



Future of Bevacizumab in Metastatic Breast Cancer

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Editorial

Bevacizumab is an interesting antitumor drug in clinical cancer medicine, its effect on survival of patients confirmed in metastatic colon cancer, renal cancer, lung cancer, ovarian cancer and glioblastoma but despite using with many combination chemotherapy in metastatic breast cancer with capecitabine, taxanes, gemcitabine, doxorubicin no effects on overall survival and progression free survival seen [1,2]. It is a humanized monoclonal antibody against vascular endothelial growth factor A and impairs its binding to vascular endothelial growth factor receptor type1 and type2 and leads to tumor shrinkages. The basic mechanism is that, antiangiogenesis treatment normalizes tumor flow initially resulting in important tissue oxygenation and decreased interstitial pressure with increasing the delivery of cytotoxic agents to tumor. They confer better efficacy in combination with chemotherapy during earlier treatment because late stage breast cancer expresses many different angiogenic factors such as fibroblast growth factors in contrast early cancer expresses more vascular endothelial growth factors. If in future the biomarker would be a clinical practice, especially circulating tumor cells, it is possible to select and predict the activity of the disease and targeted therapy with bevacizumab to be done on precise time of tumors with angiogenesis activity [3,4]. Probably, considering the breast cancer carcinogenesis starting with a very small size tumor activity, it is possible to control the tumor more efficiently by antiangiogenesis drugs, may be attempting for curability of breast cancer in next decades.

Because of its antiangiogenesis effects on central nervous system metastasis, in future it can be a preventive drug therapy delaying seeding of tumor in life threatening site of body which is difficult to control by oncologist. How can we accelerate the effectiveness of bevacizumab in metastatic breast cancer? It needs to have knowledge and control the reactivation of other mechanisms of tumor growth in breast cancer biology. Activation of epidermal growth factor pathway; Here 2 pathway, endocrine redundant and cyclooxygenase 2 pathways are the common mechanisms of failure [5-8].

Conclusion

Regarding the failure of survival effects of bevacizumab in metastatic breast cancer this is not the end of the drug in this disease entity, bevacizumab therapy in future still has a major role in metastatic breast cancer.

References

1. Adhemar Longatto Filho, Jose Manuel Lopes, Fernando C Schmitt. Angiogenesis and Breast Cancer. *J Oncol*. 2010;2010.
2. Marino N, Woditschka S, Reed LT, Nakayama J, Mayer M, Wetzel M, et al. Breast cancer metastasis: issues for the personalization of its prevention and treatment. *Am J Pathol*. 2013;183(4):1084-95.
3. Steeg PS. Tumor metastasis: mechanistic insights and clinical challenges. *Nat Med*. 2006;12(8):895-904.
4. Bryan P Sneider, Kathy D Miller. Angiogenesis of breast cancer. *J Clin Oncol*. 2005;23:8.
5. Nourani Khojasteh H. Lessons about antiangiogenesis treatment of breast cancer in the current era. *Med Res Archives*. 2017;5(2).
6. Lord S, Harris AL. Angiogenesis - still a worthwhile target for breast cancer therapy? *Breast Cancer Res*. 2010;12(4):S19.
7. Fox SB, Generali DG, Harris AL. Breast tumour angiogenesis. *Breast Cancer Res*. 2007;9(6):216.
8. Castañeda-Gill JM, Vishwanatha JK. Antiangiogenic mechanisms and factors in breast cancer treatment. *J Carcinog*. 2016;15:1.

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