Histopathology of infectious bursal disease in non-lymphoid organs of chickens

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INTRODUCTION

Infectious bursal disease (IBD) was first described by COSGROVE (5). Since then much of the pathology of the disease has not been well understood. Young chickens under 2 weeks of age suffer subclinical form of the disease in absence of maternal antibodies (13, 28). The mechanism of this age-related immunity is yet to be identified. The upper age limit of susceptibility appears to depend on the involution of the bursa of Fabricius (bursa) which contains surface immunoglobulin M-bearing B cells which have been reported to be the target cells for IBD virus (IBDV) infection (12, 19). But other workers have shown...
that IBDV can replicate in other cells in vivo (3, 14, 18). In fact SCHAT et al. (26) observed more haemorrhages in the muscles and intestines of embryonally bursectomised chicken than in intact chickens infected with IBDV thereby also questioning the earlier theory of indispensability of the B cells in initiation of IBD (8, 15). Mortality in IBD has also been associated with depletion in complement level (8, 27) and to clotting abnormality (28). The involvement of immune-complex in the pathogenesis of IBD was suggested by LEY et al. (16) when they demonstrated immunofluorescence in the glomeruli of infected chickens. In this study histopathological evidence of immune-complexaemia or ARTHUS reaction is examined in some non-lymphoid organs. The microscopic changes in the lymphoid organs have been described in earlier publications (20, 23).

**MATERIALS AND METHODS**

**Flock history and IBDV**

The chickens and the local Nigerian isolate of IBDV used are the same with those already described in the studies of the persistence of the virus and the appearance of precipitins in infected chickens (21). The virus was obtained as 20 p. 100 suspension of bursae of chickens that died of confirmed field outbreaks of IBD in Nsukka area (22). The suspension was found to have a bursal lesion of $10^{4.8} / 0.5$ ml by method of REED and MUENCH (25). Two hundred chickens were given 0.05 ml of the bacteria-free suspension while 60 that served as control had 0.05 ml of only the sterile phosphate buffered saline (PBS) intra-ocularly.

**Histopathology**

Five infected and 2 control birds were sacrificed daily for 15 days post-infection (PI). The kidney, liver, muscles of the chest and thigh, pancreas, lung, brain, and proventriculus of the sacrificed and 19 dead chickens were fixed in 10 p. 100 formal saline for a minimum of 24 h. They were processed and embedded in paraffin wax. Thin sections 5μ thick were cut, stained with haematoxylin and eosin (H & E) and examined under light microscope.

**RESULTS**

Histopathological sections of the kidney of chickens sacrificed on days 1 and 2 PI were congested and haemorrhagic while some epithelial cells of the tubules and ducts were pyknotic and eosinophilic. By day 3 PI oedema and degeneration of the epithelial cells were evident. In birds that were sacrificed on day 4 PI, eosinophilic casts were present in the ducts and tubules. But in those that died of IBD on the same day, there was generalized pyknosis and karyorrhexis of the epithelial cells and more casts in the tubules and ducts. The main renal lesions observed in birds that were sacrificed on day 5 PI were congestion and haemorrhage. Degeneration was not severe. Lesions in birds that died on the same day (Photo 1) were similar — but more severe — to those of the birds that died on day 4 PI. But no casts were seen and there was hyperplasia of the epithelial cells in some ducts and tubules.

On day 6 PI, kidney of sacrificed birds had few degenerative lesions mainly in the ducts, some of which contained casts of desquamated epithelial cells. Heterophils were found around the large collecting ducts. But kidney of birds that died on that day showed severe karyorrhexis and degeneration of the epithelial cells. Hyperplasia of epithelial cells and large eosinophilic casts in the tubules and ducts were also observed (Photo 2). By day 7 PI there was much progress in recovery. Most of the tubules and ducts were normal. But severe focal mononuclear cell infiltration was observed (Photo 3). Lesions on days 8 and 9 PI were similar to those of day 7 PI. On day 12 PI infiltration by mononuclear cells was still evident. Oedema and haemorrhage were seen in few of the renal samples on day 15 PI. The kidney of control birds showed no remarkable changes.

Liver was congested and haemorrhagic on day 1 PI. By day 3 PI oedema and degeneration of some hepatocytes were present. Chickens that died on days 4 and 5 PI had degenerated hepatocytes while birds sacrificed at the same period showed only congestion and haemorrhage which were also present in the dead birds. Karyorrhexis of the hepatocytes was found in birds that died on day 6 PI. Degeneration was last observed on day 8 PI. By day 12 PI only congestion of few portal veins was observed. There was no lesion in the
liver by day 15 PI. The organ in control birds had no lesion.

Muscle lesions first appeared in birds sacrificed on day 3 PI and comprised congestion, oedema, haemorrhage, and necrosis (Photo 4). In birds that died on day 4 PI, additional lesion was the presence of large mononuclear cells that appeared to be phagocytes. Similar lesions were seen in birds that died on day 6 PI while those that were sacrificed on days 4 and 5 PI showed only congestions and haemorrhage. Oedema, congestion, haemorrhage, necrosis, and infiltration by large mononuclear cells were found on days 7 and 8 PI. The muscle fibres were still necrotic by day 9 PI when the last observation was made. The control birds had no significant muscle lesion.

In birds that were sacrificed on day 6 PI,
the proventricular glands were congested while those that died on that day also had many necrotic glandular cells in the lumina. The organ was normal by day 8 PI. No lesion was observed in the proventriculus of control chickens. Sections of the brain, lung, and pancreas of infected and control chickens showed no lesion.

**DISCUSSION**

In the kidney there was no sign of neither thickened glomerular basement membrane nor mesangial cell proliferation. Similar observations have been made by LEY et al. (17) who also reported that ultrastructural change suggestive of acute immune-complex glomeru-
lonephritis in IBD was rare. Furthermore, HENRY et al. (11) found atrophic glomeruli in birds examined on days 1 to 5 PI. The infiltration by mononuclear cells has also been described (11, 17). But the generalized and coagulative necrosis of the epithelial cells observed in this study appear to be more severe than already described for the kidney in IBD (4, 11). This could be part of the reason why IBD in Nigeria is commonly associated with abnormally high mortalities of up to 43.8 p. 100 (24), over 50 p. 100 (6) and 11.5 to 33.5 p. 100 (22). HELMOLDT and GARNER (10) and LEY et al. (17) recorded tubular necrosis in few of the infected chickens.

Apart from pyknosis in some epithelial cells on days 1 and 2 PI, chickens sacrificed on days 3 to 15 PI showed no evidence of renal necrosis. But all those that died on days 4 to 6 PI had generalized karyorrhexis and many casts in the ducts. Tubular necrosis was most severe in chickens that died on day 5 PI when the highest daily mortality (52.7 p. 100 of the entire mortality) was recorded. These observations tend to suggest that generalized karyorrhexis in the tubules and ducts may be the immediate cause of death in IBD. Previous studies of renal microscopic changes in IBD have been mainly on sacrificed chickens (4, 10, 11, 17). This could be part of the reason why these earlier workers found mild renal changes.

The histopathological lesions in the liver appear to be in agreement with observations of CH0 and EDGAR (4). Necrosis of the hepatocytes although present in less than 30 p. 100 of the cells was found in only the chickens that died on day 6 PI. The hepatic changes do not appear to be of much significance in the pathogenesis of IBD.

Arteritis was not observed in the muscles of the chest and thigh. This supports the observation of SCHAT et al. (26) who produced muscle lesions in embryonally bursectomised chickens which could not have been able to produce antibodies and immune-complexaemia.

The observations in this experiment have not shown any histopathological evidence of immune-complexaemia in the pathogenesis of IBD. Changes in the dead and sacrificed were of different types in the kidney and liver while they were similar but more severe in dead than in sacrificed chickens in the lymphoid organs (20, 23). However, microscopic changes in acute immune-complex glomerulonephritis could be subtle (2). This may be the case in IBD which has short duration. Consequently, immunofluorescence has been found to be a more sensitive method of detecting acute glomerulonephritis (7).

Earlier observations (21) support the report that the clinical signs of IBD seem to depend on the rate and amount of viral production in B lymphocytes (1). The rate of virus detection has been found to be the same irrespective of age and bursectomy in chickens (8, 26). But these workers did not determine the titre of the virus in the organs of the groups at various periods.

RESUMEN


Se han infectado pollos con el virus de la enfermedad de Gumboro aislado en Nigeria. Se observaron algunos casos de plenosis en células epiteliales de los tubos uriníferos y de los canalículos en pollos sacrificados 1 y 2 días después de la infección. Luego, sólo los pollos muertos naturalmente mostraron síntomas de necrosis renal sumamente graves al 5º día, coincidiendo con las tasas de mortalidad más elevadas. Se notaron un ataque epitelial de los canalículos y una infiltración por células mononucleares a partir del 7º día y después. No se observó la necrosis de los hepatocitos más que en los pollos muertos al 6º día.

REFERENCES

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