

Expression analysis of caspase-6, caspase-9 and BNIP3 in prostate cancer

Nam Jin Yoo¹, Min Seob Kim¹, Sang Wook Park¹, Seong Il Seo², Sang Yong Song³, Ji Youl Lee⁴, and Sug Hyung Lee¹

¹Departments of ¹Pathology and ⁴Urology, College of Medicine, The Catholic University of Korea, Seoul; Departments of ²Urology and ³Pathology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

ABSTRACT

Aims. Altered regulation of cell death is a feature of human cancer. The aim of this study was to explore whether the expression of the proapoptotic proteins caspase-6, caspase-9, and Bcl-2/adenovirus E1B-19kDa-interacting protein3 (BNIP3) is altered in prostate cancers.

Methods. We analyzed the expression of caspase-6, caspase-9, and BNIP3 in 107 prostate adenocarcinoma tissues by immunohistochemistry using a tissue microarray (TMA) method.

Results. Normal glandular cells expressed caspase-6 and BNIP3 proteins in 10 (9.3%) and 9 (8.4%) prostate tissues, respectively. By contrast, the prostate cancers expressed caspase-6 and BNIP3 in 65 (60.7%) and 69 (64.5%) cases, respectively. Prostate intraepithelial neoplasia (PIN) showed caspase-6 and BNIP3 expression in 65% and 65% of cases, respectively. We observed caspase-9 expression in 40 (37.4%) normal, 8 (40%) PIN, and 45 (42.1%) cancer tissues. None of the expression of caspase-6, caspase-9 or BNIP3 was associated with pathological characteristics such as tumor size, patient age, Gleason score, or tumor stage.

Conclusion. Our data showed that prostate cancer and PIN cells display higher expression of the proapoptotic proteins caspase-6 and BNIP3 than normal cells. Ne-expression of these proteins from the PIN stage suggests that apoptosis deregulation might occur in the early stage of prostate carcinogenesis, and that altered expression of proapoptotic proteins may be a feature of prostate cancer. **Free full text available at www.tumorionline.it**

Key words: caspase-6, caspase-9, BNIP3, prostate cancer, immunohistochemistry.

Acknowledgments: This study was supported by a grant from the Ministry for Health, Welfare and Family Affairs (A084012). The TMA sections were supplied by the Prostate Bank in Korea supported by the Korea Science and Engineering Foundation.

Correspondence to: Sug Hyung Lee, Department of Pathology, College of Medicine, The Catholic University of Korea, 505 Banpo-dong, Socho-gu, Seoul 137-701, Korea.
Tel +82-2-2258-7311;
fax +82-2-2258-7765;
e-mail suhulee@catholic.ac.kr

Received March 2, 2009;
accepted May 14, 2009.