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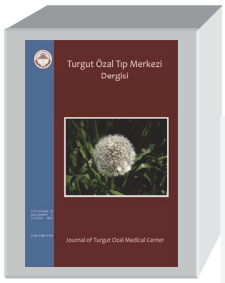

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Febrile Seizure Related with Adenovirus Gastroenteritis: A Case Report

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Abstract
Febrile seizure is the most common, age-dependant, benign, and fever-related convulsion of childhood. Its pathogenesis is still not clear. Fever causing febrile seizures is usually associated with viral infections, mostly upper respiratory tract infections, otitis media, tonsillitis, or urinary tract infections. The incidence of febrile convulsions during gastroenteritis is lower and gastroenteritis is thought to exert a protective feature in febril seizures. Although the most common pathogen in gastroeneritis is the rotavirus, adenovirus and small round viruses are also frequently reported. We aim to review the pathogenesis of febrile seizures and discuss vaccination in selected groups with known pathogens while discussing a patient with febrile seizures during adenovirus gastroenteritis.
Key Words: Adenovirus Gastroenteritis; Febrile Seizure; Pathogenesis.

Adenovirüs Gastroenteriti ile İlişkili Febril Konvülziyon: Bir Vaka Sunumu

Özet
Febril konvülziyon, çocukluk çağının en sık görülen, yaşa bağımlı, benign ve ateşle ortaya çıkan konvülziyonudur. Patogenezi halen çok iyi aydınlatılamamıştır. Febril konvülziyonda ateş genellikle viral enfeksiyonlarla ilişkilidir ve sıklıkla üst solunum yolu enfeksiyonları, otitis media, tonsillit, idrar yolu enfeksiyonlarından kaynaklanır. Gastroenterit sırasında febril konvülziyon insidansı düşük bulunmuştur ve gastroenteritin febril konvülziyondan koruyucu özellik gösterdiği düşünülmektedir. Gastroeneritlerde en sık görülen etken rotavirüs olsa da adenovirüs ve küçük yuvarlak yapılı virüsler de sık bildirilen patojenlerdir. Adenovirüs gastroenteriti sırasında nöbet geçiren bir hastayı tartışırken, febril konvülziyon patogenezini gözden geçirmek, ateşli nöbete sebep olan patojenleri saptayarak seçilmiş gruplara aşı uygulamasını tartışmak istedik.
Anahtar Kelimeler: Adenovirüs Gastroenteriti; Febril Konvülziyon; Patogenez.

INTRODUCTION

As an age-dependent and benign convulsion that develops with fever, febrile seizure is the most common convulsion of childhood. Its pathogenesis is still not very well understood. Fever in febrile seizures is generally associated with viral infections and often results from upper respiratory tract infections, otitis media, tonsillitis, or urinary tract infections. Human herpes virus type 6-7, EU influenza, adenovirus, respiratory syncytial virus, herpes simplex virus, and cytomegalovirus are the frequent factors behind febrile seizure (1).

The incidence of febrile seizures is reported to be lower during gastroenteritis and it is thought that gastroenteritis is protective against febrile seizures (2). Although the most common cause of gastroenteritis is the rotavirus (by 83%), adenovirus (by 8.2%) and small round structured viruses (by 5.8%) are also among frequently reported pathogens (3). This study presents a rare febrile convulsion case which is associated with adenovirus gastroenteritis.

CASE REPORT

A 22-month-old male patient with no prior history of seizures was admitted to our hospital after a seizure attack. We learnt that the patient had been suffering from yellowish diarrhoea 2-3 times a day in the last four days. In the day before the admission, vomiting and fever had been added to the clinical picture while practitioners in the previous centre applied intravenous fluid replacement. The clinical examination showed spasms in the whole body and 3-4-minute seizures accompanying crossed eye movements. The seizure relapsed after three hours. We terminated the seizure with anticonvulsant therapy and referred the patient to our clinic. The family medical history did not contain any incidents of febrile or afebrile seizures. The patient's growth was consistent with development stages. The body temperature was 38°C and there were milds signs of dehydration during the physical examination. Other systemic and neurological findings were normal. The laboratory examination was as follows: hemoglobin count was 12,7 mg/dL; white blood cell count was 10,2 mm3/mL; blood biochemistry and urine analysis

were normal; and C-reactive protein was negative. The direct examination of stool was normal. The rotavirus antigen was negative while adenovirus antigen and PCR blood analysis for adenovirus were both positive.

As the patient continued to vomit and suffer from diarrhoea and he did not have enough oral intake, he was given intravenous fluid support. During the monitoring when he did not have any fever, the patient had another generalised short-term tonic-clonic seizure. After a 15 mg/kg of phenytoin application, we maintained a dose of 5 mg/kg/day. EEG findings were normal. Vomiting and diarrhea decreased during monitoring and the patient began to have oral intake without any further seizures. The phenytoin treatment and fluid support that had been initiated was gradually reduced and then terminated within five days. The patient was discharged with advice. The patient continued to attend the follow-ups for a year without any story of seizures.

DISCUSSION

Febrile convulsions are fever-related convulsions that can occur between 1 month and 5 years of age and they do not necessarily follow defined causes such as central nervous system infections, acute electrolyte disturbances, or intoxication nor accompany a prior history of afebrile seizures (4). They are the most common type of childhood convulsions with a prevalence rate of 4-5% (5). The pathogenesis of febrile seizures is not fully known. It is known that seizures share a genetics-related basis but the mode of inheritance is not clear. So far polygenic, autosomal dominant, and autosomal recessive patterns have been identified for febrile seizures. Environmental factors also affect the development of seizures in varying proportions. For patients with a family history of febrile convulsions, studies have reported polymorphisms in voltage-gated sodium channel subunit genes and GABA receptor subunit genes (6). In these children, susceptibility to convulsions as well as to fever is higher. Although the mechanism behind the age factor is not exactly known, it has been suggested that the balance between stimulatory and inhibitory neurotransmitters is not yet mature at around this age and that convulsions rear as a result of the changes fever causes on these neurotransmitters (7). In recent years, researchers have also focused on the role of cytokines in the pathogenesis of febrile convulsion.

It has been proposed that proinflammatory cytokines (IL-B, TNF-Alpha, IL-6) modulate neurotransmitters during inflammation or excitation and increase the risk of febrile convulsions in young children with genetic predisposition and seizure sensitivity that comes out with fever (8).

Studies on the role of viral infections in the etiology of febrile seizures has increased in recent years. The role of viral infection in the etiology of febrile seizures is proportional to the degree of induced fever. Due to their potential to invade neurotropic and central nervous systems certain viruses may cause encephalitis and encephalopathy. It is particularly known that complex febrile seizures are formed by a different mechanism from simple seizures and that it is more difficult to have a differential diagnosis between these seizures and encephalopathy (1). The frequency of Herpes Type 6 and 7 infections in febrile convulsions suggests that these viruses might have been involved in the pathogenesis of the picture. It is thought that viruses may lower the seizure threshold through fever or may even play a certain role in the creation of seizures through neurotrophic effects or reactivation. Convulsions may occur in children with infection history though they may not have fever during the seizures. Therefore, it is still not very clear if it is the fever, infections caused by the fever, or systemic reactions that leads to febrile convulsions (9). Huang YC et al's study (10) proposes that the most commonly reported symptoms of patients with adenovirus infection and central nervous system dysfunction are seizures, altered mental status, headaches, and visual hallucinations.

Patients have most often been diagnosed with febrile seizures, encephalitis / encephalopathy, afebrile seizures, or aseptic meningitis. There may be several reasons for the neurological symptoms such as seizures, altered mental status, and paresis in children with diarrhoea. The virus itself or several different structures can cause encephalitis, encephalopathy, or meningitis by passing through the blood-brain barrier. Besides, significant fluid and electrolyte loss after developing diarrhea and vomiting can also bring about these symptoms. Thirdly, fever in children with diarrhoea can cause seizures in predisposed children under 2 years of age. Because the development of central nervous system in this period is very swift, even less important stimuli can trigger seizures (11). The current adopted mechanism to explain the seizures is the third one since a significant proportion of patients do not develop severe electrolyte imbalance or high fever during diarrhoea.

With yet unexplored pathogenesis, there are still many ongoing debates with respect to the treatment and follow-up of febrile seizures. While presenting the case of a patient with adenovirus gastroenteritis who had seizures, our aim was to review the pathogenesis of the disease and open the way to discuss the idea of developing a vaccine pinpointing pathogens that cause seizures with fever and of applying this to selected groups of patients. We believe that it is possible to reduce the incidence of febrile seizures with viral vaccines that can be developed in the future.

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