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Original Article

Clinical Profile of Neem Oil Encephalopathy in Children

R Suresh[1], A Abubackar Siddiq[1], D Rajkumar[2], S Balasankar[3]

[1]Assistant Professor, Department of Paediatrics, Institute of Child Health and Research Centre, Madurai Medical College, Madurai, Tamil Nadu, India, [2]Associate Professor, Department of Paediatrics, Institute of Child Health and Research Centre, Madurai Medical College, Madurai, Tamil Nadu, India, [3]Professor, Department of Paediatrics, Institute of Child Health and Research Centre, Madurai Medical College, Madurai, Tamil Nadu, India

Abstract

Background: Neem oil commonly used as native medicine can cause toxic encephalopathy.

Objective: The objective of the study was to study the clinical and epidemiological features of children with neem oil poisoning.

Materials and Methods: This was a retrospective analysis of case records.

Results: Among the 88 cases, the mortality was 30.68%. In survived patients are cortical blindness (5.7%) and recurrent convulsion (2.3%), no sequelae were found in 61.4%.

Conclusion: Convulsion is the most common presentation of neem oil poisoning. Death is usually associated with prolonged convulsion and is usually within first 48 h.

Key words: Children, Encephalopathy, Neem oil, Status epilepticus

INTRODUCTION

Neem oil is used as a traditional medicine in South India for treatment of various ailments such as cough, cold, and diarrhea. Consumption of neem oil can, however, result in varied toxic systemic and neurological manifestations.[1] Neem oil is a vegetable oil pressed from the fruits and seeds of Neem (*Azadirachta indica*), an evergreen tree which is endemic to the Indian sub-continent. Neem oil is generally light to dark brown, bitter and has a rather strong odor that is said to combine the odors of peanut and garlic. It comprises mainly triglycerides and large amounts of triterpenoid compounds, which are responsible for the bitter taste. Neem oil also contains steroids (campesterol, beta-sitosterol, and stigmasterol) and a plethora of triterpenoids of which Azadirachtin[2] is the most well-known and studied. The Azadirachtin content of neem oil varies from 300 ppm to over 2000 ppm depending on the quality of the neem seeds crushed.

The socio cultural practices involved in child rearing, mostly irrational cause a great impact in childhood morbidity and mortality. One such harmful practice is the instillation of vegetable oil (usually gingili oil) in the child's nose and mouth as a cure for respiratory infections. This is a widespread custom in rural Tamil Nadu. Healthy children probably having a viral respiratory illness when subjected to this practice eventually develop more serious complications. When neem oil is used for the same custom, children present with encephalopathy and seizures.[1]

Aim of the Study

The aim of the study was to study the clinical and epidemiological features of children with neem oil poisoning.

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MATERIALS AND METHODS

Study Centre

The study was conducted in a tertiary care center in South Tamil Nadu.

Corresponding Author: A Abubackar Siddiq, 55/2E, Mohammedshapuram Fourth Street, Tirumangalam, Madurai - 625 706, Tamil Nadu, India.

Study Design

It is a retrospective descriptive study for 3 years by analyzing the case records.

Inclusion Criteria

All cases with history of neem oil ingestion and developing features of encephalopathy and other toxic features were included in the study.

Exclusion Criteria

Other known cause for convulsion such as neuroinfection and neurodegenerative disorder was excluded from the study.

Methodology

All children with history of neem oil ingestion were enrolled in the study as per the criteria specified. Following a brief history and rapid evaluation, the children were resuscitated and managed as per the designed protocol. Blood samples for the biochemical estimation of glucose, urea, creatinine, and bicarbonate were collected, as soon as an intravenous access was first established for management. Supportive therapy, such as inotropes and mechanical ventilation were provided, as and when required. The status epilepticus is managed as per the standard protocol used in our institution.

Analysis

Data will be entered in excel spread sheet and analyzed using simple descriptive statistics.

RESULTS

Total number of cases admitted with neem oil encephalopathy was 88. Among these 27 children succumbed to the illness. The mortality rate is 30.68%. Male:female ratio is 1.05:1. Age ranges from 36 days to 7 years mean value 1.193 standard error of mean 1.19 standard deviation 1.42 Q1 .25 Q3 1.5. Age-specific mortality rate ranges 1 month –1 year 26.67%; 1–2 years 30.77%; 2–3 years 66.67%; 3–4 years 25%; and 4–8 years 40%. The places distribution is as follows Madurai urban 21 (23.86%), rural 43 (48.86%), and neighboring districts 24 (27.3%). Patients presented with convulsion (73.3%), postictal state (22.19%), shock (3.1%), respiratory distress (1.5%), and route of administration-oral (100%). The amount of neem oil given ranges from 1 ml to 7.5 ml the mean value 3.99 standard error of mean 0.14 standard deviation 1.36 Q1 3 Q3 5. Patients were administrated neem oil for abdominal pain (34.1%), respiratory infection (29.5%), deworming (23.9%), unknown reason (6.8%), and convulsion (2.3%). The onset of convulsion ranges from 30 min to 5 h the mean value 2.1 standard error of mean 0.11 standard deviation 1.02 Q1 1.31 Q3 2.5. The total duration of convulsion ranges from 0.17 h to 8.3 h the mean value 2.56 standard error of mean 0.22 standard deviation 2.08 Q1 1 Q3 4.

The total duration of hospital stay [Table 1] ranges from 50 min to 21 days the mean value 4.3 days standard error of mean 0.38 standard deviation 3.58 Q1 2 Q3 6. The complication while undergoing treatment is as follows: Need of ventilatory support (6.8%), need of inotropic support (8.0%), need of ventilatory and inotropic support (4.5%), aspiration (2.3%), shock (0.2%), and no specific complication (78.4%). The sequelae [Figure 1] in survived patients are cortical blindness (5.7%), movement disorder and recurrent convulsion (2.3%) no sequelae was found in (61.4%) .The laboratory values was normal in (84.1%), low glucose was presented (5.7%), elevated urea creatinine (4.5%), elevated AST ALT (2.3%), and low bicarbonate (3.4%). EEG revealed background abnormalities in the form of diffuse slowing with paroxysmal abnormalities in the form of spikes and wave discharge in 85% (49 of 57). CT scan revealed diffuse cerebral edema in 36 (of 42). CSF analysis, done in ten patients, was normal. Autopsy done in 27 cases showed following findings-diffuse cerebral edema, brain is congested more over parieto-occipital area and petechiae hemorrhages seen. No significant changes are seen in liver and lung except mild congestion. However, these changes are nonspecific and can occur in any hypoxic

Table 1: Hospital stay and outcome

	Survived	Expired	Total
0–2 Days	9	23	32
2–5 Days	28	4	32
>5 Days	24	0	24
Total	61	27	88

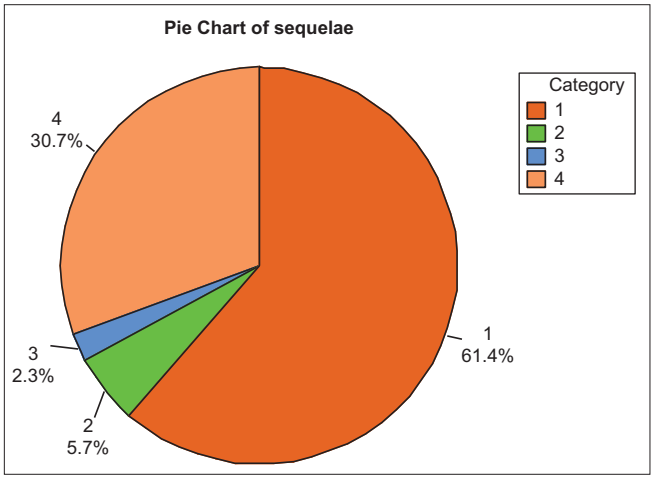

Figure 1: Sequelae. (1) Recovered without sequelae, (2) Cortical blindness, (3) Movement disorder and recurrent convulsion, (4) Expired

encephalopathy. There is no relation between gender and mortality, male expired 12 (26.66%) and female expired 15 (34.88%); $P = 0.102$ (insignificant). Duration of convulsion correlates with mortality [Table 2], but it is not statistically significant $P = 0.115$.

DISCUSSION

The incidence was decreased markedly in the 3rd year. However, this may be a transient phenomenon .In a study conducted by Lai *et al.*,[3] two children with neem oil poisoning were reported that the metabolic acidosis was not present in the present study. CSF analysis was normal as in the study and both children were recovered. In a study conducted by Sinniah *et al.*[4] most of the cases between the age group of 21 days and 4 years which is similar to this study. However, there is no recurrent vomiting and Reye syndrome was not found in this study. The mortality was 15% compare to 30.68% in this study. Eight (62%) required assisted ventilation as compare to in this study. The onset of convulsion is 30 min to 4.5 h as compare to in this study. In a study conducted in JIPMER should eight out of 218 cases were the cause of coma and the mortality was 48%. In the study by Sinnaiah *et al.* was showed that the extracted neem seed oil is toxic at least in children.

Child recovering from neem oil encephalopathy had only transient cortical blindness which recovered over 1–3 months. The severity varies from asymptomatic to death and it is probably genetic makeup of the child versus concentration of neem oil. The children are more prone for toxicity than adults, probably, and mechanism for control of seizure activity are fragile in younger children and may get disrupted with minimal abnormalities in neurofunction. Similarly, Kroczka *et al.*[5] report that SE appeared more frequently in children diagnosed with epilepsy during the first 2 years of life.

Prasad *et al.*[6] comments that longer a seizure lasts, the more difficult it becomes to control and that seizures can have immediate and long-term adverse consequences on immature and developing brain. The neem oil encephalopathy, unique to our area may be responsible for this slightly higher mortality.

Table 2: Duration of convulsion and outcome

	Survived	Expired	Total
0–2 h	44	5	49
2–4 h	14	10	24
>4 h	3	12	15
Total	61	27	88

Sagduyu *et al.*[7] report a lower case fatality rate of 21% in status epilepticus. However, the mortality rate reported in text books is around 5% in status epilepticus. Meta-analysis by Gilbert *et al.*[8] states that outcomes in India could be different for non-drug related or etiology related reasons. For example, slow transportation to the site of medical care could both increase mortality and decrease efficacy. Differences in intensive care unit practices also could alter mortality. Kroczka *et al.* add that the low frequency of SE in their study can be related to continuous access to pediatric neurologist and experienced nurse team.

CONCLUSIONS

1. Convulsion is the most common presentation of neem oil poisoning.
2. Death is usually associated with prolonged convulsion and is usually within first 48 h.
3. Neem oil encephalopathy has high mortality compared to other causes of status epilepticus.
4. Reye syndrome is not detected in this study.

Limitations

1. EEG could not be done in all cases and EEG monitoring of therapy of refractory SE was not done due to limited resources and lack of bedside EEG monitor.
2. CT brain was not done in some children due to the poor general condition of the patient, preventing transport to the CT-scan room.
3. Method of extraction of neem oil could not be elicited due to practical difficulties. Laboratory estimation of serum ammonia was not done.
4. Laboratory estimation of serum ammonia was not done.

Recommendations

1. Public awareness program should be initiated regarding neem toxicity in children especially in high-risk area.
2. Toxicological studies on neem oil should be made available in Regional forensic laboratory.
3. Large scale community based and (animal) experimental studies on neem oil is essential as it has socio-cultural background.

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