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Evaluation of Antiepileptic Activity of *Prunus amygdalus* Dry Fruit Suspension in Albino Mice

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ABSTRACT

Epilepsy, a prevalent neurological disorder, poses a substantial global health burden affecting individuals of all ages. Despite the availability of antiepileptic drugs (AEDs), a considerable proportion of patients face challenges in achieving adequate seizure control, prompting exploration into alternative therapies. This research focuses on evaluating the antiepileptic activity of *Prunus amygdalus* (PA) dry fruit suspension in albino mice following the induction of convulsions through maximum electric shock (MES) & isoniazid-induced tonic-clonic seizures, and strychnine-induced convulsive seizures. The study reveals that 100, 200, and 400 mg/kg doses of *Prunus amygdalus* suspension administered orally significantly reduces the frequency and amplitude of induced movements. The duration of convulsions was reduced significantly also for both clonic and tonic seizures as well. The antiepileptic activity of *Prunus amygdalus* is attributed to its impact on Na+ voltage-dependent channels or acting as an NMDA antagonist. The presence of alkaloids and flavonoids in PA suspension aligns with established evidence supporting their antiepileptic effects. This study offers insightful information into the development of plant-based alternatives for epilepsy management, paving the way for further investigations into the therapeutic potential of *Prunus amygdalus* in neurological disorders.

Keywords: *Prunus amygdalus,* epilepsy, strychnine, isoniazid, maximum electric-shock, convulsions.

1. INTRODUCTION

The neurological condition, epilepsy is typified by frequent, spontaneous seizures that arise from aberrant brain electrical activity. People of all ages suffer with this chronic illness, which has a major negative influence on their quality of life.[1] The World Health Organization (WHO) estimates that 50 million individuals worldwide suffer with epilepsy, making it one of the most prevalent neurological conditions.[2] Central to its pathophysiology is an increased predisposition to hyperexcitability, where neurons become prone to excessive and synchronized firing. Dysfunctional ion channels, responsible for regulating ion flow across neuronal membranes, contribute to this imbalance, affecting normal excitability. An intricate interplay between excitatory neurotransmitters like glutamate and inhibitory neurotransmitters such as GABA further disrupts the equilibrium. Structural anomalies, genetic predispositions, and neuroinflammation can also contribute, altering brain connectivity and increasing susceptibility to seizures. Excitotoxicity, causing damage from excessive stimulation, further compounds the intricate pathophysiology of epilepsy.[3]

The prevalence of epilepsy tends to increase with age, particularly in older adults. Children are estimated to be higher than adults, affecting approximately 1% of the global pediatric population. Seizure disorders can have profound consequences

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on the cognitive and social development of children and adolescents. It is estimated that proper diagnosis and treatment could potentially render up to 70% of individuals living with epilepsy seizure-free.[4]

The primary approach to managing epilepsy involves antiepileptic drugs (AEDs), although, a considerable number of individuals with AED continue to face challenges in achieving adequate seizure control including drug resistance. In recent years, there has been a growing interest in exploring plant-based drugs as potential alternatives for managing epilepsy. Natural compounds derived from plants often possess a diverse array of bioactive molecules with pharmacological properties. These compounds may offer novel mechanisms of action, and fewer side effects compared to synthetic medications.[5,6]

Prunus amygdalus, commonly known as sweet almonds, is one such plant that has shown promise due to its rich composition of bioactive compounds such as flavonoids, phenolic acids, tannins, and vitamin E.[7,8] Sweet almond suspension (PAS) has demonstrated therapeutic benefits in eczema and pimples, diabetes, hyperlipidemia, arthritis.[9-11] In addition, almonds can be used to treat stomach ulcers, head lice, renal discomfort, and gastroenteritis.[12] Almonds are incredibly helpful for maintaining brain function, building muscle, and extending life because of its anti-stress, antioxidant, and immunostimulant properties.[13] This research aims to evaluate the antiepileptic activity of *Prunus amygdalus* dry fruit suspension in albino mice following the induction of convulsions through maximum electric shock (MES) & isoniazid-induced tonic-clonic seizures, and strychnine-induced convulsive seizures.

2. MATERIAL & METHODS

2.1 Chemicals and Drugs

Prunus amygdalus were procured from the local market from Hyderabad, Telangana, India. Isoniazid, strychnine, diazepam, and phenytoin drugs were procured from standard suppliers.

2.2 Preparation of Suspension

A grinder blender was used to manually grind the sweet almond seeds. As previously mentioned, an aqueous solution of powdered almond was made at a concentration of 14.28%.[14]

2.3 Preliminary Phytochemical Screening

The primary groups of phytochemicals (tannins, saponins, flavonoids, alkaloids, phenols, glycosides, steroids, and terpenoids) contained in the extracts were determined by confirmatory qualitative phytochemical screening of plant extracts using conventional techniques.[15]

2.4 Acute Toxicity Studies

Following recommendations from the Organization for Economic Cooperation and Development (OECD), a study was conducted on the extract's toxicity. For the toxicity investigation, Swiss albino mice of both sexes weighing 20-25 g were utilized. As indicated by the guideline, a suspension of the extract dosage of 2000 mg/kg was made and given to a group of six animals. All animals were monitored for symptoms of toxicity and aberrant behavior every hour until 48 hours after the dosage was administered. Following this, up to the fourteenth day, daily observations were collected about toxicity and death. Every third day, the animals' body weights were noted. After the fourteenth day of dosage, all the mice were sacrificed.[16]

2.5 Experimental Animals

Swiss male albino mice weighing between 20 to 25 grams were utilized in the study. Prior to the trial, each animal was kept in an environmentally controlled chamber and given a week to become acquainted with their surroundings. These mice were housed in a controlled environment with a temperature of 25°C ± 2°C and a relative humidity of 45-55%, following a standard 12-hour light to 12-hour dark cycle. They had unrestricted access to both food and water, with food being withheld for 6 hours before drug administration. The study's experimental methodology was authorized by the Institutional Animal Ethics Committee (IAEC) and executed in compliance with the rules issued by the Government of India's Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA).

2.6 Assessment of Antiepileptic Activity

2.6.1 Maximum Electroshock (MES) Induced Seizure

In the MES model, the animals were divided up into five groups (n = 6). Group I animals were used as

controls, receiving 10 ml/kg, p.o. of Na CMC (0.3%), Group II animals were used as positive controls, receiving 25 mg/kg, i.p. of Phenytoin (PHT), and Groups III–V animals were given 100, 200, and 400 mg/kg, p.o. of *Prunus amygdalus* suspension (PAS). All the groups received drug treatment for 15 days. On the fifteenth day, 30 minutes after the test and standard drug treatments, all the mice received treatment with a current of (150 mA, 0.2 s) through corneal electrodes using an Electro convulsiometer (INCO, India) to cause generalized tonic-clonic seizures in all the animal groups. An evaluation of the antiepileptic action was conducted by measuring the tonic hind limb extension (THLE) extent.[17]

2.6.2 Isoniazid Induced Seizures

In isoniazid induced seizure model, the animals were divided into five groups (n = 6) with Group I animals serving as control and received Na CMC (0.3%) at a dose of 10 ml/kg, p.o, Group II served as Positive control and received diazepam (DZP) (1 mg/kg, i.p.) and Groups III-V animals were administered with the *Prunus amygdalus* suspension (PAS) at doses of (100, 200 and 400 mg/kg, p. o.). Each group received a 15-day dose of the drugs. On the fifteenth day, 30 minutes following drug therapy, mice were given conventional antiepileptic drug, isoniazid (300 mg/kg, i.p.) to induce seizures. An hour was used for observing the animals for bouts of tonic convulsion, which were classified as hind limb extension. Considerations were made for variables like the beginning of tonic convulsion, the quantity of animals convulsing or not convulsing during the observation time, and the animals' survival or demise. The animals' survival and the PAS's ability to stop or delay the initiation of hind limb extension were seen as signs of antiepileptic action.[18,19]

2.6.3 Strychnine Induced Seizures

In the strychnine-induced seizure model, the animals were divided into five groups (n = 6). with Group I animals serving as control and received Na CMC (0.3%) at a dose of 10 ml/kg, p.o, Group II served as Positive control and received diazepam (DZP) (1 mg/kg, i.p.) and Groups III-V animals were administered with the *Prunus amygdalus* suspension (PAS) at doses of (100, 200 and 400 mg/kg, p. o.). Each group received a 15-day dose of the drugs. On the 15th day, seizures were induced in mice using the standard antiepileptic drug, strychnine (0.5 mg/kg, i.p.), administered 30 minutes after drug treatment. The animals were observed for one hour to monitor tonic convulsion episodes.[20]

2.7 Statistical Analysis

The results were presented as the mean ± SEM (Standard Error of the Mean). The data was assessed using one-way analysis of variance (ANOVA) followed by Dunnett's Multiple Comparison test within GraphPad Prism (version 5.0).

3. RESULTS

3.1 Preliminary Phytochemical Screening

The preliminary phytochemical analysis of the *Prunus amygdalus* suspension indicated the presence of saponins, flavonoids, carbohydrates, proteins, and alkaloids. The results are given in Table 1.

Table 1: Phytochemistry of the *Prunus amygdalus* suspension

Phytochemical Constituent	Test	Result
Carbohydrate	Molisch's	+
	Anthrone	+
	Benedict's	+
	Fehling's	+
Tannins	Ferric chloride	-
Saponins	Frothing	+
Steroids & Triterpenoids	Liebermann-buchard's	-
Flavonoids	Lead acetate	+
	Shinoda' test	+
Proteins	Biuret's	+
	Millon's	+
Alkaloids	Dragendorff's	+
	Mayer's	+
	Hager's	+
	Wagner's	+

Note: + present; - absent.

3.2 Acute Toxicity Studies

The *Prunus amygdalus* suspension was found to be safe up to maximum dose of 2000 mg/kg body weight by oral route. No sign of toxicity and no mortality was found. The 1/5th, 1/10th, 1/20th doses for pharmacologic studies were taken as 100, 200, 400 mg/kg/b.w of the maximum tolerable dose (i.e. 2000mg/kg).

3.3 Effect of PAS in Maximum Electroshock (MES) Induced Seizure

The administration of maximal electroshock (MES) induced hind limb tonic extension seizures in all subjects. In the control group treated with the

Table 2: The effect of *Prunus amygdalus* suspension in Maximal Electroshock (MES) induced seizures

S. No.	Group	Onset of HLTE (sec) (Mean ± SEM)	Quantal Protection	% Protection
1	Vehicle Control	14.50 ± 0.76	0/0	0
2	PHT (25 mg/kg, i.p.)	0	0/6	100
3	PAS (100 mg/kg, i.p.)	11.83 ± 0.70*	2/6	33.33
4	PAS (200 mg/kg, i.p.)	7.83 ± 0.47**	4/6	66.66
5	PAS (400 mg/kg, i.p.)	5.50 ± 0.42**	5/6	83.33

*The values are expressed as mean ± SEM (n=6). ***P < 0.001, **P < 0.01, *P < 0.05 compared to the vehicle control group. Data were analyzed by one-way ANOVA, followed by Dunnett's Multiple Comparison test.*

vehicle, mice exhibited tonic limb extension with an average duration of 14.50 ± 0.76 seconds. *Prunus amygdalus* suspension (PAS) at a dosage of 100 mg/kg, orally, demonstrated protective effects in 33.33% of the mouse population and significantly altered the incidence of MES-induced seizures. Similarly, an oral dose of 200 mg/kg of the suspension provided protection in 33.33% of mice and substantially reduced the duration of hind limb tonic extension (HLTE) caused by MES. At a dose of 400 mg/kg, orally, the suspension exhibited protective effects in 83.33% of the animal population and significantly diminished the duration of seizures. The standard anti-epileptic drug, phenytoin (25 mg/kg, i.p.), conferred protection to all animals and markedly reduced the duration of HLTE (Table 2).

3.4 Effect of PAS in Isoniazid Induced Seizure

Isoniazid (300 mg/kg i.p.) induced tonic seizures in all the animals. The oral administration of *Prunus amygdalus* suspension at 100 mg/kg p.o. significantly delayed the latency and altered the incidence of seizures caused by isoniazid to a lesser extent, providing protection in 33.33% of the animal population. A dosage of 200 mg/kg p.o. significantly prolonged the latency of seizures and offered protection to 66.66% of the animal population. A dose of 400 mg/kg p.o. exhibited protection in 83.33% of the animals when compared to the standard anti-epileptic drug diazepam. Both the *Prunus amygdalus* suspension and diazepam significantly delayed the latency of seizures as shown in Table 3.

3.5 Effect of PAS in Strychnine Induced Seizure

Strychnine (2 mg/kg i.p.) induced tonic seizures in all experimental animals. The oral administration of *Prunus amygdalus* suspension at 100 mg/kg p.o. significantly delayed the latency and modified the incidence of seizures caused by strychnine to a significant extent. At a dosage of 200 mg/kg p.o., the suspension significantly prolonged the latency of strychnine-induced seizures and provided protection in 50% of the animals. An oral dose of 400 mg/kg resulted in protection in 83.33% of the animals, comparable to the standard anti-epileptic drug diazepam. Both the suspension and diazepam significantly delayed the latency of seizures as shown in Table 4.

4. DISCUSSION

Currently available medications can effectively manage epileptic seizures in approximately 50% of patients. Another 25% may experience improvement, while the remaining 25% do not benefit significantly.[21] Unfortunately, the clinically used medications often come with undesirable side effects that complicate treatment, highlighting the

Table 3: The effect of *Prunus amygdalus* suspension in isoniazid induced seizures

S. No.	Group	Onset of Tonic Convulsions (sec) (Mean ± SEM)	Quantal Protection	% Protection
1	Vehicle Control	121.8 ± 2.33	0/0	0
2	DZP (1 mg/kg, i.p.)	0	0/6	100
3	PAS (100 mg/kg, i.p.)	136.2 ± 1.07*	2/6	33.33
4	PAS (200 mg/kg, i.p.)	173.3 ± 1.542**	4/6	66.66
5	PAS (400 mg/kg, i.p.)	207 ± 2.324***	5/6	83.33

*The values are expressed as mean ± SEM (n=6). ***P < 0.001, **P < 0.01, *P < 0.05 compared to the vehicle control group. Data were analyzed by one-way ANOVA, followed by Dunnett's Multiple Comparison test.*

Table 4: The effect of *Prunus amygdalus* suspension in strychnine induced seizures

S. No.	Group	Onset of Tonic Convulsions (sec) (Mean ± SEM)	Quantal Protection	% Protection
1	Vehicle Control	52.17 ± 0.87	0/0	0
2	DZP (1 mg/kg, i.p.)	0	0/6	100
3	PAS (100 mg/kg, i.p.)	83.50 ± 1.38*	2/6	33.33
4	PAS (200 mg/kg, i.p.)	124.3 ± 1.11**	3/6	50.0
5	PAS (400 mg/kg, i.p.)	164.7 ± 1.89**	5/6	83.33

*The values are expressed as mean ± SEM (n=6). ***P < 0.001, **P < 0.01, *P < 0.05 compared to the vehicle control group. Data were analyzed by one-way ANOVA, followed by Dunnett's Multiple Comparison test.*

need for new antiepileptics. One approach to discovering novel drugs involves investigating existing compounds, potentially belonging to new structural classes. Effective medications seem to enhance amino acid-mediated junction inhibition or promote the inactivated state of voltage-activated Na+ channels in typical forms of epileptic seizures.[22,23]

The study on Prunus amygdalus (PA) suspension demonstrated that doses of 100, 200, and 400 mg/kg, orally, reduced the frequency and amplitude of movements. Preliminary phytochemical analysis of the PA suspension indicated the presence of saponins, flavonoids, carbohydrates, proteins, and alkaloids. The PA suspension was found to be safe up to a maximum dose of 2000 mg/kg, orally, with no signs of toxicity or mortality after 48 hours.

In the maximal electroshock (MES)-induced epileptic model, representing the tonic-clonic type of epilepsy, the PA suspension showed significant antiepileptic activity. The tonic extension phase was abolished, possibly due to the inhibition of voltage-dependent Na+ channels or acting as an NMDA antagonist.[24] The PA suspension exhibited its maximum effect at a dose of 400 mg/kg, orally, providing protection in 83.33% of the population compared to the standard group's 100% protection.

Commonly used to treat tuberculosis, isoniazid can have major adverse impacts on the central nervous system, including comas and seizures. It is believed to prevent the CNS from synthesizing GABA.[25] The PA suspension-treated groups showed protection in 83.33% of the animals at a dose of 400 mg/kg, orally, indicating a potential enhancement of GABA synthesis. The values were considered significant when compared with the control group. In the strychnine-induced seizure model, where strychnine acts as a potent spinal cord convulsant, selectively blocking glycine receptors to induce excitatory responses in the CNS, the PA suspension demonstrated significant inhibition at a dose of 400 mg/kg, orally, providing protection in 83.33% of the population compared to the standard group's 100%.[26] When compared to the control group, the values were determined to be significant, which may indicate interference with the transmission of glycine. Glycine inhibitory mechanisms may be indirectly enhanced by diazepam's suppression of seizures.

Numerous experimental observations have unequivocally demonstrated that alkaloids and flavonoids exhibit antiepileptic activity.[27-29] Alkaloids prevent seizers by modulating several neurotransmitter systems like N-methyl-D-aspartate, nitric oxide, and serotonin, which modulate convulsions.[27] Flavonoids, on the other hand, bind to the benzodiazepine site on the GABAA-receptor resulting in antiepileptic effects.[30] The phytochemical screening of the Prunus amygdalus suspension indicated the presence of both alkaloids and flavonoids as active phytoconstituents and consequently, the findings from the present studies provide evidence supporting the antiepileptic effects of the Prunus amygdalus suspension against tonic-clonic and convulsive seizures.

5. CONCLUSION

Based on the above investigations, it is evident that the *Prunus amygdalus* suspension demonstrated noteworthy antiepileptic activity. These results lend support to the traditional application of this plant in alleviating neuronal disorders. The likely influence of alkaloids and flavonoids in modulating and enhancing neurotransmitter and GABAergic systems may account for the observed significant activity of the *Prunus amygdalus* suspension. These findings provide valuable insights into the phytochemical

foundation of the antiepileptic activity of *Prunus amygdalus* suspension, underscoring the need for additional research to fully elucidate its therapeutic potential.

Conflict of Interest: The authors declare that they have no conflicts of interest.

REFERENCES

1. Sirven JI. Epilepsy: A Spectrum Disorder. Cold Spring Harb Perspect Med. 2015 Sep 1;5(9):a022848.
2. Fiest KM, Sauro KM, Wiebe S, Patten SB, Kwon CS, Dykeman J, Pringsheim T, Lorenzetti DL, Jetté N. Prevalence, and incidence of epilepsy: A systematic review and meta-analysis of international studies. Neurology. 2017 Jan 17;88(3):296-303.
3. Stafstrom CE, Carmant L. Seizures, and epilepsy: an overview for neuroscientists. Cold Spring Harb Perspect Med. 2015 Jun 1;5(6):a022426.
4. Anwar H, Khan QU, Nadeem N, Pervaiz I, Ali M, Cheema FF. Epileptic seizures. Discoveries (Craiova). 2020 Jun 12;8(2):e110.
5. Dalic L, Cook MJ. Managing drug-resistant epilepsy: challenges and solutions. Neuropsychiatr Dis Treat. 2016 Oct 12;12:2605-2616.
6. Veeresham C. Natural products derived from plants as a source of drugs. J Adv Pharm Technol Res. 2012 Oct;3(4):200-1.
7. Gallardo-Rivera CT, Lu A, Treviño-Garza MZ, García-Márquez E, Amaya-Guerra C, Aguilera C, Báez-González JG. Valorization of Almond (*Prunus serotina*) by Obtaining Bioactive Compounds. Front Nutr. 2021 May 31;8:663953.
8. Moreno Gracia B, Laya Reig D, Rubio-Cabetas MJ, Sanz García MÁ. Study of Phenolic Compounds and Antioxidant Capacity of Spanish Almonds. Foods. 2021 Sep 30;10(10):2334.
9. Zeichner JA, Berson D, Donald A. The Use of an Over-the-Counter Hand Cream with Sweet Almond Oil for the Treatment of Hand Dermatitis. J Drugs Dermatol. 2018 Jan 1;17(1):78-82.
10. Moezi L, Arshadi SS, Motazedian T, Seradj SH, Dehghani F. Anti-Diabetic Effects of *Amygdalus lycioides* Spach in Streptozocin-Induced Diabetic Rats. Iran J Pharm Res. 2018 Winter;17(1):353-364.
11. Tarmoos AA, Kafi LA. Effects of sweet almond (*Prunus amygdalus*) suspension on blood biochemical parameters in experimentally induced hyperlipidemic mice. Vet World. 2019 Dec;12(12):1966-1969.
12. Mushtaq A, Khaliq M, Saeed A, Azeem MW, Ben Ghania J. Almond (*Prunus amygdalus* L.): A review on health benefits, nutritional value, and therapeutic applications. Int J Chem Biochem Sci. 2015;8:103-106.
13. Tripathi J, Bhola S, Vashishth R. A review on the therapeutic role of Vadam Taila through Nasya (*Prunus amygdalus* Batsch) in Avabahuka as a single drug. World J Pharm Res. 2018;7(19):382-395.
14. Al-Taee RAK, Kafi LA. Comparative Study of Anticoagulant Effect of Sweet Almond Suspension (*Prunus amygdalus*) with Aspirin in Male Albino Mice. In: The 2nd Scientific Conferences of Kerbala University, College of Pure Sciences; 2013:216–220.
15. Kammari S, Padmavathi R, Samad MA, Dixith AS. Comparative Evaluation of Aqueous Extracts of Peel, Seed, and Whole Fruit of Punica Granatum L. on Ethylene Glycol Induced Urolithiasis in Rats. Int J Pharm Pharm Sci. 2020 Sep;19(2):507-518.
16. Organization for Economic Co-operation and Development. Test No. 420: Acute Oral Toxicity-Fixed Dose Procedure. OECD Publishing; 2002.
17. Xiang C, Li ZN, Huang TZ, Li JH, Yang L, Wei JK. Threshold for maximal electroshock seizures (MEST) at three developmental stages in young mice. Zool Res. 2019 May 18;40(3):231-235.
18. Asehinde S, Ajayi A, Bakre A, Omorogbe O, Adebesin A, Umukoro S. Effects of Jobelyn® on Isoniazid-Induced Seizures, Biomarkers of Oxidative Stress and Glutamate Decarboxylase Activity in Mice. Basic Clin Neurosci. 2018 Nov-Dec;9(6):389-396.
19. Obese E, Biney RP, Henneh IT, Adakudugu EA, Anokwah D, Agyemang LS, Woode E, Ameyaw EO. The Anticonvulsant Effect of Hydroethanolic Leaf Extract of *Calotropis procera* (Ait) R. Br. (Apocynaceae). Neural Plast. 2021 Jun 26;2021:5566890.
20. Amabeoku G, Chandomba R. Strychnine-induced seizures in mice: the role of noradrenaline. Prog Neuropsychopharmacol Biol Psychiatry. 1994 Jul;18(4):753-63.
21. Goldenberg MM. Overview of drugs used for epilepsy and seizures: etiology, diagnosis, and treatment. P T. 2010 Jul;35(7):392-415.

22. Lee JW, Dworetzky B. Rational Polytherapy with Antiepileptic Drugs. Pharmaceuticals (Basel). 2010 Jul 26;3(8):2362-2379.
23. Khateb M, Bosak N, Herskovitz M. The Effect of Anti-seizure Medications on the Propagation of Epileptic Activity: A Review. Front Neurol. 2021 May 27;12:674182.
24. Piovesan EJ, Randunz V, Utiumi M, Lange MC, Kowacs PA, Mulinari RA, Oshinsky M, Vital M, Sereniki A, Fernandes AF, e Silva LL, Werneck LC. Influence of NMDA and non-NMDA antagonists on acute and inflammatory pain in the trigeminal territory: a placebo control study. Arq Neuropsiquiatr. 2008 Dec;66(4):837-43.
25. Navalkele B, Bueno Rios MX, Wofford JD, Kumar V, Webb RM. Seizures in an Immunocompetent Adult from Treatment of Latent Tuberculosis Infection: Is Isoniazid to Blame? Open Forum Infect Dis. 2020 Apr 23;7(5):ofaa144.
26. Yevenes GE, Zeilhofer HU. Allosteric modulation of glycine receptors. Br J Pharmacol. 2011 Sep;164(2):224-36.
27. Bhutada P, Mundhada Y, Bansod K, Dixit P, Umathe S, Mundhada D. Anticonvulsant activity of berberine, an isoquinoline alkaloid in mice. Epilepsy Behav. 2010;18(3):207-210.
28. Auwal MS, Saka S, Mairiga IA, Sanda KA, Shuaibu A, Ibrahim A. Preliminary phytochemical, and elemental analysis of aqueous and fractionated pod extracts of Acacia nilotica (Thorn mimosa). Vet Res Forum. 2014 Spring;5(2):95-100.
29. Loshali A, Joshi BC, Sundriyal A, Uniyal S. Antiepileptic effects of antioxidant potent extract from *Urtica dioica* Linn. root on pentylenetetrazole and maximal electroshock induced seizure models. Heliyon. 2021 Feb 5;7(2):e06195.
30. Diniz TC, Silva JC, de Lima-Saraiva SR, Ribeiro FP, Pacheco AG, de Freitas RM, Quintans-Júnior LJ, Quintans Jde S, Mendes RL, Almeida JR. The role of flavonoids on oxidative stress in epilepsy. Oxid Med Cell Longev. 2015;2015:171756.

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