In acute ischemic stroke, core of the brain tissue where the blood perfusion is minimum, damages irreversibly in minutes. Reperfusion of ischemic area may results in free radicals production. Pregaboline has been shown to promote neuroprotection in spinal ischemia.

In this study, 36 Wistar Albino type male rats was used. Rats were divided into six groups: control, pregabalin, ischemia, pregabalin+ischemia, ischemia+reperfusion and pregabalin+ischemia+reperfusion. Pregabalin was administered with oral gavage through two days in 50 mg/kg/day dose. Ischemia was performed by occluding the right internal carotid artery with bulldog clamp for 30 minutes, then reperfusion was performed through 20 minutes. TBARS, NO, SOD, GSH-Px, CAT levels and NMDA-NR2A and NR2B receptor concentrations were evaluated.

TBARS and NO levels in ischemia and ischemia+reperfusion groups were significantly more increased in comparison to control group and were significantly more decreased in pregabalin+ischemia+reperfusion group. NO level in
ischemia+pregabaline group was significantly more
decreased than was in ischemia group. GSH-Px level
was decreased in both damage group showing the

usage of enzyme. GSH-Px levels in pregabaline+is
chemia+reperfusion group were significantly more
increased in comparison to ischemia+reperfusion
group. NR2B receptors concentrations were
significantly reduced in Pregabaline+ischemia and
pregabaline+ischemia+reperfusion groups.
Pregabaline led to decreased NMDA-NR2B
receptor levels and NO levels in ischemia and
ischemia+reperfusion groups, supporting a
significant role of pregabaline in decreasing
oxidative stress along with neuroprotection in
ischemia-reperfusion damage and suggests that
pregabaline may be used in acute ischemic brain
injury for both to decrease the oxidative stress and
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chemia+reperfusion group were significantly more
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group. NR2B receptors concentrations were significantly reduced in Pregabaline+ischemia and pregabaline+ischemia+reperfusion groups. Pregabaline led to decreased NMDA-NR2B receptor levels and NO levels in ischemia and ischemia+reperfusion groups, supporting a significant role of pregabaline in decreasing oxidative stress along with neuroprotection in ischemia-reperfusion damage and suggests that pregabaline may be used in acute ischemic brain injury for both to decrease the oxidative stress and also to provide neuroprotection.