RESEARCH PAPER

ANTIEPILEPTIC AND ANTIOXIDANT EFFECTS OF PRUNUS DULCIS VAR. AMARA: EVIDENCE FROM IN VITRO, IN VIVO, AND BIOCHEMICAL STUDIES

Sneha Yadav[1]*, Brijesh Sirohi[2], Naveen Gupta[3], Pinky Singh[4], Ganesh Prasad Patel[5]

[1*]Patel college of Pharmacy, Madhyanchal Professional University, Madhya Pradesh, Email Id: ysneha.yadav16@gmail.com ORCID:0000-0002-7378-922X
[2]Radharaman college of pharmacy Bhopal Madhya Pradesh, Email Id: brijeshsirohi.rcp@gmail.com ORCID:0009-0006-7913-1530
[3]Patel college of Pharmacy, Madhyanchal Professional University, Madhya Pradesh, Email Id: naveenmpharm@gmail.com
[4]Patel college of Pharmacy, Madhyanchal Professional University, Madhya Pradesh, Email Id: pinkysingh1790@gmail.com ORCID:0000-0001-4627-8564
[5]Patel college of Pharmacy, Madhyanchal Professional University, Madhya Pradesh, Email Id: ganeshpatel269@gmail.com

***Corresponding Author**
Email: ysneha.yadav16@gmail.com

Received: 25th May, 2026; **Revised:** 6th June, 2026; **Accepted:** 9th Aug, 2026; **Available Online:** 20th Aug, 2026

ABSTRACT

Background: Epilepsy is a chronic neurological disorder characterized by recurrent seizures resulting from abnormal neuronal hyperexcitability. Although currently available antiepileptic drugs effectively control seizures in many patients, their long-term use is often associated with adverse effects and drug resistance, highlighting the need for safer and more effective therapeutic alternatives. Prunus dulcis var. amara (bitter almond) is a medicinal plant rich in flavonoids and phenolic compounds with reported antioxidant and neuroprotective properties; however, its antiepileptic potential remains insufficiently explored.

Objective: The present study aimed to evaluate the antioxidant and antiepileptic activities of Prunus dulcis var. amara through integrated in vitro and in vivo investigations.

Materials and Methods: The hydroalcoholic extract of P. dulcis var. amara was subjected to phytochemical screening and evaluated for antioxidant activity using standard in vitro assays, including DPPH and ABTS radical scavenging assays, with ascorbic acid as the reference standard. Antiepileptic activity was assessed in pentylenetetrazole (PTZ)-induced seizure model in Wistar rats. Animals were treated with different doses of the extract, while diazepam served as the standard antiepileptic drug. Seizure latency, seizure duration, seizure score, and mortality were recorded. Biochemical estimations of oxidative stress markers, including superoxide dismutase (SOD), catalase (CAT), reduced glutathione (GSH), and malondialdehyde (MDA), were performed to evaluate the neuroprotective effect of the extract.

Results: The extract exhibited concentration-dependent free radical scavenging activity in all in vitro antioxidant assays, demonstrating significant antioxidant potential. In the PTZ-induced epilepsy model, P. dulcis var. amara significantly delayed seizure onset, reduced seizure severity and duration, and improved seizure protection compared with the disease control group. Treatment also restored endogenous antioxidant enzyme activities by significantly increasing SOD, CAT, and GSH levels while reducing MDA concentration, indicating attenuation of seizure-induced oxidative stress. The observed pharmacological effects are likely attributable to the presence of flavonoids and phenolic constituents, particularly quercetin, kaempferol, rutin, catechin, and chlorogenic acid.

Conclusion: The findings demonstrate that Prunus dulcis var. amara possesses significant antioxidant and antiepileptic activities, which may be mediated through its ability to suppress oxidative stress and protect neuronal tissue from seizure-induced damage. These results provide experimental evidence supporting the therapeutic potential of P. dulcis var. amara as a promising plant-derived candidate for epilepsy management. Further mechanistic investigations and clinical studies are warranted to validate its efficacy and safety.

Keywords: Prunus dulcis var. amara; epilepsy; pentylenetetrazole; antioxidant; flavonoids; neuroprotection; oxidative stress; anticonvulsant activity.

How to cite this article: Yadav S, Sirohi B, Gupta N, Singh P, Patel GP., Antiepileptic and Antioxidant Effects of Prunus dulcis var. amara: Evidence from In Vitro, In Vivo, and Biochemical Studies. Int J Drug Deliv Technol. 2026;16(76s): 464; DOI: 10.25258/ijddt.16.76s.40

Source of support: Nil.

Conflict of interest: Nil.

INTRODUCTION

Epilepsy is one of the most common chronic neurological disorders, affecting more than 50 million people worldwide. It is characterized by recurrent, unprovoked seizures caused by abnormal, excessive, and synchronized neuronal discharges in the brain. The disorder has a multifactorial etiology, including genetic predisposition, traumatic brain injury, stroke, central nervous system infections, brain tumors, and developmental abnormalities. Seizures are broadly classified as focal or generalized according to the origin and propagation of abnormal electrical activity. Despite substantial advances in diagnosis and therapeutic interventions, epilepsy remains a major global health challenge due to its high prevalence, risk of disability, reduced quality of life, and significant socioeconomic burden (1,2).

The pathogenesis of epilepsy involves a complex interplay of molecular and cellular mechanisms. A hallmark of epileptogenesis is the imbalance between excitatory glutamatergic and inhibitory γ-aminobutyric acid (GABA)-ergic neurotransmission, resulting in neuronal hyperexcitability and hypersynchronous firing. In addition, accumulating evidence indicates that oxidative stress, neuroinflammation, mitochondrial dysfunction, apoptosis, ion channel abnormalities, and dysregulated intracellular signaling significantly contribute to seizure initiation and progression. Excessive generation of reactive oxygen species (ROS) during seizures promotes lipid peroxidation, protein oxidation, DNA damage, and neuronal cell death, thereby exacerbating epileptic pathology. Consequently, agents capable of simultaneously suppressing seizure activity and attenuating oxidative stress represent promising therapeutic candidates for epilepsy management (3).

Antiepileptic drugs (AEDs) remain the primary therapeutic option for seizure control and exert their effects by modulating voltage-gated ion channels, enhancing GABAergic neurotransmission, or inhibiting glutamatergic excitation. Although currently available AEDs successfully control seizures in many patients, approximately 30% develop drug-resistant epilepsy. Furthermore, prolonged AED therapy is frequently associated with adverse effects, including sedation, cognitive impairment, dizziness, hepatotoxicity, teratogenicity, psychiatric disturbances, and metabolic complications, which often compromise treatment adherence and quality of life. These limitations underscore the need for safer therapeutic agents possessing both anticonvulsant and neuroprotective properties (4).

Natural products have emerged as an important source of novel pharmacologically active molecules for neurological disorders. Numerous medicinal plants contain flavonoids, phenolic acids, alkaloids, terpenoids, and other secondary metabolites that exhibit antioxidant, anti-inflammatory, antiapoptotic, and neuroprotective activities. Experimental studies have demonstrated that these phytoconstituents can reduce seizure susceptibility through modulation of GABAergic signaling, inhibition of glutamate-mediated excitotoxicity, suppression of oxidative stress, attenuation of neuroinflammation, and preservation of mitochondrial integrity. Therefore, medicinal plants continue to attract considerable interest as complementary or alternative therapeutic approaches for epilepsy (5).

Prunus dulcis var. *amara* (bitter almond), a member of the family Rosaceae, has long been used in traditional systems of medicine, including Ayurveda, Unani, Persian, and European herbal medicine. Traditionally, processed bitter almond preparations have been employed as sedatives, antispasmodics, analgesics, expectorants, and remedies for inflammatory disorders and muscular pain. Although raw bitter almonds contain the cyanogenic glycoside amygdalin, appropriate detoxification procedures allow their safe medicinal use. Recent phytochemical investigations have demonstrated that *P. dulcis* var. *amara* is a rich source of bioactive constituents, including flavonoids (quercetin, kaempferol, rutin, isorhamnetin, catechin, and epicatechin), phenolic acids (chlorogenic acid), phytosterols (β-sitosterol and stigmasterol), triterpenoids (ursolic and oleanolic acids), tannins, alkaloids, and volatile compounds such as benzaldehyde. These phytochemicals possess potent antioxidant and anti-inflammatory activities that are closely associated with neuroprotection (6).

Among these constituents, flavonoids such as quercetin and kaempferol have attracted particular attention because of their ability to modulate GABAergic neurotransmission, scavenge reactive oxygen species, inhibit neuroinflammatory mediators, and protect neurons against excitotoxic injury. Previous pharmacological studies have demonstrated that *P. dulcis* var. *amara* exhibits antioxidant, anti-inflammatory, anxiolytic, analgesic, sedative, antispasmodic, and neuroprotective activities. However, systematic evaluation of its anticonvulsant potential and the contribution of its antioxidant properties to seizure protection remains limited (7).

Considering the critical role of oxidative stress in epileptogenesis and the abundance of neuroprotective phytochemicals present in *P. dulcis* var. *amara*, further investigation of its antiepileptic activity is scientifically warranted. Therefore, the present study was undertaken to evaluate the antiepileptic potential of *P. dulcis* var. *amara* using integrated in vitro and in vivo approaches. The antioxidant activity of the extract was assessed using established in vitro assays, while anticonvulsant efficacy was evaluated in a pentylenetetrazole (PTZ)-induced experimental model of epilepsy. In

addition, oxidative stress-associated biochemical parameters were determined to elucidate the possible mechanisms underlying its neuroprotective effects. The findings of this study may provide experimental evidence supporting the development of *P. dulcis* var. *amara* as a promising plant-derived therapeutic candidate for epilepsy (8).

1.0 Materials and Methods

2.1 Plant identification

PDVA seeds were collected from local regions of Prayagraj (Uttar Pradesh). Further it is verified from Dr. Alok Shrivastava from the Department of Botany at Mahatma Jyotiba Phule Rohilkhand University (Bareilly), India

2.2 Plant Extract

The finely powdered Prunus dulcis var. amara seeds were first defatted using petroleum ether (60–80 °C) in a Soxhlet apparatus for 6 hours to remove fixed oils. The defatted residue was then soaked in distilled water (1:5 w/v) for 4 hours to allow endogenous emulsin enzymes to hydrolyze and volatilize hydrogen cyanide (HCN) from cyanogenic glycosides. The aqueous layer was decanted, and the residue was repeatedly washed with distilled water until no characteristic almond odor of HCN was detected. The detoxified powder was then air-dried and used for extraction. The treated seed powder was extracted with 80% methanol in a Soxhlet extractor for 8 hours. The extract was filtered and concentrated under reduced pressure using a rotary evaporator to obtain a semi-solid methanolic extract of Prunus dulcis var. amara (MEPDA). This crude extract, free of cyanogenic glycosides, was subjected to chromatographic separation for the isolation of quercetin (9).

2.3 In-vitro Antioxidant activity:

2.3.1 DPPH antiradical capacity

The evaluation of the DPPH radical scavenging ability was one of the methods used to ascertain the in vitro antioxidant capacity. A DPPH solution (0.004% concentration in methanol) was combined with 1 ml of the extract, which had been prepared at various concentrations ranging from 10 to 100 µg/ml, for this test. After that, these mixes were allowed to sit at room temperature for half an hour in the dark. Using ascorbic acid as the reference standard, the change in absorbance at 517 nm was measured using a UV Visible spectrophotometer (Labindia 3000+). A control, consisting of 1 milliliter of methanol and 3 milliliters of the DPPH solution, was also created by substituting methanol for the extract or ascorbic acid. The formula was used to determine the percentage of DPPH radical inhibition in order to assess the IC50 value (10).

2.3.2 Nitric oxide scavenging activity

In order to test for nitric oxide scavenging capacity, sodium nitroprusside ($Na_2[Fe(CN)_5NO]$) was added to water that had been adjusted to a physiological pH, which naturally causes nitric oxide to develop. Following an interaction with oxygen, this NO (nitric oxide) forms NO^{-}_2 (nitrite ions), which can be detected using the Griess Ilosvoy technique. This approach involved mixing equimolar volumes of phosphate-buffered saline (pH 7.4) and plant extract (0.5 ml) at different concentrations with 2 ml of 10 mM sodium nitroprusside. For two and a half hours, the mixture was incubated at room temperature. One milliliter of naphthylethylenediamine dihydrochloride (0.1% w/v) was added to half a milliliter of this mixture, which had been incubated for 30 minutes at room temperature after being treated with Griess reagent and allowed to stand for 5 minutes. Using ascorbic acid as a reference standard and the following formula, the percentage of nitric oxide scavenged was determined by spectrophotometric measurement of absorbance at 546 nm (64): % NO radical scavenging activity = [(Acontrol – A test)/Acontrol] × 100 (11)

2.4 Animals

Animals were purchased From CDRI (Central drug research institute) animal house having weight ranges in between 150-210 gm and 10-12 weeks old of albino wistar rats., for our research purpose. Storage condition maintained at 12hr dark condition and 12hr light condition, maintained at 24± 2°C conditions is maintained. During research protocol diet was pellet and ad libitum water. Accommodation of the experimental animals were kept in isolation specified area in animal house at-least before the 14 days prior to start the protocol for the experiment, which is approved by I.A.E.C Institutional Animal Ethics Committee having.

2.5 Drugs and Chemicals and Experimental design

2.5.1 Chemicals and Reagents

Pentylenetetrazole (PTZ; CAS No. 54-95-5) was procured from Sigma-Aldrich (Merck Life Science Pvt. Ltd., Bengaluru, Karnataka, India) and used for the induction of experimental seizures. Diazepam (CAS No. 439-14-5) was obtained from Sigma-Aldrich (Merck Life Science Pvt. Ltd., Bengaluru, Karnataka, India) and served as the standard antiepileptic drug. L-Ascorbic acid (CAS No. 50-81-7) was purchased from Sigma-Aldrich (Merck Life Science Pvt. Ltd., Bengaluru, Karnataka, India) and used as the reference antioxidant in the in vitro antioxidant assays.

All other analytical-grade chemicals and reagents, including methanol, ethanol, dimethyl sulfoxide (DMSO), hydrochloric acid, sulfuric acid, sodium carbonate, sodium hydroxide, aluminum chloride, Folin–Ciocalteu reagent, 2,2-diphenyl-1-picrylhydrazyl (DPPH; CAS No. 1898-66-4), 2,2′-azinobis-(3-ethylbenzothiazoline-6-sulfonic acid) (ABTS; CAS No. 30931-67-0), potassium persulfate (CAS No. 7727-21-1), ferric chloride (CAS No. 7705-08-0), ferrous sulfate (CAS No. 7782-63-0), trichloroacetic acid (CAS No. 76-03-9), thiobarbituric acid (CAS No. 504-17-6), hydrogen

peroxide (CAS No. 7722-84-1), and nitroblue tetrazolium (NBT; CAS No. 298-83-9) were of analytical grade and purchased from Sigma-Aldrich (Merck Life Science Pvt. Ltd., Bengaluru, Karnataka, India), HiMedia Laboratories Pvt. Ltd. (Mumbai, Maharashtra, India), and SRL Pvt. Ltd. (Mumbai, Maharashtra, India). Double-distilled water was used throughout the study for the preparation of all solutions and reagents.

2.5.2 Pentylenetetrazole (PTZ) induced convulsion

The use of PTZ to induce seizures in rodents is considered a standard experimental model for investigating myoclonic convulsions similar to those seen in humans. Clonic convulsions were induced in rats via subcutaneous injection of PTZ at a dosage of 100 mg/kg. Following PTZ administration, each rat was placed in a transparent Plexiglas enclosure and observed for 30 minutes to identify the onset of clonic seizures. The seizures were characterized by prolonged whole-body clonus lasting over 3 seconds, along with the absence of the righting reflex (12).

2.5.3 Estimation of biochemical parameters in rat brain homogenate

2.5.3.1 Determination of superoxide dismutase

The Kakar et al. approach was used to find out if P. alata extract might boost antioxidant enzymes like Superoxide dismutase. First, the crude brain solution was centrifuged at 1500 rpm for 10 minutes and 10,000 rpm for 15 minutes. The supernatant was then collected to make the rat brain homogenate solution. There were only a few changes made to the assay. It began by mixing 0.1 ml of 5-Methylphenazinium methyl sulfate (186 µmol), sodium pyrophosphate-buffer (0.052 mmol, 1.2 ml, pH 7.0), and 0.3 ml of the homogenate that had been prepared. The process started when 0.2 ml of NADH (780 µmol) was added, and it stopped after one minute when 1 ml of acetic acid (glacial) was added. We measured the absorbance at 560 nm to find out how much chromogen was made, and we reported the results in units per mg of tissue (13).

2.5.3.2 Determination of catalase

The effect of the extract on brain catalase activity was assessed by combining 2.5 ml of 50 mmol phosphate buffer (PB, pH 5.0), 0.4 ml of 5.9 mmol hydrogen peroxide (H2O2), and 0.1 ml of the prepared rat brain homogenate solution. Absorbance was recorded at 240 nm following a one-minute interval. A deviation in absorbance of 0.01 units/min was defined as one unit of catalase activity (14).

2.5.3.3 Determination of MDA

The concentration of Malondialdehyde (MDA), an indicator of lipid peroxidation, was evaluated by measuring the formation rate of thiobarbituric acid-reactive substances (TBARS), expressed as equivalents of MDA. This method involved the combination of an aliquot of brain homogenate with 1 ml of 25% trichloroacetic acid (TCA) and 1 ml of 0.67% thiobarbituric acid (TBA). The mixture was heated in a boiling water bath for 20 minutes. After the heating process, the mixture was subjected to cooling and centrifugation at a force of 4000 × g for 20 minutes. The optical density of the supernatant was measured at a wavelength of 535 nm. The MDA level was calculated using a designated molar extinction coefficient and is expressed in nanomoles per gram of wet tissue (15).

2.7 Statistical analysis

The results of the experimental procedures and observations are presented as the mean value, along with the standard error of the mean (SEM). The statistical evaluation of the data was conducted using GraphPad Prism software, version 9.0, which employed appropriate analytical methods to determine the statistical significance of the findings.

2.0 Results and Discussion

3.1 In-vitro antioxidant assay

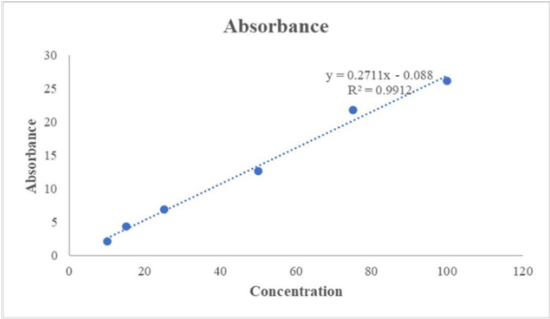
3.1.1 DPPH radical scavenging assay

In this investigation, PDWA demonstrated notable antioxidant capabilities, showing a dose responsive increase in free radical scavenging, relative to ascorbic acid. The relationship was quantified with a standard curve ($Y = 0.9422x + 13.089$, $R^2 = 0.9906$) illustrated in figure 1, based on varying ascorbic acid concentrations. At a higher dose of 100 µg/mL, the methanolic extract from the leaves showed an 81.66% inhibition rate (IC50 = 50.18), closely following ascorbic acid's 94.23% inhibition (IC50 = 40.91), with the methanol extract from the stem showing a 79.17% inhibition rate (IC50 = 54.63). This indicates the substantial antioxidant potential of the leaf extract, as depicted in Table 6 and Figure 16. Among the tested extracts, the leaf extracts were superior in antioxidant activity, and methanol proved to be an effective solvent for extraction.

Figure 1: Effect of PDWA on DPPH radicals

Figure 1: Represent Effect of PDWA on DPPH radicals

Concn µg/ml	Ascorbic acid	PDWA
100	94.23	73.61
75	82.14	51.34
50	61.62	38.14
25	40.37	20.95
15	25.65	11.74
10	20.55	7.36
IC50	40.91	68.63



3.1.2 Nitric oxide radical scavenging activity

In comparison to ascorbic acid, the methanolic extract from PDWA leaves demonstrated notable nitric oxide (NO) scavenging capabilities, with the activity increasing in correlation with the dosage (as outlined in Table 1). At a concentration of 100 µg/mL, the inhibition rate of the leaf methanolic extract reached 68.77%, with an IC50 value of 59.35µg/ml, closely followed by the stem methanolic extract which showed a 67.34% inhibition rate with an IC50 value of 68.64µg/ml. In contrast, ascorbic acid exhibited a higher inhibition rate of 90.34% with an IC50 value of 42.17µg/ml. The presence of polyphenolic compounds, such as tannins and flavonoids, within PDWA, is believed to counteract the free radicals produced by nitroprusside in the experimental setup, thereby potentially offering cellular stress mitigation within the body.

Table 1: Effect of PDWA on NO radicals

Conc µg/ml	**Ascorbic acid**	**PDWA**
100	90.34	56.85
75	80.53	38.24
50	59.14	28.22
25	41.27	21.84
15	24.43	17.93
10	21.72	15.25
IC50	**42.17**	**93.41**

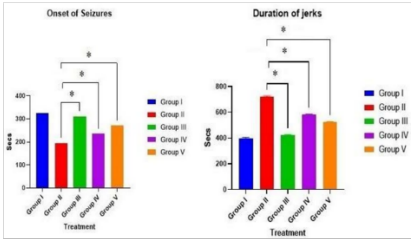
3.2 Pharmacological studies

3.2.1 In-vivo anticonvulsant activity

PTZ produced convulsions in the experimental animals, and clonic and tonic seizures were observed. When comparing the experimental results of Group III (standard diazepam) with Group IV (PLM-200mg/kg) and Group V (PLM-400mg/kg), it can be observed that the standard diazepam showed better anticonvulsant effects followed by both groups treated with PDWA. The onset of seizures in Group III was significantly delayed (310±0.73 sec) compared to Group IV (235±1.46 sec) and Group V (271±1.2 sec). The duration of jerks in Group III was also shorter (424±1.61 sec) compared to Group IV (584±0.92 sec) and Group V (524±1.43 sec). All the animals in Group III survived, while only 66.67% and 83.3% of the animals in Group IV and Group V respectively survived (Table2 & figure 2). Group V, which was treated with PDWA at a dose of 400 mg/kg per oral, showed the best results next to Group III (standard diazepam) in terms of anticonvulsant activity. The onset of seizures in Group V was delayed compared to the disease control group (Group II) and was also reduced compared to the group treated with 200 mg/kg per oral (Group IV). The duration of jerks in Group V was also decreased compared to the disease control group and was shorter than the duration in Group IV. Additionally, the percentage of animals that survived in Group V was 83.3%, which was higher than the disease control group (0%) and also higher than the group treated with 200 mg/kg per oral (80%). These results highlight the potential of PDWA at a dose of 400 mg/kg per oral as an effective anticonvulsant drug.

Figure 2: Represent behavioural responses in tabular and figure form

S.No	Onset of Seizures (Sec)	Duration of jerks (Sec)	Animals survived	Percentage of protection
Group I: Normal group	323.4±1.03	399.1.88	5/5	100
Group II: Disease Control	195±1.58	722±2.06	0/5	0
Group III: Standard diazepam (1mg/kg per oral)	310±0.73*	424±1.61*	5/5	100
Group IV: (PLM-200 mg/kg, per oral)	235±1.46*	584±0.92*	4/5	80
Group V: (PLM-400 mg/kg, per oral)	271±1.2*	524±1.43*	4/5	80



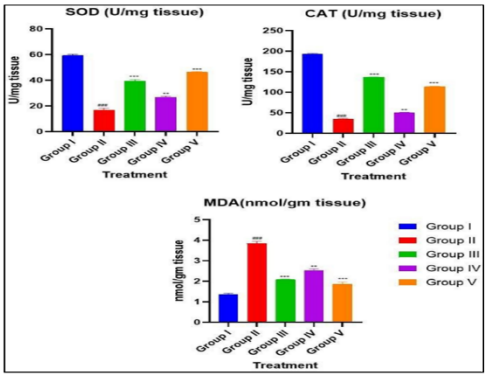
All values are expressed as Mean±SEM, statistical analysis by One-way ANOVA followed by Dunnett's test, *P<0.05 indicates statistically significance, comparison with disease control."

3.22 Biochemical parameters

As the primary antioxidant enzymes in the brain, catalase (CAT), and superoxide dismutase (SOD) are essential for the treatment of oxidative stress and related disorders. MDA is an indicator that is produced by the lipid peroxidation in oxidative stress. The PTZ has increased the MDA and decreased SOD and CAT levels in the experimental animals. The standard Diazepam and methanolic leaf extract of *PDWA* has reverted the elevated MDA concentrations and revived the antioxidant enzymes (SOD&CAT). The results of the experiments show the effect of different treatments (PTZ, Standard Diazepam, PLM-200 mg/kg, and PLM-400 mg/kg) on the levels of SOD, CAT, and MDA in the tissue. High levels of SOD and CAT indicate increased antioxidant activity, while high levels of MDA indicate oxidative stress. Comparing the PTZ group with the other groups, the PTZ group has lower levels of SOD & CAT (16.8±3.27 & 34.8±3.11) compared to the normal group (59.2±2.16 & 193±3.80), and higher levels of MDA (3.84±0.21) compared to the normal group (1.36±0.11). This suggests that the PTZ treatment induced oxidative stress, but the treatment increased antioxidant activity was indicated by the higher levels of SOD & CAT. The standard diazepam group had the highest levels of both SOD (46.2±1.30) and CAT (136±1.34) compared to all the other groups, indicating strong antioxidant activity. The PLM-200 mg/kg (26.4±2.07 & 50.6±1.14) and PLM-400 mg/kg (39.4±2.40 & 113±2.16). The results suggest that the standard diazepam treatment has the strongest effect in reducing oxidative stress and increasing antioxidant activity, followed by the PDWA200 mg/kg and PDWA-400 mg/kg treatments.(Figure 3).

Figure 3: Represent various responses of biochemical parameters

	SOD (U/mg tissue)	CAT (U/mg tissue)	MDA (nmol/gm tissue)
Group I: Normal group	59.2±2.16	193±3.80	1.36±0.11
Group II: PTZ group (100 mg/kg)	16.8±3.27###	34.8±3.11###	3.84±0.21###
Group III: Standard diazepam (1mg/kg per oral)	39.4±2.40***	113±2.16***	1.8±0.23***
Group IV: (PDWA-200 mg/kg, per oral)	26.4±2.07**	50.6±1.14**	2.5±0.19**
Group V: (PDWA-400 mg/kg, per oral)	46.2±1.30***	136±1.34***	2.1±0.05**



All values are expressed as Mean±SEM, statistical analysis by One -way ANOVA followed by Dunnett's multiple comparison test.
*****P<0.001, **P<0.01 when compared to the PTZ group**
###P<0.001when compared to the Normal group"

4.0 Coclusion

Both in vitro and in vivo studies in this study showed that PDVA has strong antioxidant and antiepileptic properties. In antioxidant tests, the extract demonstrated a strong ability to scavenge free radicals, suggesting that it can counteract reactive oxygen species and lessen oxidative stress. When compared to the disease control group, therapy with PDVA significantly delayed the onset of seizures, decreased the severity and duration of seizures, and enhanced seizure protection

in the PTZ-induced epilepsy model. Additionally, by increasing antioxidant enzyme activity and decreasing lipid peroxidation, the extract restored natural antioxidant defence systems, indicating a neuroprotective impact against oxidative damage brought on by seizures. Bioactive phytochemicals, especially flavonoids and phenolic compounds like quercetin, kaempferol, rutin, catechin, and chlorogenic acid, which are known to have antioxidant, anti-inflammatory, and neuroprotective qualities, are probably responsible for the observed pharmacological effects. PDVA may reduce epileptogenesis by reducing oxidative stress and maintaining neuronal integrity, according to the combination anticonvulsant and antioxidant actions. Overall, the results show PDVA potential as a viable natural therapeutic candidate for the treatment of epilepsy and offer scientific proof for its traditional medicinal use. To determine its safety, effectiveness, and therapeutic applicability in human epilepsy, however, more research involving the isolation and characterization of active constituents, thorough molecular mechanism studies, pharmacokinetic evaluation, chronic toxicity assessment, and well-designed clinical studies is required.

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