İzmir Tıp Fakültesi Dergisi
Journal of İzmir Faculty of Medicine

Case Report
DOI: 10.57221/izmirtip.1683084

İzmir Tıp Fak Derg. 2024;4(2):63-65.

Rarely Used Hemodialysis Treatment in Encephalopathy: Case Report

Ensefalopati Tablosunda Nadir Kullanılan Hemodiyaliz Tedavisi: Olgu Sunumu

Kenan Çalışkan, Serhat Koyuncu, Oğuzhan Bol, Necmi Baykan

Kayseri City Hospital, Department of Emergency, Kayseri, Turkey

Abstract

Aim: Valproic acid (VPA) is an anticonvulsant and mood stabilizer used in various psychiatric and neurological conditions with a wide therapeutic window. This case report aims to emphasize that taking a drug in patients receiving psychiatric treatment can be toxic even if it is not at a toxic level.

Case: A 33-year-old male patient was brought to the emergency room with complaints of decreased speech, slow movements, unresponsiveness, and blank stare for several days. According to the anamnesis taken from his father, it was learned that he had a known bipolar disorder and that his current complaints had increased for 2-3 days. Vital values were stable.

The patient's laboratory and radiological examinations were within normal limits. When it was learned that VPA treatment had just started, drug level and ammonia level tests were requested considering the possibility of side effects. A diagnosis of hyperammonemia induced encephalopathy was made due to high ammonia levels.

Conclusion: While the treatment for VPA side effects is to discontinue the drug and follow up, in our patient, simply discontinuing the drug was not enough and he required hemodialysis treatment. With this case, we wanted to remind that hemodialysis can also be used in VPA-induced encephalopathy.

Keywords: Valproic acid; hemodialysis; hyperammonemia;

Öz

Amaç: Valproik asit (VPA), çeşitli psikiyatrik ve nörolojik durumlarda kullanılan geniş bir terapötik pencereye sahip bir antikonvülzan ve ruh hali dengeleyicidir. Bu olgu sunumu, psikiyatrik tedavi gören hastalarda ilacın toksik düzeyde olmasa bile toksik olabileceğini vurgulamayı amaçlamaktadır.

Olgu: Otuz üç yaşında erkek hasta, birkaç gündür olan konuşmada azalma, hareketlerde yavaşlama, tepkisizlik ve boş bakış şikayetleriyle acil servise getirildi. Babasından alınan anamnezde, bilinen bipolar bozukluk olduğu ve mevcut şikayetlerinin 2-3 gündür arttığı öğrenildi.

Vital değerleri stabil seyretti. Hastanın laboratuvar ve radyolojik incelemeleri normal sınırlardaydı. Valproik asit tedavisine yeni başlandığı öğrenilince yan etki olasılığı düşünülerek ilaç düzeyi ve amonyak düzeyi testleri istendi. Yüksek amonyak düzeyi nedeniyle hiperamonyemi kaynaklı ensefalopati tanısı konuldu.

Sonuç: Valproik asit yan etkilerinin tedavisi ilacı kesmek ve takip etmek iken, bizim hastamızda sadece ilacı kesmek yeterli olmamış ve hemodiyaliz tedavisine ihtiyaç duyulmuştur. Bu vaka sunumu ile hemodiyalizin VPA kaynaklı ensefalopatide de kullanılabileceğini hatırlatmak istedik.

Anahtar sözcükler: Valproik asit; hemodiyaliz; hiperammonemi

Corresponding Author: Necmi Baykan
Kayseri City Hospital, Department of Emergency, Kayseri, Turkey
E-mail: drnecmibaykan@gmail.com
Geliş ve Kabul Tarihi: 24.04.2025/22.05.2025

INTRODUCTION

Valproic acid (VPA) is an anticonvulsant and mood stabilizer used in various psychiatric and neurological conditions with a wide therapeutic window (1). Nausea, vomiting, diarrhea, increased appetite, weight gain, decreased leukocyte and platelet counts, insulin resistance, obesity, and menstrual irregularities can be observed due to VPA use, while hyperammonemic encephalopathy is rarely seen. Valproic acid can rarely cause hyperammonemia and delirium; failure to recognize high ammonia levels can lead to worsening of the clinical picture and death (2). In its treatment, discontinuation of the drug, low-protein diet, lactulose, and symptomatic treatment are usually sufficient, but hemodialysis may also be required (3). This case report aims to emphasize that taking a drug in patients receiving psychiatric treatment can be toxic even if it is not at a toxic level.

CASE

Written consent was obtained from the patient that his medical data could be published. A 33-year-old male patient was brought to the emergency room with complaints of decreased speech, slow movements, unresponsiveness, and blank stare for several days. The patient was not cooperative or oriented. According to the anamnesis taken from his father, it was learned that he had a known bipolar disorder and that his current complaints had increased for 2-3 days. His vital values on arrival were TA: 110/80mmHg, Nb: 72/min, Fever: 36.5°C, SpO_2: 96%, fingertip blood sugar 156mg/dl, and there were no acute pathological findings on his ECG. The patient's laboratory values were; pH: 7.46, lactate: 2.1, pCO_2: 33, HCO_3: 24.6, K: 4.2, Na: 141, Cre: 0.98, Bun: 9, AST: 18, ALT: 10, CRP: 4, WBC: 6700. The patient's cranial imaging was performed for the purpose of differential diagnosis of central pathology and no pathological findings were detected in these imagings. It was learned that the patient's anemnesis was deepened and VPA treatment was started by the psychiatrist 10 days ago. Valproic acid and ammonia level tests were requested with the preliminary diagnosis of encephalopathy secondary to hyperammonemia. The patient's valproic acid level was seen as 122 mEq/lt (50-100), and the ammonia level was 181.7 mcg/dl (27-102). The patient was transferred to the intensive care unit for follow-up purposes, considering that delirium and encephalopathy could occur due to high ammonia levels. The patient was taken to hemodialysis treatment when the clinical picture did not improve despite stopping VPA and applying symptomatic treatment during the follow-ups. The clinical picture improved after hemodialysis treatment and he was discharged with recovery on the 3rd day of intensive care follow-up.

DISCUSSION

Written consent was obtained from the patient regarding the publication of his medical data.

Valproic acid-induced hyperammonemia is a reversible adverse drug reaction that can occur at therapeutic or supratherapeutic doses of VPA in patients with normal liver function (4). As in our case, the incidence of hyperammonemia in patients with psychiatric disorders was also found to be significantly higher in another study. In addition, as in our case, serum VPA levels were shown to be significantly higher with the additional use of risperidone (5). It has been theorized that drugs such as risperidone increase VPA levels by competing for binding to albumin. With concomitant administration, risperidone binds to albumin and effectively increases ammonia levels, leaving increased VPA levels (6). Valproic acid-induced hyperammonemia varies from subclinical cases to potentially fatal encephalopathy. Although no treatment changes are required in patients with asymptomatic hyperammonemia, it is important to monitor these patients closely for symptoms of encephalopathy. There are multiple treatment methods for patients diagnosed with VPA-induced encephalopathy, the primary treatment is discontinuation of VPA. It usually results in complete recovery in most psychiatric patients (7). In Iqbal et al. patients, sufficient results were obtained by discontinuing VPA, but in our patient, the clinical picture improved after hemodialysis (8).

CONCLUSION

With this case, we wanted to remind that hemodialysis can also be used in VPA-induced encephalopathy. Clinicians who prescribe VPA should be careful about drug interactions and should be careful about the state of altered consciousness for timely intervention.

Author Contribution

Written consent was obtained from the patient that his medical data could be published.

The authors declare no conflict of interest.

The authors disclose that no grants or support resources were used.

The authors approved the final version of the manuscript.

The authors declared that this manuscript has not been published before and is not currently being considered for publication elsewhere.

Presented as a oral presentation at the 4th International Congress on Emergency and 1st International Sonocritical Congress on Emergency Medicine, 2024 in Antalya.

REFERENCES

1. Apa F, Ateşci FÇ, Varma GS. Valproik asit kullanımına bağlı gelişen hiperamonyemik ensefalopati: olgu sunumu. Klinik Psikiyatri. 2018;21:102-6.
2. Pradeep RJ. Valproate monotherapy induced-delirium due to hyperammonemia: a report of three adult cases with different types of presentation. Indian J Psychiatry. 2008;50:121-3.
3. Wu YF. Recurrent hyperammonemia associated with olanzapine. J Clin Psychopharmacol. 2017;37:366-7.
4. Sousa C. Valproic acid-induced hyper-ammonemic encephalopathy: a potentially fatal adverse drug reaction. Springerplus. 2013;2:13.
5. Hosseini H, Shafie M, Shakiba A, Ghayyem H, Mayeli M, Hassani M, et al. Valproic acid-induced hyperammonemia in neuropsychiatric disorders: a 2-year clinical survey. Psycho-pharmacology (Berl). 2023;240:149-56.
6. Rodriques-Silva N, Venâncio Â, Bouça J. Risperidone, a risk factor for valproate-induced encephalopathy. Gen Hosp Psychiatry. 2013;35:452.e5-6.
7. Chopra A, Kolla BP, Mansukhani MP, Netzel P, Frye MA. Valproate-induced hyperammonemic encephalopathy: an update on risk factors, clinical correlates and management. Gen Hosp Psychiatry. 2012;34:290–8.
8. Iqbal K, Kummamuru H, Dasari N, Koritala T, Jain NK, Deepika K, et al. A case of valproic acid induced hyperammonemic encephalo-pathy. Cureus. 2021;13:e20380.