ISSN No:-2456-2165

Study of Family History in Simple Febrile Seizure

Dr Anjali C S [1], Dr Rena Sara James[2] , Dr Vijaykumar B[3]
1&2 post graduates, 3Professor and HOD Department of paediatrics, KVG medical college, Sullia,

Abstract

➢ ***Background and Objectives***

Febrile seizures occurs between the age of six and sixty months with a temperature of 38°C (100.4°F) or higher, and are not due to central nervous system infection or any metabolic imbalance, and which occur in the absence of history of prior afebrile seizures[1]

A simple febrile seizure is a primary generalized, most often tonic–clonic, attack along with fever, lasting for a maximum of 15 minutes, and not recurrent within a 24-hour period.

A simple febrile seizure is a primary generalized, usually tonic–clonic, attack associated with fever, lasting for a maximum of 15 minutes, and not recurrent within a 24-hour period. The objective of the study was to find family history in simple febrile seizure.

➢ ***Methods***

Total of 100 children with simple febrile seizure were included in our study. History of the presenting complaints in detail were recorded including duration of fever, time of onset of seizures, type of seizures, duration of seizures, past history of seizures and family history of seizures. Blood was sent for serum blood sugar, serum levels of sodium, magnesium, potassium, total calcium & ionised calcium.

➢ ***Results***

The mean age of presentation of simple febrile seizure was found to be 2.6years.Out of 100 children 63% were males and 37% were females.7(7%) patients who had history of febrile seizures in father 15(15%) among mother and 19(19%) among siblings is seen. There were 7(7%) patients who had history of seizures in father 2(2%) among mother and 3(3%) among siblings is seen.

➢ ***Conclusion***

Genetic predisposition have a significant role in febrile seizures. There is definite evidence of genetic role in febrile seizures however the mode of inheritance is not yet identified.

Keywords:- *Hypomagnesemia; Simple Febrile Seizure; Hypocalcemia; Hyponatremia.*

I. INTRODUCTION

Febrile seizures occurs between the age of six and sixty months with a temperature of 38°C (100.4°F) or higher, and are not the due to central nervous system infection or any metabolic imbalance, and which occur in the absence of history of prior afebrile seizures[1]

A simple febrile seizure is a primary generalized, most often tonic–clonic, attack along with fever, lasting for a maximum of 15 minutes, and not recurrent within a 24-hour period. A complex febrile seizure is more prolonged (>15 minutes), focal, and may reoccur within 24 hours. Febrile status epilepticus is another subtype which lasts longer than 30 minutes. Some use the term simple febrile seizure plus for those with recurrent febrile seizures within 24 hr.

Altered levels of blood sodium (Na+), potassium (K+), calcium (Ca+) and magnesium (Mg+) have been entailed in the pathogenesis for developing seizures. Normal levels of these electrolytes are needed for maintaining functions of central nervous system. Alterations in cell membrane ion gradient can cause direct and indirect impact on discharges of nerves and thus facilitating convulsion like activities[5]The American Academy of Pediatrics Practice does not recommend investigating aforementioned electrolytes in serum routinely for evaluating a child with febrile convulsion, unless clinically desirable.

II. METHODOLOGY

➢ *Source of Data Collection*

Patients between 6 months of age and 60 months of age with simple febrile convulsion who presented to the department of Pediatrics, Kurinji Venkataramana Gowda Medical College Hospital, Sullia, Karnataka satisfying inclusion criteria.

- *Inclusion Criteria:*

Patients between ages of 6 month and 60 months of either sex with simple febrile seizures with or without fever or seizure during presentation.

- *Exclusion Criteria*

✓ CNS infections like meningitis or encephalitis.
✓ Metabolic disorder.
✓ Neurological or structural abnormalities in the brain or skull.
✓ PEM
✓ Complex febrile seizures
✓ Known case of seizure disorder
✓ Other congenital disorder causing seizure.

ISSN No:-2456-2165

➢ *Study Period:*
November 2017 to April 2019

➢ *Methods*
A prospective observational clinical study on patients belonging to the age group between 6 months and 60 months with simple febrile convulsions, first or recurrent episode, admitted in the department of paediatrics, K.V.G Medical College and Hospital and willing to participate in this study was taken. The data collection was from December 2017 to June 2019, during which patients presenting with febrile seizures was included irrespective of gender, social status, cast, creed, and religion.

Ethical committee clearance was obtained. Before including the children in the study, a history of the presenting complaints in detail were obtained, which included duration of fever, time of onset of seizures, type of seizures, duration of seizures, past history of seizures and family history of seizures. Additionally, history indicative of any triggering factors for the febrile seizure like cough, cold, nasal discharge, ear discharge, burning micturition or crying during micturition were also recorded.

Vital signs such as heart rate; respiratory rate and blood pressure were also recorded. The axillary temperature in children were recorded with the digital thermometer kept in the axilla for a minute. Anthropometric measurements such as weight, height, mid-arm circumference and head circumference were recorded in accordance with standard recommendations. These were followed by general examination and systemic examination. Children who had any features of chronic congenital or acquired illnesses were not included. Children with features indicative of intracranial infection like altered sensorium, meningeal signs, bulging anterior fontanel etc were also excluded.

➢ *Statistical Method*
Patients information and biochemical investigations was entered in Microsoft Office Excel 2007 and IBM SPSS Statistics 20 was used for analysis. Frequencies, Percentages, Mean & Standard deviation were used to analyse the data.

III. OBSERVATION AND RESULTS

This study was conducted at KVG Medical College Sullia to find the incidence of hypomagnesemia in simple febrile seizure. The data collected were analysed by a biostatistician with SPSS software. A total of 100 children in the age group of 6months and 5years were included in my study

➢ *Sex Wise Distribution of Patients:*
In this study the sex distribution of patients showed that the majority were males and the males: females ratio was found to be 1.7.Out of 16 patients with hypomagnesemia 8 were males and 8 were females

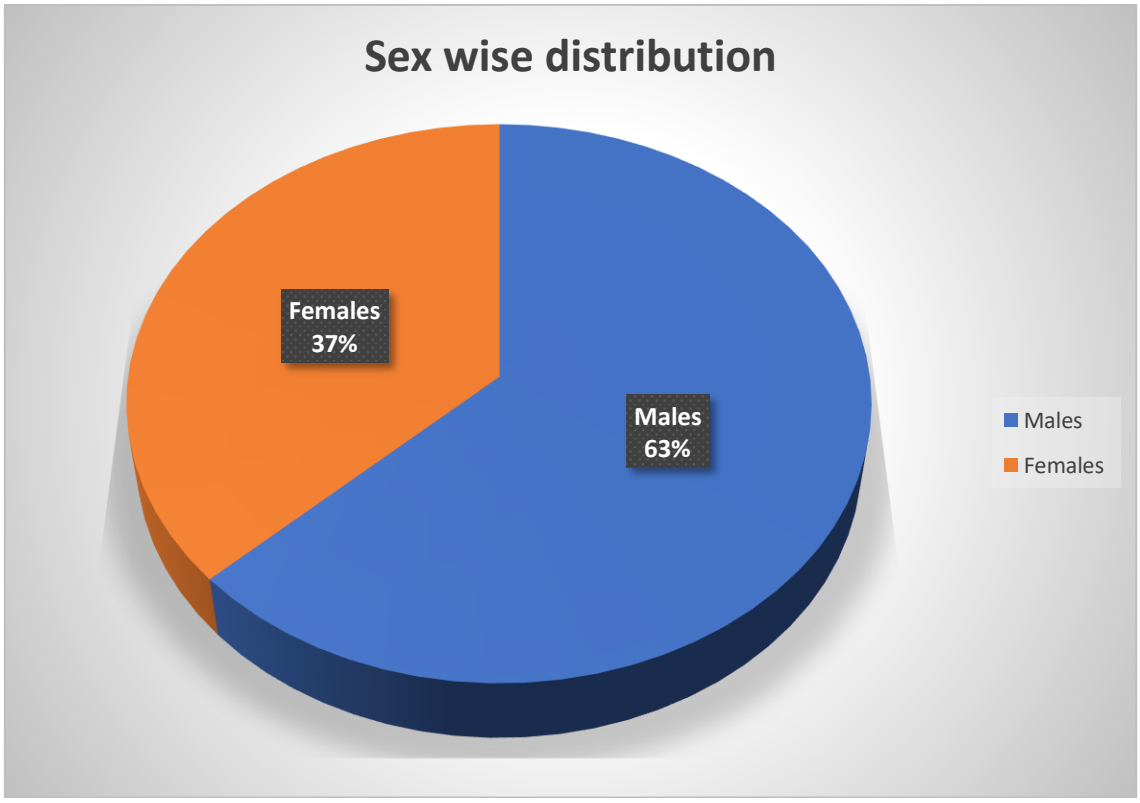

Fig 1

ISSN No:-2456-2165

➢ *Age Wise Distribution of Patients:*

In our study most of the patients belonged to the age group of 2 years one month to 3 years of age. The mean age of the study participants was found to be 2.6year with standard deviation of 1.1 years

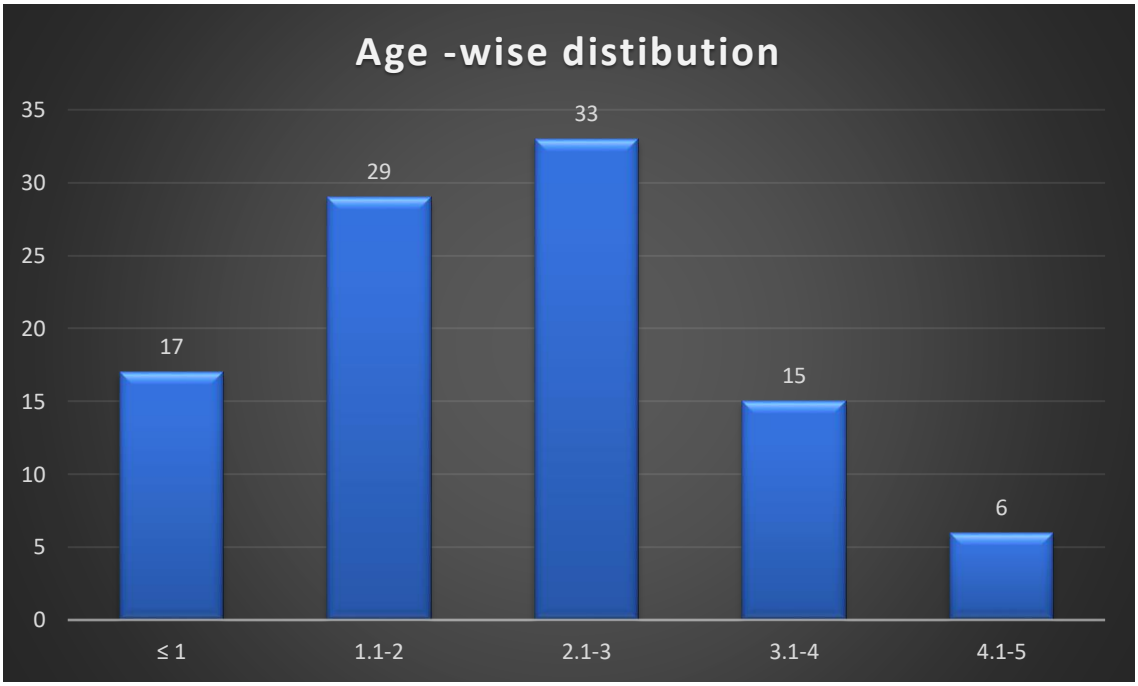

Fig 2

➢ *Distribution of Patients Based on History of Febrile Seizure in Family:*

In our study we had very less patients with history of febrile seizures in family. There were 7(7%) patients with history of febrile seizures in father 15(15%) among mother and 19(19%) among siblings is seen.

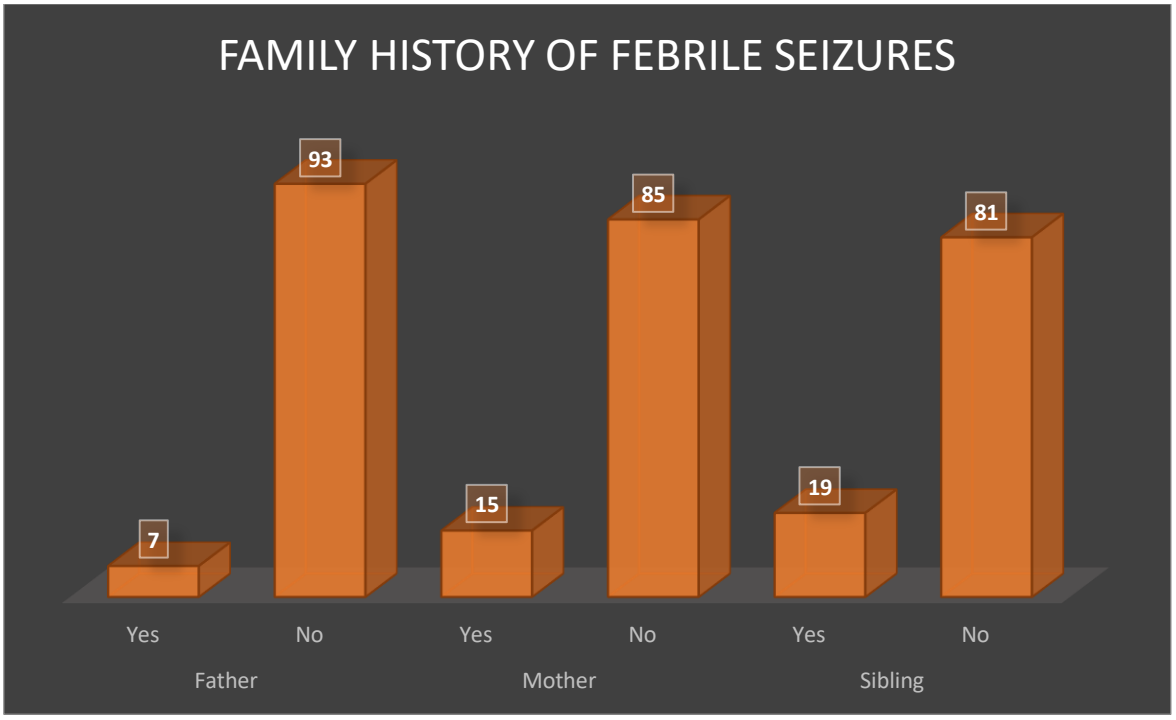

Fig 3

➢ *Distribution of Patients Based on Family History of Epilepsy:*

In our study we had very less patients who had history of seizures in family. There were 7(7%) patients who had history of seizures in father 2(2%) among mother and 3(3%) among siblings is seen.

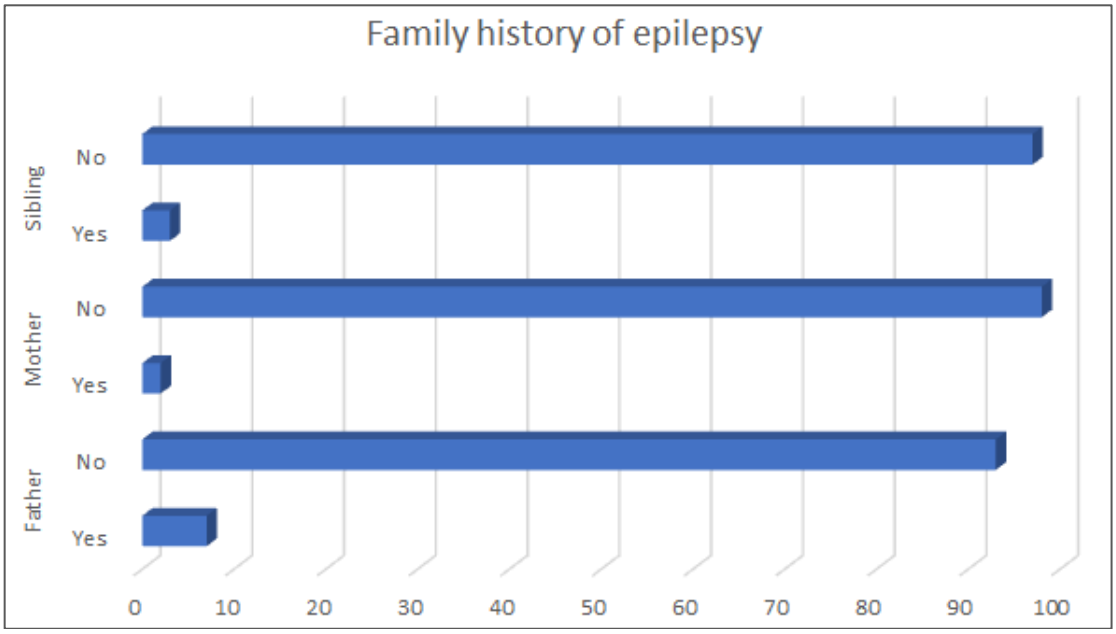

Fig 4

IV. DISCUSSION

Febrile convulsion is an illness which can be an emotional trauma to most parents causing parental anxiety. Many studies had been done to identify the predisposing risk factors and associated metabolic conditions like hyponatremia, hypomagnesemia, decreased zinc level and anemia.

➢ *Age*

In our study most of the patients belonged to the age group of 2 years one month to 3 years of age. The mean age of the study participants was found to be 2.6year with standard deviation of 1.1 years.

- In Namakin K et al study, a mean age of 24 months were reported and few other studies reported mean age of onset between 20 and 27 months.
- Studies have shown that febrile seizures occur within first 3 years of age which is similar to our study where majority are less than 3 years of age.
- Usha Kiran CB et al had mean age in her study of 25 months (2 years 1 month) this is similar to our study.

➢ *Sex*

Inthe study the sex distribution of patients showed that the majority were males and the males: female's ratio was found to be 1.7. And among 16 children with hypomagnesemia 8 were males and 8 were females.

- Sreenivasaiah Bharathi et alhad 47.36% males while in our study it was 63% and were majority unlike the above mentioned study.
- Studies have shown predominance among male pts which is similar in our study where majority are males.
- Usha Kiran CB et al had equal males and females while in our study we had majority males.

➢ *Family history of febrile illness.*

In our study we had very less patients who had history of febrile illness in family. There were 7(7%) patients who had history of febrile seizures in father 15(15%) among mother and 19(19%) among siblings is seen.

- Studies which show genetic predisposition have shown increased risk of developing seizures when siblings have history which is about 10%, this is similar in our study but the percentage is higher in our study.
- Usha Kiran CB et al had 14% cases with family history of febrile seizures while in our study it was 41%.

➢ *Family history of epilepsy*

In our study we had very less patients with history of seizures in family. There were 7(7%) patients with history of seizures in father 2(2%) among mother and 3(3%) among siblings is seen.

V. CONCLUSION

Genetic predisposition have a significant role in febrile seizures. There is definite evidence of genetic role in febrile seizures however the exact mode of inheritance is not yet identified.[30]Polygenic inheritance probably occur in families. The clinical description of 'febrile seizure susceptibility trait' in certain families reveals autosomal dominant type of inheritance with decreased penetrance. Sodium and GABA channel gene mutations are also noted in these families. Febrile seizure genes have been mapped to chromosome 19p and 8q 13-21 in accordance with linkage studies.

ISSN No:-2456-2165

Genes associated with febrile seizures include *SCN1A, SCN1B, SCN9A,* and*CPA6.* In terms of other etiologies, a dysregulation betweenthe proinflammatory IL-1β, IL-6, and IL-8 cytokines and antiinflammatory ILR-1A cytokines has been associated with **febrile status epilepticus.** A decreasedILR-1A/IL-8 ratio (suggestive of an overall proinflammatory state) is predictiveof hippocampal abnormalities on MRI done after febrile status epilepticus. TheILR-1A/IL-8 ratio may thus prove to be a potential biomarker for identifyingfebrile seizure patients who may be at higher risk for developing mesial temporal lobe epilepsy later in life.[1]

Febrile seizure is an ideal example of complex interplay between isgenetic susceptibility and environmental factors. Most probably, all children have mild increased susceptibility to seizures due to fever during a specific age window, this being increased significantly by an underlying genetic influence. History of febrile seizures can be present in any type of epilepsy. A few epilepsy syndromes mostly start with febrile seizures; such as generalized epilepsy with febrile seizures plus (GEFS+), severe myoclonic epilepsy of infancy (SMEI or Dravet syndrome), and, in many patients, temporal lobe epilepsy secondary to mesial temporal sclerosis. GEFS+ syndrome is a highly variable phenotype with autosomal dominant inheritance which occurs typically in early childhood, and remits in mid-childhood. This is characterized by multiple febrile seizures and several subsequent types of afebrile generalized seizures, including generalized tonic-clonic, absence, myoclonic, atonic, or myoclonic astatic seizures with variable degrees of severity. A focal febrile seizures plus epilepsy variant, in which the seizures are focal rather than generalized, has also been described.[1]

Dravet syndrome is the most in severity of the phenotypic spectrum of febrile seizure–associated epilepsies. It is a distinct entity, with onset in infancy. Initially characterized by febrile and afebrile unilateral clonic seizures that recur every one or two months. Seizures subsequently occur with lower fevers and then without fever.. This syndrome is usually caused by a denovo mutation, although rarely it is inherited in an autosomal dominant manner or may be inherited from a nonaffected carrier parent. Mutations in the *SCN1A*gene are the most common cause of Dravet syndrome (causing ~ 80% of all cases). The same gene is mutated in the GEFS+ spectrum; but, in Dravet syndrome there is loss of function due to mutation and so a more severe phenotype. There are several milder variants of Dravet syndrome that manifest some but not all of the above features and that are referred to as Dravet syndrome spectrum or SMEI-Borderland. Rarely the *GABRG2, SCN1B,* and*SCN2A* genes may cause Dravet syndrome; however, in 10–20% of the cases aspecific gene mutation is not identified.

REFERENCES

[1]. Kliegman R M, Stanton B F, St Geme.Seizures in childhood.Nelson textbook of Pediatrics.21th edition.Elsevier; 2020:Chapter 611(2824-2831).

[2]. Swaiman K F, Ashwal S, Ferriero D M, Schor F Nina.Swaimans Pediatric Neurology Principles and Practice.6th edition. Elsevier; 2017: Chapter65 (519-523).

[3]. Consensus statement: febrile seizures-long term management of children with fever-associated seizures. Pediatrics. 1980; 66(6):1009-1012.

[4]. Hughes HK, Kahl LK. Blood chemistries and body fluids.The Harriet Lane Handbook, 21st edition.Elsevier 2018;chapter 27:714

[5]. Van PaerschanW,BodianC,Marker H, metabolic abnormalities and new onset seizure in human immunodeficiency virus-seropositive patients Epilepsia 1995;36:146-150.

[6]. Kaplan PW, Neurologic aspects of eclampsia.neurolxlin 2004:22;841-861

[7]. LennoxWG.Febrile convulsions(hyoerthermic-precipitation epilepsy) epilepsy and related conditions.Boston:Little,Brown and company 1960:393-413.

[8]. Denis W, and Talbot FB. Calcium in blood of children.Amer J Dis Child 1921; 21:29-37.

[9]. Fisher et al. Instruction manual for the ILAE 2017 operational classification of seizure types.Epilepsia2017;58(4):522-530

[10]. Audenart D, Van Broorkhoven. Genes and Loci Involved In Febrile Seizures and Related Epilepsy Syndromes. Hum Mutat, 2006. 27(5): 391-401

[11]. Frantzen, Lennox-Buchthal et al. A Genetic Study of Febrile Convulsions.Neurology, 1970. 20: 909-917.

[12]. Seregi A, ForstermannU, Herldt R, Hertting G et al. The Formation and Regional distribution of prostaglandins D2 and F2α in the brain of spontaneously convulsing gerbils. Brain res 1985; 337:171-174.

[13]. Saper CB &Breder CD. The neurologic basis of fever. New Engl J Med 1994; 330:1880-1886.

[14]. Nemichandra, SC,Prajwala HV, Harsha S. and Narayanappa, 1,3- Department of Neurology, JSS Medical College;2,4-Department of Pediatrics, JSS Medical College and Hospital, JSS University, Mysuru, Karnataka. Implications of alteration of serum trace elements in febrile seizures. International Journal of Current ResearchVol.9, Issue, 07, pp.55044-55047, July, 2017.

[15]. Amouian S, Mohammadian S, Behnampour N, Tizrou M. Trace elements in febrile seizure compared to febrile children admitted to an academic hospital in Iran, 2011. Journal of clinical and diagnostic research: JCDR. 2013 Oct;7(10):2231.

[16]. Khosroshahi N, GhadirianL,Kamrani K. Evaluation of Magnesium Levels in Serum and Cerebrospinal Fluid of Patients with Febrile Convulsion Hospitalized in Bahrami Hospital in Tehran in 2010-2011. ActaMedicaIranica 2015. 53(12):778-781.

[17]. Chhaparwal BC, Kohli G, Pohowalla JN. et al. Indian J Pediatr (1971)38: 241.
[18]. Wong H B. An association between serum magnesium and tremor and convulsion in infants and children..TheLancet , Volume 292 , Issue 7558 , 18 - 21
[19]. Rutter N, Smales OR. Calcium, magnesium, and glucose levels in blood and CSF of children with febrile convulsions. Archives of Disease in Childhood 1976; 51:141-143.
[20]. Friedman M J,GhazalaT Q. Sharieff. Seizures in Children. Pediatric Clinics North America 53 (2006) 257– 277
[21]. VisudhiphanP,VisudtibhanA,Chiemchanya S,Khongkhatithum C. Neonatal Seizures and Familial Hypomagnesemia with Secondary Hypocalcemia.Pediatric neurology.September2005Volume 33, Issue 3, Pages 202–205
[22]. Weisleder P, Tobin JA, Kerrigan JF, Bodensteiner JB. Hypomagnesemic seizures: case report and presumed pathophysiology. Journal of child neurology. 2002 Jan;17(1):59-61.
[23]. Derakhshan R, Balaee P, Bakhshi H, Darakhshan S. The relationship between serum magnesium level and febrile convulsion in 6 months to 6 years old children. Zahedan Journal of Research in Medical Sciences. 2010 Jul 1;12(2):40-2.
[24]. Benga I, Baltescu V, Tilinca R, Pavel O, Ghiran V, Muschevici D, Benga G. Plasma and cerebrospinal fluid concentrations of magnesium in epileptic children. Journal of the neurological sciences. 1985 Jan 31;67(1):29-34.
[25]. Patel S, Rayanagoudar G, GeldingS.Familial hypomagnesaemia with secondary hypocalcaemia.BMJ Case Reports 2016.
[26]. Namakin K, ZaradastM,Sharifzadeh GH, BidarT,Zargarian S. Serum Trace Elements In Febrile Seizures: A Case Control Study . Iron J Child Neural, 2016. 10(3): 57-60.
[27]. Talebian A, Vakili Z, Talar S A. Assessment of The Relation Between Serum Zinc & Serum Magnesium Levels In Children With Febrile Convulsion. Iranian Journal of Pathology, 2009. 4(4): 157-160
[28]. Journal of biomedical sciences Vol.2 No. 1:2 doi:10.3823/1013