



# MULTIDISCIPLINARY *SCIENCE JOURNAL*

VOLUME - 3



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**ISSN**  
INTERNATIONAL  
STANDARD  
SERIAL  
NUMBER  
UNITED STATES  
ISSN:3067-1221; CODEN: USA



# EVALUATION OF ANTIEPILEPTIC ACTIVITY OF *HELIOTROPIMUM INDICUM* IN PTZ INDUCED SEIZURE MODEL

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**Article Info:** Received: 14 Aug 2026, Accepted: 25 Sep 2026, Published: 09 Oct 2026

## ABSTRACT:

Epilepsy is a neurological disorder marked by recurrent seizures arising from abnormal neuronal excitability and disrupted excitatory-inhibitory balance, with oxidative stress contributing significantly to disease progression. This study evaluated the antiepileptic potential of hydro alcoholic extract of *Heliotropium indicum* using a Pentylene-tetrazole (PTZ)-induced seizure model in Wistar albino rats. Phytochemical screening confirmed the presence of alkaloids, flavonoids, Phenolics, tannins, saponins, Terpenoids, steroids, and glycosides in the extract. Male Wistar rats (n=6/group) were divided into five groups and administered PTZ (80 mg/kg, i.p.) to induce seizures, followed by treatment with the extract (200 and 400 mg/kg, p.o.) or sodium valproate (200 mg/kg, p.o.) as the standard. PTZ significantly shortened seizure latency and prolonged Clonic and tonic seizure durations relative to controls. Extract treatment produced a dose-dependent anticonvulsant effect, delaying seizure onset and reducing seizure duration, with the 400 mg/kg dose showing effects comparable to the standard drug. Biochemical analysis revealed that PTZ elevated brain malondialdehyde (MDA) while depleting antioxidant enzymes (SOD, CAT) and glutathione (GSH); extract administration reversed these changes, restoring antioxidant status and lowering lipid peroxidation. These findings indicate that *H. indicum*, likely through its flavonoid and phenolic constituents, exerts anticonvulsant, antioxidant, and neuroprotective effects, potentially by enhancing inhibitory neurotransmission and mitigating oxidative neuronal damage. The extract represents a promising candidate warranting further investigation for epilepsy management.

**Keywords:** *Heliotropium indicum*, Anticonvulsant activity, Pentylene-tetrazole (PTZ), Oxidative stress, Antioxidant activity, Neuroprotection.

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**Citation:** Chippa Ramya et.al, (2026). Evaluation of Antiepileptic Activity of *Heliotropium Indicum* In Ptz Induced Seizure Model. Eco Science Journal. 2026 3(10).



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**INTRODUCTION:**

Epilepsy is a chronic neurological disorder characterized by recurrent seizures resulting from abnormal, excessive, and synchronized neuronal activity in the brain. The development of epilepsy involves complex molecular, cellular, and structural alterations that progressively transform normal neuronal networks into hyperexcitable circuits capable of generating recurrent seizures. An imbalance between excitatory and inhibitory neurotransmission, particularly involving glutamatergic and GABAergic pathways, together with alterations in ion-channel function, contributes significantly to neuronal hyperexcitability and seizure generation<sup>[1]</sup>. Recurrent seizures can produce neuronal injury and functional disturbances, increasing the risk of cognitive impairment and other neurological complications<sup>[2]</sup>.

Oxidative stress is considered an important mechanism in epileptogenesis. Excessive generation of reactive oxygen species (ROS) can cause lipid peroxidation, protein oxidation, DNA damage, mitochondrial dysfunction, and neuronal apoptosis. In addition, neuroinflammatory mediators such as interleukin-1 $\beta$ , tumor necrosis factor- $\alpha$ , and interleukin-6 may further enhance neuronal excitability and seizure susceptibility. Repeated seizure activity can therefore promote neuronal degeneration, gliosis, synaptic remodeling, and hippocampal damage, contributing to the progression of epilepsy<sup>[1]</sup>.

The management of epilepsy mainly involves antiepileptic drugs (AEDs), which act through mechanisms including modulation of voltage-gated sodium and calcium channels, enhancement of GABAergic neurotransmission, reduction of glutamatergic excitation, and regulation of synaptic transmission. Although AEDs provide effective seizure control in many patients, their use may be associated with adverse effects and drug interactions. Moreover, approximately 30% of patients continue to experience seizures despite treatment with multiple AEDs and are considered to have drug-resistant epilepsy. Alternative approaches include epilepsy surgery, neuromodulation, ketogenic dietary therapy, and emerging therapies, highlighting the continuing need for novel and safer antiepileptic agents.

Medicinal plants and their bioactive phytochemicals have gained considerable attention as potential sources of new antiepileptic agents. Flavonoids, alkaloids, Terpenoids, phenolic compounds, and saponins have been reported to possess anticonvulsant, antioxidant, anti-inflammatory, antiapoptotic, and neuroprotective properties. These compounds may exert anticonvulsant effects by enhancing GABAergic neurotransmission, reducing glutamate-mediated excitotoxicity, modulating ion channels, attenuating oxidative stress and neuroinflammation, and protecting neurons from degeneration.

Experimental models such as maximal electroshock (MES), Pentylenetetrazole (PTZ)-induced seizures, strychnine-induced seizures, kainic acid-induced epilepsy, Pilocarpine-induced status epilepticus, and kindling models are widely used for evaluating the anticonvulsant potential of natural products<sup>[3]</sup>.

Among these models, the PTZ-induced seizure model is widely employed for investigating agents that can prevent chemically induced seizures and for evaluating mechanisms associated with impaired inhibitory neurotransmission. Therefore, the identification of medicinal plants possessing both anticonvulsant and antioxidant properties may provide a promising approach for developing safer and more effective therapeutic strategies, particularly for patients with drug-resistant epilepsy<sup>[3]</sup>.

*Heliotropium indicum* (Indian heliotrope or Indian turnsole) is an erect, annual herb belonging to the family Boraginaceae and is widely distributed throughout tropical and subtropical regions of Asia, Africa, and the Americas, commonly occurring in cultivated fields, roadsides, gardens, and wastelands. The plant possesses a hairy, branched stem, simple alternate ovate to elliptic leaves, and characteristic scorpioid cymes bearing small white to pale violet flowers, with schizocarpic fruits containing hard nutlets. Traditionally used in Ayurveda and folk medicine, *H. indicum* has been employed for wounds, inflammation, pain, fever, respiratory and gastrointestinal disorders, and skin infections. Phytochemical studies have identified alkaloids, particularly pyrrolizidine alkaloids, along with flavonoids, phenolics, tannins, saponins, terpenoids, steroids, glycosides, and essential oils. These constituents are associated with diverse pharmacological activities, including antioxidant, anti-inflammatory, antimicrobial, analgesic, wound-healing, anticonvulsant, hepatoprotective, antidiabetic, and neuroprotective effects. Its reported antioxidant and anticonvulsant properties, particularly those associated with flavonoid and phenolic constituents, suggest that *H. indicum* may be a promising source of bioactive compounds for investigating novel therapeutic approaches for epilepsy; however, the presence of pyrrolizidine alkaloids necessitates careful evaluation of its safety and toxicity.

### Toxicological Studies:

**Insecticidal and toxicity studies of *Heliotropium Indicum* leaf extracts for control of insect pests of stored grains:** The leaf extracts of *Heliotropium indicum* obtained by sequential extraction method in hexane, ethyl acetate and methanol were screened for insecticidal activity on the weevils of rice, sorghum, maize and cowpea at different extract concentrations of 0.01 g/ml, 0.02 g/ml, 0.04 g/ml and 0.08 g/ml and exposure time of 24-96 h. This cage experiment was performed in triplicate and the number of dead weevils were counted and recorded each day for four days. Following phytochemical analysis of the extracts, a toxicology study was conducted on the extracts using Wistar rats. The serum liver and kidney function tests were carried out through enzyme function tests after repeated administration of the extracts. The parameters assessed were the biochemical, hematological, and histopathological functions. The result showed significant elevation in the level of ALT, AST and ALP in Hexane extract group when compared to the control group, whereas there was no significant difference between the methanol group and control group. However, a significant reduction was observed the level of Ethyl acetate extract group when compared to the control group. Similarly, compared with those of the control group, the hematological test of the rat groups showed consistent leukocyte and reticulocyte levels. The liver and kidney were assayed by histopathology, and the results showed no noticeable alteration in the architectural integrity of the investigated tissues, except for minor lesions in the livers of Hexane extract group. The study established the scientific rationale for the use of *Heliotropium indicum* as safe biopesticides to control stored grain insect pests.

### Phytochemical and Toxicological Effect of Ethanol Extract of *Heliotropium Indicum* on Liver of Male Albino Rats:

*Heliotropium indicum* is a plant of the Boraginaceae family reported to have multiple beneficial health effects, including analgesic, anti-malaria, anti-microbial, anti-tumor, anti-inflammatory, amongst others. 42 Male Wistar rats were used for this study. Group A served as control and received distilled water only. Groups B, C, D, E, and F were fed with the extract of *Heliotropium Indicum* in different concentrations. (500 mg/kg, 1000 mg/kg, 1500 mg/kg 2000 mg/kg and 3000 mg/kg) respectively. This study showed that *Heliotropium indicum* contains phytochemicals such as saponins tannins, cardiac glycosides, and terpenoids. Our results revealed that *Heliotropium indicum* decreased the ALT, AST, ALP, and bilirubin levels, especially at doses from 1500 mg/kg B.W. Also, inflammatory cytokine expressions were significantly reduced at similar doses. However, the level of antioxidants (GSH, CAT, GST, SOD, GPx) were significantly increased by *Heliotropium indicum* administration at doses ranging from 1500 mg/kg to 3000 mg/kg.

The antioxidative potentials of *Heliotropium indicum* are further complemented by its ability to reduce inflammatory cytokine expression, including TNF- $\alpha$ , IL-1 $\beta$ , IL-6, COX-2 as well as NO and LPO. Ethanol extract of *Heliotropium indicum* at doses between 1500 mg/kg and 3000 mg/kg has antioxidative, anti-inflammatory effects and can counteract lipid peroxidation.

**Evaluation of the bio-toxicity and cytogenetic effects of methanolic leaf extract of *Heliotropium indicum* L:** *Heliotropium indicum* L. has been shown to be very important in traditional healing medicine. However, many reports have indicated that the toxicity is not unconnected with pharmaceutical properties. This study therefore investigated the cytotoxic, genotoxic and biotoxic activities of methanolic leaf extract of *H. indicum* using Brine Shrimp lethality assay. The results revealed that the leaf extract of *H. indicum* showed lethality against the Brine Shrimps nauplii. The highest mortality was recorded at a concentration of 1000  $\mu$ g/ml. The LC<sub>50</sub> value of the Brine Shrimps mortality of the extract was recorded to be 461.04 $\pm$ 10.02  $\mu$ g/ml. In addition, the extract inhibited mitotic division in *A. cepa* root meristematic cells. The mitotic index was reduced from 59.99% in the control to 2.2% at 1000  $\mu$ g/ml of the leaf extract. Chromosomal aberrations were observed at different concentrations. These include sticky chromosomes at metaphase and anaphase, chromosome breakage, degenerated and disoriented chromosomes at metaphase plate. It can be concluded from this present study that methanolic leaf extract of *H. indicum* was cytotoxic and genotoxic to Brine Shrimps and *A. cepa* cells respectively. Hence, caution should be exercised with respect to the consumption of *H. indicum* leaf in any form.

**Sub-Acute Toxicity of Ethanol Leaf Extract of *Heliotropium indicum* in Male Wistar Rats: A Dose Response Study:** This study investigated the phytochemical profile and sub-acute toxicity of the ethanolic leaf extract of *Heliotropium indicum* in male rats following 28-day oral administration to evaluate its safety for traditional medicinal use. Phytochemical screening revealed high levels of alkaloids (6.3%), flavonoids (8.8%), phenolics (15.0%), and tannins (9.0%), with pyrrolizidine alkaloids likely contributing to observed toxic effects. Acute toxicity testing (OECD 423) classified the extract under GHS Category 5 (LD<sub>50</sub> > 3000 mg/kg), indicating low acute risk; however, 5000 mg/kg caused over 50% mortality within 48 hours. In the sub-acute toxicity test, doses  $\geq$ 3200 mg/kg significantly suppressed body weight gain and induced haematological alterations, including reduced RBCs, haemoglobin, and platelets, alongside elevated WBCs and neutrophilia; indicating systemic inflammation. Serum biomarkers showed dose-dependent increases in ALT, AST, urea, and creatinine, showing significant hepatorenal impairment. Morphometric analysis using Image J confirmed organ damage, with increased hepatocyte, glomerular, and cardiomyocyte dimensions; while histopathology revealed steatosis, tubular necrosis, myocardial disarray, and pulmonary oedema at high doses. Although LH, testosterone, and oestrogen remained stable, FSH reduction suggests possible reproductive toxicity. The NOAEL was established at 1600 mg/kg, with LOAEL at 3200 mg/kg. These findings significantly contribute to the understanding of *H. indicum*'s toxicity threshold, highlighting its multi-organ risk at high doses despite moderate safety at lower levels.

## MATERIALS AND METHODS:

**Collection and authentication of Plant material:** *Heliotropium indicum* was picked out for study and came from a close place in the Ranga Reddy district of Hyderabad. The parts of the plants were named correctly and checked by Dr. Madhava Chetty, who is in charge of botany at Sri Venkateshwara Academy in Tirupathi, Andhra Pradesh.

Getting plant oil ready It took 8 hours of work at 55–600C and a Soxhlet extractor to get the methanol out of the *Heliotropium indicum*. The plant was left to dry out in the shade. After passing the supernatant through Whatman filter paper No.1, it was concentrated in

in an IKA ® RB 10 Rota Evaporator (India) that was set to 44 100C and low pressure and vacuum. The extract was kept at 220C in a container with seeds that kept air out.

The percentage of extract yield was calculated by using the formula

$$\% \text{ of extract yield} = \frac{\text{weight in gm of extract obtained}}{\text{weight in gm of plant material taken}} \times 100$$

**Preliminary phytochemical screening:** The extract was subjected to qualitative phytochemical analysis following standard procedures to determine the presence of major secondary metabolites. Tests were performed for alkaloids (Dragendorff's, Mayer's, Wagner's and Hager's tests), glycosides (Test A), naphthoquinones (juglone test), anthraquinones (Bornträger's and modified Bornträger's tests), iridoidal glycosides (Trim–Hill test), coumarin glycosides (ammonia and alkaline solution tests), cyanogenetic glycosides (Grignard's test), sterols (Salkowski and Liebermann tests), triterpenoids (Liebermann–Burchard test), flavonoids (Shinoda test), and tannins (FeCl<sub>3</sub> test). The results were recorded based on the characteristic qualitative reactions observed.

**Experimental Animals<sup>[4]</sup>:** Healthy male Wistar albino rats (8–10 weeks old; 180–220 g) were acclimatized for 7 days and housed under standard conditions (22 ± 2°C, 50–60% humidity, 12 h light/dark cycle) with standard pellet diet and water *ad libitum*. The study was conducted in accordance with CPCSEA guidelines following prior IAEC approval.

PTZ-induced seizures were used to evaluate anticonvulsant activity. Five groups of six rats each (n=6) were used. The test extract of *Heliotropium indicum* and standard anticonvulsant drug were administered orally once daily. After 30 min, **PTZ (80 mg/kg, i.p.)** freshly prepared in normal saline was administered to induce seizures.

#### Experimental animal grouping<sup>[5]</sup>

Group I – Normal Control: Vehicle (0.5% Carboxymethyl Cellulose, 10 mL/kg, p.o.)

Group II – PTZ Control: Vehicle (0.5% CMC, 10 mL/kg, p.o.) + PTZ (80 mg/kg, i.p.)

Group III – Standard: Sodium Valproate (200 mg/kg, p.o.) + PTZ (80 mg/kg, i.p.)

Group IV – Test Low Dose: *Heliotropium indicum* hydro alcoholic extract (200 mg/kg, p.o.) + PTZ (80 mg/kg, i.p.)

Group V – Test High Dose: *Heliotropium indicum* hydro alcoholic extract (400 mg/kg, p.o.) + PTZ (80 mg/kg, i.p.)

**Assessing seizure latency**<sup>[6]</sup>: Five groups of six male Wistar albino rats (n = 6) were used to evaluate the anticonvulsant activity of *Heliotropium indicum* hydro alcoholic extract (200 and 400 mg/kg, p.o.) using a PTZ-induced seizure model, with sodium valproate (200 mg/kg, p.o.) as the standard. PTZ (80 mg/kg, i.p.) was administered following treatment, and animals were observed for 30 min. Seizure latency and the duration of Clonic and tonic seizures were recorded in seconds and expressed as mean  $\pm$  SEM <sup>[7,8]</sup>. Brain lipid peroxidation was assessed by estimating TBARS as MDA. Brain homogenate (10% w/v) prepared in 0.1 M phosphate buffer (pH 7.4) was centrifuged, and the supernatant was reacted with acetic acid, TBA, and SDS, heated at 95–100°C for 1 h, extracted with n-butanol:pyridine (15:1), and the absorbance was measured at 532 nm. MDA levels were expressed as nmol/mg protein <sup>[9]</sup>. SOD activity was estimated based on inhibition of NBT reduction by superoxide radicals<sup>[10]</sup>.

**Statistical analysis:** All data are expressed as Mean  $\pm$  SEM (n = 6 animals/group). Statistical comparisons among groups were performed by one-way analysis of variance (ANOVA) followed by Tukey's post-hoc multiple comparison test using GraphPad Prism software. A value of p < 0.05 was considered statistically significant. In the tables, \*p < 0.05, \*\*p < 0.01 and \*\*\*p < 0.001 denote significance versus the PTZ control group (Group II).

**Acute oral toxicity / toxicological evaluation:** The acute and sub-chronic oral toxicity of *Heliotropium indicum* has been characterised in independent published studies. Owolabi et al.<sup>[12]</sup> reported an oral LD50 of 9.78 g/kg body weight for the 70% ethanolic leaf extract in Swiss mice, and found no adverse hepatic or renal changes on 5-month repeated oral dosing of Sprague-Dawley rats at 0.5–2.0 g/kg/day, although testicular inflammatory changes were observed at the highest dose (2.0 g/kg/day). Dash and Murthy [14] reported that an OECD-423 limit-dose acute oral toxicity study of the methanolic extract (300, 600 and 2000 mg/kg) produced no mortality or overt toxicity in rats over 14 days. Boye et al. [13] similarly found the aqueous extract (1–5 g/kg) to be well tolerated acutely in mice, although 14-day repeated oral dosing at 1–2 g/kg in Sprague-Dawley rats produced pathological changes in the heart, kidney, liver and lungs, indicating a risk of cumulative toxicity with prolonged high-dose exposure. The 200 and 400 mg/kg doses employed in the present single-daily-dose PTZ-seizure protocol are therefore 24- to 49-fold below the reported acute LD50 and within the dose range shown to be free of mortality in OECD 423 limit-dose testing. Nevertheless, because *H. indicum* contains hepatotoxic pyrrolizidine alkaloids, these acute-model findings should not be extrapolated to long-term use, and a dedicated sub-chronic (28–90-day) toxicity study with liver and kidney histopathology and serum biochemistry at 200 and 400 mg/kg is recommended before the extract is considered for extended antiepileptic use.

## RESULTS :

Percentage of yield = (Weight of crude extract / Weight of dried material)  $\times$  100

Percentage of yield = (19 g / 50 g)  $\times$  100 = **38%**

| Phytochemical Constituents | Observation / Result |
|----------------------------|----------------------|
| Alkaloids                  | + Present            |
| Flavonoids                 | + Present            |
| Phenolic compounds         | + Present            |
| Tannins                    | + Present            |
| Saponins                   | + Present            |
| Terpenoids                 | + Present            |
| Steroids                   | + Present            |
| Glycosides                 | + Present            |
| Carbohydrates              | + Present            |
| Proteins                   | + Present            |
| Amino acids                | + Present            |
| Fixed oils and fats        | – Absent             |
| Anthraquinones             | – Absent             |
| Coumarins                  | + Present            |
| Quinones                   | – Absent             |

Table 1. Phytochemical Test Results.

| Groups    | Treatment                                                   | Seizure Latency (sec) |
|-----------|-------------------------------------------------------------|-----------------------|
| Group I   | Normal control (Vehicle)                                    | No seizures observed  |
| Group II  | PTZ control (80 mg/kg, i.p.)                                | 92.50 ± 6.24          |
| Group III | Sodium valproate (200 mg/kg, p.o.) + PTZ                    | 356.40 ± 18.52***     |
| Group IV  | <i>Heliotropium indicum</i> extract (200 mg/kg, p.o.) + PTZ | 198.70 ± 12.46**      |
| Group V   | <i>Heliotropium indicum</i> extract (400 mg/kg, p.o.) + PTZ | 286.80 ± 15.73***     |

Table 2. Assessing Seizure Latency.

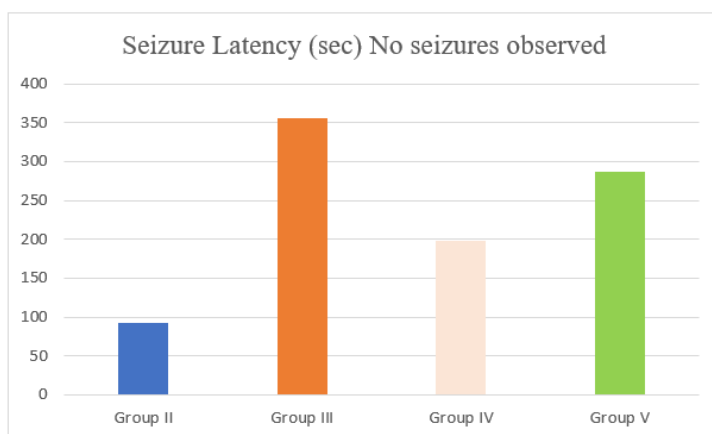
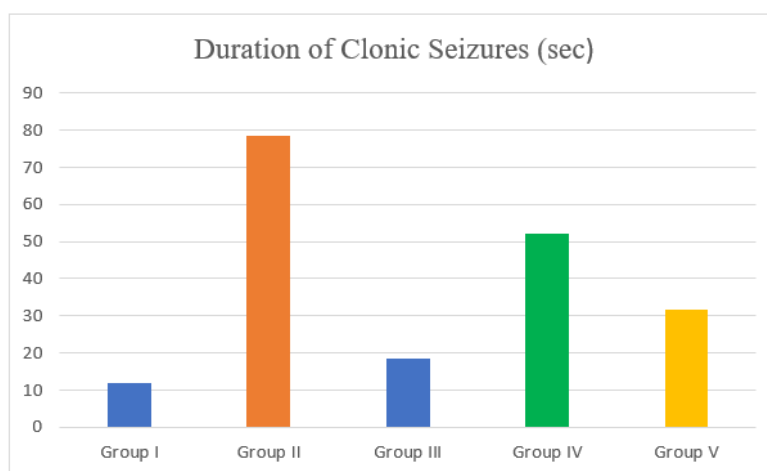


FIG-1. Seizure Latency (Sec).

**CLONIC SEIZURES:**

| Groups    | Treatment                                            | Duration of Clonic Seizures (sec) |
|-----------|------------------------------------------------------|-----------------------------------|
| Group I   | Normal control (Vehicle)                             | 0.00 ± 0.00                       |
| Group II  | PTZ control (80 mg/kg, i.p.)                         | 78.40 ± 5.62                      |
| Group III | Sodium valproate (200 mg/kg, p.o.) + PTZ             | 18.60 ± 2.14***                   |
| Group IV  | Heliotropium indicum extract (200 mg/kg, p.o.) + PTZ | 52.30 ± 4.28**                    |
| Group V   | Heliotropium indicum extract (400 mg/kg, p.o.) + PTZ | 31.70 ± 3.06***                   |

**Table 3: Duration of Clonic Seizures (sec).****FIG-2. Duration of Clonic seizures (Sec).****TONIC SEIZURES:**

| Groups    | Treatment                                            | Duration of Tonic Seizures (sec) |
|-----------|------------------------------------------------------|----------------------------------|
| Group I   | Normal control (Vehicle)                             | 0.00 ± 0.00                      |
| Group II  | PTZ control (80 mg/kg, i.p.)                         | 46.80 ± 4.35                     |
| Group III | Sodium valproate (200 mg/kg, p.o.) + PTZ             | 12.40 ± 1.86***                  |
| Group IV  | Heliotropium indicum extract (200 mg/kg, p.o.) + PTZ | 30.60 ± 3.12**                   |
| Group V   | Heliotropium indicum extract (400 mg/kg, p.o.) + PTZ | 19.20 ± 2.45***                  |

**Table 4: Duration of Tonic Seizures (sec).**

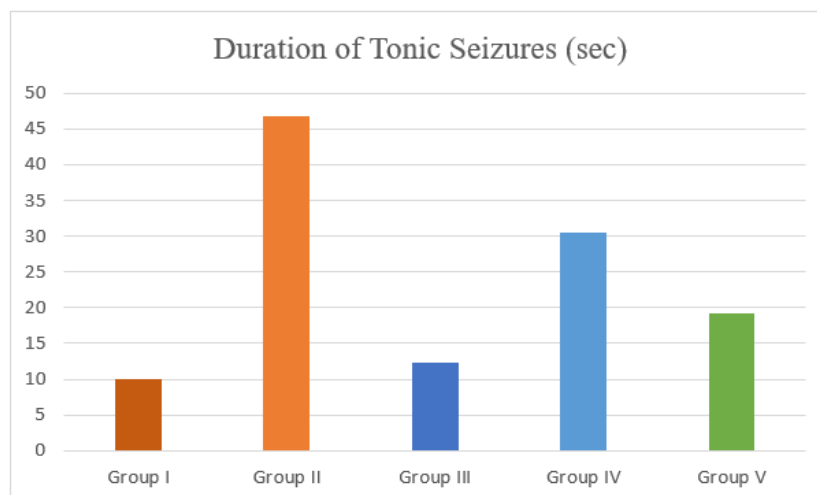


FIG-3. Duration of Tonic seizures (Sec).

Effect of *Heliotropium indicum* on Oxidative Stress Parameters in Brain Tissue:

| Groups    | Treatment                            | MDA (nmol/mg protein) | SOD (U/mg protein)    | CAT ( $\mu\text{mol H}_2\text{O}_2/\text{min/mg protein}$ ) | GSH ( $\mu\text{mol/mg protein}$ ) |
|-----------|--------------------------------------|-----------------------|-----------------------|-------------------------------------------------------------|------------------------------------|
| Group I   | Normal control                       | $1.82 \pm 0.12$       | Group I               | Normal control                                              | $1.82 \pm 0.12$                    |
| Group II  | PTZ control (80 mg/kg, i.p.)         | $4.96 \pm 0.28$       | $4.21 \pm 0.31$       | $32.60 \pm 2.24$                                            | $3.14 \pm 0.26$                    |
| Group III | Sodium valproate (200 mg/kg) + PTZ   | $2.14 \pm 0.16^{***}$ | $8.24 \pm 0.42^{***}$ | $59.80 \pm 3.05^{***}$                                      | $6.42 \pm 0.34^{***}$              |
| Group IV  | H. indicum extract (200 mg/kg) + PTZ | $3.42 \pm 0.22^{**}$  | $6.35 \pm 0.38^{**}$  | $46.70 \pm 2.86^{**}$                                       | $4.92 \pm 0.31^{**}$               |
| Group V   | H. indicum extract (400 mg/kg) + PTZ | $2.68 \pm 0.18^{***}$ | $7.58 \pm 0.41^{***}$ | $55.90 \pm 3.12^{***}$                                      | $5.86 \pm 0.35^{***}$              |

Table 5. Effect of *Heliotropium indicum* on Oxidative Stress Parameters in Brain Tissue.

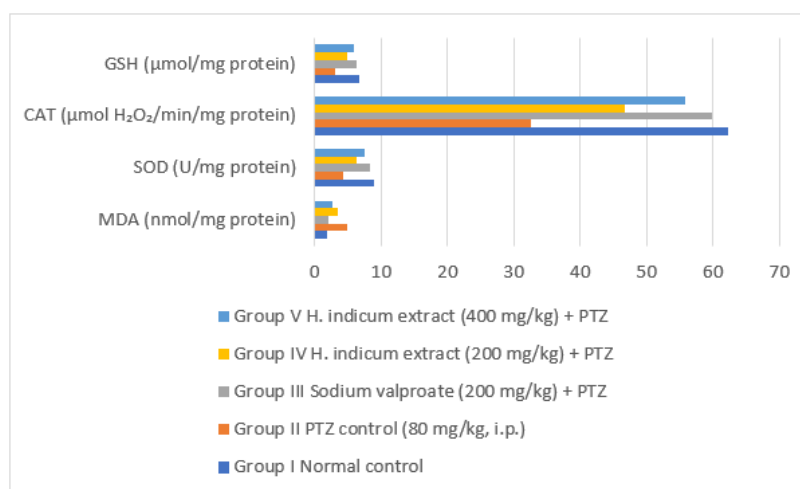


FIG-4. Graph showing information of Effect of *Heliotropium indicum* on Oxidative Stress Parameters in Brain Tissue.

## DISCUSSION:

Epilepsy is characterized by recurrent unprovoked seizures resulting from abnormal neuronal hyperexcitability and an imbalance between excitatory and inhibitory neurotransmission. The present study evaluated the anticonvulsant and antioxidant effects of the hydroalcoholic extract of *Heliotropium indicum* in PTZ-induced seizures in Wistar albino rats. PTZ (80 mg/kg, i.p.) effectively induced seizures, characterized by reduced seizure latency and increased durations of clonic and tonic seizures. Sodium valproate significantly increased seizure latency and reduced seizure duration, confirming the validity of the experimental model. Treatment with *H. indicum* extract at 200 and 400 mg/kg significantly increased seizure latency and reduced clonic and tonic seizure durations compared with the PTZ control, with the **400 mg/kg dose showing greater anticonvulsant activity**. Preliminary phytochemical screening revealed the presence of alkaloids, flavonoids, phenolic compounds, tannins, saponins, terpenoids, steroids, glycosides, and other constituents. These phytoconstituents may contribute to the observed activity through modulation of inhibitory neurotransmission and antioxidant mechanisms. PTZ administration also produced marked oxidative stress, evidenced by increased MDA and decreased SOD, CAT, and GSH levels. Treatment with *H. indicum* reduced MDA levels and restored SOD, CAT, and GSH levels, suggesting significant antioxidant potential. The anticonvulsant activity may therefore be associated with attenuation of oxidative stress and protection against seizure-induced neuronal damage. Overall, the findings suggest that *H. indicum* hydroalcoholic extract possesses **dose-dependent anticonvulsant and antioxidant properties** and may serve as a potential source for the development of novel antiepileptic agents. Further studies are required to elucidate its active phytoconstituents, molecular mechanisms, long-term efficacy, and safety profile.

## CONCLUSION:

This study evaluated the antiepileptic potential of hydro alcoholic extract of *Heliotropium indicum* in a PTZ-induced seizure model in Wistar albino rats. PTZ administration successfully induced seizures, characterized by reduced seizure latency and prolonged Clonic and tonic convulsions, confirming neuronal hyperexcitability and disrupted excitatory-inhibitory balance. Treatment with *H. indicum* extract produced significant anticonvulsant effects, increasing latency to first Clonic seizure and reducing seizure duration

compared to PTZ controls. These effects were dose-dependent, with the 400 mg/kg dose showing greater efficacy, suggesting the presence of active neuroprotective constituents capable of lowering seizure susceptibility and severity. Biochemical analysis showed that PTZ-induced seizures were accompanied by heightened oxidative stress, evidenced by elevated MDA levels and depleted antioxidant defences (SOD, CAT, GSH), confirming oxidative injury as a key contributor to PTZ-induced neuronal damage. Extract treatment significantly reversed these changes, lowering MDA and restoring antioxidant enzyme activity, indicating that its neuroprotective action likely involves reduced reactive oxygen species generation and preservation of neuronal integrity during seizures.

Phytochemical screening identified alkaloids, flavonoids, Phenolics, tannins, saponins, Terpenoids, steroids, and glycosides in the extract, which may collectively underlie its antiepileptic activity through mechanisms such as enhanced GABAergic inhibition, reduced neuronal excitation, and improved antioxidant defence.

Overall, findings indicate that *H. indicum* possesses notable anticonvulsant, antioxidant, and neuroprotective properties, with efficacy potentially comparable to standard antiepileptic therapy in controlling seizure progression and limiting oxidative brain damage. These results support *H. indicum* as a promising natural candidate for novel antiepileptic drug development. Further research—including isolation and characterization of active constituents, neurotransmitter and molecular pathway analysis, chronic seizure models, pharmacokinetic profiling, and comprehensive toxicity studies—is warranted to establish its clinical therapeutic potential and safety.

#### ACKNOWLEDGEMENTS:

We are very thankful to our principal Dr. A. Ramesh, staff members and honorable chairman Sri. K. V. Vishnu Raju garu of Vishnu Institute of Pharmaceutical Education and Research (VIPER) for providing a wonderful facility to do the research.

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