PATHOLOGY OF PASTEURELLA MULTOCIDA INFECTION IN CHICKENS

Shilpa Sood1 and P.C. Verma

CCS Haryana Agricultural University, Hisar - 125 004, India

ABSTRACT

The present study was conducted on 42, 12-15 weeks old birds to study the gross and histopathological changes induced by Pasteurella multocida infection. The birds were divided into two groups viz., control and infected consisting of 21 birds each. The birds in infected group were administered with LD50 of local isolate of Pasteurella multocida A:1. Three birds from each group were randomly sacrificed and subjected to thorough post mortem examination. Tissues were collected in 10% buffered formalin for histopathological studies. Initially pathological lesions were of acute septicaemic nature, congestion, hemorrhage and presence of bacterial colonies. Later on fibrinous pericarditis, necrosis of liver and kidney and lymphoid depletion and RE cell hyperplasia in the spleen were observed.

INTRODUCTION

Fowl cholera is a septicaemic bacterial disease of poultry. It is one of the earliest reported diseases of chicken caused by Gram negative Pasteurella multocida and is characterized by high mortality. The outbreaks of this disease have been reported in India from time to time and more so in the recent past (Char et al., 1982; Ramadevi et al., 2000). Currently, the disease has reemerged as a major cause of economic losses to the poultry industry all over the world. Therefore, the present study was undertaken to study pathology and pathogenesis of a local isolate of P. multocida in chickens.

MATERIAL AND METHODS

In the present study, a total of 42, 12-15 weeks old disease free white Leghorn birds were kept under strict hygienic conditions. They were given commercial layer ration and boiled drinking water ad lib. They were divided into two groups viz. control group (Group-I) and infected group (Group-II) consisting of 21 birds each. The infected group was administered LD50 (1.8 x 10^7.23 CFUs) of local isolate of P. multocida serotype A:1 intravenously. The control group was administered with equal amount of plain sterile BHI broth @ 0.2 ml I/V. Three birds from each group were slaughtered at 12 hours, 1 day, 2 days, 5 days, 7 days, 10 days and 15 days post infection (DPI). Detailed post-mortem was conducted on sacrificed birds and gross lesions if any, were recorded. The tissues namely liver, heart, kidneys, lungs, spleen, brain, ovaries, pancreas, proventriculus and oesophagus were collected in 10% buffered formalin and processed for H and E staining.

RESULTS AND DISCUSSION

Grossly the birds in infected group revealed lesions like congestion of lungs, liver, spleen, kidney and heart and petechial/ecchymotic hemorrhages on the surface of heart in early stages (Fig. 1). Later on pericardium showed fibrinous pericarditis. The liver and spleen revealed mottling and congestion, splenomegaly and occasionally necrotic foci in liver. Hemorrhages were also observed on the junction of oesophagus and proventriculus and occasionally on proventriculus only. Kidneys reveal edema and ovaries also revealed congestion initially. Conjunctivitis was also observed during the later half of the experiment. These changes have also been

Present address:
1Department of Veterinary Pathology, SKUAST, Jammu.
Fig. 1. Petechial hemorrhages on heart

Fig. 2. Vasculitis with thickening of blood vessel walls due to presence of bacterial colonies.
Fig. 3. Fibrinous thickening of interlobular septa and pneumonic changes in lungs

Fig. 4. Hemorrhages and thickening of pericardium
earlier observed (Char et al., 1982; Panda et al., 1981; Khan et al., 1977 and Gustafson et al., 1998).

There was generalized vasculitis, (Fig. 2) hyperemia, fibrinous thrombi and presence of bacterial colonies were noticed in heart, liver, spleen, ovaries, lungs and kidneys. Similar changes have earlier been reported by Rhoades and Rimler (1991).

The changes in the oesophagus consisted of congestion of serosal vessels initially and in the later stages there was hyperactivity of the gland, degeneration and leukocytic infiltration which consisted initially of heterophils and then of mononuclear cells. Similar changes in oesophagus have been earlier reported by Morishita et al. (1997). The salient changes in the proventriculus consisted of congestion and desquamation of glandular epithelium, cellular infiltration in the mucosa, glandular degeneration and desquamation of glandular epithelium, infiltration of heterophils initially and mononuclear cells in later stages in the submucosa. In the chronic cases, fibrinous thickening and fibroblastic lesion, proliferation in interglandular tissue and necrotic changes in submucosal glands were also observed.

The changes in the lungs initially consisted of congestion, hemorrhages and vasculitis upto 24 hours P.I but later on pneumatic changes were evident which consisted of heterophilic infiltration of alveoli and fibrinous thickening of alveolar walls (Fig. 3). Later on there was degeneration of bronchi and desquamation of epithelial cells of bronchioles. Occasionally focal areas of necrosis with bacterial colonies involving groups of alveoli were also noticed. Beyond 7 DPI, the changes consisted of interstitial pneumonia characterized by severe thickening of alveolar walls and walls of blood vessels with fibrinous exudates.

The microscopic changes observed in the liver consisted initially of congestion, hemorrhages and mild degeneration with presence of bacterial colonies upto 24 hours P.I but in later stages necrotic changes involving groups of hepatic parenchymatous cells with heterophilic infiltration were prominent. Occasionally, a few microgranulomas consisting of masses of mononuclear cells and heterophils were also observed. In later stages, the changes were less severe and consisted of Von Kupffer cell hyperplasia and thickening of portal triad due to leukocytic infiltration consisting chiefly of mononuclear cells at 10 and 15 D.P.I. Similar changes characterized by acute focal hepatitis and necrosis with heterophilic infiltration have been reported earlier (Rhoades, 1964; Hunter and Webster, 1979).

The sections of kidneys examined revealed congestion generally upto 5 DPI. Other changes observed were glomerular degeneration and hypercellularity, tubular degeneration, tubular necrosis and leukocytic infiltration and presence of bacterial colonies. The changes of chronic interstitial nephritis characterized by thickening and replacement of intertubular tissue with fibrinous exudates, leukocytic infiltration and fibroblastic tissue proliferation was observed in later stages of infection. In heart, initially the changes consisted of congestion and hemorrhages. The pericardium and myocardium were thickened with leukocytic infiltration and fibrinous exudates (Fig. 4). In many cases, coagulative necrosis and bacterial colonies were also noted. Pericarditis was fibrinous in nature during later stages with heterophilic infiltration initially but later on infiltration was of mononuclear cells. Myocardial changes consisting of degeneration, muscular disruption, leukocytic infiltration and fatty changes were observed especially at 10 DPI.
Microscopically spleen revealed congestion hemorrhages, depletion of lymphoid elements hyperplasia of reticuloendothelial cells and presence of bacterial colonies. Salient changes in the ovary revealed congestion and degeneration of ovarian follicles, leukocytic infiltration and presence of bacterial colonies. In pancreas congestion was occasionally observed at some intervals. There was degeneration of acinar cells and leukocytic infiltration around the acini and blood vessels at 5 and 7 DPI. In brain, no appreciable change except congestion in a few cases was observed in cerebellum up to 5 DPI. On 7 and 10 DPI, there was congestion, satellitosis, neuronophagia and purkinji cells degeneration.

ACKNOWLEDGEMENT
Authors are thankful to Professor and Head, Department of Veterinary Pathology for providing necessary facilities during the investigation.

REFERENCES