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Stress Amelioration Potential of Active Constituent of *Ocimum sanctum*

OBJECTIVE

To evaluate the potential roles of triterpenic acid: ursolic acid (UA) in the activation of the nuclear factor-erythroid 2-related factor 2 (Nrf2) pathway and the underlying mechanism

MATERIALS AND METHODS

Male Imprinting Control Region (ICR) mice (28–32 g) were used for this study. The TBI model was induced utilizing a modified weight drop device. Mice were divided into following groups: (1) Sham; (2) TBI; (3) TBI + vehicle; (4) TBI + UA; (three subgroups were administrated UA at 50, 100, and 150 mg/kg). All mice underwent the TBI with the exception of the sham group. Ursolic acid was dissolved in 1% dimethylsufoxide (DMSO)-phosphate-buffered saline (PBS) and injected into the mice intraperitoneally immediately following TBI. An equal volume of 0.1% DMSO in PBS served as the vehicle control (vehicle). Protein was extracted from the lesioned hemisphere and glutathione peroxidase (Gpx), superoxide dismutase (SOD), and malondialdehyde (MDA) content in the total protein were measured using kit-based assay protocols.

RESULTS

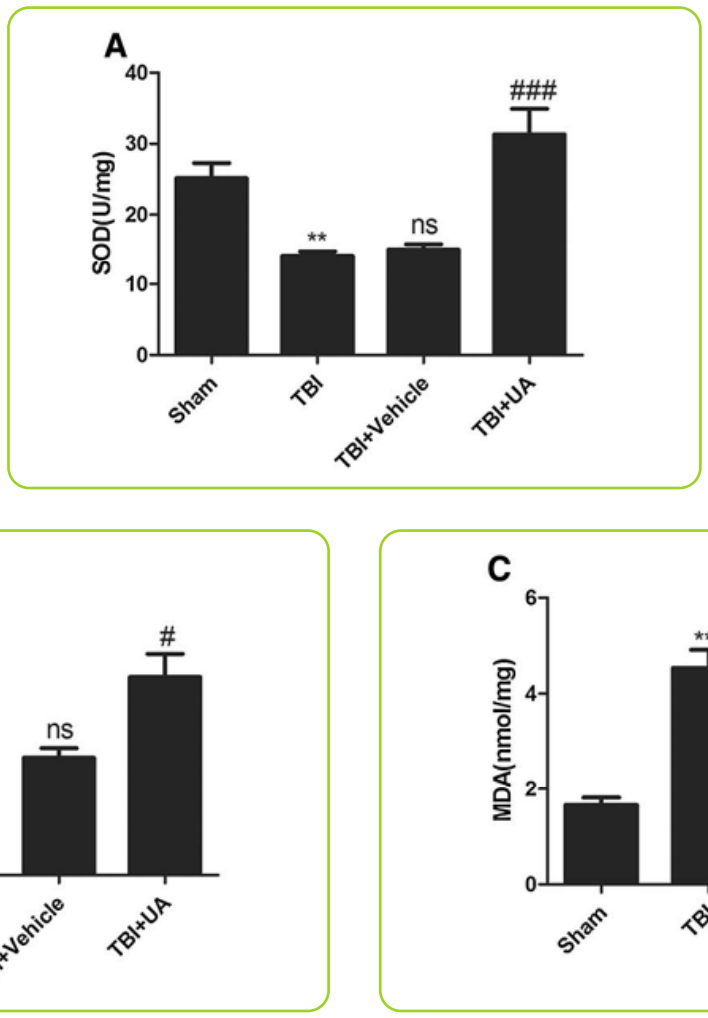

Figure : Ursolic acid (100 mg/kg) reduced oxidative stress in the brain following TBI

Oxidative stress was assessed by (A) SOD activity, (B) GPx activity and (C) MDA level. Data represent mean ± SD; n=6 per group; **p<0.01 and ***p<0.001 versus sham group; nsp>0.05 versus TBI group; ###p<0.001 and #p<0.05 versus TBI+vehicle group

CONCLUSIONS

- SOD and GPx levels nearly returned to normal following the administration of UA (p<0.001 and p<0.05, respectively).
- Administration of UA reduced the elevation of MDA significantly (p<0.001 Vs TBI+vehicle group).

OUTCOME

These data depicted that UA increases the activity of antioxidant enzymes and attenuated brain injury via Nrf2 factor.

Reference:
Ding H, Wang H, Zhu L et al. Ursolic Acid Ameliorates Early Brain Injury After Experimental Traumatic Brain Injury in Mice by Activating the Nrf2 Pathway. Neurochem Res. 2017;42(2):337-346.