EPILEPTIC SEIZURES AND HYDATID CYST RUPTURE

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ABSTRACT

***Introduction**: Diagnosis and treatment of a hydatid cyst continues to be a problem in endemic areas, such as Turkey. Hydatid cyst rupture into the peritoneum can cause severe anaphylactic reactions. The most common cause of intraperitoneal hydatid cyst rupture is cyst rupture occurring after trauma.*

***Case presentation**: A 25-year-old male patient was brought to the emergency department with a complaint of a generalized tonic-clonic seizure while he was in the bathroom. For 3 days he had been receiving treatment for an influenza infection. His vital signs were blood pressure 140/70 mmHg, pulse 90/min, temperature 37 ºC, O2 saturation 95%. The patient's physical examination was normal, and his electrocardiogram showed a normal sinus rhythm. His cranial computed tomography was normal. His laboratory findings were normal except for hyponatremia, and fluid-electrolyte treatment was provided. During monitoring, he did not develop any acute problem, his hyponatremia resolved and he was discharged with suggestions. The patient was found dead in his bed 5 hours after discharge. The autopsy showed that his death occurred due to a hydatid cyst rupture in his liver.*

***Conclusion**: We concluded that the rupture was caused by a minimal trauma occurring during possible tonic-clonic convulsions. This condition should always be considered in a differential diagnosis in Turkey, where hydatid cyst cases are common.*

***Key words**: hydatid cyst rupture, seizure, trauma.*
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Introduction

Cyst hydatid disease is still endemic in many parts of the world, and it maintains its importance as a common health problem for both Turkey as a whole and its regions[(1)]. This disease is zoonoz, which is caused by the larval sestods of Echinococcus granulosus; about 50-70% of cysts settle in the liver. It is still endemic in Africa, the Mediterranean coast of Europe, the Middle East, South America and New Zealand.

The most common cause of intraperitonea (HC) rupture is a cyst rupture after trauma. Studies show that a traumatic HC rupture occurs mostly after a fall from height. The complications of HC rupture are abdominal pain, urticaria, anaphylaxis and sudden death[(2)].

In the case of HC disease rupturing into the peritoneum, the anaphylactic reaction rate is in the range of 1% to 12.5%, and previous studies have reported a sudden death rate of 25% for patients who developed anaphylaxis. Significant risk factors for a ruptured liver HC include age, a cyst diameter >10 cm and a superficial cyst location[(3)]. In our presentation, we emphasize that epileptic patients should also be carefully monitored for complications from seizures while they are being evaluated in the emergency department.

Case presentation

A 25-year-old male patient was brought to the emergency department with a complaint of a generalized tonic-clonic seizure while he had been in the

bathroom. The patient's relatives stated that the patient had been using analgesic antipyretic medications for 3 days due to a viral upper respiratory tract infection. The patient had known hypothyroidism and epilepsy and had been regularly taking his existing medications. He had had nausea and vomiting for a few days but was in a good general state, conscious, cooperative and oriented when he was admitted to our department. His vital signs were as follows: blood pressure 140/70 mmHg, pulse 90/min, temperature 37 °C, O2 saturation 95%. The patient's physical examination was normal. For the differential diagnosis of the seizure, the patient had a haemogram, biochemistry and blood gas tests and underwent a brain computed tomography (CT) scan and electrocardiogram (EKG).

White blood cell N(4-11 K/ul)	7.9 K/ul
Hemoglobin N (13-17.5 K/ul)	15.5 K/ul
Platelets N (150-400 K/ul)	265 K/ul
Glucose N(70-110 mg/dl)	151 mg/dl
Urea N(0-50 mg/dl)	64 mg/dl
Creatinine N(0,6-1,3 mg/dl)	1.2 mg/dl
SGOT N(<35 U/L)	46 U/L
SGPT N(<35 U/L)	15 U/L
Na N(136-145 mmol/l)	116 mmol/l
K N(3.5-5.1 mmol/l)	4.7 mmol/l

Table 1: Laboratory test results of patient.
N: normal range, SGOT: serum glutamic oxaloacetic transaminase, SGPT: serum glutamic-pyruvic transaminase.

Table 1 presents the patient's laboratory test results. The EKG showed normal sinus rhythm. His CT scan of the brain was normal (Figure 1). Hyponatremia was thought to be due to oral intake disturbance from a viral upper respiratory tract infection he had had for a few days, and fluid-electrolyte treatment was provided. Monitoring did not show any acute problem, his hyponatremia resolved and he was discharged with suggestions. Five hours after discharge, the patient was found dead in his bed. Following a complaint from the patient's relatives, the case was referred to the prosecutor's office, and an autopsy was performed.

The autopsy report indicated that death was caused by the rupture of a liver HC.

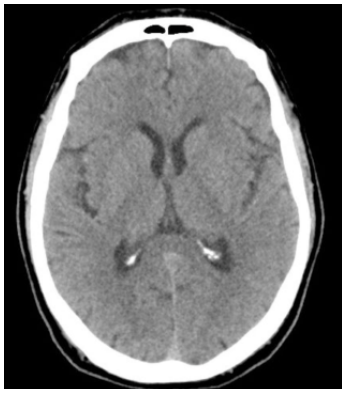

Figure 1: Cranial CT was considered to be normal.

Discussion

Patients may present to the emergency department not only because of seizures but also due to the indirect complications of seizures. Complications of epileptic seizures are often associated with injuries occurring after convulsive activity or directly resulting from sudden loss of consciousness[4]. Important complications in terms of morbidity and mortality include foreign body aspiration and cognitive decline; these are in addition to the direct effect of traumas, such as head trauma, fractures, falls, burns, drowning, motor vehicle accidents and collisions seen from epileptic seizures[5]. We believe that our patient had an HC rupture following a trauma caused by a generalized tonic-clonic seizure. The patient showed no symptoms during his monitoring and treatment in the emergency department, and similar cases have been reported in the literature[3].

A study on post-traumatic ruptures of an HC identified a total of 68 patients (38 males, 30 females) aged 8-76 years between 2000 and 2011. Despite optimal surgical and antihelmintic treatment, seven patients developed recurrence, five patients were found to have a biliary fistula, two patients had an incisional hernia, and one patient developed a gastrocutaneous fistula. One patient died of intraoperative anaphylactic shock, and one patient died of gastrointestinal bleeding and pulmonary failure[6].

Rupture of an HC spreads to the pleura, diaphragm and peritoneum. Anaphylactic shock induced by the rupture of an HC can be seen. The rupture of an HC should be considered in the case

of anaphylactic shock, the reason for which cannot be explained in endemic areas[7].

Surgical excision is a common treatment option because of the risk of anaphylaxis and recurrence of septic peritonitis. The rupture of an HC requires both emergency surgery and careful postoperative care. Patients with traumatic or spontaneous rupture of an HC must undergo emergency surgery and postoperatively receive 1 to 6 months of medical treatment (Albendezol 10-15 mg/kg/d). Recurrence may develop after unsatisfactory surgical or medical treatment[3].

In our case, the autopsy report showed that the patient developed a seizure-induced rupture of an HC, which further resulted in anaphylactic shock. The absence of signs of shock during monitoring of the patient suggests that the patient had no established shock clinic and that he developed a delayed anaphylactic shock afterwards. We failed to diagnose the patient's ruptured HC during his monitoring and treatment in the emergency department so we did not have the chance to provide surgery followed by medical treatment.

Conclusion

HC is not a disease that can be diagnosed just by a physical examination. In our case, we believe that a minimal trauma during occurring during tonic-clonic seizures resulted in the rupture of a superficial HC. The rupture of an HC should always be considered in the differential diagnosis of patients who present with trauma in Turkey where HC cases are common.

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