PSGL-1 expression is associated with immunosuppression and aggressive clinicopathological parameters in breast cancer patients.

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Breast cancer is the most common malignancy in women, and remains the leading cause of cancer-related death. It is a complex and heterogeneous disease, with variable clinical, pathological and biological aspects. Immunotherapy targeting immune checkpoints represents a new therapeutic avenue that has shown promising results in a wide range of cancers. Nevertheless, in approximately 70% of cases, existing therapies prove ineffective, underscoring the need to investigate other pathways associated with immunosuppression within breast tumor microenvironment.

Objective: The aim of our work is to explore the expression patterns of PSGL-1 molecule in relation to clinicopathological parameters of the disease, and to analyze its association with genes of the mammary immune microenvironment in breast cancer patients, with a focus on the transcriptomic scale.

Materials and methods: We conducted a real-time quantitative PCR analysis on 52 biopsies, including tumor tissues and corresponding adjacent uninvaded tissues, taken from 26 Moroccan breast cancer patients, as well as a bioinformatics analysis using international transcriptomic datasets.

Results: Real-time quantitative PCR (qRT-PCR) revealed a significant increase in PSGL-1 expression in triple-negative breast cancer (TNBC) compared to luminal B molecular subtype. In addition, PSGL-1 expression showed a strong association with a profuse infiltration of protumoral immune cells, exhaustion markers, and molecules that inhibit the antitumor immune response. Furthermore, PSGL-1 expression showed a strong positive correlation with VISTA compared to other ligands of this pathway, namely IGSF11 and VSIG3.

Conclusion: All in all, our findings indicate that PSGL-1 may serve as a promising therapeutic target in breast cancer, encouraging further research into this molecule and its role in immunosuppression in this disease.

Key words: PSGL-1, breast cancer, immune checkpoint, immunosuppression, antitumoral immune response.