

# Iron-Deficiency Anemia as a Risk Factor for the First Simple Febrile Convulsion

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## Abstract

**Background:** Febrile seizure is the most common convulsive disorder in childhood. The role of iron in the metabolism of neurotransmitters and carrying oxygen to the brain suggests the possibility of a relationship between iron-deficiency anemia and febrile seizures. **Objectives:** The aim of study is to study the role of iron-deficiency anemia as a risk factor for febrile seizures. **Materials and Methods:** This is a hospital-based case-control study performed in 30 children with febrile seizures (case) and 80 febrile children without seizures (control) who were aged between 6 and 60 months. The groups were matched in terms of age, gender, and the use of supplemental iron. Laboratory tests consisted of complete blood count, serum iron, plasma ferritin were done for each patient. **Results:** Thirty-four percent of the cases with febrile seizure had iron-deficiency anemia compared with 20% of children with febrile illness without seizure and 12% of healthy children. The odds ratio for iron-deficiency anemia in the patients with febrile seizure was 3.059 (95% confidence interval (CI) = 0.969–9.657) compared with the febrile children without convulsion and 5.608 (95% CI = 1.834–17.146) compared with the healthy group. **Conclusion:** Based on the result of this study, iron deficiency could be an important risk factor for the development of febrile convulsion. This encourages the evaluation of iron status in children with febrile seizure.

**Keywords:** Convulsion, iron-deficiency anemia, risk factor

## INTRODUCTION

Febrile seizures (FSs) are the commonest cause of seizures in children, occurring in 2%–5% of children<sup>[1]</sup> aged between 6 months and 5 years. FSs are a relatively common childhood condition referring to a child having a seizure when he/she has a high temperature of 38°C or above by rectal measurement<sup>[2]</sup> with no previous history of FSs or without acute systemic metabolic abnormality that may produce convulsions or nervous system infection.<sup>[3]</sup> The exact cause of febrile convulsion is unknown, but genetic and environmental factors have influence on its occurrence. A simple febrile seizure (FS) is a primary, generalized, usually tonic-clonic, attack associated with fever, lasting for a maximum of 15 minutes and not recurrent within a 24-hour period. A complex FS is more prolonged (>15 minutes), is focal, and/or recurs within 24 hours. Febrile status epilepticus is a FS lasting >30 minutes.<sup>[4]</sup> Iron deficiency is the most common nutritional deficiency and hematological disease of infancy and childhood<sup>[5]</sup> especially between 6 and 24 months of age.<sup>[6]</sup> Iron is a nutritional element not only

needed for the synthesis of hemoglobin but also essential for enzymes involved in neurochemical reactions<sup>[5]</sup> such as myelin formation,<sup>[7]</sup> brain energy metabolism,<sup>[8]</sup> and some neurotransmitters and enzymes metabolism such as monoaminoxidase and aldehyde oxidase.<sup>[9]</sup> Neurological symptoms such as poor attention span, learning deficits, weak memory, delayed motor development, and behavioral disturbance caused by iron deficiency are well known.<sup>[9]</sup> Despite these facts, some studies show that antipyretics do not affect the prevention of seizures caused by fever.<sup>[10]</sup> So other variables that affect this process have special circumstances, and considering that fever can intensify the anemia symptoms by increasing body metabolism,<sup>[9]</sup> there is a need to explore relation between FS and iron-deficiency

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anemia. The aim of study is to study the role of iron-deficiency anemia as a risk factor for FSs.

## MATERIALS AND METHODS

This study has been carried out in Babil Teaching Hospital of Maternity and Children from April 1 to July 1, 2014. This hospital received 6834 inpatient, 2670 outpatients, and 9460 patients in emergency room. In addition, 126 referral cases have been received from other hospitals in province. A hospital-based case-control study has been carried to determine the risk of iron-deficiency anemia among study groups (febrile convulsion, febrile without convulsion, as well as healthy group). Three groups have been collected from the hospital: the first group was febrile convulsion patients, and the second group was febrile patients without convulsion. In addition, healthy group without fever or iron-deficiency anemia has been collected from other hospital wards. Thirty patients with FS admitted to the emergency unit and wards of hospital. Their ages ranged from 6 months to 5 years. They had temperature of 38°C or more without a history of previous seizure attacks. Control group included 80 children (6 months to 5 years of age) and had visited Babil Teaching Hospital during the same period of the study; they had a temperature of 38°C or more without a history of seizure attacks. They attended outpatient clinic and emergency room for upper respiratory tract infection, gastroenteritis, urinary tract infection, or nonspecific causes of fever. For each child with febrile convulsion, two children were selected in the control groups: one with a similar febrile illness, who was admitted to the hospital at about the same time as the subject, but did not show seizure, and another was a healthy child. Children of the control groups were selected using an objective-based method and matched with the cases in terms of age (with a maximum difference of 1 month), gender, and the place of residence (urban or rural).

For patients suffering from febrile convulsions, aged less than 1 year, lumbar puncture has been done to exclude patients with central nervous system (CNS) infection. For the patients aged more than a year, lumbar puncture has been done according to the clinical signs symptoms.

After making sure that the participants had not taken iron supplement in the last 3 days, a 5mL blood sample was taken and sent to the laboratory. The blood tests of complete blood count (CBC) difference, mean corpuscular hemoglobin (MCH), mean corpuscular volume (MCV), iron, and serum ferritin all these investigations were done. Serum ferritin levels were evaluated by using multidevices, VIDAS Ferritin device made in France (bioMerieux) model 2009, which is a automated test for the determination of human ferritin using enzyme-linked fluorescent assay technique by ferritin kit (for measuring ferritin): 100mL of blood introduced into the device and centrifuged; this process lasts for 2 hours. CBC (Hb concentration, hematocrit

[HCT], MCH, MCV) was done by Ruby device made in USA (Abbott Diagnostic Device, model 2006), which need 150mL of blood and takes 5 minutes for the result. S. iron is measured by Spectrophotometer (Cecil) device made in France, it takes 500mL of clotted blood and then centrifuges it, and this process takes 2 hours. All the blood tests were performed by a laboratory technician who was not informed of the purpose of the study. The diagnosis of anemia was made by a pediatrician who studied the results of tests without knowing the groups to which each of the test results belonged. For children aged 6 months to 2 years, iron deficiency anemia (IDA) was defined as Hb < 10.5g/dL, HCT < 33%, MCV < 70 fL, MCH < 23 pg. For children aged 2–5 years, IDA was defined as Hb < 11.5g/dL, HCT < 34%, MCV < 75 fL, MCH < 24 pg. MCH, MCV, and peripheral blood smear were used to rule out other causes of anemia. The normal level of serum iron was determined as Fe > 40 µg/dL for children younger than 1 year, and Fe > 50 µg/dL for children over 1 year of age. The normal range of ferritin established was more than 12ng/dL for healthy children and more than 30ng/dL for children with infection.

## Classification of cases

According to the type of fit, the cases of febrile convulsions divided into two types (simple and complex).

A case was considered as a complex if one or more of the following criteria were present:

1. Seizure duration is >15 minutes
2. Repeated seizures occur within 24 hours
3. Focal seizure activity or focal findings are present.

## Exclusion criteria

The following are the exclusion criteria: a history of any afebrile seizure, a history of neurodevelopmental delay, an age below 6 months and above 5 years, cases of CNS infection. Also, patients with following clinical features were excluded: lethargy, irritability, vomiting, nuchal rigidity, bulging fontanel headache, drowsiness, toxicity, and coma, and/or positive laboratory tests. Finally, cases of complex febrile convulsion were excluded from the study.

## Statistical analysis

Statistical analysis was done using SPSS program version 20. Continuous data were presented as mean and standard deviation. Meanwhile, categorical data were presented as frequency and percentage. Pearson's chi square was used to find the association between categorical variables. One-way analysis of variance was used to compare between more than two groups. A *P* value of less than or equal to 0.05 was considered as statistically significant.

## Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of

Helsinki. The study was carried out with patients' verbal and analytical approval before a sample was taken. The study protocol, subject information, and consent form were reviewed and approved by a local ethics committee according to the document number 255 on January 5, 2014 to get this approval.

## RESULTS

In the case of the personal characteristics, no significant differences were found among the groups in terms of age, gender, the place of residence, and the type nutrition during infancy. However, there were significant differences between the study groups regarding the family history of convulsion. Table 1 shows that the means of Hb, HCT, and serum iron were significantly different among the groups. However, no significant differences were found among the groups in terms of MCV and MCH. Table 2 shows that the febrile convulsion group and the group of

fever without convulsion had no significant difference in terms of the disease causing fever, but body temperatures were significantly different at the time of admission. In the febrile convulsion group, the ratio of boys to girls was nearly 2:1. According to the parameters for the diagnosis of ID, for children aged 6 months to 2 years, IDA was defined as Hb < 10.5 g/dL, HCT < 33%, MCV < 70 fL, MCH < 23 pg, and for children aged 2–5 years, IDA was defined as Hb < 11.5 g/dL, HCT < 34%, MCV < 75 fL, MCH < 24 pg. Table 3 shows that the presence of IDA was 43.3% in the convulsion group, 20% in the group with fever without convulsion, and 12% in the healthy group. Table 4 shows that odds ratio of IDA for the febrile convulsion group was nearly three times that of the febrile group and slightly more than five times higher than that of the healthy group as shown in Figures 1-5 the distribution of patients according to the gender, residence, history of convulsion, infant nutrition, and infection.

**Table 1: Mean differences of different indices of iron-deficiency anemia by three groups (healthy, febrile without convulsion, and febrile convulsion)**

| Variable           | Healthy       | Febrile without convulsion | Febrile convulsion | P      |
|--------------------|---------------|----------------------------|--------------------|--------|
| Hb                 | 12.72 ± 1.42  | 12.71 ± 1.41               | 11.65 ± 2.02       | 0.010* |
| MCV                | 75.60 ± 7.57  | 74.39 ± 5.43               | 72.83 ± 8.08       | 0.254  |
| MCH                | 27.26 ± 5.99  | 25.33 ± 3.75               | 27.31 ± 6.00       | 0.257  |
| Serum iron         | 53.10 ± 11.78 | 50.37 ± 12.65              | 44.13 ± 15.20      | 0.014* |
| Serum ferritin     | 18.46 ± 7.88  | 17.87 ± 4.93               | 17.40 ± 1.38       | 0.8    |
| Packed cell volume | 36.13 ± 4.47  | 36.26 ± 4.75               | 33.60 ± 5.43       | 0.048* |

\*P ≤ 0.05 (significant)

**Table 2: Association of study groups with children personal information**

| Variable                     | Study groups  |                                |                        | P      |
|------------------------------|---------------|--------------------------------|------------------------|--------|
|                              | Healthy (%)   | Febrile without convulsion (%) | Febrile convulsion (%) |        |
| Gender                       |               |                                |                        |        |
| Male                         | 27 (54.0)     | 20 (66.7)                      | 19 (63.3)              | 0.486  |
| Female                       | 23 (46.0)     | 10 (33.3)                      | 11 (36.7)              |        |
| Residence                    |               |                                |                        |        |
| Urban area                   | 32 (64.0)     | 13 (43.3)                      | 16 (53.3)              | 0.190  |
| Rural area                   | 18 (36.0)     | 17 (56.7)                      | 14 (46.7)              |        |
| Family history of convulsion |               |                                |                        |        |
| Yes                          | 2 (4.0)       | 10 (33.3)                      | 7 (23.3)               | 0.002* |
| No                           | 48 (96.0)     | 20 (66.7)                      | 23 (76.7)              |        |
| Infancy nutrition            |               |                                |                        |        |
| Breastfeeding                | 15 (30.0)     | 15 (50.0)                      | 12 (40.0)              | 0.377  |
| Formula feeding              | 11 (22.0)     | 6 (20.0)                       | 8 (26.7)               |        |
| Both                         | 24 (48.0)     | 9 (30.0)                       | 10 (33.3)              |        |
| Infectious diseases          |               |                                |                        |        |
| Respiratory                  |               | 15 (50.0)                      | 16 (53.3)              | 0.819  |
| Gastrointestinal tract       |               | 13 (43.3)                      | 11 (36.7)              |        |
| Others                       |               | 2 (6.7)                        | 3 (10.0)               |        |
| Age                          | 25.96 ± 15.31 | 23.60 ± 15.69                  | 24.27 ± 14.18          | 0.772  |
| Temperature                  |               | 38.5 ± 0.50                    | 38.8 ± 0.45            | 0.867  |

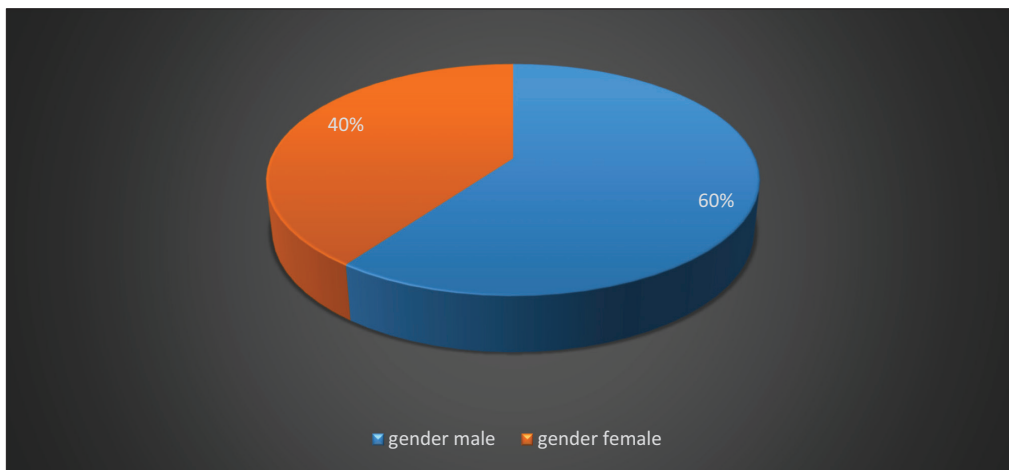
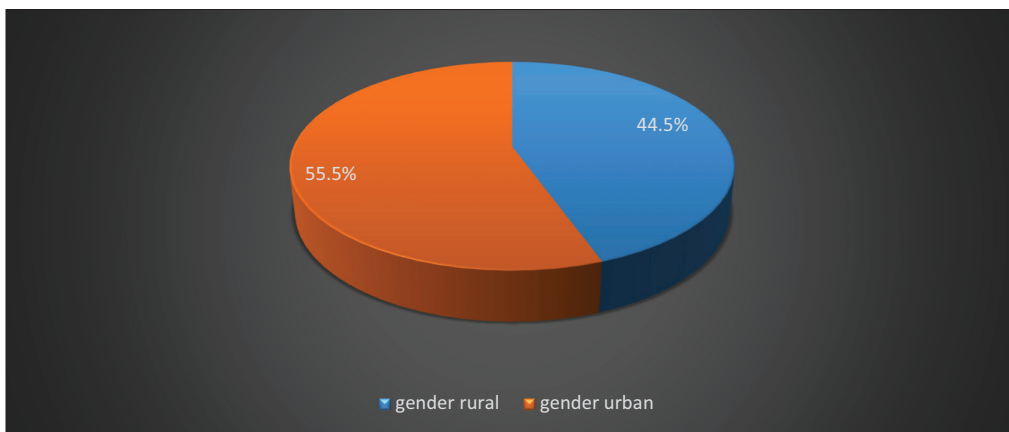
\*P ≤ 0.05 (significant)

**Table 3: Association of study groups with iron-deficiency anemia**

| Iron-deficiency anemia groups | Positive (%) | Negative (%) | P      |
|-------------------------------|--------------|--------------|--------|
| Healthy (control)             | 6 (24.0)     | 44 (51.8)    | 0.005* |
| Febrile without convulsion    | 6 (24.0)     | 24 (28.2)    |        |
| Febrile convulsion            | 13 (52.0)    | 17 (20.0)    |        |
| Total                         | 25 (100.0)   | 85 (100.0)   |        |

\* $P \leq 0.05$  (significant)**Table 4: Comparison of iron-deficiency anemia by study groups**

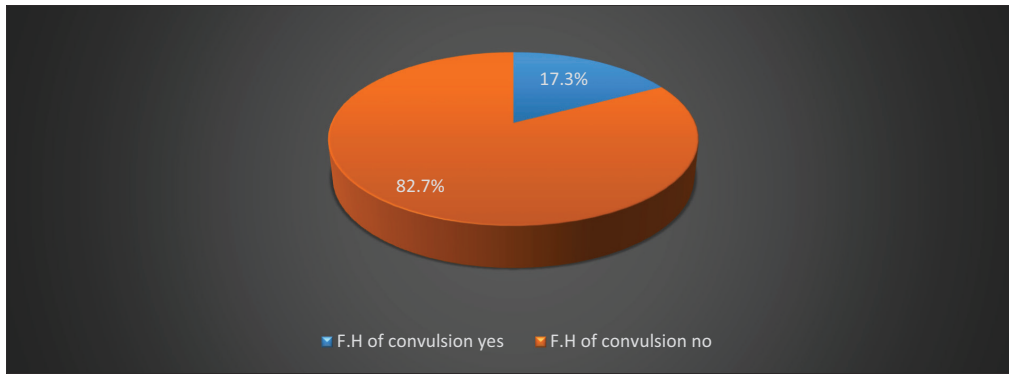
| Compared groups                                   | Odds ratio | 95% confidence interval | P      |
|---------------------------------------------------|------------|-------------------------|--------|
| Febrile convulsion and febrile without convulsion | 3.059      | 0.969–9.657             | 0.050* |
| Febrile convulsion and healthy                    | 5.608      | 1.834–17.146            | 0.001* |
| Febrile without convulsion and healthy            | 1.833      | 0.533–6.310             | 0.941  |

\* $P \leq 0.05$  (significant)**Figure 1:** Distribution of patients and control by gender**Figure 2:** Distribution of patients and control by residence

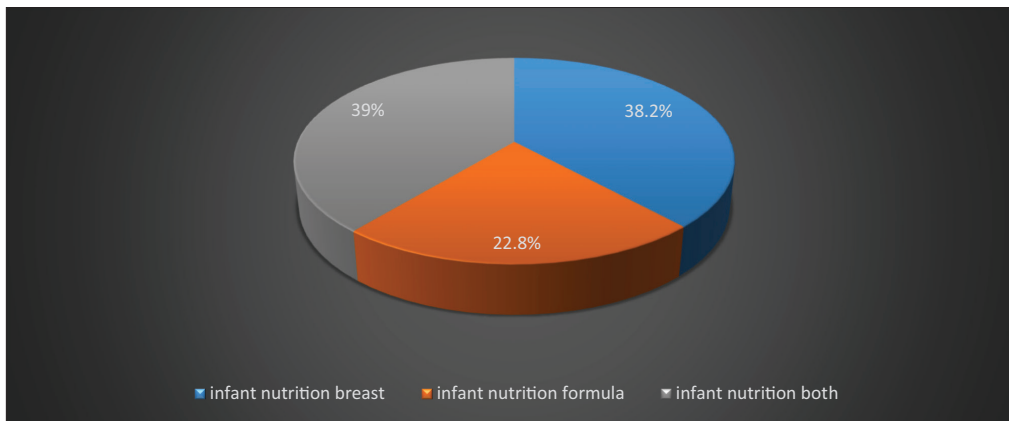
## DISCUSSION

Iron deficiency can alter brain synaptic neurotransmitters. The increase of glutamate excitatory neurotransmitters, decrease of GABA inhibitory neurotransmitters, decrease of monoamines, and hypoxemia from iron-deficiency anemia may be responsible for the induction of seizure

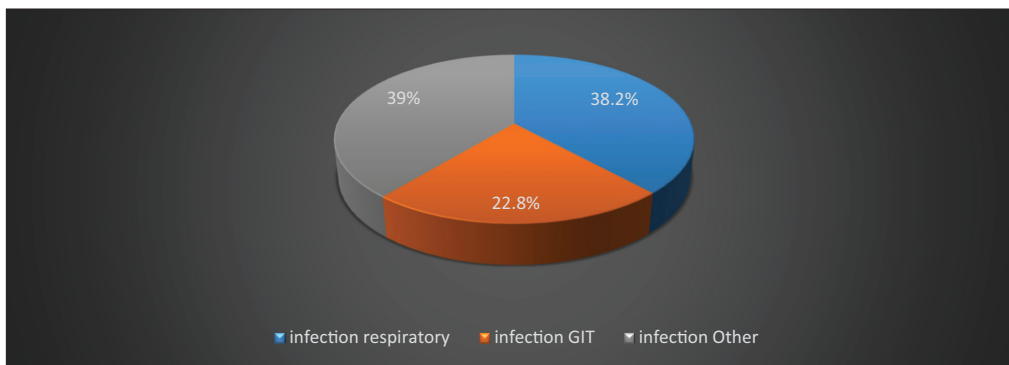
due to iron deficiency.<sup>[11]</sup> Clinically neurological symptoms such as poor attention span, learning deficits, poor memory, delayed motor development, and behavioral changes caused by iron deficiency are well known.<sup>[12]</sup> Its association with FSs was first observed and published in mid 90's in an Italian study.<sup>[13]</sup> This was followed by few



**Figure 3:** Distribution of patients and control by family history of convulsion



**Figure 4:** Distribution of patients and healthy by infant nutrition



**Figure 5:** Distribution of patients by infection

more international studies.<sup>[14]</sup> In 2009, Hartfield *et al.*, from the University of Alberta, Canada reported in a retrospective study that children with FSs were twice as likely to have iron deficiency as those with febrile illness alone. Some international studies denied any role of iron insufficiency in FSs. In fact, in an Iranian study, Bidabadi and Moushaf from the University of Guilan concluded that iron deficiency is less frequent in children with the first FS.<sup>[15]</sup> In 2001, Naveed-ur-Rehman and Billoo conducted the only local study at the Aga Khan University Hospital that convincingly associated iron-deficiency anemia as a risk factor for the FSs.<sup>[16]</sup> In this

study, the odds ratio of IDA for the febrile convulsion group was nearly three times that of the febrile group and slightly more than five times higher than that of the healthy group, namely, the incidence of IDA in the febrile convulsion group was significantly higher than in the other two control groups. Similar to our study, Pisacane *et al.* reported that anemia in their case group (30%) was higher than in hospital control group (14%) and healthy group (12%).<sup>[17]</sup> Also, in a study by Vaswani *et al.* from Mumbai in India, 68% of cases were iron deficient compared with 30% of controls.<sup>[18]</sup> Dawn *et al.*<sup>[19]</sup> also found similar results with children with FSs almost twice likely to have

iron deficiency compared to controls. In our study, three criteria, i.e., hemoglobin, S. iron, serum ferritin and the S. iron, hemoglobin were significantly different among cases and controls, for diagnosis of iron deficiency anemia. There was no significant difference in plasma ferritin. Serum ferritin, a nonspecific acute phase reactant, can rise in any inflammatory conditions, although both cases and controls were having fever at the time of enrollment. It is indeed evident that ferritin is an acute phase-reactive substance in nonspecific response to any febrile disease.<sup>[15]</sup> This can be confirmed by the higher plasma ferritin levels in the patient groups than in the healthy group, and fever can cause the lack of difference in ferritin levels between the two patient groups. In any case, the use of plasma ferritin cannot simply be an efficient criterion for the diagnosis of ID in febrile children. Regarding the comparison of multiple blood indices (Hb%, MCV, MCH, S. iron) between cases and control, the result of Hb%, HCT, MCV, MCH was lower in the cases than in the control group, but there was significant difference seen in the serum iron level, Hb, and HCT ( $P = 0.014$ ,  $0.010$ , and  $0.048$ , respectively). Hartfield *et al.*<sup>[14]</sup> conducted a retrospective case-control study in Canada and determined iron status using MCV, and hemoglobin observed similar results. In the study, the sex distribution shows that the male (60%) cases were more than female (40%), and this agrees with Al-Atrushi's study,<sup>[20]</sup> which shows 59.8% boy and 40.2% girl. In our study, the mean age of all studied children was  $24.27 \pm 14.18$  months. This is in nearly agreement with Bidabadi and Moushaf,<sup>[15]</sup> which shows it to be  $22.86 \pm 12.86$ . This indicates the strong relationship between age and febrile convulsion so that the most of FS occurs between 6 months and 3 years of age. Regarding genetic factors, this study had shown that positive family history of febrile convulsion was statistically significant risk factor for developing febrile convulsion; the same result was reported by many studies done by Anne *et al.* in the USA,<sup>[21]</sup> Murat *et al.* in Turkey,<sup>[22]</sup> Talebian and Honarpisheh in Kashan, Iran,<sup>[23]</sup> Vestegaard *et al.* in Denmark,<sup>[24]</sup> Huang in southern Taiwan,<sup>[25]</sup> and Ghasemi *et al.* in Iran.<sup>[26]</sup> In this study, the positive family history of FS was higher among cases than controls (62% vs. 6%); this was in agreement with Al-Atrushi's<sup>[20]</sup> study and also agrees with Mahyar *et al.*,<sup>[27]</sup> which reveal that 55% vs. 6.3% of cases and control, respectively, had positive family history of febrile convulsion. Fever can potentiate the effects of anemia or ID on the brain and therefore cause convulsions. In addition, anemia can be associated with the severity of febrile disease, and patients with more severe symptoms may be affected by convulsions. However, febrile convulsion usually occurs at the onset of a febrile disease, before the reduction of Hb due to the infectious disease.<sup>[17]</sup> In a study conducted in Thailand, the rate of Thalassemia children with febrile convulsion reported as being 4.4 times less than the general population of children. The researchers suggested

that it might be due to higher levels and the role of iron in brain metabolism, which leads to the reduced occurrence of febrile convulsion in those children.<sup>[28]</sup> Regarding fever, its peak, duration, and the onset of fit after fever were not found related to the type of FS. The causes of fever were studied; it was found that the URTIs were the most common cause of fever; this is similar to the result that was reported by Tahir in Pakistan.<sup>[29]</sup> This may reflect that these infections are one of the common diseases in our country, especially the first period of the study included the time at which respiratory tract infections were more. In the present study, 53.3% had respiratory tract infection (RTI), 36.7% had acute gastroenteritis, and 10% had other infections as UTI; this is in agreement with Chan *et al.*<sup>[30]</sup> The mechanisms proposed include the generation of fever, the degree of fever, or an elevated/inappropriate response to cytokines generated in association with infection.<sup>[31]</sup>

## CONCLUSION

Depending on the results of this study, iron-deficiency anemia seems to be a significant risk factor for the development of febrile convulsion. This encourages the evaluation of iron status in children with FS.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

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