Mechanism of Angiogenesis Signaling and Anti-VEGF Therapy in Neuroblastoma

Mohammad Amin Dehghani - Student Research Committee of Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran
Fatemehe Dehghani - Department of Genetics, Faculty of Medicine, Shiraz University of Medical Sciences, Shiraz, Iran
Maryam Shirani - Student Research Committee of Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran
Seyyed Hossein Hassanpour - Young Researchers and Elite Club, Yasooj Branch, Islamic Azad University, Yasooj, Iran

Neuroblastoma is the most common pediatric extracranial and heterogeneous solid tumor in children. Owing to intense research efforts in NB therapy, including the application of multimodal therapeutic strategies, the survival rate has markedly improved in patients with low- and intermediate-stage NB. Angiogenesis is the process of the development of the intrinsic vascular network, and it is an essential event in the progression and metastatic spread of solid tumors such as neuroblastoma, where new capillaries spread from preexisting vessels and the transition from avascular to vascular phase occurs via neovascularization. Malignant neuroblastoma is a highly vascularized solid tumor that requires access to blood vessels for growth, invasion and metastasis, being highly dependent on angiogenesis. VEGF signaling plays a regulatory role in neuroblastoma angiogenesis via a paracrine mechanism through two specific tyrosine kinase VEGF receptors, -VEGFR-1 and VEGFR-2, located at the surface of endothelial cells. Therefore, antiangiogenic strategies can be effective in inhibiting tumor cell dissemination and metastasis in highly vascular neuroblastoma. In general for children having neuroblastoma, the anti-angiogenic therapy works as a boon when administered along with gene therapy. Treatment of neuroblastoma in both in vitro and in vivo models with an anti-VEGF agent results in antitumor activity with a decrease in both tumor vascularity and in the number of intratumoral vessels and a decrease in VEGF expression suggesting a diversified role of VEGF in the progression of advanced stage neuroblastoma.

Keywords:
Neuroblastoma; Angiogenesis; Anti-angiogenic therapies; VEGF; Anti VEGF.
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