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Necrotic Enteritis by *Clostridium perfringens* in Ostrich (Struthio Camelus)

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Abstract: After the report of sudden death in one of the ostrich farms of Tabriz, the carcasses were necropsied. Symptoms that were seen include petechia and ecchymosis hemorrhagic in the end area of duodenum and the outset of jujenom along with gaseous gangren. For perfect study and certain diagnosis, sampling of the liver and intestine was done and *Clostridium Perfrigenes* was confirmed. Histological examination revealed multifocal necrosis of hepatocytes with infiltration of hetrophils and also apoptosis. Extensive superficial necrosis associated with fibrin and serocellular deposits was shown in intestine. Generally, sudden changes in diet, stress and nutrition from soil and sands that cause sudden death in young ostriches because of their high sensitivity. Application of effective antibiotics, adjustment and change of diet can be effective in controlling of this disease.

Key words: Ostrich · Clostridium Perfrigenes · Tabriz

INTRODUCTION

Gastrointestinal diseases are considered one of the most frequent and economically important diseases in ostrich farms. Predisposition factors such as poor management conditions, stress and other concomitant diseases contribute to the development of these lesions, which are primarily caused by bacterial infections. *Escherchia Coli, Campylobacter Jejuni, Pseudomonas Aeruginosa, Salmonella* and *Clostridium* are the pathogen bacteria most frequently involved in infectious enteritis in ostriches [1].

Neonatal mortality is a major problem in raising ostriches [2, 3]. The majority of mortalities is in ostriches less than three months of age. In one study more than 80% of the ostriches submitted to a veterinary diagnostic laboratory for disease diagnosis were less than 12 weeks of age [2]. The causes are variable but majority of the diseases and disorders have been attributed to poor management of the birds, predisposing them to variety of infectious agents [2, 4]. Common conditions and diseases include edematous chicks, yolk sac infection, yolk sac retention, diarrhea, musculoskeletal problems, impactions, congenital disorders, cloacae prolapsed and respiratory signs [2, 5]. *Clostridial Enteritis* is a common disorder of ratites of all ages [5]. Enteritis due to *Clostridium Perfringens* and *Clostridium Difficile* are common findings in neonatal ostrich chicks submitted to the laboratory for disease diagnosis.

Case Report: In one the ostrich farms around Tabriz with 140 birds (5 month up to reproductive) in different pens during February of 2007 - May of 2007 sudden losses of 5-9 month an ostrich was reported. It was distinguished that 3 of these losses were died because of sudden change in diet with high moisture a barley and after the putting 3-9 month ostriches in new pen and big sands and soil existed in litter. Death was happened suddenly in 24-48 hour. Abdomens were distending and all of them were necropsied. In necropsy, intestine was distending with petechia and ecchymosis which intensity of losses was great in 1/3 distal of duodenum and outset of jujenom. Mucosa of intestine in some parts of duodenum was stricken to necrosis and easily was peeled but other parts of mucosa have much change from the microscopic view. Liver was distending and its lobules front parts had shown bleeding (Figure 1). Gizzard was full of large amount of soil and big sands. Gizzard mucosa and its cuticul layer could easily be peeled by hand.

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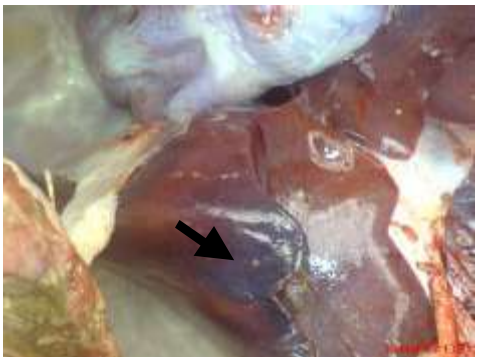

Fig. 1: Hemorrhage in front lobe of liver. Yellow necrotic foci in liver

For detection of agent, liver and intestine of mucous were cultured aerobically on 5% sheep blood agar and MacConkey agar plates (Remel, Lenexa, KS, USA). Culture for *Campylobacter* species was attempted on Campylobacter FDA media (Remel, Lenexa, KS, USA) under microaerophilic conditions. The plates were incubated at 37°C with 5% carbon dioxide. Caecal contents were culture for salmonella by enrichment in selenite broth (Remel, Lenexa, KS, USA), incubated at 37°C for 24 hours and streaked onto Brilliant green agar plates. The Brilliant green agar plates were incubated at 37°C for 24 hours and examined for any colonies of bacteria.

Samples were gotten from all dead ostriches in 1-1.5 hour, interval after death and reporting the farm manager was done and they were sent to laboratories. After culture in blood agar and observing opaque and hemolyse colonies in SIM was registered which is one of the manifest characteristics of *Colostridium Perfrigens* (other results of differential culture: negative endol, gelatin hydrolysis, lecittin hydrolysis in agaros of yolk, foul fermentation in tornosol milk and sugar fermentation in TSI).

Gross lesions were recorded. Tissue samples including liver and intestine were fixed in 10% buffered formalin and were embedded in paraffin blocks. Sections were cut at 4-7 µm, mounted on glass slides and were stained with haematoxylin and eosin. The sever hemorrhage in intestine mucosa was completely obvioused with necrosis and separation of epithelium that was affected the atrophy and downing the intestine villies (Figure 2). The sever hemorrhage was appeared in liver sinusoid with degenerative change. The fatty change liver and necrosis of hepatocytes with otolytic change and apoptosis (fragmentation of chromatin) were shown in liver (Figure 3).

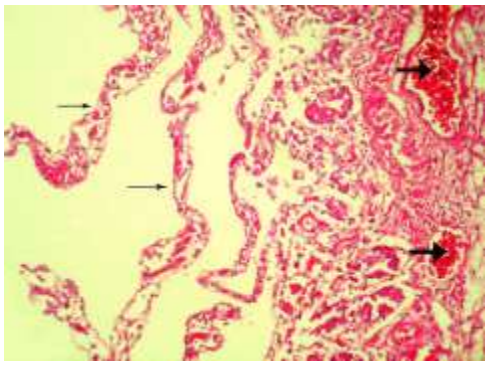

Fig. 2: Intensive hemorrhage in mucosa of intestine (thick arrow) along with necrosis and separation of superficial epithelium which caused atrophy and humility of intestine fuzz and their tenacity (thin arrow) (haematoxylin and eosin, 60 X)

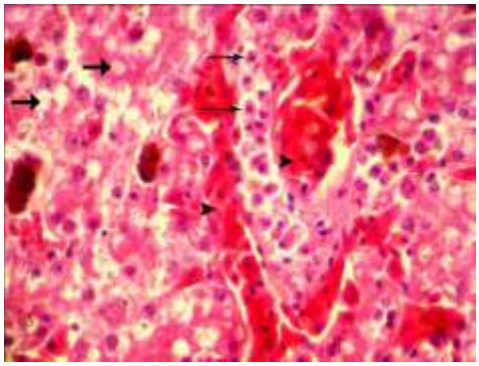

Fig. 3: Intensive hemorrhage (thick arrow) in liver sinozoeids along with degenerative changes in the form of aggregation of transparent vacuel (thick arrow) in cytoplasma of hepatocyte and alo necrosis hepatocyte along with otolytic changes in them and symbolic apoptosis changes in liver cells in the compact from and sectionaling of chromatin (thin arrow) and spreaded hemosiderosis. (haematoxylin and eosin, 400 X)

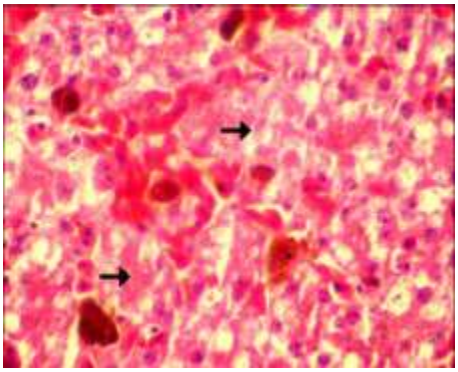

Fig. 4: sever necrosis of hepatocytes with otolytic changes and mucosa of sinosoids and spreaded hemosideosis. (haematoxylin and eosin, 400 X)

The hepatocytes sever necrosis was cleared integrally with otolytic changes (Figure 4).

In order to control and treatment of the diseases by observing the mortality and history of flock ie. sudden changes in diet (with grade 2 barley) and stress of handling and transportation of new ostriches to the farm and also according to necropsy finding, probable recognition was on necrotic enteritis and basitrasin was used 60 gram in 10 liter water during 7 consecutive day and the results were satisfactory.

RESULTS AND DISCUSSION

The intestinal lesions described in our animals comprised sever fibrino-necrotic in association with fibrinous peritonitits and multifocal serosal hemorrhages. Gastrointestinal diseases in ostrich have been noted to be complex and multifactorial in origin when virus, parasites and bacteria have been involved [1, 6, 7]. Only a small variety and low incidence of viruses have tropism for the digestive tract of ostrich, including *Coronavirus, Circovirus, Enterovirus* and the *Newcastle* disease virus, which is considered the most important and frequent virus [1, 8]. Enteric parasites in ostrich are also described to have low incidence and the susceptibility to coccidian, which frequently cause necrotic enteritis in other avian species, is still controversial in ostrich pathology [6, 7, 9]. Among pathogens bacteria, *Salmonella* and Clostridium are considered to be the most important species causing necrotic enteritis in ostriches [10].

Generally necrotic intestine inflation or entrotoxaemia with *Colostridium Perfrigens* is one of the prevalent diseases in young and adult ostriches. As young ostriches are sensitive to these diseases to the extent that sometimes in less than 24 hour sudden death occurs without any necropsy signs. It seems that some of the main occurrence reasons of these diseases are sudden changes in diet, incorrect nutrition, stress of transportation and access to materials that are unlike of nutriment like sand and soil especially in young ostriches. Prevention of this disease occurs in some countries by live vaccines of entrotoxaemia and in some breeding systems during the circuit drugs like basitrasian or tetracycline is used. But generally, prevention of stress related to sudden changes in diet can be useful in control of diseases. To treat via drinking water, one can use effective antibiotics like Ampicillin 1-2 gram in 10 liter), Basitrasian-Zinc (4 gram in 10 liter) or Tetracycline. Birds are unable to drink water because of intensity of injuries could be manually under treatment [11-15].

According to researches done by Voon and Lee in 2004, it was distinguished that young ostriches are stricken to this disease more than adults [13]. Generally this disease engenders because of environmental stress, disease or sudden change in diet and chicks are more endanger to these changes [13]. Involved ostriches show symptoms like squirt, jadish, slothing and sudden death. In autopsy signs intensive bleeding in jojenom, liver and a little ascites were shown [13]. Also according to Shivaprasid's researches in 2003 on involved ostriches, liver Hepatitis along with focal necrosis of liver especially in the surface of liver capsule was shown. In both of these studies Colostridum bacteria was segregated and even produced toxin was detected and confirmed by ELISA. According to our surveys about necrotic intestine inflation occurrence in other researches it was shown that susceptible factors are necessary for outbreak of this disease, which in our report sudden change of diet (by grade 2 barley that was mildewed to some extent) in first case and stress of handling and transportation in second case are known as the susceptible factors of this disease.

Colostridium Perfrigens is one of the bacterias that are inhabited in intestine and also in normal situation they exist in intestine and we can segregate them from healthy samples of intestine. About report of this disease by regard to mortality, necropsy finding and history of flock about sudden change of allotment and transportation of them, early diagnosis was on necrotic intestine inflation. In intestine samples except Colostridum Perfrigens *E. coli* was also segregated in some cases but regarding the autopsy signs, flock history and segregation of disease factor in large amount from liver samples (normally they should not exist in liver) occurrence of this disease is confirmed.

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