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Hot Topics in This Issue:

Thyroid Tumor Carcinogenesis

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This issue of Journal of Basic & Clinical Medicine publishes manuscripts from a symposium; "Molecular carcinogenesis of thyroid neoplasia and theoretical interpretations" conducted by the 18th Annual Meeting of Japan Endocrine Pathology Society, being held in Tokyo, Japan on 1st of November, 2014 and two more papers were invited to enrich discussion and for better understanding by readers. These five papers focus on thyroid tumor carcinogenesis.

Kakudo M et al. introduced current knowledge of molecular aspects and genetic alterations of thyroid tumors from the multistep carcinogenesis theory (JBCM 2015; 4(1):13-21). However, there are several issues in thyroid carcinogenesis, which do not fit to the multistep carcinogenesis theory. These problems are discussed by Takano et al. from a view point of fetal cell carcinogenesis in last issue (JBCM 2014; 3(1):6-11). The fetal cell carcinogenesis theory can explain heterogeneous nature of tumors from benign to highly aggressive carcinomas, different from linear model of the multistep carcinogenesis theory. Another problem in adapting thyroid neoplasms in the multistep carcinogenesis is due to the lack of known precursor lesions between normal follicular cells and early well differentiated thyroid carcinomas. All thyroid tumors in WHO classification are coded either /0 (benign tumors) or /3 (malignant tumors) by SNOMED (Systematized Nomenclature of Medicine) and there are no tumors which are coded as /1 (borderline or uncertain) or /2 (*in situ* carcinomas). Follicular adenoma is a candidate of precursor lesions for thyroid carcinomas in many reviews and text books, while follicular adenoma is not regarded as a high-risk precancerous lesion of follicular or papillary thyroid carcinomas due to the infrequent progression from follicular adenoma to thyroid carcinomas. Possible precursor lesions of thyroid carcinomas are reviewed in this issue by Kakudo K et al., which include extremely low-grade thyroid carcinomas currently labeled as malignant tumors (JBCM 2015, 4(1):2-7). It is important for pathologists to differentiate biologically benign, borderline and low-risk thyroid carcinomas from biologically malignant high-risk thyroid carcinomas. These genuine carcinomas that develop through progression of precancerous lesions are summarized by Kakudo K as moderate-risk and high-risk thyroid carcinomas in last issue (JBCM 2014, 3(1):12-17).

One more important theory for carcinogenesis is the cancer stem cell theory reviewed by Nakashima et al. (JBCM 2015, 4(1):8-12). Cancer stem cells were first identified in acute myeloid leukemia and the thyroid cancer stem cells remain to be confirmed. Accumulating evidence supports that thyroid cancer is initiating by tumor-initiating cells commonly known as cancer stem cells. This hypothesis also provides an explanation not only for heterogeneity of thyroid tumors but also for why radiation exposure in childhood (younger than 5 years old) leads to them at high-risk for developing thyroid carcinoma.

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