Vasculogenic mimicry

From:
http://www.sciencemag.org/sites/default/files/images/60617NF_vascularMimicry_web%20%282%29.jpg

Vasculogenic mimicry is the formation of microvascular channels by aggressive, metastatic and genetically deregulated tumour cells [1) 2)].

Treatments targeting VM are lacking due to the poor understanding of the molecular mechanism involved in VM formation. By analyzing the TCGA database, microRNA-29a-3p (miR-29a-3p) was found to be highly expressed in normal brain tissue compared with glioma. An in vitro study revealed an inhibitory role for miR-29a-3p in glioma cell migration and VM formation, and the further study confirmed that ROBO1 is a direct target of miR-29a-3p. Based on this, we engineered human mesenchymal stem cells (MSCs) to produce miR-29a-3p-overexpressing exosomes. Treatment with these exosomes attenuated migration and VM formation in glioma cells. Moreover, the anti-glioma role of miR-29a-3p and miR-29a-3p-overexpressing exosomes were confirmed in vivo. Overall, the present study demonstrates that MSCs can be used to produce miR-29a-3p-overexpressing exosomes, which have great potential for anti-VM therapy and may act as supplements to anti-angiogenetic therapy in the clinic [3)].

Vasculogenic mimicry (VM) has been observed in melanoma and in some nonmelanoma tumor types.

45 astrocytomas (including World Health Organization grade II 15 cases, grade III 15 cases, and grade IV 15 cases) by CD34 endothelial marker periodic acid-Schiff (PAS) dual staining to see if VM existing in these tumors. The results demonstrated that endothelium-lined vessels dominated the tumor microvasculature and stained positively for PAS, laminin, and endothelial marker. PAS-positive pattern of VM was found in two grade IV astrocytomas. Channels stained positively for PAS, laminin, and negatively for CD34 of the VM entrapped in the tumor tissue. Erythrocytes could be observed in some of these channels. In these networks of PAS-positive pattern, spots of weak reaction for CD34 were observed, suggesting the incorporation of VM channel and normal vessel. Furthermore, in astrocytoma, especially glioblastoma, focus of anaplastic tumor cells appeared with CD34 expression, whereas some tumor cells lost glial fibrillary acid protein expression. It is assumed that genetically deregulated tumor cells in astrocytoma could lose the astrocyte-specific protein and express inappropriate markers not expected in cells of astrocyte lineage. The present results suggest that VM phenomenon exists in some malignant astrocytoma [4)].

Studies have shown that, under hypoxic conditions, glioblastoma (Glioblastoma) cells display the ability to drive blood-perfused vasculogenic mimicry (VM).

Vasculogenic mimicry (VM) was an important tumor blood supply to complement the endothelial cell-dependent angiogenesis, while leptin and receptor (ObR) involved in angiogenesis in glioblastoma has

been reported on previous study, but the relationship between ObR expression and VM formation in human glioblastoma tissues, as well as their prognostic significance still remains unclear.

Vasculogenic mimicry (VM) has been reported to be a novel glioma neovascularization process. Anti-VM therapy provides new insight into glioma clinical management.

Ling et al., assessed whether the Transforming growth factor beta (TGF-β-induced EMT) contributed to vasculogenic mimicry (VM) formation in glioma, they established an SHG44 cell line stably transfected with TGF-β cDNA loaded lentivirus. SB203580 was employed to inhibit the TGF-β-induced EMT. The results showed that the VM forming ability of cells could be improved by TGF-β over-expression. The migration and invasion capabilities of cells were also enhanced due to EMT. SB203580 was able to weaken cell migration, invasion and VM forming abilities via blocking p38/MAPK signaling pathways, but it had tiny influence on MMP/LAMC2 chain. Consequently, we concluded that EMT inhibition via p38/MAPK signaling pathways would partly impair TGF-β-induced VM formation in glioma [5].

1)
Folberg, R; Hendrix, MJ; Maniotis, AJ (February 2000). “Vasculogenic mimicry and tumor angiogenesis”. The American Journal of Pathology. 156 (2): 361–81. doi:10.1016/s0002-9440(10)64739-6. PMC 1850026 Freely accessible. PMID 10666364.
2)
Folberg, R; Maniotis, AJ (July–August 2004). “Vasculogenic mimicry”. APMIS : acta pathologica, microbiologica, et immunologica Scandinavica. 112 (7–8): 508–25. doi:10.1111/j.1600-0463.2004.apm11207-0810.x. PMID 15563313.
3)
Zhang Z, Guo X, Guo X, Yu R, Qian M, Wang S, Gao X, Qiu W, Guo Q, Xu J, Chen Z, Wang H, Qi Y, Zhao R, Xue H, Li G. MicroRNA-29a-3p delivery via exosomes derived from engineered human mesenchymal stem cells exerts tumour suppressive effects by inhibiting migration and vasculogenic mimicry in glioma. Aging (Albany NY). 2021 Feb 1;12. doi: 10.18632/aging.202424. Epub ahead of print. PMID: 33535172.
4)
Yue WY, Chen ZP. Does vasculogenic mimicry exist in astrocytoma? J Histochem Cytochem. 2005 Aug;53(8):997-1002. Epub 2005 May 27. PubMed PMID: 15923371.
5)
Ling G, Ji Q, Ye W, Ma D, Wang Y. Epithelial-mesenchymal transition regulated by p38/MAPK signaling pathways participates in vasculogenic mimicry formation in SHG44 cells transfected with TGF-β cDNA loaded lentivirus in vitro and in vivo. Int J Oncol. 2016 Oct 7. doi: 10.3892/ijo.2016.3724. PubMed PMID: 27748800.