Number of fish	Route of infection	Water temperature	Dose of infection	Morbidity		Mortality	
					Number	Percent	Number	Percent
1	20	I/P	22 ± 1°C	0.2 ml. B.S. /fish	12	60	5	25
2	20	immersion	22 ± 1°C	2 L B.S.	7	35	2	10
3	20	Control	22 ± 1°C	PBS treated	0	0	0	0
4	20	I/P	30 ± 1°C	0.2 ml. B.S. /fish	20	100	11	55
5	20	immersion	30 ± 1°C	2 L B.S.	19	95	8	40
6	20	Control	30 ± 1°C	PBS treated	0	0	0	0

B.S.: Bacterial Suspension (1.3×10^7 CFUs ml^{-1})

Fig. (1) : Naturally infected O.niloticus with Y.ruckeri showing severely congested mouth and haemorrhages on the operculum.

Fig. (2) : Naturally infected O.niloticus with Y.ruckeri showing inflamed mouth opening and deep seated haemorrhages in the head area .

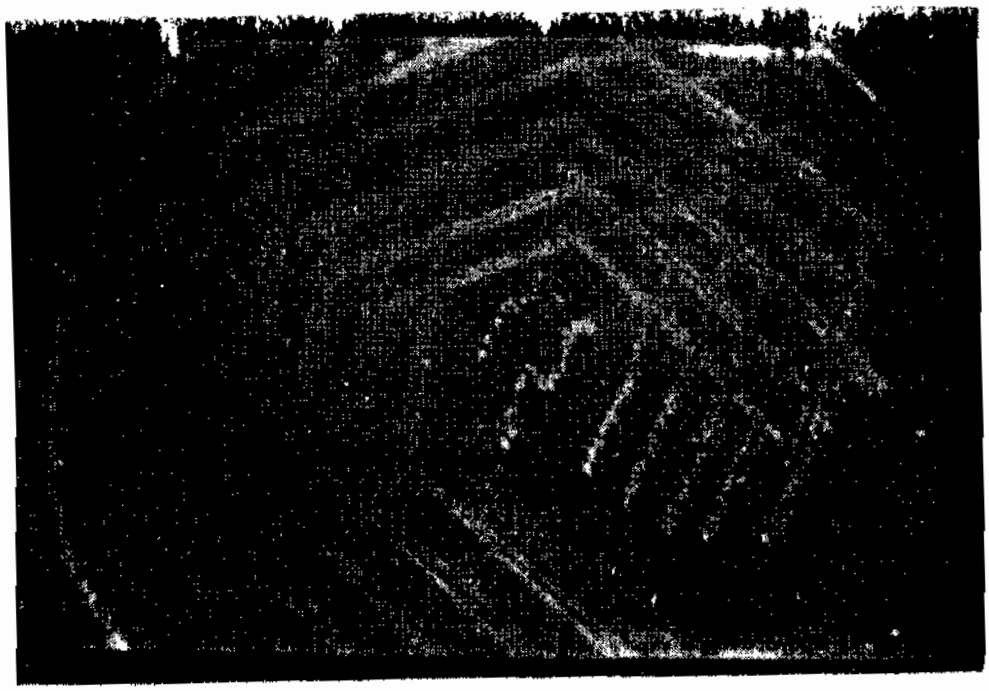

Fig. (3) : Culture of *Y.ruckeri* on TSA after 48 h incubation showing yellowish white round colonies.

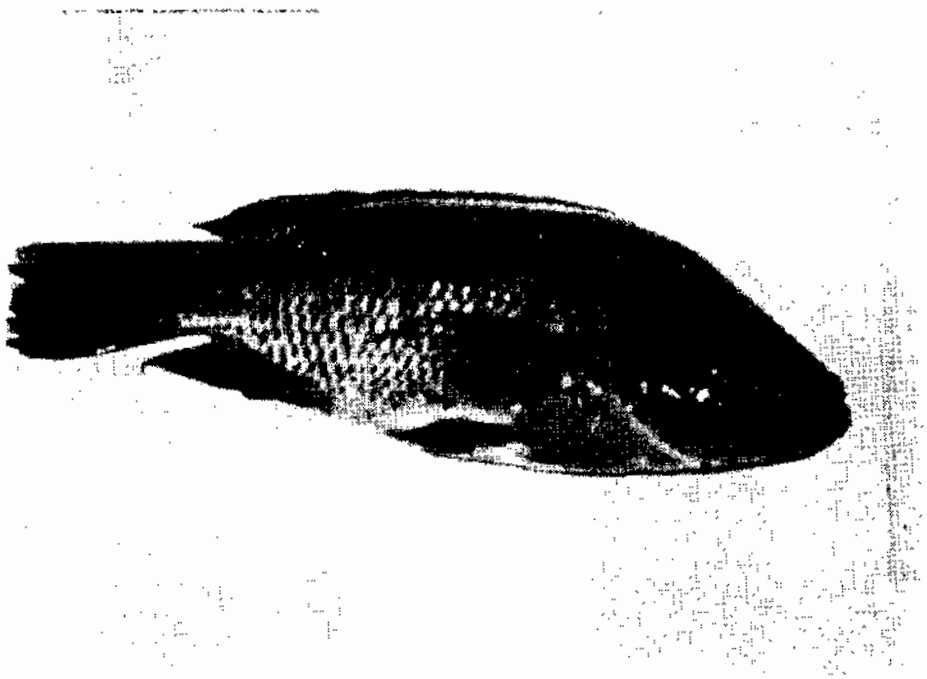

Fig. (4) : Intraperitoneally infected Cyprinoids with *Y.ruckeri* at 30 ± 1°C showing dark skin coloration as well as haemorrhages on the head , skin and pectoral fin .

Fig. (5) : Experimentally infected O.niloticus by immersion with Y.ruckeri at 30 ± 1°C showing congestion of the mouth opening.

Fig. (6) : Spleen of naturally infected O.niloticus with Y.ruckeri showing depletion of lymphocytes and focal areas of haemosidrosis . H & E (X 100) .

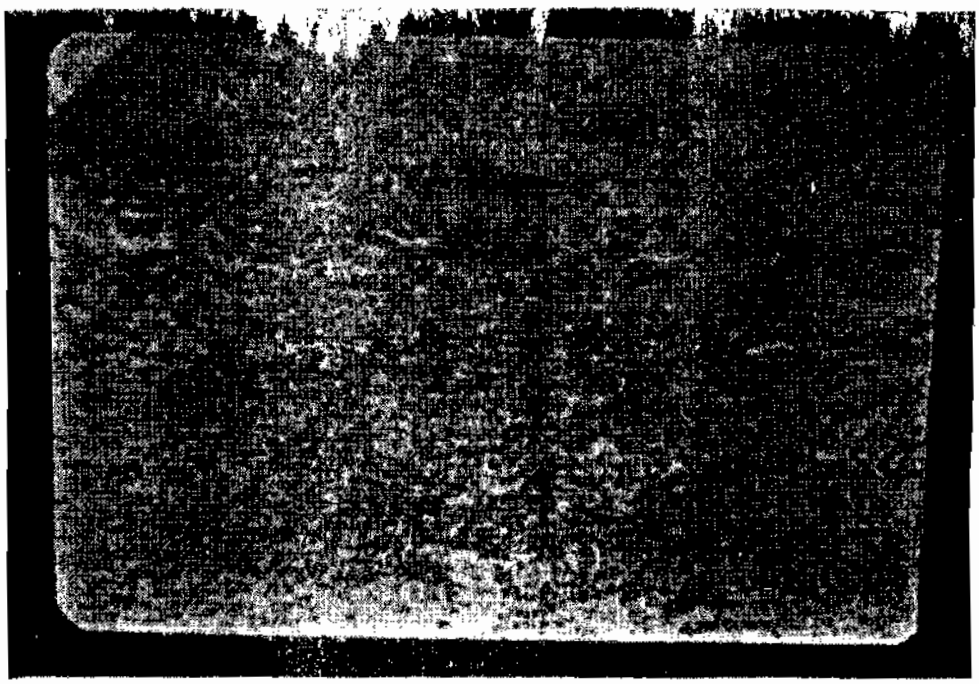

Fig. (7): Kidney of intraperitoneal infected O.niloticus with Y.ruckeri at 30 ± 1°C showing amyloid infiltration. H & E (X 100).

Fig. (8) : Intestine of natural asymptomatic infected O.niloticus with Y.ruckeri showing edema.H & E (X 100).

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الملخص العربى

إرتباط الإصابات المتكررة بمرض الفم الأحمر فى أسماك البلطى المستزرعة بالرعاية الغير جيدة

المشتركون فى البحث

عصام سعد الدين عبدالزيز

قسم بحوث أمراض الأسماك - معهد بحوث صحة الحيوان - الدقى - الجيزة - مصر

تم دراسة مرض اليرسينيوزيس ومسببه بكيتيريا يرسينيا روكيرى فى إحدى مزارع أسماك البلطى فى مصر، وقد تكررت الحالة المرضية فى فصل الصيف وارتبطت بالرعاية غير الجيدة للأسماك والتى تمثلت فى سوء جودة مياه الاستزراع مع التشغيل المستمر للدورات الإنتاجية.

وقد تم تسجيل الصور الإكلينيكية وغير الإكلينيكية للمرض فى الأسماك المصابة طبيعياً وتجريبياً، وقد تمثلت الأعراض الإكلينيكية الرئيسية فى إحتقان منطقة الرأى وقواعد الزعانف والأحشاء الداخلية، طبيعياً، بلغت النسب المئوية لعزل يرسينيا روكيرى من الأسماك المصابة إكلينيكياً وغير إكلينيكياً والسليمة ظاهرياً وأيضاً من عينات مياه أحواض تلك الأسماك ٨٨٪، ٧٦٪، ٥٦٪، ١٠٠٪، ٨٠٪، ٤٠٪ على التوالى.

تجريبياً، بلغت النسب المئوية للأسماك المريضة والنافقة المحقونة فى التجويف البروبتونى ببكتيريا يرسينيا روكيرى عند درجة حرارة ٢٢±١°م، ٣٠±١°م ٦٠٪، ١٠٠٪ و٢٥٪، ٥٥٪ على التوالى، بينما بلغت تلك النسب ٣٥٪، ٩٥٪ و١٠٪، ٤٠٪ فى مجموعات الأسماك التى تعرضت لبكتيريا يرسينيا روكيرى فى الماء عند نفس درجات الحرارة على التوالى.

وقد تمثلت طبيعة التغيرات الهستوباثولوجية لأنسجة الأسماك المريضة أساساً فى الالتهابات والتحلل.