Role of PI3K/AKT/mTOR pathway as a therapeutic target in estrogen receptor positive breast cancer

Background and aim: The activation of phosphoinositide 3 kinase (PI3K)/Akt/mammalian target of rapamycin (mTOR) (PAM) pathway has been reported in the breast cancer, among which PI3KCA is the most significant gene prevalently mutated in estrogen receptor (ER)-positive breast cancer. Human tumor samples have shown lower ER levels of the PAM signaling pathway, meaning the endocrine resistance. Materials and methods: The articles searched on PubMed database in English was based on the keywords of ER-positive breast cancer, PI3K/AKT/mTOR pathway, Mammalian target of rapamycin, Endocrine resistance and Endocrine therapy. Results: The inhibition of PAM pathway enhanced significantly the expression of ER gene and ER-inducible target genes, and elevated sensitivity to tamoxifen treatment in ER-positive breast cancer cell lines. The PAM pathway as an important intracellular signaling system can cause the cell growth and vitality. Highly activated pathway can affect the tumorigenesis of ER-positive breast cancer and the endocrine therapy resistance. Based on preclinical and clinical trials, the -inhibition of PAM pathway can be effective in the endocrine therapy from the first line setting and beyond in the ER-positive breast cancer.Conclusion: According to the findings, it is pivotal to scrutinize the mechanism of PAM pathway signaling in maintaining the ER-positive breast cancers and starting the endocrine therapy resistance, whose inhibition can be effective to treat the ER-positive breast cancer, suggesting endocrine therapy coupled with PAM pathway inhibition.

ER-positive breast cancer; PI3K/AKT/mTOR pathway; Mammalian target of rapamycin; Endocrine resistance; Endocrine therapy.
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