

Letter

Addition of granulocyte-colony stimulating factor (G-CSF) may further increase chemosensitive state in premenopausal node-positive breast cancer patients with induced angiogenesis after surgery

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See related research article by Retsky *et al.*, <http://breast-cancer-research.com/content/6/4/R372>, and related commentary by Baum, <http://breast-cancer-research.com/content/6/4/160>

We read with great interest the article by Retsky and colleagues in this issue of *Breast Cancer Research* [1]. They hypothesize that there is induced angiogenesis after breast cancer surgery in approximately 20% of premenopausal node-positive patients, which stimulates division of dormant micrometastatic cells and synchronizes them into a highly chemosensitive state. Therefore, adjuvant chemotherapy works particularly well for that patient category. Breast cancer stem cells in the thousands of micrometastatic cancer cells have the capacity to repopulate and metastasize. Granulocyte-colony stimulating factor (G-CSF) stimulates the pluripotent stem cell and neutrophil precursors to divide and increase the production of mature neutrophils, thus enhancing the function of mature neutrophils, which ameliorates neutropenia and the associated complications. These breast cancer stem cells have not been totally characterized, and may carry antigens almost identical to those carried by hematopoietic stem cells [2]. We hypothesize that G-CSF use in premenopausal node-positive breast cancer patients, especially in the era of induced angiogenesis, may activate and repopulate these dormant breast cancer stem cells more efficiently as well as stimulating blood stem cells. Consequently, activated breast cancer stem cells become chemosensitive to cell cycle-specific various chemotherapeutic agents.

Competing interests

None declared.

References

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2. Altundag K, Altundag O, Elkiran ET, Cengiz M, Ozisik: **Addition of granulocyte-colony stimulating factor (G-CSF) to adjuvant treatment may increase survival in patients with operable breast cancer: interaction of G-CSF with dormant micrometastatic breast cancer cells.** *Med Hypotheses* 2004, in press.