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REHABILITATION FOLLOWING VALPROIC ACID-INDUCED HYPERAMMONEMIC ENCEPHALOPATHY (VHE) IN A PATIENT WITH INTRACTABLE EPILEPSY



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INTRODUCTION

- Valproate is an anticonvulsant widely used to treat seizures. Its use has been linked to Hyperammonemic Encephalopathy (HE), a rare complication.
- VHE is characterized by decreased levels of consciousness, focal neurologic deficits, cognitive slowing, drowsiness and lethargy.
- The pathophysiology behind these manifestations is not entirely understood.
- These findings may even occur with normal therapeutic drug levels.
- VHE is more common in patients who employ polytherapy with anticonvulsants such as phenobarbital or topiramate.

DIAGNOSTICS



CT BRAIN: STABLE LEFT FRONTAL ENCEPHALOMALACIA. NO ACUTE PROCESS.

AMMONIA LEVEL	
7/19/19	108
7/22/19	67
7/23/19	66
7/26/19	23
7/27/19	66
7/28/19	90
7/29/19	64
7/30/19	63
7/31/19	83
8/1/19	43

TEST	LEVEL
Valproic Acid Level	91
Lamotrigine Level	9.0
Topiramate Level	9.1
Levetiracetam Level	22.2

CASE PRESENTATION

- We present a case of a 29-year-old African American male with intractable epilepsy secondary to traumatic brain injury and a remote history of a left frontal CVA with associated right hemiparesis. He was initially admitted to the hospital with complaints of headache, confusion, weakness and multiple seizures. He continued to experience seizures in acute care despite treatment with 5 different antiepileptic medications, including Lamotrigine, Levetiracetam, Lacosamide, Clobazam, and Valproic Acid.
- On admission, his ammonia levels were found to be elevated and peaked at 108. Valproic acid was discontinued and he was started on Lactulose, Rifaximin, and Clobazam. The patient's ammonia levels improved to 43 at the time of admission to the inpatient rehabilitation facility (IRF), and he remained seizure-free for two days. His initial course in rehabilitation was complicated by recurrent seizures leading to a short return to acute care. However, upon readmission to IRF, he continued to have intermittent seizures leading to ongoing neurologic insult hindering his progress.
- The patient's seizures eventually subsided, and significant progress was made with extensive rehabilitation. Comprehensive physical, occupational, and speech therapies lead to meaningful improvement in FIM scores (51 to 87).

DISCUSSION

- This patient was at increased risk for developing VHE due to receiving treatment with several antiepileptics, despite therapeutic levels of Valproic Acid.
- Diagnosis of VHE is supported by elevated ammonia levels and EEG findings consistent with severe encephalopathy. EEG typically demonstrate continuous generalized slowing, a predominance of theta and delta activity, occasional bursts of frontal intermittent rhythmic delta activity, and triphasic waves.
- Management focuses on reducing ammonia levels with Lactulose and Rifaximin, as well as stopping the offending agent. Some studies also suggest carnitine supplementation, as valproic acid can lead to carnitine deficiency.

CONCLUSION

- This case demonstrates the unique challenges of a patient with intractable epilepsy with a complex and carefully balanced medication regimen complicated by continued neurologic insult during his rehabilitation course.

REFERENCES

1. Segura-Bruna, N, et al. "Valproate-Induced Hyperammonemic Encephalopathy." *Acta Neurologica Scandinavica*, U.S. National Library of Medicine, July 2006, www.ncbi.nlm.nih.gov/pubmed/16774619.