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ORIGINAL ARTICLE **OPEN ACCESS**

Antiseizure Effect of Aqueous Extract of *Pergularia Daemia* Forsk. in Pentylenetetrazole-Induced Wistar Albino Rats

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ABSTRACT
Epilepsy is a Neurological disease. It disturbs the brain's regular electrochemical processes. Similar to a seizure, Epilepsy is an indication of irregular brain activity. The purpose of the present study is to determine whether the Aqueous leaf extract of Pergularia daemia Forsk. (AEPD) has the anti-seizure activity of Pentylenetetrazole (PTZ) induced rats. Wistar strains of albino rats were used for the experiments, and the animals were randomly divided into various groups. To determine the antiepileptic potential of rats given Pentylenetetrazole-induced epilepsy, AEPD extract was administered at doses of 100, 200, and 400mg/Kg BW. Physical behaviors like immobility, swimming, and motor activities were assessed in the experimental animals. By analyzing the levels of neurotransmitters including GABA, Glutamate, Norepinephrine, serotonin, and Acetylcholinesterase in the animal's brain, the effect of PTZ was examined. The results showed that on the 30th day, acute administration of PTZ, in a dose of (75 mg/kg body weight) produced seizure, which increased immobility and reduced swimming time. Although the plant treatment in PTZ-induced groups showed restored levels of glutamate, dopamine, serotonin, acetylcholinesterase, and GABA. The PTZ increased oxidative stress followed by degeneration of neural and non-neural cells (Glial cells), which altered levels of neurotransmitters. The results indicate that Pergularia daemia Forsk. a traditional source, can be utilized to treat seizures.
Keywords: *Epilepsy, GABA, Seizure, Pergularia daemia Forsk., Pentylenetetrazole.*

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INTRODUCTION

The rapid and unexpected modification in motor activity as well as behavior, with or without abnormalities in awareness, is known as epilepsy, a chronic neurological disorder marked by recurring, unexplainable convulsions or seizures. The condition changes as a result of the group of epileptic neurons in the brain firing excessively and abnormally hypersynchronized [1]. Around 50 million people are suffering from epilepsy worldwide and among them, 80% are in upward countries. Epilepsy affects all age groups, particularly young people in the first two decades of life and the elderly. It is estimated that the mortality rate for those with epilepsy is 2-3 times more than the general population, and is significantly higher for young age people [2]. Epileptic foci were shown to have neurotransmitter abnormalities [3]. Therefore, ictogenesis is exacerbated by a neurotransmitter imbalance characterized by decreased GABA activity through GABA A receptors as well as increased glutamate activity through ionotropic glutamate receptors [4]. Moreover, ictogenesis is also heavily impacted by the actions of other neurotransmitters, namely (dopamine, serotonin hypoactivity, and noradrenaline hyperactivity) [4]. In addition to adverse effects, teratogenic consequences, chronic toxicity, and dose-related around 30% of people with epilepsy still suffer seizures after being treated with new antiepileptic medicines [5]. Therefore, the discovery of natural anticonvulsants that are both efficacious and benign is of the utmost importance. Up to 80% of the population in certain underdeveloped nations relies on folk cures or traditional medicine [6]. Many people believe that medicinal plants might be a significant source of new therapeutic chemical compounds [7]. Several plants utilized in traditional medicine to treat epilepsy have yet to be scientifically examined although some have demonstrated action in recent bioassays for the identification of anticonvulsant activity [8]. Herbaceous *Pergularia daemia* Forsk**.** is a member of the Asclepiadaceae family. This kind of plant is native to subtropical as well as tropical regions, mainly found in Tamil Nadu, India. Extremely

widespread in the tropical regions of Africa, reaching as far east as Arabia. *Pergularia daemia* Forsk**.** has various medicinal properties for example antibacterial, cardioprotective, hepatoprotective, antidiabetic, wound healing, and antifertility [9-13]. The plant is effective in vatha, poisoning, asthma, and convulsion; while the root is beneficial in piles, [14] leprosy, anemia, and mental disturbance. The Plant contains stomachic, diuretic, and laxative effects, beneficial in sore eyes, biliousness, and cough. Leaf paste combined with castor oil is utilized for joints in spleen enlargement and liver problems; leaves have hypoglycemic action. This research was conducted to determine whether or not an Aqueous extract of *Pergularia daemia* Forsk has anticonvulsant effects in animal models.

MATERIAL AND METHODS

Collection and authentification of plant material

The plant source was collected in the surrounding area of Trichy, and the specimen that was deposited in the RAPINAT Herbarium, Department of Botany, St. Joseph's College, Trichy, was used for authentication.

Preparation of Aqueous extract

The dried leaves were powdered using a commercial electrical blender. 200g of dry powder was mixed with six times water and mixed well. The content was boiled at 100^{o}C for till reduce 1/3rd level of water. The final crude content was filtered utilizing muslin cloth and evaporated to dryness by water. The final obtained crude extract was used for experimental studies.

ANTIEPILEPTIC SCREENING

Experimental Animals

Healthy adult Two to three-month-old Wistar strain of Albino rats weighing 150g-200 g were purchased from Biogen, in Bangalore. Before the experiment, the animals were allowed five days to acclimatize to the laboratory conditions. Animals were housed in standard polypropylene cages. The animals were housed in standard polypropylene cages. Animals received regular rat chow pellets from Sai Durga Foods and Feeds in Bangalore, India, and water *ad libitum*. All the studies were carried out following CPCSEA ethical principles after obtaining the required approval from the committee (Approval No: 790/03/ac/CPCSEA).

Chemicals

The PTZ was purchased from Sigma Chemicals, USA. Diazepam was purchased from Ranbaxy Lab, Mumbai.

PTZ-induced seizures

In this experiment, the rats were divided into six groups comprising six rats each. Group I was considered normal and Group II rats were administered PTZ at the dose of 75 mg/kg BW i.p. to induce clonic convulsions in the rat. Group III, IV, and V were treated with doses of 100, 200, and 400mg/kg of the AEPD extract were administered orally into test groups and Group VI was treated with oral administration of Diazepam (4mg/BW). The PTZ was given after 60 minutes from the oral administration of the extract, and diazepam. The antiepileptic activity was accessed by its ability to delay the onset of myoclonic jerks and clonic convulsions. After the experimental period, animals were sacrificed by cervical decapitation. Blood was collected and serum was separated by centrifuging at 3000 rpm for 10 minutes. The Brain was dissected out and washed in ice-cold saline. The Brain was homogenized in 0.1 M phosphate buffer, pH 7.4, and used for the various experiments.

The methods were carried out the physical behavioural tests like onset and duration of convulsion, Forced Swimming test [15] and Hole cross test [16]. The homogenate was used for the determination of the Neurotransmitter assay like Glutamate [17], GABA, [18] Dopamine [19], Serotonin [20], Noradrenaline [21] and Aceylcholinesterase [22].

STATISTICAL ANALYSIS

All the results were expressed as mean ± SD. The data were statistically analyzed by SPSS (use any statistical software) one–way analysis of variance (ANOVA) and P values <0.05 were considered significant.

RESULTS

Effect of *Pergularia daemia* Forsk on PTZ induced seizure.

Clonic and tonic seizures are caused by PTZ. The most common preliminary screening test for evaluating likely anticonvulsant medicines is the prevention of seizures in PTZ-induced animals. In the present study, when administered orally diazepam and aqueous leaf extract of *Pergluaria daemia* Forsk. at a dose of 4mg/kg and (high dose (400mg/Kg BW), medium dose (200mg/Kg BW), and low dose(100mg/Kg BW)) decreased the onset of seizures compared to control. These results suggest that the leaf extract of *Pergularia daemia* Forsk possesses clinically appropriate antiseizure activity. It is useful in overcoming PTZ induced convulsions, in animal models.

Figure 1. showed that the AEPD delay the onset and duration of PTZ induced seizures.

Figure 2 showed that the PTZ exposure affected all the physical behaviors of the animals as it significantly increased immobility and decreased locomotor function of swimming in Group II rats compared to the normal group($p<0.05$). In contrast, treatment with AEPD was effective in decreasing immobility ($p<0.05$) and in another way, in increasing locomotor function in swimming significantly($p<0.05$), which helps the brain on a molecular and behavioral level by regulating neurotransmitters, which could be used to manage stress and mood reducing hormones.

Figure 3 showed that PTZ (75mg/kg) administration in Group II significantly ($p<0.05$) decreased the nociceptive activity by affecting sensory neurons that respond to damaging or potentially damaging stimuli by distribution of possible treatment signals to the spinal cord and the brain. PTZ showed a significant reduction in spontaneous motor activity. The aqueous extract of *Pergularia daemia* Forsk at the dose level of (100,200 and 400mg/kg BW) was given to Group III, IV, and V rats. After the induction with PTZ the Group III, IV, and V rats do not show any alteration in the nociceptive function of the central nervous system. Diazepam (4mg/kg BW) treated rats also showed no modulation in the nociceptive activity of the central nervous system in group VI rats.

Table: 1 depicts that PTZ administration increased the Glutamate level significantly ($p<0.05$) in the forebrain of epileptic control animals. AEPD at the dose level of 100, 200, and 400mg/kg body weight and standard drug diazepam-treated animals showed a significant ($p<0.05$) decline in Glutamate levels in the forebrain of PTZ-induced rats. In the present study, GABA, Serotonin, Norepinephrine, Dopamine, and Acetylcholine esterase levels were significantly ($p<0.05$) decreased in the forebrain of PTZ-induced epileptic control animals were observed. AEPD at the dose level of 100, 200, and 400mg/kg, standard drug diazepam-treated animals showed a significant ($p<0.05$) increase in the levels of GABA, Serotonin, Norepinephrine, Dopamine, and Acetylcholine esterase in the forebrain of PTZ induced rats.

DISCUSSION

The findings of the present study suggest that AEPD had Antiseizure activity. The onset and duration of convulsion results showed that no death was found in the rats besides PTZ-induced seizure. After the treatment with an AEPD dose level of 400 mg/Kg significantly late the onset of convulsions, significantly reduced the interval of convulsion. The standard anti-epileptic drug diazepam (4mg/Kg) completely antagonized the seizure induced by PTZ. A forced swim test is a commonly used animal model for assessing the depression caused by medication molecules [23]. Depression and psychiatric conditions are caused by antiepileptic drugs. The preparation of herbal compounds or herbal medicine with antiepileptic activity without causing depression is among the most interesting in drug development. This herbal medicine reduces depression [24]. In the present study, a locomotor activity test was used to evaluate for possible sedative effects of the antioxidant activity of AEPD. In this test, the locomotor activity of the AEPD was inhibited. As was observed in the diazepam control, the effective dosages of AEPD 400 mg/kg were shown to cause a significant decrease in locomotor activity and a decrease in motor coordination. Diazepam is also moderately effective and is being widely used for absence seizures due to its facilitating GABAergic activity [25]. However, there is suppression of the locomotor activity seen with Diazepam and other Benzodiazepines [26]. Gamma-aminobutyric acid (GABA), the major inhibitory neurotransmitter in the cerebral cortex, maintains the inhibitory tone that counterbalances neuronal excitation. When this balance is disturbed, seizures may occur. GABA is formed within GABAergic axon terminals and released into the synapse, where it acts at one of two types of receptor, GABA-A, which controls chloride entry into the cell, and GABA-B, which increases potassium conductance, decreases calcium entry inhibits the presynaptic release of other transmitters [27]. PTZ may cause convulsions by preventing GABA from acting on GABA-A receptors [28]. Decreasing and enhancing convulsion occurs by increasing and decreasing GABA neurotransmission [29]. In the present study treatment with effective doses of AEPD 400 mg/Kg has been shown to cause enhances the GABA levels. Diazepam and other common antiepileptic medications are thought to be effective by improving GABA-mediated inhibition and acting as an anticonvulsant against PTZ seizures. Glutamate is the most rich excitatory neurotransmitter in the vertebrate nervous system. At chemical synapses, glutamate is stored in vesicles. Nerve impulses prompt the release of glutamate from the pre-synaptic cell. In the opposing post-synaptic cell, glutamate receptors, such as the NMDA receptor, bind glutamate and are activated. In brain injury or disease, they can work in reverse, and excess glutamate can accumulate outside cells. This process causes calcium ions to enter cells via NMDA receptor channels, leading to neuronal damage and eventual cell death, and is called excitotoxicity [30]. In the present study, the established antiepileptic drugs such as diazepam restored the glutamate levels in the brain. Similarly AEPD at the dose level of 400mg/kg significantly ($p<0.05$ & $p<0.01$) decreased glutamate levels in the forebrain of rats.Increasing the dopamine, noradrenaline, and serotonin (Monoamine) levels in the brain by inhibiting monoamine oxidase (MAO). MAO is an enzyme that breakdown down biogenic amines and

tends to increase the seizure threshold. [31] Serotonin (5-Hydroxy tryptamine) is an inhibitory neurotransmitter involved in the regulation of mood, sleepy, anxiety, arousal and aggression. It has been suggested that serotonin agonists, precursors, and inhibitors of neuronal uptake increase narcoleptic catalepsy [32**]**. In many animal test systems, an increase in serotonergic transmission lowers the threshold of pentylenetetrazole (PTZ)-induced seizures, protecting against PTZ-induced convulsions [32] For an effective internal encoding of motor skills, dopamine activation seems to be important.

Dopamine is also thought to act as a teaching signal to the brain regions responsible for learning new behaviors. Octopamine, a chemical related to dopamine, has been shown to have a comparable effect on insects [33**]**. Dopaminergic receptors, found in the substantia nigra and other areas of the brain, mediate these actions. Noradrenaline, which is primarily involved with blood pressure control, also has a function to play in the control of seizures, though less significantly than other biogenic amines. The cerebellum may have a biphasic impact of glutamate, which would block glutamate release at low concentrations [34]. In the present study, AEPD at the dose of 400 mg /Kg elevates monoamines such as serotonin, dopamine and noradrenaline ,which also causes an inhibition of seizure activity

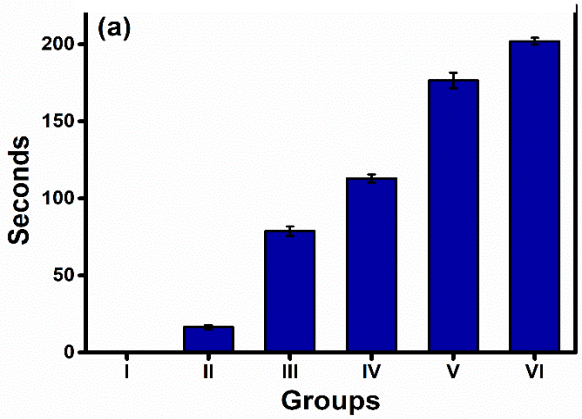
The central cholinergic system is essential for the development of memories [35]. In the elderly, acetylcholine-containing neurons become dysfunctional, leading to memory loss [36]. According to the results of this study, PTZ injections to epileptic rats considerably reduced their levels of acetylcholine. The rats' memory is altered along with this decrease in memory. Furthermore, acetylcholinesterase activity biochemical study shows that AEPD extract caused a suppression of the enzyme's activities. An improvement in memory was observed during the physicochemical test, and this was followed by an increase in the level of acetylcholine in the hippocampus of treated rats when given various extract doses. While the treatment with AEPD at the dose level of 400mg/kg showed that AChE activity was significantly decreased and acetylcholine activity increased.

Table: 1 Effect of *Pergularia daemia Forsk*. on Glutamate, GABA, Serotonin, Norepinephrine, Dopamine, and Acetylcholine esterase activity in PTZ-induced epileptic rats

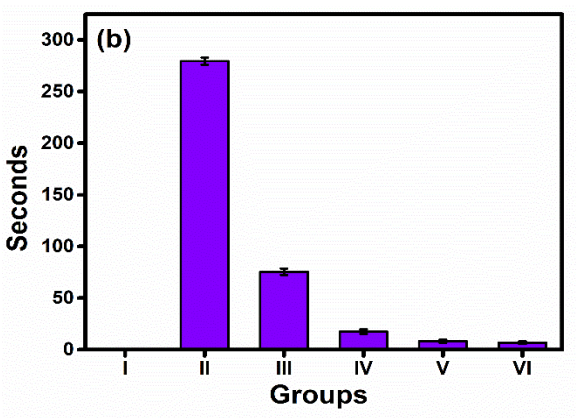
Group	Glutamate (µ mole/g tissue)	GABA (µg/ g tissue)	Serotonin (ng/ g tissue)	Nor Epinephrine (Pg/ g tissue)	Dopamine (Pg/ g tissue)	Acetylcholine esterase (µg of acetylcholine hydrolyzed/ g tissue)
I	41.29*±0.94	2.725*±0.009	50.76*±0.43	2.05*±0.129	1.37*±0.045	7.978*±0.305
II	62.17*±0.54	0.375*±0.170	31.16*±0.53	0.845*± 0.06	0.832* ± 0.052	1.556*±0.094
III	58.69±0.70	0.682±0.013	44.73±0.51	1.26 ± 0.069	0.813 ± 0.042	1.574±0.067
IV	54.9±0.53	1.136±0.010	46.48±0.16	1.557 ± 0.129	1.077 ± 0.040	2.166±0.056
V	51.42**±0.70	1.246**±0.017	47.33**±0.17	1.932** ± 0.068	1.164** ± 0.039	4.182**±0.244
VI	40.66**±0.53	1.423**±0.024	48.93**±0.29	2.01** ± 0.077	1.27** ± 0.039	6.956**±0.028

*p<0.05 statistically significant compared between Normal and Disease Control Group, **p<0.05 statistically significant compared between Disease Control and Drug treated Group

Figure 1 (a): Onset of seizure in experimental Rats

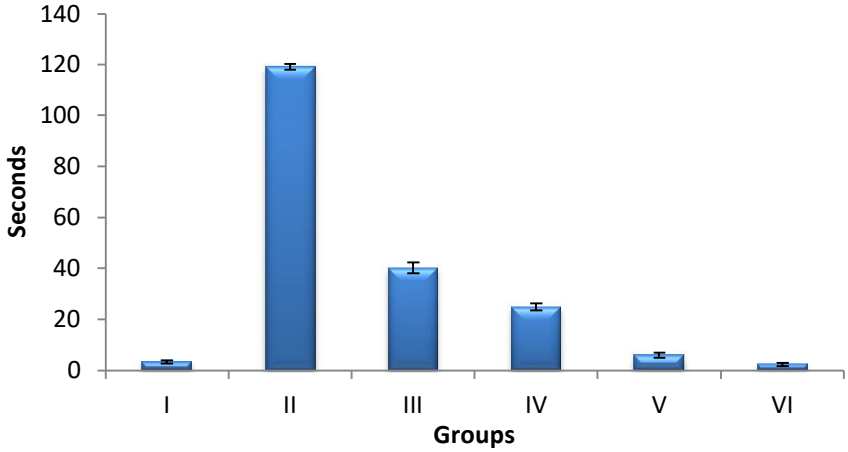


(b): Duration of seizure in experimental rats.



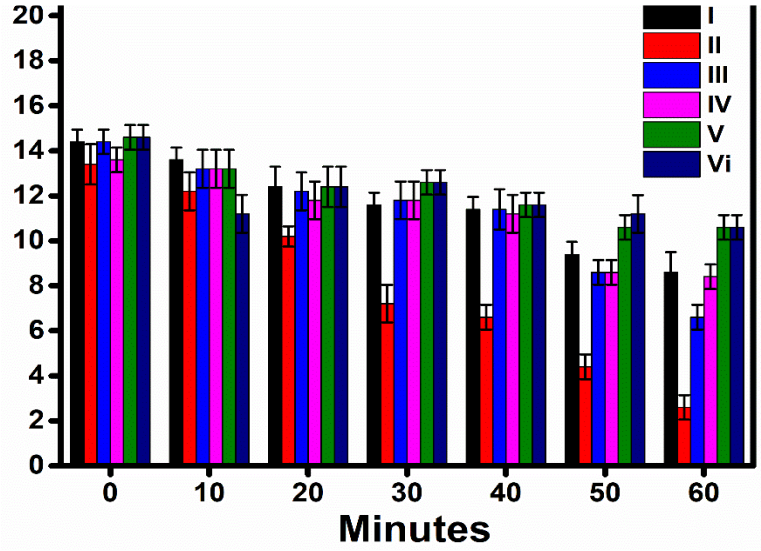
*p<0.05 statistically significant compared between Normal and Disease Control Group, **p<0.05 statistically significant compared between Disease Control and Drug treated Group

Figure: 2 Effect of *Pergularia daemia* Forsk. on the immobility of rats during forced swimming test



*p<0.05 statistically significant compared between Normal and Disease Control Group, **p<0.05 statistically significant compared between Disease Control and Drug treated Group

Figure:3 Effect of *Pergularia daemia Forsk*. on Hole cross test of PTZ induced epileptic rats



*p<0.05 statistically significant compared between Normal and Disease Control Group, **p<0.05 statistically significant compared between Disease Control and Drug treated Group

CONCLUSION

The present study, which examines convulsion models induced by PTZ, indicates both neurotransmitters and neuropeptides are altered in epileptic foci in the hippocampus. Ictogenesis is by a inequality neurotransmitter imbalance between glutaminergic neurons with excitotoxicity and presynaptic GABAergic neurons with hypoactivity. Since serotonin hypoactivity via 5-HT receptors and dopamine hypoactivity via D2 receptors have a proconvulsant effect, post-synaptic excitatory neurotransmitters are also involved in neural networks. The animals treated with Aqeous extract of *Pergluaria daemia* Forsk. significantly restore all neurotransmitter levels in the brain. We conclude that our Aqeous extract of *Pergularia daemia* Forsk. acquires antiepileptic activity.

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AUTHOR'S CONTRIBUTIONS

Jasmin Vinitha formation and design of study, sample assets, analysis, explanation of data and framing the manuscript. Agnel Arul John have read and authorized the final version of the manuscript.

COMPETING INTEREST

The authors have no conflicts of interest to disclose.

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