A STUDY OF VITAMIN D STATUS IN CHILDRAN WITH FERBILE SEIZURES

Dr.CHIDVITHA.SAI KURRA

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Dr.A.S.AKKI

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LIST OF ABBREVATIONS USED:

NCPP : National Collaborative Perinatal Project

CHES : Child health and education study

NIH : National Institute Of Health

FS : Febrile Seizure

IL : Interleukin

DPT : Diphtheria , Pertussis, Tetanus

MMR : Measles , Mumps , Rubella

SPECT : Single Photon Emission Computed Tomography

CSF : cerebrospinal fluid

HHV : Human Herpes Virus

CMV : Cytomegalovirus

LP : Lumbar Puncture

EEG : Electroencephalogram

CT : computerized tomography

MRI : Magnetic Resonance Imaging

AAP : American Academy of Pediatrics

FS : Febrile Seizure

Ca : Calcium

Mg : Magnesium

URTI : Upper respiratory tract infections

GE : Gastroenteritis

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INTRODUCTION:

The most prevalent neurological condition in infants and young children is febrile seizures. It is an age-dependent condition that affects 2% to 5% of kids under the age of five.[1]

Generalized tonic-clonic seizures classified as simple febrile seizures are those that persist for under 15 minutes and don't repeat for more than 24 hours. In early infancy, they typically return in one-third of children, but they are benign.

Complex febrile seizures are classified as focused, lasting more than 15 minutes, and happening more than once during a 24-hour period. They are a heterogeneous group of events that are linked to an elevated risk of recurrence throughout early childhood and an increased likelihood of future afebrile seizures.

Every child will experience between 3% and 4% of febrile seizures. Its frequency will peak in the following 12 to 18 months.

Definitions:[1]

Seizure:

The term "seizure" refers to the progressive onset of symptoms and signs that are brought on by abnormally high levels of synchronous neuronal activity in the brain.

FS:

Those seizures that:

1) Are frequently observed in children between the ages of 6 and 60 months.
2) They are not brought on by metabolic disturbances or CNS infections.
3) Not previously known to have had afebrile seizures.

Febrile status epilepticus:

These are the FS which last for more than 30 mins of duration and is commonest type of status epileptics occurring.

VITAMIN D:

In people with epilepsy, the role of vitamin D is well recognised. Only a few studies have been done to support the role of vitamin D in febrile seizures and their recurrence. This area of study is still being done.

Low vitamin B6 and low Na levels have been added to the list of risk factors that raise the likelihood of developing a simple febrile seizure. The causes of febrile seizures are the subject of extensive research. [(2)]

There is growing concern that the aetiology of febrile seizures may be significantly influenced by vitamin D. With an estimated 1 billion cases worldwide, vitamin D deficiency is a serious health issue for children. It affects children in India to a prevalence of 50–90%. [3]

AIMS AND OBJECTIVES OF THE STUDY

1. To assess vitamin D levels in children with febrile seizures .
2. To see the correlation between vitamin D levels and recurrence of febrile seizures.
3. To study the focus of fever .
4. To study the risk factors of febrile seizures coming to our institute .

REVIEW OF LITERATURE

Seizures are unusual electrical activity of neurons located in brain which result in gradual stereotypical signs and symptoms where involuntary movements and altered consciousness are noted .[4]

Definitions:

Seizures which occur due to a known etiology are known as **symptomatic seizures**[4].

Provoked seizure – which occur due to acute conditions like fever, decreased glucose levels, consumption of any toxic substance, intracranial infection, injury, or any other factors precipitating to this condition . [4,5]

Unprovoked seizure – seizure which do not occur due to any acute condition, which consists of 3 types[5]:

1. Caused by pre – existing brain abnormality or any insult leading to **remote symptomatic seizure**
2. Seizure due to genetic cause leading to idiopathic seizure
3. Cryptogenic seizure – where the cause is not known

- **Febrile seizure** – seizure which occur in children aged between 6 months to 5 years having a temperature $\geq 38^0$C (100.4^0F) and without CNS infection, disturbances in metabolic, or previous history of afebrile seizure .
- **Infantile spasms** – they are a type of myoclonic-tonic seizure where there is flexor, extensor, or mixed flexor-extensor spasms where it occurs in infants, and unlikely in children who are very young .

- **Epilepsy** : It is the disease of the brain where two or more unprovoked or reflex seizure which occur in less than 24 hours apart in duration (or) 1 unprovoked seizure and with a high risk (≥ 60%) of reappearing of seizures over the next ten years .
- Diagnosis of an epilepsy syndrome

- **Status epilepticus** – it's a condition which results due to either from lack of mechanisms which are responsible for seizure cessation or due to mechanisms which initiate the seizure, this leads to seizures which prolong , and which can lead to long-term complications, which includes death of neuron, injury to neuron , neuron networks alteration, or depend on seizure type and duration.

FEBRILE SEIZURES

Febrile seizures are the most common seizures in children with an uniformly excellent prognosis and the prevalence is between 3-4%.[6]

There has been 3 studies based on population :

- NCPP had included approximately 54,000 American women in the years between 1959 & 1966 and h a v e b e e n their children were followed until the age of seven years.
- Rochester epidemiology project was used to identify the residents of Rochester , Minnesota ,USA who have had seizures for which system of medical records were linked to this project .
- CHES it's a birth cohort study where sixteen thousand neonates live born were enrolled in UK from first week of April 1971 and were followed for 10 years[7].

Prevalence:

Febrile seizures prevalence reported was ranging from 0.1 percent to 15.1 percent with an average prevalence of 5.3%.[8] The wide variation is attributable to different ways of finding the case and definitions of FS.

Figures which were most accurate were probably those attained from population containing children longitudinally. Ross and his colleagues have used the 1958 British cohort and found 2.5 perecent of children had FS and Verity & associates reported a prevalence rate of 2.3% in a longitudinal study conducted in US ,w h e r e Nelson & Ellenberg noticed prevalence of FS of 3.5 perecent at 7 years of age in white children and in black children was 4.2 percent. Prevalence of febrile convulsions in tropics range from 2.4/1000 and in Eucador is also high i.e., 14% in Mariana Island[9-12].

In Parsi community in Mumbai, India, Bharucha et al.[9] demonstrated the prevalence of febrile seizures to be 5.4/1000. Upto 10 percent of children experience FS which was suggested by Indian studies. Incidence rate is similar to that of western countries was suggested by recent data collection.[9,13,14]

Waruiru C et al.[15](2004) carried out a clinical review on Febrile Seizure and their main focus of the review was on the recent information and knowing of FS & outlined the very useful information of the management of children who come with an apparent "**Febrile seizure**". However, they did haven't discussed detailed FS management. According to their review, a cumulative incidence of 2 percent to 5 percent of FS were reported in the population studies in Western Europe and the USA. Incidence in India is 5-10% , 8.8 percent in Japan and 14 % in Guam and noted that incidence of FS varies .[15]

Incidence rate is 3-4% worldwide according to Michael V. Johnston.[16]

Definitions:

By definition according to AAP criteria, the child to have fever, and who was neurologically well between six months to five years of age where the episode of seizure is of short duration i.e less than fifteen minutes, generalized tonic clonic type of seizure which occurs once only i . e . simple FS or multiple times i.e complex FS within a period of 24-hour following a fever.[9] The most widely accepted definition is by the National Institute of Health(NIH) Consensus panel (USA 1980) which defines febrile Convulsions, as an event in infancy or childhood occurring between 3 months and 5 years of age, associated with fever but without evidence of intra cranial infection.[17]

FS was defined by The International league against epilepsy as seizure activity which occurs during the age of 3 month to five yrs , w h i c h i s f o l l o w e d b y f e v e r b u t n o t i n a s s o c i a t i o n w i t h a n y CNS infection ,where the child was seizure free since birth.[18]

A temperature checked axillary of either > 38 degree Celsius or more than 37.8 degree Celsius as a simple cut off level has been proposed to diagnose a febrile seizure, but there are still no consensus.[19]

The Joint Working Group of the research unit of Royal College of Physicians and the British Paediatric Association defines Febrile Convulsions as an Epileptic Seizure occurring in a child aged from 6 months to 5 years precipitated by fever arising from infection outside the nervous system in a child who is otherwise neurologically normal.[19]

A Seizure is defined as a “paroxysmal involuntary brain function that may manifest as an impairment or loss of consciousness, abnormal motor activity, behavioural abnormalities, sensory disturbance, or autonomic dysfunction.”

Epilepsy is defined as a disorder of brain characterized by an endurating predisposition to generate seizures and by the neurobiological, cognitive, psychosocial,and social consequences of this condition.[16]

Status Epilepticus:

The guidelines published by the International League Of Epileptic Society defines Status Epilepticus as,

"A single seizure lasting for more than 30 minute duration or a series of epileptic seizure during which function is not regained between ictal events in a >30 minute period".[7]

Age:

Febrile convulsions occur mainly in the age group of 6 months to five years. These are not seen in a child aged >5 years which indicates a particular possibility of young children where fever is precipitant. Although there are some exceptions, children outside this age range are less likely to have febrile convulsions. The onset of febrile convulsions in the majority of children occurs before 3 years with the average age of onset between 18 and 22 months. The reported prevalence of febrile convulsions in the United States is about 3-4%.[20]

The incidence of FS and its relation with age is not understood clearly where it is said that due to lack of myelination of underdeveloped brain, where chemical composition changes and there are water and electrolyte balance differences, consumption of oxygen is increased , decreased dendritic connections and there's a difference between adult brains electrophysiologic to that of younger children where all of this are shown as probable reasons for correlation between FS and a targeted age group.

Girls have more rapid cerebral maturation which accounts fro rapid decline rate of FS after the second year of life. [20]

Gender :

In most of the studies, it is seen that simple febrile convulsions have occurred more frequently in males, with a male to female ratio ranging from 1.1: 1 to 4:1.[17]

Aetiological aspects:

A) Preconceptual factors:

- Chronic maternal health predating the conceptions of children with febrile convulsions have been found significantly frequently.[21]
- Parental sub fertility is common in families where children particularly males, have seizures when pyrexial. Maternal smoking, maternal motor deficits and mental retardation in an older half sibling are also reported to increase the risk for febrile convulsions.
- Thus, in some affected children the prenatal environment may not be conducive to optimal cerebral development.[22]

B) Prenatal factors:

- Vaginal bleeding either in early or late pregnancy, maternal medications during pregnancy in particular anti-epileptic drugs, anti-emetics, antibiotics, and anti-depressants might have adverse effects on the developing nervous system.[23]
- In a case control study in Western Washington, mothers smoking cigarette and alcohol consumption at the time of during pregnancy were identified as risk factors for FS in their children. Maternal smoking Prenatally has shown to have a two fold risk of developing a simple FS.
- These results says that stoppage of alcohol and smoking during pregnancy, was

routinely advised at the time of pregnancy will be useful in preventing FS .[23] Vestergaard M et al.[24] also confirms the association between prenatal exposure to cigarettes and the risk of febrile seizures in his 2 population based cohorts study.[24]

C) Perinatal factors:

Some studies have shown febrile convulsions are more common in breech deliveries and small for gestational age babies.[7] But in the analysis of the information gathered during the National Collaborative Perinatal Project (NCPP), Nelson and Ellenberg (1990) did not find any of a large number of factors related to pregnancy, labour, and delivery that increase the risk for febrile convulsions.[22,25]

D) Family history of seizures**:**

- Twenty five to forty percent of children may present with FS who has a family history positive for FS and reported frequency of febrile seizures in their siblings ranges from 9- 22%. FS occurring in families with genetic susceptibility where it could be transmitted from both the parents[11].
- If the child's sibling also has FS then its 1 in 5 risk of getting FS & its 1 in 3 risk of getting FS if both the parents are effected with FS.[26]
- The first degree family history of febrile seizures is important in assessing the recurrence risk of FS , whereas 2nd & 3rd degree family history are given as minor risk factor . if the 1st degree relative is affected with FS it gives the highest recurrence risk for FS.[27]
- A family history of non febrile seizures has been reported in several studies as a risk factor for the development of non febrile seizures following febrile seizures. In one large study by Nelson et el.[11] in 1978, it was seen that the risk of epilepsy was

increased among children who had a family with a history of non febrile convulsions . A increase in risk of epilepsy associated with a positive family history was not marked unless there was a previous neurological abnormality or a lengthy focal or multiple first seizure in the proband.[25]

A British National cohort study observed that children with a positive family history were more likely to have a complex first febrile seizure than those with a negative history. In both American and British studies, if onset is in the early age and if family history is positive & these were considered as predictors of FS.[25]

NCPP conducted studies where it has shown that a if family history is positive for seizure disorder and this makes it an important contribution for risk of getting FS & if mother has FS then that will have the highest influence .[11,25]

Febrile convulsions are 2-3 times more likely in family members of affected children than in general population, whereas no clear association exists between febrile convulsions and family history of afebrile seizures.[8,25]

If family history positive for FS then it could show twenty five to forty percent of patients with FS . If more than one family member has a history of febrile convulsions, the risk of febrile convulsions in siblings of probands with febrile convulsions significantly rises.. Rosenberg found that if two or more members of a family had a history of febrile convulsions, their incidence in the siblings of probands with febrile convulsions was 45%, compared with incidence of 0.8% if no family member had febrile convulsion. The NCPP data indicate a positive family history of convulsions in approximately 7% of individuals with febrile convulsions.[28]

In a prospective study, Van Esch A et al.[27], from Netherlands studied 115 children to determine the value of a detailed family history for the assessment ofthe risk of recurrence

of febrile seizures. A positive first-degree family history of febrile seizures elevated the chance of recurrence to 27–52%. Children with second degree relatives who had febrile seizures did not significantly enhance their likelihood of having febrile seizures again.. [27]

Genetic factors in febrile convulsions:

The Febrile seizure susceptibility genes have recently been linked to FEB1 (chromosome 8q13-q21) and FEB2 (chromosome 19p13.3) in multiple large Japanese families, demonstrating an autosomal dominant pattern with reduced penetrance..[29,30]

Furthermore, a family with the clinical subtype known as generalised epilepsy with febrile seizures plus syndrome (GE FS (+)) was found to have a mutation in the voltage-gated sodium (Na+) channel beta subunit gene (SCN1B) at chromosome 19q13.1. One affected sibling increases the likelihood of another kid developing febrile seizures by 1 in 5, and two parents and a previous child increase the risk by 1 in 3.[31]

The mode of genetic inheritance is generally believed to be autosomal dominant with incomplete penetrance but polygenic mechanisms may also be involved and the mode of inheritance may vary from family to family.[12,32,33]

Twin studies:

It has been acknowledged that a sizable hereditary component contributes to the risk of febrile seizures.[32] According to studies, monozygous twins have a greater concordance rate of febrile convulsions than dizygotic twins.[23] In a study done by Marianne J K et al.[34] , he reported that a significantly higher proband wise concordance rates for monozygotic twins compared to dizygotics.[34] Monozygotic twins matched for similar neurological development showed a concordance rate of 80% for febrile convulsions, according to Lennox-Buchthal. Bower et al.[35] have found monozygotic twins had a lower concordance rate of febrile convulsions

(46%), despite the fact that the rate was significantly larger than that of dizygotic twins (13%). Bower discovered 31% in monozygotics as opposed to 14% dizygotes, nevertheless..[35]

Risk of Afebrile convulsions:

While it appears clear that siblings of patients with epilepsy have an increased risk for febrile convulsions, it is not clear whether the siblings are also at increased risk for epilepsy. 3.4% of parents and 4.7% of siblings of febrile convulsion probands have afebrile convulsions. However other investigators have not found an increased risk of epilepsy among siblings of febrile convulsion probands.[35]

Lesions resulting from febrile illness of childhood:

During the age of susceptibility to febrile convulsions, the brain is in a phase of active growth and maturation. On general grounds therefore, it could be due to excess neuronal discharge and hypoxia, occurring during prolonged and / or recurrent convulsions might be particularly likely to cause brain damage. They described cortical neuronal necrosis, sometimes widespread, occasionally laminar, and often most evident in the walls and depths of sulci in 11 children who died within 1 to 13 days of onset of severe febrile convulsions. The distribution of lesions included cerebral cortex, hippocampus, amygdaloid nucleus, thalamus and basal ganglia. On viewing retrospectively, patients with temporal lobe epilepsy have a greater than normal probability of having had febrile convulsion in early life. Furthermore patients with temporal lobe epilepsy and a preceding history of febrile convulsion are more likely to have siblings who have also had febrile convulsions.[12,25]

Iron deficiency and Febrile Seizures

Iron deficiency anemia is one of the most prevalent micronutrient deficiencies in young children in India and other parts of the world, and it is strongly associated with persistent cognitive and motor delays even after the anemia and Iron deficit have been repaired.[36]

Iron deficiency though commonest micronutrient deficiency worldwide is a preventable and treatable condition.[37]

Fevers can increase low serum ferritin's detrimental effects on the brain and lead to seizures.[20] For the metabolism of many neurotransmitters, monoamine, and aldehyde oxidase in the brain, iron is used as a co-factor.[38,30] Thus, iron deficiency may affect a child's seizure threshold. Iron insufficiency is a disorder that is easily treatable and is posited as a risk factor for febrile seizures in children.. [40,41]

In order to determine the association between iron deficiency and febrile seizures, Pisacane A et al.[42] conducted a case-control study in 156 infants younger than 2 years in 1996. It was found that fever can exacerbate the negative effects of anaemia and iron deficiency on the brain, and that seizures can result as a result..[42]

Another study by in 2002 used a prospective case-control design to examine the relationship between iron stores and the first febrile seizure. They came to the conclusion that there may be a role for iron deficiency in the occurrence of first febrile seizures since ferritin levels are low in children who had their 1st FS than in the reference group..[30]

A study in Karachi was done in 2005 to see whether there is a connection between iron deficiency anaemia and FS. They came to the conclusion that FS are more common in kids with iron deficient anaemia...[43]

Hartfield et al.[44] studied in children with febrile seizures were almost twice as likely to be iron deficient as those with febrile illness alone suggesting that screening for iron deficiency

should be considered in children presenting with febrile seizures.[44]P Leela Kumari et al.[45] in their study have shown that iron deficiency is a significant risk factor for simple febrile seizures in children of age group between 6 months to 3 years.[45]

Fever and Febrile Seizures:

Fever provokes seizures in case of a child with febrile convulsions and hence termed febrile seizures.[46]Temperature influences numerous cellular processes, including the electrical activity of neurons.[47]The functions of several neuronal ion channels are dependent markedly on temperature in the physiological and fever ranges, approximately 36– 42^0C .[48]

The temperature also modulates the amplitude and kinetics of major ionic currents.[46]Thus these facts suggests that an increase in the temperature of neuronal tissue could enhance the rate, magnitude or synchrony of neuronal firing, leading to seizures; this notion is supported by the fact that, in children hyperthermia induced by hot bath or anticholinergic medications might also provoke seizures .[29]

Fever mediators also may contribute to the generation of febrile seizures.[46]Fever mediators like cytokines and specifically interleukin IL_1b, enhance neuronal excitability in part by augmenting glutamate – receptor function .[49]

Brain hyperthermia also elicits rapid release of endogenous IL_1b. [50]Which in turn contributes to the generation of Seizures [51.] Mutations in the IL_1b gene promotes, that result in increased production of cytokine, have been reported in individuals with febrile seizures .[52]Thus the available data support a significant role of fever in case of febrile seizures.

Exogenous pyrogens released during viral and bacterial infections cause an upward setting of the thermoregulatory centers in the hypothalamic/ pre optic areas. It has been suggested that the associated release of acetylcholine in the caudal hypothalamus with

subsequent activation of nicotinic receptors concerned in thermogenesis might be directly related to the precipitation of febrileconvulsions.[53]

Eighty percent of febrile seizures take place during the first day of a fever before the parent is aware of the fever, beginning within the first few hours of an acute infectious illness.[25]Whether the most important factor in the induction of convulsions is the level of temperature or the rapidity of its rise but both remains the subject of de bate.[35,54]

When seizure begins temperature is at its peak at 39 to 40^0celsius.[54]Although some studies indicate that the height of body temperature is a primary predictor of seizures associated with hyperthermia, the rapidity with which the temperature rose appears to be more significant than the temperature at which febrile convulsions started.[33,55]

Berg AT et al[56] is credited with the idea that the pace of temperature increase is significant in 1888, & conducted the first thorough investigation in cats and published his findings in 1939.[56]In his investigations on animals, he noted that there was no correlation between the rate at which the temperature rose and the occurrence of convulsions and that animals would seize once the temperature reached a particular point, regardless of the rate at which the temperature rose.[56]

The likelihood of subsequent febrile seizures has also been linked to the peak body temperature at the time of the initial febrile seizure.[33]

Hence with above data it appears that the suddenness in rise in temperature may be the triggering aspect of seizure than the height of fever.[55]

Precipitating Event:

Only when a fever illness occurs during the key age range do febrile convulsions occur in kids with risk characteristics.[22]It has been hypothesised that the convulsion is influenced by the type of bacteria causing the infection, as well as fever, age, and genetic predisposition.

According to epidemiological research conducted in western Europe and the USA, upper respiratory tract infections and other viral disorders are linked to febrile convulsions.[31]

Children who are hospitalised after their first febrile convulsion are most frequently unwell due to viruses. Upper respiratory tract infections, otitis media, gastro-intestinal infections, and roseola infantum are some of the conditions that are frequently linked to one another.[10] Febrile convulsions are less usually linked to measles.. Lewis et al.[35]using Elaborate and Extensive testing implicated a viral etiology in 86% of children admitted to the hospital for first febrile convulsion.[35]Amarendra et al.[57] has noticed 86% children had predisposing URI, 8% had Acute Gastroenteritis.[57]

Some seasonal variations has been observed in the occurence of febrile seizures. Peaks occur in November and January, perhaps related to common respiratory infections. Peaks also occur from June through August when gastrointestinal infections are prevalent.[25]Helen M Lewis studied 73 children (37 boys, 36 girls) with febrile convulsions. A viral illness was demonstrated by isolating a virus from the CSF, blood or urine in 27% of 73 children who were admitted to the hospital after a first episode of febrile convulsion.

Interferon tests and complement fixation tests

However, just 4% of children had the pathogen isolated from the CSF, blood, or urine, according to parallel bacterial cultures that detected a potential infection in 29% of the children. There was no connection between the type of pathogen and the intensity of the convulsion, the level of fever, the CSF protein, or the CSF white cells. The findings imply that a febrile convulsion may be a reaction to a virus or other microorganism invading the bloodstream. Because successful separation of the virus from the blood is not more frequently performed, invasion may be of a short duration.[31]

Human herpes virus 6(HHV-6) has been demonstrated to be the causative agent in Roseola Infantum. It has been suggested that HHV-6 may have neurotropic properties and can be involved in the pathogenesis of febrile seizures in infants. Hukin et al, in their study in British Columbia have concluded the incidence of primary HHV-6 infection is similar in patients with febrile seizures and age matched controls. Hence HHV-6 does not seem to be a major factor in the pathogenesis of first and second febrile seizures.[57]Recurrences of febrile seizures in the respiratory seasons has been associated with Influenza Type A virus.[58]

In a study conducted by Akpede.G.O et al[59] in Nigeria in children less than 5 years with convulsions associated with fever of acute onset, 74% had no localising signs of infection. Of these 68% had malaria, 4% bacteremia and 7% malaria with bacteremia. All the malarial parasites were Plasmodium falciparum. Among children with bacteremia (11%), Staphylococcus Aureus was the commonest single isolate (33%), 56% of the bacteremic children had simple febrile convulsions while 44% had complex convulsions[59].

In a retrospective study (1980-82) conducted at Liverpool by McIntyre et al.[60] All blood sample cultures' results were examined. 2919 blood samples were cultured during the course of the two years. Out of them, 205 (7%), an organism grew, and 110 (4%), were thought to be harmful to the patient. The most prevalent group of microbes was Enterobacteriaceae (28%) with Ecoli accounting for 2/3 of these.[60]

Children who reported with fever and convulsions were routinely given blood and urine cultures as part of a 12-month prospective trial at the Children's Hospital in Brisbane. Of the 282 patients, 12 (4.3%) had bacteraemia. In half of these cases, there was no clinical suspicion. Of the 272 patients, 7 (2.6%) had UTIs, and clinical suspicion for it was present in 6 of these cases. Nine of the 12 individuals who had undetected bacteremia or UTIs experienced simple febrile convulsions. All 12 patients had a prolonged fever. Patients under the age of 2 years had a considerably higher prevalence of bacteremia. It was determined that routine culture

testing increases the likelihood of finding bacteremia and UTI in children who are admitted to the hospital with febrile convulsions.[61]

Febrile seizures and Immunization:

There has been a wide range of studies with respect to relationships between vaccination and febrile seizures. There are lot of evidence showing that vaccination does trigger the onset of febrile seizures in certain genetically susceptibility children.[62]

A study was done by Center for Disease Control and prevention vaccine safety data link working group in regard to association of certain vaccines and febrileconvulsions. They discovered that receiving the DPT vaccine was only connected with a higher risk of febrile seizures on the day of vaccination, whereas receiving the MMR vaccine was linked to a higher risk of febrile seizures 8 to 14 days later. A higher incidence of non-febrile seizures was not linked to any immunisation. However, they discovered that there was no evidence linking the aforementioned risks to any long-term negative effects.[63]

Seizures occurring after immunization are likely to be febrile occurring in response to temperature elevation especially those occurring within 48 hours of DPT and 7-10 days after measles immunizations.[10]

Farrington et al found a higher incidence of convulsions within the first three days following DPT vaccination. The third dose of the vaccination, for which the attributable risk (across all ages), was 1 in 12,500 doses, had the only effect. After the UK switched to an accelerated immunisation schedule, there may have been a 4-fold drop in febrile convulsions linked to the DPT vaccine if the vaccination was completed by 4 months rather than 10 months. The measles component of the MMR vaccine was to blame for 67% of admissions for convulsions that appeared 6–11 days after receiving the immunisation. Only vaccines containing the Urabe

mumps strain were reported to have an increase of hospitalizations for convulsions 15–35 days following MMR immunisation (1 in 2600 Urabe doses).[64]

The prevalence of febrile seizures after primary immunization was found by Harker (1977) in oxford to be 0.09 per 1000 doses for DPT and 0.93 for measles. With newer measles vaccine the prevalence is lower.[33]

Pathophysiology:

Neither experimental nor clinical experience has revealed a definitive mechanism for febrile seizures. Immaturity of the thermoregulatory mechanism and limited capacity of young animals to increase cellular energy metabolism at elevated temperatures have been implicated. Animal studies suggest that Arginine Vasopressin may be an important mediator in the pathogenesis of hypothermia induced seizures.[9,35]

During infancy there is a lowered threshold to convulse in the presence of fever. The brain in children being immature is unstable, hence it reacts to fever by sudden outburst of abnormal activity resulting in convulsive episode.

The pathophysiology of febrile convulsion can be examined on the basis of :

1)Cerebral development prior to and at the critical age.

2)Cerebral damage at the time of the convulsion.

Cerebral development :

Between the ages of 6 months and 3 years, when febrile convulsions are more common, both organization and myelination are occurring in the child's brain. Since prenatal factors and adverse events in early pregnancy may predispose to febrile convulsions, it is possible that abnormal neuronal proliferation and migration might be contributory events, but there is no pathological evidence to confirm this possibility.

There is however evidence from Single Photon Emission Computed Tomography (SPECT) that focal areas of hypoperfusion can be found in a proportion of children with febrile convulsions.[22]

Cerebral damage in association with febrile convulsion:

There is a reasonable amount of circumstantial evidence that single brief (less than 15 minutes) generalised convulsions with fever do not cause recognisable cerebral damage. Biochemical evidence of cerebral hypoxia is lacking when the CSF pyruvate and Lactate levels are measured after brief convulsions with fever. Either prolongation of seizure for more than 30 minutes or repetition within a 24hour period is associated with raised lactate levels and lactate:Pyruvate ratios suggesting that cerebral hypoxia has occurred.[22]

Clinical features:

The disease is characterised by sudden onset of seizures within 24 hours of the child developing fever. The parents sometimes may only complain of seizures but on examination the child appears febrile. They are usually generalized tonic clonic seizure which lasts for a brief period (<15minutes) but however in some children the seizure duration can exceed 15 minutes and may have focal seizures and in rarest of the cases the child may also have febrile status epilepticus. After the seizure episode the child usually has a small duration of postictal

drowsiness for a very brief period but still certain children appear drowsy for a longer duration. A detailed history has to be taken and a complete clinical examination has to be done.

Simple febrile seizure:

A simple febrile seizure is a generalised seizure in a neurologically healthy child that lasts less than 15 minutes and only happens once in 24 hours.[5]

Complex FS:

A complex febrile seizure is one that has one or more of the following characteristics:

• The seizure has a focal onset, displays focal features while occurring, or is followed by a neurological impairment.

• Lengthy time (more than 15 minutes).

• Recurring in the same febrile episode or within 24 hours.

Numerous studies have revealed that between 9 and 35% of people experience complicated febrile seizures..

Differential Diagnosis:

Other causes of acute loss of consciousness or rhythmic involuntary movements in early childhood are:

- Breath holding spells
- Reflex anoxic seizures.
- Syncope
- Rigors and Tetany.
- In breath holding spells & reflux anoxic seizures, the episodes are acute reactions to

noxious stimuli, which are usually unexpected.

- Syncope is associated with limpness & bradycardia rather than tonic clonic movements & tachycardia.
- Consciousness is usually not lost during rigors & tetany.
- Benign paroxysmal vertigo, in which sudden acute episodes ofunsteadiness occur, is not associated with loss of awareness.[65,66]

Investigations:

No investigations are routinely necessary in all children after febrile convulsion. It is prudent to measure the blood glucose concentration with a glucose oxidase strip in any child who is still convulsing or not arousable when seen with or without fever.[14]

The 2 important aims o f sending investigations in a child with FS are to evaluate any CNS infection or fever of unknown cause .Child who is admitted for the first time due to FS is most commonly due to virus.[67]

There is no evidence in the literature that investigations are helpful in determining the origin of convulsions in the absence of a specific clinical illness..[35]

However, Hb, MCV, MCH, and MCHC along with serum Ferritin can be done so as to evaluate for Iron deficiency anaemia which has a strong association withfebrile convulsions.[36-39,68]

To pinpoint the source of the fever and determine the cause of the fever, a thorough clinical examination is required. There is no routine requirement for testing electrolytes, sugars, Ca, Mg, or total counts unless clinically necessary.[11] Urine analysis is recommended in children with no obvious focus of infection to rule out urinary tract infection.[71]

Although neutropenia usually related to viral illness occurs commonly, blood counts are not helpful in management.

Viral studies are seldom of value in the management and prognosis of febrile convulsion. Though detailed studies will often give evidence of viral infection of the CNS the outcome does not seem to be influenced by this, though it is suggested CMV infection may play a part in the evolution from febrile convulsion to epilepsy.[3]

In a study from United Kingdom showed an incidence of 2.3% for occult bacteraemia in children admitted with febrile convulsion. The probability of a serious bacterial infection rose to 80% when

the segmented neutrophil count was greater than 10 x 109 or the band neutrophil count was greater than 5 x 109/L[60]

Bacteraemia is much more prevalent in individuals under the age of 2 years old, according to studies done in Australia in 1984 by Mcintyre PB et al.[61] Leucocytosis (>15,000) was a sensitive (75%) but unreliable (57%) diagnostic tool for bacteremia.[61]

Studies by Gombos M et al.[70] USA, showed occult bacteremia to affect approximately 5% of febrile children aged 2 to 36 months.

Since different studies have proved the occurrence of occult bacteraemia in febrile convulsions, the following investigations are done in all cases of febrile convulsions taken up for the study.

- TLC & DLC
- PBS
- BC / sensitivity
- Urine microscopy
- Stool microscopy

In selected cases, Ear swab for culture & sensitivity in presence of ear discharge, Throat swab for culture & sensitivity in presence of acute pharyngitis or tonsillitis, CSF analysis, EEG , Neuro Imaging are done.

CSF analysis:

Because meningitis signals may be weak or non existent in patients under the age of 12 months who appear with fever and seizures, the AAP strongly advises LP in these cases. Children between the ages of 12 and 18 months should be evaluated for LP because meningitis symptoms and signs might be modest. Children who have gotten antibiotics and are experiencing febrile seizures may also have

meningitis symptoms and indications that are concealed, thus LP is also necessary in these situations.[73]In case of older children LP is indicated whenever a doubt of meningitis exists.[72]

It is not routinely recommended to do CSF analysis in all cases of febrile convulsions.[15] CSF analysis is indicated when:

• Young children (under 18 months)

• Excessive or inexplicable sleepiness or agitation.

• if sings of Meningitis are seen .[15]

Electro-encephalogram (EEG):

Since the EEG is of questionable value following febrile convulsions, routine EEGs are not necessary . EEG does not predict which children progress to a seizure disorder. Epileptiform abnormalities are relatively common in children with benign febrileseizures.[73]Further EEG has a low sensitivity in children under 3 years of age following an unprovoked seizure.[74]

Hence for all these above reasons EEG is not recommended in case of febrile seizures. There is no evidence so far that EEG abnormalities if present also help predict either recurrence of febrile seizures or the development of subsequent epilepsy. [71]

If an EEG is performed soon after a convulsion, it typically reveals a considerable generalised slowing that can last for a week or longer and may be asymmetrical. Abnormalities that become apparent quickly after the post-ictal period include spikes, 4-6/sec slow waves, or spike waves.. Specific abnormalities are more frequently observed in older kids, those who have had several prior febrile convulsions, minor abnormalities that were present before, or those who have had focal seizures. However, these abnormalities are not predictive of recurrent epilepsy nor of recurrences. When febrile convulsions are followed by focal neurological abnormalities, an EEG may

occasionally be required to help rule out an underlying structural lesion. In spite of this, neuroimaging is more helpful.[15]

Neuro Imaging in Febrile Seizures :

A young child with simple febrile seizures should not be handled with a CT scan or an MRI head.[11]

On the basis of the available evidence and consensus, the AAP recommends that neuroimaging not be performed in the routine evaluation of the child with a first simple febrile seizure.[71]

Even though laboratory tests, taking cultures, and imaging are performed in daily practices when approaching febrile seizures, the association with serious infections is rare and usually overestimated. The diagnostic approach should be individualized to each case.[75]

Management of febrile convulsions

The management of febrile convulsions can be considered under the following headings:

- Attention to the effect of the seizure
- Anti-epileptic drugs in the acute stage
- Identification and treatment of the underlying infection
- Recognition of and allaying of parental anxieties
- Attention to the effects of seizure.

It is crucial to actively normalise body temperature in febrile convulsions in addition to maintaining a clear airway, risk of aspiration will be less in semi prone position , and vitals should be monitored. Lowering the body temperature is achieved by removal of excess clothes, tepid sponging and use of antipyretic drugs.

Sponging with ice water will cause cutaneous vasoconstriction and actually increase core body temperature. Sponging with tepid water slowly brings down the body temperature but may induce shivering which is a potent mechanism of thermogenesis and is uncomfortable. Since sponging

does not reset the hypothalamic thermal set point, fever may return once sponging is stopped.[76]The commonly used antipyretic is Paracetamol. Dose: 15 mg/kg/dose q 6 to 8th hrly orally . Rectal suppositories are also available and given when child not able to take orally.

Antiepileptic drugs in the acute stage:

By the time a child is taken to a doctor, the majority of febrile convulsions would have passed. If the infant is still convulsing, it must be treated like any other convulsion, with medication if necessary.

• Preservation of an open airway

• The semiprone position, which reduces the aspiration risk

• Vital signs observation.

Diazepam or Lorazepam administered intravenously can stop seizures. 0.1 mg/kg/dose of lorazepam is intravenously given at a rate of 1 mg per minute.

It is secure and efficient to use rectal diazepam at 0.5 mg/kg. Within 5 to 10 minutes, it is totally absorbed and plasma concentration is attained, virtually as quickly as when it is administered intravenously.

After rubbing the anus with Vaseline, a tiny syringe is used to draw in the undiluted intravenous anticonvulsant mixture. The polythene tube is then carefully pushed 4-5 cm into the anus.

In a pilot research, children with epilepsy who received midazolam by nasal inhalation showed effective seizure control. Recent research has demonstrated that intranasal administration of midazolam is effective just like intravenous diazepam for treating febrile seizures .

If seizure fails in responding to the first dose of diazepam after 15 mins, a further comparable dose can be given. If still there is no response, child should be managed with phenobarbitone/phenytoin loading followed by maintenance dose Intravenously. Febrile convulsions is an important cause of status epilepticus in children & should be managed intensely.[22,65]

Identification & treatment of the underlying infection:

Since febrile convulsion is always a symptom of a generalized illness, it is clearly important that a good physical examination is performed & that treatment for any remediable condition is instituted. Almost 90% of febrile convulsions are related to viral infections and for these children symptomatic therapy will be appropriate. Of the 10% with bacterial infections, it's particularly important to consider and in most cases to remove bacterial meningitis. Children with febrile convulsions may have modest amounts of bacteraemia and urinary tract infections, which are frequently undiagnosed and should be ruled out.[22]

Risk of recurrence:

While relatively few children who experience febrile convulsion develop epilepsy, many children develop recurrence of febrile convulsions.[35]

The recurrence rate is 25-30%. In the NCPP study about 1/3 rd of children had at least one recurrence, 9% had three or more attacks. 50% ofthe second attacks occurred within 6 months of first convulsion, 75% in a span of 1 year and 90% in span of 2 yrs.[6] Recurrence of febrile seizures can be divided into major and minor risk factor.

Major factor :

- Age < 2 yr
- Duration of fever < 24hours
- Fever 38 – 39^{0}C

Major factors :

- Family history of febrile seizures

- Family history of epilepsy
- Complex febrile seizures
- Male Gender
- Low serum sodium

Having no risk factors carries a risk of about 12%, 1 risk factor (25 – 50%), 2risk factors (50 – 59%), 3 or more (73 – 100%).[77]Recurrence risk is not uniform for all children, the most important factor appears to be age of onset of onset of first febrile convulsion. The younger the child at the first attack, the more likely are the chances.[11,31,35]

In the NCPP study, males & females and white & blacks did not differ significantly in their vulnerability to recurrence.[6]

Numerous factors have been discovered to be linked to a higher chance of febrile convulsions returning. These are:

- Younger age of onset (< 15 months)
- Family history of febrile convulsions.
- Epilepsy in first degree relative.

The likelihood of recurrence has not been strongly linked to complex traits.

In a study done by S.G.Ling et al.[78] in Singapore the following factors were attributed to as the risk factors for complex first febrile seizures and subsequent development of recurrence. They are age of 15months or less, birth weight of 2 kg or less and an initial temperature of less than 38 degree Celsius. [78]

It was seen a shorter duration of fever before the initial febrile seizure and a lower temperature at the onset of febrile seizure were associated with an increased risk of recurrence in children who have febrile seizures.[77]

A neurological anomaly that develops later on may raise the risk, however it is impossible to determine how much.

It is currently understood that none of the risk variables by themselves can classify kids as having a high or low risk of recurrent seizures. A combination of risk indicators that function cumulatively and can identify groups of different risk categories can more accurately estimate the risk. According to this subgrouping, the majority of kids (65–75%) have a low recurrence risk of less than 30%, while only a small minority (3–10%) have a high risk and others have an intermediate risk.

In a Nigerian study, they have shown that the younger the age at first febrile convulsion the more likely the recurrence rate. Those with moderate degree of pyrexia were 10 times more likely to have subsequent recurrent convulsions compared to those with high degree of pyrexia. Further, the recurrence rate was 5 times higher in first borns compared second born or more. A male preponderance was observed.[31]

Risk of epilepsy:

Although febrile convulsions are benign in nature but a low proportion ofchildren (2 to 4%) may go ahead and develop epilepsy later in life.[79]

Vestergaard M et al.[80]and his follow investigators investigated into the long term risk of epilepsy after febrile seizures in susceptible subgroups in Denmark. They concluded that children with a history of febrile seizures had a higher rate of epilepsy that lasted into adult life, but less than 7% of children with febrile seizures developed epilepsy during 23 years of follow up. The risk was high for those who had a family history of epilepsy, cerebral palsy, or low APGAR scores at 5 minutes at birth.[80]

In one study which was done in U.K. to know the risk of epilepsy after febrile convulsions concluded that the risk of epilepsy after FS is very less than reported in many hospital based studies.[81]

A study reported that 15% of children with epilepsy have had febrile seizures and only 2% to 7% of children who experience febrile seizures proceed to develop epilepsy later in life. They have reported several predictors of epilepsy after febrile seizure.[77]

The risk factors for epilepsy are:

- Complex febrile seizures.
- Family history of febrile convulsions.

- Presence of neurodevelopmental abnormality

Overall risk after febrile convulsions – 2% to 2.5 percent. Epilepsy risk after one febrile convulsion is ,

- 1% have no risk factors.
- 2.5% for 1 risk factor
- Three risk factors: 5% to 10%

Children who experience epilepsy following prior febrile seizures may experience absence, complex partial, and generalised tonic-clonic seizures.

Intellectual & motor function:

There is no proof that febrile convulsions promote intellectual decline or raise the risk of death, cerebral palsy, or mental impairment.[67,82]

In the British National Child Development study, children with febrile convulsions did not show deficits in school performance at 7 and 11 years of age.

In the NCPP report of intellectual performance in children with febrile convulsions, there was no difference between children with febrile convulsions and their convulsion free siblings on IQ testing at 7 yrs. Neither recurrent convulsions nor those lasting > 30 minutes were associated with IQ deficit.[70]

Prophylaxis :

Patients with febrile convulsion have good outcome. Several studies have found benign outcome. The long term prognosis in terms of subsequent epilepsy, neurological, cognitive & scholastic ability was not influenced by the type of treatment given. There is no evidence that the treatment to prevent recurrence can prevent the subsequent development of epilepsy.

Diazepam, sodium valproate, phenobarbitone & clobazam have been used to prevent recurrence of febrile convulsion. Prescription of prophylaxis should be reserved for the rare cases in which:

Multiple seizures have occurred in a child below 1 year.

- Abnormal neurological development
- Had focal paralysis following a seizure
- Parents anxiety will remain very high even after reassurance.

Prophylaxis are of 2 types :

a) Long term – Phenobarbital and Sodium Valproate are used.

b) Intermittent – Diazepam orally or rectally when the child has got fever in 3 divided doses to total of 1 mg/kg/day in three divided doses. If side effects like lethargy or ataxia occurs, the dosage is halved. Clobazam 0.1 to 1mg/kg/day is also used in intermittent prophylaxis.

A potential drawback of intermittent medication is that seizure could occur before fever is noticed

In children with febrile seizures, this treatment does not reduce the likelihood of developing epilepsy in the future.

Phenobarbital 5mg/kg/day has been used as continuous prophylaxis in one or two divided doses.

Valproate is as effective as phenobarbitone in preventing recurrence.In controlled trials, just 4% of children receiving valproate experienced a second febrile seizure, compared to 35% of control individuals. Adverse effects include fatal hepatotoxicity, thrombocytopenia, pancreatitis, GI disturbances.

Neither valproate nor phenobarbitone is effective in reducing the risk of epilepsy in children with febrile seizures

Finally, a meta-analytical assessment of the preventive measures against febrile seizure recurrence was conducted. Continuous Phenobarbital prophylaxis versus intermittent diazepam prophylaxis was compared & found to have adverse effects with long term use of phenobarbitone & thus long prophylaxis of febrile convulsion is not recommended.[67]

Metabolism of vitamin D:

It's a fat soluble vitamin . Dairy products, eggs, some seafood & foods enriched with D vitamin. But less than 10% of the vitamin D needs are met by food[83]. Cutaneous synthesis is this vitamin's main source. After exposure to particular UV wavelengths in sunshine, 7-dehydrocholesterol is converted to vitamin D photochemically in the skin. In the spring, summer, and fall, fair-skinned people who are exposed to sunshine for ten to fifteen minutes between ten AM and three PM have adequate vitamin D synthesis. Darker skinned need to spend more time in the sun .[84]

Vitamin D Deficiency's Effect on Bone Health:

As the level of vitamin D falls, parathormone level raises as they are inversely related .The intestinal absorption of calcium diminishes when 25-hydroxyvitamin D levels drop below this threshold, and parathyroid hormone levels start to rise.[83]

Subsequent increase in tubular calcium absorption, promotion of phosphorus loss in the urine, and stimulation of kidneys to create Vitamin D3 all result from the release of parathyroid hormone. Additionally, osteoblasts are activated by parathyroid hormone, which promotes the development into mature osteoclasts. Bone has collagen matrix which is mineralised & is broken down by osteoclasts, which can cause bone loss and make bones weak.

Ideal vitamin D levels are still under research , vitamin D should atleast be higher as 30ng/ml cause less levels of vitamin D lead to higher parathyroid hormone levels[84,85]. These definitions, however, are based on data for adults; there are no comparable statistics for kids.

Vitamin D Status Among Children With Epilepsy:

Uncontrollable epilepsy in children frequently don't get enough protein, vitamins, and minerals in their meals.[86] But among ambulatory children whose epilepsy is well-controlled on monotherapy, vitamin D deficiency is also extremely common. Offerman et al.[87]observed in 1979 that vitamin D values are below 15 ng/mL in 72% of 83 children with epilepsy who were 10 to 16 years old as opposed to 50% of 16 control patients.[87]

Levels showed seasonal variation, peaking in June and troughing in December. Since then, a number of cross-sectional studies have looked at how common vitamin D deficiency is among kids with epilepsy.

One scientific trial looked at how vitamin D supplementation affected the bone mineral density of kids with epilepsy. Contradictory research suggests that vitamin D and bone health are complicated issues for kids with epilepsy in a number of different areas.

Verrotti et al.[89]reported that the vitamin D levels of all participants were "normal." However, in all groups, the mean concentration of vitamin D less than thirty ng/ml . Due to the fact that vitamin D levels below 30 ng/mL have an impact on metabolism. [89] These findings indicate that these participants' vitamin D status was indeed poor on the whole.

Due to worries about confounding results, the majority of studies deliberately omit individuals who are not mobile. However, Baer and his associates study looked at vitamin D levels in a huge sample of at-home 3-6-year-old kids in connection to ambulatory status. A total of 236 subjects—23 ambulatory and using antiepileptic medications, 43 non ambulatory

but not using medication of epilepsy, and forty six nonambulatory & using epilepsy medications—were ambulatory.

and observed even after controlling for covariates, non-ambulatory children had a risk of developing deficiency of vitamin. Unrelated to the use of antiepileptic medications, also discovered low values of z-scores for density of bone mineral in non-ambulatory patients. Other research found no correlation between ambulatory status and vitamin D levels in non-institutionalized children with intractable epilepsy, although it's possible that these studies lacked the capacity to detect such a difference.[91]

Children With Intractable Epilepsy's Vitamin D Status:

Kids who have uncontrollable epilepsy are susceptible to having poor nutritional condition. [86] and have shown a high rate of insufficiency of vitamin D. Bergqvist et al.[91] children. Who have uncontrollable seizures who were using medications which are new antiepileptics were investigated before and during their ketogenic diet treatment. Before the diet was started, 51% of the 45 patients had insufficient levels of vitamin D, while 4% of them had insufficiency of vitamin D, which is defined as levels of 11 ng/mL. Bergqvist et al.[93] noticed valuable polypharmacy effect, seven ng/mL drop in vitamin D for every new antiepileptic medication. High values of vitamin D were linked to less use of antiepileptic medicines, generalised seizures, and appropriate vitamin D consumption through diet.

Antiepileptic Drugs :

Because many antiepile ptic medications stimulate hepatic CYP450 mechanism,as increase vitamin D metabolism, which causes levels of vitamin D to decline, parathyroid hormone levels to increase, and turnover of bone to abnormally increase. But even non-enzyme inducing drugs were linked to deteriorating bone health.. The majority of research on vitamin D status

and bone density in kids with epilepsy were not powered or created for examining an impact of certain medications. However , a number of research examined the results of valproic acid and carbamazepine.

Carbamazepine

Children using carbamazepine were the subjects of three studies looking at vitamin D levels[88,92,93]. These two investigations disproved the idea that hepatic enzyme induction is the cause of hypovitaminosis D in epileptic patients by finding no significance statistically between vitamin D levels in controls and kids receiving carbamazepine treatment.

Nicolaidou et al. [93]saw no distinctions between the groups receiving carbamazepine and valproic acid. That study likewise discovered a seasonal variation in vitamin D levels as well as a negative association with vitamin D and parathyroid hormone. Five studies that looked at density of bone in connection to carbamazepine medication found no meaningful effect.[94-98]. While one study found no change in vitamin D levels compared to healthy control subjects, another found that children using carbamazepine had poorer bone mineral density than those taking valproic acid.[99].

Valproic Acid

Many believe that valproic acid should have a less significant impact on bone health than enzyme-inducing antiepileptic medications since it inhibits hepatic enzymes. According to the research, valproic acid has complicated effects on indices of bone health and density. Chou saw valproic acid recipients had better bone mineral density than carbamazepine users, it was observed.[99] Conversely, Babayigit et al. [92] showed that children given valproic acid and healthy control participants did not significantly differ in their levels of 25-hydroxyvitamin D, but that the group comtaining valproic-acid had reduced density of bone mineral.[92] Five

investigations have no evidence of difference in bone mineral density in children who are taking valproic acid.[100]

Oxcarbazepine :

Since oxcarbazepine is a less potent enzyme-inducer than carbamazepine, one may predict that it should have a smaller influence on bone health. Studying how oxcarbazepine affects bone health is crucial because new guidelines indicate that it should be used as the first line treatment for persons with partial epilepsy syndromes[101].

Cansu et al.[102] 34 ambulatory children with newly diagnosed idiopathic localization-related epilepsy who had no additional neurologic or medical diagnosis were prospectively enrolled[102] .

Mintzer et al.[103] in adult patients receiving carbamazepine were switched to oxcarbazepine monotherapy after comparison to control subjects..Oxcarbazepine and carbamazepine individuals' levels of 25-hydroxyvitamin D were much lower than those of control subjects' levels, but there was no significant difference between the two groups, indicating that the newer agent may also have a significant effect on health of the bone .

Lamotrigine

In a study they examined the both the medication i.e valproic acid and lamotrigine separately & together, on bone mass & growth of epileptic kids.[100] The study included 53 individuals who were taking one or both antiepileptic medications and did not have a bone or metabolic disorder, any medications that might affect the bones. Without any discernible differences between patients receiving lamotrigine or valproic acid treatment, 23 participants (43%) had density of bone mineral z-score below 1.5 and nine (24.3%) were less than the 10th

percentile for height. Lower levels of 25-hydroxyvitamin D and worse bone-health markers were correlated with low physical activity ratings. They were linked to low bone mineral density and short stature .

In 2010, Abhijeeth Mishra et al [104]conducted a study on Carbamazepine Therapy effect on Vitamin D and Parathormone in Epileptic Children. 47 kids with partial epilepsy who had just received a diagnosis and started on carbamazepine were included in the study. According to the study, after six months, carbamazepine levels were estimated. 6.72 2.22 years S.D. was the average age.

Kija Edward in their According to a study, vitamin D deficiency affected 11 (16.2%) of the children with epilepsy compared to 6 (8.8%) of the control group (p = 0.29). Compared to the 27 (39.7%) children in the control group, 30 (44.1%) children with epilepsy had vitamin D deficiency. In comparison to the control group, children on ASMs exhibited decreased mean vitamin D levels (p = 0.02). In comparison to controls, children on enzyme-inducing ASMs had higher mean parathyroid hormone levels (p = 0.03) and lower mean vitamin D levels (p = 0.08), vitamin D2 levels (p = 0.0018), vitamin D3 levels (p = 0.004), and serum phosphate levels (p = 0.000). There was no difference in dietary intake or ancestry, despite the fact that both groups' diets were deficient in vitamin D-containing foods.[105]

In 2019, a study conducted a meta analysis observed a decrease in the mean Vit D level in children with epilepsy on valproate monotherapy compared with healthy children with a Standard Mean Difference = -0.313 [-0.457, -0.169]. Cumulative meta-analysis showed progressive negative effect of valproate therapy on Vit D levels across time. Other antiepileptic medications caused a similar effect on Vit D status.[106]

According to a study from 2022 on changes in serum vitamin D levels and to compare changes in serum levels of calcium, phosphate, alkaline phosphatase, and parathyroid hormone after 3 months, the median (IQR) vitamin D levels in the control group were significantly lower than

those in the intervention group, with a difference of 6.64 (8.4, 2.65) vs. 5.66 (1.81, 7.12; p 0.001). Compared to 12.5% in the deficient group and 37.5% in the control group, only 5% of children in the intervention group had vitamin D insufficiency ($p = 0.005$). The group that did not receive supplements experienced significant changes in serum phosphate ($p = 0.02$), ionised calcium ($p = 0.02$), and alkaline phosphatase level ($p = 0.003$)

MATERIALS AND METHODOLOGY

Study design: Cross sectional Observational Study

Study population: Children presenting with febrile seizures are included in the study. Detailed history which is relevant about the present episode will be recorded .The established risk factors for febrile seizures also will be noted in all cases . Then a detailed examination and basic investigations to find the focus of fever will be sent.

- A 5ml of blood sample will be collected and Vitamin D levels would be assayed using chemiluminescent immunoassay. Serum calcium, phosphorus and alkaline phosphatase levels will also be assayed in all children.
- Vitamin D levels will be graded by using IAP grading sufficiency(>20ng/ml), insufficiency (12-20ng/ml), deficient (<12ng/ml).
- Relationship of vitamin D and recurrence of febrile seizures will be assessed by Pearson's chi square test

Study setting : PICU and Paediatric Ward of Shri. B. M. Patil Medical College, Hospital & Research centre, Vijayapura .

Study duration : January 2021 to June 2022

Sample size : With anticipated mean SD +/- of vitamin D level in children (of age 6 months to 60 months) 24.41+/-11.21. This study will require a sample size of 61 patients with 95% level of confidence and precision of 3 .

Formula for sample size : $n=z^2 S^2/ d^2$

Where Z= Z statistic at α level of significance

d^2= Absolute error

P= Proportion rate

q= 100-p

INCLUSION CRITERIA:

Children aged from 6 months to 60 months with febrile seizures.

EXCLUSION CRITERIA:

Children presenting with previous history of having features of rickets and those with liver, renal and endocrine disorders.

Statistical analysis: the date obtained will be entered in Microsoft excel and statistical analysis will be performed using statistical package for social sciences (verson 20)

- Results would be presented as Mean (Median) ±SD, counts and percentages and diagrams.
- For normally distributed continuous variables would be compared using Independent t test. For not normally distributed variables Mann Whitney U test would be used.
- Chi square test will be used for categorical variables
- Person's/ Spearman's Correlation is used to calculate correlation between variables.
- p<0.05 will be considered statistically significant.

- All statistical tests will perform two tailed.

RESULTS

Table 1: Sex distribution of participants

SEX	N	%
Female	29	47.5
Male	32	52.5
Total	61	100.0

In our study, the incidence of male children were 52.5% (32) and females were 47.5% (29) respectively. It was observed that Male: Female ratio was 1.103:1.

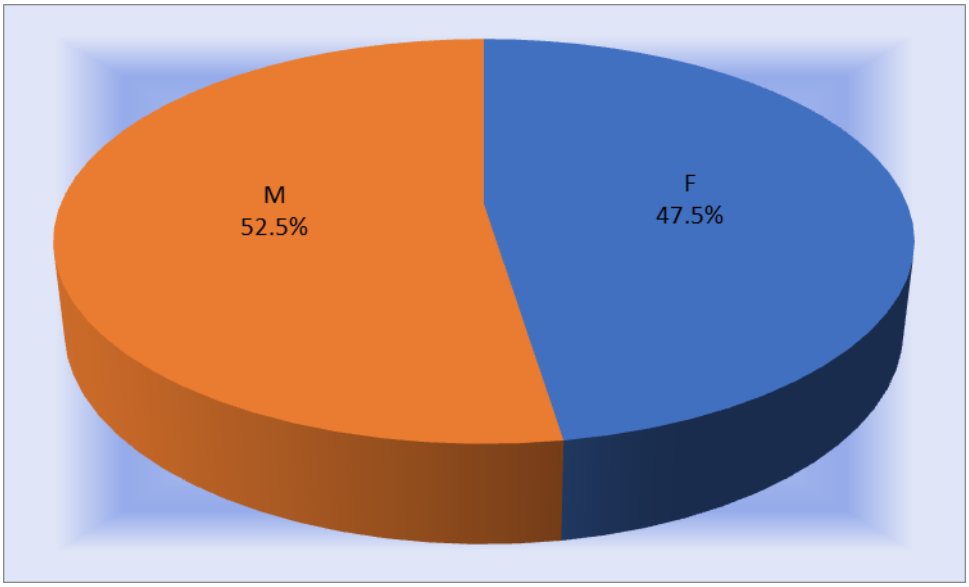

Figure 1: Sex distribution of participants

Table 2: Mean age in male and female

AGE (months)						
SEX	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P value**
FEMALE	29	21.86	12.78	7	48	0.318
MALE	32	25.56	15.61	6	60	
Total	61	23.80	14.34	6	60	

In our study mean age noticed was 23.80± 14.34 months. Age ranges from 6 months to 60 months. The difference between mean age of two sexes of participants was statistically non-significant ($p > 0.05$, t test)

Table 3: Mean vitamin D in male and female

Vitamin D level (ng/ml)						
SEX	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P value**
FEMALE	29	20.37	9.69	10.29	42.32	0.378
MALE	32	18.36	7.95	10.24	34.22	
Total	61	19.31	8.80	10.24	42.32	

In our study mean vitamin D level was 19.31± 8.8 ng/ml. Vitamin D level ranges from 10.24 to 42.32 ng/ml in this study population. The difference between mean vitamin D level of two sexes of participants was statistically non- significant ($p > 0.05$, t test)

Table 4: Vitamin D levels in study population

Vitamin D level	N	%
Deficient	9	14.8%
Insufficient	35	57.4%
Sufficient	17	27.9%
Total	61	100.0%

In our study, Vitamin D sufficiency (>20 ng/ml) was found in only **27.9%** participants (17 out of 61), insufficiency (12-20ng/ml) was found in **57.4%** (35 out of 61) cases and deficiency noticed in **14.8%** (9 out of 61)

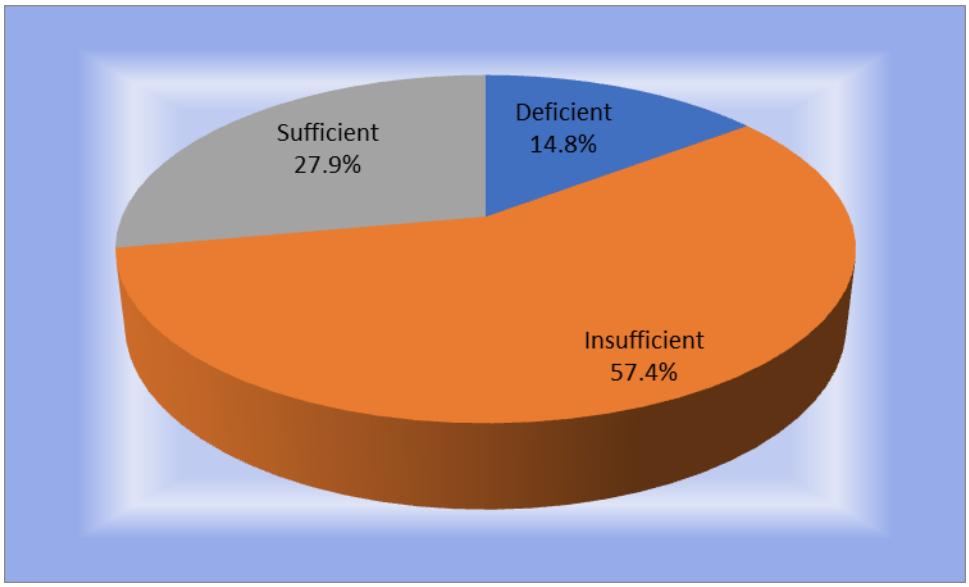

Figure 2: Vitamin D levels in study population

Table 5: Vitamin D levels in two sexes of participants

Vitamin D level	Sex					
	Female		Male		Total	
	N	%	N	%	N	%
Deficient	3	10.3%	6	18.7%	9	14.%
Insufficient	17	58.6%	18	56.3%	35	57.4%
Sufficient	9	31.0%	8	25.0%	17	27.9%
Total	29	100.0%	32	100.0%	61	100.0%

Chi-square = 0.942 with 2 degrees of freedom; P = 0.624

In our study, 31.0% Female population had sufficient Vitamin D (>20 ng/ml) as compared to Vitamin D sufficiency in Male i.e 25.0%. By chi-square test, the p value obtained was >0.05, which was considered to be statistically non- significant.

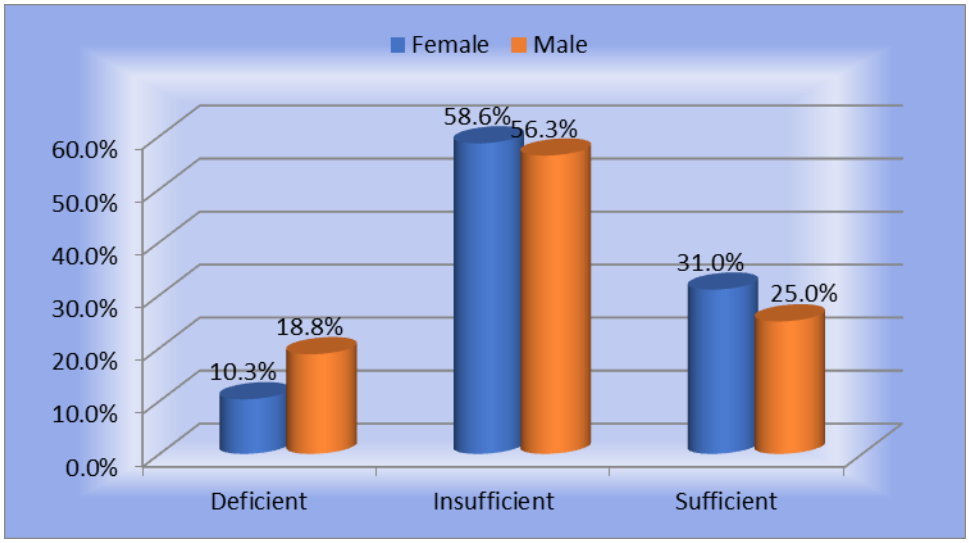

Figure 3: Vitamin D levels in two sexes of participant

Table 6: Vitamin D metabolites in participants

	N	Minimum	Maximum	Mean	SD	Normal range
Calcium (mg/dl)	61	7.9	10.8	9.17	0.62	9 -11
ALP (U/L)	61	118	218	127.23	16.00	100-350
Phosphorous (mg/dl)	61	2.7	7.4	3.79	0.67	2.5-4.5

Table 6 provides an overview of the mean, standard deviations, and ranges of vitamin D metabolites. which don't have statistical significance.

Table 7: Pearson Correlation Coefficient between vitamin D level and age

N	R	P value
61	.079	.547

In our research, we observed a weakly positive association between age and vitamin D level that was statistically non-significant ($p > 0.05$).

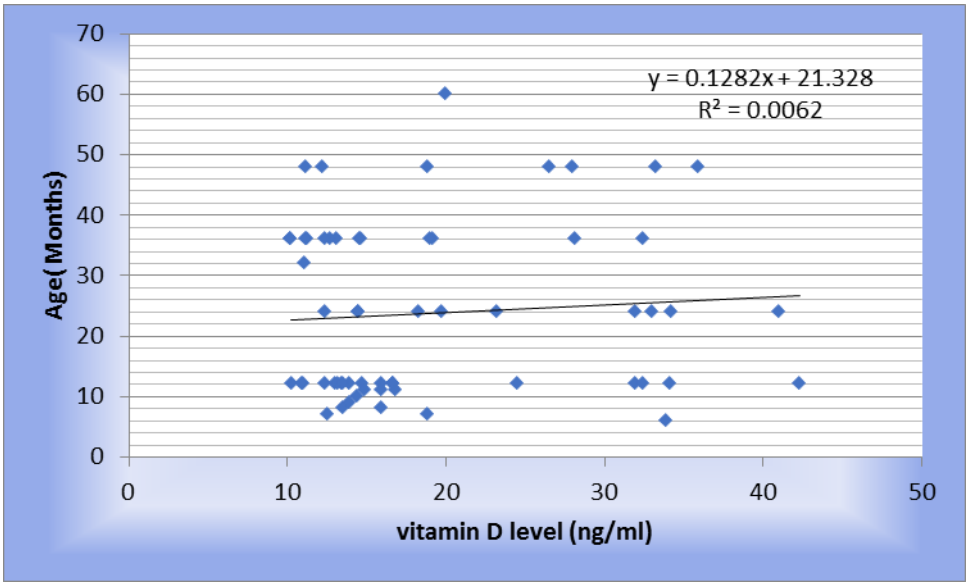

Figure 4: The correlation between vitamin D level and age

Table 8: Pearson Correlation Coefficient between vitamin D level and Calcium

N	R	P value
61	.072	.579

In this study, we found a weakly positive association between calcium levels and vitamin D levels that was statistically non-significant ($p > 0.05$).

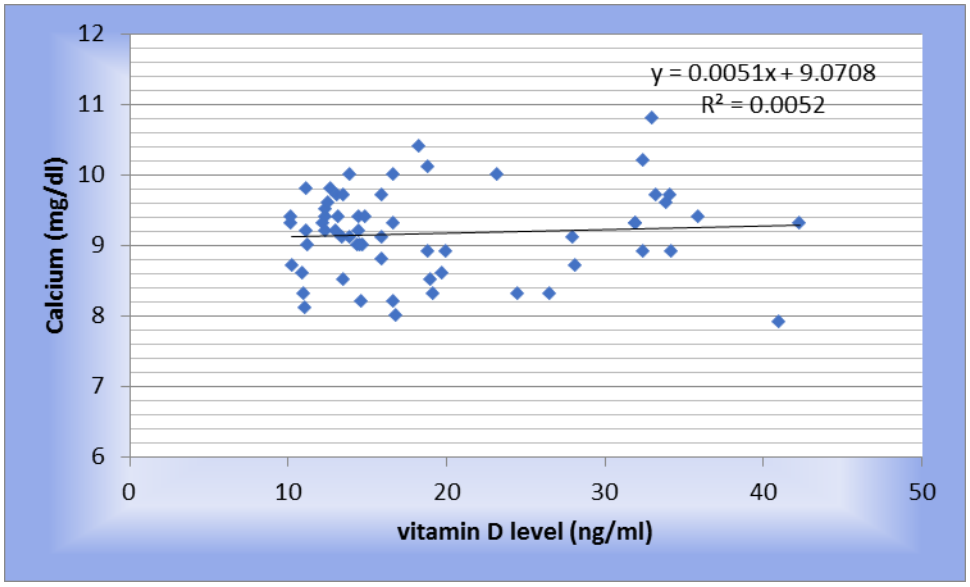

Figure 5: The correlation between vitamin D level and Calcium

Table 9: Pearson Correlation Coefficient between vitamin D level and ALP

N	R	P value
61	.083	.527

In our study, it was found statistically non- significant (p >0.05) and is weakly positive relation between ALP and vitamin D .

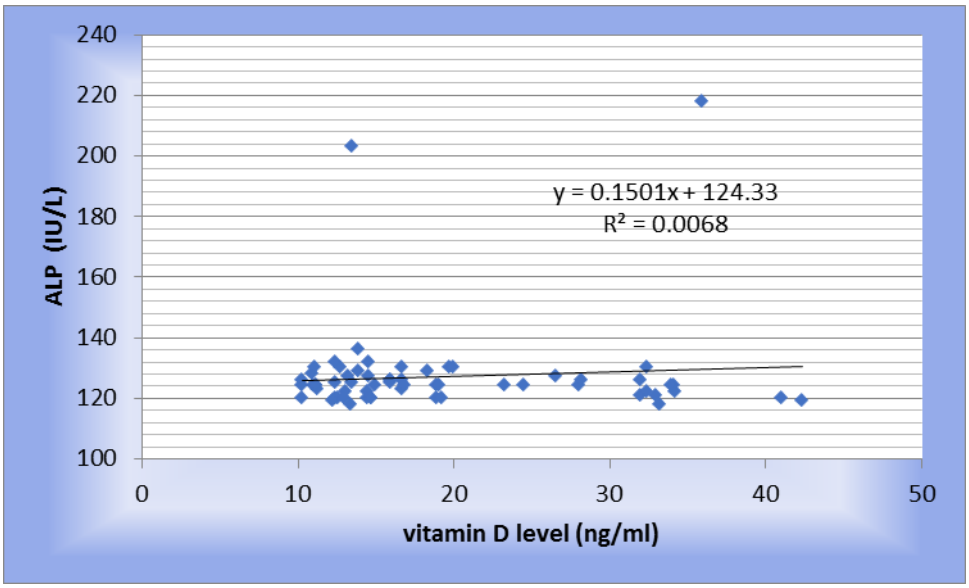

Figure 6: The correlation between vitamin D level and ALP

Table 10: Pearson Correlation Coefficient between vitamin D level and Phosphorous

N	R	P value
61	-.099	.447

In our study, we found statistically non- significant and correlation was weak negative between vitamin D level & Phosphorous.

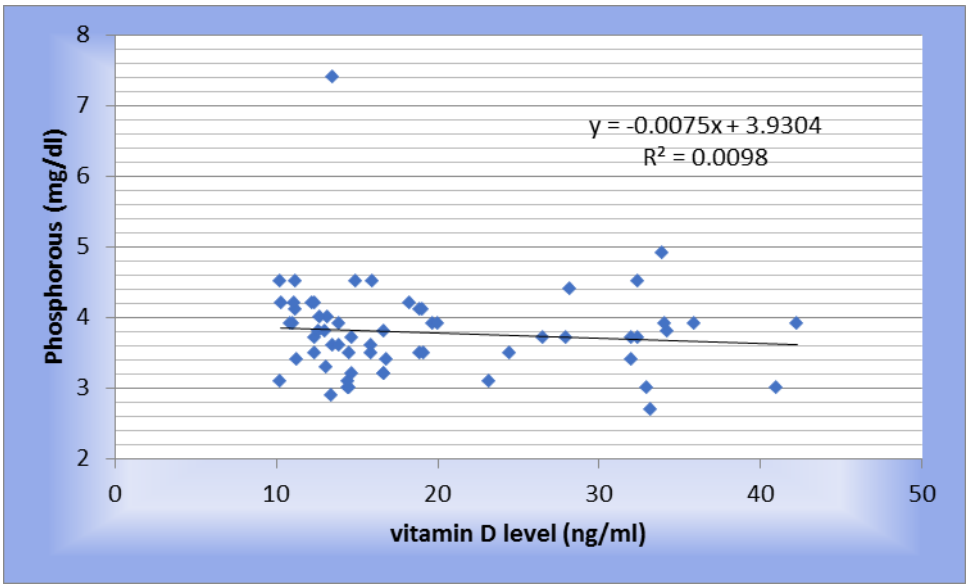

Figure 7: The correlation between vitamin D level and Phosphorous

Table 11: Duration of seizure

Duration of seizure	N	%
<5 Minutes	26	42.6
5-10 minutes	35	57.4
Total	61	100.0

In our study mean duration of seizure was 4.81± 1.89 minutes. 42.6% patients had seizure less than 5 minutes and 57.4% had seizure duration between 5-10 mins .

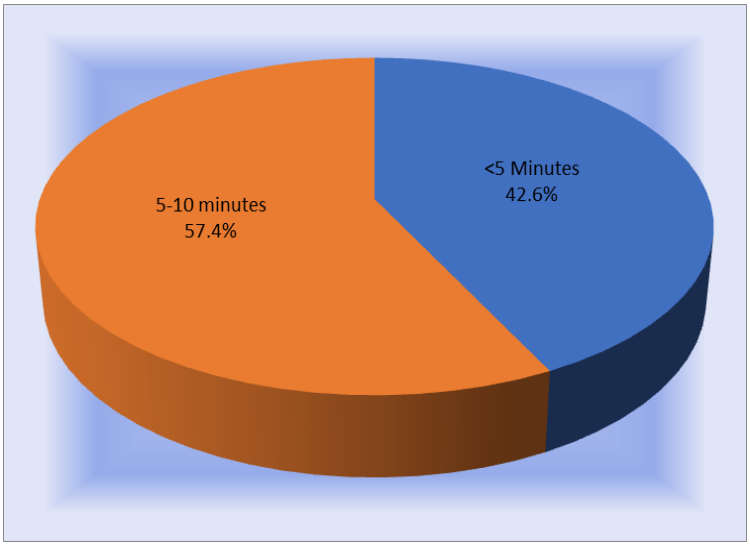

Figure 8: Duration of seizure

Table 12: The mean of vitamin D level according to recurrence of febrile seizure

Vitamin D level (ng/ml)						
Recurrence	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P value**
No	53	19.76	9.11	10.24	42.32	0.313
Yes	8	16.36	6.07	10.24	28.00	
Total	61	19.31	8.80	10.24	42.32	

In our study, the recurrence of febrile seizure was found in 8 participants. The mean vitamin D level was 16.36± 6.07 ng/ml in recurrence group. The difference between mean vitamin D level of two group of participants according to recurrence was statistically non-significant ($p > 0.05$)

Table 13: Recurrence of febrile seizure

Recurrence	Number of cases	Percentage
Yes	8	13.1
No	53	86.9
Total	61	100.0

In our study, the recurrence of febrile seizure was found in 13.1% participants.

Figure 9: Recurrence of febrile seizure

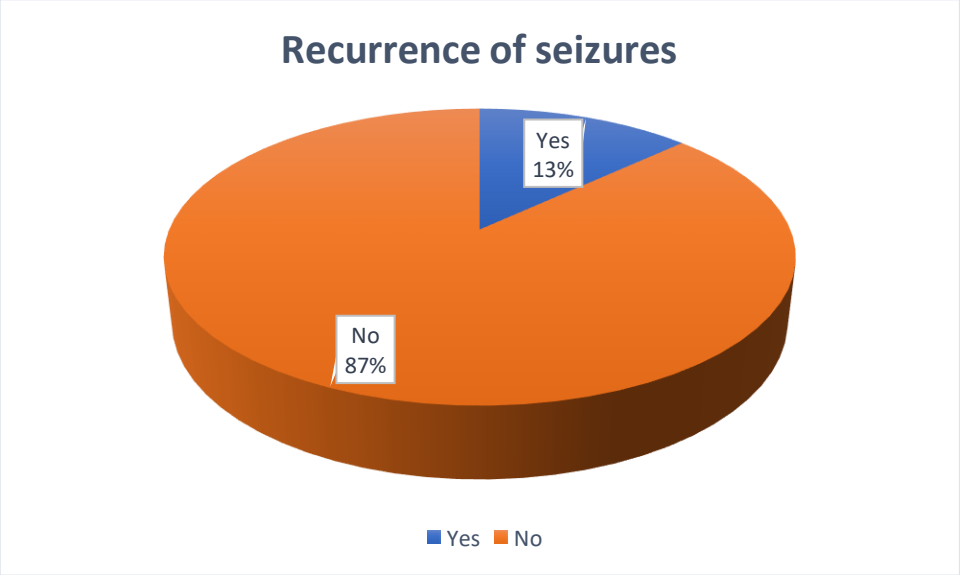

Table 14 : Vitamin D levels according to recurrence of febrile seizure

Vitamin D level	Recurrence					
	No		Yes		Total	
	N	%	N	%	N	%
Deficient	6	11.3%	3	37.5%	9	14.8%
Insufficient	31	58.5%	4	50.0%	35	57.4%
Sufficient	16	30.2%	1	12.5%	17	27.9%
Total	53	100.0%	8	100.0%	61	100.0%

Chi-square = 4.096 with 2 degrees of freedom; P = 0.129

In our study, 37.5% of recurrence group had deficiency of Vitamin D as compared to no recurrence group i.e 11.3%.In recurrence group, vitamin D insufficiency was seen in 50% of the cases. By chi-square test, the p value obtained was >0.05, which was considered to be statistically non-significant.

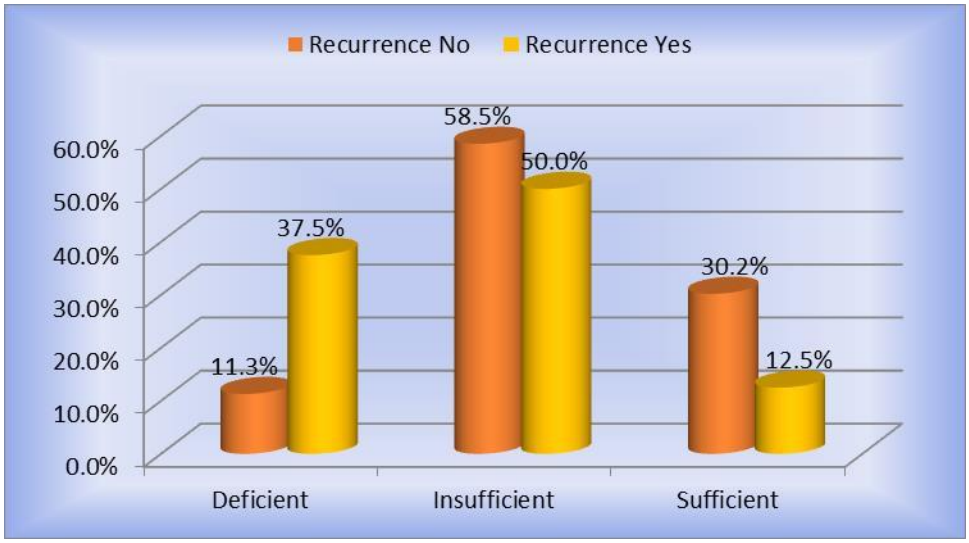

Figure 10: Vitamin D levels in recurrence FS

Table 15: Age group distribution of participants

Age group	N	%
<18 Months	28	45.9%
>18 Months	33	54.1%
Total	61	100.0%

In our study, 54.1 % participants are aged more than 18 months .

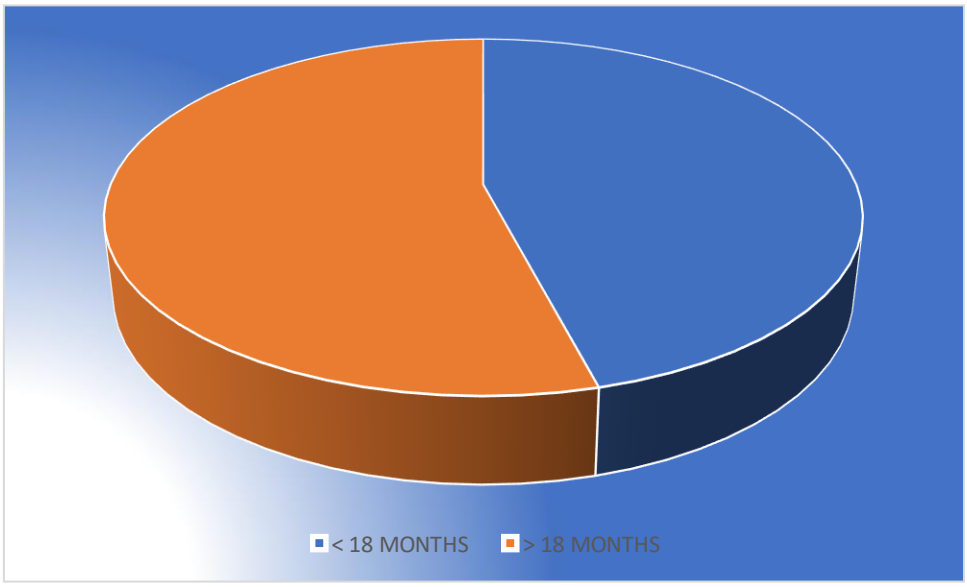

FIGURE 11: AGE DISTRIBUTION

Table 16: Temperature distribution of participants

Temperature	N	%
<100.4˚F	27	44.3%
100.4-102.2˚F	34	55.7%
Total	61	100.0%

In our study , 55.7 % children had temperature of more than 100.4 degree Fahrenheit .

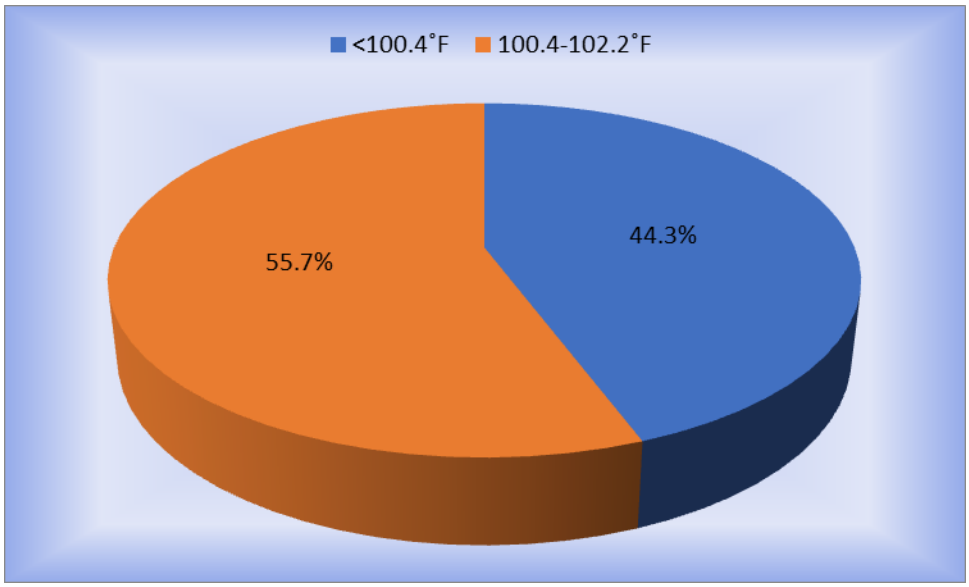

FIGURE 12: TEMPERATURE DISTRIBUTION

Table 17 : Fever duration distribution of participants

Duration of fever	N	%
1 day	38	62.3%
>1 day	23	37.7%
Total	61	100.0%

In our study, in 62.3 % participants fever duration was of shorter duration i.e 1day.

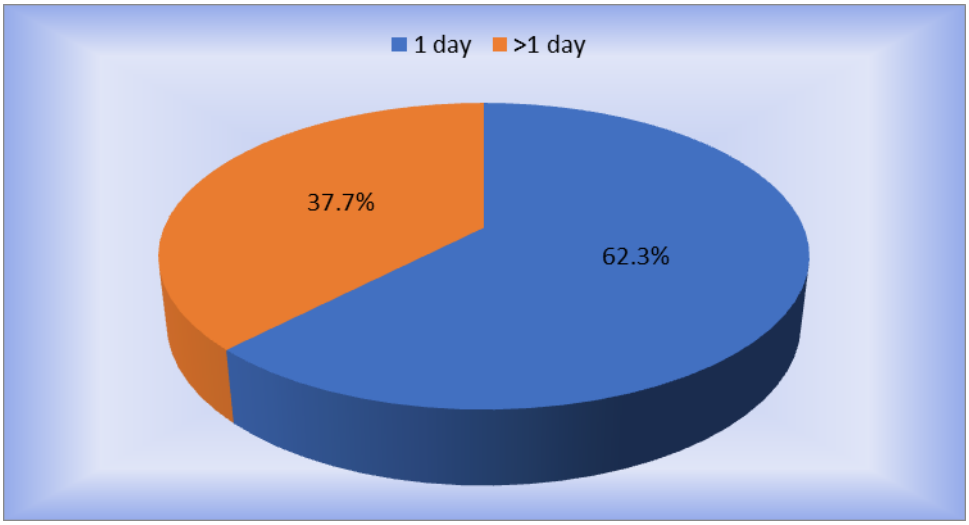

FIGURE 13: FEVER DURATION

Table18: Foci of infections in participants

Foci of infections	N	%
GE	**14**	**23.0**
URTI	**20**	**32.8**
Undetected	**27**	**44.3**
Total	**61**	**100.0**

In our study, focus of infection were found in 55.8% of the cases where, 32.8 % of cases were URTI , 23 % in GE cases and 44.3% were fever without focus .

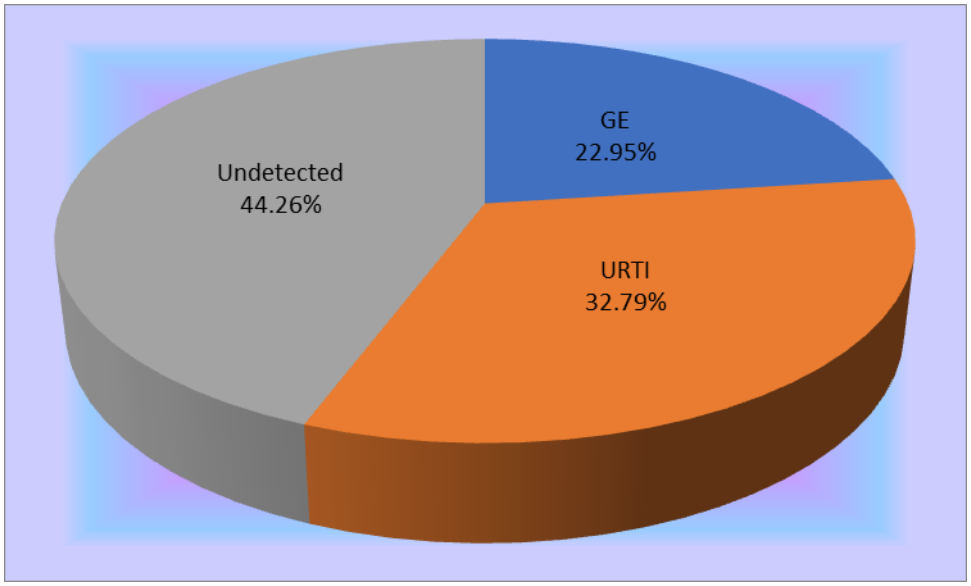

FIGURE 14: FOCI OF FEVER

TABLE NO 19: CRP AND DISTRIBUTION OF PARTICIPANTS

CRP	NUMBER OF CASES	PERCENTAGE
<10 (NEGATIVE)	26	43%
>10 (POSITIVE)	35	57%
TOTAL	61	100%

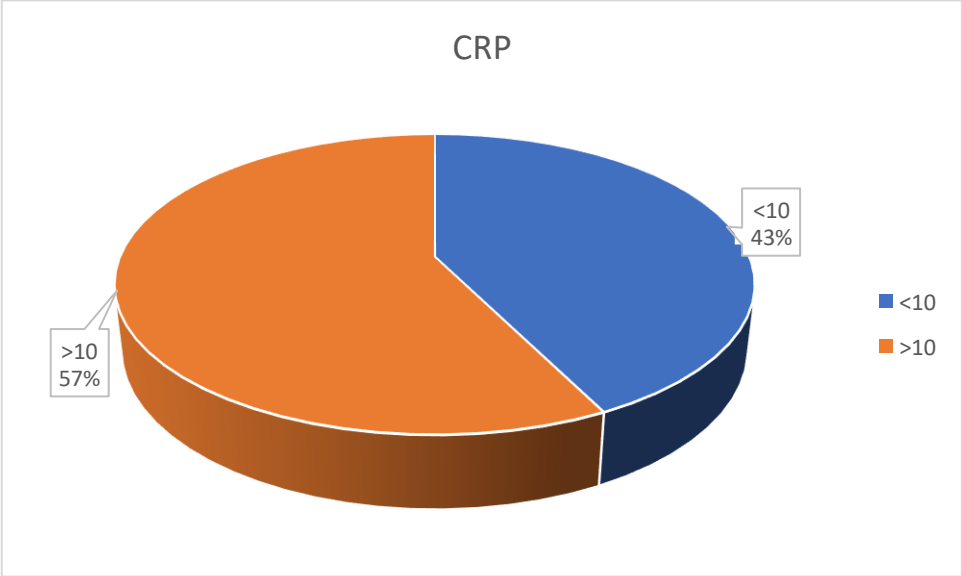

FIGURE 15: CRP

DISCUSSION :

In children, febrile convulsions are a frequent cause of medical emergencies.

seen all around the world. From 6 months to 5 years of age, febrile seizures can happen. 3 - 4% of people experience febrile convulsions each year worldwide.

The incidence is the same all throughout the world. At least 3-4% of kids might have under the age of five, experience one episode. According to estimates, incidence in India is about 10%. However, current research show that the incidence is nearly equivalent to Western-based people [1] . Some feverish children may get FS and some children may not experience febrile convulsions. The fundamental mechanism is still unclear.

Several causes which includes family history and genetics, vitamin D deficiency and a history of febrile convulsions were suggested.

Studies have indicated that a lack of vitamin D is a risk factor for convulsions to occur . In our study, we have observed that low levels of vitamin D levels could cause febrile convulsions to develop.[2]

In current study 23.80 +/- 14.34 months was the mean age observed , similar results were seen in Mahyar et al.[6] study in 2006 which includes 52 children with FS and fifty two healthy children as controls and showed 27.13 ± 15.72 months as the mean age . In Waqar Rabbani et al.[108] study where they had got 23.97±14.45 months as mean age .

In our study, the incidence of febrile seizure episodes in males and females were 52.5% (32) and 47.5% (29) respectively, Male: Female ratio was 1.103:1, mean age was calculated as 23.80 ± 14.34 months. Similar studies were done by Motlaghzadeh et al .[109]& Shariatpanahi et al.[3] revealed the similar results stating that there's no such gender variation of levels of vitamin D was noticed.

GENDER DIFFERENCE ACCORDING TO DIFFERENT AUTHORS:

S.NO	AUTHOR	% OF MALE	% OF FEMALE
1	Motlaghzadeh et al	61.9%	70.8%
2	Shariatpatnani et al	45%	63%
3	Our study	52.5%	47.5%

In our study, mean vitamin D level was 19.31 +/- 8.8 ng/ml and the difference between mean vitamin D level of two sexes of participants was statistically non- significant (p >0.05, t test) . In Ghazal Shariatpanahi et al [3] study, the average levels of vitamin D were 24.41 +/- 11.21 ng/ml.

In our study, vitamin D insufficiency was the most common among children who experienced febrile seizures i.e 57.4% ,followed by sufficiency -27.9% & deficiency -14.8% .Studies by Motlaghzadeh et al [109], Ghazal Shariatpanahi et al[3], Jehangir A.Bhat et al.[2], Virender Singh et al[110]. also stated that similar results with high incidence of insufficiency of vitamin D in children with FS.

Like Rabbani et al.[108] we have also found a strikingly high prevalence of vitamin D insufficiency, they found that insufficient vitamin D levels was 46.6%, and that vitamin D deficiency affected 7.9% of children under the age of two years

Ghazal Shariatpanahi et al. [3] studied levels of vitamin D and its association with febrile seizures in children in Iran . The study was performed on forty children with FS and were screened. The levels of calcium, phosphorus, PTH, and were measured. There is was no significant

correlation between vitamin D and its metabolites in their study, similar results were observed in our study.

VITAMIN D STATUS ACCORDING TO DIFFERENT AUTHORS

	AUTHOR	INSUFFICIENCY	DEFICIENCY	SUFFIENCY
1	Ghazal Shariatpanahi et al	72.5%	7.5%	20%
2	Virender Singh *et al.*	59.5%	13.5%	27%
3	Jehangir A.Bhat et al	43.56%	30.89%	25.56%
4	Present study	57.4%	14.8%	27.9%

Jehangir A.Bhat et al.[2] in their study found a strong negative connection between 25-hydroxy vitamin D and febrile seizure recurrence. He noticed 3rd, 4th episode of recurrent FS had more of vitamin D deficiency . The % of kids who experienced 2nd episode of FS had approximately equal in deficiency and insufficiency groups and least (1.4%) in normal group. Similar results were noticed in our study as well .

In our study, children with recurrence episode of febrile seizure ,50% of recurrence group had vitamin D insufficiency and of which 37.5% of recurrence group had deficient Vitamin D this is similar to study conducted by Jehangir A.Bhat et al.[2]

Amarendra et al.[57] have reported common cause of fever in febrile seizures were upper respiratory tract infections followed by acute gastroenteritis. In our study, focus of infection were found in 34 (55.8%) cases, 14 (23%) cases had acute GE, 20(32.8%) cases had URTI and 27(44.2%) cases had undetermined focus of fever (excluding CNS infection) which is similar to the study conducted by Amarendra et al.[57]

In a study, conducted by Krystyna Gontko et al.[111] have observed CRP was significantly lower in children with FS compared to children with fever (15.73 vs 58.50 ; $p<0.001$). In our study, CRP was positive in 57% of cases and negative in 43% of cases.

In our study, we have included children between the age six months to sixty months, we found < 18 months are 45.9% and > 18 months are 54.1 % . Nadirah rasyid ridha et.al [112] in her study revealed that children having first FS within 18 months are 71.37 times more prone to develop recurrent episode of FS.

In our study ,males were 52.5 % and females were 47.5% . Z.Habib et al.[113] in her study concluded that 57% male had recurrence of FS and mentioned that males are 1.3 times more prone for recurrent FS compared to females. In our study ,out of 61 cases , we observed 8 cases of recurrence FS which constitutes to 13.1% where 87% were males .

In our study, duration of fever of 1 day was noticed in 62.3% , 37.7% of the study group had duration of fever more than 1 day. Berg AT et al[114] mentioned in their study that 67% of children had recurrent episode of FS where child had shorter duration of fever at first episode

of febrile seizure. In our study, out of 8 recurrence FS, 7 had 1 day of fever. Children from our study needs regular follow up as they might develop recurrent episode of FS.

CONCLUSION :

- In our study, we noticed 57.4% of cases had insufficiency of vitamin D levels in febrile seizures.

- In recurrent episode of febrile seizures, we have observed low vitamin D levels in 87.5% of cases.

- In our study, focus of fever was unknown/idiopathic (44.3%) followed by URTI (32.8%) and Gastroenteritis (23%).CRP is positive in 53% of cases with FS.

- In this current study ,>18 months , duration of fever of 1 day and male sex are the risk factors for febrile seizures and needs regular follow up to notice recurrence of febrile seizures.

RECOMMENDATION :

Vitamin D supplementation could be given in all cases of febrile seizures so as to reduce the risk of recurrence. However, further studies with larger sample size are required to analyse the relationship of vitamin D and recurrence of febrile seizures .

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B.L.D.E. (DEEMED TO BE UNIVERSITY) IEC/No-09/2021 Date-22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11-00 am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: A study of Vitamin D status in children with febrile seizures

Name of PG student: Dr Kurra Chidvitha Sai, Department of Paediatrics

Name of Guide/Co-investigator: Dr A S Akki, Professor of Paediatrics

DR .S.V.PATIL
CHAIRMAN, IEC

Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

1

PARENTS / GUARDIAN CONSENT STATEMENT:

We confirm that Dr. Kurra Chidvitha Sai is doing "A STUDY ON VITAMIN D STATUS IN CHILDREN WITH FEBRILE SEIZURES "in Paediatrics ward and PICU In Shri B. M. Patil Medical College Hospital, Vijayapura, Karnataka. Dr. Kurra Chidvitha Sai has explained to us the purpose of research and the study procedure. We are willing to allow our child to get treated in Shri B.M. Patil Medical College Hospital, Vijayapura. We have been explained about the study, benefits and possible discomforts in detail in our native language and we understand the same. We are aware that child will get best treatment, and no compensation like financial benefits will be given if our child's condition deteriorates and any untoward complication happens, and we will not sue anyone regarding this. Therefore, we agree to give our full consent for child's participation as a subject in this research project.

_____ _____

(Parents / Guardian) (Witness to signature)

DATE : DATE:

PROFORMA :

IDENTIFICATION:

1) OPD /IPD NUMBER:

2) NAME:

3) AGE and SEX

HISTORY

4) HISTORY OF PRESENT SEIZURE:

A) NUMBER:

B) TYPE OF SEIZURE:

C) DESCRIPTION (DURATION AND RELATION WITH FEVER)

5) HISTORY OF PREVIOUS SEIZURE:

6) HISTORY OF FEVER: a) DURATION:

- b) SEVERITY:

7) FAMILY HISTORY OF FEBRILE SEIZURES

8) FAMILY HISTORY OF EPILEPSY

9) HISTORY RELATED TO FEVER:

10) BIRTH HISTORY INCLUDING TERM, PLACE AND MODE OF DELIVERY AND CRY AT BIRTH

ANTENATAL:

PERINATAL:

POSTNATAL:

11) DEVELOPMENTAL HISTORY :

GROSS MOTOR, VISION &FINE MOTOR, HEARING &LANGUAGE, SOCIAL

DEVELOPMENTAL AGE:

12) IMMUNIZATION:

13) SOCIOECONOMIC:

14) DIET HISTORY

15) ANTHROPOMETRY

a) WEIGHT IN KGS:

b) HEIGHT(CM):

c)HEAD CIRCUMFERENCE(CM)

d)MID-ARM CIRCUMFERENCE(CM)

ANTHROPOMETRY STATUS BASED ON WHO AND IAP GROWTH CHARTS

16)CLINICAL EXAMINATION

a) GENERAL PHYSICAL EXAMINATION

VITALS (TEMPERATURE, HR, RR, BP)

PALLOR, ICTERUS, CYANOSIS, CLUBBING, LYMPHADENOPATHY, EDEMA:

ANY NEUROCUTANEOUS MARKERS ON HEAD TO TOE:

b) SYSTEMIC EAXMINATION:

CVS:

RS:

CNS:

PA:

17) INVESTIGATIONS:

VITAMIN D :

CBC:

TC: DC: HB: PCV:

RBC: PLATELETS: MCV/MCH MCHC:

SERUM ELECTROLYTES:

SERUM CALCIUM:

SERUM POTASSIUM :

SERUM SODIUM:

ALP:

PHOSPHOROUS:

CRP:

CSF :

ADDITIONAL INVESTIGATIONS TO FIND FOCUS OF FEVER :

CXR:

URINE ROUTINE :

DIAGNOSIS:

SR NUM	IPD NUM	NAME	AGE (MONTHS)	SEX	DOA	DOD	TEMP	HR	FAMILY H/O EPILEPSY	FAMILY H/O FS	RR	EPISODE NUM	DURATION OF FEVER(DAYS)	DURATION OF SX (MINS)	TYPE	DEVELOPMENT	IMMUNISATION	TC	N/L	HB	PCV	PLATELETS	GRBS	NA	K	CAL	CRP	CATEGORY VITD	IT D	CAT(ALP)	ALP	CAT(PHOS)	PHOSPHORUS	CSF	UR	X RAY	RTI SYMPTOMS	GE SYPMTOMS
											24	1		1	6 GTCS	NORMAL	ACC TO AGE	6730	58/31	10.1	30.7	396000	92	131	4.1	9.2	14	INSUFFICENCY	12.42	NORMAL	122	NORMAL	4.2	NORMAL	NORMAL	NORMAL	PRESENT	ABSE
2	289495	VEDANSHA	24	MALE	27-06-2021	30-06-2021	100	102	ABSENT	ABSENT	32	1	1	5	5 GTCS	NORMAL	ACC TO AGE	10850	77/16	11.2	34	96000	89	138	4.5	10.8	6.2	SUFFICENCY	33	NORMAL	121	NORMAL	3	NORMAL	NORMAL	NORMAL	ABSENT	PRES
3	35560	AYUSHMAN PARNKAR	48	MALE	20-06-2021	23-06-2021	101.2	100	ABSENT	ABSENT	26	1	3	6	6 GTCS	NORMAL	ACC TO AGE	25320	75/18	10.3	32.1	329000	78	137	4.4	9.7	26.3	SUFFICENCY	33.23	NORMAL	118	NORMAL	2.7	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
4	80434	AARIZ INAMDAR	12	MALE	16-06-2021	18-06-2021	101	120	ABSENT	ABSENT	38	1	1	3	3 GTCS	NORMAL	ACC TO AGE	7630	63/30	9.2	29.7	304000	86	133	4.2	8.3	5	DEFICIENCY	11.02	NORMAL	124	NORMAL	3.9	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
5	162153	BHAGYASHREE	36	FEMALE	31-08-2021	02-09-2021	101.3	112	ABSENT	ABSENT	27	1	5	5	5 GTCS	NORMAL	ACC TO AGE	20280	62/40	9.6	25.5	337000	69	139	4.8	8.3	15	INSUFFICENCY	19.19	NORMAL	120	NORMAL	3.5	NORMAL	NORMAL	NORMAL	PRESENT	ABSE
6	141997	UTAVIKA SATISH NAIK	12	FEMALE	13-08-2021	14-08-2021	99.2	126	ABSENT	ABSENT	36	1	2	7	7 GTCS	NORMAL	ACC TO AGE	11450	79/13	10.7	32.9	227000	76	130	3.2	8.7	18	DEFICIENCY	10.29	NORMAL	120	NORMAL	4.2	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
7	152892	SIYANATH SHEIKH	36	MALE	22-08-2021	23-08-2021	99.6	102	ABSENT	ABSENT	34	2	1	5	5 GTCS	NORMAL	ACC TO AGE	7790	79/14	11.2	35.3	194000	87	134	3.9	9.3	14	DEFICIENCY	10.24	NORMAL	126	NORMAL	4.5	NORMAL	NORMAL	NORMAL	ABSENT	PRES
8	190075	SAMARTH RAMAPPA	48	MALE	24-09-2021	27-09-2021	102.2	98	ABSENT	ABSENTV	26	1	2	8	8 GTCS	NORMAL	ACC TO AGE	11330	89/8	15.7	47.3	132000	75	138	4.4	8.9	13	INSUFFICENCY	18.9	NORMAL	120	NORMAL	3.5	NORMAL	NORMAL	NORMAL	PRESENT	ABSE
9	187774	LALASAB BANSUDE	48	MALE	23-09-2021	27-09-2021	102	102	ABSENT	ABSENT	26	1	1	5	5 GTCS	NORMAL	ACC TO AGE	7960	68/25	12	35.8	138000	86	138	4	9.3	5	INSUFFICENCY	12.24	NORMAL	119	NORMAL	4.2	NORMAL	NORMAL	NORMAL	PRESENT	ABSE
10	179409	PRATEEK	48	MALE	15-09-2021	17-09-2021	101	100	ABSENT	ABSENT	28	1	1	3	3 GTCS	NORMAL	ACC TO AGE	14600	81/15	9.6	28.4	359000	95	132	4.2	8.3	5	SUFFICIENCY	26.53	NORMAL	127	NORMAL	3.7	NORMAL	NORMAL	NORMAL	ABSENT	PRES
11	178335	SUPRIYA	24	FEMALE	14-09-2021	18-09-2021	100	120	ABSENT	ABSENT	40	1	1	5	5 GTCS	NORMAL	ACC TO AGE	1580	69/24	10.7	35.2	306000	79	139	5.6	10.2	30	SUFFICIENCY	32.42	NORMAL	130	NORMAL	4.5	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
12	166943	KUSHI	24	FEMALE	04-09-2021	07-09-2021	100.1	112	ABSENT	ABSENT	38	1	1	6	6 GTCS	NORMAL	ACC TO AGE	5300	59/29	10.2	32.7	27800	86	136	4.8	9.3	10	SUFFICIENCY	32	NORMAL	126	NORMAL	3.4	NORMAL	NORMAL	NORMAL	ABSENT	PRES
13	164564	SHAYED PATEL FAROOQ	22	MALE	02-09-2021 5.9.21		100	98	ABSENT	ABSENT	34	1	3	3	3 GTCS	NORMAL	ACC TO AGE	17590	75/19	10.8	35.3	318000	96	141	4.5	8.9	18	SUFFICIENCY	34.22	NORMAL	122	NORMAL	3.8	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
14	175779	DEEPAK Y SHINDE	33	MALE	13-09-2021	16-09-2021	99.3	100	ABSENT	ABSENT	28	1	1	3	3 GTCS	NORMAL	ACC TO AGE	22060	78/14	11	34.2	485000	78	136	4.7	9.4	13	DEFICIENCY	10.24	NORMAL	124	NORMAL	3.1	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
15	182833	SHANVI RAJPUT	38	FEMALE	18-09-2021	19-09-2021	100.2	95	ABSENT	ABSENT	26	2	1	10	10 GTCS	NORMAL	ACC TO AGE	19110	94/4	10.2	32	312000	77	141	4.5	9	12	DEFICIENCY	11.29	NORMAL	123	NORMAL	3.4	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
16	213316	ANIRUDHA	24	MALE	13-10-2021	15-10-2021	100.2	89	ABSENT	ABSENT	28	2	2	3	3 GTCS	NORMAL	ACC TO AGE	7180	66/23	9.6	30.9	189000	68	137	4.4	8.6	10	INSUFFICENCY	19.72	NORMAL	130	NORMAL	3.9	NORMAL	NORMAL	NORMAL	PRESENT	ABSE
17	215609	PRETHAMGOUDA	36	MALE	15-10-2021	17-10-2021	100.1	78	ABSENT	ABSENT	34	1	2	2	2 GTCS	NORMAL	ACC TO AGE	3730	75/18	10.8	31.5	213000	89	135	4.2	8.9	15.5	SUFFICIENCY	32.42	NORMAL	122	NORMAL	3.7	NORMAL	NORMAL	NORMAL	PRESENT	ABSE
18	198849	AYUSH MAHESH	33	MALE	01-10-2021	04-10-2021	99.7	85	ABSENT	ABSENT	34	2	1	6	6 GTCS	NORMAL	ACC TO AGE	12140	83/12	11.5	35.9	342000	100	136	4.1	9.2	30	DEFICIENCY	11.2	NORMAL	124	NORMAL	4.5	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
19	256765	SANKET SHRISHAIL	34	MALE	17-11-2021	20-11-2021	100	78	ABSENT	ABSENT	38	2	1	5	5 GTCS	NORMAL	ACC TO AGE	5810	68/25	10.8	39	442000	98	138	5	9.7	14.3	INSUFFICENCY	13.09	NORMAL	122	NORMAL	3.3	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
20	277120	ANVI JITESH	9	FEMALE	02-12-2021	05-12-2021	101	100	ABSENT	ABSENT	38	1	1	4	4 GTCS	NORMAL	ACC TO AGE	6000	68/21	9.5	28.9	175000	100	140	4.2	9.1	14	INSUFFICENCY	13.9	NORMAL	136	NORMAL	3.6	NORMAL	NORMAL	NORMAL	ABSENT	PRES
21	282391	SWATHI BAILAPATTAR	12	FEMALE	05-12-2021	07-12-2021	101	89	ABSENT	ABSENT	35	1	1	4	4 GTCS	NORMAL	ACC TO AGE	13500	78/12	9.2	30.1	542000	120	135	4.2	10	15	INSUFFICENCY	13.9	NORMAL	129	NORMAL	3.9	NORMAL	NORMAL	NORMAL	ABSENT	PRES
22	10830	KASHYA TORPI	7	FEMALE	10-12-2021	13-12-2021	101	119	ABSENT	ABSENT	38	1	3	5	5 GTCS	NORMAL	ACC TO AGE	7170	43/37	11.8	36.7	347000	120	141	4.4	10.1	9.2	INSUFFICENCY	18.89	NORMAL	124	NORMAL	4.1	NORMAL	NORMAL	NORMAL	PRESENT	ABSE
23	294641	KAVERI NINGAYYA	12	FEMALE	13-12-2021	16-12-2021	101.2	110	ABSENT	ABSENT	34	1	4	5	5 GTCS	NORMAL	ACC TO AGE	21170	79/12	9.9	29	458000	89	139	4.1	9.7	18	SUFFICIENCY	34.12	NORMAL	124	NORMAL	3.9	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
24	3442	MAYURI	23	FEMALE	03-01-2022	05-01-2022	101.98		ABSENT	ABSENT	28	1		2.10	GTCS	NORMAL	ACC TO AGE	8920	57/33	10.7	32.6	467000	86	140	4.4	10	5	SUFFICIENCY	23.24	NORMAL	124	NORMAL	3.1	NORMAL	NORMAL	NORMAL	ABSENT	PRES
25	6090	ARADHYA	12	FEMALE	05-01-2022	08-01-2022	101	89	ABSENT	ABSENT	28	1	2	6	6 GTCS	NORMAL	ACC TO AGE	16480	79/14	10.2	34.7	311000	78	140	4.2	9.4	37	INSUFFICENCY	13.19	NORMAL	127	NORMAL	4	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
26	17776	UMERA INAMDAR	13	FEMALE	14-01-2022	16-01-2022	100	102	ABSENT	ABSENT	36	1	2	5	5 GTCS	NORMAL	ACC TO AGE	1810	61/31	8.9	29.8	530000	68	140	4.2	9.3	12	SUFFICIENCY	32	NORMAL	121	NORMAL	3.7	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
27	20101	SHREENIDI SHIVANAND	12	FEMALE	15-01-2022	17-01-2022	101	98	ABSENT	ABSENT	26	1	1	3	3 GTCS	NORMAL	ACC TO AGE	12280	74/20	10.8	33.1	347000	89	139	4.1	9.3	5	INSUFFICENCY	16.72	NORMAL	126	NORMAL	3.2	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
28	61060	ARUD S HEBBAL	14	MALE	17-02-2022	19-02-2022	102.1	115	ABSENT	ABSENT	38	1	1	2	2 GTCS	NORMAL	ACC TO AGE	10320	86/5	10.8	33.7	355000	84	141	3.8	9.5	10.5	INSUFFICENCY	12.37	NORMAL	125	NORMAL	3.7	NORMAL	NORMAL	NORMAL	PRESENT	ABSE
29	42908	RIDHA MD RIFIQ	7	MALE	02-02-2022	06-02-2022	101	120	ABSENT	ABSENT	38	1	2	5	5 GTCS	NORMAL	ACC TO AGE	10360	87/11	10.1	29.8	318000	78	134	4.5	9.6	15	INSUFFICENCY	12.6	NORMAL	120	NORMAL	3.8	NORMAL	NORMAL	NORMAL	ABSENT	ABSE
30	73113	WAGESH	45	MALE	26-02-2022	28-02-2022	101	98	ABSENT	ABSENT	26	2	1	4	4 GTCS	NORMAL	ACC TO AGE	21500	82/13	10.6	30.4	220000	68	134	3.6	9.1	5	SUFFICIENCY	28	NORMAL	124	NORMAL	3.7	NORMAL	NORMAL	NORMAL	PRESENT	ABSENT
31	65381	SAHANA RAJU MADAR	14	FEMALE	20-02-2022	23-02-2022	100	87	ABSENT	ABSENT	28	1	2	5	5 GTCS	NORMAL	ACC TO AGE	8430	65/27	8.2	26.4	459000	78	140	3.8	9.3	6.1	SUFFICIENCY	42.32	NORMAL	119	NORMAL	3.9	NORMAL	NORMAL	NORMAL	ABSENT	PRESENT
32	90783	ATIKSHA GALASARGI	13	FEMALE	14-03-2022	16-03-2022	101	98	ABSENT	ABSENT	30	1	2	5	5 GTCS	NORMAL	ACC TO AGE	14180	45/44	7.6	27.9	314000	68	139	3.3	9.2	5.2	INSUFFICENCY	13.02	NORMAL	120	NORMAL	3.8	NORMAL	NORMAL	NORMAL	PRESENT	ABSENT
33	83021	SWARA	38	FEMALE	07-03-2022	10-03-2022	100.2	89	ABSENT	ABSENT	26	1	3	4	4 GTCS	NORMAL	ACC TO AGE	16630	77/19	12.2	36	416000	75	139	4	9.8	36	INSUFFICENCY	12.73	NORMAL	130	NORMAL	4	NORMAL	NORMAL	NORMAL	ABSENT	PRESENT
34	129402	PREMA GANGADHAR	36	FEMALE	17-04-2022	19-04-2022	101	70	ABSENT	ABSENT	24	1	2	2	2 GTCS	NORMAL	ACC TO AGE	9560	78/16	11.8	37.8	361000	89	139	4.3	9	24	INSUFFICENCY	14.57	NORMAL	127	NORMAL	3.5	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
35	129393	MARYAM	8	FEMALE	17-04-2022	18-04-2022	101	110	ABSENT	ABSENT	36	1	2	3	3 GTCS	NORMAL	ACC TO AGE	6100	48/46	9.5	30.1	318000	67	131	4.6	9.7	5	INSUFFICENCY	15.92	NORMAL	126	NORMAL	3.6	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
36	121921	ANJALI VITTAL	12	FEMALE	10-04-2022	12-04-2022	100.6	106	ABSENT	ABSENT	34	1	1	5	5 GTCS	NORMAL	ACC TO AGE	3320	40/49	9.9	33.1	335000	60	135	3.8	8.2	20	INSUFFICENCY	16.72	NORMAL	123	NORMAL	3.2	NORMAL	NORMAL	NORMAL	PRESENT	ABSENT
37	124095	BIBIMARIAM	35	FEMALE	11-04-2022	14-04-2022	101.3	78	ABSENT	ABSENT	24	1	1	8	8 GTCS	NORMAL	ACC TO AGE	15200	80/16	12.3	41.1	411000	84	149	3.3	8.7	5	SUFFICIENCY	28.2	NORMAL	126	NORMAL	4.4	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
38	173236	AMBRESH	12	MALE	22-05-2022	24-05-2022	100.6	89	ABSENT	ABSENT	32	1	2	4	4 GTCS	NORMAL	ACC TO AGE	19470	79/15	9	28	423000	90	132	4.5	9.1	5.9	INSUFFICENCY	13.42	NORMAL	118	NORMAL	2.9	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
39	182551	MUTTU	24	MALE	29-05-2022	31-05-2022	102.2	100	ABSENT	ABSENT	36	1	1	6	6 GTCS	NORMAL	ACC TO AGE	1850	78/17	10.5	33.7	456000	87	135	4.6	9.4	23.7	INSUFFICENCY	12.42	NORMAL	120	NORMAL	3.5	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
40	174655	CHRANJEEVI	12	MALE	23-05-2022	25-05-2022	99.9	96	ABSENT	ABSENT	25	1	1	4	4 GTCS	NORMAL	ACC TO AGE	6420	69/25	10.5	32.5	206000	67	142	4	9	9.3	INSUFFICENCY	14.72	NORMAL	120	NORMAL	3.2	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
41	172684	SAHITHYA	24	FEMALE	22-05-2022	24-05-2022	100.2	89	ABSENT	ABSENT	28	1	1	9	9 GTCS	NORMAL	ACC TO AGE	18420	52/43	11.3	31.9	286000	92	136	4	9.2	24	INSUFFICENCY	14.48	NORMAL	120	NORMAL	3.1	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
42	151769	GANGADEEP J	38	MALE	05-05-2022	07-05-2022	100.2	98	ABSENT	ABSENT	26	2	1	5	5 GTCS	NORMAL	ACC TO AGE	14700	84/8	9	25.8	291000	60	133	3.8	8.5	9.6	INSUFFICENCY	19.04	NORMAL	124	NORMAL	4.1	NORMAL	NORMAL	NORMAL	ABSENT	PRESENT
43	26496	ARJUN BARANAL	12	MALE	31-05-2022 3/6/22		100.2	78	ABSENT	ABSENT	23	1	1	5	5 GTCS	NORMAL	ACC TO AGE	16920	89/9	7.2	23.5	552000	78	134	3.9	8.8	7.3	INSUFFICENCY	15.92	NORMAL	126	NORMAL	3.5	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
44	156713	KRITI H PATIL	24	FEMALE	10-05-2022	13-05-2022	101	98	ABSENT	ABSENT	24	1	2	4	4 GTCS	NORMAL	ACC TO AGE	26800	53/41	12.2	34.5	212000	98	130	3.6	7.9	16	SUFFICIENCY	41	NORMAL	120	NORMAL	3	NORMAL	NORMAL	NORMAL	ABSENT	PRESENT
45	179057	SAMAIRA AMMENSAB	48	FEMALE	26-05-2022	01-06-2022	100.3	89	ABSENT	ABSENT	30	1	2	5	5 GTCS	NORMAL	ACC TO AGE	26890	59/32	8.6	25.8	310000	83	139	4.5	9.8	9	DEFICIENCY	11.2	NORMAL	124	NORMAL	4.1	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
46	210978	AMAIRA AMEERPASHA	36	FEMALE	20-06-2022	23-06-2022	99.2	79	ABSENT	ABSENT	23	1	1	6	6 GTCS	NORMAL	ACC TO AGE	9520	86/8	12.3	36	212000	85	135	4.2	8.2	20	INSUFFICENCY	14.67	NORMAL	120	NORMAL	3.7	NORMAL	NORMAL	NORMAL	PRESENT	ABSENT
47	215363	KASHIM NADAF	11	MALE	24-06-2022	25-06-2022	99.9	113	ABSENT	ABSENT	38	1	1	4	4 GTCS	NORMAL	ACC TO AGE	12640	54/42	10	32.2	397000	90	137	5	9.1	8.7	INSUFFICENCY	15.96	NORMAL	125	NORMAL	4.5	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
48	215330	SONU BHIMASHANKAR	60	MALE	23-06-2022	25-06-2022	100.6	86	ABSENT	ABSENT	24	1	1	4	4 GTCS	NORMAL	ACC TO AGE	16100	77/20	11.5	35.7	405000	100	139	4.6	8.9	8.8	INSUFFICENCY	20	NORMAL	130	NORMAL	3.9	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
49	215329	KRISH APPANASAHEB	24	MALE	23-06-2022	25-06-2022	99.8	100	ABSENT	ABSENT	30	2	1	5	5 GTCS	NORMAL	ACC TO AGE	19730	82/16	11.6	36.3	452000	122	142	4.7	10.4	16	INSUFFICENCY	18.29	NORMAL	129	NORMAL	4.2	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
50	218818	SATVIK RATHOD	12	MALE	26-06-2022	28-06-2022	99.6	86	ABSENT	ABSENT	25	1	1	6	6 GTCS	NORMAL	ACC TO AGE	10490	51/40	11.1	34.2	175000	109	140	3.9	8.3	5	SUFFICIENCY	24.5	NORMAL	124	NORMAL	3.5	NORMAL	NORMAL	NORMAL	PRESENT	ABSENT
51	208625	SAMPATH MARIWALAR	32	MALE	18-06-2022	20-06-2022	102	102	ABSENT	ABSENT	28	1	1	5	5 GTCS	NORMAL	ACC TO AGE	4380	65/29	9.6	31	226000	89	130	3.6	8.1	10	DEFICIENCY	11.09	NORMAL	130	NORMAL	4.2	NORMAL	NORMAL	NORMAL	PRESENT	ABSENT
52	209450	PREETHAM POLICEPATIL	6	MALE	19-06-2022	24-06-2022	99.8	114	ABSENT	ABSENT	36	1	1	6	6 GTCS	NORMAL	ACC TO AGE	7270	80/20	12.4	40.2	245000	68	132	4.6	9.6	22	SUFFICIENCY	33.93	NORMAL	124	HIGH	4.9	NORMAL	NORMAL	NORMAL	PRESENT	ABSENT
53	231342	KISHORE HUNASANGI	10	MALE	05-07-2022	08-07-2022	101.2	120	ABSENT	ABSENT	38	1	1	10	10 GTCS	NORMAL	ACC TO AGE	13120	50/40	11.7	35.1	423000	125	135	4	9	10.1	INSUFFICENCY	14.43	NORMAL	122	NORMAL	3	NORMAL	NORMAL	NORMAL	ABSENT	PRESENT
54	245921	ANJANAYA KATTIMANI	8	MALE	16-07-2022	18-07-2022	100.6	108	ABSENT	ABSENT	32	1	1	5	5 GTCS	NORMAL	ACC TO AGE	17280	60/34	9.1	24.9	522000	110	134	4.7	9.7	30	INSUFFICENCY	13.51	NORMAL	125	NORMAL	3.6	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
55	274336	JAKSHINI GALAGALI	24	FEMALE	08-08-2022	11-08-2022	101.5	96	ABSENT	ABSENT	28	1	4	4	4 GTCS	NORMAL	ACC TO AGE	20260	75/20	12.4	37	404000	100	135	4	9.4	7.4	INSUFFICENCY	14.52	NORMAL	132	NORMAL	3	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
56	268487	SHREYA MEDEGALE	11	FEMALE	04-08-2022	09-08-2022	100.8	110	ABSENT	ABSENT	32	1	2	10	10 GTCS	NORMAL	ACC TO AGE	6890	63/30	11.2	31.5	142000	124	132	4.3	8	10	INSUFFICENCY	16.82	NORMAL	124	NORMAL	3.4	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
57	268493	MAHADEVAPPAGOUDA	12	MALE	04-08-2022	05-08-2022	100.9	99	ABSENT	ABSENT	28	1	1	3	3 GTCS	NORMAL	ACC TO AGE	18910	55/38	11	32	469000	86	134	4.5	10	17	INSUFFICENCY	16.72	NORMAL	130	NORMAL	3.8	NORMAL	NORMAL	NORMAL	PRESENT	ABSENT
58	280095	SHREEVARDHAN JADAV	12	MALE	13-08-2022	16-08-2022	101	90	ABSENT	ABSENT	26	1	1	2	2 GTCS	NORMAL	ACC TO AGE	12080	53/42	12	32.5	298000	84	135	4.5	8.5	5	INSUFFICENCY	13.5	NORMAL	203	HIGH	7.4	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
59	269868	SAKSHI	11	FEMALE	04-08-2022	06-08-2022	100.4	102	ABSENT	ABSENT	32	1	1	3	3 GTCS	NORMAL	ACC TO AGE	13800	58/38	12.7	37	331000	98	132	3.8	9.4	11	INSUFFICENCY	14.92	NORMAL	124	NORMAL	4.5	NORMAL	NORMAL	NORMAL	PRESENT	ABSENT
60	264544	AZAN SAYYAD	12	MALE	01-08-2022	07-08-2022	99.6	89	ABSENT	ABSENT	24	1	1	4	4 GTCS	NORMAL	ACC TO AGE	10500	66/28	8.3	25.7	135000	87	136	4.4	8.6	12	DEFICIENCY	10.92	NORMAL	128	NORMAL	3.9	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT
61	267088	ARNA GAJANAND	48	FEMALE	03-08-2022	08-08-2022	100.2	96	ABSENT	ABSENT	28	1	1	2	2 GTCS	NORMAL	ACC TO AGE	11920	71/25	11.1	32.3	214000	98	132	3.8	9.4	11.8	SUFFICIENCY	35.95	NORMAL	218	NORMAL	3.9	NORMAL	NORMAL	NORMAL	ABSENT	ABSENT