Abstract:

Objectives: Acrylamide is a toxic compound that forms during food processing at high temperatures. Acrylamide has been reported to cause toxicity in various organs in the body including the CNS, liver, kidney, reproductive system, and gastrointestinal tract. Acrylamide is considered as a carcinogenic and potent neurotoxic agent that can be absorbed through the skin, gastrointestinal and respiratory tracts. Acrylamide promotes oxidative and inflammatory processes in the CNS leading to neurological disorders. Carvedilol is an adrenergic antagonist that has been demonstrated to exhibit potent anti-oxidant and anti-inflammatory effects. The aim of this study was to evaluate the potential protective effect of carvedilol and its underlying mechanisms in acrylamide-induced brain injury in a mouse model.

Materials and Methods: The experiments were conducted on mail Swiss albino mice. Acrylamide was administered in mice at the dose of 50 mg/kg/day intraperitoneally for 11 days to induce brain injury. Mice were treated with carvedilol at two doses (5 and 10 mg/kg/day) orally for 11 consecutive days. At the end of the experiment, after the gait assessment mice were sacrificed and brain tissues were dissected for histological and biochemical analysis.

Results: The results of the present study showed that treatment of mice with carvedilol decreased acrylamide-induced bodyweight loss, abnormal gait, and histopathological damage in the brain tissue. Carvedilol treatment significantly reduced the levels of malondialdehyde (MDA), carbonyl protein and myeloperoxidase (MPO) activity. Furthermore, administration of carvedilol significantly increased the levels of glutathione (GSH), catalase, superoxide dismutase (SOD) and heme oxygenase-1 (HO-1) in the brain tissue of mice.

Conclusion: These findings indicate that carvedilol is able to decrease acrylamide-induced brain injury by inhibition of oxidative stress and inflammation.