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The Risk Factors Associated with Adverse Outcome in Pediatric Convulsive Status Epilepticus in a Tertiary Care Hospital Kashmir India

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ABSTRACT

Objective: The study aimed to ascertain various risk factors associated with adverse outcomes of convulsive status epilepticus in children. **Methods**: Hospital based prospective observational study performed in department of Pediatrics in Children Hospital Government Medical College Srinagar Kashmir India from October 2020 to September 2022. Data were recorded with the help of a pre-formed proforma. **Results**: In this study 148 cases (85.1.0%) recovered, 26 (14.9%) died, and 10 (5.5%) left against medical advice. Among recovered, 10 cases (5.5%) developed new neurological sequelae. The various risk factors associated with significant mortality in this study were longer duration of status, acute symptomatic etiology, the requirement of early ventilatory support, impairment in hemodynamics, and alteration in metabolic environment of the patients. Our results were consistent with various other studies. **Conclusion**: Early termination of seizure activity at the earliest, and prudent management of associated co-morbidities like respiratory or circulatory impairment in these children would result in an improved outcome.

Keywords: Etiology, Outcomes, Convulsive Status Epilepticus, Children.

INTRODUCTION

Status epilepticus (**SE**) is a neurological emergency and is defined as seizure continuous in character for more than five minutes or multiple seizures occurring consecutively during which patients were unable to regain consciousness over a period of 30 minutes.[1] There are two broad categories of SE: convulsive status epilepticus (CSE) and non-convulsive status epilepticus (non-CSE). The identification of non-CSE from behavioral signs is difficult and electroencephalography (EEG) is often a crucial diagnostic tool.[2]

There are four phases of status epilepticus. They are classified based on duration as follows: [3-6]

1) Early phase or premonitory status: in which the convulsion continuesfor more than 5 minutes. At this stage, the first-line treatments (benzodiazepines) are used to control the seizure either prior to arrivalat the hospital, by the patient's parents or paramedics, or at a hospital in the emergency department.
2) Established status epilepticus: in which the seizure activity continuesfor more than 10 and up to 30 minutes with the loss of consciousness between seizures. In this stage second line treatments such as intravenous phenytoin, phenobarbital or levetiracetam are used to try and terminate it. This phase of status epilepticus may be termed benzodiazepine-resistant status epilepticus.
3) Refractory status epilepticus: in which the seizure activity (convulsion) lasts for more than 30 minutes or has failed at least one dose of the benzodiazepine and a dose of second-line intravenous AED, or both.

Non-CSE is not considered a medical emergency since patients do not loseconsciousness and usually return to normal within minutes of its resolution(either spontaneously or in response to treatment). In CSE, however, early institution of treatment is essential to avoid irreversible brain injury through both metabolic decompensation and respiratory depression which

further exacerbates the cerebral metabolic injury through anoxia, and subsequently, profound hypotension.[8]
The formal etiology of status epilepticus has been classified by the International League Against Epilepsy into five divisions: acute symptomatic, remote symptomatic, idiopathic epilepsy-related, cryptogenic epilepsy-related and unclassified.[9]

In 2006, the North London Convulsive Status Epilepticus in Childhood Surveillance Study (NLSTEPSS) classified the etiology into 7 groups: prolonged febrile seizure, acute symptomatic, remote symptomatic, remote with acute causes, idiopathic epilepsy related, cryptogenic epilepsy relatedand unclassified (Table II).[10]

Table I: Etiology of status epilepticus

Etiology	Definition	Causes
Prolonged febrileseizure	CSE that occurred in normal children who hadno history of central nervous system (CNS) infection and aged between 6 months and 5 years with a temperature at least 38.0C	Febrile seizure
Acute symptomatic	CSE that occurred in otherwise healthy children who had neurological insult withinthe past week	Meningitis Viral CNS Infection Head injury
		Hypoxia
Remote symptomatic	CSE reported in children who had a pre- existing CNS abnormality for more than 1week	Tuberous sclerosis Encephalopathy
Remote withacute causes	CSE that occurred in children within a week from febrile illness or acute neurological insult and associated with a history of previous neurological abnormalities	Cerebral palsy Hydrocephalus
Idiopathic epilepsy related	CSE that occurred in children who had a history of idiopathic epilepsy with no symptomatic causes for the seizure	Idiopathic epilepsy
Cryptogenic epilepsy related	CSE that occurred in children who had ahistory of cryptogenic epilepsy with no symptomatic causes for the seizure	Cryptogenic epilepsy
Unclassified	All other SE	------

The incidence of CSE in children ranges from 10 to 38 in 100,000 per year. [10-12]. The higher incidence of CSE is seen in children aged less than 4 years with a peak in first year of life.[13]

Children with a history of pre-existing epilepsy constitute the highest proportion of SE patients (10-20%). [14-15] Further febrile SE was most reported in children less than 2 years of age, while cryptogenic and symptomatic SE were more frequently documented in older children.[15]

AIMS AND OBJECTIVES
To study the clinical spectrum, etiology and outcome of status epilepticus in children aged between 1month to 18 years.

INCLUSION CRITERIA: All children aged between 1 month to 18 years who at presentation or during the hospital stay had convulsive status epilepticus - defined as continuous seizure activity or recurrent seizure activity without regaining consciousness lasting for >30 min.

EXCLUSION CRITERIA: Patients in whom the information regarding seizure duration was incomplete or unclear.
SAMPLE SIZE: Totally 184 cases whopresented with status epilepticus in our pediatric emergency during the study period were included in study.

METHOD
This was a hospital based prospective observational study performed in department of Pediatrics in Children Hospital Government Medical College Srinagar Kashmir India from October 2020 to September 2022. Ethical approval for this study was obtained from the hospital ethics committee. During this period a total number of 184 cases who were presented to the emergency with status epilepticus were included in study. All children aged between 1 month to 18 years who at presentation or during the hospital stay had convulsive status epilepticus - defined as continuous seizure activity for more than 5 minutes or recurrent seizure activity without regaining the consciousness in between.
Patients in whom the information regarding seizure duration was incomplete or unclear.

Procedure: Every sick child who was presenting to emergency department with
CSE had been assessed and triaged on arrival and a rapid cardiopulmonary assessment was made with immediate monitoring of heart rate, blood pressure, SPO2, signs of shock, pupil size and reaction to light. Before starting IV resuscitation, a blood

sample was taken for baseline investigations. Then the cases were managed according to the protocol followed in our emergency room/pediatric intensive care unit. After early management and stabilization of patient a detailed history was obtained including duration of seizure, distance fromthe hospital where the fits started, mode of transport, any prehospital and or treatment during transportation, precipitating factors, prior seizures/SE, drug history and compliance, any chronic medical or neurological illness, developmental milestones, and prior neurological status. Demographic and baseline data was recorded with the help of a preformed proforma. Variables included age, sex, type of status epilepticus, cause, duration of convulsions, duration of unconsciousness, precipitating factors, EEG, and number of anti-epileptic drugs required to control the seizures, history of convulsions and fever, any complications occurred and ultimate survival or death. Baseline investigations were also carried out including complete blood count, blood glucose levels, serum electrolytes, serum calcium and magnesium, blood urea and creatinine, urinalysis, lumbar puncture, MRI and EEG were performed.

Operational definition of status epilepticus used in this study was that a seizure continuous in character for more than five minutes or multiple seizures occurring consecutively during which patients were unable to regain consciousness over a period of 30 minutes.

Data thus collected was subjected to statistical analysis with the help of computer software SPSS version 23. Chi square test was applied and p value less than or equal to 0.005 was considered significant. The relationship of various demographic, clinical characteristics and etiology with outcome was evaluated employing the Chi-square test, Fischer's exact test for categorical data, and independent t-test for continuous data with normal distribution.

Outcome was determined by the following variables:

- Complete recovery without neurological sequelae.
- Recovery with neurological sequelae.
- Death

RESULTS

Table 1. FINAL OUTCOME

Outcome	No of cases N=184	Percentage
Recovered without sequelae	138	75.0
Recovered with sequelae	10	5.5
Death	26	14.0
LAMA	10	5.5
Total	184	100

In this study, out of 184 cases, 138 cases (75.0%) recovered without sequelae; 10 cases (5.5%) recovered with new neurological sequelae; 26cases (14%) died, and 10 cases (5.5%) left against medical advice.

ANALYSIS OF OUTCOME

Out of 184 cases enrolled in study, 10 left against medical advice and didn't complete the study to know the outcome. So, the outcome of the remaining 174 cases wereanalyzed here.

Table 2: AGE AND OUTCOME

Age	Recovered	Death	Total
<1yr	38 (86.4%)	6 (13.6%)	44 (100%)
1-5yrs	78 (85.7%)	13 (14.3%)	91 (100 %)
6-10yrs	44 (89.8%)	5 (10.2%)	49 (100%)
>10yrs	6 (75.0%)	2 (25.0%)	8 (100%)
Total	148 (85.1%)	26 (14.9%)	174 (100%)
Chi^2: 0.519 df: 3 p- Value: **0.915**			

The above table explains the outcome across the various age groups. Of thetotal 174 patients, 85.1% have recovered and 14.9% have died. Almost thesame proportion is maintained across all age groups. So, age is not havinga significant association with the outcome (p-Value> 0.05).

Table 3: DISTANCE TRAVELLED TO REACH PEDIATRIC HOSPITAL & OUTCOME

Distance KM	Recovered	Death	Total
<5	30 (93.8%)	2(6.3 %)	32 (100%)
5-10	36 (100%)	0 (0 %)	36 (100 %)
11-20	26 (81.3 %)	6 (18.8 %)	32 (100 %)
21-40	44 (88%)	6 (12%)	50 (100 %)
>40	12 (50 %)	12 (50%)	24 (100 %)
Total	148 (85.1 %)	26 (14.9 %)	174 (100%)
Chi^2: 16.070 df: 4	p- Value: **0.003**		

The above table explains the relationship between travelled distance to reach hospital and the outcome. The outcome is good among cases who travelled less than 10 km. Mortality was very low among children who travelled less than 10 km. Mortality was very high among children who had those 40 children who had travelled more than 40 km. Out of 24 cases, 12 cases (50%) recovered, and 12 cases (50%) died. It clearly depicts that smaller the travel distance or time interval to reach facility, better the outcome. The association is also statistically significant (p–Value < 0.05).

Table 4: PRE-REFERAL THERAPY

Prehospital therapy	Recovered	Death
Yes	34 (73.9 %)	12 (26.1 %)
No	114 (89.1%)	14 (10.9 %)
Total	148 (85.1%)	26 (14.9%)

The cases that had prehospital therapy had poor outcome (26.1%) than the cases who didn't receive prehospital therapy (10.9%). But this difference is not statistically significant (p- value > 0.05).

Table 5: DURATION OF CONVULSION BEFORE ARRIVAL

Duration of seizure Minutes	Recovered	Death
30-60	132(93.0%)	10(7.0 %)
>60	16(50.0 %)	16(50.0 %)
Total	148(85.1 %)	26(14.9 %)

The prolonged duration of the convulsions had poor outcome, statistically significant (p- value <0.05).

Table 6: FEVER ASSOCIATION AND OUTCOME

H/O Fever	Recovered	Death
Febrile	88 (85.4%)	15 (14.6%)
Afebrile	60 (84.5%)	11 (15.5%)
Total	148 (85.1%)	26 (14.9%)

The table depicts that 14.6% died among patients who had fever and CSE, 15.5% died among patients who had unprovoked CSE. The difference is not statistically significant (pvalue > 0.05).

Table 7: PREVIOUS NEUROLOGICAL STATUS

Neurologicalstatus	Recovered	Death
Normal	96 (90.6%)	10(9.4%)
Abnormal	52(76.5%)	16(23.5%)
Total	148(85.1%)	26(14.9%)

Among the previously neurological abnormal children (68), 23.5% (16) cases died, while only 9.4% (10) patients died who were previously neurologically normal (n=106). But this difference was not again statistically significant (p- value >0.05).

Table 8: COMORBID FACTORS

Risk factors	Recovered(n=148)	Death(n=26)	Total (n=174)	p value
Shock	12(8.11%)	4(15.38%)	16(9.2%)	0.000
Fever	88 (59.5%)	15 (57.7%)	103(59.2%)	0.766
Raised ICT	12 (8.10%)	0 (0%)	12(6.897%)	0.287
Intubation	22(14.86%)	16(69.54%)	38(21.84%)	0.000
SpO2 on arrival(low)	60(40.54%)	18(69.23%)	78(44.83%)	0.004
Hypoglycemia	10(6.76%)	8(30.76%)	18(10.34%)	0.015
Hypocalcemia	16(10.81%)	16(69.54%)	32(18.39%)	0.000
Acidosis	16(10.81%)	16(69.54%)	32(18.39%)	0.000
Delay in regaining consciousness after theseizure	44(29.73%)	4(15.38%)	48(27.58%)	0.942

The above table depicts various risk factors which were present at arrival significantly affected the outcome. Hypoxia at the time of arrival, decompensated shock, respiratory failure requiring intubation, hypoglycemia, hypocalcemia, and acidosis. (p-value <**0.05**).

Table 9: AED TREATMENT RESPONSE AND OUTCOME

AED Treated	Recovered	Death	Total
Diazepam 1	28(93.3%)	2(6.7%)	30(100%)
Diazepam 2	44(95.7%)	2(4.3%)	46(100%)
Phenytoin 1	36(85.7%)	6(14.3%)	42(100%)
Phenytoin 2	14(100%)	0(0%)	14(100%)
Phenobarbitone	12(66.7%)	6(33.3%)	18(100%)
Midazolam 1µgm/Kg/min	8(80%)	2(20%)	10(100%)
Midazolam 2µgm/Kg/min	2(100%)	0(0%)	2(100%)
Midazolam 3µgm/Kg/min	0(0%)	2(100%)	2(100%)
Midazolam 4µgm/Kg/min	4(40%)	6(60%)	10(100%)
Total	148(85.1%)	26(14.9%)	174(100%)
Chi^2: 20.427 df: 8 p- Value: **0.009**			

If the seizure control is poor with the initial first line AED, mortality is very high. In children receiving Midazolam infusion (3-4µgm/Kg/min) had poor outcome. This is also statistically significant p- Value

Table 10: ETIOLOGY AND OUTCOME

Final diagnosis	Recovered	Death	Total Percentage
Febrile seizure	34 (100%)	0(0%)	34(100%)
Acute CNS infection	26(76.5%)	8(23.5%)	34(100%)
Remote causes	38(76%)	12(24%)	50(100%)
Idiopathic SD	24(92.3%)	2(7.7%)	26(100%)
Neurocutaneoussyndrome	04(100%)	0(0%)	4(100%)
IE metabolism	02(50%)	02(50%)	4(100%)
Non compliance	08(80%)	02(20%)	10(100%)
CNS haemorrhage	04(100%)	0(0%)	4(100%)
Systemic illness	06(100%)	0(0%)	6(100%)
Tumour	02(100%)	0(0%)	2(100%)
	148(85.1%)	26(14.9%)	174(100%)

In this study, out of 184 cases, 174 children completed the study. 148 casesrecovered and 26 cases died due to varied etiology. All the febrile seizure cases presented as SE had good outcome. Remote causes, Acute CNS infection, IEM, and non-compliance of AED are the etiological factors thatinfluence the poor outcome

Table 11: RECOVERED WITH SEQUELAE AND OUTCOME

Etiology	Total cases	Recovered withsequelae
Acute CNS infection	36	4 (11.11%)
CNS hemorrhage	4	2 (50%)
Idiopathic SD	30	2 (6.66%)
Remote cause	50	2 (4.0%)
Total	120	10 cases

Among 174 cases, 10 cases developed neurological sequelae. The most common etiological agents associated with sequelae are acute CNS infection, CNS hemorrhage, Idiopathic SD, and remote cause.

DISCUSSION

In this study, 184 cases of convulsive status epilepticus were included in the prescribed study period. The mean age of the patients in the present study was 4.9 years. The youngest age was 2 months and the eldest was 15 years. Children < 1-year account for 23.9 % of the total cases (n=184) and 67.4% were children < 5 years. The number of cases >10yrs was only 6.5%. Out of 184 children, male children were 102 (55.4%) and female childrenwere 82 (44.6%).

Out of 184 cases, 42.4 % cases were referred from the rural health care system and 57.6% came directly from their respective homes. Out of 184 children, 32 cases had travelled distance of less than 5 km; 44 cases 5-10 km; 32 cases 10-20 km; 50 cases 20-40 km, and 26 cases more than 40 km to reach hospital.

In our study around 48 patients (26.1%) had received pre-referral treatment at rural health care settings and the remaining 136 cases (73.9%) were referred without any pre-referral treatment for convulsive status epilepticus.

In this study 148 cases (85.1.0%) recovered, 26(14.9%) died, and 10 (5.5%) left against medical advice. Among recovered, 10 cases (5.5%) developed new neurological sequelae. The mortality rate in children with CSE ranged from 14- 33% in Indian studies, while studies from developed countries report mortality of 9-11%. [15-18] The various risk factors associated with significant mortality in this study were longer duration of status, acute symptomatic etiology, the requirement of early ventilatory support, impairment in hemodynamics, and alteration in metabolic environment of the patients. Our results were consistent with various other studies. [19,20] In our study children who had previous neurological and developmental issues had a poor outcome and results were consistent with, and Thandavarayan et al. [21] and Kwong et al. [22]

The outcome was good among cases who had traveled less than 10 km. There was 50% mortality among those 24 cases, who were brought from more than 40 km distance from our hospital. The study showed that the lower the travelling distance from the pediatric critical care center, the better is outcome of the patients. The association is also statistically significant (p-Value < 0.05). K. Eriksson, et al.'s [23] study also revealed the significant association between treatment delay and outcome of patient.

The prolonged duration of the seizure before arrival at ER had a poor outcome. This is also statistically significant (p-value <0.05). **Gulati S etal, Karla Veena et al. [76]** in their study have also found that a seizure duration of more than 45 minutes is significantly associated with higher mortality. **KL Kwong et al.'s** [21] study also supports that a seizure duration of more than 60 minutes had an adverse outcome.

The other risk factors which significantly affected the outcome were hypoxia at the time of arrival, decompensated shock, respiratory failure requiring intubation, and acidosis. (p-value <0.05). **Kalra Veena et al**.'s [15] in their study also showed the presence of shock (p-0.001) was associated with significant mortality.

Among 148 cases that survived, 10 cases (6.7%) developed neurological sequelae; (Aphasia-4, Hemiparesis-2, Cognitive dysfunction-4). Among them 6 cases were less than 1year and 4 cases were more than 10 years. The children who had developed neurological sequelae, 8 had severe metabolic acidosis on admission, 2 were in shock. The study conducted by **Maytal J et al.** [24] showed that newneurologic deficits were found in 17 (9.1%) of the 186 survivors. Status epilepticus in children < 1 year accounted for 23.9 % of the cas Almost 68% of them were in the group < 5 years. The mean age of the patient in the present study was 4.9 yrs. The study further revealed that the 14.9 % mortality in convulsive status epilepticus were associated with some risk factors like prolonged duration of seizures, long distance from treating hospital and poor response to initial first-line AED. The outcome was also poor in patients who required early ventilation, Midazolam infusion (3-4µgm/Kg/min), were hypoxic at the time of admission to hospital.

CONCLUSION

Status epilepticus in children is associated with significant mortality and morbidity. Longer duration of convulsions increases the risk of respiratory failure, shock, and deranged metabolic environment in patients which in turn increase morbidity and mortality in CSE. Therefore, early termination of seizure activity at the earliest, and prudent management of associated co-morbidities like respiratory or circulatory impairment in these children would result in an improved outcome. The findings from this study suggest new directions for research. Research should be done to determine which interventions may lead to improved outcomes. Right treatment of CSE, at right time with right drugs invariably demands upgradation of rural health care delivery system and proper transportation of such patients to the nearest pediatric intensive care facility. Our results may also carry implications for improvement of patient care. Early identification and treatment of some of these aggravating factors may not only prevent immediate mortality, but it may also reduce long-term complications and limit neurological dysfunction. Beyond these implications, the development of treatment paradigms may also help reduce variability in care and outcomes and thereby decrease hospital costs.

Limitations: First, it is a hospital-based single-center study.
Second, the variation in SE management protocols in different centersmay influence results in those centers, and caution should be taken when generalizing single-center study results.

REFERENCES

1. Al-Mufti, F; Claassen, J (Oct 2014). "Neurocritical Care: Status Epilepticus Review". Critical Care Clinics. 30 (4): 751–764.
2. Walker M, et al., Nonconvulsive status epilepticus: Epilepsy Research Foundation workshop reports. Epileptic Disorders, 2005. **7**(3): p. 253-296.
3. Clark P and Prout T, Status epilepticus: a clinical and pathological study in epilepsy [part 1]. American Journal of Psychiatry, 1903. **60**(2): p. 291-306.
4. Clark P and Prout T, Status epilepticus: a clinical and pathological study in epilepsy [part 2]. American Journal of Psychiatry, 1904 **60**(4): p. 645-698-7.
5. Clark P and Prout T, Status epilepticus: A clinical and pathological study in epilepsy [part 3]. American Journal of Psychiatry, 1904. 61(1): p. 81-108-3.
6. Shorvon S and Ferlisi M. The treatment of super-refractory status epilepticus: a critical review of available therapies and a clinical treatment protocol. Brain, 2011. 134 (part 10): p. 2802-18.
7. Applton R and Anthony M, Epilepsy: The Facts. 3rd Edition. 2009: Oxford University Press.ILAE, Proposal for revised classification of epilepsies and epileptic syndromes. Commission on Classification and Terminology of the International League Against Epilepsy. Epilepsia, 1989. 30(4): p. 389- 99.
8. Chin R, et al., Incidence, cause, and short-term outcome of convulsive status epilepticus in childhood: prospective population- based study. TheLancet, 2006. 368(9531): p. 222-229.
9. Coeytaux, A., et al., Incidence of status epilepticus in French- speakingSwitzerland (EPISTAR). Neurology, 2000. 55(5): p. 693-697
10. Hesdorffer, D., et al., Incidence of status epilepticus in Rochester, Minnesota, 1965-1984. Neurology, 1998. 50(3): p. 735-741.
11. Govoni V, et al., Incidence of status epilepticus in southern Europe: apopulation study in the health district of Ferrara, Italy. European neurology, 2008. 59(3-4): p. 120-126.
12. Sillanpää M and Shinnar S, Status epilepticus in a population-based cohort with childhood-onset epilepsy in Finland. Annals of neurology, 2002. 52(3): p. 303-310.
13. Berg A, et al., Status epilepticus after the initial diagnosis of epilepsy in children. Neurology, 2004. 63(6): p. 1027-1034.
14. Shinnar S, et al., In Whom Does Status Epilepticus Occur: Age- Related Differences in Children. Epilepsia, 1997. 38(8): p. 907-914.
15. Gulati S, Kalra V, Sridhar MR. Status epilepticus in Indian children in a tertiary care center. Indian J Pediatr. 2005 Feb;72(2):105-8.
16. Kumar M, Kumari R, Narain NP. Clinical Profile of Status epilepticus(SE) in Children in a Tertiary Care Hospital in Bihar. J Clin Diagn Res: JCDR. 2014;8(7):14-7
17. Murthy JM, Jayalaxmi SS, Kanikannan MA. Convulsive status epilepticus: clinical profile in a developing country. Epileps. 2007;48(12):2217-23
18. Thandavarayan M, Ramaswamy S, Bose P, Thirumalaikumarasamy S. Immediate outcome and risk factors determining the outcome of status epilepticus in children attending tertiary care centre. Int JContemp Pediat. 2017;4(4):1289-95
19. Murthy JM, Jayalaxmi SS, Kanikannan MA. Convulsive status epilepticus: clinical profile in a developing country. Epileps. 2007;48(12):2217-23
20. Das NK, Soren B, Gupta D. Clinical Profile, Aetiology, and Short- Term Outcome of Convulsive Status Epilepticus in Children in Eastern India.JMSCR.2017; 5(1):15914
21. Thandavarayan M, Ramaswamy S, Bose P, Thirumalaikumarasamy S. Immediate outcome and risk factors

determining the outcome of status epilepticus in children attending tertiary care centre. Int J Contemp Pediat. 2017;4(4):1289-95
22. Kwong KL, Lee SL, Yung A, Wong VC. Status epilepticus in 37 Chinese children: aetiology and outcome. J Paediatr Child Health. 1995;31(5):395-8.
23. K. Eriksson, Treatment delay and the risk of prolonged status epilepticus Lancet. 2006 Jul 15;368(9531):222-9.
24. Maytal J,Shinner S, Moshe SL, Alvarez LA, Low morbidity and mortality of SE inchildren, Pediatrics, 1989 Mar;83(3):323-31.

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