

HYPERBARIC OXYGEN THERAPY (HBOT) PREVENTS SUBARACHNOID HAEMORRHAGE (SAH)-INDUCED APOPTOSIS AND IMPAIRED CONTRACTILITY OF THE RABBIT BLADDER

Hypothesis / aims of study

In stroke patients, after the initial detrusor areflexia in the acute period, the progression of the urinary bladder dysfunction varies depending of the type of stroke. To the best of our knowledge, no study exists specifically regarding the effects of experimental SAH on urinary bladder. The aim of our study was to investigate the effects of experimental SAH on urinary bladder and to evaluate the possible effects of HBOT on urinary bladder after experimental SAH.

Study design, materials and methods

The creation of experimental SAH was performed through a midline occipitocervical incision, where the atlanto-occipital membrane was punctured by a 23-gauge needle to withdraw cerebrospinal fluid (CSF) from the cisterna magna and a 1 ml nonheparinized autologous blood was injected gently into the cisterna magna. The animals were then kept in a head down position at an angle of 30 degrees for 30 minutes to achieve dissemination of blood through the basal cisterns.

A total of 15 male New Zealand White Rabbits were assigned randomly to one of three groups: Group I were not subjected to SAH and served as the control group (n = 5), Group II were subjected to SAH, received no treatment, and served as the SAH group (n = 5) and Group III were subjected to SAH and received 5 sessions of HBO (started 12 hours after SAH induction and was given twice daily for the first two days and once on the third day) and served as the treatment group (n = 5). At 72 hours after the SAH induction procedure, bladders from all animals subjected to experimental SAH and animals in Group I were removed for in vitro organ bath experiments, biochemical analyses and histological examination.

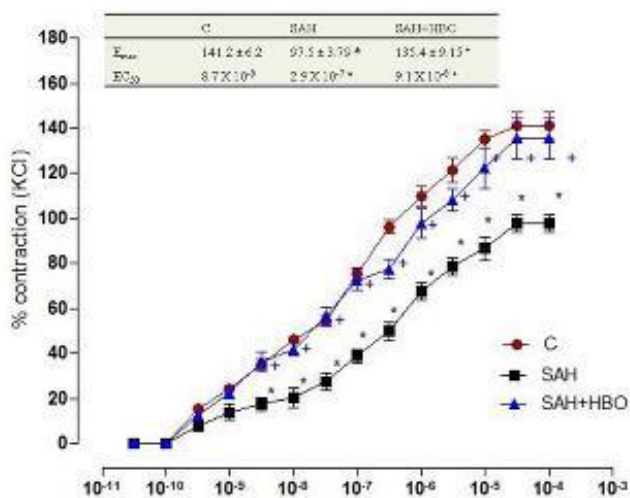


Figure 1. Concentration–response curves obtained by cumulative addition of CCh to rabbit urinary bladder strips of the groups (*p < 0.05 versus control group; +p < 0.05 versus SAH group). Inset table 1. E_{max} and EC_{50} values for carbachol in bladder strips.

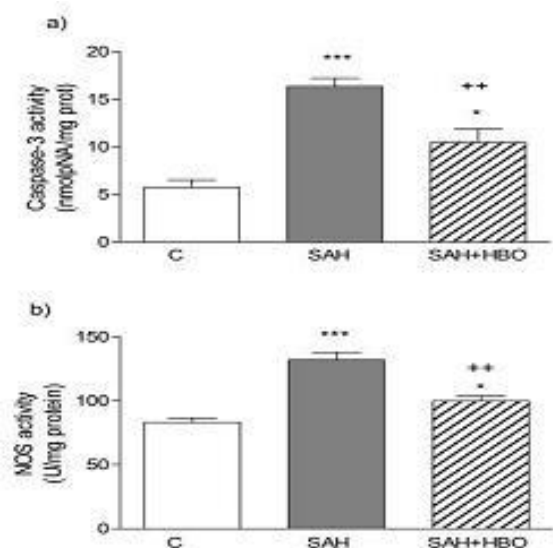


Figure 2. a) Caspase-3 and b) NOS activities in the bladder tissues of groups. (* p<0.05, *** p<0.001 vs control group; **p<0.01 vs SAH group).

Results

Results of isolated organ bath experiments demonstrated that compared to the control group, the contractile responses of the strips to carbachol (CCh) in the SAH group were significantly decreased whereas HBOT restored the contractile responses ($p < 0.05$ – figure 1). Caspase-3 activity of bladder tissues was significantly elevated in the SAH group, indicating SAH-induced apoptosis ($p < 0.01$), whereas SAH-induced increase in caspase-3 activity was significantly depressed in the tissues of the HBOT group. Furthermore in the SAH group, nitric oxide synthase (NOS) activity were found to be significantly increased ($p < 0.01$), when compared to those in the control group, and again HBOT provided a decreased NOS activity ($p < 0.01$ - figure 2).

Interpretation of results

SAH impairs the contractile response of the rabbit bladder and HBOT in the acute period protects the bladder from the damage of SAH. SAH also induces apoptosis in the rabbit bladder and again HBOT in the acute period prevents SAH-induced apoptosis significantly.

Concluding message

HBO treatment created a protective effect from SAH-induced changes in bladder tissues of the rabbit that needs to be investigated by further studies.

Disclosures

Funding: None **Clinical Trial:** No **Subjects:** ANIMAL **Species:** Rabbit **Ethics Committee:** Marmara University Laboratory Animals Ethical Committee