INTRODUCTION

Febrile convulsions are the most common form of childhood convulsion that occurs in 2-5% of them, which represents the most common childhood convulsion disorder and occurs only in association with an elevated temperature. Evidence suggests, however, that they have little connection to cognitive function, so the prognosis for normal neurologic function is excellent with children with febrile convulsions (Swaiman, 2006). In the United States and Western Europe, they occur in 2–4% of all children; in Japan, however, 9–10% of all children experience febrile convulsions, and rates as high as 14% have been reported from the Mariana Islands in Guam (Shinnar, 2002). Febrile convulsions occur in young children at a time in their development when the convulsion threshold is low. This is a time when young children are susceptible to frequent childhood infections such as upper respiratory infections and viral syndromes, and they respond with comparably higher temperatures (Landreau-
Mascaro, 2002). Animal studies suggest a possible role for endogenous pyrogens, such as interleukin-1 beta, that, by increasing neuronal excitability, may link fever and convulsion activity (Matsuo, 2006). Viral illnesses are the predominant cause of febrile convulsions. Recent literature documented the presence of human herpes simplex virus 6 (HHSV-6) as the etiologic agent in roseola in about 20% of a group of patients presenting with their first febrile convulsions. Shigella gastroenteritis has also been associated with febrile convulsions (Millichap, 2006). Febrile convulsions tend to occur in families. In a child with febrile convulsions, the risk of febrile convulsions for the sibling is 10%, and it is almost 50% for the sibling if a parent has febrile convulsions as well. Although clear evidence exists for a genetic basis of febrile convulsions, the mode of inheritance is unclear (Audenaert, 2006). Risk factors for febrile convulsions are as follows: family history of febrile convulsions, High temperature, Parental report of developmental delay, neonatal discharge at an age greater than 28 days Daycare attendance and maternal alcohol intake and smoking during pregnancy (two-fold) The presence of two of these risk factors increases the probability of a first febrile convulsion by about 30% (Vestergaard, 2002). There are two types of febrile convulsions: Simple febrile convulsions are usually over in a few minutes, but in rare cases they can last up to 15 minutes. During this type of convulsion, a child's whole body may convulse, shake, and twitch; their eyes may roll; and they may moan or become unconscious. Children can sometimes vomit or urinate on themselves during the convulsions. Complex febrile convulsions can last longer than 15 minutes or happen more than once in 24 hours. They may also involve the movement or twitching of just one part of the body (Gupta, 2016). During generalized febrile convulsions, the body will become stiff and the arms and legs will begin twitching. The child loses consciousness, although their eyes remain open. Breathing can be irregular. They may become incontinent (wetting or soiling themselves); they may also vomit or have increased secretions (foaming at the mouth). The convulsion normally lasts for less than five minutes. The child's temperature is usually greater than 38 °C (100.4 °F) (Symptoms of febrile convulsions, 2014). Patients with active convulsions should be treated with airway management, high-flow oxygen, supportive care, and anticonvulsants as necessary. Acute treatment such as rectal (0.5 mg/kg), buccal (0.4–0.5 mg/kg), or intranasal (0.2 mg/kg) diazepam is effective and can be given at home for a convulsion lasting longer than five minutes (Sadleir, 2007).

Materials and Method:

Experiment Designs:
The samples were divided into two groups: the control group with 30 samples and the infected group with 60 samples. The trial period is from 2 August until 15 November, 2022.

Sample Collection:
The questionnaire form for the study was designed and included all the required information. Five milliliters of venous blood were drawn from each participant. One milliliter was placed in the anticoagulant tube to determine the complete blood picture as quickly as possible. Four milliliters were placed in tubes. These are left to curdle for 15 minutes at room temperature. The serum was separated by a centrifuge at 5000 rpm/min for five minutes. Serum was used to determine biochemical agents.

Biochemical and Physiological parameters:

Determination of malondialdehyde (MDA) in serum:
The researchers (SHAH and Guidet) used a modified thiobarbituric acid reaction method to measure MDA, which is one of the final products of the lipid meta-oxidation process, and its level is an indicator of this process, as the measurement depends on the interaction between fat peroxides, particularly malondialdehyde, and TBA in a pH-dependent medium. (Guidet and Shah, 1989).
Determination of acetylcholinesterase (AChE) in serum:

**Basic test principle:** Acetylcholinesterase was measured in serum by using the modified Ellman method (Ellman et al., 1961; Jewad et al., 2011). Ellman’s procedure is commonly used for the determination of acetylcholinesterase and also for monitoring of the ACh hydrolysis by acetylcholinesterase (AChE) or butyrylcholinesterase (BChE) in vitro. The Ellman’s method is based on the reaction of thiocholine (one of the products of the enzymatic hydrolysis of ACh by acetylcholinesterase) with 5,5-dithiobis-2-nitrobenzoic acid (DTNB), also called Ellman’s reagent), which forms a yellow product (5-mercapto-2-nitrobenzoic acid and its dissociated forms) at pH 8. The maximum absorption coefficient was found at 412 nm.

**Determination of Glutathione (GSH) in serum:**

Reaction principle: The level of glutathione in blood serum was measured by using the Ellman reagent method (Ellman et al., 1961).

**Statistical Analysis:**

The results obtained from the current study were analyzed by using SAS 2001. The ANOVA test was used. The significant differences between the arithmetic averages were tested by using the Duncan multiple range test to compare between the groups at a significance level of 0.05.

**The Results and Discussion:**

**Complete blood count (CBC):**

Some hematological parameters were studied, where Table (1) showed a significant decrease (P≤0.05) in the level of hemoglobin Hb (11.61±1.05g/dL) and the presence of a significant increase (P≤0.05) in the number of white blood cells counted WBCc (11.91±4.80×10³/µL) and the percentage of lymphocytes LYM (37.2±15.3%) and the number of platelets PLT (355±100×10³/µL) in the blood serum of children in the infected group, Compared with the level of hemoglobin Hb (12.557±0.626g/dL), the number of white blood cells counted WBCc (9.16±2.16×10³/µL), the percentage of lymphocytes LYM (31.48±6.04%), and the number of platelets PLT (314.1±56.5×10³/µL) in the pediatric serum of the control group. The results of the study did not show a significant difference (P≥0.05) in the form of Red Cell Distribution Width RDW (39.63±3.34 fL) and the number of Red Blood Cells Count RBCc (4.609±0.437×10⁶/µL) in the blood serum of children in the affected group compared with the Red Cell Distribution Width RDW (39.42±2.88 fL) and Red Blood Cells Count RBCc (4.747±0.328×10⁶/µL) in the blood serum of children in the control group.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Patients</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Numbers</td>
<td>60</td>
<td>30</td>
</tr>
<tr>
<td>Hemoglobin g/dL (Hb)</td>
<td>11.61±1.05</td>
<td>12.557±0.626</td>
</tr>
<tr>
<td>Red Cell Distribution Width RDW (fL)</td>
<td>39.63±3.34</td>
<td>39.42±2.88</td>
</tr>
<tr>
<td>Red blood cells count 10⁶/µL (RBCc)</td>
<td>4.609±0.437</td>
<td>4.747±0.328</td>
</tr>
<tr>
<td>white blood cells count 10⁶/µL (WBCc)</td>
<td>11.91±4.80</td>
<td>9.16±2.16</td>
</tr>
<tr>
<td>lymphocytes LYM %</td>
<td>37.2±15.3</td>
<td>31.48±6.04</td>
</tr>
<tr>
<td>platelets PLT 10⁶/µL</td>
<td>355±100</td>
<td>314.1±56.5</td>
</tr>
</tbody>
</table>

Note: ***/ indicates a significant difference , ns/no significant difference. ±/average ± standard deviation

Anemia has been linked to febrile convulsions in terms of the effect of iron at the nervous system level in terms of neurocellular metabolism because iron affects the level of neurotransmitters as well as enters into the synthesis of hemoglobin in the blood and thus the delivery of oxygen to the brain. Iron
is also required for nervous system functions such as neuron metabolism, myeloid sheath formation, and energy production (Lozoff et al., 2006). There are some iron-dependent enzymes such as toptophan and hydroxylase, which are involved in the synthesis of serotonin, and tyrosine hydroxylase, which is involved in the synthesis of dopamine and norepinephrine, so iron deficiency affects monoamine metabolism (Yadav and Chandra, 2011). Viral upper respiratory tract infections and viral gastrointestinal diseases occur in children, as do bacterial infections of the respiratory tract and gastrointestinal tract. All viral and bacterial conditions increase the number of leukocytes, particularly lymphocytes and platelets, and anemia increases the number of platelets (Stafstrom, 2002).

Concentrations of Glutathione (GSH) and Malondialdehyde (MDA) and Acetylcholine Esterase (AChE) in serum:

The study indicates a significant decrease (P≤0.05) in the concentration level of acetylcholine esterase AChE (246.3±50.6U/L) in the blood serum of the affected group compared to the concentration level of acetylcholine esterase AChE (272.4±42.0U/L) in the blood serum of the control group. The results of the study showed that there was no significant difference (P≥0.05) in the level of glutathione concentration GSH (1.009±0.125 μmol/L) and the level of malondialdehyde concentration (9.84±1.68 μmol/L) in the blood serum of the affected children compared to the level of glutathione concentration GSH (0.978±0.071 μmol/L) and the level of malondialdehyde concentration (10.25±1.72 μmol/L) MDA in the control children's blood serum.

Table (2): Shows the concentrations of GSH, MDA, and AChE in serum

<table>
<thead>
<tr>
<th>Parameters group</th>
<th>Numbers</th>
<th>Acetylcholine esterase AChE (U/L)</th>
<th>Malondialdehyde MDA (μmol/L)</th>
<th>Glutathione GSH (μmol)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>60</td>
<td>246.3 ± 50.6 **</td>
<td>9.84 ± 1.68 ns</td>
<td>1.009 ± 0.125 ns</td>
</tr>
<tr>
<td>Control</td>
<td>30</td>
<td>272.4 ± 42.0 ns</td>
<td>10.25 ± 1.72 P≥0.012 P≥0.282</td>
<td>0.978 ± 0.071 P≥0.148</td>
</tr>
</tbody>
</table>

Note: ** indicates a significant difference. ns / no significant difference. ± / average ± standard deviation

Acetylcholine esterase (AChE) is an important enzyme because of its role in transmitting nerve impulses within the body of an organism. It stops the transmission of nerve impulses through the hydrolysis of acetylcholine in nerve connections, neuromuscular connections, blood, and others. The enzyme acetylcholine esterase is very sensitive to a large number of chemical compounds that inhibit the enzyme, as well as having an optimal temperature (37 °C) and an optimal pH (7.4). Children with bacterial or viral infections have a high body temperature of more than 38.4 °C, and diarrhea or vomiting cause a high pH, which causes a decrease in the concentration of the enzyme, which leads to the non-decomposition of acetylcholine, so heat convulsions occur in children. (Pohanka and Naringrekar, 2014).

REFERENCES


