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Field Observations on a Devastating Respiratory Syndrome in Broilers in Pakistan

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Abstract

A respiratory syndrome causing heavy mortality in broilers is widespread throughout the country. It involves all ages of broilers mostly; layers are also susceptible. Initially there is sinusitis, followed by pneumonia, mucoid to haemorrhagic tracheitis and air-sacculitis. Mortality varies from 5 percent to as high as 70 percent. Target organs included all respiratory tract showing congestion, haemorrhages and necrosis. Tracheal epithelium contained basophilic intranuclear inclusions. Lungs showed multiple colonies of coca-bacilli. The disease appears to be virus-bacteria interaction.

Introduction

There were a few diseases in poultry industry in Pakistan around two decades ago. With the growth of poultry industry, many diseases have marched in. Amongst these the appearance of hydropericardium syndrome in 1987 (Anjum et al., 1989), infectious bursa' disease in 1989 and avian influenza in 1994 are worth mentioning (Anjum, 1997). The diseases are still continuing and causing heavy losses to poultry farmers. Once again poultry farmers are experiencing heavy losses with a previously unknown respiratory disease described here.

Materials and Methods

The study was conducted on 45 broiler flocks and 11 layer flocks in and around Faisalabad. The flocks included all ages. All possible factors of aetiological and epidemiological significance were recorded. Clinical signs were observed on flock visits and postmortem examination of sick and freshly dead birds were conducted. Specimens were collected and processed for histopathology (Anjum, 1980) and agent isolation.

Results

The disease started in Khurrianwala (Faisalabad) around 2nd week of March 1999 around Faisalabad. There was a rapid spread in the vicinity within two weeks. The disease was primarily seen in broilers; layer flocks were also involved.

Clinical signs: A rhinitis with mucoid to purulent discharge was the first finding by the farmer, which was visible as sticky nose or was confirmed by pressing upon nostrils. The nature of nasal discharge was mucoid to purulent. Some birds may sneeze or rub their nose with toes and a very few showed swelling of inter-mandibular space. Changes in feed and water intake were usually insignificant. There was not much morbidity. Birds started losing live body weight. Body temperature was very high. Respiratory sounds increased with the passage of time. Few birds in some flocks showed swelling of face.

The disease is usually seen from 12 days of age onwards in broilers. As old as 55 weeks old layers were also involved.

Mortality: Mortality shows a typical pattern. It started in 4-6 days after rhinitis. Initially, 2 to 5 died in a flock of 1000 birds which attains peak during the next 4 to 5 days and as many as 40 to 60 die daily. Mortality continues for several days. The disease causes more mortality in broilers than in layers.

Gross lesions: Lesions were mainly seen throughout respiratory system starting from nasal sinuses to trachea, lungs and air sacs. Carcase was almost always congested and sometimes slightly cyanotic. There was mucoid to purulent discharge in nares. The trachea was inflamed and contained clear mucoid or purulent secretion in lumen. Tracheal exudate was sometimes blood tinged. Mild to severe haemorrhages were present in trachea in advanced cases. Initially, lungs were congested, oedematous and consolidated. Cut pieces of lung tissue settled in 10 percent formalin. With the passage of time a white fibrino-necrotic layer covered the lungs. There was inflammation of the thoracic air sacs which first became foamy, and then changed into a thick white layer around lungs. Rarely thoracic air sacs contained inspissated pus. Abdominal air sacs were not commonly involved.

Changes in other organs were non-specific. Liver was commonly dark red in colour and sometimes enlarged. Spleen was normal, pale or reactive. Bursa was mostly normal or regressed. Kidneys were generally normal in appearance but urates were commonly present in ureters. Many birds in some flocks also showed vesicular dermatitis like lesions in hip region.

Histopathology: Lungs showed engorgement of vessels in inter-alveolar spaces. Oedematous fluid was seen filling the alveoli, Small to large haemorrhages were seen throughout lung tissue. Scattered patches of necrosis were present throughout. Mononuclear cell infiltration was predominating. Several colonies of coca-bacilli were seen in lung tissue in necrotic areas.

Trachea mucosa was sloughed and haemorrhagic with mononuclear cell infiltration. Epithelium in exudate contained in lumen of trachea showed basophilic intranuclear inclusion bodies. Coco-bacilli were also seen in tracheal exudate. **Experimental transmission:** Experimental transmission with trachea and lung homogenate (10 percent with gentamicin added at the rate of 1 mg/mL) in day-old and 2-week-old cockerels did not succeeded.

Discussion

Havocs have been threatening poultry industry in Pakistan since late 1980's. The current respiratory syndrome is an addition to the already existing wide spectrum of diseases. A variety of respiratory diseases of chicken were included in differential diagnosis. Rhinitis and facial swelling resemble coryza, tracheal lesions resemble infectious laryngotracheitis and infectious bronchitis and chronic cases involving air sacs resemble very much to mycoplasmosis and colisepticaemia. Congested carcase indicates febrile reaction of the body.

The presence of intranuclear inclusions in epithelium of tracheal exudate suggests presence of a virus. Amongst the already known agents causing such inclusions is infectious laryngotracheitis (ILT) but in ILT the inclusions are eosinophilic (Jordan and Pattison, 1996; Calnek *et al.*, 1997). The inclusions in the current outbreak resemble to adenovirus inclusions in liver (Anjum *et al.*, 1989; Anjum, 1997).

Colonies of coco-bacilli organisms in lung tissue and in trachea exudate may have a primary role in disease or may be secondary invaders. Studies are needed to isolate and characterize the organism. Have isolated Haernophllus paragallinarum from nares of birds affected with the respiratory syndrome, however, they have not characterized its pathogenicity in the chicken. There was no correlation of the disease with area, feed, breed, chick origin, sex or any other factor.

A priori, the concurrent presence of basophilic intranuclear inclusion bodies in tracheal epithelium and bacterial colonies in lung tissue suggest that the syndrome is a complex of virus-bacteria aetiology. It seems as if a virus which is unable to cause disease on its own and under circumstances, yet unknown, may trigger a respiratory disease alone or in collaboration with the bacteria to complicate the situation. Work is in progress for isolation and characterization of virus and bacteria.

In conclusion, the respiratory disease syndrome is result of virus-bacteria interaction.

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