**Infectious bursal disease (IBD)** is a highly contagious disease of young chickens caused by IBD virus (IBDV) belonging to the *Birnaviridae* family (1). Two serotypes have been recognized to naturally infect chickens. In the pathogenic serotype I, the large genomic segment A encodes for four viral proteins, the two capsid proteins VP2 (48 kDa) and VP3 (32 to 35 kDa), the viral protease VP4 (24 kDa), and a nonstructural protein VP5 (17 to 21 kDa). The smaller segment B encodes for the RNA-dependent RNA polymerase VP1 protein (94 kDa) (2). Coinfection with very virulent IBDV (vvIBDV) and attenuated IBDV strains has led to the exchange of double-stranded genomic RNA segments to generate new reassortment viruses (3–5).

The BGE14/ABT1/MVC/India strain was isolated from four-week-old commercial broiler flocks in India. This isolate caused immunosuppression, low weight gain, and bursal atrophy with high mortality. The presence of IBDV was diagnosed by agar gel immunodiffusion using specific hyperimmune serum and reverse transcriptase (RT)-PCR by amplifying the VP2 hypervariable region (6). The complete genome sequencing was carried out by RT-PCR using overlapping consensus primers and Sanger’s dideoxy sequencing in both directions by M/s Shrimpex Biotech, Chennai, India. Sequences were compiled and edited using the SeqMan program (Lasergene). Multiple sequence alignments were performed with MEGA5 (7), and a phylogenetic tree was constructed using PhyML.

The complete genome of segment A showed the highest nucleotide similarity (99.4%) with vvIBDV strain SH-99 (LM651365). In segment B, the highest similarity (99.9%) was found with an attenuated strain D78 (AF499930). The phylogenetic analysis indicated that segment A formed a cluster with vvIBDV strains, while segment B formed a cluster with attenuated and classical virulent reference strains. The results revealed that segment A of BGE14/ABT1/MVC/India was derived from a vvIBDV strain and that segment B was derived from an attenuated strain (8). There were three unique amino acid (aa) substitutions (L74I, P125S, and W133R) in VP5 compared to other vvIBDV isolates. The difference observed in the polycationic C-terminal region (aa residues 132 to 143) might be responsible for the variation in virulence and adaptability (9).

Analysis of the VP2 sequence of IBDV showed that there are four unique aa residues—222(A), 242(I), 256(I), and 299(S)—for the vvIBDV strains. Isoleucine was substituted by valine at residue 294(I-V). The serine-rich heptapeptide positions from aa 326 to aa 332 (SWASG5) were found conserved, indicating that this isolate belonged to vvIBDV subtypes (10). The VP1 protein of the vvIBDV strains contains 15 characteristic amino acid residues (11). In contrast, the BGE14/ABT1/MVC/India strain carried the differences in all of the amino acid residues (V4I, K13T, I61V, T145N, D146E, N147G, E242D, A287T, M390L, D393E, K508R, S511R, P562S, P687S, and P695K), which made it identical to most of the classical, variant, and attenuated IBDV strains. These data are useful for analyses of epidemiology and evolutionary characteristics of IBDV in India.

**Nucleotide sequence accession numbers.** The full-length sequence of the BGE14/ABT1/MVC/India isolate has been deposited in GenBank with accession numbers KT884452 (segment A) and KT884453 (segment B).

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REFERENCES
1. Müller H. 1986. Replication of infectious bursal disease virus in lymphoid cells. Arch Virol 87:191–203. http://dx.doi.org/10.1007/BF01315299.
2. Jackwood DJ, Saif YM, Moorhead PD. 1985. Immunogenicity and antigenicity of infectious bursal disease virus serotypes 1 and 2 in chickens. Avian Dis 29:1184–1194.
3. Hon CC, Lam TY, Drummond A, Rambaut A, Lee YF, Yip CW, Zeng F, Lam PY, Ng PT, Leung FC. 2006. Phylogenetic analysis reveals a correlation between the expansion of very virulent infectious bursal disease virus and reassortment of its genome segment B. J Virol 80:8503–8509. http://dx.doi.org/10.1128/JVI.00585-06.
4. Le Nouën C, Rivallan G, Toquin D, Darlu P, Morin Y, Beven V, De Boisseson C, Cazaban C, Comte S, Gardin Y, Eterradossi N. 2006. Very virulent infectious bursal disease virus: reduced pathogenicity in a rare natural segment-B-reassorted isolate. J Virol 80:209–216. http://dx.doi.org/10.1099/vir.0.81184-0.
5. Wei Y, Li J, Zheng J, Xu H, Li L, Yu L. 2006. Genetic reassortment of infectious bursal disease virus in nature. Biochem Biophys Res Commun 350:277–287. http://dx.doi.org/10.1016/j.bbrc.2006.09.040.
6. Jackwood DJ, Sommer-Wagner SE. 2005. Molecular epidemiology of infectious bursal disease viruses: distribution and genetic analysis of newly emerging viruses in the United States. Avian Dis 49:220–226. http://dx.doi.org/10.1637/7289-101404R.
7. Tamura K, Peterson D, Peterson N, Stecher G, Nei M, Kumar S. 2011. MEGAS: molecular evolutionary genetics analysis using maximum likelihood, evolutionary distance, and maximum parsimony methods. Mol Biol Evol 28:2731–2739. http://dx.doi.org/10.1093/molbev/msr121.
8. Wei YW, Yu XP, Zheng JT, Chu WY, Xu H, Yu XM, Yu L. 2008. Reassortant infectious bursal disease virus isolated in China. Virus Res 131:279–282. http://dx.doi.org/10.1016/j.virusres.2007.08.013.
9. Hernández M, Villegas P, Hernández D, Banda A, Maya L, Romero V, Tomás G, Pérez R. 2010. Sequence variability and evolution of the terminal overlapping VP5 gene of the infectious bursal disease virus. Virus Genes 41:59–66. http://dx.doi.org/10.1007/s11262-010-0485-4.
10. Eterradossi N, Arnauld C, Toquin D, Rivallan G. 1998. Critical amino acid changes in VP2 variable domain are associated with typical and atypical antigenicity in very virulent infectious bursal disease viruses. Arch Virol 143:1627–1636. http://dx.doi.org/10.1007/s007050050404.
11. Ren X, Xue C, Zhang Y, Chen F, Cao Y. 2009. Genomic analysis of one Chinese strain YS07 of infectious bursal disease virus reveals unique genetic diversity. Virus Genes 39:246–248. http://dx.doi.org/10.1007/s11262-009-0379-5.