Histopathology of bursa of fabricius and post-mortem findings in infectious bursal disease affected broiler chickens in Haryana

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Abstract
The aim of the present study was to diagnose infectious bursal disease (IBD) using gross and histopathological approaches. The study period was from January, 2015 to February, 2016. Gelatinous exudates around bursa, swollen and haemorrhagic bursa, atrophied bursa and haemorrhages on thigh and pectoral muscles were the major necropsy findings. During post-mortem bursae of fabricius were taken for histopathological study.

Keywords: Infectious bursal disease, histopathology, post-mortem findings

Introduction
Infectious bursal disease (IBD), a highly contagious and immunosuppressive viral disease of chickens, was first described by Cosgrove in 1962 as “Avian Nephrosis” because of prominent lesions in kidneys. The disease is also known as ‘Gumboro’ disease because of an outbreak in Gumboro area of Southern Delaware, USA. Etiological agent of infectious bursal disease was first identified in 1962 (Winterfield and Hitchner, 1962) [29]. Later on due to pathognomonic lesions in bursa of fabricius, the disease was termed as infectious bursal disease (Hitchner, 1970) [9]. Infectious bursal disease virus (IBDV) mainly affects chicken at 3-6 weeks of age and has a predilection for the bursa of fabricius where the virus infects actively dividing and differentiating B-lymphocytes (Burkhardt and Muller, 1987; Li et al., 2015) [1, 17]. The disease is clinically characterized by dullness, depression, loss of appetite, white watery diarrhoea, soiled vent, ruffled feathers, severe prostration, vent picking and subnormal temperature followed by death (Cui et al., 2013) [6]. Infectious bursal disease virus (IBDV) belongs to the family Avibirnaviridae of the genus Avibirnavirus. The virus is double stranded RNA, bi-segmented (segments A and B), non-enveloped and icosahedral (Dobos et al., 1979; Murphy et al., 1995; Vera et al., 2015) [7, 21, 28]. Two serotypes: serotype-1 and serotype-2 have been reported on the basis of virus neutralization test. Serotype-1 has variation in virulence and pathogenicity, causes disease and immunosuppression in chickens. On the basis of heterogenous antigenecity and sequence analysis, serotype-1 viruses are classified as attenuated, classical virulent, intermediate virulent, very virulent (vv) and antigenic variant strains (Kataria et al., 2001) [10]. Viruses of serotype-2 were isolated from turkeys and are non-pathogenic to both turkeys and chickens. Serotype-2 viruses produce neither disease nor immunity against pathogenic strains of serotype-1 (Muller et al., 2003; Cortey et al., 2012; Vera et al., 2015) [20, 4, 28].

In India, the disease was first reported by Mohanty et al. (1971) [19]. However, the virus, IBDV was isolated for the first time in the country by Jayaramaiah and Mallick (1974) [15]. Later on, the disease was recorded in different forms from all the states. In late 1980s very virulent (vv) IBD viruses were initially reported in Europe (Chettle et al., 1989; Van den Berg et al., 1991) [2, 27] and thereafter were reported around the world, except in North America and Australia (Sharma et al., 2000; Van den Berg, 2000) [23, 26]. The vvIBD viruses are showing antigenetic similarity to the classical strains but differ in virulence and pathogenicity causing 60-100% mortality in infected flocks (Van den Berg, 2000) [20]. Classical virulent strains cause bursal inflammation and severe lymphoid necrosis in infected chickens, resulting in immunodeficiency and 20–30% mortality. Antigenic variant strains cause rapid atrophy of the bursa without inflammation, haemorrhage, or mortality (Snyder et al., 1988; Jackwood and Jackwood, 1994; Jackwood et al., 2008; Li et al., 2015) [23, 12, 14, 17].
Due to the high mortality rates in acute infections, reduced growth, excessive condemnation of carcass and severe immunosuppression by subclinical infections; IBD is of major economic importance to the poultry industry (Choudhary et al., 2012) [3]. Due to immunosuppressive effect of the IBD virus, incidence and severity of secondary and opportunistic infections increase, leading to vaccination failure (Dormitorio et al., 1997; Jackwood and Sommer, 1998) [8, 13].

Materials and Methods

Postmortem examination on 85 carcasses of broiler chicken upto the age of 3-7 weeks brought to Department of Veterinary Public Health and Epidemiology, LUVAS, Hisar conducted during the period of one year (January, 2015 to February, 2016) from different regions of Haryana. Representative and appropriate bursa of fabricius showing post-mortem lesions were collected in 10% buffered formalin for histopathological studies. The formalin fixed tissues of bursa were processed for paraffin embedding technique. The section were cut at the thickness of 3-4 µ and stained with H & E stain (Luna, 1968) [18].

Results and Discussion

In almost all IBD-affected birds, the post-mortem lesions were observed in bursa of fabricius. Haemorrhages on thigh (Fig. 1a) and pectoral muscles, presence of gelatinous exudates around bursa (Fig. 1b), oedematous and swollen bursa (Fig. 1c), haemorrhages in bursal follicles (Fig. 1d) were recorded. These changes were observed in acute form of the disease. However, in chronic form of the disease, the bursal changes comprised of atrophy and presence of cheesy core inside the bursa. The haemorrhages on thigh and pectoral muscles were of milder degree in sub-acute form of disease. In some of the flocks, haemorrhages at the junction of proventriculus and gizzard were also recorded. Besides these, swollen kidneys and enlargement of liver were also noticed during post-mortem examination of broiler chicks.

The histopathological changes in the bursa of fabricius mainly showed the congestion and marked haemorrhages in bursa of fabricius (Figs. 2a, 2b). There was marked depletion of lymphocytes in bursal follicles and fibrous connective proliferation with mild lymphocytic infiltration in inter-follicular areas (Figs. 2c, 2d). Atrophic changes at microscopic level, which were in the consonance with the gross observations. The cystic changes in which there was formation of cystic spaces were observed in bursal follicles (Fig. 2e). Similar pathological findings were reported by Hoque et al. (2001) [10], Rudd et al. (2001) [22], Islam et al. (2008) [11] and Singh et al. (2015) [24].
Fig 2: Photomicrographs showing histopathological changes in bursa of fabricius in infectious bursal disease affected broiler chickens
a) Congestion and marked haemorrhages in bursa of fabricius (H&E × 400)
b) Congestion and marked haemorrhages in bursa of fabricius (H&E × 100)
c) Depletion of lymphocytes in bursal follicles and fibrous connective tissue proliferation with mild lymphocytic infiltration in inter-follicular areas (H&E × 100)
d) Cystic areas with accumulated necrotic debris in bursal follicle and fibrous connective proliferation with mild lymphocytic infiltration in inter-follicular areas (H&E × 200)
e) Atrophy of bursal follicles (arrow) and formation of cystic spaces in bursal follicle with arrow head (H&E × 40).

Conclusion
From the present study, it can be concluded that outbreaks of IBD occur throughout the year in broiler chicken flocks in Haryana state. Infectious bursal disease can be diagnosed on the basis of clinical signs, gross examination and histopathological studies. It is necessary to follow biosecurity measures and vaccinate birds regularly with appropriate strain of vaccine. Furthermore, regular surveillance and identifications of field strains would help in making effective control strategies.

References


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