Poster | Disorders of Nervous Systems and Treatment

[2P]Cerebrovascular Disease and Ischemia

Thu. Jul 30, 2020 1:30 PM - 3:30 PM Poster Session
*Videos are available throughout the meeting period.

[2P-198]Investigation of glial cells by chronic cerebral hypoperfusion in B-cell translocation gene 2 (BTG2) knockout mice

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An analysis of RNA-Seq revealed that B-cell translocation gene 2 (Btg2) is up-regulated in old amyloid precursor protein transgenic mice with diabetes (APP; *ob/ob*). Btg2 is one of the BTG/TOB family genes and reported to have tumor suppressive function in cancer cells. Btg2 is also reported to be up-regulated in white matter lesion, induced by cerebral hypoperfusion in mouse brain (Ohtomo et al., 2018). It is well known that both diabetes and Alzheimer's disease (AD) cause white matter lesions. Thus, we generated Btg2 knockout (KO) mice, treated them with bilateral common carotid artery stenosis (BCAS) and examined behavioral phenotypes, severity of white matter lesions, and brain inflammation.
We performed two behavioral tests, open-field test and Morris water maze test in BCAS/sham-operated wild type and Btg2 KO mice. In the open-field test, total distance travelled and average speed of BCAS-treated Btg2 KO mice were significantly increased than BCAS-treated wild type mice. In the Morris water maze test, escape latency was tended to be longer in the BCAS-treated Btg2 KO mice compared to other groups at final trial test (5th day), though significant difference was not observed.
White matter lesion was assessed at the corpus callosum and optic tract by the method previously developed by Wakita et al. (Acta Neuropathol., 1994). Degeneration of myelin fiber and vacuolation were increased in the white matter of BCAS-treated mice compared to sham-operated mice, though the severity was not significantly different between wild type and Btg2 KO mice. Immunohistochemical analysis was performed using antibodies against GFAP, Iba-1 and Mac2 (Galectin-3) to examined the astrocytes and microglial cells. In the white matter of BCAS-treated mice, immunoreactivities of GFAP and Mac2 were higher than sham-operated mice. In hypoperfusion area in the cortex of BCAS-treated mice, Mac-2 immunoreactivity was increased and some of Mac2-positive cells were immunoreactive with anti-Btg2 antibody. Deletion of Btg2 may affect behavioral phenotype in chronic cerebral hypoperfusion model, though its role in glial cells remains to be further investigated.