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Research paper

Tannic acid alleviates lead acetate-induced neurochemical perturbations in rat brain

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HIGHLIGHTS

- Neurotoxicity is induced by lead acetate in Wistar rats.
- Tannic acid ameliorated lead acetate induced neurotoxicity.
- Neuroprotective efficacy of tannic acid is mediated by mitigating oxidative stress.
- Tannic acid is a potential candidate for attenuating heavy metal neurotoxicity.

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ABSTRACT

Oxidative stress has been projected as a promising mechanism involved in lead exposure. The lead predisposition catalyzes oxidative reactions and generates reactive oxygen species. The present study was carried out to investigate the effect of oral administration of tannic acid (TA) on behavioral deficit, antioxidative deterioration induced by lead acetate (LA) exposure on experimental rat brain. Male Wistar rats were treated with 50 mg/kg body weight of LA and TA for three times a week for two weeks. Our data showed LA-induced profound elevation of ROS production and oxidative stress, as evidenced by increased levels of oxidative stress markers such as lipid peroxidation and protein carbonyl observed in LA treated rats, whereas significant depletion in the activity of non-enzymatic antioxidants, enzymatic antioxidants, neurotoxicity biomarker and histological changes were observed in LA treated rat brain. However, TA administration restored antioxidant status of brain significantly when compared to control. Our results demonstrate that TA exhibits potent antioxidant properties and suppresses oxidative damages in rat brain induced by LA treatment. These findings were further supported by the neurotoxicity biomarker and histopathological findings in the brain tissue showed that TA protected tissue from deleterious effects of LA exposure. It is concluded, these data suggest that LA induces oxidative stress and supplementation of TA has a powerful antioxidant effect, and it protected rat brain from poisonous effect of LA exposure in experimental rat.

1. Introduction

Lead (Pb) is a pervasive and ubiquitous heavy metal in nature [1]. Its persistent occupational and environmental exposure may contribute to hematopoietic, gastrointestinal, urinary, cardiovascular, and nervous systems are well described in human and animals [2]. Pb exposure to the brain is considered as a major risk factor for the development of neuronal dysfunction especially in developing brain [3]. Pb induces wide range of direct neurotoxic effects including apoptosis, excitotoxicity, oxidative stress and damage to neuronal mitochondria [4]. Moreover, experimental studies performed on Pb intoxicated rats have shown alterations in neurobehavioral deficits. [5].

Pb acts as a catalyst in oxidative deterioration of cellular damage mediated by free radicals involved in pathology associated with Pb intoxication in rats [6]. Furthermore, generation of reactive oxygen species (ROS), subsequent stimulation of lipid peroxidation (LPO) and weakening of endogenous antioxidant system have been hypothesize to be major contributors to Pb toxicity [7]. Brain is particularly susceptible to oxidative damage since it has

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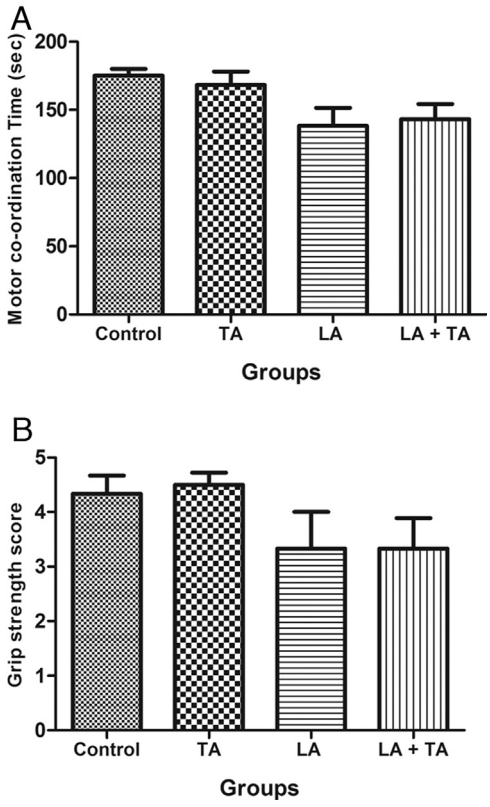

Fig. 1. Effect of TA treatment on muscular coordination skill and grip test in LA treated rats. LA administration showed no significant impairment in motor coordination as well as in grip test when compared to control. Also TA alone treated group showed no significant alteration when compared to control. Each value represented as mean ± SE (n = 6).

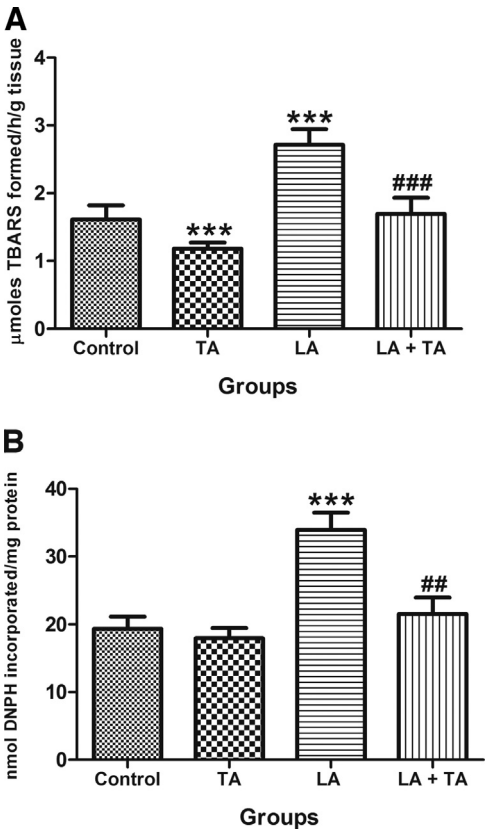

Fig. 2. Effect of TA treatment on LPO in term of TBARS (A) and PC (B) content in rat brain homogenate. LPO and PC content was significantly increased in LA treated group rats as compared to control ($^{***}P < 0.001$). TA treatment has decreased the content of LPO and PC significantly in ($^{###}P < 0.001$ and $^{##}P < 0.01$). LA + TA group as compared to LA group rats. The LPO level in TA alone showed significant difference ($^{***}P < 0.001$) when compared to control. Each value represented as mean ± SE (n = 6).

potential sources of phospholipids and high amounts of polyunsaturated fatty acids (PUFA) and rapid oxidative metabolic activity, but its ability to combat oxidative stress is limited [8]. Thus pharmacological agents with antioxidant property may shield the system to stay normal against the oxidative damage induced by Pb intoxication. In the present study tannic acid (TA) is used to mitigate neuronal damage in rat model of lead acetate (LA) intoxication. TA is a naturally occurring plant polyphenol [9], possesses antioxidant and anticancer and ant-inflammatory effect and also a very efficient chelator for metals in animal model [10].

Protective efficacy of TA may be due to the scavenging free radicals and lower oxidative damage. Since the antioxidant activity of several polyphenols involving prevention of free radicals formation and lipid peroxidation has been correlated with their metal chelating properties [11]. Therefore this study was formulated to investigate the pretreatment effect of TA on biochemical and morphological alterations in lead acetate intoxicated rat brain.

2. Materials and methods

2.1. Animals

The experiments were carried out on male Wistar rats weighing 300–450 g obtained from the Central Animal House Facility of Hamdard University. The animals were used in accordance with the procedure approved by the Animal Ethics Committee of Jamia Hamdard New Delhi.

2.2. Experimental design

Experiments were carried out to evaluate the antioxidant effect of 50 mg/kg body weight of TA against LA induced oxidative stress, for 2 weeks. 24 rats were randomly divided into four groups of six animals each (n = 6). Group I was vehicle-treated control, the second group was treated with LA (50 mg/kg body weight; intraperitoneally) three times a week for two weeks, the third group received LA (*i.p.*) and TA (orally) at the dose of 50 mg/kg body weight three times a week for two weeks and the fourth group was treated with TA (orally) three times a week for two weeks. After the completion of two weeks of dosing regime, all rats were sacrificed by cervical dislocation under anesthesia (diethyl ether) and the brains were excised for further investigation of biochemical and histological evaluations. The doses of TA and LA were selected on the basis of previous studies and literature reports [12–14].

2.3. Neurobehavioral test

Rotarod and grip strength tests were performed as described in our previous reported study [15]. The rotarod unit was used to evaluate the motor deficiency of the rat to hold the rotating rotor whereas the grip strength test was used to evaluate limb strength that has been used to investigate the effects of Pb on neuromuscular deficiency.