Research Article

Evaluation of the anticonvulsant and anti-inflammatory effects of ethanolic extract of *Allium cepa* peel in a pentylenetetrazol-induced seizure model in Swiss albino mice

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ABSTRACT

Background: Epilepsy is a chronic neurological disorder characterized by recurrent seizures resulting from abnormal neuronal activity. Limitations of current antiepileptic drugs, including adverse effects and drug resistance, have prompted the search for safer plant-derived alternatives. *Allium cepa* (onion) peel is a rich source of flavonoids and other phytochemicals with antioxidant and anti-inflammatory properties. **Objectives:** The purpose of this study was to evaluate the anticonvulsant and anti-inflammatory effects of ethanolic *A. cepa* peel extract (OPE) in a pentylenetetrazol (PTZ)-induced seizure model in Swiss albino mice. **Materials and Methods:** OPE was prepared by Soxhlet extraction and subjected to phytochemical screening. Swiss albino mice were divided into six groups ($n = 5$). Treatments were administered for 7 days. Seizures were induced with PTZ (45 mg/kg, i.p.). Diazepam served as the standard drug, while OPE was administered orally at 100 and 200 mg/kg, with one group receiving OPE (100 mg/kg) plus Darolac syrup. Seizure latency, severity, duration, mortality, motor coordination, brain tissue levels of interleukin-6 (IL-6) and interleukin-1β (IL-1β), and hippocampal histopathology were evaluated. Data were analyzed using one-way analysis of variance followed by Dunnett's multiple comparison test. **Results:** OPE contained flavonoids, alkaloids, tannins, and glycosides. Pre-treatment with OPE significantly delayed seizure onset, reduced seizure severity, seizure duration, and mortality, and decreased IL-6 and IL-1β levels compared with the PTZ group. The OPE (100 mg/kg) plus Darolac group showed the greatest protective effect. Histopathological examination demonstrated reduced neuronal degeneration and preservation of hippocampal architecture. **Conclusion:** OPE exhibited significant anticonvulsant and anti-inflammatory effects, suggesting its potential as a natural therapeutic candidate for epilepsy.

Keywords: *Allium cepa*, quercetin, neuroinflammation, pentylenetetrazol, epilepsy

Introduction

Epilepsy is one of the most common chronic neurological disorders, characterized by recurrent, unprovoked seizures caused by abnormal electrical activity in the brain. It affects nearly 50 million people worldwide and remains a major cause of neurological disability. Although several antiepileptic drugs (AEDs) are available, approximately one-third of patients continue to experience uncontrolled seizures due to drug-resistant epilepsy. In addition, long-term use of AEDs is associated with adverse effects such as sedation, cognitive impairment, hepatotoxicity, and drug interactions, highlighting the need for safer and more effective therapeutic agents.[1,2]

Seizures result from an imbalance between excitatory and inhibitory neurotransmission in the central nervous system. Reduced γ-aminobutyric acid (GABA)-mediated inhibition and excessive glutamatergic activity increase neuronal excitability and promote seizure generation. Besides neurotransmitter imbalance, oxidative stress, mitochondrial dysfunction, neuroinflammation, and neuronal apoptosis, these factors play important roles in epileptogenesis. Among these, neuroinflammation has emerged as a key contributor, with activated microglia and astrocytes releasing pro-inflammatory cytokines such as interleukin-1β (IL-1β) and interleukin-6 (IL-6),

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which enhance neuronal excitability and contribute to seizure progression.

The pentylenetetrazol (PTZ)-induced seizure model is widely used to evaluate anticonvulsant activity because PTZ antagonizes GABA_A receptor-mediated inhibition, producing generalized seizures similar to those observed in human epilepsy. This model allows assessment of seizure latency, seizure severity, seizure duration, mortality, and the neuroprotective effects of investigational compounds.[3]

Medicinal plants have gained increasing attention as potential sources of novel anticonvulsant agents due to their antioxidant, anti-inflammatory, and neuroprotective properties. Plant-derived phytochemicals, particularly flavonoids and phenolic compounds, have been shown to reduce oxidative stress, suppress inflammatory signaling, modulate neurotransmitter systems, and protect neurons from seizure-induced damage.[4]

Allium cepa L. (onion), a member of the family Amaryllidaceae, is widely cultivated and traditionally used for treating various inflammatory and metabolic disorders. While the edible bulb has been extensively studied, the dry outer peel is generally discarded despite containing higher concentrations of bioactive compounds than the edible portion. Onion peel is particularly rich in flavonoids such as quercetin and its glycosides, along with tannins, alkaloids, and phenolic acids. These phytochemicals possess potent antioxidant, anti-inflammatory, and neuroprotective activities. Quercetin has been reported to scavenge reactive oxygen species, inhibit the production of inflammatory cytokines, reduce oxidative stress, and modulate GABAergic neurotransmission, suggesting its potential role in seizure prevention.[5,6]

Previous studies have demonstrated that flavonoid-rich plant extracts delay seizure onset, reduce seizure severity, and protect neuronal tissue in experimental models of epilepsy. Furthermore, suppression of inflammatory cytokines such as IL-1β and IL-6 has been recognized as an important mechanism underlying the neuroprotective effects of natural products.[7,8]

Recently, increasing evidence has highlighted the important role of the gut–brain axis in the pathogenesis and progression of epilepsy. Alterations in the composition and metabolic activity of intestinal microbiota (gut dysbiosis) have been associated with increased neuroinflammation, disruption of blood–brain barrier integrity, altered neurotransmitter metabolism, and enhanced neuronal excitability. Consequently, probiotic supplementation has emerged as a potential adjunctive therapeutic approach for neurological disorders, including epilepsy. Beneficial microorganisms may restore gut microbial homeostasis, strengthen intestinal barrier function, modulate immune responses, and reduce systemic inflammation. In addition, probiotic bacteria belonging to the genera *Lactobacillus* and *Bifidobacterium* are capable of producing neuroactive metabolites such as GABA and short-chain fatty acids, which have been implicated in regulating neuronal excitability and seizure susceptibility. Darolac® syrup, a commercially available multistrain probiotic formulation, possesses immunomodulatory and anti-inflammatory properties and may enhance the neuroprotective effects of phytochemicals by modulating the gut–brain axis. Therefore, investigating Darolac as an adjunct to *A. cepa* peel extract may provide insight into the potential synergistic interaction between probiotic-mediated modulation of the gut microbiota and the antioxidant and anti-inflammatory properties of onion peel in improving anticonvulsant efficacy.

Therefore, the present study was undertaken to investigate the anticonvulsant and anti-inflammatory effects of ethanolic *A. cepa* peel extract (OPE), alone and in combination with Darolac®, in a PTZ-induced seizure model in Swiss albino mice. The study evaluated seizure latency, seizure severity, seizure duration, mortality, motor coordination, brain IL-1β and IL-6 levels, and histopathological changes to assess the neuroprotective potential of *A. cepa* peel and to determine whether probiotic supplementation could enhance its anticonvulsant efficacy.

Materials and Methods

Plant material and extraction

Fresh red onion (*A. cepa* L.) peels were collected from Rajkamal Chowk Market, Amravati, Maharashtra, India, in November 2025. The plant material was authenticated by Dr. Prashant A. Gawande, Department of Botany, Sant Gadge Baba Amravati University, Amravati. A voucher specimen (No. AM/AL/CE/510/1/1–12/2025) was deposited in the Department of Pharmacognosy, Dr. Rajendra Gode Institute of Pharmacy, Amravati.

The collected peels were washed, shade-dried, and powdered. About 120 g of powder was extracted with 650 mL of ethanol using a Soxhlet apparatus for 24 h. The extract was filtered, concentrated, and stored at 4–8°C until further use [Figure 1].

Phytochemical screening[9]

The OPE was screened qualitatively for major phytoconstituents. Flavonoids were identified using Shinoda, alkaline reagent, sulfuric acid, zinc-HCl, and lead acetate tests. Alkaloids were detected using Dragendorff's and Wagner's tests, glycosides by the Keller–Kiliani test, and tannins by standard qualitative methods.

Chemicals and animals

PTZ, diazepam, Darolac syrup, and other analytical-grade chemicals were used. Healthy Swiss albino mice (7–8 weeks, 25–30 g) of either sex were obtained from the National Institute of Biosciences, Pune, Maharashtra (CCSEA No. 2240/PO/Re/S/23/CCSEA). Animals were maintained under standard laboratory conditions (25 ± 2°C, 60–65% humidity, 12 h light/dark cycle) with food and water ad libitum.

Ethical approval and experimental design

The study was approved by the IAEC of Dr. Rajendra Gode Institute of Pharmacy, Amravati (Approval No. 2240/IAEC/25–26/01) and conducted in accordance with CCSEA guidelines.

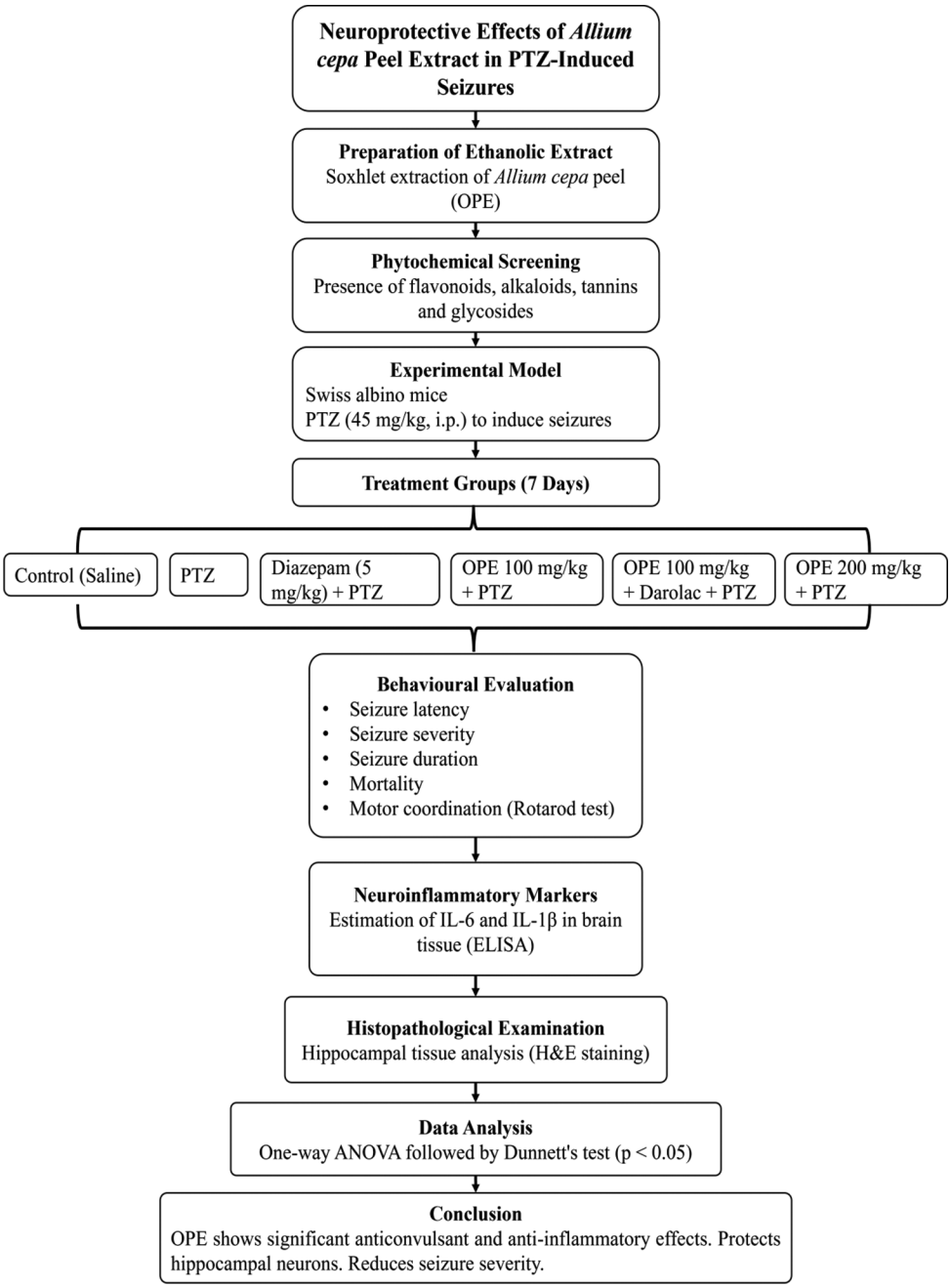

Figure 1: Schematic representation of the study methodology and evaluation of the neuroprotective effects of ethanolic *Allium cepa* peel extract in pentylenetetrazol-induced epilepsy

Thirty mice were divided into six groups ($n = 5$):

- Group I: Normal saline (1 mL/kg, i.p.)
- Group II: PTZ (45 mg/kg, i.p.)
- Group III: Diazepam (5 mg/kg, i.p.) + PTZ
- Group IV: OPE (100 mg/kg, p.o.) + PTZ
- Group V: OPE (100 mg/kg p.o) + Darolac (0.5 mL, p.o.) + PTZ
- Group VI: OPE (200 mg/kg, p.o.) + PTZ.

Treatments were administered for 7 days. On day 7, PTZ was administered 60 min after the final treatment, and seizure activity was recorded for 10 min.

PTZ-induced seizure assessment[10]

Anticonvulsant activity was evaluated by recording seizure latency, seizure severity, seizure duration, mortality, and motor coordination.

Seizure severity was scored using the modified Racine scale:

- 0: No response;
- 1: Facial and whisker twitching;
- 2: Myoclonic jerks without rearing;
- 3: Myoclonic jerks with rearing;
- 4: Clonic seizures with loss of posture;
- 5: Generalized tonic–clonic seizures.

Motor coordination was evaluated using a rotarod apparatus at 15, 20, and 25 rpm.

Estimation of neuroinflammatory markers[9]

Following behavioral assessment, brain tissues were collected and homogenized in phosphate buffer (pH 7.4). After centrifugation, the supernatants were used to estimate IL-6 and IL-1β using mouse ELISA kits. Absorbance was measured at 450 nm using a microplate reader.

Histopathological evaluation

Brain tissues were fixed in 10% neutral buffered formalin, processed, embedded in paraffin, sectioned (5 μm), and stained with hematoxylin and eosin. Hippocampal sections were examined for neuronal degeneration, vacuolization, inflammatory changes, and tissue architecture.

Statistical analysis

Data were expressed as the mean ± standard error of the mean and analyzed using GraphPad Prism 10. One-way analysis of variance followed by Dunnett's multiple comparison test was applied. Statistical significance was considered at $P < 0.05$.

Results

Preliminary phytochemical screening

The ethanolic extract of *A. cepa* peel (OPE) showed the presence of major phytoconstituents, including flavonoids, alkaloids, glycosides, and tannins. Flavonoids were confirmed by Shinoda, alkaline reagent, sulfuric acid, zinc-HCl, and lead acetate tests, while alkaloids and glycosides were detected using Dragendorff's/Wagner's and Keller–Kiliani tests, respectively [Table 1].

Effect of OPE on PTZ-induced seizure behavior

PTZ administration produced significant seizures characterized by reduced latency, increased seizure severity, prolonged seizure duration, and mortality. Treatment with OPE showed protective effects against PTZ-induced seizures. OPE 100 mg/kg alone showed moderate protection, whereas OPE 100 mg/kg combined with Darolac and OPE 200 mg/kg significantly delayed seizure onset, reduced seizure severity, and decreased seizure duration compared with the PTZ group.

The OPE + Darolac group showed the maximum anticonvulsant activity, increasing seizure latency to 68.6 ± 1.2 s, reducing the seizure severity score to 3.8 ± 0.49, and decreasing seizure duration to 38.8 ± 1.2 s. Diazepam showed complete protection against PTZ-induced seizures, as no seizure event occurred during the observation period [Table 2]. The effects of the treatments on latency to first seizure, seizure severity, seizure duration, and mortality are illustrated in Figure 2a-d, respectively.

Effect of OPE on motor coordination

The rotarod test demonstrated that OPE-treated animals maintained normal motor coordination at 15, 20, and 25 rpm. No significant motor impairment was observed, indicating that the anticonvulsant activity was not associated with severe neuromuscular dysfunction [Table 3 and Figure 3].

Effect of OPE on neuroinflammatory cytokines

PTZ administration significantly increased brain IL-6 and IL-1β levels compared with the control group. Treatment with OPE significantly reduced inflammatory cytokine levels; IL-6 levels decreased from 43.32 ± 0.16 pg/mL in the PTZ group to 28.40 ± 0.63 pg/mL in the OPE 100 mg/kg + Darolac group. Similarly, IL-1β levels decreased from 42.34 ± 0.21 pg/mL to 30.45 ± 0.51 pg/mL [Table 4; Figure 4a and b].

Table 1: Preliminary phytochemical screening of ethanolic *Allium cepa* peel extract

Phytochemical test	Observation
Shinoda test (Flavonoids)	Positive (+)
Sulphuric acid test (Flavonoids)	Positive (+)
Alkaline reagent test (Flavonoids)	Positive (+)
Lead acetate test (Flavonoids)	Positive (+)
Dragendorff's test (Alkaloids)	Positive (+)
Wagner's test (Alkaloids)	Positive (+)
Keller–Kiliani test (Glycosides)	Positive (+)
Test for tannins	Positive (+)

Data are presented as (+), phytochemical detected (positive test); (−), phytochemical not detected (negative test). OPE: Ethanolic *Allium cepa* peel extract

Table 2: Effect of OPE on behavioral parameters in the PTZ-induced seizure model

Group	Latency to seizure (sec)	Seizure severity score	Seizure duration (sec)	Mortality
Control	0.0±0.0	0.0±0.0	0.0±0.0	0/5
PTZ	40.2±2.0	5.0±0.0	77.8±1.1	5/5
Diazepam	0.0±0.0	0.0±0.0	0.0±0.0	0/5
OPE 100 mg/kg	47.4±2.0	5.0±0.0	60.8±0.9	4/5
OPE 100 mg/kg +Darolac	68.6±1.2	3.8±0.49	38.8±1.2	3/5
OPE 200 mg/kg	66.8±1.4	4.6±0.24	50.4±1.3	4/5

Values are expressed as mean±SEM (n=5). Data were analyzed using one-way analysis of variance followed by Dunnett's multiple comparison test. $P<0.05$ was considered statistically significant. OPE: Ethanolic *Allium cepa* peel extract, PTZ: Pentylenetetrazol, SEM: Standard error of the mean, sec: Seconds, mg/kg: Milligrams per kilogram

Table 3: Effect of OPE on rotarod performance

Treatment	15 rpm (min)	20 rpm (min)	25 rpm (min)
OPE 100 mg/kg	5.4±1.2	5.8±0.8	5.7±0.7
OPE 100 mg/kg+Darolac	5.0±1.3	5.2±0.9	5.4±1.4
OPE 200 mg/kg	5.3±1.1	5.4±1.2	5.4±1.2

Values are expressed as mean±SEM ($n=5$). Data were analyzed using one-way analysis of variance followed by Dunnett's multiple comparison test. No statistically significant differences were observed among treatment groups ($P>0.05$). OPE: Ethanolic *Allium cepa* peel extract, rpm: Revolutions per minute, SEM: Standard error of the mean, min: Minutes

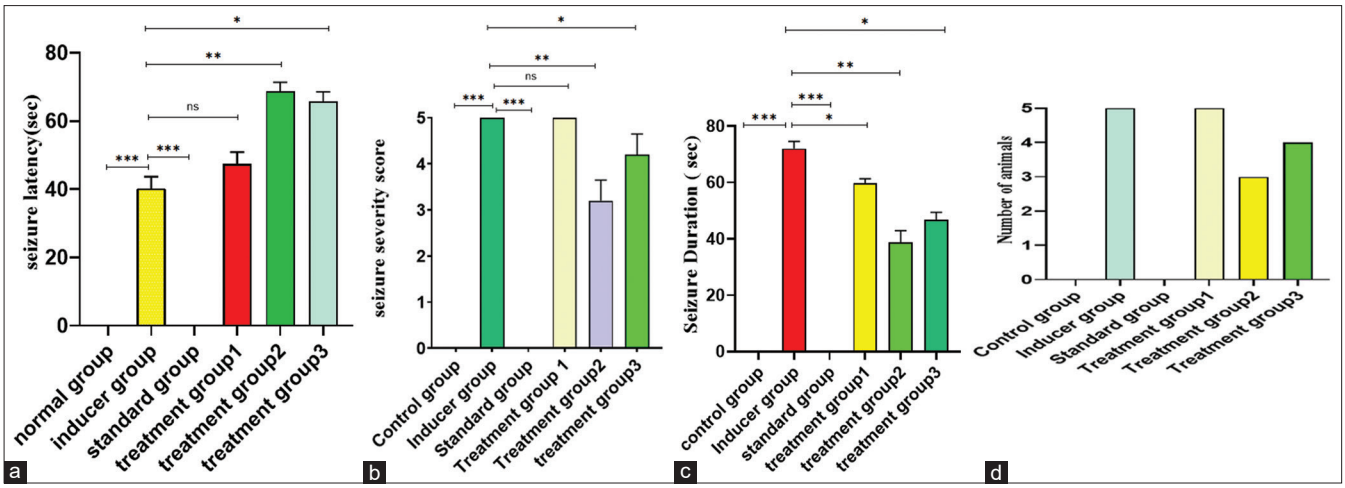

Figure 2: (a) Latency to first seizure. (b) Seizure severity assessment. (c) Seizure duration assessment. (d) Mortality assessment

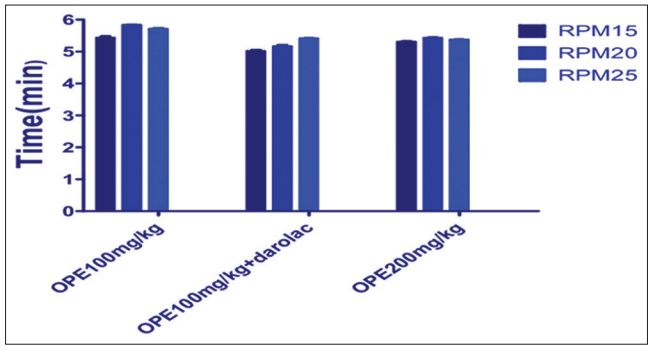

Figure 3: Rotarod test

Table 4: Effect of OPE on inflammatory cytokines in brain tissue

Group	IL-6 (pg/mL)	IL-1β (pg/mL)
Control	19.73±0.14	26.46±0.18
PTZ	43.32±0.16	42.34±0.21
Diazepam	25.65±0.43	29.45±0.51
OPE 100 mg/kg	33.43±0.58	38.23±0.49
OPE 100 mg/kg+Darolac	28.40±0.63	30.45±0.51
OPE 200 mg/kg	40.45±0.82	35.34±0.51

Values are expressed as mean±SEM (n=5). Data were analyzed using one-way analysis of variance followed by Dunnett's multiple comparison test. IL-6: Interleukin-6, IL-1β: Interleukin-1 beta, pg/mL: Picograms per milliliter, OPE: Ethanolic *Allium cepa* peel extract, PTZ: Pentylenetetrazol, SEM: Standard error of the mean

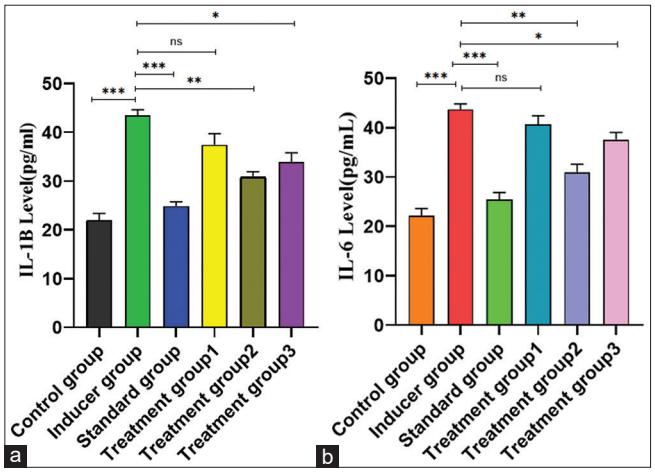

Figure 4: (a) Interleukin-1β levels. (b) Interleukin-6 levels

Histopathological evaluation

Histological examination revealed normal hippocampal architecture in control animals. PTZ administration caused neuronal shrinkage, vacuolization, irregular cellular arrangement, and increased glial cell infiltration. Diazepam and OPE treatments reduced these pathological alterations. The OPE 100 mg/kg + Darolac group showed the greatest preservation of hippocampal architecture, followed by the OPE 200 mg/kg treatment [Figure 5].

Seizure behavioral observations

PTZ-treated animals exhibited facial twitching, myoclonic jerks, rearing, clonic seizures, hind limb extension, Straub tail response, and generalized tonic–clonic seizures. OPE treatment reduced the progression and severity of seizure manifestations, demonstrating protective activity against PTZ-induced seizures [Figure 6].

Discussion

Epilepsy is a chronic neurological disorder characterized by recurrent seizures resulting from abnormal neuronal excitability. Increasing evidence indicates that oxidative stress, neuroinflammation, mitochondrial dysfunction, and neurotransmitter imbalance contribute significantly to seizure initiation, progression, and neuronal injury. AEDs are available for clinical management; their long-term use is frequently associated with adverse effects and drug resistance, highlighting the need for safer and more effective therapeutic alternatives, particularly those derived from natural products.

In the present study, the ethanolic extract of *A. cepa* peel (OPE) demonstrated significant anticonvulsant and neuroprotective effects in the PTZ-induced seizure model. Preliminary phytochemical screening confirmed the presence of flavonoids, alkaloids, tannins, and glycosides. Onion peel is particularly rich in flavonoids,

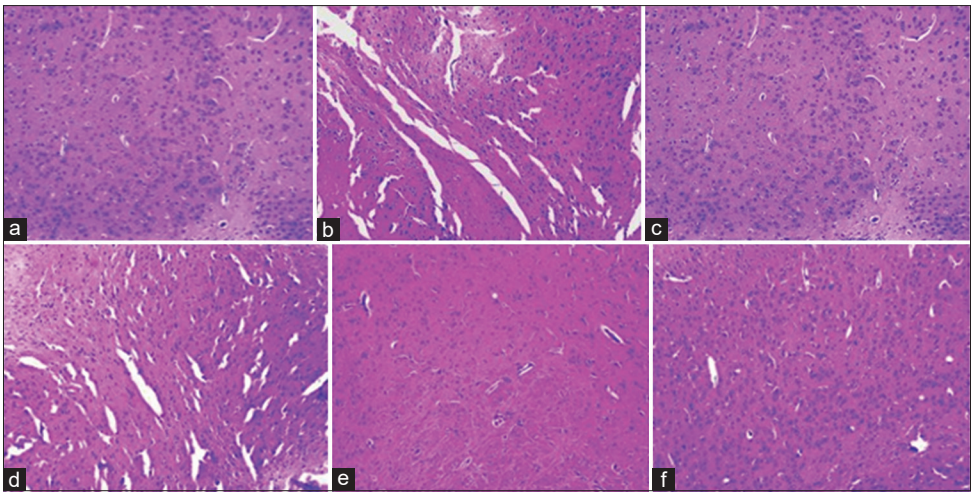

Figure 5: Histopathological changes in the disease control group compared with the control group. (a) Control Group, (b) Disease Control Group, (c) Standard Group, (d) Treatment Group (OPE 100 mg/kg), (e) Treatment Group (OPE 100 mg/kg+Darolac), and (f) Treatment Group (OPE 200 mg/kg). OPE: Ethanolic *Allium cepa* peel extract

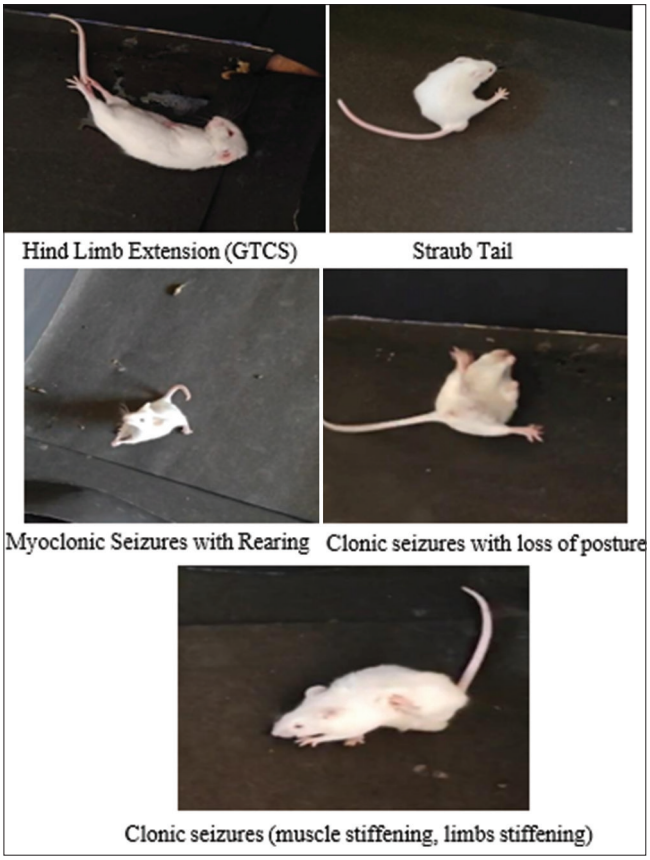

Figure 6: Different phases of seizures

especially quercetin and its derivatives, which are well recognized for their antioxidant, anti-inflammatory, and neuroprotective activities.[11,12]

PTZ administration produced characteristic seizure manifestations, including reduced seizure latency, increased seizure severity, prolonged seizure duration, and mortality. PTZ induces seizures primarily through antagonism of GABAergic neurotransmission, resulting in enhanced neuronal excitability, and is therefore widely used for evaluating anticonvulsant agents. Treatment with OPE significantly delayed seizure onset and reduced seizure severity and duration, with the greatest protection observed in the OPE 100 mg/kg + Darolac and OPE 200 mg/kg groups. These findings suggest that the anticonvulsant activity of OPE may be attributed to flavonoid-mediated antioxidant effects, modulation of inhibitory neurotransmission, and stabilization of neuronal excitability.[9]

An important finding of the present study was that the combination of OPE (100 mg/kg) with Darolac produced greater anticonvulsant activity than OPE administered alone. The combination-treated group exhibited prolonged seizure latency, reduced seizure severity and duration, lower levels of pro-inflammatory cytokines, and improved histopathological architecture. Although the exact mechanism underlying this enhanced effect was not investigated, the findings suggest a possible additive or synergistic interaction between the neuroprotective phytochemicals present in onion peel and the probiotic formulation.

Growing evidence indicates that the gut–brain axis plays an important role in regulating central nervous system function through bidirectional communication involving immune signaling, microbial metabolites, neurotransmitter production, and maintenance of intestinal barrier integrity. Alterations in gut microbiota have been associated with increased neuroinflammation, enhanced neuronal excitability, and seizure susceptibility. Probiotic formulations such as Darolac may contribute to the restoration of microbial homeostasis, the suppression of systemic inflammatory responses, and the production of neuroactive metabolites, including GABA and short-chain fatty acids, which have been reported to modulate neuronal excitability, improve blood–brain barrier integrity, and reduce microglial activation. Simultaneously, the flavonoids and phenolic compounds present in *A. cepa* peel, particularly quercetin, possess potent antioxidant and anti-inflammatory properties, that

may attenuate oxidative stress and inflammatory signaling pathways. Therefore, it is plausible that probiotic-mediated modulation of the gut microbiota, together with the neuroprotective phytochemicals present in onion peel, may have contributed to the superior anticonvulsant and anti-inflammatory effects observed in the combination-treated group.

Oxidative stress is recognized as a major contributor to seizure-induced neuronal injury through the excessive generation of reactive oxygen species, mitochondrial dysfunction, lipid peroxidation, and neuronal apoptosis. The antioxidant properties of onion peel flavonoids, particularly quercetin, may therefore contribute to neuronal protection by scavenging reactive oxygen species, enhancing endogenous antioxidant defense systems, and preserving mitochondrial function.[13]

Neuroinflammation is another critical factor involved in epileptogenesis and seizure progression. In the present study, PTZ administration significantly increased brain IL-1β and IL-6 levels, indicating activation of inflammatory pathways. Elevated concentrations of these cytokines are known to enhance neuronal excitability and disrupt neurotransmitter homeostasis, thereby facilitating the development of seizures. Treatment with OPE significantly reduced IL-1β and IL-6 levels, suggesting that suppression of neuroinflammatory responses may contributes to its anticonvulsant activity. Similar anti-inflammatory effects of onion-derived flavonoids have been attributed to inhibition of NF-κB signaling and downregulation of pro-inflammatory cytokine production.[14]

Histopathological evaluation further supported the neuroprotective potential of OPE. Brain sections from PTZ-treated animals showed marked neuronal degeneration, vacuolization, and disruption of normal hippocampal architecture. In contrast, OPE-treated groups exhibited substantially reduced neuronal damage and improved preservation of hippocampal structure. The combination treatment demonstrated comparatively greater histological protection, consistent with the behavioral and biochemical findings, suggesting that attenuation of oxidative stress and neuroinflammation contributed to preservation of neuronal integrity.

Overall, the present study demonstrates that OPE exerts significant anticonvulsant and neuroprotective effects against PTZ-induced seizures by improving behavioral outcomes, suppressing neuroinflammatory cytokines, and preserving hippocampal architecture. Furthermore, coadministration with Darolac produced superior protective effects, suggesting that probiotic supplementation may enhance the therapeutic efficacy of onion peel extract. These findings support the potential of combining phytochemicals with probiotics as an adjunctive strategy for epilepsy management, although further mechanistic and clinical studies are warranted.

Limitation

Despite these promising findings, the present study has certain limitations. Although Darolac enhanced the anticonvulsant and anti-inflammatory effects of OPE, changes in gut microbial composition, short-chain fatty acid production, GABA-producing bacterial populations, intestinal barrier integrity, and gut–brain signaling pathways were not evaluated. Consequently, the involvement of the gut–brain axis remains a biologically plausible but unconfirmed mechanism. Future investigations incorporating microbiome profiling, metabolomic analysis, neurotransmitter quantification, and molecular pathway studies are required to elucidate the mechanistic basis of the observed synergistic effects.

Conclusion

The present study demonstrates that the ethanolic extract of *A. cepa* peel exhibits significant anticonvulsant and neuroprotective activity against PTZ-induced seizures in mice. OPE improved seizure latency, reduced seizure severity and duration, decreased neuroinflammatory cytokine levels, and protected hippocampal neuronal architecture. These effects may be attributed to the presence of flavonoids and other bioactive phytoconstituents with antioxidant and anti-inflammatory properties. The findings suggest that *A. cepa* peel may be a potential natural therapeutic candidate for the management of epilepsy.

Acknowledgments

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