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# EPIZOTIOLOGICAL STUDIES ON PROLIFERATIVE KIDNEY DISEASE IN TILAPIA (*OREOCHROMIS NILOTICUS*) AND AFRICAN CATFISH (*CLARIAS GARIEPINUS*)

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**A B S T R A C T**

The proliferative kidney disease (PKD) has been documented to cause particular economic loss in fish farms worldwide. The present investigation aims to determine the prevalence and the etiology of (PKD) in *Oreochromis niloticus* and *Clarias gariepinus* obtained from El-Riah El-Tawfiki and its tributaries. To achieve this goal, 500 fishes were used of which 266 were *O. niloticus* and 234 were *C. gariepinus*. The fishes were dissected and the kidneys were examined macroscopically for the presence of morphological abnormalities and nodules. Fresh and Giemsa-stained slides of kidney tissue were also examined for determination of the causative agent. The results showed the prevalence of PKD in *O. niloticus* was higher in autumn (95.08%) and spring (91.94%) than in summer (76 %) and winter (73.53%). The prevalence of PKD in *C. gariepinus* was high in winter season (76%) and low in spring season (36.84 %). The overall prevalence of PKD was higher in *O. niloticus* (83.46%) than in *C. gariepinus* (46.58%). The Clinical signs of fish affected with PKD may be non-specific included distended abdomen with dark color, anaemia with pale gills and emaciation. Some fish have nodules in the eye around the iris forming a ring. Macroscopically, there was enlargement of the kidney with appearance of some kidney nodules of approximately 0.2 – 0.6 mm diameter. Based on the spore morphology, the causative agent was identified as different types of Myxosprean spores in *O. niloticus* and *C. gariepinus*. The high prevalence of PKD in both types of fishes suggests the need to establish strict control measure to overcome the great economic losses imposed by the disease.

**KEY WORDS**: *Clarias gariepinus, Myxosporean, Oreochromis niloticus,* PKD, Prevalence.

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## 1. INTRODUCTION

Proliferative kidney disease (PKD) is a serious disease principally affecting populations of farmed and wild salmonid fish [16]. The disease is characterized by a severe swelling of the kidney induced by the host immune response to the presence of extrasporogonic stages of a Myxozoan parasite [8, 18, 26]. PKD is an example of devastating disease which is a result of infection by a Myxozoan parasite. Although the organism causing PKD was recognized for many decades it was only determined to be a Myxozoan in the 1980s [17]. The recent discovery based on molecular evidence that freshwater Bryozoans are hosts for the causative agents of PKD [4]. The parasite was described as *Tetracapsuloid bryosalmonae* and finally identified the long sought source of PKD which is not transmitted from fish to fish [7]. PKD has been documented to cause particular economic loss to rainbow trout fish farms in the UK and Europe and to salmon hatcheries in North America [16]. PKD is also a disease of wild and feral fishes [13, 22, 32] and appears to be on the increases possible as a sequence of increasing temperatures [11, 32]. In Egypt, proliferative kidney disease was observed to be epidemic among Tilapian fishes and known as Tilapian proliferative kidney disease (TPKD) [10]. The disease is characterized by a severe swelling of the kidney induced by the host immune response to the presence of extrasporogonic stages of a Myxozoan parasite [8, 18, 26]. The disease is manifested as a massive immune response to the presence of *Tetracapsuloid Bryosalmonae* but the temperature dependence of the fish's immune systems means that the disease does not develop until temperatures exceed 15 °C although fishes may be infected at lower temperatures [15]. Because of the serious and the apparently growing impact of PKD in farmed and wild fishes, this study was conducted to investigate the prevalence of PKD in *Oreachromus niloticus* and *Clarias gariepinus*. The clinical and macroscopical findings were recorded. The morphology of disease-causing spores was presented.

## 2. MATERIAL AND METHODS

2.1. *Fish*

In this study 500 fish were investigated, of which 266 *Oreochromis niloticus* with average weight of 120±10 g and 234 *Clarias gariepinus* with average weight of 200±15g. The fishes were collected from El-Riah El-Tawfiki and its tributaries during the period from March 2010 to August 2011. The fishes were transmitted alive in large plastic containers with sufficient quantity of water obtained from where the fishes were collected. The freshly dead fishes were labeled and packed in clean plastic bags and kept in an ice boxer. The collected fishes were transported as rapid as possible to the laboratory of department of fish diseases and management, faculty of veterinary medicine, Benha University where clinical and parasitological examination were conducted. Freshly dead fishes were subjected to immediate examination.

2.2. *Aquaria*

Clean glass aquaria measured 100×30× 50 cm were used for holding the fishes during the examination. The Aquaria were supplied with sufficient aeration by using electric air pump. The fishes were fed on pelleted commercial ration containing 25 % crude protein.

2.3. *Clinical examination*:

The fishes were examined externally for any abnormalities as previously described by Noga [24].

2.4. *Postmortem examination*

Dissection of fish and post-mortem examination for any internal abnormalities of the body cavity, kidneys, and urinary bladders were done according to Noga [24]. Particular attention was given to examination of kidney for any change in size, colour or presence of nodules or cysts.

2.5. *Microscopic examinations of kidney preparations*

Specimens from examined kidneys and nodules if present with few drops of saline were squashed and examined under microscope. In addition, air-dried smears fixed with methanol and stained with Giemsa used for further examinations [21]**.** Images were captured using Sony digital camera (SDC-p92, Sony Corporation).

2.6. *Statistical analysis*

The prevalence of PKD in both *O. niloticus* and *C. gariepinus* in different seasons was determined as the percentage of the affected fish from the total population examined using excel program.

## 3. RESULTS

3.1. *The Prevalence of PKD*

The prevalence of PKD in *O. niloticus* was higher in autumn (95.08%) and spring (91.94%) and lower at winter season

(73.53%). Moderately low infection was described in summer season (76 %). On the other hand, the prevalence of PKD infection in *C. gariepinus* was high in winter season (76%) and low in spring (36.84 %), summer (38.71 %) and autumn (41.30 %) seasons. The overall prevalence of PKD in *O. niloticus* (83.46%) was higher than the *C. gariepinus* (46.58%) (Table1, Fig.1).

Table 1 Seasonal prevalence of PKD in *Oreochromis niloticus* and *Clarias gariepinus*

| Season | *Oreochromis niloticus* | | | *Clarias gariepinus* | | |
|---|---|---|---|---|---|---|
| | Examined | Diseased | Incidence (%) | Examined | Diseased | Incidence (%) |
| Spring | 62 | 57 | 91.94 | 76 | 28 | 36.84 |
| Summer | 75 | 57 | 76.00 | 62 | 24 | 38.71 |
| Autumn | 61 | 58 | 95.08 | 46 | 19 | 41.30 |
| Winter | 68 | 50 | 73.53 | 50 | 38 | 76.00 |
| Totals | 266 | 222 | 83.46 | 234 | 109 | 46.58 |

### 3.2. *Clinical signs of PKD*

The clinical signs may be absent or unspecific. Some affected fishes showed distended abdomen with dark color (Fig. 2), exophthalmos, appeared anaemic with pale gills and emaciation. Some fishes have nodules in the eye around the iris forming a ring (Fig. 3) and some fish appeared to be normal with no pathognomonic external lesions or signs. However, microscopical and histopathological examination of the kidneys of these apparently normal fish revealed various Myxosporeans.

### 3.3. *Macroscopical examination*

All the affected fishes showed enlargement of the kidneys (Fig. 4) and spleen (Fig. 5) and ascites but the pathognomonic PM lesion was the enlargement of the posterior kidney that may be protruded ventrally toward the abdominal cavity (Fig. 6) and sometimes distended urinary bladder. Also, we found macroscopic yellowish white nodules (2-3 mm or more in diameter) mostly appeared at the underlying side of the kidney in *O. niloticus* where at the surface of kidney in *C. gariepinus* (Fig. 7 and 8). When these nodules were examined it found to contain myxosporean spores.

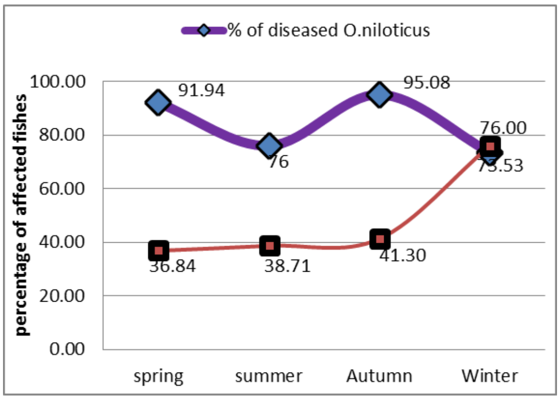


Fig.1. Seasonal prevalence of PKD infection in *oreochromus niloticus* and *Clarias gariepinus*.

Fig.2. Distended abdomen with dark color in *Oreochromis niloticus* affected with PKD

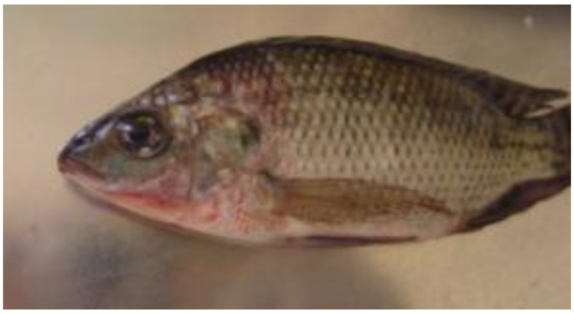

Fig. 3 A tilapia fish with PKD have nodules in the eye around the iris forming a ring.

Fig. 4. Enlargement of the kidney in *Clarias gariepinus* affected with PKD

Fig. 5. Enlargement of kidneys and spleen in Oreochromis *niloticus* affected with PKD.

Fig. 6. Enlargement of the kidney that is protruded ventrally toward the abdominal cavity in *Oreochromis niloticus* affected with PKD

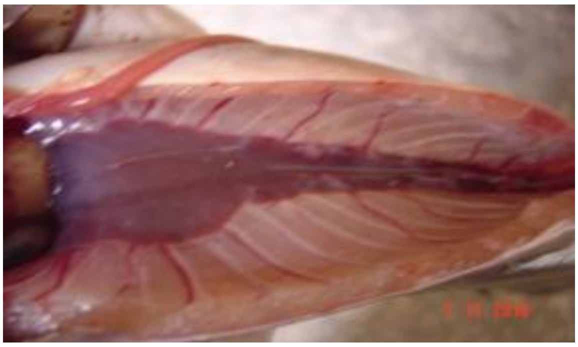

Fig. 7. Enlargement of the kidney with macroscopic yellowish white nodules of different sizes in *Clarias gariepinus* affected with PKD.

### 3.4. *Microscopical examination*

Different types of myxosporean spores were identified by microscopical examination of the fresh preparations of affected kidneys. The infection demonstrated as mixed types of spores and almost no single infection within the fish observed (Fig. 9). Microscopic nodules were also observed in some affected cases. These nodules were either surrounded by intact capsule or the capsule was ruptured with release of the spores (Fig. 10)

Fig. 8. Macroscopic white nodules of different sizes in the kidney of *Clarias gariepinus* affected with PKD.

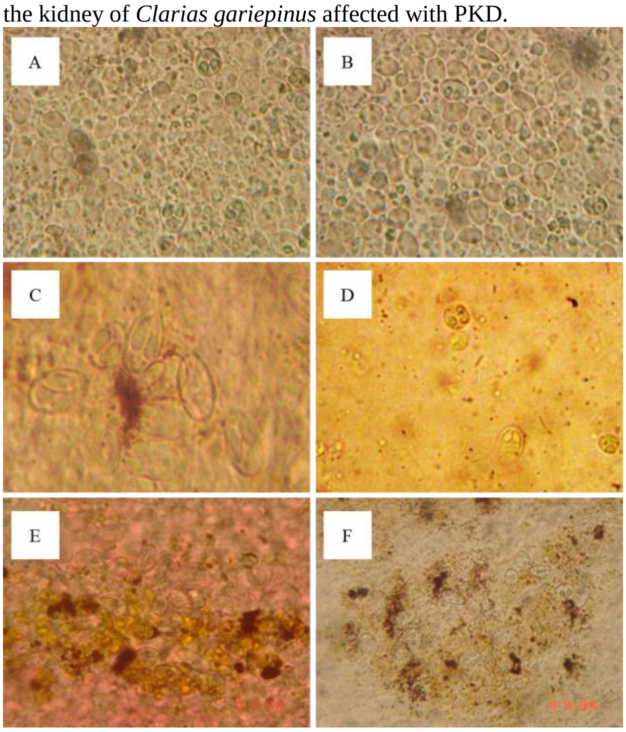


Fig.9. Fresh preparations of kidneys affected with PKD examined by microscopic examination showing different Myxosporean spores (A-F). All preparation showed mixed types of spores within the same slide.

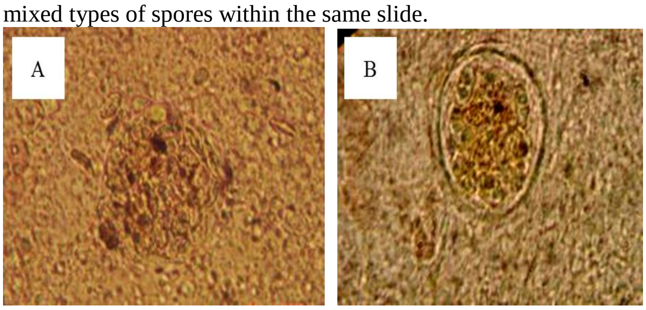


Fig. 10. Fresh preparations of kidneys affected with PKD showing microscopic nodules.

Different types of spores of myxosporea were identified in Giemsa-stained preparation as shown in Fig. 11. The spores have different sizes and morphology. However, in all stained preparation, the polar bodies are densely stained than the sporoplasm.

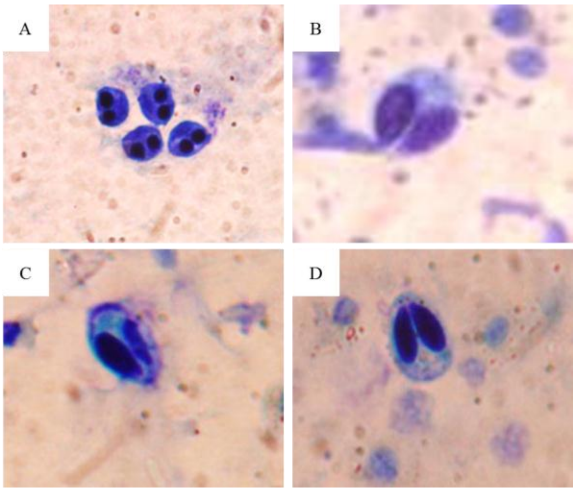


Fig. 11. Stained kidney tissue preparation (A-D) showing different myxosporean spores of varying sizes and morphology. Notice that the polar bodies are densely stained than the sporoplasm.

## 4. DISCUSSION

This study demonstrated that the overall prevalence of PKD in *O. niloticus* was 83.64%. This result coincided with that reported by Eissa *et al*. [9] who reported that heavy infection with Myxosoma spp. was recorded among cultured *O. niloticus* from Sharkiya governorate with prevalence infection exceeded 80 % of all examined fish. The high prevalence (more than 80 %) of Myxosporean infection among the examined fish highly suggests that such infection is endemic in the ponds used for rearing of these fish. This result is also comparable to that observed by Feist and Longshaw [12] who recorded that PKD is highly problematic for fish farms and hatcheries where up to 100% of stock can be infected. In consistent with this result, Okamura *et al*. [25] observed that PKD prevalence was variable range from 0 to 90-100%. On the other hand, Shehab El-Din [28] demonstrated a lower rate of infection (10.56%) in tilapian fish. The prevalence of PKD in *O. niloticus* was higher in autumn ((95.08%) and spring (91.94%) and lower at winter season (73.53%). These results coincided with those reported by Hedrick *et al*. [16]. They reported that the PKD is often seasonally dependent occurring at water temperature above 15 ˚C in the fall months of the year. These results were also confirmed by higher prevalence during the month of October and November which are considered as the fall months in Egypt. Gay *et al*. [15] demonstrated that the disease does not develop until temperature exceeds 15 °C. Therefore, the disease prevalence will be reduced during the winter season, a result that coincided with the result of this study where the lowest prevalence of the disease was observed in winter season. The occurrence of PKD is associated with seasonal increase in water temperature above 15 °C [8]. In addition, the seasonal elevation of water temperature causes reduction of water dissolved oxygen and increases the susceptibility to the disease. The results of this study disagree with that reported by Tops *et al.* [30] and Tops and Okamura [31] who mentioned that the peak prevalence of PKD in rainbow trout *Oncorhynchus mykiss* in the fish farm occurred in summer (up to 100%). This can be explained by the difference in temperature between Europe and Egypt where the summer temperature in Europe is significantly lower than that in Egypt.

In this study, a lower prevalence of PKD was observed in the summer season (76 %) compared to autumn (95.08%) and spring (91.64%). A more recent study in rainbow trout *Oncorhynchus mykiss* demonstrated that at 18°C, fish affected with PKD showed a gradual recovery of normal kidney morphology which was associated with a decline in parasite numbers and infection prevalence than fish kept at 12°C [27]. This implies that the increase of water temperature at 18°C or above has a negative effect on the disease prevalence. The common consistent finding with previous studies was that the prevalence increase in warmer temperature regions [29] as the temperature promotes disease development, enhances bryozoan biomass and increases spore production [25]. The discrepancies in the prevalence rate between this study and other studies conducted in United Kingdom demonstrated by Tops et al. [30] suggested that other factors rather than water temperature could have an effect on the prevalence of the PKD disease. This is particularly true since some previous studies on PKD demonstrated the existence of strong correlation between organic pollution of water, the presence of bryozoa and the outbreak of PKD [11]. In addition, Wahli *et al*. [32] reported that water contaminants can influence the PKD via development of the parasite in the bryozoan host or in its fish host. Therefore, the factors affecting prevalence of PKD in *O. niloticus* in Egypt may require further investigations and studies.

The prevalence of PKD infection in *C. gariepinus* was high in winter season (76%) and low in spring (36.84 %), summer (38.71 %) and autumn (41.30 %) seasons. The overall prevalence of PKD in *C. gariepinus* was 46.6%. These findings partially agreed with those reported by Abbass *et al*. [1] who found that the prevalence of Myxozoan infestation in *C. gariepinus* was 42.5% and the maximum prevalence was observed in the winter (70%) and spring (80 %) seasons. The results also agreed with Abdel-latif [2] who demonstrated that the highest prevalence of parasitic gill infestation by Myxosporeans in *C. gariepinus* occurred in winter season (22%). This was relevant to the study of Feist *et al*. [13] who verified that *Tetracapsuloides bryosalmonae* used skin and gills for entry then it disseminate to the kidneys through the lymphatic tissues or blood.

Compared to the prevalence of PKD in *O. niloticus*, the prevalence in *C. gariepinus* was lower in all seasons. The general prevalence of PKD in *C. gariepinus* (46.6 %) was lower than *O. niloticus* (83.5 %). The exact cause for this observation is not well-understood. It is well-known that the PKD infection rate is dependent on the host immune system [15]. Also, previous studies revealed that the total leucocytic count is higher in *C. gariepinus* (24.05±0.050) [3] than in *O. niloticus* (15.0±4.5) [23]. This could partially explain the lower prevalence of the PKD in *C. gariepinus* than *O. niloticus*. This observation may require further investigations.

Regarding the clinical signs, some affected fishes showed distended abdomen with dark color, exophthalmos, appeared anaemic with pale gills and emaciation. These findings agreed with those reported by others [10, 11, 19 25]. The anaemia observed with PKD could be attributed to the loss of haemopoeitic tissues in the kidney and spleen caused by the Myxozoan parasites. Fernandez-de-Luco et al. [14] reported that abdominal distension and anaemia of rainbow trout affected with PKD were the most obvious clinical signs. Internally, all the affected fishes showed enlargement of the kidneys and spleen and may be ascites but the pathognomonic PM lesion was the enlargement of the posterior kidney that may be protruded ventrally toward the abdominal cavity. These internal findings were in line with those reported by others [9, 18, 12, 26, 29]. In the current study no specific clinical signs were observed in most of the fish although the microscopical, the histopathological and PCR results demonstrated the occurrence of the disease. This was relevant to the results recorded by Okamura *et al*. [25] who demonstrated that the identification of the disease may be hindered by absence of clinical signs and hence there was a great possibility for misdiagnosis. Similar observations were reported by Buck *et al*. [5] as they were No gross changes observed in fishes affected with PKD, however histological examination revealed various Myxosporean stages in renal tubules.

Microscopically, different types of mature Myxosporean spores were identified by examination of the fresh preparations of affected kidneys. Similar results were recorded by El-Mansy and Abdel-Ghaffar [10]. They identified seventeen Myxosporean species belong to the genera Myxobolus, Tiangula and Chloromyxum in Tilapian fishes from the River Nile at El-Rahawy drain. The presence of different Myxosporean spores together in the kidney of almost all examined Tilapian fishes could be attributed to two reasons, first that mature spores might come from their specific organs to the kidney via blood and in this case the kidney infection could be used as a diagnostic evidence for the presence of Myxosporean parasites and the other reason that some spores may originate as a final stage of the development of PKD cells within the kidney tissues because mature spores were already found together with these stages surrounded with cyst like structures [10]. From this study, it was concluded that PKD occurs with high prevalence in Egypt in both *O. niloticus* and *C. gariepinus.* The prevalence was usually higher in *O. niloticus* than *C. gariepinus*. This may suggest the urgent need to place a strict control measure because of the great economic losses imposed by the disease since these kinds of fishes represent main source for protein most of Egyptian population.

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مجلة بنها للعلوم الطبية البيطرية
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# دراسات وبائية عن مرض الكلى التكاثرى فى أسماك البلطى النيلى وأسماك القط الإفريقى

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## الملخص العربى

مرض الكلى التكاثرى من الامراض التى تسبب خسائر اقتصادية كبيرة في المزارع السمكية في جميع أنحاء العالم. وتهدف هذه الدراسة لتحديد معدل الإصابة ومسببات أمراض الكلى التكاثري (PKD) في البلطي النيلي واسماك القط الإفريقى التى تم الحصول عليها من الرياح التوفيقى وروافده. لتحقيق هذا الهدف، تم تجميع عدد 500 من الأسماك منها 266 من البلطي و 234 من اسماك القط الإفريقى. وتم تشريح الأسماك و فحص الكلى ظاهريا وملاحظة أى تغيرات شكلية او عقد مرضيه .تم الفحص المباشر للعينات الكلوية تحت الميكروسكوب وايضا تم الفحص بعد صباغة الشرائح بصبغة جيمسا. أظهرت النتائج حدوث المرض في البلطي النيلي وكانت أعلى معدلات الاصابة في فصل الخريف (95.08%) والربيع (91.94 %) واقل فى فصل الصيف (76.00%) والشتاء (73.53%). وكانت نسبة حدوث وقوع المرض في أسماك القط عالية في فصل الشتاء (76.00%) ومنخفض في فصل الربيع 36.84% .كان المعدل العام للمرض أعلى في البلطي النيلي 83.46 % عن أسماك القط 46.58%. وكانت معظم العلامات المرضية غير محدد وهى عبارة عن بطن منتفخ مع اللون الداكن، وفقر الدم وشحابة الخياشيم مع الهزال .بعض الأسماك لديها عقد في العين حول القزحية تظهر وكانها حلقة، كان هناك تضخم في الكلى مع ظهور بعض العقد الكلوية من 0.2– 0.6 مم .بناء على اشكل الظاهرى تم التعرف على العامل المسبب للمرض مثل أنواع مختلفة من ميكسوسبورين في البلطي النيلي واسماك القط الإفريقى. خلصت النتائج الى ضرورة وضع نظام محكم للسيطرة على مرض الكلى التكاثرى فى هذه الانواع من الاسماك نظرا لانها تمثل مصدر بروتينى هام لمعظم طوائف الشعب المصرى.

**(مجلة بنها للعلوم الطبية البيطرية: عدد 23 (1)، يونيو 2012: 150–158 )**