Idazoxan does not prevent but worsens focal hypoxic-ischemic brain damage in neonatal Wistar rats

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Résumé en anglais	We examined the neuroprotective efficacy of a post-treatment with idazoxan (Idaz): an alpha2-adrenoceptor antagonist with activity at the I1- and I2-subtypes of the imidazoline receptor (I-receptor), in an experimental model of perinatal hypoxic-ischemic (HI) brain damage. Seventy-two, 7-day-old Wistar rats were subjected to permanent unilateral ligation of the common carotid artery and transient (2 hr) hypoxia (8% O(2)). The surviving animals were sub-divided into 3 groups: one "control" group received intraperitoneal (i.p.) injection of saline (Sigma; n = 21) and two "treated" groups received, 10 min post-HI, i.p. treatments with Idaz (I3: 3 mg/kg; n = 19) or (I8: 8 mg/kg; n = 20). Idaz effects were assessed by TTC-staining 72 hr post-HI for Sigma (n = 13), I3 (n = 11), and I8 (n = 12) groups and by MRI-examination 5 weeks post-HI for Sigma (n = 8), I3 (n = 8), and I8 (n = 6) groups. Total ratio of brain infarct areas were significantly (P < 0.01) different between Sigma and Idaz-treated rats: 20.9 +/- 4.0%, 35.6 +/- 5.9 % and 36.8 +/- 5.8% for Sigma, I3 and I8, respectively, when determined with TTC-staining and; 23.3 +/- 3.7%, 39.8 +/- 4.2%, and 43.2 +/- 10.1%, for Sigma, I3, and I8, respectively, when assessed by MRI. Our results suggest that Idaz, given as a post-HI treatment, does not exert neuroprotective effects but enhances the brain injury induced by focal neonatal cerebral HI. The deleterious mechanism may result from an overactivity of sympathetic tone and/or the immaturity of central I-receptors in newborn rats.
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