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Review Article

Infectious Bursal Disease Virus Evolution, Current Situation and its Role as an Immunosuppressive Factor Threatening Chicken Production in Egypt

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Abstract

Infectious bursal disease virus (IBDV) is a double stranded RNA virus of the Birnaviridae family that targets the B lymphocytes in chickens bursa, causing immunosuppression and increased susceptibility to secondary infections. IBDV remains an economically important pathogen worldwide due to direct morbidity, mortality and vaccine failures linked to virus evolution and immune suppression. Globally, IBDV evolution has been characterized by diversification into classic, variant, very virulent IBDV (vvIBDV), antigenic variants, and reassortant strains; ongoing mutation, recombination, and genome segment reassortment drive antigenic drift and shifts that undermine the vaccine protection. In Egypt, multiple IBDV lineages co-circulate, including vvIBDV strains and reassortants gaps in surveillance, vaccine matching, and biosecurity increase disease impact. IBDV contributes substantially to immunosuppression in Egyptian chickens, exacerbating losses from other pathogens and reducing vaccine efficacy against diseases such as Newcastle disease and avian influenza. Effective control requires integrated measures: updated surveillance and molecular characterization, use of properly matched vaccines (including homologous, immune-complex, or vector vaccines where appropriate), strict biosecurity, and monitoring of immunosuppression consequences in flock health management. Due the endemic status of IBDV in the Egyptian chicken flocks reared intensively in some governorate. We recommended continuous surveillance and choice of combatable vaccines, biosafety and biosecurity measures.

Keywords: chickens; ibd, ibdv; prevention; control; egypt

Introduction

Infectious bursal disease virus (IBDV) is a double stranded RNA virus of the Birnaviridae family that targets the bursal of Fabricius and B lymphocytes in chickens, causing immunosuppression and increased susceptibility to secondary infections. IBDV remains an economically important pathogen worldwide due to direct morbidity/mortality and vaccine failures linked to virus evolution and immune suppression (Mundt 2003; van den Berg et al. 2000). The IBDV evolution has been characterized by diversification into classic, variant, very virulent (vvIBDV), antigenic variants, and reassortant strains; ongoing mutation, recombination, and genome segment reassortment drive antigenic drift and shifts that undermine vaccine protection (Islam et al. 2017; Jackwood 2012). In Egypt, multiple IBDV lineages co-circulate, including very virulent strains and reassortants; gaps in surveillance, vaccine matching, and biosecurity increase disease impact (Amer et al.,2007a, 2007b and 2008, Zohair et al., 2017, Ghetas et al., 2022a). IBDV contributes substantially to immunosuppression in Egyptian poultry, exacerbating losses from other pathogens and reducing vaccine efficacy against diseases such as Newcastle disease and avian influenza (Abdel-Alim et al. 2020; El Naggar et al. 2018). Also, it was reported that IBDV infection affects the intestinal mycobiota, and microbiota in chickens (Ghetas et al., 2022b, Mosa et al., 2024a and b). For the effective prevention and control of IBDV infection many vaccines are used in Egypt (Awad et al, 2023, Mosad et al., 2024). Surveillance and molecular characterization, use of properly matched vaccines (including homologous, immune-complex, or vector vaccines where appropriate), strict biosecurity, and monitoring of immunosuppression consequences in flock health management (Mahgoup et a., 2012, Ghetas et al., 2022, Ramon et al. 2022).

Virology and pathogenesis

Agent and genome: IBDV is a non-enveloped, bi-segmented double-stranded RNA virus (segments A and B). Segment A encodes the polyprotein

(pVP2-mature VP2 is the major capsid protein and primary antigenic determinant-VP3, and VP4 protease); segment B encodes VP1, the RNA-dependent RNA polymerase. VP2's hypervariable region (HVR) largely determines antigenicity and virulence (Mundt 2003, Coulibaly et al. 2005).

Pathogenesis: IBDV selectively infects immature B cells in the bursa of Fabricius, causing B-cell depletion, bursal atrophy, and immune dysfunction. Clinical disease ranges from subclinical immunosuppression to acute, hemorrhagic disease with high mortality depending on strain virulence and host factors (age, maternal antibodies, concurrent infections) (van den Berg et al. 2000, Müller et al. 2003).

Immunosuppression: By destroying B lymphocyte populations and impairing humoral responses, IBDV decreases responsiveness to other vaccines and increases susceptibility to secondary bacterial and viral infections leading to production losses beyond direct IBD mortality (Hassan et al. 2013, Schat 2003).

Virus Evolution and epidemiology

Historical overview: Classic IBDV strains were first recognized in the 1950s; antigenic variants emerged in the 1980s in the United States; very virulent IBDV (vvIBDV) strains emerged in the late 1980s/early 1990s in Europe and spread globally, causing high mortality and production losses (Müller et al. 2003, Jackwood 2012).

Mechanisms generating diversity: High mutation rates, point mutations in the VP2 HVR, reassortment between genome segments A and B, and recombination contribute to the emergence of antigenic variants and novel pathogenic strains that can partially escape vaccine-induced immunity (Islam et al. 2017, Jackwood et al. 2018). Current global landscape: Today, multiple genogroups coexist. vvIBDV (genogroup 3/very virulent) remains widespread; antigenic variants (notably in the Americas and Asia) and reassortant strains continue to arise. Region specific genotypes and vaccine-escape mutants have been reported in Europe, Asia, Africa, and the Americas, complicating control (Mundt 2014, Islam et al. 2019).

Vaccine pressure and evolution: Widespread use of live attenuated and intermediate vaccines exerts selection pressure; incomplete immunity (due to maternal antibodies or inappropriate vaccine timing) can facilitate field virus replication in vaccinated flocks and selection of escape mutants (Jackwood 2012; van den Berg 2000).

Genetic changes to pathogenicity and immunosuppression

Very virulent IBDV (vvIBDV):

Genetic correlates: vvIBDV strains (first reported late 1980s/early 1990s) carry characteristic amino acid substitutions in VP2 HVR and distinct segment B lineages. These changes are associated with enhanced pathogenicity and mortality (Müller et al. 2003; Mundt 2014).

Phenotype: vvIBDV causes severe bursal destruction, profound B cell depletion, high mortality in young chickens, and profound immunosuppression in survivors (van den Berg et al. 2000).

Classical vs. antigenic variant strains

Genetic correlates: Variant strains possess mutations in neutralizing epitopes of VP2 that reduce recognition by antibodies elicited by classical vaccines (e.g., antigenic variants described in the US in the 1980s and later in Asia) (Jackwood 2012).

Phenotype: Reduced vaccine-induced neutralization and failure of classical vaccines to fully protect, leading to subclinical immunosuppression and production losses (Mundt 2003).

Reassortant strains in field outbreaks

Example: Several field reports describe reassortants combining a vvIBDV-like segment A with a different segment B (or vice versa). Such reassortants can show altered virulence or replication kinetics compared with parental strains (Jackwood et al. 2018; Islam et al. 2019).

Phenotype: Some reassortants retain high pathogenicity and immunosuppressive capacity; others exhibit attenuated or modified clinical signs but still cause immunosuppression sufficient to reduce vaccine responses to other pathogens.

Specific amino acid substitutions and functions

Example residues: Substitutions at VP2 positions (such as 222, 256, 279, 284, 330 numbering depends on alignment) have been implicated in antigenic change and virulence modulation (Coulibaly et al. 2005; Mundt 2003).

Phenotype: Single or multiple residue changes in HVR can reduce binding by neutralizing antibodies, permitting viral replication in vaccinated birds and causing immunosuppression without classic hemorrhagic disease.

The current situation in Egypt

Circulating lineages: Multiple reports over the last decade indicate co-circulation in Egypt of vvIBDV strains, classic strains, antigenic variants, and reassortants. Molecular studies show mutations in the VP2 HVR and occasional reassortment between segments A and B, producing strains with altered antigenicity and pathogenicity (Abdel-Alim et al. 2020; Hassan et al. 2015).

Outbreak pattern and control challenges: Egypt's dense poultry industry, mixed farm types (backyard and commercial), variable vaccination practices, and limited coordinated surveillance contribute to persistent IBD outbreaks. Vaccine failures have been reported and attributed to mismatch between vaccine strains and circulating viruses, improper vaccine handling/administration, and interference by maternal antibodies (El Naggar et al. 2018, Elbestawy et al. 2019).

Economic and production impact: IBD-related immunosuppression increases mortality from secondary infections, reduces growth and feed conversion in broilers, and impairs antibody responses to other vaccines in layers and breeders—causing egg production losses and reduced flock value (Abd El Rahman et al. 2017, Abdel-Alim et al. 2020).

Surveillance and diagnostics in Egypt: Diagnostic capacity includes virus isolation, RT-PCR and sequencing of VP2, and histopathology. However, systematic nation-wide molecular surveillance is limited; published studies are often regional and episodic, making it hard to track spread and evolution comprehensively (Hassan et al. 2015, El Naggar et al. 2018).

IBDV immunosuppression, mechanisms and consequences

Mechanisms of immunosuppression: Destruction of immature B cells, bursal atrophy, decreased B-cell repertoire and antibody production; possible effects on T-cell function and innate responses have also been described. Immunosuppression is dose-, age-, and strain-dependent (van den Berg et al. 2000, Schat 2003).

Consequences for poultry health: Increased susceptibility to bacterial pathogens (E. coli, Salmonella), viral infections (Newcastle disease virus, infectious bronchitis virus, avian influenza), and parasitic burdens; reduced vaccine responses lead to outbreaks of other vaccine-preventable diseases. The combined effect increases mortality, morbidity, and production losses and raises antimicrobial usage (Hassan et al. 2013, Müller et al. 2012).

Examples from Egypt: Studies report concomitant infections and higher bacterial septicemia in flocks with IBDV; vaccination programs against other diseases show reduced seroconversion when IBDV-induced immunosuppression occurs, aggravating endemic disease burdens in Egypt's flocks (El Naggar et al. 2018, Abdel-Alim et al. 2020).

Control strategies:

Surveillance and molecular characterization: Strengthen coordinated, continuous surveillance with routine sequencing (VP2 HVR and whole genomes where possible) to detect emergent strains and reassortants and to inform vaccine strain selection (Mundt 2014, Islam et al. 2019).

Vaccination policy: Use vaccines matched as closely as possible to circulating strains. Consider the strategic use of inactivated, recombinant (vector), immune complex, or properly attenuated live vaccines depending on flock type, maternal antibody levels, and local strain antigenicity. Regular evaluation of vaccine efficacy with challenge and serological studies is essential (Jackwood 2012, van den Berg 2000).

Biosecurity and management: Improve farm biosecurity to limit introduction/spread, segregate age groups, control movement of people/equipment, and manage environmental stressors that exacerbate disease. Educate farmers on vaccine handling and timing to avoid interference by maternal antibodies (Müller et al., 2003).

Addressing immunosuppression: Monitor flocks for bursal health and antibody response profiles; implement measures to prevent and control secondary infections (e.g., targeted antimicrobial stewardship, improved hygiene) and adjust vaccination schedules for other diseases following recovery from IBD outbreaks (Schat, 2003, Hassan et al., 2013).

Policy and coordination: National veterinary authorities should coordinate surveillance data sharing, update official vaccine recommendations, and support diagnostic capacity building to respond to evolving IBDV strains-especially in countries with high poultry density such as Egypt.

Conclusions

IBDV remains a dynamically evolving pathogen whose antigenic and genetic diversification driven by mutation, recombination, and reassortment complicates control worldwide. In Egypt, co-circulation of vvIBDV and variant/reassortant strains, variable vaccine matching and coverage, and management challenges sustain the virus's impact. The virus's capacity to induce immunosuppression amplifies its threat by undermining responses to other vaccines and increasing susceptibility to secondary infections, leading to substantial economic losses. Integrated strategies—enhanced molecular surveillance, vaccine matching and proper vaccination practices, improved biosecurity, and monitoring for immunosuppression are needed to reduce the threat to poultry production.

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Authors' contributions

Mohamed. M. Amer collected data, wrote the manuscript, and revised the original draft. The authors read and approved the final manuscript.

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Competing interests

The authors declare that they have no competing interests.

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