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Chromosomal Translocations Fusing the BCL6 Gene to Different Partner Loci Are Recurrent in Primary Central Nervous System Lymphoma and May Be Associated With Aberrant Somatic Hypermutation or Defective Class Switch Recombination.

Original Articles

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Schwandt, Heinrich MD; Akasaka, Takashi PhD; Zuhke-Jenisch, Reina; Hans, Volkmar MD; Schaller, Carlo MD; Klapper, Wolfram MD; Dyer, Martin J. S. MA, DPhil, FRCP, FRCP Path; Siebert, Reiner MD; Deckert, Martina MD

Abstract:

Primary central nervous system lymphomas (PCNSLs) are diffuse large B cell lymphomas confined to the brain. Only minimal data exist on chromosomal aberrations underlying PCNSLs. We studied 41 PCNSLs by fluorescence in situ hybridization for breakpoints affecting the BCL6 locus in chromosomal band 3q27. Of 37 cases evaluable, 14 (38%) carried a breakpoint in the BCL6 locus. Two of these showed juxtaposition of BCL6 to the IGH locus. In 4 cases, the BCL6 breakpoints were cloned using long-distance inverse polymerase chain reaction. All breakpoints were located within the BCL6 major translocation cluster. The translocation partners were the IGH gene in 14q32.33, the IGL gene in 22q11.22, and the histone 1 H4I gene in 6p22.1. In the fourth case, a deletion in 3q leads to loss of an 837-kb fragment extending from the first intron of BCL6 to the third intron of the lipoma-preferred partner (LPP) gene. This deletion may bring the BCL6 gene under the control of regulatory elements of the LPP gene or the miRNA-28 gene located in intron 4 of LPP. DNA sequence analysis of the junctional sequences provided evidence that aberrant class switch recombination or somatic hypermutation may be involved in the generation of BCL6 translocations.

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