Anticonvulsant Activity of Fruit and Leaf Extract of *Emblica officinalis* against Strychnine Induced Convulsions in Albino Mice – A Comparative Study
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**Abstract:** Epilepsy is the term used for a group of disorders characterized by recurrent spontaneous seizures that apparently result from complex processes involving several neurotransmitter systems such as glutamairgic, cholinergic, and gabaergic system. Seizures can arise from virtually any cerebral pathology that increases the excitability of brain tissue. The etiologies can be divided into genetic and acquired brain lesions. *Emblica officinalis* is plant and which is well known fruit and it is called as *Usiri or usirikai* (Telugu), *amalika* (Sanskrit), *aamla* (Hindi), *aamla* (Gujarati), *aavalaa* (Marathi), *amloki* (Bengali), *Aula* (Punjabi), *nellikka* (Malayalam), *nellikkai* (Tamil and Kannada). The present study is aimed to evaluate the anticonvulsant activity of Fruit and leaf extracts of *Emblica officinalis* by Strychnine Induced Convulsions in Mice Model. Here the animals are divided into nine groups Phenytoin sodium 40mg/kg p.o is used as for standard group and test groups received low dose 200mg/kg p.o, medium dose 400mg/kg p.o, high dose 600mg/kg p.o for Fruit and leaf extracts respectively. The results demonstrate the strong anticonvulsant activity of *Emblica officinalis* fruit and leaf extracts (200, 400 & 600 mg/kg) in strychnine induced convulsions. The present study provides the benefits of *Emblica officinalis* fruit and leaf extracts using in the treatment of epilepsy.

**Keywords:** Anticonvulsant Activity, *Emblica officinalis*, Strychnine, Convulsions, Mice, Epilepsy.

**INTRODUCTION**
Mental, behavioral and social health problems are the major health problems in the world. Yet these have received scant attention outside the wealthier, industrialized nations.

The World Health Organization (WHO) has declared 2001 as the year for mental health in recognition of the burden that mental and brain disorders pose on people and families affected by them. In the last ten years of the 20th century is called in neuroscience “decade of the brain”. Epilepsy usually begins in childhood, potentially impeding education, employment, social relationships and development of a sense of self-worth. Epilepsy is among the disorders that are strongly associated with significant psychological and social consequences for everyday living. There is no doubt that epilepsy belongs to the most encountered neurological conditions since the disease affects approximately 1% of the population. Epilepsy is one of the most common neurological disorders with reported prevalence of 6 - 8/100,000, incidence of 30-50/100,000 per year and cumulative incidence of 3%. It requires prolonged and sometimes life-long drug therapy. The prevalence of epilepsy in developing countries is usually higher than in developed countries [1].

Epilepsy is the term used for a group of disorders characterized by recurrent spontaneous seizures that apparently result from complex processes involving several neurotransmitter systems such as glutamairgic, cholinergic, and gabaergic system. Cognitive impairment is a frequently occurring secondary consequence of epilepsy. The interest in the cognitive side effects of antiepileptic drug (AED) treatment is of recent origin. The first studies are from the early 1970s and were probably stimulated by the widening range of possibilities for drug treatment during that period [i.e., the introduction of Carbamazepine (CBZ) and valproante (VPA)]. The major commonly used AED include CBZ, VPA, and Phenytoin [2].

**EPILEPSY**
Epilepsy is a seizure disorder resulting from sudden bursts of electrical energy in the brain. These electrical discharges produce seizures which vary from one person to another in frequency and form. Sometimes the electrical signal only reaches part of the
brain where a part of the body, like an arm or a leg may move on its own. If the signal goes through all of the brain, the person may shake all over, fall and lose consciousness. It is not a disease, psychological disorder of contagious [3].

CAUSES OF EPILEPSY

Epilepsies constitute a large group of neurological diseases with an incidence of 0.5-1% in the general population. The type of seizure depends on the site of the focus in the brain. Epileptic attack can be caused by biochemical insults to the brain, such as hypoglycaemia, anoxia, hypocalcaemia, hyperventilation, water intoxication and sudden withdrawal of certain drugs such as barbiturates or alcohol. Epilepsy can also be caused by previous active pathology, such as birth trauma to the brain, during or following meningitis, trauma to the skull and brain later in life, cerebral abscesses, cerebral infarction, cerebral haemorrhage or subarachnoid haemorrhage. Other causes included head injury with/without intracranial haemorrhage, central nervous system and systemic [3].

TYPES SEIZURE

Seizures are classified according to their clinical features and patterns seen on the EEG. Epileptogenic seizures are divided into two broad categories based on the symptoms and EEG findings observed at the onset of the seizure. If the initial onset indicates involvement of both sides of the brain, the seizures are referred to as generalized seizures and if involvement of only a localized area of the brain, they are referred to as partial seizures[3].

Partial (focal, local) seizure

- Simple partial seizure
- Complex partial seizure
- Partial seizure evolving to secondary generalized seizure.

Generalized seizure

- Absence seizure
- Myoclonic seizure
- Clonic seizure
- Tonic seizure
- Tonic-clonic seizure
- Atonic seizure

PARTIAL SEIZURES

Partial seizures happen when the disturbance occurs in just one part of the brain. With these seizures, the activity can start in one place in the brain and then move to another or it could just stay in the one area. These may produce relatively simple symptoms without loss of consciousnesses, such as involuntary muscle contractions, abnormal sensory experiences.

Fig-1: Partial seizure: discharge remains localized

Simple partial seizures

They are remarkably different from person to person, depending on the part of the brain where they begin. The patient does not lose consciousness and therefore is able to tell what happened, but the experience may be so strange that he may not be able to express himself properly. What happens is dependent on the location of the affected area.

Complex partial seizures

Complex partial seizures generally last 30 seconds to 2 minutes and are also followed by a short period of confusion or impairment (typically 15 minutes). Here the patient has impaired consciousness, there is no complete loss of consciousness, he is slightly aware of what is going on, but he cannot respond to anything, neither can he change his behaviour during an attack.

Partial seizures secondary generalized

If the seizure starts as a partial seizure, then spreads to include the entire brain, it is referred to as a partial seizure secondarily generalized. This means it started as a partial seizure then became a generalized seizure.

Fig-2: Secondary generalized seizure: epileptic discharge initially localized and then spreads to trigger a generalized seizure

GENERALIZED SEIZURES

Generalized seizures are produced by electrical impulses from throughout the entire brain, once initiated; it spreads quickly into the entire or at least the greater part of the brain. The primary generalized seizures are characterized by a complete loss of consciousness and the absence of an aura.
They come on suddenly and unexpectedly, and if the patients fall, they may injure themselves. The generalized seizures consist of six different seizure types, of which the primary generalized tonic-clonic seizure (GTCS) is the most common.

Absence seizures (petit mal seizure)
Absence seizures (also called petit mal (little illness) because they occurred so frequently) are a lapse of awareness, sometimes with staring, that begins and end abruptly, lasting only a few seconds.

More common in children than in adults. Absence seizures are characterized by a brief impairment of consciousness, which usually lasts no more than a few seconds.

Myoclonic seizures
Myoclonic seizures are rapid, brief contractions of bodily muscles, which usually occur at the same time on both sides of the body. Occasionally, they involve one arm or a foot. People usually think of them as sudden jerks or clumsiness. A variant of the experience, common to many people who do not have epilepsy, is the sudden jerk of a foot during sleep.

Clonic seizures
These seizures are generalized seizures, where the tonic component is not present, only repetitive clonic jerks (clonic jerks are repetitive rhythmic flexing and stretching of limbs).

Tonic seizures
Tonic seizures are sudden sustained muscle contractions, fixing the limbs in some strained position. There is immediate loss of consciousness. Often there is a deviation of the eyes and head towards one side, sometimes rotation of the whole body. They are seen mainly in paediatric practice.

Generalized Tonic Clonic Seizures (GTCS)
Generalized tonic clonic seizures (grand mal seizures) are the most common and best known type of generalized seizure. Generalized seizures happen when the electrical disturbance sweeps through the whole brain at once, causing loss of consciousness falls and convulsions. They begin with stiffening of the limbs (the tonic phase), followed by jerking of the limbs and face (the clonic phase). During the tonic phase, breathing may decrease or cease altogether, producing cyanosis (blueing) of the lips, nail beds, and face. This clonic phase usually lasts less than a minute [3].

Emblica officinalis Gaertn., commonly known as amla (Hindi) and gooseberry (English), has been used in the traditional system of medicine to reduce fever, alleviate asthma, treat constipation and enhance digestion, strengthen the heart, benefit the eyes, enhance intellect and as a health tonic[4]. The fruits of E. officinalis have potent antioxidant activity due to the presence of tannoids, tannins, vitamin C and flavonoids [5]. The pharmacological studies on E. officinalis fruit have revealed that it has good antioxidant, cytoprotective and immunomodulatory, anti-diabetic, hypolipidemic, antitussive, cardioprotective, antiulcerogenic and hepatoprotective activity [6-14]. Since, the primary underlying pathology of epilepsy is attributable to increased oxidative stress and in view of the fact that E. officinalis possesses very high antioxidant activity, it was interesting to investigate the effect of Methonolic extract of E. officinalis Fruit and leaves on Strychnine induced Convulsions in Mice model.

MATERIALS AND METHODS

Plant materials
Emblica officinalis - Fruits
Emblica officinalis - Leaves

Collection of plant material
Plant materials Emblica officinalis fruits and leaves were collected in the surroundings of sathupally, the collected plant materials are washed thoroughly in water, dried under shade and the dried plant materials are Authenticated then the dried materials are pulverized in electrical grinder to a fine powder.

Extraction of plant materials
The crushed materials of Emblica officinalis fruits and leaves were extracted with Ethanol by Soxhlet apparatus. The extracts obtained were evaporated to dryness under low pressure and stored in refrigerator for pharmacological studies. The yield of the fruit and leaf extracts were found to be 24%w/w and 20 % w/w respectively.

PHYTOCHEMICAL STUDIES [15-16]
Collected extracts were subjected to various chemical tests for the preliminary determination of phytoconstituents. All extracts were mixed with equal proportion of alcohol and water (to get a hydro-alcoholic sample), before subjected to various chemical reagents.
PHYTOCHEMICAL SCREENING

Phytochemical examinations were carried out for all the extracts as per the standard methods.

Detection of alkaloids

Extracts were dissolved individually in dilute hydrochloric acid and filtered.

- **Mayer’s Test**
  Filtrates were treated with Mayer’s reagent (potassium mercuric iodide). Formation of a yellow colored precipitate indicates the presence of alkaloids.

- **Wagner’s Test**
  Filtrates were treated with Wagner’s reagent (iodine in potassium iodide). Formation of brown/reddish precipitate indicates the presence of alkaloids.

- **Dragendorff’s Test**
  Filtrates were treated with Dragendorff’s reagent (solution of potassium bismuth iodide). Formation of red precipitate indicates the presence of alkaloids.

- **Hager’s Test**
  Filtrates were treated with Hager’s reagent (saturated picric acid solution). Presence of alkaloids was confirmed by the formation of yellow colored precipitate.

Detection of carbohydrates

Extracts were dissolved individually in 5 ml distilled water and filtered. The filtrates were used to test for the presence of carbohydrates.

- **Molisch’s Test**
  Filtrates were treated with 2 drops of alcoholic α-naphthol solution in a test tube. Formation of the violet ring at the junction indicates the presence of carbohydrates.

- **Benedict’s Test**
  Filtrates were treated with Benedict’s reagent and heated gently. Orange red precipitate indicates the presence of reducing sugars.

- **Fehling’s Test**
  Filtrates were hydrolysed with dilute HCl, neutralized with alkali and heated with Fehling’s A & B solutions. Formation of red precipitate indicates the presence of reducing sugars.

Detection of glycosides

Extracts were hydrolysed with dilute HCl, and then subjected test for glycosides.

- **Modified Borntrager’s Test**
  Extracts were treated with ferric chloride solution and immersed in boiling water for about 5 minutes. The mixture was cooled and extracted with equal volumes of benzene. The benzene layer was separated and treated with ammonia solution. Formation of rose-pink color in the ammonical layer indicates the presence of anthranol glycosides.

- **Legal’s Test**
  Extracts were treated with sodium nitroprusside in pyridine and sodium hydroxide. Formation of pink to blood red color indicates the presence of cardiac glycosides.

Detection of saponins

- **Froth Test**
  Extracts were diluted with distilled water to 20ml and this was shaken in a graduated cylinder for 15 minutes. Formation of 1 cm layer of foam indicates the presence of saponins.

  - **Foam Test**
    0.5 gm of extract was shaken with 2 ml of water. If foam produced persists for ten minutes it indicates the presence of saponins.

Detection of phytosterols

- **Salkowski’s Test**
  Extracts were treated with chloroform and filtered. The filtrates were treated with few drops of conc. sulphuric acid, shaken and allowed to stand. Appearance of golden yellow color indicates the presence of triterpenes.

- **Libermann-Burchard’s test**
  Extracts were treated with chloroform and filtered. The filtrates were treated with few drops of acetic anhydride, boiled and cooled. Conc. sulphuric acid was added. Formation of brown ring at the junction indicates the presence of phytosterols.

Detection of phenols

- **Ferric Chloride Test**
  Extracts were treated with 3-4 drops of ferric chloride solution. Formation of bluish black color indicates the presence of phenols.

Detection of tannins

- **Gelatin Test**
  To the extract, 1% gelatin solution containing sodium chloride was added. Formation of white precipitate indicates the presence of tannins.

Detection of flavonoids

- **Alkaline Reagent Test**
  Extracts were treated with few drops of sodium hydroxide solution. Formation of intense yellow color, which becomes colorless on addition of dilute acid, indicates the presence of flavonoids.
• **Lead acetate Test**
  Extracts were treated with few drops of lead acetate solution. Formation of yellow colour precipitate indicates the presence of flavonoids.

**Detection of proteins and aminoacids**

• **Xanthoproteic Test**
  The extracts were treated with few drops of conc. Nitric acid. Formation of yellow color indicates the presence of proteins.

• **Ninhydrin Test**
  To the extract, 0.25% w/v ninhydrin reagent was added and boiled for few minutes. Formation of blue color indicates the presence of amino acid.

• **Detection of diterpenes Copper acetate Test**
  Extracts were dissolved in water and treated with 3-4 drops of copper acetate solution. Formation of emerald green color indicates the presence of diterpenes.

**Pharmacological comparative evaluation of emblica officinalis plant extracts**

All the study protocols were approved by the Institutional Animal Ethical Committee (IAEC). Animals were procured from Sainath labs, Hyderabad. Kept under laboratory standard conditions on a 12-night/dark cycle with light, in a temperature (22°C) controlled room and were acclimatized for a minimum of 7 days before experiment was performed. They were housed in cages with free access to food & water. Animals were acclimatized to laboratory conditions for 48 hours prior to experimental protocol to minimize non-specific stress. Animals were maintained on a standard mouse diet. Before the experiments. Food was withheld for 12h before the start of experiments.

**Acute toxicity study and dose selection:** (OECD Guidelines-420, 2006)

Acute toxicity study for the two extracts of *Emblica officinalis* fruit and leaf were carried out on mice according to OECD guidelines.

Four mice were used for each extraction. Each animal received single dose of plant extract (2000 mg/kg body weight).

**Annexure – 2c – OECD Guidelines: Test Procedure Starting Dose of 300 mg/kg B.W.**

Start

300 mg/kg 3 animals

0-1 (None of animals died)

1000 mg/kg 2-3 (None of animals died)

2000 mg/kg (None of animals died)

LD50 cut off 2000 mg/kg B.W.

The maximum safe dose was found to be 2000mg/kg so the therapeutic dose 1/10th of the LD50 is 200 mg/kg B.W.

**Anticonvulsant activity against Strychnine induced convulsions in mice**

**Experimental design [17]**

Animals are dividing into 9 groups with uniform weight. Treat the animals daily with the test compound or standard for 7 days. Challenge the animals with strychnine 4mg/kg i. p on day 7 just 30 Min. after giving the test or standard compounds. Then place the animals into individual cages & observe the animals for the symptoms of convulsions and death during the next 30 min. Record the time taken for the onset of seizures, duration of convulsion period & death. At least 80-90% of animals in the control group must show the convulsions. The onset of convulsions and the number of animals convulsing or not convulsing within the observation period was noted. The ability of the test drug to prevent or delay the onset of the convulsions exhibited by the animals was taken as an indication of anticonvulsant activity. Time interval between inducing agent and onset of seizures was measured.

**PROCEDURE**

The Swiss albino mice weighing 15-20 g were used. Animals were divided into seven groups (n=6) as follows-

**Group-I (Normal control)**

The animals of this group received vehicle distilled water 0.5ml/kg p.o route for seven days.
Group-II (Disease control)  
The animals of this group received vehicle distilled water 0.5ml/kg p.o route for seven days and challenged with strychnine 4mg/kg i.p on 7th day 30 min. after dosing.

Group-III (Standard control)  
The animals of this group received Phenytoin sodium 40mg/kg p.o for seven days and challenged with strychnine 4mg/kg i.p on 7th day 30 min. after dosing.

Group-IV (EFFEO (Low))  
The animals of this group received *Emblica officinalis* fruit extract 200mg/kg p.o for seven days and challenged with strychnine 4mg/kg i.p on 7th day 30 min. after dosing.

Group-V (EFFEO (Medium))  
The animals of this group received *Emblica officinalis* fruit extract 400mg/kg p.o for seven days and challenged with strychnine 4mg/kg i.p on 7th day 30 min. after dosing.

Group-VI (EFFEO (High))  
The animals of this group received *Emblica officinalis* fruit extract 600mg/kg p.o for seven days and challenged with strychnine 4mg/kg i.p on 7th day 30 min. after dosing.

Group-VII (ELEEO (Low))  
The animals of this group received *Emblica officinalis* leaf extract 200mg/kg p.o for seven days and challenged with strychnine 4mg/kg i.p on 7th day 30 min. after dosing.

Group-VIII (ELEEO (Medium))  
The animals of this group received *Emblica officinalis* leaf extract 400mg/kg p.o for seven days and challenged with strychnine 4mg/kg i.p on 7th day 30 min. after dosing.

Group-IX (ELEEO (High))  
The animals of this group received *Emblica officinalis* leaf extract 600mg/kg p.o for seven days and challenged with strychnine 4mg/kg i.p on 7th day 30 min. after dosing.

STATISTICAL ANALYSIS  
The collected data was analysed by using the suitable statistical model and test the significance at P>0.05 level.

RESULTS AND DISCUSSION  
Phytochemical analysis  
Dried powder of leaves & Fruits of *Emblica officinalis* was subjected to soxhlet extraction and the % yield was found to be 24% & 20% respectively.

![Table 1: Extraction of plant materials](http://saspublisher.com/sajp/)

<table>
<thead>
<tr>
<th>Plant name</th>
<th>Solvent used</th>
<th>Method of extraction</th>
<th>Percentage yield</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Emblica officinalis</em>(fruits)</td>
<td>Ethanol</td>
<td>Soxhletation</td>
<td>24%</td>
</tr>
<tr>
<td><em>Emblica officinalis</em>(leaves)</td>
<td>Ethanol</td>
<td>Soxhletation</td>
<td>20%</td>
</tr>
</tbody>
</table>

After complete evaporation of the solvent the extract was preserved in the refrigerator for the further Phytochemical & Pharmacological studies.

![Table 2: Phytochemical analysis of plant extracts](http://saspublisher.com/sajp/)

<table>
<thead>
<tr>
<th>Chemical list</th>
<th><em>Emblica officinalis</em> (fruit)</th>
<th><em>Emblica officinalis</em> (leaf)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alkaloids</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Carbohydrates</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Proteins</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Amino acids</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Steroids</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Terpenoids</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Glycosides</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Flavonoids</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Tannins</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>

PHARMACOLOGICAL STUDIES  
Acute Toxicity Study (OECD Guidelines-420, 2006)  
Acute Toxicity study for the plant extracts was carried out on mice according to OECD guidelines. No, mortality or any other autonomic or behavioural responses such as tremors, convulsion, salivation, diarrhoea, lethargy, sleep and/or coma were observed during first 14 days. Fruit and leaf extracts were found to be safe up to dose levels of 2000mg/kg body weight.

Anticonvulsant activity  
In the present study an attempt has been made to evaluate the anticonvulsant activity of *Emblica officinalis* leaf and fruit extract against strychnine induced convulsions in mice in comparison with
phenytoin as standard. The formulations were subjected to acute toxicity studies as per OECD guidelines since no death was observed at 2000 mg/kg, 200, 400 & 600 mg/kg doses of the extracts were taken as effective doses for screening anticonvulsant activity.

The Anticonvulsant activity was evaluated by using strychnine induced convulsion method. In the present study, mice were treated with respective standard drug and test extracts for 7 days by oral rout. On 7th day 30 min after administration of standard and test extracts all the groups were challenged with strychnine (4mg/kg i.p.) except the Normal Control to induce convulsions. Strychnine interferes with postsynaptic inhibition mediated by glycine. Glycine is an important inhibitory transmitter and amino acid. Strychnine acts as a selective, competitive antagonist to block the inhibitory effects of glycine at all glycine receptors.

Disease control group received only strychnine on 7th day and the occurrence of jerks, clonic and tonic seizures were found to be 1.16±0.35, 1.38±0.19, and 1.35±0.18 min respectively. The third group received standard drug phenytoin for 7 days and strychnine 4mg/kg on 7th day, onset time for jerks; clonic and tonic seizures were found to be increased when compared to normal group and are as follows 24.38±0.78, 26.2±1.3, 31±0.83 min respectively. Results represented in the table No. 5 & in graphs 1, 2 & 3.

In the present study group 4, 5, and 6 were treated with Emblica offcinalis fruit extract (EFEEO) 200, 400 & 600 mg/kg respectively for 7 days and 7, 8 & 9 groups were treated with Emblica officinalis leaf extract (ELEEO) 200, 400 & 600 mg/kg respectively for 7days and all these groups were challenged with strychnine on 7th day. All the doses (EFEEO 200, 400 & 600 mg/kg and ELEEO 200, 400 & 600 mg/kg) of extracts were showed anticonvulsant activity by increasing the onset time for jerks (26.4±0.83, 28.8±0.70 & 29.1±0.69 and 15.8±1.30, 16.4±1.14 & 17.2±1.21), clonic (28.6±0.54, 30±0.70 & 30.6±0.70 and 18.1±1, 15.6±0.83 & 16.1±0.91) and tonic seizures (31.7±1.98, 32±1.14 & 32.3±1.04 and 22.8±0.54, 27±0.54 & 27.8±0.42) in treated groups when compared to untreated group respectively. Anticonvulsant activity of the extracts was compared with that of the standard drug phenytoin. Both 200, 400 & 600 mg/kg doses of EFEEO were showed significant anticonvulsant activity when compared to standard. Further all doses of EFEEO showed more anticonvulsant activity than the phenytoin. EFEEO 400 & 600 mg/kg was found to be more potent than EFEEO 200 mg/kg and phenytoin. Results represented in the table No. & graphs 1, 2 & 3.

MLEEO 200, 400 & 600 mg/kg showed significant anticonvulsant activity when compared to standard disease control. But leaf extract was found to be less potent than the standard as well as fruit extract.

![Fig-4: Onset of jerking movements in strychnine induced convulsions](image)

Results were expressed as Mean±SD;(n=6). The data was analyzed by one way ANOVA followed by Dunnet test, significance at p<0.01** when compared with the standard.
Fig-5: Onset of clonic seizures in strychnine induced convulsions

Results are expressed as Mean±SD; (n=6). The data was analyzed by one way ANOVA followed by Dunnet test, significance at p<0.01** when compared with the standard.

Fig-6: Onset of tonic Seizures in strychnine induced convulsions

Results were expressed as Mean±SD; (n=5). The data was analyzed by one way ANOVA followed by Dunnet test, significance at p<0.01** when compared with the standard.

Fig-7: % protection against mortality

Results were expressed as Mean±SD; (n=6). The data was analyzed by one way ANOVA followed by Dunnet test.
Table 3: Effect of standard drug and test extracts on onset time of jerks, tonic and clonic seizures in mice against strychnine induced convulsions

<table>
<thead>
<tr>
<th>GROUP</th>
<th>TREATMENT (p.o)</th>
<th>DOSE (mg/kg)</th>
<th>ONSET OF JERKS (min)</th>
<th>ONSET OF CLONIC SEIZURES</th>
<th>ONSET OF TONIC SEIZURES</th>
<th>STATUS OF THE ANIMAL</th>
<th>% OF PROTECTION AGAINST MORTALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal control</td>
<td>Vehicle</td>
<td>0.5</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0/5</td>
<td>0%</td>
</tr>
<tr>
<td>Disease control</td>
<td>Vehicle</td>
<td>0.5</td>
<td>1.16±0.35</td>
<td>1.38±0.19</td>
<td>1.35±0.18</td>
<td>5/0</td>
<td>0%</td>
</tr>
<tr>
<td>Standard</td>
<td>Phenyoitn</td>
<td>4</td>
<td>24.38±0.78**</td>
<td>26.2±1.30**</td>
<td>31±0.83**</td>
<td>0/5</td>
<td>100%</td>
</tr>
<tr>
<td>EFEEO (Low)</td>
<td>EFEEO</td>
<td>200</td>
<td>26.4±0.83**</td>
<td>28.6±0.54**</td>
<td>31.7±1.98**</td>
<td>0/5</td>
<td>100%</td>
</tr>
<tr>
<td>EFEEO (Medium)</td>
<td>EFEEO</td>
<td>400</td>
<td>28.8±0.70**</td>
<td>30±0.70**</td>
<td>32.3±1.4**</td>
<td>0/5</td>
<td>100%</td>
</tr>
<tr>
<td>EFEEO (High)</td>
<td>EFEEO</td>
<td>600</td>
<td>29.1±0.69**</td>
<td>30.6±0.70**</td>
<td>32.3±1.04**</td>
<td>0/5</td>
<td>100%</td>
</tr>
<tr>
<td>ELEEO (Low)</td>
<td>ELEEO</td>
<td>200</td>
<td>15.8±1.30**</td>
<td>18.1±1**</td>
<td>22.8±0.54**</td>
<td>3/2</td>
<td>40%</td>
</tr>
<tr>
<td>ELEEO (Medium)</td>
<td>ELEEO</td>
<td>400</td>
<td>16.4±1.14**</td>
<td>15.6±0.83**</td>
<td>27±0.54**</td>
<td>2/3</td>
<td>60%</td>
</tr>
<tr>
<td>ELEEO (High)</td>
<td>ELEEO</td>
<td>600</td>
<td>17.2±1.21**</td>
<td>16.1±0.91**</td>
<td>27.8±0.42**</td>
<td>2/3</td>
<td>60%</td>
</tr>
</tbody>
</table>

Results are expressed as Mean ± SD; (n=6). The data was analyzed by one way ANOVA followed by Dunnet test, significance at p<0.01** when compared with the standard.

CONCLUSION

The results demonstrate the strong anticonvulsant activity of Emblica officinalis fruit and leaf extracts (200, 400 & 600 mg/kg) in strychnine induced convulsions. The present study provides the benefits of Emblica officinalis fruit and leaf extracts using in the treatment of epilepsy.

On the basis of results, it can be concluded that, Emblica officinalis fruit and leaf extracts (200, 400 & 600 mg/kg), exert significant anticonvulsant activity when compared to the standard drug phenytoin. Fruit extract is having more anticonvulsant activity than leaf extract. These could be due to different concentration of active principles, each with single or diverse range of biological activities, which shows synergistic action. Further fruit extract was showing more anticonvulsant activity than the standard drug and leaf extract. This study emphasized the need for the preparation of herbal formulations making use of standardized extracts and to carry out in-depth pharmacological evaluation of these extracts and ascertain their claims in the light of modern scientific understanding such that their potentials may be tapped for better use as alternate and safe herbal drugs.

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REFERENCES
1. Carpio RA. Campostrini; Mortality of Epilepsy in Developing Countries; Epilepsia, 46(Suppl. 11):28–32, 2005.


