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Protective role of *Ashwagandharishta* and flax seed oil against maximal electroshock induced seizures in albino rats

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Abstract

Ashwagandharishta, an Ayurvedic classical formulation, is the remedy for *Apasmara* (epilepsy), *Murchha* (syncope), *Unmada* (psychosis), etc. Recent studies in animal models have shown that n-3 PUFAs can raise the threshold of epileptic seizures. The indigenous medicinal plant, called *Atasi* (*Linum usitatissimum* Linn.) in Ayurveda, or flax seed, is the best plant source of omega-3 fatty acids. The present study is designed to investigate whether *Ashwagandharishta* and *Atasi taila* (flax seed oil) protect against maximal electroshock (MES) seizures in albino rats. Further, a possible protective role of flax seed oil as an adjuvant to *Ashwagandharishta* in its anticonvulsant activity has also been evaluated in the study. MES seizures were induced for rats and seizure severity was assessed by the duration of hind limb extensor phase. Phenytoin was used as the standard antiepileptic drug for comparison. Both flax seed oil and *Ashwagandharishta* significantly decreased convulsion phase. Pre-treatment with flax seed oil exhibited significant anticonvulsant activity by decreasing the duration of tonic extensor phase. Contrary to the expectations, pre-treatment with flax seed oil as an adjuvant to *Ashwagandharishta* failed to decrease the tonic extensor phase; however, it significantly decreased the flexion phase ($P < 0.001$) and duration of the convulsions ($P < 0.05$). Both the drugs exhibited an excellent anti–post-ictal depression effect and complete protection against mortality.

Key words: Antiepileptic, *Ashwagandharishta*, flax seed oil, maximal electroshock seizure, omega-3 fatty acid

Introduction

Epilepsy is a neuropsychological disorder, which occurs due to over discharge of neurotransmitter substance.[1] Epilepsy, anxiety, and depression are all common disorders. It is therefore not surprising that the conditions coexist in a significant number of patients. Indeed, some authors estimate the lifetime prevalence of depression in association with epilepsy to be as high as 55%.[2] Despite this, there has been remarkably little research into the mechanism of depression and anxiety in epilepsy, and even less on its treatment.[3] There are number of drugs available for the treatment of epilepsy in modern therapy. But the major disadvantage being faced is their side effects. One patient out of three is resistant to antiepileptic drug;[1] thus, there is a need for new drugs which have least side effects and minimum interaction and provide more effectiveness.

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Ashwagandharishta, an Ayurvedic classical formulation, is the remedy for *Apasmara* (epilepsy), *Shosha* (tuberculosis), *Murchha* (syncope), *Unmada* (psychosis), *Mandagni* (poor digestive power), etc.[4] *Ashwagandha* (*Withania somnifera* D.), a main ingredient of *Ashwagandharishta* [Table 1], has anti-stress and anxiolytic activities. It works as an antidepressant by enhancing 5-HT neurotransmission and omega-3s promote transmission of the chemical messengers that facilitate communication between nerve cells and are associated with emotional stability (e.g., serotonin) and positive emotions (e.g., dopamine).[5] Further, it also affects brain-derived neurotrophic factor (BDNF), which encourages synaptic plasticity, provides neuroprotection, enhances neurotransmission, and has antidepressant effects.[6]

Omega-3 fatty acids (or n-3 PUFAs) are essential for normal brain development and a deficiency in these fatty acids can contribute to the emergence of neurologic dysfunctions. With respect to epilepsy, recent studies in animal models have shown that n-3 PUFAs can raise the threshold of epileptic seizures.[7] The indigenous medicinal plant, called *Atasi* in Ayurveda, or flax seed, is the best plant source of omega-3 fatty acids. It is the richest source of alpha-linolenic acid (ALA) which can be endogenously converted to longer chain omega-3 fatty acids,

eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Flax seed oil delivers greater amount of omega-3s compared to ground flax seed and it is the easiest form to take [Table 2]. Fatty acids may play an important role in the management of seizures[8] and ALA may have anticonvulsant effects.[9]

Lipids are important constituents of the neuronal membrane and fatty acid profile changes may alter membrane functions. Essential fatty acids and phospholipids may: i) offset the deleterious effects of substances that induce seizures, such as iron, that have been shown to increase lipid peroxidation; ii) offer stability in membrane fluidity that may otherwise be associated with seizures; and iii) control the alteration in neuronal membrane phospholipid metabolism that may result from the high prevalence of excitatory amino acid receptors in the epileptic focus. A genetic link may exist between epilepsy and PUFA deficit,[10] and a mixture of DHA and EPA may provide some measure of correction. Furthermore, essential fatty acids may serve as neuroprotectors in the brain, similar to the effects observed in the heart, as shown in recent studies where ALA (but not palmitic acid) was found to protect against ischemic-induced neuronal death, and to prevent kainate-induced seizures and its associated neuronal death.[11]

Thus, the present study was undertaken to evaluate the anticonvulsant activity of *Ashwagandharishta* and flax seed oil which is the richest plant source of omega-3 fatty acids. Further, protective role of flax seed oil as an adjuvant to *Ashwagandharishta* has also been evaluated to know its effect on post-ictal depression.

Table 1: Ingredients of *Ashwagandharishta*

Drugs	Latin name	Part used	Quantity (g)
Kwath Dravyas Ashwagandha	*Withania somnifera* Dunal.	Root	2400
Musali	*Chlorophytum tuberosum* Baker.	Root	960
Manjishtha	*Rubia cordifolia* Linn.	Root	480
Haritaki	*Terminalia chebula* Retz.	Pericarp	480
Haridra	*Curcuma longa* Linn.	Rhizome	480
Daruharidra	*Berberis aristata* DC.	Stem	480
Madhuka	*Glycirrhiza glabra* Linn.	Root	480
Rasna	*Pluchea lanceolata* C. B. Clarke	Root	480
Vidari	*Pueraria tuberosa* Linn.	Root tuber	480
Arjuna	*Terminalia arjuna* Roxb.	Stem bark	480
Mustaka	*Cyperus rotundus* Linn.	Rhizome	480
Trivrita	*Ipomoea terpethum* R. Br.	Root	480
Ananta (*Shweta Sariva*)	*Hemidesmus indicus* R. Br.	Root	384
Shyama (*Krishna Sariva*)	*Cryptolepis buchanani* Roem and Schult.	Root	384
Shveta Chandana	*Santalum album* Linn.	Heart wood	384
Rakta Chandana	*Pterocarpus santalinus* Linn. f.	Heart wood	384
Vacha	*Acorus calamus* Linn.	Rhizome	384
Chitraka	*Plumbago zeylanica* Muell Arg.	Root	384
Water for decoction – reduced to one fourth			41.856 L 10.464 L
Makshika (*Madhu*)	Honey		14,400
Dhataki	*Woodfordia fruticosa* Kurz.	Flower	768
Prakshepa Dravyas			
Shunthi	*Zinziber officinale* Roxb.	Rhizome	96
Maricha	*Piper nigrum* Linn.	Fruit	96
Pippali	*Piper longum* Linn.	Fruit	96
Twak	*Cinnamomum zeylanicum* Breyn.	Stem bark	192
Ela	*Elleteria cardomomum* Maton.	Seed	192
Patra	*Cinnamomum tamala* Nees and Eberm.	Leaf	192
Priyangu	*Callicarpa macrophylla* Vahl.	Flower	192
Nagakesara	*Mesua ferrea* Linn.	Stamen	96

Table 2: Comparative essential fatty acids of flax seed oil and ground flax seeds

Source	Total essential fatty acids (g)	Omega-6 FA (g)	Omega-3 FA (g)	Ratio (omega 6:omega3)
Flax seed oil (1 tbsp – 14 g)	8.9	1.7	7.2	1:4
Ground flax seed (1 tbsp – 7 g)	2	0.4	1.6	1:4

Materials and Methods

Test drugs

Flax seeds were purchased from a commercial dealer (Lallubhai Vrajlal Gandhi Ltd., Gandhi Bridge, Ahmedabad, Gujarat) of Ahmedabad in the month of December 2010. The drug was identified and authenticated by pharmacognosist of the institute. Oil was obtained (25%) by traditional grinding and expression pressing method.[12] *Ashwagandharishta* was procured from the pharmacy attached to SDM college of Ayurveda, Udupi, Karnataka. Milk (which was used as vehicle with flax seed oil) of reputed brand (Amul Taaza) was purchased from local market freshly on the day of experiment.

Animals

Wistar strain albino rats weighing 180 ± 10 g were obtained from animal house (Registration No. 548/2002/CPCSEA) attached to Pharmacology laboratory. Six animals were housed in each cage made up of polypropylene with stainless steel top grill. Dry wheat (post hulled) waste was used as bedding material and was changed every morning. The animals were acclimatized for 7 days before commencement of the experiment in standard laboratory conditions 12 ± 01 h day and night rhythm, maintained at 25 ± 3°C and 50–70% humidity as per Committee for the purpose of control and supervision of experiments on Animals (CPCSEA) guidelines. Animals were provided with balanced food (Amrut brand rat pellet feed supplied by Pranav Agro Mills Pvt. Ltd., Vadodara, Gujarat) and water *ad libitum*. Protocol used in this study for the use of animals was approved by the Institutional Animal Ethics Committee (Approval No. IAEC/09/11/12). Before subjecting them to experimentation, the animals were given a week's time to get acclimatized with laboratory conditions.

Dose selection and schedule

The dose of *Ashwagandharishta* is 50 ml/day,[13] whereas the human dose of the flax seed oil is 20 ml/day. The dose for experimental animals was calculated by extrapolating the human dose to animals based on the body surface area ratio by referring to the standard table of Paget and Barnes (1964).[14] On this basis, rat dose of *Ashwagandharishta* was found to be 4.5 ml/kg rat and the dose of flax seed oil was found to be 1.8 ml/kg rat. Flax seed oil was suspended in milk of suitable concentration depending upon the body weight of animals and administered. Phenytoin sodium was selected as the standard antiepileptic drug and administered at a dose of 25 mg/kg.[15] In all groups of animals, the drug was administered orally with the help of gastric catheter sleeved to syringe.

Anticonvulsant activity

The maximal electroshock-induced seizures (MES) test is the most frequently used in an animal model for identification of anticonvulsant activity of drugs like phenytoin for the generalized tonic–clonic seizures.[16] The MES test serves to identify compounds which prevent seizure spread, corresponding to generalized tonic–clonic seizures in humans. This model is based on observation of the stimulation induced by repeated electrical pulses in different neuronal structures.[17]

The rats were pre-tested 24 h prior to administration of test drugs for sensitivity to electric shock and those failing to give hind limb tonic extension were rejected. Thus, the screened animals were divided into six groups of six animals each. Group 1 served as control and received equivalent amount of distilled water. Group 2 served as vehicle control and received equivalent amount of milk. Group 3 received *Ashwagandharishta*. Group 4 received flax seed oil with milk, whereas Group 5 received flax seed oil along with milk followed by *Ashwagandharishta*. Sixth group received phenytoin sodium as the standard antiepileptic drug. Experiment was conducted between 9 a.m. and 11 a.m. Seizures were induced in all the groups by using an electro convulsiometer and elicited by a 60-Hz alternating current of 150 mA intensity for 0.2 sec. A drop of electrolyte solution (0.9% NaCl) with lignocaine was applied to the corneal electrodes prior to application to the rats. The animals were observed for the extensor phase as well as its duration and post-ictal depression. The abolition of extensor phase in drug-treated group was taken as the criterion for anticonvulsant activity.[18]

Statistical analysis

The data were expressed as mean ± standard error mean (SEM). Difference between the groups was statistically determined by student's *t* test for unpaired data. Level of significance was set at $P < 0.05$.

Results

The duration of extensor phase in the control group was 8.83 ± 0.47 sec [Table 3]. Pre-treatment with flax seed oil exhibited significant anticonvulsant activity by decreasing the duration of tonic extensor phase ($P < 0.05$). However, flax seed oil followed by *Ashwagandharishta* failed to protect MES-induced convulsion. The standard drug phenytoin significantly abolished the extensor phase in the animals, with 81.20% protection. Pre-treatment with vehicle, flax seed oil, and *Ashwagandharishta* significantly shortened the flexion phase of MES-induced seizure, while flax seed oil as adjuvant with *Ashwagandharishta* significantly decreased the duration of convulsion. Further, both flax seed oil and *Ashwagandharishta* significantly decreased the convulsion phase. The standard drug phenytoin had exhibited significant anticonvulsant effect by abolishing the tonic extension phase. Further, it significantly shortened all other phases induced by MES.

Discussion

Phenytoin is effective in therapy of generalized tonic–clonic and partial seizures and has been found to show strong anticonvulsant action by increasing the brain content of gamma-amino butyric acid (GABA) in MES test.[19,20]

In the present study, a significant reduction ($P < 0.05$) in the time taken for post-ictal depression was noted in *Ashwagandharishta* treated group of animals. It also shortened the flexion phase of MES-induced seizures, but failed to decrease the tonic extensor phase [Table 3]. Various ingredients of *Ashwagandharishta* have proven anticonvulsant activity. *Ashwagandha* (*W. somnifera* Dunal) itself possesses antiepileptic properties.[21,22] The major bioactive chemical principles

Table 3: Effect on MES-induced seizures in rats

Groups	Flexion (sec)	Extension (sec)	Convulsion (sec)	Recovery time (min)	% protection
Water control	5.17 ± 0.31	8.83 ± 0.47	25.67 ± 4.14	4.13 ± 0.59	-
Vehicle control (milk)	3.50 ± 0.34*	8.20 ± 0.58	24.18 ± 3.79	3.20 ± 0.65	07.65
Ashwagandharishta	3.50 ± 0.34*	9.40 ± 0.93	17.90 ± 3.98	2.59 ± 0.27*	-
Flax seed oil	2.80 ± 0.20***	6.75 ± 0.48*	16.60 ± 3.16	2.44 ± 0.16*	76.44
Flax seed oil + *Ashwagandharishta*	3.17 ± 0.75***	8.17 ± 0.48	12.60 ± 3.25*	3.52 ± 0.28	07.48
Phenytoin	1.33 ± 0.05***	1.66 ± 0.55***	14.83 ± 1.28*	1.13 ± 0.04***	81.20

Data: Mean ± SEM, *$P < 0.05$, ***$P < 0.001$

of *W. somnifera* D. appear to be the glycol-withanolides (WSG).[23,24] WSG have been shown to inhibit ibotenic acid-induced cognitive deficits in rats by reversing rat brain frontal cortical and hippocampal decreases in acetylcholine, choline acetylase, and cholinergic muscarinic receptors produced by the neurotoxin[22] and also exert significant antioxidant effect in various rat brain areas, including striatum *n.pl.*[23] The other ingredients of *Ashwagandharishta* like *Cyperus rotundus*,[25,26] *Curcuma longa*,[27-30] *Terminalia chebula*,[31,32] *Acorus calamus*,[33-36] *Pueraria tuberosa*,[37,38] and *Glycyrrhiza glabra*[39,40] are also proven to be effective against experimentally induced seizure models. Thus, the observed effect may be attributed to the combined role of these ingredients.

Flax seed oil exhibited significant anticonvulsant activity by decreasing the duration of tonic extensor phase ($P < 0.05$), flexion phase ($P < 0.001$), and recovery time ($P < 0.05$), with 76.44% protection against MES. This observation confirms the protective effect of omega-3 fatty acid against the seizures as reported earlier.[41] It has been also reported that chronic treatment with omega-3 promotes neuroprotection and positive plastic changes in the brain of rats with epilepsy,[42] with a decrease in neuronal death in CA1 and CA3 subfields of the hippocampus. This could be attributed to n-3 PUFAs' ion channel modulation[43-45] and anti-inflammatory action. In *in vitro* studies, DHA has been reported to inhibit epileptiform activity and synaptic transmission mainly through the frequency-dependent blockade of Na^+ channels in the rat hippocampus,[43] and to stabilize neuronal membrane by suppressing voltage-gated Ca^{2+} currents[46] and Na^+ channels.[41]

Contrary to the expectations, pre-treatment with flax seed oil as an adjuvant to *Ashwagandharishta* failed to decrease the tonic extensor phase; however, it significantly decreased the flexion phase ($P < 0.001$) and duration of the convulsion ($P < 0.05$). The reason for this observation needs further investigations and also can be tried in other animal models representing epilepsy.

Conclusion

The present study reveals that both *Ashwagandharishta* and flax seed oil are having antiepileptic activity; besides, they are having excellent anti–post-ictal depression effect. The observed activity may be mediated through inhibition of voltage-dependant Na^+ channels or by blocking glutaminergic excitation mediated by the N–methyl–d–aspartate (NMDA) receptor. However, further detailed investigations are needed to explore the exact mechanism involved. Further, both the drugs can play a major role as an adjuvant therapy with modern antiepileptic drugs; however, this needs detailed investigations.

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हिन्दी सारांश

अश्वगन्धारिष्ट एवं अतसी तैल का विद्युतीय तरंग जनित आक्षेप के प्रति संरक्षणात्मक प्रभाव का अध्ययन

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आयुर्वेदिक ग्रन्थों में अश्वगन्धारिष्ट का उल्लेख अपस्मार, मूर्च्छा, उन्माद आदि रोगों की चिकित्सार्थ पाया जाता है । नवीनतम अध्ययनों से ज्ञात हुआ है कि ओमेगा ३ फैटि एसिड आक्षेप के प्रति सहनशीलता के स्तर को बढ़ाता है । आयुर्वेद में वर्णित वनौषधि अतसी के बीज में ओमेगा ३ फैटि एसिड प्रचुर मात्रा में पाया जाता है । प्रस्तुत अध्ययन का उद्देश्य यह जानना था कि क्या अतसी बीज तैल एल्बिनो चूहों में विद्युतीय तरंग द्वारा उत्पन्न किये गए आक्षेप के प्रति संरक्षणात्मक प्रभाव दिखाता है ? साथ ही, अतसी तैल को अश्वगन्धारिष्ट के साथ सहायक चिकित्सा के रूप में देने पर आक्षेपहर क्रिया का भी अध्ययन इस शोध में किया गया है । चूहों में विद्युतीय तरंग जनित आक्षेप उत्पन्न कर उनकी तीव्रता का आंकलन पश्च पाद विस्तार की कालावधि के आधार पर किया गया । तुलनात्मक अध्ययन हेतु मानक आक्षेप विरोधी औषधि phenytoin का चयन किया गया । अतसी तैल एवं अश्वगंधारिष्ट दोनों ने आक्षेप की कालावधि को सांख्यिकीय दृष्टि से सार्थक रूप से कम किया । अतसी तैल द्वारा प्रारंभिक चिकित्सा के पश्चात् विद्युतीय तरंग जनित आक्षेप के tonic extensor phase में पर्याप्त कमी पाई गई । परंतु अतसी तैल को जब अश्वगंधारिष्ट के सहायक के रूप में विद्युतीय तरंग जनित आक्षेप उत्पन्न करने के पूर्व चिकित्सार्थ दिया गया तो आक्षेप के tonic extensor phase में कमी दृष्टिगोचर नहीं हुई, यद्यपि आक्षेप के flexion phase एवं कालावधि में सांख्यिकीय दृष्टि से सार्थक कमी पाई गई । दोनों औषधियों द्वारा post ictal depression की अवधि में कमी पाई गई, साथ ही प्रायोगिक चूहों पर विद्युतीय तरंग जनित आक्षेप के सद्यः प्राणहर प्रभाव के प्रति पूर्ण संरक्षण भी पाया गया ।