DOI: 10.7860/JCDR/2025/73308.20975

Original Article

Paediatrics Section

Assessment of Blood pH and pCO_2 as Predictors of Febrile Seizures in Paediatric Patients: A Cross-sectional Study

SUHIT BANERJEE[1], TAPAN KUMAR BISWAS[2], GOBINDA MONDAL[3], BANASREE ROY[4], ANISH CHATTERJEE[5]

ABSTRACT

Introduction: Febrile seizures commonly occur in the age group of six months to five years. Hypocarbia and alkalosis are known to enhance neuronal excitability and promote epileptiform activity. Neuronal excitability and seizure activity are strongly suppressed by various manoeuvre that lead to a decrease in brain pH, including exposure to elevated Carbon Dioxide (CO_2) levels, which results in hypercapnia.

Aim: To study blood pH and partial pressure of Carbon Dioxide (pCO_2) levels in febrile children and to correlate these parameters with the occurrence of febrile seizures.

Materials and Methods: This cross-sectional study was conducted in the Department of Paediatrics at Dr. BC Roy Postgraduate Institute of Paediatric Sciences, Kolkata, West Bengal, India, from January 2019 to December 2019. Infants and children aged six months to five years with clinical features compatible with the diagnosis of febrile seizures were enrolled as cases, while children with fever but without convulsions were included as controls. A total of 50 children were selected as cases and 50 as controls. Venous blood gas analysis was performed within one hour and 24 hours after the onset of seizures in cases and one hour after the febrile episode in controls. Data were statistically analysed using t-tests, one-way analysis of variance, Chi-square tests, or Fisher's exact test. A p-value ≤0.05 was considered statistically significant.

Results: The mean age of the febrile seizure cases was 24±16 months, while that of the controls was 31±23 months. Of the cases, 40 (80%) were male and 33 (66%) of the controls were male. The blood pH of cases within one hour of the febrile seizure was (mean±SD) 7.43±0.03 (indicative of respiratory alkalosis) and 24 hours after the febrile seizure, it was 7.41±0.05, showing a significant difference (p=0.017). The blood pCO_2 of cases measured within one hour of the febrile seizure was (mean±SD) 30±5 (indicative of hypocapnia), while the pCO_2 of controls measured one hour after the febrile episode was 36.5±6.5 mmHg. A significant difference (p<0.0001) was found between these two pCO_2 values.

Conclusion: Respiratory alkalosis and hypocapnia are associated with febrile seizures. Medical carbogen may be used as treatment or prophylaxis for febrile seizures; however, further studies are required to establish this.

Keywords: Convulsion, Fever, Hypocarbia, Respiratory alkalosis

INTRODUCTION

Febrile seizures are one of the most common phenomena encountered in paediatric emergencies. A febrile seizure is defined as a seizure that occurs in children between the ages of 6 and 60 months (with a peak incidence at 12-18 months) and is associated with a temperature of 38°C (100.4°F) or higher. These seizures are not due to a central nervous system infection or any metabolic imbalance and there is an absence of a history of afebrile seizures in the past [1].

It is documented that 2% to 5% of children under the age of 5 experience febrile seizures [2], with a peak incidence occurring at 12-18 months [3]. The rate of recurrence of febrile seizures has been reported to range from 20% to 50% [4-6]. They are classified as "simple febrile seizures" if they are brief (lasting less than 15 minutes) with no lateralising features, or as "complex febrile seizures" if they are prolonged (lasting more than 15 minutes), have focal features, or recur within 24 hours [5]. Although most febrile seizures are benign, one-third of them are classified as 'complex' [3,7].

It is known that changes in pH and hypocarbia play a central role in the control of seizure activity in the brain. Alkalosis and hypocarbia are known to enhance neuronal excitability and promote epileptiform activity, both in-vitro and in-vivo [8]. Hyperventilation, which leads to a net loss of CO_2 and consequent respiratory alkalosis, is a standard method to provoke absence seizures, complex partial seizures and other epileptiform manifestations in human patients. The hyperventilation-induced Electroencephalography (EEG) changes are more pronounced in children than in adults [9]. Various procedures that decrease brain pH levels can suppress neuronal excitability and thus reduce seizure activity [10].

There are only a few studies related to hypocapnia and its association with febrile seizures and most of them have been conducted outside India [11-14]. A study by Kilicaslan B et al., involving 36 children in Turkey, with 18 patients experiencing febrile convulsions as cases and 18 fever patients as a control group, revealed significantly lower pCO_2 levels in the febrile seizure patients compared to the control group based on venous blood sampling taken one hour later; however, no significant difference in pH was observed [11]. An Egyptian study by Marzouk H, which included 43 children with 22 febrile seizures and 21 controls, showed a significant difference in mean blood pH and pCO_2 levels [12]. Another study from Iran by Arshi S et al., involving 44 children with febrile convulsions and 39 controls, found significant hypocapnia in the febrile seizure group compared to the controls [13].

Only one Indian study was identified after an extensive literature search, conducted by Sachan D and Goyal S, involving 45 children. In this study, venous blood gas pH and pCO_2 values were measured within two hours and after two hours of the seizure episode. A total of 91% of children exhibited hypocapnia after a febrile seizure, which was significant; however, no significant difference in pH values was noted [15]. The lack of data from this eastern part of India inspired us to plan the present study.

With this background, the present study was designed to investigate blood pH and pCO_2 levels in febrile children and to correlate these parameters with the occurrence of febrile seizures.

MATERIALS AND METHODS

The present cross-sectional study was conducted in the Department of Paediatrics at Dr. BC Roy Postgraduate Institute of Paediatric Sciences, a tertiary care paediatric hospital in Kolkata, West Bengal, India, from January to December 2019, lasting for a period of one year. The study was initiated after obtaining informed, written consent from the parents and ethical clearance from the Institutional Ethics Committee (No. BCH/ME/PR/-3734).

Inclusion criteria: Infants and children from all backgrounds, irrespective of sex, religion, residence, or socio-economic status, aged six months to five years with clinical features compatible with the diagnosis of febrile seizures [1], were enrolled as cases. These children were admitted to the study Institute. Febrile seizures were diagnosed if a child exhibited fever (temperature >37.8°C) not caused by meningitis, encephalitis, or any other condition affecting the brain and were otherwise neurologically healthy, without any neurological abnormalities identified through examination or developmental history [1]. Additionally, children aged six months to five years with fever but without convulsions were included in the study as controls.

Exclusion criteria: Children with seizures occurring in the context of fever caused by meningitis, encephalitis, or any other illness affecting the brain, as well as those presenting more than one hour after the seizure episode, were excluded from the cases. Children with fever and respiratory distress and/or gastroenteritis were excluded from the controls.

Sample size calculation: Sample size was calculated according to the following formula:

$n=z^2\ p\ (1-p)/c^2$ and it was 50.

n=required sample size, z=confidence level at 95% (standard value of 1.96)

p=estimated prevalence of disease 4.2 [16], c=margin of error at 5%.

Estimated sample size 63

50 cases and 50 controls were taken for the study.

Study Procedure

All cases were evaluated clinically. Information regarding demographic data, including age, sex, religion and type of seizure, was collected and examinations were conducted to identify cases of febrile seizures. For the present study, the following measures were undertaken:

1. Measurement of axillary temperature using a digital thermometer, placed in the axilla for one minute.
2. Venous blood gas analysis [12,13] was performed within one hour and 24 hours after the onset of seizures in cases and one hour after the febrile episode in controls.

For venous blood gas analysis, a 0.2 mL blood sample was collected from a peripheral vein using a heparinised syringe, which was then analysed using the OPTI Clinical Chemistry Analyser-Test System (OPTI CCA-TS) blood gas analyser. All aseptic precautions were taken during peripheral venous blood collection. The results were compared with standard values: venous blood pH of 7.31-7.41 and pCO_2 of 40-50 mm Hg [14].

STATISTICAL ANALYSIS

For statistical analysis, data were entered into a Microsoft Excel spreadsheet and subsequently analysed using Statistical Package for the Social Sciences (SPSS) (version 25.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Data were summarised as mean and standard deviation for numerical variables and count and percentages for categorical variables. Two-sample t-tests were used to assess the difference in means between independent samples or unpaired samples. One-way analysis of variance (one-way ANOVA) was employed to compare the means of three or more samples for numerical data (using the F distribution). Unpaired proportions were compared using the Chi-square test or Fisher's exact test, as appropriate.

Once a t-value is determined, a p-value can be derived using a table of values from the Student's t-distribution. A p-value of ≤0.05 was considered statistically significant.

RESULTS

In the present study, 50 children were designated as cases and 50 as controls. The mean age of the febrile seizure cases was 24±16 months, while for the controls it was 31±23 months. There was no statistically significant difference in age between the cases and controls. Among the 50 cases, 40 (80%) were males and 10 (20%) were females. In the control group, 33 (66%) were males and 17 (34%) were females. No significant differences (p=0.115) were found between cases and controls regarding sex. Among the cases, 14 were Muslim and the remainder were Hindu. In the control group, 11 were Muslim and 39 were Hindu. The mean temperature of the febrile seizure patients was 39.3±1.2°C, while for the controls it was 39.6±1.5°C. No statistically significant difference (p=0.272) was found between cases and controls regarding mean temperature [Table/Fig-1].

Variables		Cases (n=50)	Controls (n=50)	p-value
Age (months) (Mean±SD)		24±16	31±23	0.080
Gender	Male	40 (80%)	33 (66%)	0.115
	Female	10 (20%)	17 (34%)	
Religion	Hindu	36	39	0.490
	Muslim	14	11	
Temperature (°C)		39.3±1.2	39.6±1.5	0.272

[Table/Fig-1]: Distribution of age, sex, religion and temperature in participants.

Among the 50 cases, 49 (98%) children experienced Generalised Tonic-Clonic Seizures (GTCS) and 1 (2%) child had focal seizures. Additionally, 11 (22%) patients had prolonged seizure episodes (lasting more than 15 minutes), while the remaining 39 (78%) had seizures lasting less than 15 minutes. A total of 16 (32%) patients had a past history of febrile seizures. A total of 5 (10%) patients reported a history of febrile seizures in their parents, 3 (6%) of them were preterm and no mothers reported any comorbidities during pregnancy [Table/Fig-2]. Patients with active convulsions were managed with benzodiazepines.

Variables		n (%)
Type of seizure	GTCS	49 (98)
	Focal seizures	1 (2)
Duration >15 minutes <15 minutes		11 (22)
		39 (78)
Past history of febrile seizure		16 (32)
History of febrile Seizures in parents		5 (10)
Preterm		3 (6)

[Table/Fig-2]: Data from participants' clinical histories.

The blood pH of cases within one hour of the febrile seizure was 7.43±0.03 and 24 hours after the seizure, it was 7.41±0.05. A significant difference (p=0.017) was observed between the pH measured within one hour of the febrile seizure and the pH measured 24 hours later. The blood pH of cases within one hour of the febrile seizure was 7.43±0.03, while the pH of controls measured one hour after the febrile episode was 7.4±0.05. The difference (p=0.09) between these two pH values was not significant [Table/Fig-3].

Variables	Cases (Mean±SD)	Controls (Mean±SD)	p-value
pH (1 h)	7.43±0.03	7.4±0.05	0.9
pH (24 h)	7.41±0.05	7.4±0.05	0.3198
p-value	0.017	-	
pCO_2 (1 h)	30±5	36.5±6.5	<0.0001
pCO_2 (24 h)	34±5	36.5±6.5	0.0484
p-value	0.0001	-	

[Table/Fig-3]: Comparison between pH and pCO_2 of cases and controls within one hour and 24 hours. Unpaired t-test was applied to calculate the p-value.

The pCO_2 value of cases measured within one hour of the febrile seizure was 30±5 mmHg and after 24 hours, it was 34±5 mmHg. A significant difference (p=0.0001) was found between these two pCO_2 values. The blood pCO_2 of cases measured within one hour of the febrile seizure was 30±5 mmHg, while the pCO_2 of controls measured one hour after the febrile episode was 36.5±6.5 mmHg. A significant difference (p<0.0001) was found between these two pCO_2 values. There was also a significant difference (p=0.0484) between the pCO_2 of cases (34±5 mmHg), measured 24 hours after the febrile seizure and the pCO_2 of controls (36.5±6.5 mmHg), measured 24 hours after the febrile episode [Table/Fig-3].

DISCUSSION

Febrile seizures are the most common cause of seizures among children. The exact pathogenesis is unknown but is thought to involve factors such as genetic predisposition and various alterations [17]. In the present study, the authors enrolled 50 cases of febrile seizures and 50 controls (fever without convulsions). They measured pH and pCO_2 in the cases within one hour and after 24 hours of the febrile seizure and in the controls one hour after the febrile episode.

In the present study, the mean age of the febrile seizure cases was 24±16 months, while for the controls it was 31±23 months. There was no statistically significant difference (p>0.05) between the cases and controls regarding age. Kilicaslan B et al., conducted a study where the mean age of patients with febrile seizures was 20.8±7 months [11]. In another study by Namakin K et al., the mean age of febrile seizure cases was reported as 24.1±13.4 months [17]. These findings are consistent with those of the present study.

In the present research, among the 50 cases, 40 (80%) were male and 10 (20%) were female. Among the 50 controls, 33 (66%) were male and 17 (34%) were female. There were no significant differences (p=0.115) between the cases and controls regarding sex. A study by Nickavar A et al., enrolled 70 male and 55 female patients with febrile seizures [18]. Usha KC and Suresh R enrolled 35 male and 25 female febrile seizure patients in their study [19]. These studies also indicate a male predisposition for febrile seizures, similar to the present findings.

The mean temperature of patients with febrile seizures was 39.3±1.2°C, while that of the controls was 39.6±1.5°C. No statistically significant difference (p=0.272) was found between the cases and controls regarding mean temperature. Similarly, Marzouk H showed that there was no significant difference between the febrile seizure cases and the controls concerning mean temperature (p=0.3) [12]. Kilicaslan B et al., also found that the difference between the mean temperature of febrile seizure cases and controls was not significant. The present study findings corroborate those of this study [11].

In the present study, the mean blood pH of cases measured within one hour of the febrile seizure was 7.43±0.03 and after 24 hours, it was measured at 7.41±0.05. There was a significant difference (p=0.017) between these two pH values. There was a significant difference (p=0.004) between the pH of cases measured within one hour of the febrile seizure (7.43±0.03) and the pH of controls taken one hour after the febrile episode (7.4±0.05). However, no significant difference (p=0.319) was found between the pH of cases measured 24 hours after the febrile seizure (7.41±0.05) and the pH of controls (7.4±0.05). Marzouk H found a significant difference (p<0.001) between the pH of cases measured within one hour of the febrile seizure (7.47±0.06) and the pH of controls measured one hour after a febrile episode (7.37±0.03) [12]. This result aligns with the present findings. Schuchmann S et al., conducted a study where they observed that respiratory alkalosis (pH 7.46±0.04) occurred in children with febrile seizures, with pH measured immediately after admission [20]. Respiratory alkalosis was also detected in the present study, measured one hour after the febrile seizure episode. Arshi S et al., conducted a study that reported a statistically significant difference between the pH of febrile seizure patients (pH=7.41) and the pH of patients with simple fever (pH=7.39) [13]. The comparison of these two groups yielded p=0.011. The present study also produced similar results.

Sachan D and Goyal S found that there was no statistical significance between the pH in samples drawn within two hours of the seizure (7.43±0.05) and the pH in samples drawn two hours after the seizure (7.41±0.04) [15]. They did not find any statistical significance (p=0.209) between these two pH values, perhaps because they measured the second pH value too early. Kilicaslan B et al., conducted a study which reported no statistical significance (p=0.29) between the pH of cases (7.39±0.05) measured 24 hours after the febrile seizure and the pH of controls (7.37±0.05) [11]. The present study findings (p=0.31) corroborate these results.

In the present study, the mean blood pCO_2 of cases measured within one hour of the febrile seizure was 30±5 mmHg, while the pCO_2 measured after 24 hours of the febrile seizure was 34±5 mmHg. A significant difference (p<0.001) was observed between these two pCO_2 values. There was also a significant difference (p<0.001) between the pCO_2 of cases measured within one hour of the febrile seizure (30±5 mmHg) and the pCO_2 of controls taken one hour after the febrile episode (36.5±6.5 mmHg). A statistically significant difference (p=0.034) was found between the pCO_2 of cases measured 24 hours after the febrile seizure (34±5 mmHg) and the pCO_2 of controls (36.5±6.5 mmHg).

Marzouk H found a significant difference (p<0.001) between the pCO_2 of cases measured within one hour of the febrile seizure (29.89±2.98 mmHg) and the pCO_2 of controls measured one hour after a febrile period (37.98±3.9 mmHg) [12]. The present study corroborates this finding. Schuchmann S et al., conducted a study where they found hypocapnia (pCO_2 29.5±5.5 mmHg) in children with febrile seizures; the pCO_2 was measured immediately after admission. Hypocapnia in venous blood was also observed in the present study, measured one hour after the febrile seizure [20].

Arshi S et al., conducted a study that reported a statistically significant difference between the pCO_2 of febrile seizure patients (pCO_2=28.4 mmHg) and the pCO_2 of patients with simple fever (pCO_2=33.4 mmHg) [13]. The comparison of these two groups yielded p=0.011. The present study also produced comparable results. Sachan D and Goyal S found a statistically significant difference (p=0.001) between the pCO_2 in samples drawn within two hours of the seizure (23.24±3.44 mmHg) and the pCO_2 in samples drawn two hours after the seizure (29.29±4.99 mmHg). The present study results are related to the findings of this study [15].

Kilicaslan B et al., conducted a study in which there was a statistical significance (p=0.006) between the pCO_2 of cases (29.28±7.3 mmHg) measured one hour after the febrile seizure and the pCO_2 of controls (37.28±8.98 mmHg) [11]. The present study findings (p<0.001) corroborate this result. A comparison of the findings from the present study with contrasting studies is shown in [Table/Fig-4] [11-15,20].

Author (Year of publication)	Place/year of study	Sample size	Mean age (in months)	Comparison of pH between cases and controls at 1 hour/ after admission	Comparison of pH between cases and controls at 24 hours	Comparison pCO_2 between cases and controls just after seizure	Comparison pCO_2 between cases and controls after >2-24 h	Findings
Kilicaslan B. et al., (2014) [11]	Turkey October 2012-January 2013	18 case, 18 control	20.8±7.0 30.4±19.8	7.40±0.06 7.37±0.05	7.39±0.05	29.28±7.30 37.28±8.98	33.33±8.84	Blood pCO_2 was significantly lower in the febrile seizure group at 1 hr
Marzouk H [12] (2015)	Egypt June 2013-October 2014	22 case, 21 control	19.3±11.7 20.3±13.1	7.47±0.06 7.37±0.03	7.42±0.04	29.89±2.98 37.98±3.90	36.01±3.41	Hypocapnia associated with febrile seizures
Arshi S et al., [13] (2019)	Iran 2013-2015	44 case, 39 control		7.41 (mean)	7.39 (mean)	28.4 (mean)	33.4 (mean)	Hypocapnia is associated with febrile seizure
Abd El-Moneim HM et al., [14] (2020)	Egypt	50 cases, 50 contols	2.16±0.85 years 2.12±0.82 (years)	Case- 38% alkalosis Control- 0 Alkalosis		Case- 74% hypocapnia Control- 12% hypocapnia		Hypocapnea is significantly associated with febrile seizures
Sachan D and Goyal S (2018) [15]	India November 2014-March 2016	45 cases	22.87±12.98	7.43±0.05	7.41±0.04	23.24±3.44	29.29±4.99	pCO_2 values <2 hr is significantly lower than >2 hr
Schuchmann S et al., [20] (2011)	Germany 2005-August 2007	213 Febrile seizure cases, 220 Gastroenteritis patients	6.0-58.9 9.0-59.7	Case 7.46±0.04 GE' 7.44±0.04		Case 29.5±5.5 GE' 26.3±5.9		Respiratory alkalosis with increased pH, reduced pCO_2, in febrile seizure group
Present study	India January-December 2019	50 cases, 50 controls	24±16 m 31±23 m	7.43±0.03 7.4±0.05	7.41±0.05	30±5 36.5±6.5	34±5	Hypocapnia and alkalosis are associated with febrile seizure group

[Table/Fig-4]: Findings of previous studies in comparison to the present study [11-15,20].

Limitation(s)

The study was conducted in only one tertiary care hospital, making it a single-centre study; therefore, hospital bias cannot be ruled out.

CONCLUSION(S)

Respiratory alkalosis followed by hypocapnia may be a mechanism contributing to the occurrence of febrile seizures. Hypocapnia is a probable precipitating factor for febrile seizures, as demonstrated in the present study. Effective and early control of fever is one of the most important methods to prevent febrile seizures by decreasing tachypnoea, thus reducing CO_2 washout (hypocarbia). Measures to reduce hypocapnia could be a useful treatment approach for febrile seizures; however, this requires detailed multicentre studies with larger sample sizes.

Acknowledgement

The authors would like to thank Head of the Institution, Head of the Department, Senior and Junior colleagues and the participants.

REFERENCES

[1] Nelson Textbook of Pediatrics, 21th edn. Philadephia: WB Sanders; 2019:1619-29.
[2] Dreier JW, Li J, Sun Y, Christensen J. Evaluation of long-term risk of epilepsy, psychiatric disorders, and mortality among children with recurrent febrile seizures: A national cohort study in Denmark. JAMA Pediatrics. 2019;173(12):1164-70.
[3] Sawires R, Buttery J, Fahey M. A review of febrile seizures: Recent advances in understanding of febrile seizure pathophysiology and commonly implicated viral triggers. Front Pediatr. 2022;9:801321.
[4] Shang J, Yamashita T, Fukui Y, Song D, Li X, Zhai Y, et al. Prevalence, incidence, and recurrence of febrile seizures in Korean children based on national registry data. J Clin Neurol. 2018;14(1):43-47.
[5] Kumar N, Midha T, Rao YK. Risk factors of recurrence of febrile seizures in children in a tertiary care hospital in Kanpur: A one year follow up study. Ann Indian Acad Neurol. 2019;22(1):31-36.
[6] Shen F, Lu L, Wu Y, Suo G, Zheng Y, Zhong X, et al. Risk factors and predictors of recurrence of febrile seizures in children in Nantong, China: A retrospective cohort study. BMC Pediatrics. 2024;24(1):420.
[7] Castellazzi ML, La Vecchia A, Scali M, Agostoni C, Di Pietro G, Milani GP. Clinical and laboratory parameters associated with febrile seizure recurrence within the first 24 h: A ten-year cohort study. Front Pediatr. 2024;12:1373848.
[8] Helmy MM, Tolner EA, Vanhatalo S, Voipio J, Kaila K. Brain alkalosis causes birth asphyxia seizures, suggesting therapeutic strategy. Ann Neurol. 2011;69(3):493-500.
[9] Salvati KA, Souza GM, Lu AC, Ritger ML, Guyenet P, Abbott SB, et al. Respiratory alkalosis provokes spike-wave discharges in seizure-prone rats. E-life. 2022;11:e72898.
[10] Tolner EA, Hochman DW, Hassinen P, Otáhal J, Gaily E, Haglund MM, et al. Five percent CO2 is a potent, fast-acting inhalation anticonvulsant. Epilepsia. 2011;52(1):104-14.
[11] Kilicaslan B, Erol I, Ozkale Y, Saygi S, Sariturk C. Association between hypocapnia and febrile seizures. J Child Neurol. 2014;29(5):599-602.
[12] Marzouk H. Relevance of hypocapnia to febrile seizures in children. Egyptian Pediatric Association Gazette. 2015;63(3-4):98-102.
[13] Arshi S, Bemanian MH, Ershadi D, Ehsanipoor F. A study on the relationship between hypocapnia and febrile seizure at HazratRasool Hospital in Iran during a three-year period of 2013-2015. Altern Integ Med. 2019;8(2):1000280.
[14] Abd El-Moneim HM, Hawary B, Kholef EFM, El Tayeb YMH. Association between hypocapnea and febrile seizures. The Egyptian Journal of Hospital Medicine. 2020;81(6):2252-58.
[15] Sachan D, Goyal S. Association of hypocapnia in children with febrile seizures. J Pediatr Neurosci. 2018;13(4):388-91.
[16] Canpolat M, Per H, Gumus H, Elmali F, Kumandas S. Investigating the prevalence of febrile convulsion in Kayseri, Turkey: An assessment of the risk factors for recurrence of febrile convulsion and for development of epilepsy. Seizure. 2018;55:36-47.
[17] Namakin K, Zardast M, Sharifzadeh G, Bidar T, Zargarian S. Serum trace elements in febrile seizure: A case-control study. Iran J Child Neurol. 2016;10(3):57-60.
[18] Nickavar A, Hasanpour H, Sotoudeh K. Validity of serum sodium and calcium screening in children. Acta Medica Iranica. 2009;47(3):229-31.
[19] Usha KC, Suresh R. Reduced serum calcium is a risk factor for febrile seizures. Int J Contemp Pediatr. 2017;4:1506-08.
[20] Schuchmann S, Hauck S, Henning S, Grüters-Kieslich A, Vanhatalo S, Schmitz D, et al. Respiratory alkalosis in children with febrile seizures. Epilepsia. 2011;52(11):1949-55.

PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Paediatrics, Santiniketan Medical College, Bolpur, West Bengal, India.
2. Assistant Professor, Department of Paediatrics, Deben Mahato Government Medical College and Hospital, Purulia, West Bengal, India.
3. Associate Professor, Department of Paediatrics, Dr. B.C. Roy Postgraduate Institute of Paediatric Sciences, Kolkata, West Bengal, India.
4. Associate Professor, Department of Paediatrics, Deben Mahato Government Medical College and Hospital, Purulia, West Bengal, India.
5. Professor, Department of Paediatrics, Rampurhat Government Medical College, Bolpur, West Bengal, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Banasree Roy,
19/C/3, Abinash Chandra Banerjee Lane, Kolkata-700010, West Bengal, India.
E-mail: drbr1978@rediffmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jun 06, 2024
- Manual Googling: Dec 06, 2024
- iThenticate Software: Dec 24, 2024 (13%)

ETYMOLOGY: Author Origin

EMENDATIONS: 8

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Jun 05, 2024**
Date of Peer Review: **Sep 04, 2024**
Date of Acceptance: **Dec 26, 2024**
Date of Publishing: **May 01, 2025**