Abstract

Introduction: The pathophysiology of cerebral ischemia is essential for early diagnosis, neurologic recovery, the early onset of drug treatment and the progosis of ischemic events. Experimental models of cerebral ischemia can be used to evaluate the cellular response phenomena and possible neurological protection by drugs. Objective: To characterize the cellular changes in the neuronal population and astrocytic response by the effect of Dimethyl Sulfoxide (DMSO) on a model of ischemia caused by cerebral embolism. Methods: Twenty Wistar rats were divided into four groups (n = 5). The infarct was induced with bovine thrombin (40 NIH/Unit.). The treated group received 90 mg (100 ul) of DMSO in saline (1:1 v/v) intraperitoneally for 5 days; ischemic controls received only NaCl (placebo) and two non-ischemic groups (simulated) received NaCl and DMSO respectively. We evaluated the neuronal (anti-NeuN) and astrocytic immune reactivity (anti-GFAP). The results were analyzed by densitometry (NIH Image J-Fiji 1.45 software) and analysis of variance (ANOVA) with the Graph pad software (Prism 5). Results: Cerebral embolism induced reproducible and reliable lesions in the cortex and hippocampus (CA1), similar to those of focal models. DMSO did not reverse the loss of post-ischemia neuronal immune reactivity, but prevented the morphological damage of neurons, and significantly reduced astrocytic hyperactivity in the somato-sensory cortex and CA1 (P <0.001). Conclusions: The regulator effect of DMSO on astrocyte hyperreactivity and neuronal-astroglialcytoarchitecture, gives it potential neuroprotective properties for the treatment of thromboembolic cerebral ischemia in the acute phase.

Keywords
Brain cerebral ischemia, dimethyl sulfoxide (DMSO), immunohistochemistry, astrocytes, neuroglia gliosis.