MOLECULAR PATHOGENETIC MECHANISMS IN THYROID FOLLICULAR CELL NEOPLASM – A LITERATURE REVIEW
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ABSTRACT

Background: Thyroid carcinoma is the most common malignancy of the endocrine organs. Most of this malignant tumor derived from thyroid follicular cell and had well differentiation. Various signaling pathways known have a role in the development of well differentiated thyroid carcinoma to poorly and undifferentiated carcinoma. This literature review was aimed to understand the molecular genetic effect on thyroid follicular cell carcinogenesis, so it could be useful in developing new targeting therapeutic strategies.

Methods: This article was based on literature review of various books, journals, and recent research. The effect of various molecular aspects were studied on the pathogenesis of thyroid follicular cell neoplasm.

Results: The MAPK/ERK (mitogen-activated protein kinase/extracellular signal-regulated kinase signaling pathway) and PI3K/Akt (lipid kinase phosphoinositide-3-kinase signaling pathway) played an important role in transmission of cell signals through transduction systems as ligands, transmembrane receptors, and cytoplasmic secondary messengers to cell nucleus, where they influence the expression of genes that regulate important cellular processes, include: cell growth, proliferation, and apoptosis. The genes, coding the signaling cascade proteins (\textit{RET}, \textit{RAS}, \textit{BRAF}, \textit{PI3K}, \textit{PTEN}, \textit{AKT}), are mutated or aberrantly expressed in thyroid cancer derived from follicular cell. Genetic and epigenetic alternations, concerning MAPK/ERK and PI3K/Akt signaling pathways, in consequence of malignant follicular thyroid cell transformation. Further, \textit{TP53} dan \(\beta\)-catenin mutation affected the development of well differentiated thyroid carcinoma to poorly and undifferentiated thyroid carcinoma.

Conclusion: The understanding of this molecular pathogenetic mechanism could be provide powerful ancillary diagnostic tools, and also could be used to access novel molecular targeting therapeutic strategies.

Keywords: molecular pathogenetic, follicular cell, thyroid neoplasm