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The expression of TAM and EPHA2 genes in breast cancer

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Objective: Receptor tyrosine kinases (RTKs) are involved in cell growth, motility, and differentiation. Deregulation of RTK signaling is associated with tumor development and therapy resistance. Potential RTKs such as TAM (TYRO3, AXL, MERTK), RON, EPH, and MET have been evaluated in many cancers such as lung, prostate, and colorectal, but less is known in breast tumors. In this study, 51 luminal breast cancer tissue and 8 triple-negative breast cancer (TNBC) subtypes were evaluated by qPfor the expression of TAM, RON, EPHA2, and MET genes. **Methodology:** The study was carried out with women assisted at Hospital Barão de Lucena – Recife (PE), enrolling 59 women, aged from 32 to 100 years, divided into two groups: 51 patients diagnosed with luminal breast cancer and 8 patients diagnosed with TNBC. This study was approved by the Research Ethics Committee of the Health Sciences Center of the Federal University of Pernambuco (CAAE: 47869315 0 00005208). **Results:** Among the 59 patients, 21 had luminal A-like, 30 had lumina B-like, and 8 had TNBC. The median age at diagnosis of breast cancer was 56 years, and patients were divided into four groups: 10.34% of patients 30–40 years; 22.41% of patients 41–50 years; 32.75% of patients 51–60 years, and 34.4% of patients over 60 years. Parity was reported by 89.83%, smoking habit was reported by 22.03%, and obesity was observed in 37.29% of patients. Luminal breast cancer was the most prevalent subtype, accounting for 86.44% of patients, of which 58.82% were luminal B-like. Regarding tumor staging, 11.86% of tumors were in stage I, 42.37% of tumors were in stage II, and 44.06% of tumors were in stage III. **Conclusion:** The TAM receptors show potential for targeting therapy once the expression of the three genes coding for the trimer is related to breast cancer subtypes and influenced by the patient's aging and habits, besides, the crosstalk between TAM and other RTKs like MET and EPH receptors.

Keywords: receptors tyrosine kinase; TNBC tumors; luminal patients; resistance; gene expression; target therapy.