miR 206

Early brain injury (EBI) is the most important potentially treatable cause of mortality and morbidity following subarachnoid hemorrhage (SAH). Apoptosis is one of the main pathologies of SAH-induced EBI. Numerous studies suggest that human umbilical cord derived mesenchymal stem cells (hucMSCs) may exert a neuroprotective effect through exosomes instead of transdifferentiation. In addition, microRNA-206 (miR-206) targets BDNF and plays a critical role in brain injury diseases. However, the therapeutic effect of miR-206 modified exosomes on EBI after SAH and its regulatory mechanism have not been elucidated. To identify whether hucMSCs-derived miR-206-knockdown exosomes have a better neuroprotective effect, Zhao et al. established SAH rat model and treated with the exosomes to research the mechanism of miR-206 in EBI after SAH. They found that treatment with hucMSCs-derived miR-206-knockdown exosomes has a greater neuroprotective effect on SAH-induced EBI compared to treatment with simple exosomes. The miR-206-knockdown exosomes could significantly improve neurological deficit, brain edema and suppress neuronal apoptosis by targeting BDNF. Moreover, the BDNF/TrkB/CREB pathway was activated following treatment with miR-206 modified exosomes in vivo. In summary, these findings indicate that the hucMSCs-derived miR-206-knockdown exosomes prevent early brain injury by inhibiting apoptosis via BDNF/TrkB/CREB signaling. This may serve as a novel therapeutic target for the treatment of SAH-induced EBI [1)].

1)
Zhao H, Li Y, Chen L, Shen C, Xiao Z, Xu R, Wang J, Luo Y. HucMSCs-Derived miR-206-Knockdown Exosomes Contribute to Neuroprotection in Subarachnoid Hemorrhage Induced Early Brain Injury by Targeting BDNF. Neuroscience. 2019 Aug 7. pii: S0306-4522(19)30542-1. doi: 10.1016/j.neuroscience.2019.07.051. [Epub ahead of print] PubMed PMID: 31400488.