Abstract

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Valproic acid metabolism and its effects on mitochondrial fatty acid oxidation: a review.

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Abstract

Valproic acid (VPA; 2-n-propylpentanoic acid) is widely used as a major drug in the treatment of epilepsy and in the control of several types of seizures. Being a simple fatty acid, VPA is a substrate for the fatty acid beta-oxidation (FAO) pathway, which takes place primarily in mitochondria. The toxicity of valproate has long been considered to be due primarily to its interference with mitochondrial beta-oxidation. The metabolism of the drug, its effects on enzymes of FAO and their cofactors such as CoA and/or carnitine will be reviewed. The cumulative consequences of VPA therapy in inborn errors of metabolism (IEMs) and the importance of recognizing an underlying IEM in cases of VPA-induced steatosis and acute liver toxicity are two different concepts that will be emphasized.

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