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PAGES: 157-159

ORIGINAL PDF URL: <https://dergipark.org.tr/tr/download/article-file/81993>

Case Report / Olgu Sunusu

Hemodialysis: the life delivering therapy in a case of acute isoniazid intoxication

Hemodiyaliz: akut izoniazid zehirlenmesinde yaşam kurtarıcı tedavi

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Başvuru Tarihi/Received :
21-11-2014

Kabul Tarihi/Accepted:
27-11-2014

ÖZET

Akut izoniazid zehirlenmesi kronik şekline nazaran oldukça nadir görülür. Genellikle tekrarlayan nöbetler, metabolik asidoz ve koma ile ortaya çıkar. Acil olarak tedavi edilmezse hayatı tehdit edebilir. Tedavisinde piridoksin kullanılır. Bu yazıda akut izoniazid zehirlenmesi nedeniyle acil servise şuuru kapalı olarak getirilen ve hemodiyaliz ile yaşama dönen 16 yaşında bayan hastamızı sunuyoruz.

Anahtar Kelimeler: akut zehirlenme, hemodiyaliz, izoniazid, metabolic asidoz, piridoksin

ABSTRACT

Acute isoniazid intoxication is rarely seen compared to chronic intoxication. It usually manifests with repetitive seizures, metabolic acidosis and coma. If it isn't treated urgently, it can be life threatening. Pyridoxine is used to be given in treatment. In this article, we present a case of 16 years old woman who was brought to emergency service with unconsciousness because of the acute isoniazid intoxication and whose life was delivered by hemodialysis.

Key words: acute intoxication, hemodialysis, isoniazid, metabolic acidosis, pyridoxine

INTRODUCTION

Isoniazid (INH) is an antituberculosis drug which is the most preferred agent for prophylaxis and treatment of tuberculosis, because of its cheapness, accessibility and effectiveness (1, 2). This intense using brings about the risk of acute or chronic INH intoxication in clinical practice. Chronic intoxication cases can be detected easily, with symptoms, physical examination and laboratory findings and can be treated with using oral pyridoxine. However, intoxication with acute large doses of INH can emerge with variety of clinical conditions include repetitive seizures, metabolic acidosis with a high anion gap, coma and finally death. (1, 3).

CASE

16 years old female who has no illness, alcohol using or any drug which can make physical and psychological dependence, was brought by her relatives to emergency department with unconsciousness and convulsion. Repetitive generalize tonic-clonic convulsion and unconsciousness despite vigorous stimulation were detected at the first physical examination. There was no sign of trauma and i.v. injection. Blood pressure was 110/60 mmHg, heart beats were 120/mn and regularly, body temperature was 37.5 °C and respiration type was cheyne-stoke.

The electrocardiogram showed sinus tachycardia. Cranial computed tomography was performed and revealed nothing. She was taken intensive care unit immediately and administered anticonvulsant drugs (diazepam and phenytoin) for her convulsions but there was no recovery. Her breath got worse in a few minutes and she was intubated and connected to mechanical ventilatory support.

Laboratory findings were serum glucose 96 mg/dl, blood urea nitrogen 29 mg/dl, creatinine 0.89 mg/dl, alanine transaminase 28 U/L, aspartate transaminase 29 U/L, creatin phosphokinase (CK) 340 U/L, sodium 145 mmol/L, clorur 104 mg/dl, potassium 3.61 mmol/L, calcium 9.35 mg/dl; hemogram; wbc

5100 mm³, hb 15.1 g/dL, hct 42.2, plt 469000 mm³; PT 17.6 seconds, INR 1.72, APTT 32.5 seconds; arterial blood gases analyzes; pH 7.39, pCO₂ 23.8 mmHg, pO₂ 168 mmHg, HCO₃ 14.6 mmol/L, SO₂ 96 % (compansated metabolic acidosis with high anion gap was detected).

In second evaluation, it was learned that; 'her elderly sister was lung tuberculosis patient and INH drugs had given all family members for prophylaxis by a healthy center which they had applied one week ago. After this time, diagnosis was accepted as 'acute INH intoxication'. and hemodialysis treatment was performed after central catheterization. After the first two hours of HD treatment, convulsion stopped and did not repeat. Meantime, adequate doses of pyridoxine obtained and i.v. administration was performed. Large doses of pyridoxine were planned, but acidosis which produced by 15 g/d doses of pyridoxine was detected on blood gas measurements and pyridoxine was interrupted.

On the following days we make the diagnosis of rhabdomyolysis (CK 11216 U/L) due to repetitive seizures. Appropriate hydration, urine alkalization and controlled doses of pyridoxine were performed. In second day, she was extubated and weaned from ventilatory support. In forth day only amnesia was detected on neuro-psychiatric evaluation. In eighth day, she was discharged with complete recovery.

DISCUSSION

Limited number of cases and a few data are present regarding the acute INH intoxication and its treatment on literature. Pyridoxine-therapy that containing administration of pyridoxine equal to the amount of INH ingested and supportive care have been appeared as the most effective couple among all treatment modalities (1, 3, 4). However, seizures caused by overdose of INH are often refractory to anticonvulsant therapy and large doses of pyridoxine can be insufficient to reverse consciousness status (1, 3). In addition, losing time while waiting to stop the seizures with

conventional therapy can contribute to increase the complications (etc. overdoses of anticonvulsants, central nerve system and respiratuvar depression, cardiac arrhythmias, trauma, hypercapnia, lactic acidosis, rhabdomyolysis) and pyridoxine can cause metabolic acidosis as an adverse effect. Okutur et al. reported a case of acute isoniazid intoxication presenting with convulsions, metabolic acidosis with consequent rhabdomyolysis (5). However, patient was recovered only pyridoxine treatment- Eyüboğlu and Derinöz reported a 16-year-old male case of acute INH poisoning who was treated with pyridoxine. In their case, rhabdomyolysis was occurred on follow-up and resolved with the supportive treatment (6). Both of the patients have not required the dialysis therapy on follow-up period. In our case we observe that hemodialysis was a life saving treatment in the condition of severe INH intoxication and pyridoxine therapy may exacerbated the acidosis. Rhabdomyolysis was moderate and we managed with the supportive treatment like former two cases.

According to us, hemodialysis should be considered immediately as a prior treatment which can remove the substance from plasma effectively. Thereby the clinician can preclude the development of all these complications; especially, in cases of severe intoxication with unknown doses of INH.

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