ROLE OF KYNURENINE SYSTEM IN THE PACAP RELATED TRIGEMINOVASCULAR ACTIVATION

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The trigeminovascular system (TS) forms bridge between the meninges, the pial vessels and the nociceptive second order neurons in the caudal trigeminal nucleus (TNC) through the trigeminal ganglion (TRIG). Activation of the TS is accompanied by peripheral and central sensitization. During these processes the glutamatergic transmission, particularly the kynurenine system and several neuropeptides, for example the pituitary adenylate cyclase-activating polypeptide-38 (PACAP-38) have pivotal role. Our aim was to examine the concentration changes of PACAP-38 in the rat model of activated TS during pre-treatment by a potential NMDA receptor antagonist molecule, named kynurenic acid derivative 1 (KYNA1).
Young adult Sprague-Dawley rats were anaesthetized and injected with intravenous KYNA1 solution 30 min prior to the electrical stimulation (ES) of the right TRIG. 180 minutes after the stimulation blood samples were taken from the right cranial vena cava, moreover the TNC and TRIGs were dissected. The PACAP-38–like immunoreactivity (-ir) was measured in the above samples by radioimmunoassay method.
The ES of the TS caused significantly elevated PACAP-38-ir in the TNC, which was diminished by KYNA1 pre-treatment. Similar tendency was also observed in the stimulated TRIG and the KYNA1 seemed to be protective in the plasma. Significant changes were not detected in the unstimulated TRIG.
These results suggest that the activated TS induced PACAP-38 release from the peripheral and central terminals of the TRIG can be partly prevented by the KYNA1. It needs to test further NMDA-modulators in this model, which may confirm relationship between the NMDA and PACAP receptors.