Pakistan Vet. J., 23 (4): 2003

FOWL CHOLERA IN A BREEDER FLOCK

Z. Parveen, A. A. Nasir, K.Tasneem and A. Shah
Veterinary Research Institute, Lahore Cantt.

ABSTRACT

During January, 2003 *Pasteurella multocida* the causative agent of fowl cholera was isolated from a breeder flock in Lahore District. The age of the flock was 245 days. Increased mortality, swollen wattles and lameness were the clinical findings present in almost all the affected birds, while gross lesions were typical of fowl cholera. To prove the virulence of the organism, mice and six-week old cockerals were infected and *P. multocida* was reisolated.

Key words: Fowl cholera, breeding flock.

INTRODUCTION

Fowl cholera or avian haemorrhagic septicaemia is a contagious disease caused by *P. multocida*, a gram negative rod affecting almost all species of fowl. It usually appears as a septicaemic disease resulting in high morbidity and mortality (Brand, 1984). It can persist as a chronic disease producing localized infection of hock joints, foot pads, peritoneal cavity, oviduct and sometimes results in torticollis. Fowl cholera is more prevalent in late summer, fall and winter. This seasonal occurance is one of the circumstances rather than lowered resistance except that chickens become more susceptible as they reach maturity (Rimler and Gllsson, 1997). Free flying birds and most farm animals can be carriers of *P. multocida.* Generally these organisms except for those from swine and cats are avirulent for fowl, (Curtis and Ollerhead, 1980). Once the disease is introduced into a flock, it spreads very rapidly by contamination of open watering system and cannibalism of contaminated carcasses (Pabs-Garnon and Soltys, 1971). Cholera has rarely been reported in breeders and very little information regarding the incidence and nature of the disease is available in Pakistan. This report describes an out break of fowl cholera in a breeder flock in Lahore District, Pakistan.

CASE HISTORY AND FINDINGS

A flock of approximately 25,000 breeders suffered unusually with high mortality beginning from 245 day of age. The main complaints were sudden death with mortality varied from 20 to 25 birds per day. The birds were getting down on legs before death. The birds were fed a commercial feed. The flock had no previous history of illness and had not received any medication.

At necropsy, the birds were found to be dehydrated, with enlarged liver and spleen showing focal areas of necrosis. Hemorrhages were present in intestinal mucosa. Samples of liver, spleen and heart blood were collected for bacteriological investigation. The tissues were seared with a hot blade, the seared surface was cut with a sterile blade and a sterile loop was rubbed across the fresh cut surface. The loops were then inoculated to blood agar, nutrient agar, tryptose agar and MacConkey agar and incubated for 24 hours aerobically at 37^0C. Numerous bipolar gram negative bacteria were demonstrated on blood agar, nutrient agar and tryptose agar. No growth was found on MacConkey agar. Biochemical reactions were typical for *P. multocida* (Carter, 1984).

EXPERIMENT

In order to establish that *P. multocida* was the etiological agent of the disease in breeders, four albino mice and four six-week cockerals were placed in separate cages and grouped as A and B. The *P. multocida* isolated from initial culture, was grown in peptone water. Albino mice (group A) was given 0.4 ml of culture intraperitoneally and cockerals (group B) were given 0.5 ml of culture subcutaneously. All the albino mice and birds in both groups died within 48 hours of challenge. Gross lesions were similar to those in the field cases except that lesions were more pronounced. *P. multocida* was reisolated from liver, spleen and heart blood of dead birds and mice.

DISCUSSION

It is very difficult to ascertain the mode of introduction of the fowl cholera into a flock because natural route of entry of *P. multocida* is through the mucous membrane. In this case free flying birds could

have transmitted infection. It is also possible that some of the affected flock had early exposure to infectious bursal disease with resulting immunosuppression, making the birds more susceptible (Rimler and Gllsson, 1997). Open trough waterers may have aided in rapid spread of the disease.

The gross lesions of early E. coli, Salmonella and staphylococcal septicaemias are similar to the lesions in birds with fowl cholera. In addition, the purulent synovitis often occuring as chronic sequele to P. *multocida* is difficult to differentiate from synovitis caused by other bacteria (Jean and Gllsson, 1989).

The diagnostician should always consider pasteurellosis as a possibility when confronted with a case of acute rapidity increasing mortality in breeders regardless of whether gross lesions are present. The organism can be readily cultured from liver or spleen on blood, nutrient and tryptose agar, allowing for antibiotic sensitivity testing and proper antibacerial therapy.

REFERENCE

Brand, C.J., 1984. Avian cholera in the central and Mississippi fly ways during 1979-80. J. Wildl. Manage., 48: 399-406.

Carter, G.R., 1984. Diagnostic Procedures in Veterinary Bacteriology and Mycology. 4th ed. Charles C. Thomas, Springfield, Illinois. Pp. 11-121.

Curtis, P.E. and G.E. Ollerhead, 1980. *P. multocida* infection of cats on poultry farms. Vet. Rec., 110: 13-14.

Jean, E. S. and J. R. Gllsson, 1989. Fowl cholera in broilers. Avian Dis., 33: 816-819.

Pabs-Garnon, L.F. and M.A. Soltys, 1971. Mehtods of transmission of fowl eholera in turkeys. American J.Vet. Res., 32: 1119-1120.

Rimler, R.B and J.R. Gllsson, 1997. Fowl cholera. In: Diseases of Poultry, B.W Calnek H.J, Barnes, C.W. Beard, L.R. Mc Dougald and Y.M Saif (Eds.) 10th ed. Iowa State Uni. Press Ames, Iowa USA. pp: 143-159.