CLINICO PATHOLOGICAL CHANGES OF EXPERIMENTALLY INDUCED FOWL CHOLERA IN JINDING DUCKS

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ABSTRACT

Clinicopathological changes of duck cholera were studied in 16-week-old 10 susceptible (PHIA titer ≤ 8.00) finding trend ducks during the period from October 2002 to March 2003. Each of the experimental duck was inoculated with a virulent chicken isolate of Pasteurella multocida @ 5.4 × 10⁷ CFU/ml per bird intranasally. The incubation period varied from 12 to 42 hours with 100% mortality rate between 24 to 72 hours of infection. These affected ducks showed clinical signs of fever, anorexia, diarrhea, depression, ruffled feathers, severe weakness, drooling, which diarrhoea with mucus, exudation from eyes, lameness and unable to hold the head with head touching the ground before death. Necropsy examination of the dead ducks showed septicaemic changes, blood vascular congestion, haemorrhagic splenitis, increased amount of peritoneal and pericardial fluids, swollen and sometimes congested liver with multiple necrotic foci on the portal surface, milled and exudative heart with eutrophic haemorrhages. Trachea and lungs were severely congested and haemorrhagic and had distinct reddish appearance seen in the lung, liver and heart. The P. multocida organism was isolated from the swabs of liver and heart of all the dead ducks by standard bacteriological techniques. This study was confirmed the virulence and pathogenicity of chicken isolate of P. multocida in ducks.

Key words: Experimental infection, Duck cholera, pathogenicity, clinicopathological changes

INTRODUCTION

Duck cholera is one of the most important and devastating endemic avian disease in Bangladesh (Chowdhury et al., 1985; Baki et al., 1993). Israel and Quader, 1967 found isolated Pasteurella multocida from a case of Muscovy duck and subsequently Ali et al. (1974) isolated P. multocida from two ducks and described the disease condition as Duck cholera. Baki et al. (1993) reported high mortality rates of 54.55% in chickens and 10.91% in ducks, caused by P. multocida in Bangladesh. Although some reports on experimental studies on fowl cholera in chickens have been made (Kumal et al., 1988; Khat et al., 1997; Hossain et al., 1999) but similar reports are very limited in ducks in Bangladesh (Baki et al., 1991). This paper describes the pathogenicity and clinic-necropsy changes of experimentally induced in Jindung ducks with a virulent chicken isolate of P. multocida.

MATERIALS AND METHODS

Four-week-old 50 Jindung breed of ducks were purchased from the Government Poultry Farm, Kishorgonj, of which only 10 birds were randomly selected for experimental P. multocida of fowl cholera at the age of 16 weeks. These birds of both sex with no previous history of either vaccination or fowl cholera infection (specific pathogens free) were selected for this study. These ducks were maintained in the newly constructed duck house in the Department of Veterinary Medicine, Bangladesh Agricultural University (BAU), Mymensingh with intensive care and complete commercial feed (Quality Foods Ltd., Dhaka) and water supply throughout the experimental period. In addition to general feed supply, vitamin-mineral premix (Megaglo2, Novartis Bangladesh Ltd.) was also supplied in the drinking water daily.

Venous blood samples were collected from the wing vein of each of the ducks at the age of 16 weeks before infection and sera were separated as per standard conventional method and tested by the Passive haemagglutination assay (PHIA) to detect the level of maternal derived antibodies as described by Tripathy et al. (1970). The detected antibody titre ranged from 4.0 to 8.0 with an average of 5.6 ± 1.96 (SD) against the protective level of ≥ 2:64. A local virulent chicken isolate of P. multocida (PM-38), serotype-1 (X-73), preserved in laboratory at room temperature in mineral oil was used for experimental infection in this study. The virulence of the P. multocida isolate was reverted by serial culture on blood agar as described by Chowdhury et al. (1997). Khan et al. (1997) and Hossain et al. (1999). Accordingly, bacterial inoculum containing approximately 5.4 × 10⁷ CFU/ml in Pint was prepared by standard dilution method to produce experimental infection in ducks. Each of the 10 experimental ducks was intranasally inoculated with 1.0 ml of 5 × 10⁷ CFU/ml live virulent P. multocida organism at the age of 16 weeks. All the experimentally infected ducks were observed daily to record the signs and symptoms up to the time of death.

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Necropsy and histopathological analysis

Necropsy examination was performed as soon as the experimentally infected ducks were found dead. Swabs or tissue material from liver and heart were streaked onto blood agar plates. The plates were incubated at 37 °C for 24 hours to detect the growth of organisms. The positive cases showing growth of P. multocida were confirmed by standard bacteriologic procedure as described by Cowan and Steel (1985).

RESULTS AND DISCUSSION

A violent chicken isolate of P. multocida (1.5 ± 10^5 CFU/ml) was used for experimental production of infection in standing breed of ducks at 1.0 ml/kg intramuscularly. A study in pathogenesis and clinical-pathological changes. The incubation period of intramuscular inoculation of P. multocida in 10 breeding ducks varied from 12 to 43 hours, of which 12 hours were recorded in 4 (40%) ducks, 24 hours in 5 (50%) and 48 hours in 1 (10%) duck. This observation supports the earlier reports of Baki et al. (1991) who reported 16 to 24 hours incubation period in ducks inoculated with 2 × 10^5 CFU - bird intramuscularly, and Khan et al. (1997) who reported 10 to 12 hours incubation period in chickens inoculated intramuscularly with 5.0 ± 10^6 CFU/ml P. multocida organisms. These findings of incubation period in this study also support the report of Bhat and Banerjee (1990) who detected the P. multocida that colonize in the dorsal pharynx within 6 hours of experimental infection and then invade the blood system within 6 to 12 hours of infection. Tussi and Manchanda (1989) reported that the birds enteritis enterotoxemia and circulatory they were expelled in liver spleen and peritoneal fluid. The suppurated bacteria easily escaped from the pharyngeal process and produced disease rapidly (Kapan et al., 1987).

The clinical signs first observed a 12 hours of post-infection (PI) that include distention and depression, slight rise of body temperature (41.3 °C) and ruffled feathers (Fig. 1). At 24 hours PI, ducks showed the signs of severe weakness, drooling, anorexia, fever (43.5 °C), and emaciation from eyes (Fig. 2). Lassitude, whitish diarrhoea and cr粽w and unable to hold the head (Fig. 3) with treacle squeezing the ground below the head was noticed in some ducks. At 48 hours of PI, there was slight hypotension characterized by dark red colouring winters, comb and ear lobes. The other signs included anorexia, ileus, gastro-intestinal obstruction with mucous and submucosal chomac tachment (40.5°C). Labored breathing, cyanosis and dehydration, followed by moribund and death (Fig. 4). Death of ducks occurred at 24 hours, three at 48 hours and other three died at 72 hours PI. Death of ducks due to P. multocida infection might be caused by acute shock (Parker, 1982; Lee et al., 1992). However, Kisan et al. (1989) suggested that the renal degeneration, cardiorespiratory, severe proventriculitis, extensive lesions in liver, blood, heart due to haemorrhages, hystolethrosis and mucosal edema in the lung may have also contributed to the clinical manifestations and death of birds. The presence of lesions in the liver and kidneys in fowl cholera might have resulted definitive metabolism of uric acid and wastes and the dephosphorylation of which diaphragm might be related to the presence of urea head and uric in the form (Khan et al., 1994; Hasnain et al., 1999). The occurrence of leg weakness, immobility and lameness in Duck cholera might be due to arthrophythesis and tenosynovitis (Karin, 1987). Labored breathing and dyspnea might be due to edema and edema of the lungs (Hasnain et al., 1999). The clinical findings of Fowl cholera recorded in standing ducks in this study are in conformity with the earlier reports made on ducks (Baki et al., 1991) and chickens (Khan et al., 1997; Hasnain et al., 1999).

All the 10 ducks experimentally infected with P. multocida died within 24 to 72 hours post-infection and the post-mortem examination of these ducks showed severe septicaemic conditions with blood vascular congestion (Fig. 5) and histopathological examination of the internal organs with characteristics in the lungs, abdominal fat and intestinal mucosa (Fig. 6) and occasionally 'pseudo' haemorrhage in the brain, mesenteric lymph nodes with increased pericardial fluid and edematous haemorrhage in the heart (Fig. 7) and peritoneal fluid. Swollen and congested liver with multiple necrotic foci on the visceral surface (Fig. 8), and enlarged and inflamed heart (Fig. 9) were recorded. Trachea and lungs were hystolethrotic (Fig. 10) and contained increased amount of sputum fluid and viscid mucus in the trachea, crop and intestines. These post-mortem findings of fowl cholera in ducks are in conformity with the earlier reports made on ducks (Karin, 1987; Baki et al., 1991; Kisan et al., 1999) and chickens (Kisan et al., 1998; Khan et al., 1997).

Bacteriological isolation from dead ducks

Swabs were collected from liver and heart of the 10 dead ducks and streaked onto blood agar plates, and the plates were incubated at 37 °C for 24 to 48 hours. All the swabs cultured from 10 ducks showed growth of P. multocida in the inoculated plates and were further confirmed by standard bacteriological procedure (Cowan and Steel, 1985). Briefly, the growth of P. multocida organisms in blood agar yielded small colonies with white opaque, mucoid and translucent appearance and no haemolysis (Fig. 11).
Fig. 1. A 16-week-old Jindung duck experimentally infected with Duck cholera showing clinical signs of diarrhoea, depression and ruffled feathers at 12 hours of post-infection.

Fig. 2. Experimentally produced Duck cholera in a 16-week-old Jindung duck showing exudation from eyes with matted feathers at 24 hours of post-infection.

Fig. 3. Experimentally induced Duck cholera in a 16-week-old Jindung duck showing characteristic whitish diarrhoea and unable to hold the beak at 24 hours of post-infection.

Fig. 4. Photograph of three Jindung ducks, of which left two are dead and right one at moribund stage at 24 hours of post-experimental injection with virulent Pasteurella multocida.

Fig. 5. Post-mortem lesions of a duck died of experimental Duck cholera showing severe congestion and haemorrhage of almost all the internal organs.

Fig. 6. A portion of a rectal slime of a duck died of experimental Duck cholera showing extensive congestion and haemorrhage of the intestinal mucosa.
Fig. 7. Necropsy changes of a duck died due to experimental Duck cholera showing anemic haemorrhage in the heart.

Fig. 8. A liver of a duck died due to experimental Duck cholera showing swollen and congested the whole liver.

Fig. 9. A heart of a duck died due to experimental Duck cholera showing congestion and sub-epicardial haemorrhages.

Fig. 10. Post-mortem changes in a duck died due to experimental Duck cholera showing congested and haemorrhagic trachea, lungs, liver and kidneys.

Fig. 11. A pure culture colony of Pasteurella multocida on blood agar isolated from a duck dead of experimental Fowl cholera infection.

Fig. 12. Results of biochemical test of P. multocida isolated from a dead duck due to experimental Fowl cholera showing fermentation of dextrose, sucrose and mannitol but not fermentation of lactose and maltose.

Clinicopathology of experimental Duck cholera
Gran's staining of the immersion smears of heart and lung, and smears prepared from culture materials revealed Gran's negative, exo-bacillarity and bipolar organisms. Biochemical tests of the isolated organisms showed complete fermentation of dextrose, mannite, and mannitol but did not ferment lactose and maltose (Fig. 12) which are the characteristics of P. multocida organisms (Calnek et al., 1997).

It appears from the results of this study that P. multocida isolated from chickens is also highly pathogenic which caused 100% mortality in ducks with dose same (5.4 x 10^4 CFU/ml) of necrosis.

ACKNOWLEDGMENTS
The authors are grateful to the Smallholder Livestock Development Project-2 (SLDP-2) for financial support to conduct this research work. We also wish to thank Dr. Jonathan Bet, Senior Adviser, SLDP-2 for assistance and cooperation during this study.

REFERENCES