Transient global cerebral ischemia

A novel 21-aminosteroid (U-74389G), a new potent antioxidant, was evaluated for its protective effect on transient global cerebral ischemia. Ischemia was induced by 20 minutes of four-vessel occlusion in adult male Wistar rats. Injection of 21-aminosteroid (U-74389G, 5 mg/kg intraperitoneally injected) was repeated three times. The second injection was performed 30 minutes after the first injection, and the third injection was performed 210 minutes after that. Experimental animals were divided into five groups according to the time drug administration was initiated. Group I (n = 8) began vehicle administration 30 minutes before occlusion. Group II (n = 9) started 21-aminosteroid administration 30 minutes before occlusion. Drug administration in Group III (n = 9) began at the time of reperfusion, in Group IV (n = 8), 30 minutes after reperfusion, and in Group V (n = 6), 60 minutes after reperfusion. Animals in the control group (n = 5) underwent sham operations. One week after ischemia, the number of viable pyramidal neurons was counted in the hippocampal CA1 subfield. The results were as follows: the number of living neurons in Group I was 18.8 +/- 8.7; in Group II, was 44.7 +/- 9.5; in Group III, was 46.4 +/- 9.4; in Group IV, was 40.3 +/- 6.6; in Group V, was 10.2 +/- 2.5; and in the control group was 131 +/- 3.3. Groups II, III, and IV demonstrated significantly higher numbers of living neurons compared with Group I ($P < 0.05$). The present study revealed that U-74389G attenuated delayed neuronal death in global cerebral ischemia when it was administered before or soon after the ischemic episode [1)].

1)
Lee SH, Kondoh T, Camarata PJ, Heros RC. Therapeutic time window for the 21-aminosteroid, U-74389G, in global cerebral ischemia. Neurosurgery. 1996 Mar;38(3):517-21; discussion 522. PubMed PMID: 8837804.