Hyponatremic Seizure of Carbamazepine Poisoning

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ABSTRACT

Carbamazepine is a commonly used anticonvulsant and is generally considered to be a safe drug. However, it may sometimes be responsible for some serious toxic effects and even deaths. A 14 month-old girl patient was hospitalized because of convulsion. General condition of this patient without a history of trauma was moderate and her consciousness was lethargic. The patient without other risk factors and a history suggestive of intoxication was admitted to hospital for observation of the clauses of seizure. The patient's mother was epileptic and had a history of using carbamazepine. Carbamazepine can seriously induce hyponatremia. At children's cases with evidence of poisoning but not be shown, drugs used by parents should be investigated.

Key words: *Carbamazepine, child, intoxication, convulsion*

Karbamazepin İntoksikasyonunda Hiponatremik Konvülziyon

ÖZET

Karbamazepin yaygın kullanılan antikonvülzan ilaçtır ve genellikle güvenli bir ilaç olarak kabul edilmektedir. Ancak bazen ciddi toksik etkilere, hatta ölümlerle de ilişkili olabilir. Ondört aylık kız hasta konvülsiyon geçirme şikayetiyle getirildi. Travma öyküsü olmayan hastanın fizik muayenesinde genel durumu orta, bilinç letarjikti. Konvülsiyon için bir risk faktörü olmayan hastada mevcut durumun bir intoksikasyona bağlı olabileceği düşünüldü. Hastamızın annesinin epilepsi hastası olduğu ve Karbazepin kullandığı öğrenildi. Karbazepin ciddi şekilde hiponatremi yapabilir. Çocuklarda açıklanamayan intoksikasyon durumlarında ebeveynlerin kullandığı ilaçlar sorgulanmalıdır.

Key words: *Karbamazepin, çocuk, zehirlenme, konvülsiyon*

INTRODUCTION

Carbamazepine (CN) is a drug commonly used and secure by default. However, sometimes it may be responsible for serious toxic effects and even death. CN offers pharmacological properties such as sedation, anticholinergic properties, antidysrhythmic, muscle relaxants, antidiuretic and antidepressant effects (1). In this report we wanted to remind the hyponatremic effects of CN and also wanted to emphasize the importance of suspicion intoxication with CN used by a member of the family in unexplained cases in children.

CASE

A 14-month-old girl patient was hospitalized because of convulsion. The patient without seizures before, suffers from generalized tonic-clonic type convulsion lasting up to five-minutes to a period of without fever. General condition of the patient with no trauma was moderate, her consciousness was lethargic. In the neurological examination, the light reflex has been was bilaterally obtained. The other system examinations were normal.

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Laboratory investigations were performed. The leukocyte count was 10700/mm3, the hemoglobin value was 10.7 gr/dL, and the platelet count was 328.000/mm3 in the complete blood count analysis. Biochemical parameters were as follows: the serum sodium 114 mmol/L and chloride 87 mmol/L. The spot urine sodium was 94.56 mmol/L and the urine density was 1005. The other biochemical parameters and the blood gases were regular. The brain magnetic resonance imaging was normal.

The observation of convulsions in this patient who does not present carry any risk factor can may be linked to an intoxication event. The information about the of history of epilepsy of the patient's mother and her use of CN were obtained. After the observation of a CN value of 19.14 Ug/mL in the patient's serum (normal value: 4-8 Ug/mL), the gastric lavage was performed and activated charcoal treatment at six times per day of 1 g /kg/dose and a limited fluid treatment has been used. A day after the treatment, the patient regained consciousness. Control CN level decreased to 5.64 Ug/mL, the sodium level reached to 139 mmol/L in the two days later. After the patient recovered was discharged without any complications. The patient was seen six times in six months and no symptoms have been observed.

DISCUSSION

The CN intoxication primarily influences the neurological system. Symptoms such as nistagmus, ataxia, disartria, latergia, dystonic reactions, seizures and respiratory depression can develop (2). In adults, values over 40 Ug/mL can be considered as the upper limit of serious toxicity. Coma, respiratory depression, seizures and dysrhythmia are observed in these serum values (3). In children, these peak values are varying between 27 and 35 Ug/mL (4,5). Status epilepticus is rarely observed (1).

CN causes 4.8 to 40% hyponatremia cases in patients suffering from psychiatric deficiencies, mental retardation and epilepsy (6). The hyponatremic effect of CN can be explained by different mechanisms: a) its antidiuretic effect, b) the increase of the endogenous antidiuretic hormone (ADH) release by the neurohypophyse, c) its effect on distal collector canal cell receptor independent of its ADH effect, d) the increase of the sensitivity of the receptors in the liver to the ADH in the circulating blood, e) the increase of the half-life time of the vasopressin by inhibiting the activity of the serum vasopressinase (4). In our case, the convulsion was associated with sudden onset of hyponatremia (114 mmol/L). This value reached its normal level (139 mmol/L) after two-days restricted fluid therapy. Cardiac toxicity symptoms associated CN poisoning is sinus tachycardia, hypotension, and transmission delays (2). No cardiac complication has developed in follow-up. The treatment of CN intoxication is often an auxiliary treatment. The activated charcoal efficiently binds CN. Multiple doses of activated charcoal must be used given there is no contraindication (7). Hemodialysis has no effect on acute CN intoxication. Our patient has recovered by general support and limited fluid treatment.

In conclusions, at unexplained cases of poisoning in children, the drugs used by parents should be questioned.

REFERENCES

1. *Spiller HA. Management of carbamazepine overdose. Pediatr Emerg Care 2001;17:452-6.*
2. *Perez A., Wiley JF. Pediatric Carbamazepine Suspension Overdose-Clinical Manifestations and Toxicokinetics. Ped Emerg Care 2005:21;252-4.*
3. *Hojer J, Malmlund HO, Berg A. Clinical features in 28 consecutive cases of laboratory confirmed massive poisoning with carbamazepine alone. J Toxicol Clin Toxicol 1993; 31: 449-58.*
4. *Zarate CA, Tohen M. Carbamazepine. Sadock BJ and Sadock VA (eds) Kaplan and Sadock's Comprehensive Textbook of Psychiatry. 7th. ed., volume 2. Lippincott Williams and Wilkins, Philadelphia, USA, 2000 p.2282-8.*
5. *Macnab AJ, Birch P, Maccready J. Carbamazepine poisoning in children. Pediatr Emerg Care 1993;9:195-8.*
6. *Yassa R, Iskandar H, Nastase C, Camille Y. Carbamazepine and hyponatremia in patients with affective disorder. Am J Psychiatry 1988;145:339-42.*
7. *Bertilsson L, Tomson T. Clinical pharmacokinetics and pharmacological effects of carbamazepine and carbamazepine-10,11-epoxide. An update. Clin Pharmacokinet 1986;11:177-8.*