Print ISSN: 2319-2003 | Online ISSN: 2279-0780

IJBCP International Journal of Basic & Clinical Pharmacology

DOI: http://dx.doi.org/10.18203/2319-2003.ijbcp20163278

Case Report

A case of valproate induced non-hepatic hyperammonemic encephalopathy

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Received: 17 April 2016
Revised: 22 July 2016
Accepted: 06 August 2016

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ABSTRACT

Sodium valproate is a broad spectrum anticonvulsant. Valproate induced hyperammonemic encephalopathy with normal liver function is a serious, but less common entity.

Keywords: Sodium valproate, Hyperammonemia

INTRODUCTION

Sodium valproate is a broad spectrum anticonvulsant. Valproate induced hyperammonemic encephalopathy with normal liver function is a serious, but less common entity. Total of 12 cases reported so far. A 42 year old male patient with bipolar disorder on treatment with Tab. Sodium valproate 1500 mg, Tab. Citalopram 10 mg, Tab. Olanzapine 10 mg daily for the past 11 months presented with slurring of speech, memory impairment ,decreased social interaction, restlessness, tremor, and was hospitalized.

CASE REPORT

Investigations

Blood R/E-WNL, urine R/E-WNL, liver function test-WNL, renal function test-WNL, plasma ammonia level-0.5mg/dl (normal-0.02-0.08), valproate was discontinued and olanzapine and citalopram were continued. Acetyl -L carnitine orally 50 mg/kg/day was administered. Patient improved clinically after 1 month. According to WHO and Naranjo scale for assessment of adverse drug reaction a diagnosis of probable sodium valproate induced nonhepatic hyperammonemic encephalopathy was made.

Sodium valproate is a well-tolerated broad spectrum antiepileptic drug also approved for bipolar disorders, prophylaxis of migraine, neuropathic pain and social anxiety disorder. Most of the adverse effects are mild but there are also rare and severe adverse effects like hepatotoxicity, pancreatitis, teratogenicity, thrombocytopenia, hyperammonemia and encephalopathy. Valproate complexes with carnitine - a carrier molecule required for transport and oxidation of fatty acid in mitochondria facilitating its renal excretion. This leads to carnitine deficiency causing diminished mitochondrial function with inhibition of urea cycle. Valproate inhibits the activity of carbamoyl phosphate synthetase1-first enzymatic reaction in urea cycle there by hindering excretion of ammonia producing hyperammonemia. Hyperammonemia leads to an increase in glutathione level in brain which produces astrocyte swelling, cerebral oedema and encephalopathy. Plasma ammonia level should be routinely checked if patients on valproate therapy develop altered sensorium.[2]

CONCLUSION

Mental status of patients is to be periodically evaluated especially in psychiatric patients on multiple drugs.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Thottol RT, Mothalanpet AM, Mohamedali SPM, Hemalatha T. Thyagarajan. A case of valproate induced non-hepatic hyperammonemic encephalopathy. Int J Basic Clin Pharmacol 2016;5:2286-7.