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Research Article

AES a Presentation of Ascaris Toxin

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Abstract

Acute encephalitic syndrome (AES) though primarily caused by virus but other pathogens cannot be ignored as prompt and correct clinical acumen save majority of life.

Treatment and evaluation of 147 cases of AES admitted at our centre managed based on previous experience of similar AES prevalence in 1985, in nutritionally deprived patients of poor socio economic status with history of passing Round worm, saved all 147 cases without any adversity or adjuvant required or mortality and all passed round worm on deworming (Albendazole and Ivermectin) in therapeutic dose for 3 consecutive days after 7th day of discharge. Majority patients regained consciousness within 48 hours of therapy while seizure seized in all cases by 12 hours of therapy.

Thus, consider Round worm Encephalopathy in nutritionally deprived patients of AES in addition to other pathogens as right approach will save life, time and cost of therapy. Round worm causes encephalopathy due to competitive inhibition of Pyridoxal 5 phosphate Co enzyme, a prime co enzyme for Gama Amino Butyric Acid synthesis and metabolism in brain by its poly peptide secretion in adverse situation.

Keywords: *AES; Round Worm Encephalopathy; Pyridoxal 5 Phosphate; GABA*

Introduction

Febrile convulsion not a new presentation but continuing since long and disease gravity is increasing progressively. Initially it was claimed solely due to viral infection and was termed Japanese encephalitis but these days the presentation being termed as AES (acute encephalitic syndrome). In spite of available therapeutics and advanced diagnostic tool the mortality remain the same even at higher centre [1-5].

Acute encephalitis is the clinical diagnosis of children with acute onset of symptoms and signs of inflammatory lesions in the brain. Changes in sensorium, seizures and upper motor neuron type of altered muscle tone point to cerebral dysfunction [6].

The clinical picture usually consists of a prodromal phase (one to three days) with fever, malaise and headache and an encephalitic phase with continued fever, decreasing level of consciousness, seizures, abnormal movements or paralysis. Signs of meningeal inflammation are absent or minimal.

In summary, all that presents with fever and cerebral dysfunction are not acute encephalitis.

Acute encephalitis is mostly caused by any of the many 'neurotrophic' viruses, many of which are vector-transmitted (arthropod-borne) arboviruses. In India, Japanese encephalitis (JE) virus is the commonest. Clinical neurologic manifestations caused by wide range of viruses, bacteria, fungus, parasites, spirochetes, chemicals and toxins. correct management will depend on the correct diagnosis [7].

Considering the similar disease prevalence among the down trodden and nutritionally deprived children in 1985 who proved to be a manifestation of Ascaris lumbricoidis toxin and majority saved.

Thus, the similar line of therapeutic was evaluated in patients with AES presented at RA Hospital and Research Centre Warisaliganj (Nawada) Bihar with prime motive of ensuring cure in majority.

Objective of the Study

Ensure cure in majority and ascertain the cause of presentation.

Materials and Methods

Material

Patients with complaints of AES attending Medical emergency of RA Hospital and Research Centre during May to July 5th 2019 were considered for the study.

Methods

Parents of the admitted patients were thoroughly interrogated for onset of the disease and its progression, patients were clinically evaluated, investigated and provided basic life support and administered.

Oxygen inhalation
Ryles tube intubation for feeding (Bland, sweet, liquid oral) and Antacid with Oxetacaine (Ancool Gel) 2.5 ml every 6 hrs
IV Mannitol 10% with Glycerin 10 % in dose of 10 ml/kg every 12 hours
Inj Sodium Valproate infusion with pediatric intravenous solution
Inj Amikacin 7.5mg /Kg every 12 hours
I.V Paediatric solution plus Methyl cobalamin, Pyridoxin and Nicotinamide ½-1ml slow infusion
Pulmosafe Ointment for local chest application
Syr Neurovit through feeding tube 1.25 ml to 2.5 ml every 12 hours
Frequent change of posture
Cold sponging

Table A

Patients were observed for:

- Fever
- Convulsion
- Consciousness status
- Any evident paresis
- Any unusual presentation

On discharge on 5th day patients were advised

Susp Ancool 2.5 ml three times daily
Syr Neurovit 1.25 - 2.6 ml every 12 hourly
Syr Sodium Valproate 1.25-2.5 ml every 8 hours
Syr Becomplex 2.5 ml twice daily
Bland, simple and sweet oral liquid diet
After a week for deworming patients were advocated
Albendazole plus Ivermectin suspension in dose of 5 ml - 10 ml at bed time for 3 consecutive days
After deworming patients were advised-
Syr Neurovit 1.25-2.5 ml every 12 hours for 2 month
Syr B complex 2.5-5 ml twice daily for 2 month
Deworming every month for 3 days for 3 consecutive month every year
High protein diet
Restricts Biscuits, Kurkure etc.

Table B

Observation

Selected 147 patients were of age group 2 - 14 years and majority (38.8%) were of age group 5 - 8 years while 10.9% were of age group 11-14 years (Table 1) out of them 99 were male and 48 female respectively (Figure 1).

Age group (in years)	Number of patients			
	Male	Female	Total	Percentage
2 - 5	31	17	48	32.7
5 - 8	40	17	57	38.8
8 - 11	17	09	26	17.6
11 - 14	11	05	16	10.9

Table 1: *Showing distribution of patients as per age and sex.*

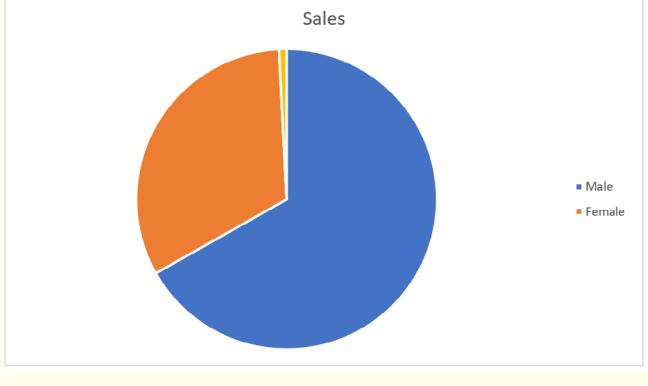

Figure 1: *Pie diagram showing male: female composition.*

Majority (49.8%) were suffering since 6 - 12 hours while 7.4% patients were from more than 24 hours (Figure 2).

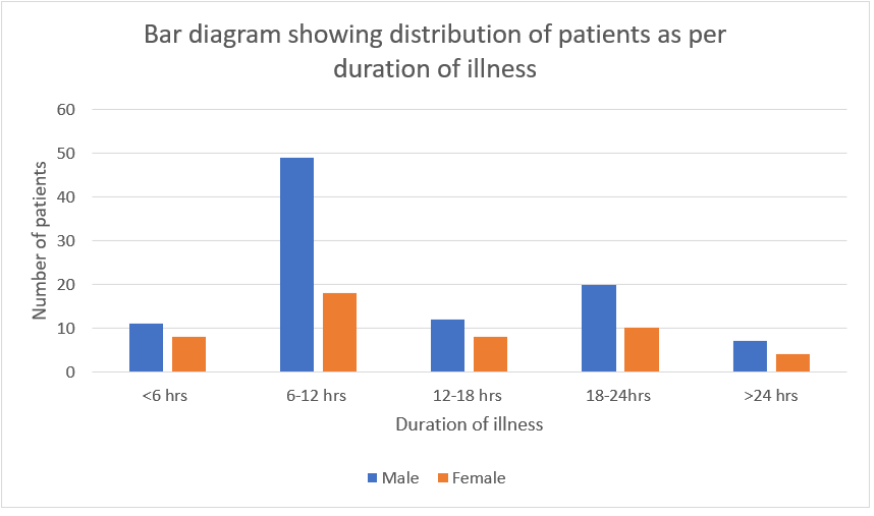

Figure 2: *Bar diagram showing duration of illness.*

Majority (55.8%) patients attended the Centre after 12 - 18 hours of onset of disease while 9.5% after > 24 hours (Figure 3).

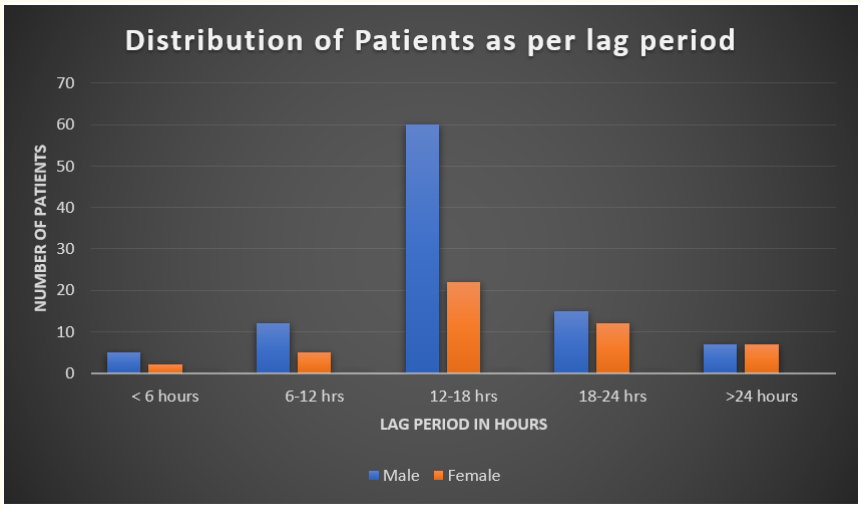

Figure 3: *Bar diagram showing distribution of patients as per lag period in attending our centre.*

Out of all 44.9% were having temperature > 102°F, 95.2% with convulsion and unconsciousness and loss of sensorium in the extremity, 70.7% with port belly abdomen, history of passing round worm, abdominal distension and urticarial rashes in past in 59.2%, 72.8% shows hemoglobin < 10 gram %, signs of malnutrition and raised eosinophil count in all the cases (Table 2).

Particulars	Number of patients
Fever	
> 102°F	66
< 102°F	81
Convulsions	
Tonic clonic	140
Mild jerks	07
Loss of sensation	147
Consciousness	
Unconscious	140
Conscious	07
Abnormal behaviour	07
History of Helminthiasis	87
Recurrent lose motion	147
Abdominal distension	147
Urticarial rash	87
Clinical examination	
Port belly abdomen	104
Signs of mal nutrition	147
Palpable liver	90
Investigation	
CBCs shows raised eosinophil	147
Haemoglobin	
< 10gm %	107
> 10gm%	40
CSF	No abnormality seen
X ray Chest	No abnormality detected
X ray abdomen	Distended intestinal loop
Blood for Malarial antigen	None
Widal	Non reactive
Blood and CSF for Viral analysis	No virus detected
EEG	No evident pathology
CT Brain	No evident pathology

Table 2: *Distribution of patients as per their presenting features.*

Result

Majority patients regained consciousness in 12 hours though 27 cases taken 40 hours to regain consciousness.

Convulsion seized within 14 hours in all the cases irrespective of their age or lag period.

Feeding tube removed after 48 hours in all the cases

No patients present with any residual paresis or neuropsychiatric changes.

After a week administration of Albendazole with Ivermectin suspension at bed time for 3 consecutive days ensure passage of plenty of round worm in all the cases

Post therapy 2 weeks follow up reveals no untoward effects or withdrawal manifestation.

All cases were repeated for their basic bio parameters shows no alteration in any of the cases.

Discussion

Nutritionally deprived Patients of Acute Encephalopathy syndrome admitted at our centre having history of passing round worm in past, vomiting and diarrhoea, occasional urticarial rash, fever been treated conventionally on the line of Round worm encephalopathy evident during 1985 shows complete recovery within 48 hours and passed round worm on deworming on 7th day after discharge [8] (Figure 4).

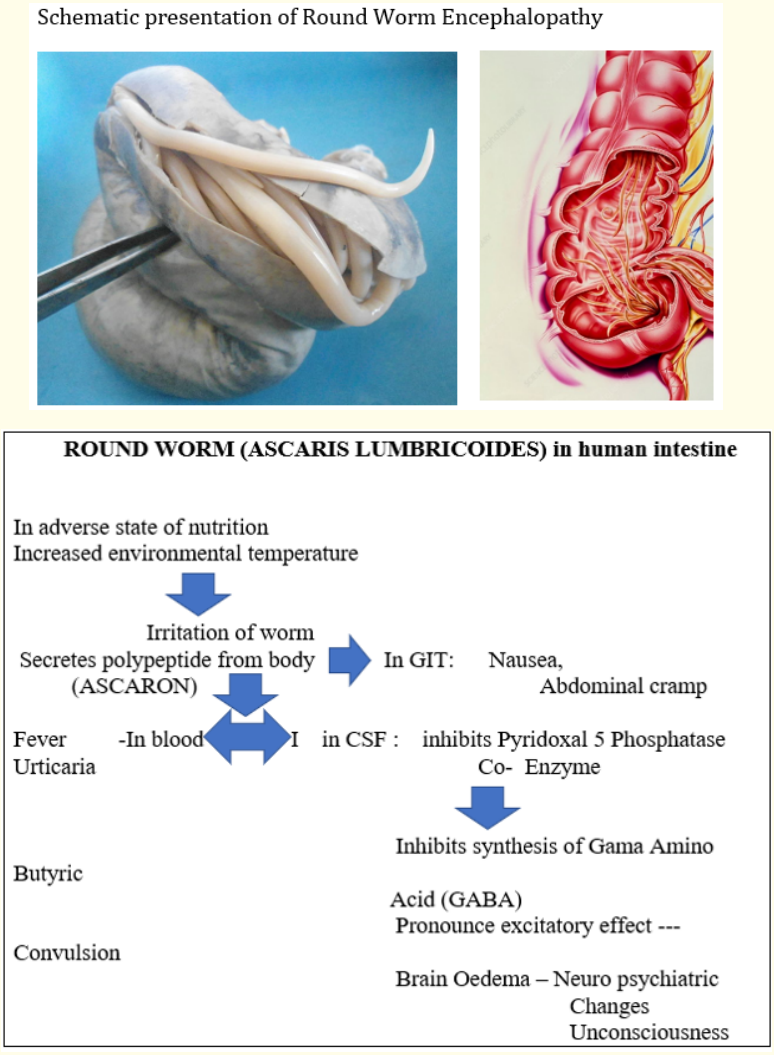

Figure 4: *Pathogenesis of Round worm encephalopathy.*

Therapeutic supremacy and achievement of clinic pathological cure can be explained as per follows [9-15] (Figure 5).

Schematic Presentation of the Administered Drug Effect

Oxygen inhalation: To ensure appropriate energy need of brain cells and check hypoxic degeneration.

IV Mannitol 10% with Glycerin 10%: Reduce brain oedema.

IV pyridoxin: Facilitate Pyridoxal 5 phosphatase a co enzyme. Responsible for formation of GABA from Glutamic Acid and its metabolism by activating enzyme Glutamate decarboxylase and GABA transaminase.

IV Sodium Valproate: To control seizure.

Through Ryle's tube

Antacid with Oxetacaine: Antacid solution coats intestinal mucosa checks toxin absorption, intestinal irritation Oxcetacain acting as local anaesthetic on round worm body calm the worm, check its irritation and secretion of polypeptide.

Neurovit Syr (Herbal neurogen): Revitalize damaged neural cells, energize the brain cells and preserve neural cells function.

Bland, simple, sweet

Liquid oral diet: Facilitate nutrition to child and round worm.

Antimicrobial therapy: To check super infection.

Pulmosafe Chest Application: To facilitate reabsorption of lung fluid and check respiratory infection

Cold sponging: To decrease body temperature and prevent neural cell integrity.

Figure 5: *Showing effect and plan of therapy.*

Conclusion

All cases of AES responded well to the regime with 100% survival without any untoward effects or sequel and proved to be due to Ascaris lumbricoides toxin, Thus AES in nutritionally deprived patients Round worm encephalopathy must be kept in mind.

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