

Modulation of crucial adenosinetriphosphatase activities due to U-74389G administration in a porcine model of intracerebral hemorrhage.

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Abstract

Spontaneous intracerebral hemorrhage (ICH) represents a partially-understood cerebrovascular disease of high incidence, morbidity and mortality. We, herein, report the findings of our study concerning the role of two important adenosinetriphosphatases (ATPases) in a porcine model of spontaneous ICH that we have recently developed (by following recent references as well as previously-established models and techniques), with a focus on the first 4 and 24 h following the lesion's induction, in combination with a study of the effectiveness of the lazaroid antioxidant U-74389G administration. Our study demonstrates that the examined ICH model does not cause a decrease in Na(+),K(+)-ATPase activity (the levels of which are responsible for a very large part of neuronal energy expenditure) in the perihematomal basal ganglia territory, nor a change in the activity of Mg(2+)-ATPase. This is the first report focusing on these crucial ATPases in the experimental setting of ICH and differs from the majority of the findings concerning the behavior of these (crucial for central nervous system cell survival) enzymes under stroke-related ischemic conditions. The administration of U-74389G (an established antioxidant) in this ICH model revealed an injury specific type of behavior, that could be considered as neuroprotective provided that one considers that Na(+),K(+)- and Mg(2+)-ATPase inhibition might in this case diminish the local ATP consumption.

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