Neuroprotective Effect of L-Theanine against Tramadol Induced Parkinson's Like Symptoms in Experimental Rats

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Parkinson's disease (PD) is a chronic neurodegenerative disorder triggered by degeneration of dopaminergic neurons in substantia nigra pars compacta. Low antioxidant level, mitochondrial failure and neuroinflammation are the major pathological mechanisms. The animals were divided into 7 groups. Group 1 served as Normal control. Group 2 received Tramadol ( $50 \mathrm{mg} / \mathrm{kg}$, i.p.) daily for 28 days. Group 3, 4 and 5 received L-theanine ( 25,50 and $100 \mathrm{mg} / \mathrm{kg}$; p.o.) from 14 to 28 day prior to the tramadol administration. Group 6 received standard drug from day 14 to day 28 prior to the tramadol administration. Behavioral observations were done on $1,7,14,21$ and 28 day after tramadol treatment. On 29 day, animals were sacrificed and striatum was isolated for biochemical, neuroinflammation, histopathological and neurotransmitters analysis. Administration of tramadol ( $50 \mathrm{mg} / \mathrm{kg}$, i.p.) for 28 days in rats produces impaired motor functions and locomotor activity as evidenced by rotarod, open field, narrow beam walk and grip strength performance. In addition, there was increased oxidative stress (MDA, nitrite) and neuroinflammatory markers (TNF- $\alpha$, IL-1 $\beta$ and IL-17) and decreased levels of catecholamines, GABA and glutamate. The treatment drug L-theanine at dose ( $25,50,100 \mathrm{mg} / \mathrm{kg}$ ) significantly and dose- dependently improved alterations in motor impairments and locomotor activity, attenuated oxidative stress, neuroinflammatory markers and restored catecholamines, GABA and glutamate level in striatum. Chronic tramadol administration produces impaired motor functions, increased oxidative stress, neuroinflammation and altered neurotransmitters level was significantly ameliorated by L-theanine, through antioxidant, antiinflammatory and neuroprotective mechanisms.

