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Gemcitabine Chemotherapy Induces Phenotypic Alterations of Tumor Cells that Facilitate Antitumor T cell Responses in a Mouse Model of Oral Cancer

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Gemcitabine (GEM) is a pyrimidine nucleoside analogue that is a new chemotherapeutic agent used for treating various cancers. Because accumulating evidence indicates that GEM may activate host immune responses, its potential as an immune modulator in cancer chemotherapy has generated considerable interest. In the present study, we sought to identify the antitumor immune effects of GEM in a mouse oral cancer model using immunological analyses. GEM induced significant oral cancer-cell apoptosis in vitro, and in vivo GEM administration markedly attenuated established mouse tumor growth. In vivo GEM administration decreased the numbers of both myeloid-derived suppressor cells (MDSCs) and B cells in tumor-bearing mice and enhanced dendritic cell maturation. Moreover, GEM treatment upregulated tumor-cell surface expressions of several immune accessory molecules and adhesion molecules, including CD80, CD86, CD40, ICAM-1, VCAM-1, and P-selectin. Remarkably, these tumor cells augmented T-cell responses. These results suggest that GEM can induce host antitumor immune responses, which would facilitate antitumor effects in the treatment of oral cancer.

Biography

Tomihara received his Ph.D. degree in 2006 from Sapporo Medical University, working on immune gene therapy by adenovirus vector. He then moved to Cancer Therapy and Research Center (CTRC) at The University of Texas Health Science Center at San Antonio (UTHSCSA) to work with Dr. Shin as a post-doctoral fellow. He obtained an assistant professor position in 2013 in the Department of Oral and Maxillofacial Surgery Graduate School of Medicine and Pharmaceutical Sciences for Research, University of Toyama, where he started independent research on cancer immunology.