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Erythromycin induced neuroprotection during prolonged deep hypothermic circulatory arrest in an acute porcine model

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Background

The present study assesses whether preconditioning with erythromycin can improve neuronal viability in the neocortex following deep hypothermic circulatory arrest (DHCA) in the porcine model.

Methods

Piglets were treated with erythromycin (25 mg/kg, iv) (n = 8) or vehicle (n = 6) and subjected to 75 minutes of DHCA at 18°C, 12 hours after pretreatment. Three served as normal controls. After gradual rewarming, treatment animals were sacrificed and brains were perfusion-fixed and cryopreserved. Motor cortex was dissected from the left hemisphere and paraffin embedded for histologic staining with hematoxylin and eosin (HE). To assess neuronal damage, HE-stained paraffin sections (10 μm) were examined by light microscopic examination at x400 magnification. Layer V of the motor cortex was counted. Neuronal injury was recorded when there was evidence of eosinophilic cytoplasm, cytoplasmic vacuolation, cell body shrinkage or nuclear pyknosis. Neuronal injury was scored on a scale of 0-5.

Results

The peri-operative physiological variables did show significant variations with erythromycin drug treatment. The motor cortex from piglets pretreated with vehicle undergoing DHCA showed diffuse edema. Neurons showed a diffuse loss of Nissl substance, shrinkage of the perikaryon, and nuclear pyknosis with a mean neuronal injury score of 3.74 + 1.47. Neuronal injury in the motor cortex was significantly lower in animals pretreated with erythromycin (2.53 + 1.22; $p < 0.01$). Normal controls showed minimal neuronal injury (0.42 + 0.51).

Conclusion

Pharmacologic preconditioning with erythromycin significantly improved neuronal viability in the motor neocortex of piglets undergoing HCA at 18°C. These findings suggest a potential clinical strategy of preemptive neuroprotection.

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