



Sudden Unexpected Death in a Child with Vomiting and Diarrhea due to Intracranial Mass Lesion

Acile Kusma ve İshalle Başvuran Çocuğun İntrakraniyal Kitleye Bağlı Ani Beklenmedik Ölümü

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Abstract

Nausea and vomiting are common sequelae of a multitude of disorders that can range from mild to severe conditions. Intracranial mass lesions can occasionally present with vomiting followed by acute neurological deterioration and sudden death, although they are usually accompanied by ongoing neurological symptoms. We aimed to report an unusual presentation of an intracranial mass resulting in death to increase awareness among pediatric emergency physicians. A previously healthy 15-month-old boy presented to the pediatric emergency department (PED) with nausea, vomiting and diarrhea. He did not have any symptom of a neurological disorder. He acutely deteriorated, developed cardiopulmonary arrest and was intubated. His cranial computed tomography showed dilatation of the third and the lateral ventricles caused by a 3x4x2 cm tumor in the posterior fossa with an evidence of hemorrhage in the tumor with minimal tonsillar herniation. An emergent extra-ventricular drainage was performed to relieve elevated intracranial pressure. He did not show any improvement and died 9 hours after admission. Common symptoms in PED like nausea and vomiting are mostly due to benign etiologies such as gastroenteritis. One should always keep in mind that there may be an underlying intracranial pathology and further investigation should not be delayed.

Keywords: Child, sudden death, intracranial mass

Öz

Bulantı ve kusma hafiften şiddetli durumlara kadar değişebilen çok sayıda hastalığın ortak semptomudur. İntrakraniyal kitle lezyonları bazen kusma ve bunu takiben akut nörolojik bozulma ve ani ölümle birlikte ortaya çıkabilir. Buna genellikle devam eden nörolojik bulgular eşlik eder. Bu çalışmada çocuk acil hekimleri arasında bilinç düzeyini artırmak için ölümle sonuçlanan intrakraniyal bir kitlenin alışılmadık bir sunumunu rapor etmeyi amaçladık. Daha önce sağlıklı olan 15 aylık bir erkek çocuk, çocuk acil servisine (ÇAS) bulantı, kusma ve ishal ile başvurdu. Herhangi bir nörolojik semptomu yoktu. Genel durumu aniden bozulan, kardiyopulmoner arrest geliştiren hasta entübe edildi. Beyin bilgisayarlı tomografisinde lateral ve üçüncü ventrikülde genişlemeye yol açan, posterior fossa'da 3x4x2 cm tümör saptandı. Tümör içinde minimal tonsiller herniasyona yol açan hemorajik alan görüldü. Artmış intrakraniyal basıncı azaltmak için acil ekstra ventriküler drenaj yapıldı. Gerekli tüm müdahalelere rağmen hasta 9 saat içinde kaybedildi. ÇAS'de bulantı ve kusma gibi sık görülen semptomlar, çoğunlukla gastroenterit gibi benign etiyolojilerden kaynaklanmaktadır. Bu tip hastalarda altta yatan intrakraniyal bir patolojinin olabileceği ve daha fazla araştırmanın geciktirilmemesi gerektiği akılda tutmalıdır.

Anahtar Kelimeler: Çocuk, ani ölüm, intrakraniyal kitle

Introduction

Sudden unexpected death is defined as death occurring within 24 hours of onset of symptoms in an apparently well individuals.¹ The most common reasons include cardiovascular, respiratory, and infectious etiologies, especially in infants. Central nervous system (CNS) diseases, such as space-occupying lesions, infarction, meningitis, and

epilepsy, rarely cause sudden unexpected death in infants.² The majority of the deaths due to intracranial mass lesions occur with progression of the disease or complications of the treatment. However, in some cases, intracranial mass is not identified and patients die in a very short time following the diagnosis.^{3,4}

We present this case to stress the possibility of intracranial mass in pediatric patients undiagnosed prior to terminal

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collapse, where the child died suddenly and unexpectedly with an intracranial mass lesion.

Case

A previously healthy 15-month-old boy presented to the pediatric emergency department (PED) with nausea and vomiting. He was in his usual state of health until 2 days prior to his presentation where he began to vomit. Reportedly, his vomiting was non-bloody, non-bilious and not projectile and followed by 2 episodes of watery stool. His mother denied fever and respiratory symptoms. He did not have any neurologic symptoms. On the day of presentation, he looked tired and weak which prompted his mother to bring him to PED. Upon arrival, his Glasgow Coma scale (GCS) score was 13, temperature - 36.5 °C, respiratory rate - 38/minute, heart rate - 130/minute (tachycardia), and blood pressure was 100/60 mm/Hg. The head and neck examination revealed normocephalic and atraumatic head. His pupils were equal, round, and reactive to light. The anterior fontanel was open and flat, there was no bulging. He had sunken eyeballs and dry oral mucosa. No nuchal rigidity was noted. His chest was clear to auscultation bilaterally. His initial neurologic examination was normal. His skin examination did not reveal any rash or hypo/hyperpigmented lesion but he had decreased skin turgor and the capillary refill over three seconds. Other system findings were essentially normal.

The patient was admitted to the emergency observation unit with moderate dehydration due to diarrhea and vomiting. Prior to intravenous (IV) fluid therapy, blood tests were performed to determine the possible electrolyte imbalance. His glucose level was 96 mg/dL. Laboratory tests were as follows: white blood cells: 12.900/mm³, hemoglobin: 10 g/dL and platelets: 203.000/mm³. Blood chemistry showed the followings: blood urea nitrogen: 22 mg/dL, creatinine: 0.63 mg/dL, calcium: 10.50 mg/dL, sodium: 136 mmol/L, and potassium: 4.30 mmol/L. There was no electrolyte imbalance. Urine analysis

showed: specific gravity: 1020, white blood cell: 7/ high-power field (hpf), red blood cell: 0/hpf, leucocyte esterase: negative, nitrite: negative, and ketone: 3⁺. Venous blood gas analysis revealed the following values: pH: 7.41, pCO₂: 29.6, HCO₃: 18.7, BE: -4.2, and lactate: 2.8. Patient history, physical examination findings and laboratory findings were accepted as compensated shock. Serum physiologic therapy was given as of 20 cc/kg IV bolus immediately after blood tests were performed. Then, 100 cc/kg increased maintenance and deficit treatment were started. During the follow-up in the emergency observation unit, skin turgor and capillary refill time improved. Three hours after his admission, he had a generalized tonic-clonic seizure that lasted 30 seconds. The seizure stopped after 0.1 cc/kg IV midazolam administration. He had another generalized tonic-clonic seizure 30 minutes after the first seizure. It lasted 1.5 minutes and stopped after 0.1 cc/kg IV midazolam administration. His clinical condition deteriorated after the seizure. The vital signs of the patient were as follows: pulse rate: 184/ minute, respiratory rate: 36/ minute, and systolic blood pressure: 70 mmHg. The patient's capillary refill time was 3 seconds and cyanosis was present. The patient was given 20 cc/kg saline therapy for the second time. Dopamine infusion was initiated due to the lack of clinical response to fluid therapy and continued hypotension. There was no reason explaining hypotension in the patient with normovolemic situation (during the follow-up in the emergency observation unit, skin turgor and capillary refill time improved) and sympathetic tonus increased. The cause of hypotension was not understood after second convulsion. May be it was due to midazolam. GCS was 4 following the second seizure. He had a prolonged post-ictal period and was not able to regain his consciousness. There was no pupillary light reflex bilaterally. His deep tendon reflexes were absent. He acutely deteriorated, was intubated immediately and developed cardiopulmonary arrest 15 minutes after his second seizure.

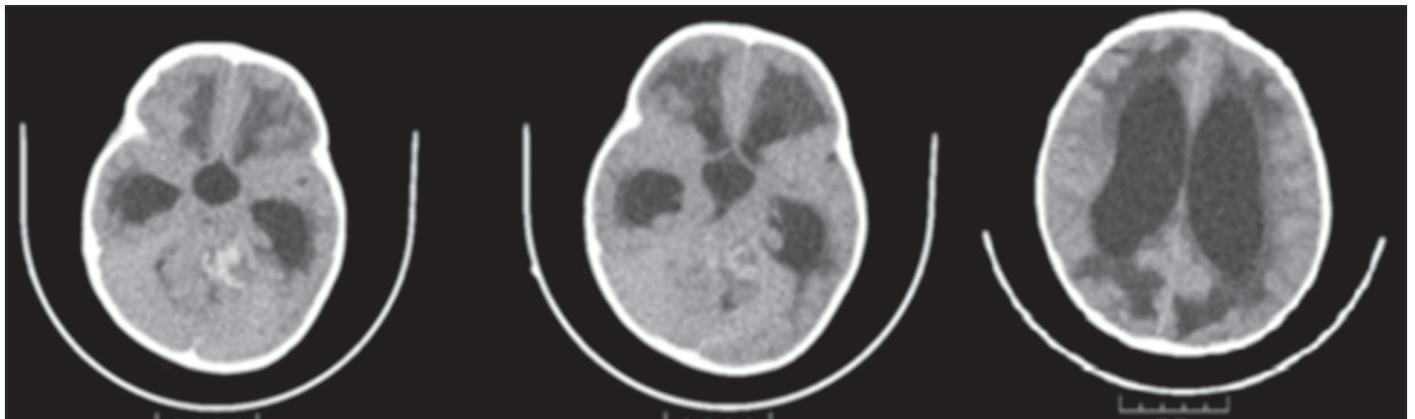


Figure 1. Patient's cranial computed tomography showed dilatation of third and the lateral ventricles caused by 3x4x2 cm tumor in posterior fossa, hemorrhage inside a tumor with minimal tonsillar herniation

Further discussion with his family revealed that the patient was reluctant to walk since last month and he became significantly inattentive since last week.

Cranial imaging was done because the patient's seizures were afebrile; no other cause or biochemical abnormality was found. Cranial computed tomography showed dilatation of the third and the lateral ventricles caused by a 3x4x2 cm tumor in the posterior fossa, and hemorrhage inside the tumor with minimal tonsillar herniation (Figure 1). The patient was started with anti-edema (methylprednisolone) treatment. He was transferred to the pediatric intensive care unit. An emergent extra-ventricular drainage was performed to relieve intracranial pressure (ICP). He died nine hours after the admission to the hospital despite aggressive respiratory and circulatory support including intubation and mechanical ventilation, administration of inotropic agents and antibiotics. Tissue diagnosis was not available because his parents did not give permission for autopsy.

Discussion

Gastroenteritis is by far the most common disorder presenting with vomiting in infants, children, and adolescents. Gastroesophageal reflux disease, gastroparesis, mechanical obstruction, anaphylaxis, Munchausen syndrome by proxy (factitious disorder by proxy), intracranial masses, peptic ulcer disease, cyclic vomiting, and diabetic ketoacidosis also may be diagnostic considerations.⁵ We present a patient who had vomiting that was misinterpreted as gastroenteritis. After his clinical deterioration, it was understood that vomiting was due to a cranial mass.

Clinical symptoms of intracranial masses differ with patient's age and type of tumor and are generally non-specific.³ In a systematic review with CNS tumors, headache (33%), nausea and vomiting (32%) and unspecified symptoms and signs of ICP (10%) were the most common symptoms.⁵ In the same study, symptoms of children under four years of age were listed as macrocephaly (41%), nausea and vomiting (30%) and irritability (24%).⁵ In these studies^{3,5}, it is examined that the symptoms accompanied by an intracranial mass and it is reported vomiting and nausea in most cases. In our case, the patient had nausea, vomiting, inattention, seizure and lethargy. Nausea and vomiting are nonspecific symptoms of ICP due to intracranial mass and are commonly present in children with CNS tumors, especially posterior fossa tumors.⁶ However, when a previously healthy patient presents to PED with these complaints, physicians immediately consider infections of the gastrointestinal system. Pediatric emergency physicians should be vigilant, and aware of the fact that vomiting and nausea are not stimulated only by one system.

In a child or infant presenting to PED with vomiting, duration of vomiting and focal gastrointestinal system symptoms are crucial for definitive diagnosis.⁵ Vomiting for more than 12 hours in a newborn, 24 hours in children under two years of age, and 48 hours in other children should be taken into consideration.^{3,7} In our case, the patient's vomiting lasted 12 hours. Sudden, unexpected death due to a CNS mass in a previously healthy individual is a rare event. Such cases have scarcely been published in the literature. Frequency of sudden, unexpected death due to a primary CNS mass is in the range of 0.11% to 0.24% in forensic autopsy series.⁸⁻¹⁴ Symptoms and death occur in patients with intracranial mass since it increases ICP and leads to cerebral herniation. A small CNS mass placed in pathophysiological critical points blocks cerebrospinal fluid (CSF) or even if it does not block CSF, it may increase ICP and lead to herniation.^{4,10,11,14} Epileptic seizures caused by CNS tumors are also considered to be a cause of sudden death.^{11,12} Especially infra-tentorial tumors can affect cardiac and respiratory centers, and lead to sudden death.¹⁵ In our case, the tumor blocked the CSF after increasing CSF pressure and eventually resulted in cerebral herniation. And it may lead to seizure, cardiac and respiratory arrest and sudden death.

The primary questions should be "Can we diagnose increased ICP before reaching the irreversible term? Can a pediatric emergency physician easily diagnose in cases having non-specific symptoms?" In some patients, symptoms are so non-specific that it is impossible to locate the mass and this may delay the diagnosis. For true diagnosis in cases with above mentioned symptoms, a CNS mass should also be kept in mind while evaluating the detailed history.

In such cases with nausea and vomiting without any other gastrointestinal system findings like abdominal defense and rebound, the most basic point is that vomiting and nausea could be a hint for neurological illnesses. Increased ICP due to intracranial masses is the main cause of nausea and vomiting. For this reason, it is necessary to search carefully for the findings of the ICP increase. For the cases with more than one or two days without diarrhea, we should investigate more and consider etiologies other than gastroenteritis.⁸

In conclusion, symptoms like vomiting and nausea that commonly encountered in PEDs are not only related with the gastrointestinal system. In cases with nausea and vomiting without any other gastrointestinal system findings like abdominal defense and rebound, other organ systems should be considered. We should keep in mind that CNS masses can lead to sudden and unexpected deaths; and clinical symptoms are usually non-specific in such cases that make definitive diagnosis difficult.

Ethics

Informed Consent: We obtained informed consent from the child's parents.

Peer-review: Externally and Internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: O.D., E.B.A., Concept: O.D., E.B.A., Data Collection or Processing: O.D., E.B.A., S.C.Ö., Analysis or Interpretation: O.D., E.B.A., S.C.Ö., Literature Search: O.D., E.B.A., E.B.A.

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