Inhaled Argon improves neurological outcome in experimental traumatic brain injury

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INTRODUCTION. While supportive treatment in the management of Traumatic Brain Injury (TBI) has progressed over the past 20 years, specific drug treatments are lacking. *In vitro* and *in vivo* models of ischemic heart and brain injury show that the gaseous agent Argon is endowed with neuroprotective potential. Whether Inhaled Argon (iAr) is protective in experimental TBI is presently unknown.
OBJECTIVES. To test the effects of inhaled Argon administered after experimental TBI in mice on neurological functions and structural outcome by longitudinal behavioural assessments and magnetic resonance imaging (MRI) including T2W and DWI sequences.
METHODS. Severe TBI was performed in anesthetized mice (C57BL/6J, 8 weeks old, male) by controlled cortical impact. Ten minutes after TBI, mice were randomized to 24h treatment by iAr 70%-O_2 30% (n=10) or air (n=10). Sensorimotor deficits were evaluated at 24h post TBI and at 1 week by neuroscore and simple neuroassessment of asymmetric impairment (SNAP) tests. MRI (7-T, Bruker)was performed at 3 days post TBI to evaluate contusion volume by T2W. The effect of iAr on acute brain edema, was analysed in a subset of mice (n=3 per group) by DWI-MRI..
RESULTS. Argon inhalation significantly improved neurological function at 24 hours and 7 days after TBI (Neuroscore 24h post TBI iAr 6.1±0.5 vs. Air 3.7±0.7, p=0.0102). Contusion volume was reduced by 16% in iAr than air breathing TBI mice. Vasogenic brain edema showed a reduction in iAr treated TBI mice close to significance (p=0.056).
CONCLUSIONS. iAr induces an acute and persistent improvement of sensorimotor function when administered for 24h starting 10 minutes after TBI. This outcome is reinforced by preliminary MRI data showing decreased edema in iAr treated mice. Our data support future studies to understand the potential of iAr as an accessible treatment in TBI.