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Electrocardiographic Changes Predicting Sudden Death in Propofol Related Infusion Syndrome

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INTRODUCTION

Under physiological conditions, glucose is the major source of energy for the brain, cardiac system, and skeletal muscle. However, under stressful conditions, there is a shift towards utilizing free fatty acids as the primary energy source for the majority of living tissues. This change in energy metabolism regulates the activity of hormone-sensitive lipases in adipose tissue. It is achieved through the activation of stress hormones such as epinephrine and cortisol. Hormone-sensitive lipase, in turn, facilitates the breakdown of triglycerides into glycerol and free fatty acids. These two triglyceride components are taken up by hepatocytes. Glycerol can be used as a source for *de novo* glucose synthesis, and free fatty acids are used for mitochondrial beta-oxidation. This change in energy source is very important and aims to bring more glucose to the central nervous system and red blood cells. Beta-oxidation of fatty acids donates electrons to the electron transport chain and is also used as an energy source. Produces a biochemical intermediate used in the citric acid cycle (aka Krebs) that can be used in the synthesis of ketone bodies. Propofol is a hydrophobic substance, so it uses a lipid emulsion as a solvent. A rabbit model showed that both lipid solvents and propofol themselves contribute to the development of hyperlipidemia and hypertriglyceridemia, which are commonly considered hallmarks of PRIS.

DESCRIPTION

However, the pathogenesis of PRIS is a highly complex process and is not solely the result of solvent lipid emulsions. His current understanding of PRIS involves propofol-mediated biochemical changes that underlie host conditions (sepsis, shock, head trauma, etc.) and complex interactions with other drug combinations. It includes the fact that Propofol inhibits the activity of the mitochondrial outer membrane enzyme carnitine palmitoyl transferase I. This enzyme transfers a fatty acyl group to carnitine to form fatty acylcarnitine. Aliphatic acylcarnitines are then transported across the inner mitochondrial membrane, where their metabolites participate in the citric acid cycle, ketogenesis, and electron transport chain. Indeed, an analysis of PRIS cases has acylcarnitine accumulation in reported patients. Fatty acids tend to accumulate in various organs (such as the liver) due to defects in propofol-mediated fatty acid beta-oxidation. Thus, a PRIS patient has elevated levels of her FFA, which indeed have to promote cardiac arrhythmias. In addition, propofol is known to directly affect the mitochondrial electron transport chain.

CONCLUSION

Animal studies have that propofol cleaves oxidative phosphorylation, inactivates cytochrome c and cytochrome a/a3, and reduces the activity of complex II, complex III, and coenzyme Q electron complex chains. Clinical data show decreased cytochrome c oxidase activity and electron transport chain complex IV activity. Other factors that may contribute to the development of PRIS have reduced carbohydrate stores, high stress, and/or catecholamine administration and glucocorticoid use. Please note that a lack of carbohydrates lowers citric acid levels and slows down fat metabolism. Animal models have that propofol inhibits beta-adrenergic receptors, explaining why patients taking propofol require high doses of exogenous catecholamines. On the other hand, increased catecholamines may lead to greater clearance of propofol, requiring higher propofol doses.

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