

Ayurveda a Boon for Epileptics

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Abstract

Apsmar denotes epilepsy, that usually persists in majority of patients with mental debility and disability, in spite of multidrug therapy with advanced anti-epileptics. But by supplementing Sodium valproate with indigenous composite of some Indian herbs as an adjuvant at 8 hourly dosing, tends to modulate the therapeutic outcome positively due to bioregulation of altered GABA bio kinetics and neurogenic action. Thus this adjunction seems a boon in epilepsy management.

Keywords: Epilepsy; Convulsions; Apsmar; Seizure; Neuronal disorder

Introduction

Apsmar, an Indian equivalent of Epilepsy, duly documented in vedic and post vedic literature of Charak and Sushrut 1000 BC is a non specific manifestation of hyper (synchronous discharge of cortical neurons and its prevalence in developing country is 57 in thousand, while in developed country like America and South Africa it is very high. Even in India also, incidences are increasing in geometric progression [1].

It is a complex brain disorder associated with both mental and physical debility, occurrence of recurrent convulsions causes neural hypoxia resulting in neuronal disorder or degeneration [2].

Improper drug scheduling among epileptics promote resistance to the receptor site causing recurrence even with wide range of available antiepileptic drugs such as; Phenobarbitone, Phenytoin, Sodiumvalproate, Carbamazepine, Ethosuccinide, Folvamate, Gabapentine, Labotegu, Oxycarbamazepine, Toperonoid, Tiagevin, Zenisonide, Tectal, Lamotrigine, Lavetiraceton, Pregabalin etc. But, non ensure complete cure of the disease except transient control of the seizure.

Almost one third of the people with epilepsy continue to have seizure (s) despite appropriate therapy with above mentioned drugs and possess considerable risk of cognitive and psychosocial dysfunction and increased health risk among the resistant epileptics. Mono therapy facilitates drug compliance, associated with lower risk of toxicity, affordability and regularity with minimized drug untoward effects.

Considering the IHO study of status of epilepsy patient taking treatment through an NGO Sanchar Kolkata and London epilepsy forum, majority cases presented with physical and mental debility in 90% cases while physical and mental handicap in 46%.

Hence a study was conducted to evaluate an indigenous composite as an adjuvant with conventional antiepileptic mono therapy in proper schedule to assess the therapeutic efficacy and safety profile [3].

Material and Methods

To evaluate the clinical efficacy of an indigenous composite as an adjuvant with widely prescribed antiepileptic monotherapy and study the status of old epileptic cases, a mass propaganda to create awareness for epilepsy patient in a multi centre epilepsy treatment camp was organized and 4568 cases of grand mal epilepsy were selected, duly interrogated (both patient and their parent), evaluated, investigated, treated and follow-up observation was given by Centre For Indigenous

Medicine & Research, RA. Hospital & Research Centre and Indian epilepsy forum.

Patients of convulsive disorder with associated other systemic diseases, patients of status epileptics and patients unable to follow the therapeutic and follow up schedule were excluded from the study.

Patient's non responsive to major therapeutics are termed resistant epileptics.

All the selected patients were investigated for CT scan, EEG, Hematological index and biochemical parameters to evaluate safety profile of the prescribed drug.

Irrespective of the disease duration, age of the patient, status of illness and previous therapeutics, all patients were given:

- Sodium valproate in recommended dose at 8 hourly schedule, strictly. (We practiced/administered doses at 6 AM, 2 PM and 10 PM)

- Indigenous composite containing (equal portions of)

- Acorus calamus (rhizome)

- Nardostachys jatamansi (root)

- Convolvulus pluricaulis

- Herpestis monneiri (leaf)

- Crotalaria verrucosa (seed)

Dose: For adult: 1 cap of 500mg daily, while in children a 1/8th decoction 5 ml in >5 yrs; <5 yrs 2.5 ml; <1 year 1.25 ml every 12 hours.

Patients were examined and data were recorded in a follow up card.

Initially, weekly for 1st month, every 15th day for next 6 months and monthly for rest of the period during the therapy and every 3 month for 2 yrs post therapy follow up during the follow up, patients were evaluated for-

- incidence of attack of seizure

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- frequency of seizure
- duration of seizure
- severity of seizure
- any untoward effect
- physical and mental status

To ensure 100% follow up with an instruction to the parent of the patient to enter the requisite information and observation in the follow up card.

To assess safety profile, patients were re-examined for hemato, hepato and renal status.

Therapeutic response was adjudged, analyzed and graded as per following index (Table 1).

Observations

• 4658 Patients of age range 5-40 years were selected for the present study. Among them 58% (2680) were male and rest female; 40.57% of all patients were of age group 10-20 years (Figure 1 and Table 2).

• Whereas disease status wise, 55.38% were recently detected, 38% were old cases of epilepsy without treatment, among them 21.9% patients were suffering from the past 1 year and others were 1-5 years old. Remaining 6.62% of patients were suffering from recurrent episodes of epilepsy in spite of a therapeutic regime (Figure 2 and Tables 3 and 4).

• As per clinical presentation 13% patients were presenting with convulsion, unconsciousness, frothing and autonomic dysfunction while old resistant cases (21%) were presenting with associated physical and mental debility (Table 5).

Grade	Characteristics
I	Complete absence of seizure, improved mental status and physical capacity without any untoward effect and Recurrence.
II	Complete absence of seizure without any untoward effect. Improved physical capabilities but unchanged mental state
III	No response

Table 1: Therapeutic response comparison as per index.

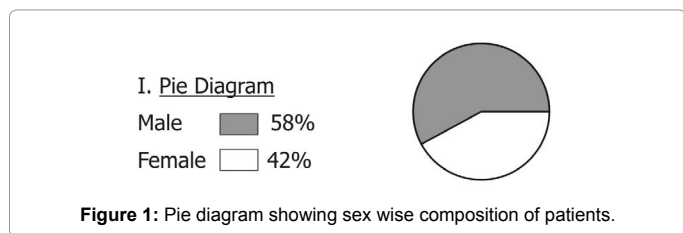


Figure 1: Pie diagram showing sex wise composition of patients.

Age Group	Number of patient			%
	Male	Female	Total	
<5 yr	72	48	120	2.6
5-10 yr	414	330	744	15.97
10-15 yr	480	320	800	17.17
15-20 yr	610	480	1090	23.4
20-25 yr	320	186	506	10.86
25-30 yr	318	236	554	11.89
30-35 yr	276	164	440	9.44
35-40 yr	190	114	304	6.52

Table 2: Distribution of patients as per age and sex.

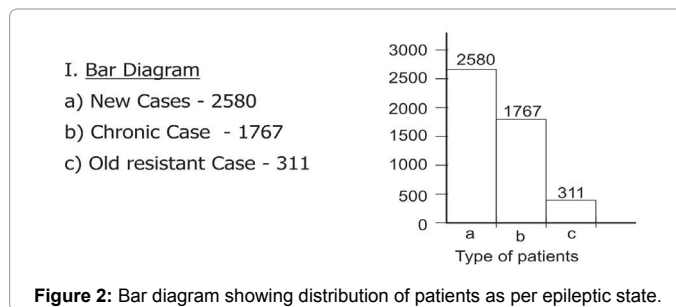


Figure 2: Bar diagram showing distribution of patients as per epileptic state.

Duration of illness	Number of patients	Percentage
<12 month	1013	21.7
1-2 yrs	1662	35.7
2-3 yrs	804	17.2
3-4 yrs	672	14.4
4-5 yrs	120	2.7
>5 yrs	381	8.3

Table 3: Distribution of patients as per duration of illness.

Therapeutic regime	Number of patients	Percentage
Phenobarbitone+Carbamezipine	49	15.75
Phenytoin sodium+phenobarbitone	52	16.72
Sodium valproate+Phenobatbitone	57	18.32
Sodium valproate+Phenytoin sodium	73	23.47
Sodium valproate+Carbamezipine	43	13.82
Phenobarbitone+Phenytoin sodium & Carbamezipine	37	11.9
	311	

Table 4: Distribution of old resistant patients as per their therapeutic regime.

Presenting feature	Number of patients	Percentage
Convulsion with unconsciousness frothing from mouth and autonomic dysfunction	607	13.05
Convulsion with unconsciousness and Frothing from mouth	2002	42.97
Convulsion with transient unconsciousness	1394	29.93
Mental debility	970	20.82
Physical debility	970	20.82

Table 5: Distribution of patients as per clinical presentation.

• Among selected patients 42.01% were with hemoglobin less or equal to 10gm%, 1.05% with altered hepatic and 1.24% with altered renal functions (Table 6).

• All newly detected cases, 96.2% of old cases and 67% of chronic resistant cases had complete cessation of epileptic attack from beginning of therapy while 23% of old cases failed to respond within 48 hours but slowly severity, frequency and duration of convulsion regressed markedly. No patient exhibited autonomic dysfunction.

• Overall 99.76% patients revealed improved mental status in terms of psychosocial behavior and intelligence (mental ability) while 0.23% (11 cases) or 3.53% of resistant cases showed no change in their mental status but all had improved physical status.

• No post therapy recurrence of seizure was reported during 2 years follow-up observation

• All newly detected cases, 96.20% of old patients and 67.52% of resistant cases had grade I clinical response while 3.8% of old and 21.8%

Particulars	Number of patients	Percentage
Hematological:		
Hemoglobin-(%)		
8-10 gm	1957	42.01
10-12 gm	2423	52.01
12-14 gm	279	5.98
Hepatic parameters:		
Serum bilirubin-		
> 1 mg%	49	1.05
<1 mg%	4609	98.95
Renal parameters:		
Blood urea-		
>30 mg%	58	1.24
<30 mg%	4600	98.76
Serum creatinine		
>1.5 mg%	54	1.16
<1.5 mg%	4604	98.84

Table 6: Distribution of patients as per their base hemato, hepatic and renal parameters.

Particulars	Number of patients		
	New cases	Old cases	
	(2580)	(1767)	Chronic resistant (311)
Convulsive attack-			
Absent	2580	1700	210
Declined	-	67	88
Non response	-	-	13
Mental status-			
Improved	2580	1767	300
Unchanged	-	-	11
Physical state-			
Improved	2580	1767	300
Unchanged	-	-	11
Safety profile-			
Recurrence of attack	None	None	109
Hepatic function-			
Altered	None	None	49
Unchanged	2580	1767	262
Renal function-			
Altered	None	None	54
Unchanged	2580	1767	257
Hematological-			
Improved	2580	1767	300
Unchanged	-	-	11
Post therapy convulsion-			
Absent	2580	1767	300
Grade of clinical response-			
Grade I	2580	1700	230
Grade II	-	67	68
Grade III	-	-	13

Table 7: Results of herbal adjunction therapy.

of resistant cases had grade II response and 4.18% of resistant cases failed to respond.

- The abnormal hemato, hepato and renal parameters of some old patients with refractory epilepsy were sustained or improved but not worsened (Table 7).

Discussion

Medical literature and study shows that available antiepileptic fails

to ensure cure and prevent recurrent intense and repeated attack of seizure associated with physical and mental debility due to hypoxia, loss of GABA mediated inhibition of dentate granule and death of GABAergic inhibitory neuron results in attenuation of inhibitory control which in turn results in hyper excitation of the remaining neuron of the hippocampus. In spite of the fact that GABA neurons are more resistant to seizure induced neuronal death than other hippocampus neurons. Mossy cells (located in dentate hilus, a part of hippocampus) are extremely sensitive to seizure induced neuronal death and damage following intense synaptic activator i.e., excite-tonic mechanism of activator of NMDA (N methyl D aspartate) a sub type of glutamate receptor which results in excessive intracellular calcium.

Release of cellular zinc attenuate GABA response and induce hyper-excitability of neurons.

GABA binds to GABA-A (coupled to calcium/chloride channel and a main target of currently prescribed drugs) as excitatory post synaptic potential are the main form of communication between neurons and is mediated by release of excitatory amino acid-Glutamate from pre-synaptic elements which is mediated by

- NMDA (N methyl D aspartic acid/aspartate)
- AMPA(Alpha-amino-3 hydroxy 5 methyl isoxazole propionic acid) kinate
- Metabotropic

GABA-B (Couples to potassium channel, a cause of latency and long duration of action) located in pre-synaptic element of an excitatory pre-neuron and inhibits pre-synaptic neuron by

- direct induction of IPSP
- inhibition of release of excitatory neuro transmitters [4,5]

Hence clinical supremacy of the adjunct indigenous composite with Sodium valproate mono drug therapy can be attributed to bioregulative action of indigenous composites active ingredients for GABA neurodynamics i.e., Nardostachys jatamansi (Jatamansin, Jatamanose, Nardostachine) Herpestis monnieri(Monnerein) bio regulate GABA biokinetics and act in synergism with Sodium valproate and prompt control of the seizure, while Acorus calamus (Acorin, Beta asarone and calaminidine) and Crotoparia verrucosa (crotallidine and verrucosin) acts as a neurogenic and helps in regeneration and repair of damaged neuron due to epileptic attacks thus improve and check neural debility and alleviate physical disability .

In addition Convolvulus pluricaulis (Covolvulin) and Herpestis monnieri (Bacoside A & B) both promote neural growth due to activation of nerve growth factor-Tyrosine kinase A receptor, preserve m RNA level of muscarnic receptors and check accumulation of lipid and protein damage ,thus improve both mental ability and capability [6].

Hence bioregulation of altered GABA neuro kinetics prompt non recurrence of seizure even after drug withdrawal and neurogenic action improve mental capability and physical capability.

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