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Microvessel density recognized by Endoglin as prognostic markers in breast carcinoma

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Breast cancer is a leading malignancy in women and the second most common cause of women mortality from cancer, and its morbidity is on the rise year by year. Newly diagnosed cases of breast cancer in China have increased rapidly by 3% yearly in the past ten years. Hence, joint detection of tumor markers is of great importance in the early discovery and judgment on progression and prognosis of disease. Endoglin (CD-105) was proposed as a marker of neovascularization in solid malignancies. Endoglin (CD105) is an accessory receptor of transforming growth factor B. The highest synthesis, as well as expression, of endoglin has been found in vascular endothelial cells. The involvement of endoglin in angiogenesis and in angiogenesis-dependent processes has been observed. The aim of the present study was to evaluate the Microvessel density (MVD) recognized by Endoglin (CD105) expression in invasive ductal carcinomas (IDC) and precancerous lesions patients including ductal carcinoma in situ (DCIS) and atypical ductal hyperplasia (ADH), as well as to analyze the relationship between the MVD recognized by Endoglin expression with other standard prognostic parameters, such as size, grade, stage of the disease, metastases, and tumour recurrence. We analyzed the MVD recognized by CD105 and CD105 expression in IDC (n=128), DCIS (n=89) and ADH (n=57). Fifty-three cases of usual ductal hyperplasia (UDH) breast tissues were selected as a control group. MVD the peripheral area adjacent to the lesion and those central areas within the lesion in every group were also assessed. All cases were immuno stained for endoglin, estrogen receptor (ER) & progesterone receptor (PR). The specimens were evaluated for CD105. Positively stained microvessels were counted in densely vascular foci in a $\times 400$ field. Results were correlated with survival and other standard prognostic parameters, such as size, grade, stage of the disease, metastases, and patient survival. Our Results show the MVD based on CD105 expression had statistical significance in IDC (31.691 ± 8.621), DCIS (27.633 ± 6.879), ADH (21.436 ± 5.112) and UDH groups (10.038 ± 3.976), MVD the peripheral area adjacent to the lesion was significantly higher than those central area within the lesion in every group ($P < 0.01$ for each group). There were significantly differences in the mean MVD recognized on CD105 between estrogen receptor & progesterone receptor positive IDC group and negative group, histological grade (I+II) and grade III IDC groups, with lymph node metastasis, distant metastasis and recurrence groups ($P < 0.05$) and without groups ($P < 0.05$). However, there was not difference in the mean MVD recognized by Endoglin between age (≤ 50 yr vs > 50 yr), tumor diameter (≤ 2 cm vs > 2 cm) ($P > 0.05$). The MVD recognized by Endoglin might be important biological markers for invasion, lymph node metastasis, distant metastasis and tumour recurrence of IDC. The over-expression of endoglin expression may be useful as an indicator of breast cancer progression and helpful for estimation of recurrence and metastasis risk. The prediction of risks for metastasis and recurrence as well as recurrence patterns based on MVD after surgery could help the design of better follow-up programmes and appropriate treatment strategies for breast cancer patients.

IKBKE promotes breast cancer metastasis through regulation of Snail

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Metastasis is the most common cause of mortality in breast cancer. Epithelial-Mesenchymal Transition (EMT) and cancer stem cell are closely associated with breast cancer metastasis. Here we report that depletion of IKBKE markedly decreases invasion, EMT and cancer stem cell self-renewal as well as breast cancer growth and metastasis in MMTV-PyMT and orthotopic models. Furthermore, we demonstrated that IKBKE directly binds to and phosphorylates Snail. The phosphorylation led to stabilization and nuclear translocation of Snail, and increase of EMT and metastasis. Taken together, these findings indicate that IKBKE plays a pivotal role in metastasis and could be a critical therapeutic target for metastatic breast cancer.