EFFECT OF CYCLOPHOSPHAMIDE ON PATHOLOGY OF HYDROPERICARDIUM SYNDROME IN EXPERIMENTALLY INFECTED CHICKENS

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Cyclophosphamide is a known immunosuppressive agent affecting primarily the humoral immune response (Glick, 1971). The present investigation was conducted to study the pathology of hydropericardium syndrome (HPS) in cyclophosphamide-immunosuppressed chickens. Twenty, day-old broiler chicks were divided into two groups A and B. Group A birds were given cyclophosphamide at 2mg per chick (0.1 mL of a 2% solution) intramuscularly daily for four days. Group B was given 0.1 mL normal saline per chick intramuscularly for same duration. At 21 days of age the chicks in both the groups were inoculated with ID$_{50}$ (0.5 ml, $10^{3}$ dilution of 20% HPS infected liver suspension).

No clinical signs except sudden mortality were observed in the first few cases. However, in other cases the bird became debilitated, dull and depressed, with ruffled feathers followed by resting the head on the chest with eyes closed. Mortality was observed as early as 36 hours post infection (h PI) in group A (total 90%) while in group B mortality (total 40%) was observed only after 48 h PI.

The most prominent gross lesions included hydropericardium and marked enlargement of the liver along with petechial haemorrhages on the liver surface. Kidneys were pale and swollen. The spleen and bursa of Fabricius were highly atrophied in group A whereas only mild changes were seen in group B. Histopathological studies revealed cellular degenerative changes in hepatocytes along with necrosis and hepatitis with mononuclear cell infiltration. Basophilic intranuclear inclusion bodies were observed in hepatocytes. Cardiac muscle fibres revealed congestion and haemorrhage along with lymphocytic and heterophilic infiltration. The spleen and bursa of Fabricius showed severe depletion of lymphocytes in the treated group and mild to moderate depletion was observed in the untreated group. There was increased mortality, more pronounced clinical signs and more gross and microscopic lesions in the cyclophosphamide-treated group.

A role for immunosuppression in precipitation and aggravation of HPS under field conditions has been suggested by several workers (Cowen et al., 1996; Toro et al., 2000), but sparse experimental information is available in the literature on the effect of immunosuppressive agents on the pathology of HPS. The present study clearly demonstrates that treatment with cyclophosphamide increases the severity of HPS in chickens.


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