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NEUROPROTECTIVE EFFECTS OF TALINUM TRIANGULARE EXTRACT ON CEREBRAL INFARCTION AND OXIDATIVE STRESS IN A RAT MODEL OF ISCHEMIA-REPERFUSION INJURY

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ABSTRACT

Context: ischemia-related stroke is a primary cause of "mortality and disability," which includes a disruption in cerebral blood circulation and the ensuing damage to regeneration. Talinum triangular, a traditional medicinal plant, has demonstrated various pharmacological properties, including antioxidative and neuroprotective effects.

Method: Male rats (n=24) were divided into four groups: Sham Control, I/R Control, TTE 200 mg/kg, and TTE 400 mg/kg, with six rats in each group. Ischemia was induced by occluding the middle cerebellar stream for ninety a few moments, then reperfusion for twenty-four hours. TTE was administered orally for seven days prior to ischemia induction.

Results: TTE treatment significantly reduced cerebral infarction in a "dose-dependent manner," with 19.74% and 35.75% reduction at "200 mg/kg and 400 mg/kg, respectively, compared to the I/R control group". TTE also significantly increased SOD, catalase, and GSH levels while decreasing MDA levels, indicating enhanced antioxidative defines and reduced oxidative stress. TTC staining corroborated these findings, showing less neural damage in TTE-treated groups.

Conclusion: Talinum triangulate extract demonstrates substantial restorative benefits, from its antioxidant activity qualities, against ischemia-reperfusion damage in rats. These results imply that TTE could be an effective treatment for ischemia-related strokes, warranting further investigation into its mechanisms and potential clinical applications.

Keywords:

Ischemic stroke, Talinum triangular, neuroprotection, antioxidative defense, cerebral infarction, ischemia-reperfusion injury.

INTRODUCTION

Globally, stroke caused by ischemic stroke continues to be the primary cause of death and permanent cognitive impairment, primarily resulting from the interruption of cerebral blood flow and subsequent reperfusion injury. This pathological process induces a series of biological processes that include inflammation, oxidative harm, including deaths of neurons, culminating in significant brain damage. Current therapeutic strategies are limited and often fail to provide comprehensive neuroprotection, underscoring the urgent need for novel and effective treatments [1]. Water lettuce, or Talinum triangulare, is a popular name for, is a traditional medicinal plant renowned for its diverse pharmacological properties, including antioxidative, anti-inflammatory, and neuroprotective effects [2]. Previous studies have highlighted the potential of Talinum triangulare extract (TTE) in mitigating oxidative stress and improving cellular defense mechanisms, giving it a therapy option for ischemia diseases. The purpose of this work is to assess TTE's preventative benefits on ischemic stroke in a rodent model of "ischemia-reperfusion (I/R)" injury [3]. Specifically, we investigate the extent of cerebral infarction, as well as the antioxidative properties of TTE, by measuring key biochemical markers such as "superoxide dismutase (SOD), catalase, reduced glutathione (GSH), and malondialdehyde (MDA)".By exploring the efficacy of TTE in reducing cerebral infarction and enhancing antioxidative defense mechanisms, this research seeks to contribute to the development of new therapeutic approaches for ischemic stroke.

METHODOLOGY

This operate objective is to evaluate the antioxidant benefits of "Talinum triangulare extract (TTE)" on stroke in dogs after exposure to "ischemia-reperfusion (I/R)" injury. The study includes four Rat groupings, with all of them having six members (n=6). The groups are as follows:

- Sham Control Group
- I/R Control Group
- "TTE 200 mg/kg Group"
- "TTE 400 mg/kg Group"

Animal Model and Treatment

- Animals: "Healthy male rats, weighing 250-300 grams", were applied in the research. The reptiles had unrestricted access to nourishment and were kept in typical conditions in laboratories and water.
- Induction of Ischemia-Reperfusion Injury: Inducing ischemia of the brain included blocking the "middle cerebral artery (MCA)" for 90 minutes, followed by reperfusion for 24 hours.
- TTE Administration: Oral TTE dosages of 200 mg/kg & 400 mg per kilogram of body weight were used each day for seven days prior to the induction of ischemia. The sham control group received no treatment, while the I/R control group received a vehicle treatment.

Assessment of Cerebral Infarction

- Measurement: Twenty-four hours before reperfusion, and the degree of brain injury was measured using "2,3,5-triphenyltetrazolium chloride (TTC)" staining. The infarcted (pale) areas were measured and expressed is a proportion of the whole head.
- Data Presentation: The results are expressed as mean ± SEM. The percentage reduction in infarction for TTE-treated groups was calculated relative to the I/R control group.

Biochemical Analysis

- Superoxide Dismutase (SOD) Levels: SOD activity was measured in brain tissue to assess the antioxidative effect of TTE.
- Catalase Levels: Catalase activity was quantified to evaluate the enzymatic breakdown of hydrogen peroxide.
- Reduced Glutathione (GSH) Levels: GSH levels were measured to determine the antioxidant capacity of the brain tissue.
- Malondialdehyde (MDA) Levels: MDA levels were assessed to evaluate lipid peroxidation and oxidative stress.

Histological Analysis

- TTC Staining: Brain sections were stained with TTC to differentiate between viable (red-stained) and infarcted (pale) tissue, providing a visual representation of neural damage.

RESULTS:

"Table 1: Showing the effect of TTE on cerebral infarction in rats subject"

Group (n=6)	"Cerebral infarction"	% Reduction of infarction"
"Sham control"	1.57 ± 0.178	-
"I/R control"	47.52 ± 2.45	-
TT extract (200 mg/kg)	38.14 ± 2.13	19.74
"TT extract (400 mg/kg)"	30.54 ± 2.57	35.75

In table 1 TTE significantly reduced cerebral infarction in a dose-dependent manner, with 19.74% reduction at 200 mg/kg and 35.75% reduction at 400 mg/kg compared to the I/R control group.

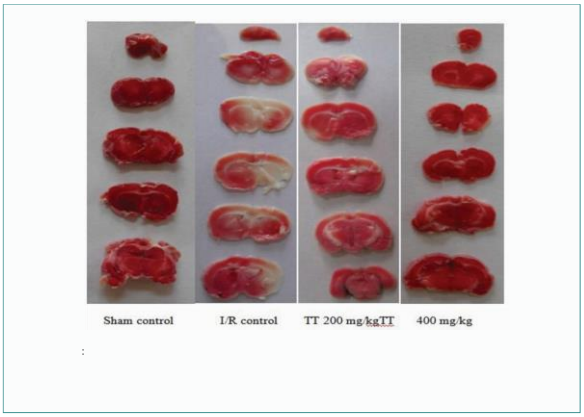
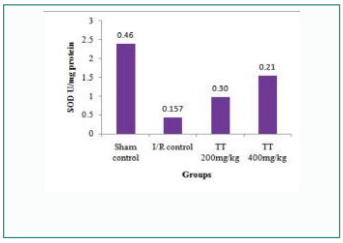
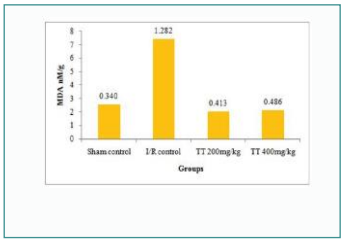
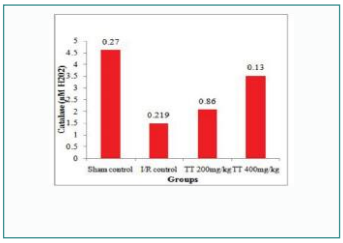


Figure 2: demonstrating the impact of a TTE-stained cerebellum segment in rats that had broad ischemia and then the reperfusion procedure: In comparison with the various groups of people, the I/R group exhibits pale spotting with TTC, indicating neuronal injury.

Figure 2 provides a visual representation of the effects of Talinum triangulare extract (TTE) on Rats' brain tissues after undergoing worldwide ischemia and recovery, using triphenyltetrazolium chloride (TTC) staining to assess neural damage. TTC staining is a widely used method to differentiate between viable and damaged tissue; viable brain tissue typically stains red, while damaged or dead tissue remains pale.





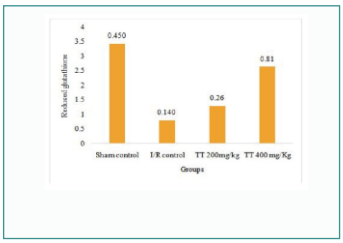


SOD : Figure 3

Catalase : Figure 4

Glutathione: Figure 5

Malonaldehye :Figure 6



The impact of TTE on SOD levels within brains of rats in reperfusion following ischemia tissue is shown in Figure 3. The findings have been contrasted to an experimental baseline group, while the Aggregate standard deviation of the standard deviation is how the statistics are shown. (n = 6).

Figure 4: Compares the findings to the ischemia control group and shows the influence of TTE on the levels of Catalase in rat brain in diabetic resuscitation area. The data are shown as mean "± SEM (n = 6)."

Figure 5: Compares the findings to the ischemia control group and shows the impact of TTE in the diabetics restorative region of rats, on lowered serotonin levels among axons. The average along with the variance of mean will be displayed for the information being shown. (n = 6).

In Figure 6, the impact of "TTE on MDA" concentrations within rat neural tissue after the reperfusion of ischemia is shown. The data are presented as "mean ± SEM (n = 6)", and the outcomes are contrasted with those of the ischaemic control population.

TTE treatment improved SOD, catalase, and GSH levels while reducing MDA levels, indicating enhanced antioxidative defense and reduced oxidative stress.

DISCUSSION

The present study demonstrates the neuroprotective effects of Talinum triangulare extract (TTE) on cerebral infarction in a rat model of "ischemia-reperfusion (I/R)" injury. The findings indicate that TTE significantly reduces cerebral infarction in a way depending on dosage, as evidenced by the 19.74% and 35.75% reduction "at 200 mg/kg and 400 mg/kg" dosages, respectively. Additionally, TTE administration enhances the antioxidative defense mechanisms, as shown by the increased levels of "superoxide dismutase (SOD), catalase, and reduced glutathione (GSH), alongside decreased malondialdehyde (MDA)" levels [6].

The reduction in cerebral infarction observed in TTE-treated groups suggests that TTE confers substantial neuroprotection against I/R injury. The dose-dependent effect, with a higher reduction in infarction at 400 mg/kg, highlights the potential for optimizing TTE dosage for maximum therapeutic benefit. This aligns with the hypothesis that TTE's antioxidative properties are essential in reducing the risk of ischemic brain injury. TTE improves the brain's antibacterial defines mechanism, as shown by increased concentrations of GSH and increases in SOD and the activity of catalase. Two crucial enzymes are glutathione and SOD that detoxify reactive oxygen species (ROS), while GSH is a major intracellular antioxidant. The reduction in MDA levels, a marker of lipid peroxidation, further supports the antioxidative effects of TTE, suggesting that it effectively reduces oxidative stress and subsequent cellular damage [7].

Several studies support the neuroprotective and antioxidative effects of natural extracts in ischemic conditions. For instance, a study by Liao et al. (2015)[4] demonstrated that Ginkgo biloba extract significantly reduced cerebral infarction and oxidative stress in a rat model of cerebral ischemia. Similarly, Sun et al. (2018)[5] found that curcumin, a compound with known antioxidative properties, provided neuroprotection against I/R injury by modulating oxidative stress pathways.

The promising results of this study suggest that TTE may be created as a stroke with ischemic attack treatment substance. But extra research is required to clarify the precise molecular mechanisms underlying TTE's neuroprotective effects. Investigations into the long-term outcomes of TTE treatment, its effects on other models of neurological injury, and potential synergistic effects with existing treatments are warranted. Additionally, exploring the pharmacokinetics and safety profile of TTE in larger animal models and clinical trials would be crucial steps toward its potential therapeutic application. Understanding the interaction between TTE and the brain's complex biochemical environment could lead to more effective and targeted treatments for

ischemic stroke and related neurovascular disorders.

CONCLUSION

This study provides compelling evidence that Talinum triangulare extract (TTE) exerts substantial preventative benefits against ischemia-reperfusion injury-induced brain damage in rats. The antioxidative properties of TTE, reflected in improved levels of SOD, catalase, GSH, and reduced MDA, play a pivotal role in its protective effects. These findings pave the way for further research into TTE as a potential therapeutic agent for ischemic stroke, offering hope for more effective treatments for this debilitating condition.

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