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VALPROIC ACID INDUCED HYPERAMMONEMIC ENCEPHALOPATHY: REPORT OF ONE CASE

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Introduction: Valproate (VPA) is widely used for acute and maintenance therapy of bipolar disease and it is the most prescribed drug for epilepsy treatment worldwide. VPA-induced hyperammonemia is a serious and unusual advers effect of VPA treatment.
Objectives: We aimed to describe the 20 year-old-woman bipolar case of VPA induced hyperammonemic encephalopaty with therapeutic VPA levels and normal liver functions.
Methods: The case will be discussed.
Results: The patient complained of confusion, disorientation, somnolance, agitation and slurred speech at the end of the first week of VPA treatment. VPA blood level was 116.5 µg/mL (N:50-125) and ammonia level was 237 µmol/L (N:10-50). Other laboratory tests including serum transaminases (both ALT and AST) and coagulation profile (PT, PTT and INR) were normal. After discontinuation of VPA treatment and hydration, these symptoms have disappeared dramatically. After 24 hours, blood ammonia level was 80 µmol/L and patient returned the baseline mental status.
Conclusions: Hyperammonemia occurs due to inhibition of mitochondrial enzymes in the urea cycle by valproic acid. Hyperammonemia and encephalopathy can develop even at therapeutic concentrations without elevated liver function enzymes. Clinicians should be aware that VPA- induced hyperammonemia and encephalopathy is still one of the most feared side effects of VPA which may require emergent management.

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