Predictive value of primary-tumor volume-based parameters on F-18 FDG PET/CT for axillary lymph node metastasis in invasive breast cancer

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Objectives: This study assessed whether volume-based parameters of primary-tumor measured by preoperative F-18 FDG PET/CT could have the predictive value for Axillary Lymph Node (ALN) metastasis prediction in invasive ductal breast cancer (IDC).

Methods: A total of 57 female patients (mean age, 49.4 ± 9.5) with IDC who underwent surgical resection of primary tumor with sentinel lymph node biopsy and/or axillary LN dissection without any neoadjuvant treatment were analyzed. Metabolically active tumor regions were delineated on pretreatment PET scans semi-automatically using custom software. Maximum standardized uptake value (SUVmax) and metabolic tumor volume (MTV) were used. To obtain the MTV, we used a threshold of SUV 2.0, 25%, 50% and 75% for the SUVmax.

Results: LN metastasis was found in 20 patients (35.1%). MTV-75% and MTV-50% were significantly higher in patient with LN metastasis (P = 0.005 and 0.015). However, SUVmax, MTV-2.0, and MTV-25% showed no significant difference. On receiver operating characteristic (ROC) curve analysis, MTV-75% (area under the curve (AUC) = 0.726, 95% confidence interval [95% confidence interval (CI)] 0.591–0.839) and MTV-50% (AUC = 0.696, 95% CI 0.560–0.811) showed good predictive performance for LN metastasis.

Conclusion: MTV-75% and MTV-50% as determined by F-18 PET/CT demonstrated a statistically significant correlation with LN metastasis in patients with IDC. Therefore, patients with high values of MTV on pretreatment F-18 FDG PET/CT should be cautiously evaluated for LN metastasis.

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Direct oncogene-targeted cancer killing and selective tumor Treg killing through the TNFR2 receptor via dominant antibody Antagonists

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A major barrier to cancer immunotherapy is the lack of selective inhibitors of the regulatory T cells (Tregs) of the cancer microenvironment. New methods to directly kill tumors through novel surface oncogenes are also desirable in this setting. Tumor Necrosis Factor Receptor 2 (TNFR2) is a target protein with restricted expression on the most potent Tregs of the tumor infiltrate and on human tumors as a newly discovered and broadly expressed human oncogene. We characterized the expression and the functional effects of newly created TNFR2 antibody antagonists on the tumor infiltrating Tregs of ovarian ascites compared to Tregs of peripheral blood from both patients and healthy controls. We also investigated if well known ovarian tumor cells lines express the TNFR2 oncogene and the effects of the TNFR2 antagonistic antibody on direct cancer killing. We found that TNFR2 antagonists inhibited Treg proliferation with exponential potency and selectivity for tumor microenvironment Tregs. Furthermore, common ovarian cancer cell lines such as OVCAR3 expressed the TNFR2 oncogene and were rapidly and completely killed by TNFR2 antagonistic antibodies, even at low concentrations. We conclude that dominant TNFR2 antagonists demonstrate tumor-specific Treg depletion with heightened specificity for the tumor microenvironment over the Tregs of peripheral blood. Blocking TNFR2 signaling with antagonist antibodies also creates a novel tool to directly eliminate ovarian tumors expressing the TNFR2 oncogene.

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