11th World Congress and Expo on
Cell & Stem Cell Research
March 25-26, 2019 | Orlando, USA

ACCEPTED ABSTRACTS JOURNAL OF CELL SCIENCE & THERAPY, VOLUME: 10 | DOI: 10.4172/2157-7013-C1-050

The granulocyte colony stimulating granulocyte colony stimulating factor promotes and improve histological outcome and neurological function recovery in ischemic rat brain

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Stroke is a leading cause of death worldwide and the most common cause of long term neurological disability in adults. The model of permanent occlusion of the common carotid arteries (POCCA) mimics ischemic event, in the early stages of the injury, which would allow us the validation of new therapeutic strategies. The objective of this study was to evaluate the restorative effects of granulocytic colony-stimulating factor (G-CSF) under ischemic conditions suggestive of its role in brain repairing processes after injury. A surgical midline incision exposed both common carotid arteries (CCA) which were dissected from surrounding nerves and fascia. Arteries receiving permanent occlusion with silk suture 3.0. After it, each animal received a daily G-CSF intradermal injection (5µg/Kg) for 5days. At the end of treatment, animals were inoculated intraperitoneally with bromodeoxyuridine (BrdU) during 5days (dose). When experimentation was complete, animals were perfused, frozen and sectioned on a cryostat. Selected sections and regions of interest (ROIs) were immunostained for NeuN, glial fibrillary acidic protein (GFAP) and nestin, anti-BrdU and c-fos. Additionally, the semi-quantitative expression of BDNF, GDNF and NGF was measured by RT- PCR as well as the transient expression of β-actin. Marked cell proliferation and gliosis were observed at hippocampus and prefrontal cortex. BDNF, GDNF and NGF were expressed in brain regions related to occlusion effects in lesioned rats totally unlike to healthy control animals. In conclusion, in spite of post-ischemic treatment with G-CSF is not addressed as neuroprotective in a model of focal brain ischemia, its markedly positive effect on cell repair or regeneration and it has relevant clinical effects if functional/behavioral results would be demonstrated in future studies.

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