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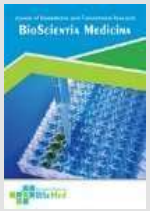

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Challenges of Diagnosis and Management of Complex Febrile Seizures in Infants: A Case Study

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A B S T R A C T

Background: Complex febrile seizures (KDK) are a type of febrile seizure that is more severe than simple febrile seizures (KDS), characterized by longer duration, recurrence within 24 hours, and/or focal onset. KDK can be a diagnostic and therapeutic challenge, especially in infants. **Case presentation:** This case report presents a 1-year-old baby girl who was treated in the emergency department (ER) with complaints of recurrent seizures and persistent fever. Physical examination revealed fever and tachypnea, while laboratory examination revealed hyperglycemia, hyperkalemia, hyponatremia, and decreased hematocrit. The patient's history indicated previous episodes of febrile seizures, but no significant family history. **Conclusion:** The patient was diagnosed with KDK and given fluid resuscitation, antipyretics, anticonvulsants, and antibiotics. The patient's parents are educated about KDK and its management. The patient's prognosis is considered good, with a small chance of long-term neurologic sequelae if treated appropriately.

1. Introduction

Febrile seizures (KD) are one of the most common acute neurological conditions that occur in children aged 6 months to 5 years, with a prevalence ranging from 2% to 5% worldwide.[1,2] KD is defined as seizures that occur at a body temperature above 38°C, without central nervous system infection or other metabolic disorders as a cause.[3] KD is categorized into two main types: simple febrile seizures (KDS) and complex febrile seizures (KDK).[4] KDS is the most common form of febrile seizure, characterized by generalized tonic-clonic seizures that last less than 15 minutes and do not recur within a 24-hour period.[5] KDS usually has a good prognosis, with a low risk of developing epilepsy later in life. On the other hand, KDK is a form of febrile seizure that occurs less frequently but has more complex characteristics and has the potential to cause more serious consequences.[6] KDK is defined as febrile seizures that last more than 15 minutes, recur within a 24-hour period, or have focal characteristics (e.g., seizures that start on one side of the body).[7] KDK can be caused by various factors, including viral or bacterial infections, metabolic disorders, or genetic abnormalities.[8] Compared with KDS, KDK has a higher risk of developing epilepsy later in life, especially if there are other risk factors such as a family history of epilepsy, previous neurological disorders, or very long or recurrent febrile seizures.[9]

The pathophysiology of KDK is not completely understood, but several mechanisms have been

proposed. One of the main hypotheses is that KDK is caused by a combination of genetic and environmental factors.[10] Genetic factors can increase an individual's susceptibility to seizures, while environmental factors such as fever can trigger seizures. Fever can trigger seizures through several mechanisms. An increase in body temperature can increase neuronal excitability, namely the ability of neurons to produce electrical impulses. In addition, fever can disrupt the balance of neurotransmitters in the brain, such as GABA (gamma-aminobutyric acid) which acts as an inhibitory neurotransmitter, and glutamate which acts as an excitatory neurotransmitter. Babies, especially those under 1 year old, have limitations in communicating and expressing symptoms. This can complicate the diagnosis of KDK, especially if the seizure is short-lived or is not accompanied by other obvious symptoms. Therefore, case studies of KDK in infants can provide valuable information about the typical clinical presentation in this age group, thereby assisting physicians in establishing an accurate diagnosis. Management of KDK in infants requires a careful and integrated approach. Stabilization of the general condition, control of seizures, and management of fever and electrolyte imbalance are important steps in initial management. Case studies of KDK in infants may provide insight into effective and safe management strategies for this age group, including appropriate selection of anticonvulsant drugs and appropriate dosage.

KDK can be caused by various factors, ranging from viral or bacterial infections to metabolic disorders or genetic disorders. Identification of the underlying cause is important for determining prognosis and planning long-term therapy. Case studies of KDK in infants can help identify risk factors and potential causes of KDK in this age group, thereby enabling early intervention and prevention of long-term complications. KDK has a higher risk of developing epilepsy later in life compared to KDS. Therefore, preventing the recurrence of seizures and epilepsy is an important goal in the management of KDK. Case studies of KDK in infants can provide information about factors that may increase the risk of recurrence of seizures and epilepsy, as well as effective prevention strategies. Education of parents and the public about KDK is very important to increase awareness about this condition, reduce anxiety, and ensure appropriate treatment at home. Case studies of KDK in infants can be used as an effective educational tool, by presenting relevant and easy-to-understand information about the symptoms, diagnosis, management, and prognosis of KDK. This case report aims to provide a comprehensive description of the clinical presentation, diagnosis, and management of KDK in infants. In addition, it is hoped that this case report will contribute to our understanding of KDK pathophysiology, risk factors, and prevention strategies.

2. Case Presentation

A 12-month-old baby girl was taken to the hospital emergency department (ER) by her parents on March 15th, 2024 at 10.30 WIB. The parents reported that the patient experienced two separate seizure episodes in less than 24 hours. The first seizure episode occurred on March 14th, 2024 at 18.00 WIB while the patient was playing at home. Seizures last about one minute and are characterized by the following symptoms: Generalized tonic-clonic movements: The patient's entire body becomes rigid (tonic phase) followed by rhythmic jerking movements (clonic phase); Loss of consciousness: The patient is unresponsive to external stimuli during the seizure; Upward eye deviation: The patient's eyes turn upward during the seizure; Perioral cyanosis: There is a bluish color around the patient's mouth, indicating mild hypoxia. After the seizure stops, the patient returns to full consciousness within a few minutes. The parents did not report any neurological sequelae after this episode. The second seizure episode occurred on March 15th, 2024 at 09.00 WIB when the patient was on her way to the hospital. The seizure lasts about a minute with characteristics similar to the first episode. The patient returned to full consciousness after the seizures stopped, with no obvious neurological deficits. Apart from seizures, the

parents also reported that the patient had had a fever since March 14th, 2024 morning. The patient's body temperature was measured at 39°C in the axilla. The parents had given oral paracetamol at the appropriate dose, but the fever did not go down.

The patient had a history of one previous episode of febrile seizure at 9 months of age. The episode occurs when the patient has a high fever and lasts less than 5 minutes. The patient experienced no neurological sequelae after the episode. The patient has received complete immunization according to schedule. There is no history of febrile seizures or epilepsy in the patient's immediate or extended family. There was no known family history of genetic or metabolic disease. There is no history of drug or food allergies. The patient is not taking any medication regularly. When she arrived at the ER, the patient was conscious but appeared fussy and uncomfortable. A complete physical examination was carried out with the following results: Body temperature 39.5°C (axillary), Respiratory rate 45 times/minute, Heart rate 150 beats/minute, Blood pressure 90/60 mmHg, Oxygen saturation 98% (room air), Consciousness shows Compos mentis (fully conscious), General condition shows mild illness, Good skin turgor, Moist oral mucosa, Lymph nodes show no enlargement, Mental function according to age, Normal cranial nerves, Normal muscle strength, normal tone, none focal motor deficit, No sensory deficit, Normal physiological reflexes, no Babinski sign, No coordination disturbance, Normal gait, Head shows Normocephalic, no trauma, Conjunctiva is not anemic, sclera is not icteric, pupils are isochorous and reactive to light, Membrane tympanic intact, no secretions, no secretions, normal mucosa, pharynx not hyperemic, tonsils not enlarged, heart sounds normal, no murmurs, vesicular, no wheezing or rales. Supporting examinations are carried out to support the diagnosis and identify the cause of KDK.

The results of supporting examinations (Table 1) showed leukocytosis with neutrophilia, which indicated an inflammatory response, possibly due to infection. However, blood and urine cultures were negative, so bacterial infection could be ruled out. Hyponatremia, hyperkalemia, and hyperglycemia indicate significant electrolyte imbalances, which can worsen seizures and require special treatment. Mild metabolic acidosis indicates a disturbance in the acid-base balance, which can be caused by dehydration, seizures, or other metabolic disorders. A normal chest X-ray shows no pneumonia or other lung abnormalities. A head CT scan was not performed because the patient's condition was unstable on arrival. A head CT scan may be performed later if structural brain abnormalities are suspected. Based on the history, physical examination, and results of supporting examinations, the working diagnosis for this patient is complex febrile convulsions (KDK). This diagnosis is supported by the presence of recurrent seizures within 24 hours, high fever unresponsive to antipyretics, and the absence of obvious signs of infection or other neurological disorders. Patients experience hyponatremia, hyperkalemia, and hyperglycemia, which can worsen seizures and require special treatment. Even though there are no signs of meningeal stimulation on physical examination and the results of the lumbar puncture are normal, meningitis still needs to be excluded as a cause of KDK. Encephalitis can cause seizures and fever but is usually accompanied by impaired consciousness and focal neurological deficits. Sepsis can cause seizures and fever but is usually accompanied by other signs of systemic infection, such as hypotension, tachycardia, and leukocytosis. Even if the EEG is normal, epilepsy should still be considered as a differential diagnosis, especially if the patient experiences recurrent seizures in the future.

Patient management focuses on stabilizing the general condition, controlling seizures, and treating fever and electrolyte imbalance. The patient was administered oxygen via nasal cannula at a flow rate of 2 L/min to ensure adequate oxygenation. The patient's vital signs (blood pressure, pulse, respiratory rate, and body temperature) are closely monitored every 15 minutes.

Table 1. Supporting examinations.

Supporting examination	Results	Reference value	Interpretation
Whole blood			
Leukocytes	18.000/µL	4.500-11.000/µL	Increased (leukocytosis)
Neutrophils	70%	40-60%	Increased (neutrophilia)
Lymphocytes	20%	20-40%	Normal
Monocytes	5%	2-8%	Normal
Eosinophils	3%	1-4%	Normal
Basophils	2%	0.5-1%	Normal
Hemoglobin	12 g/dL	11-13 g/dL	Normal
Hematocrit	36%	33-39%	Normal
Platelets	150.000/µL	150.000-450.000/µL	Normal
Serum electrolytes			
Sodium (Na)	128 mEq/L	135-145 mEq/L	Low (hyponatremia)
Potassium (K)	5.8 mEq/L	3.5-5.0 mEq/L	High (hyperkalemia)
Chloride (Cl)	98 mEq/L	98-106 mEq/L	Normal
Glucose	220 mg/dL	70-110 mg/dL	High (hyperglycemia)
Liver function test			
SGOT/AST	25 U/L	15-40 U/L	Normal
SGPT/ALT	30 U/L	7-56 U/L	Normal
Bilirubin total	0.8 mg/dL	0.3-1.2 mg/dL	Normal
Kidney function test			
Urea	30 mg/dL	10-50 mg/dL	Normal
Creatinine	0.5 mg/dL	0.3-0.7 mg/dL	Normal
Arterial blood gases			
pH	7.35	7.35-7.45	Acidosis
pCO_2	30 mmHg	35-45 mmHg	Normal
HCO_3^-	18 mEq/L	22-26 mEq/L	Low
pO_2	95 mmHg	80-100 mmHg	Normal
Culture			
Blood culture	Negative	Negative	No bacterial growth
Urine culture	Negative	Negative	No bacterial growth
Routine urine analysis			
Color	Clear yellow	Clear yellow	Normal
Specific gravity	1.015	1.005-1.030	Normal
pH	6.0	4.6-8.0	Normal
Protein	Negative	Negative	Normal
Glucose	Negative	Negative	Normal
Ketones	Negative	Negative	Normal
Bilirubin	Negative	Negative	Normal
Urobilinogen	Normal	Normal	Normal
Nitrite	Negative	Negative	Normal
Leukocyte esterase	Negative	Negative	Normal
Radiology			
Thorax photo	Normal		No abnormalities in the lungs

The patient was given intravenous diazepam at a dose of 0.3 mg/kg (total 3 mg) slowly over 3 minutes. Intravenous administration of diazepam is effective in stopping seizures within 5 minutes. After the seizures stopped, the patient was given intravenous phenytoin at an initial dose of 20 mg/kg (total 200 mg) to prevent seizure recurrence. Phenytoin is administered at an infusion rate of no more than 50 mg/minute. Continuous EEG monitoring was performed for 24 hours to assess subclinical seizure activity and determine the need for further anticonvulsant therapy. The patient was given intravenous paracetamol at a dose of 15 mg/kg every 6 hours to control fever. Warm compresses are given to the patient's forehead, armpits, and groin to help reduce body temperature. Patients were given intravenous fluids of 0.9% NaCl with the infusion rate adjusted based on serum sodium levels and vital signs. Patients are given intravenous calcium gluconate at a dose of 100 mg/kg over 10 minutes to protect the heart from the effects of

hyperkalemia. Next, the patient is given intravenous insulin and glucose to reduce serum potassium levels. Lumbar puncture is performed to collect samples of cerebrospinal fluid (CSF). CSF analysis results showed normal cell counts, protein, and glucose, ruling out the possibility of meningitis or encephalitis. Blood and urine cultures are taken to rule out sepsis. Blood and urine culture results were negative, indicating no bacterial infection. An EEG is performed to assess the brain's electrical activity and rule out epilepsy. EEG results were normal, indicating no epileptiform activity.

Based on normal supporting examination results, KDK in this patient was considered idiopathic, meaning there was no clear cause. The patient was given oral phenobarbital at a dose of 5 mg/kg/day for 3 days to prevent recurrence of seizures. Parents of patients are given education about KDK, including causes, symptoms, treatment, and how to provide first aid to children who experience seizures. The patient was scheduled for re-examination with a pediatrician and neurologist in 1 week for further monitoring and evaluation of anticonvulsant therapy. The prognosis for idiopathic KDK is generally good. Most children with idiopathic KDK recover completely without long-term neurological sequelae. However, idiopathic KDK can increase the risk of epilepsy in the future. Therefore, it is important to monitor these patients regularly and educate parents about the signs and symptoms of epilepsy.

3. Discussion

Neurons are the basic functional units of the nervous system responsible for the transmission of electrical and chemical signals. Neuronal excitability refers to the ability of a neuron to generate action potentials in response to a stimulus. An action potential is a rapid and brief electrophysiological event in which a neuron's membrane potential depolarizes (becomes more positive) and then repolarizes (returns to resting potential). This action potential propagates along the neuron's axon and allows communication between neurons. Neuron excitability is influenced by various factors, including the concentration of ions inside and outside the cell, the permeability of the cell membrane to those ions, and the activity of ion pumps that regulate the movement of ions across the membrane. The main ions involved in neuronal excitability are sodium (Na^+), potassium (K^+), calcium (Ca^{2+}), and chloride (Cl^-). Fever, or an increase in body temperature above normal, can affect ion conductance across neuronal membranes. Ion conductance refers to the ease with which ions can move across the cell membrane. Changes in ion conductance can influence neuronal membrane potential and, ultimately, neuronal excitability. An increase in body temperature can increase the kinetic energy of ions, causing faster ion movement and increased ion conductance. This can affect various ion channels in neuronal membranes, including sodium, potassium, calcium, and chloride channels. Sodium channels are essential for the initiation and propagation of action potentials. The increase in sodium conductance due to fever can cause faster depolarization of neuronal membranes and more easily reach the threshold for generating an action potential. This can increase the frequency of action potentials and increase the excitability of neurons. Potassium channels are responsible for the repolarization of neuronal membranes after action potentials. Increased potassium conductance due to fever can accelerate membrane repolarization and shorten the duration of the action potential. However, these effects may vary depending on the type of potassium channel involved. Some types of potassium channels can be inhibited by increased temperature, which can slow repolarization and increase neuronal excitability. Calcium channels play an important role in the release of neurotransmitters at the synapse, which is the meeting point between two neurons. Increased calcium conductance due to fever can increase calcium influx into presynaptic neurons, trigger greater neurotransmitter release, and increase the excitability of postsynaptic neurons. Chloride channels play a role in maintaining the resting membrane potential of neurons. Increased chloride conductance due to fever can cause hyperpolarization

of neuronal membranes and reduce neuronal excitability. Neurons have various thermosensitive ion channels, namely ion channels that are sensitive to changes in temperature. Some thermosensitive ion channels, such as transient receptor potential (TRP) channels, can be activated by an increase in body temperature. TRP channels are a large family of ion channels that respond to a variety of stimuli, including temperature, pH, and chemical compounds. Several members of the TRP family, such as TRPV1, TRPV2, TRPV3, and TRPV4, can be activated by temperature increases in the febrile range. Activation of TRP channels can increase the influx of calcium and sodium into neurons, causing membrane depolarization and increasing neuronal excitability. The sodium-potassium pump (Na^+/K^+-ATPase) is a transmembrane protein that plays an important role in maintaining the electrochemical gradient across cell membranes. This pump uses energy from ATP to pump three sodium ions out of the cell and two potassium ions into the cell. An increase in body temperature can increase the activity of the sodium-potassium pump. This can lead to increased sodium efflux and potassium influx, which can affect neuronal membrane potential and neuronal excitability. Increased neuronal excitability due to fever can cause various clinical manifestations, including febrile seizures. Febrile seizures are seizures that occur in children aged 6 months to 5 years, accompanied by fever without central nervous system infection or other metabolic disorders as a cause. The mechanisms underlying febrile seizures are not fully understood, but changes in ion conductance, activation of thermosensitive ion channels, and increased sodium-potassium pump activity are thought to play an important role in the pathogenesis of febrile seizures. Understanding these mechanisms may help in the development of strategies for the prevention and treatment of febrile seizures. For example, anticonvulsant drugs that work by modulating ion conductance or inhibiting thermosensitive ion channels can be used to prevent or control febrile seizures. Fever can increase neuronal excitability through several pathways, including changes in ion conductance, activation of thermosensitive ion channels, and increased sodium-potassium pump activity. These changes can affect the membrane potential of neurons and trigger uncontrolled action potentials, which can ultimately lead to febrile seizures.[10-12]

Neurotransmitters are chemical molecules that play a crucial role in transmitting signals between neurons in the nervous system. They are released by the presynaptic (sending) neuron into the synaptic cleft and then bind to specific receptors on the postsynaptic (receiving) neuron. Neurotransmitters can be excitatory, increasing the likelihood of the postsynaptic neuron to produce an action potential (electrical signal), or inhibitory, decreasing that likelihood. The balance between excitatory and inhibitory neurotransmitters is essential for maintaining normal brain function, including the regulation of movement, perception, emotion, and cognition. GABA is the main inhibitory neurotransmitter in the central nervous system (CNS). GABA is synthesized from glutamate through the action of the enzyme glutamate decarboxylase (GAD). After being released into the synaptic cleft, GABA binds to two main types of receptors on postsynaptic neurons, namely GABA-A receptors, which are ionotropic receptors associated with chloride ion channels. GABA binding to GABA-A receptors causes an influx of chloride ions into the cell, resulting in hyperpolarization of neuronal membranes and reduced neuronal excitability. GABA-B receptors are metabotropic receptors linked to G proteins. Binding of GABA to GABA-B receptors triggers an intracellular signaling cascade that can inhibit calcium channels, reduce the release of excitatory neurotransmitters, and increase potassium conductance, thereby causing hyperpolarization of neuronal membranes. Increased body temperature can inhibit the activity of GAD, a key enzyme in GABA synthesis. This can reduce the availability of GABA in the brain. Fever can interfere with the release of GABA from synaptic vesicles in presynaptic nerve endings. The exact mechanism is

not fully understood but likely involves changes in vesicle protein conformation or disruption of the neurotransmitter release machinery. Increased body temperature can change the conformation of GABA receptors, reduce GABA binding affinity, and decrease the responsiveness of postsynaptic neurons to GABA.[13-15]

Overall, the effects of fever on GABA can reduce the inhibitory activity of GABA in the brain, increase the excitability of neurons, and contribute to the occurrence of seizures. Glutamate is the main excitatory neurotransmitter in the CNS. Glutamate is released from the presynaptic neuron and binds to various types of glutamate receptors on the postsynaptic neuron. Glutamate receptors can be divided into two main groups, namely ionotropic receptors related to ion channels and causing rapid depolarization of the postsynaptic neuron membrane. Examples of ionotropic glutamate receptors are AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid), NMDA (N-methyl-D-aspartate), and kainite receptors. Metabotropic receptors are linked to G proteins and trigger intracellular signaling cascades that can modulate a variety of neuronal functions, including excitability, synaptic plasticity, and gene expression. An increase in body temperature can increase the release of glutamate from synaptic vesicles in presynaptic nerve endings. This may occur through increased calcium channel activity or changes in vesicle protein conformation. Fever can increase the sensitivity of glutamate receptors, especially NMDA receptors. This can occur through receptor phosphorylation or changes in the lipid environment of the cell membrane. Fever can inhibit glutamate transporters, which are proteins responsible for retrieving glutamate from the synaptic cleft. This can increase the concentration of glutamate in the synaptic cleft and prolong its excitatory effects. Overall, the effects of fever on glutamate may increase the excitatory activity of glutamate in the brain, increase neuronal excitability, and contribute to the occurrence of seizures.[16-18]

The blood-brain barrier (BBB) is a complex structure that is critical for maintaining homeostasis of the brain environment. Its main function is to protect the brain from harmful substances in the blood, while still allowing nutrients and other essential molecules to enter the brain. Endothelial cells are cells that line the walls of blood vessels in the brain. Endothelial cells in the BBB have very tight junctions, which limit the movement of molecules between the blood and the brain. Pericytes are cells that surround endothelial cells and provide structural and functional support to the BBB. Pericytes also play a role in regulating BBB permeability. Astrocytes are glial cells that are star-shaped and play an important role in maintaining brain homeostasis. Astrocytes have legs that attach to blood vessels in the brain and help maintain the integrity of the BBB. The basal lamina is a thin layer of extracellular protein located between the endothelial cells and astrocytes. The basal lamina plays a role in filtering molecules that pass through the BBB. The BBB prevents the entry of toxins, pathogens, and other harmful molecules into the brain. This is important for maintaining brain health and function. The BBB allows the entry of nutrients, such as glucose and amino acids, as well as other essential molecules, such as oxygen and hormones, into the brain. The BBB helps maintain ion and fluid balance in the brain, which is important for the normal function of neurons. The BBB helps remove metabolic waste products and toxins from the brain. Fever is often a response to infection. Infection can activate the immune system, which then releases pro-inflammatory cytokines. Pro-inflammatory cytokines, such as interleukin-1 beta (IL-1β), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6), can increase BBB permeability. Pro-inflammatory cytokines can disrupt tight junctions between endothelial cells, thereby increasing the intercellular space and allowing larger molecules to pass through the BBB. Pro-inflammatory cytokines can increase the expression of adhesion molecules on endothelial cells, which can facilitate the attachment and transmigration of leukocytes (white blood cells) to the

brain. Pro-inflammatory cytokines can damage the extracellular matrix, the protein network that supports endothelial cells, thereby disrupting the integrity of the BBB. Activation of matrix metalloproteinases (MMPs), where MMPs are enzymes that play a role in the degradation of the extracellular matrix. Fever can increase the expression and activity of MMPs in the brain, which can damage the basal lamina and tight junctions, thereby increasing BBB permeability. Fever can increase the production of free radicals, which are unstable molecules that can damage cells. Free radicals can damage endothelial cells and tight junctions, thereby increasing BBB permeability. The BBB has a selective active transport mechanism to move certain molecules between the blood and the brain. Fever can disrupt this active transport mechanism, thereby disrupting the regulation of the entry of substances into the brain. Increased BBB permeability allows the entry of harmful substances, such as toxins, pathogens, and pro-inflammatory molecules, into the brain. This can trigger inflammation, neuron damage, and brain dysfunction. Increased BBB permeability can disrupt the balance of ions and fluids in the brain, which can cause brain edema (brain swelling) and impaired neuronal function. Increased BBB permeability can disrupt neurotransmission, namely the communication process between neurons, which can cause impaired brain function. Pro-inflammatory substances entering the brain through a leaky BBB can activate inflammatory receptors on neurons and glial cells, which can increase neuronal excitability and trigger seizures. Brain edema caused by increased BBB permeability can compress brain tissue and disrupt neuron function, which can trigger seizures. Increased BBB permeability can disrupt the balance of neurotransmitters in the brain, especially GABA and glutamate, which can increase neuronal excitability and trigger seizures.[17-19]

Fever, dehydration, and electrolyte imbalance are interrelated conditions and often occur together, especially in children. These three conditions can affect brain function and increase the risk of seizures, especially in susceptible individuals. Understanding this complex relationship is critical to developing effective seizure prevention and treatment strategies. Fever increases the body's basal metabolic rate, which results in increased heat production. To maintain a stable body temperature, the body will increase heat production through the evaporation of sweat. Excessive evaporation of sweat can cause significant fluid loss, especially in children who have a larger body surface area relative to their body weight. Children who have a fever often have a decreased appetite for eating and drinking, which can reduce their fluid intake. This can worsen dehydration caused by increased fluid loss through sweat. Fever can increase the production of antidiuretic hormone (ADH), which causes the kidneys to retain more water and produce more concentrated urine. Although this mechanism aims to conserve body fluids, in children who have high fever and inadequate fluid intake, this can worsen dehydration. Dehydration not only causes fluid loss but can also disrupt the electrolyte balance in the body. Electrolytes are minerals that carry electrical charges in body fluids and play an important role in various body functions, including muscle contraction, nerve transmission, and fluid balance. Dehydration can cause hyponatremia, which is a condition where sodium levels in the blood are too low. Sodium plays an important role in maintaining fluid balance and nerve function. Hyponatremia can cause muscle weakness, confusion, seizures, and even coma. Dehydration can also cause hyperkalemia, which is a condition where potassium levels in the blood are too high. Potassium plays an important role in muscle contraction and heart function. Hyperkalemia can cause muscle weakness, cardiac arrhythmias, and even cardiac arrest. Dehydration can cause hypocalcemia, which is a condition where calcium levels in the blood are too low. Calcium plays an important role in muscle contraction, blood clotting, and nerve function. Hypocalcemia can cause muscle cramps, seizures, and heart rhythm disturbances. Hyponatremia and hypocalcemia can increase neuronal excitability by lowering the action potential

threshold. This makes it easier for neurons to fire to produce electrical impulses, which can cause seizures. Electrolyte imbalances can disrupt nerve transmission, which is the process by which neurons communicate with each other. This can cause impaired brain function and increase the risk of seizures. Electrolyte imbalances can change the permeability of cell membranes, which can cause cellular edema (cell swelling) and increase intracranial pressure. Increased intracranial pressure can trigger seizures. Children have a larger body surface area relative to their body weight compared to adults. This causes them to lose fluids more quickly through the evaporation of sweat during fever. Children have smaller fluid reserves compared to adults. Therefore, they become dehydrated more quickly if fluid intake is inadequate. Children's kidneys are not fully mature, so their ability to concentrate urine and conserve fluids is lower than that of adults. The combination of fever, dehydration, and electrolyte imbalance in children can create an environment conducive to seizures. Fever can trigger seizures directly by increasing neuronal excitability and disrupting neurotransmitter balance. Dehydration can worsen this condition by causing an electrolyte imbalance, which in turn increases neuronal excitability and disrupts nerve transmission.[18-20]

4. Conclusion

KDK is a more severe type of febrile seizure than KDS. KDK can be a diagnostic and therapeutic challenge, especially in infants. Management of KDK involves patient stabilization, identification and treatment of the underlying cause, and prevention of recurrence. The prognosis for KDK is generally good, but KDK can increase the risk of epilepsy in the future.

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