Journal of Intensive and Critical Care
ISSN:2471-8505

Open access Opinion

The Incidence of Propofol Infusion Syndrome in Critically Ill Patients

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INTRODUCTION

Propofol is a barbiturate-free intravenous anesthetic that was approved for use in 1989. Like many general anesthetics, it produces hypnotic effects by potentiating the effects of the inhibitory neurotransmitter Gamma-Aminobutyric Acid (GABA). It is an important drug that is widely used around the world. It is currently included in the World Health Organization's (WHO) Model List of Essential Medicines, which lists what WHO considers being the most effective and safe medicines necessary to meet the needs of the health system. Propofol is commonly used to induce and maintain general anaesthesia and also has many properties that favour its use as a sedative in critically ill patients. Notably, 70% of propofol used worldwide is a sedative. These properties include: Rapid onset and end of action. Sedative, anxiolytic, antiemetic, anticonvulsant properties; beneficial anti-inflammatory and antioxidant properties. Although it has an excellent safety profile, a rare and fatal complication called Propofol Infusion Syndrome (PRIS). The first reported case of this syndrome was in Denmark in 1990 when her 3 Occurred in a 12-year-old child. Two years later, a case series was published in the British Medical Journal, reporting similar symptoms in five young children. A U.S. Food and Drug Administration study earlier this year found no association between propofol and additional deaths, but the use of propofol for pediatric sedation was abandoned shortly thereafter.

DESCRIPTION

Similar symptoms in adults were reported in her late 1990s, beginning with the case of her 30-year-old woman, who was hospitalized in 1996 with an exacerbation of asthma, followed by sedation and invasive ventilation. This tutorial is intended to summarize the clinical features, pathophysiology, and management of the condition. There is considerable evidence to suggest a linear relationship between the cumulative dose of propofol and the amount and severity of symptoms. Although the definitions above refer to high doses (0.5 mg/kg/h) or prolonged administration (48 hours), many cases with low cumulative doses have been reported. Pharmacokinetic studies suggest that propofol dosing should be based on the patient's lean body mass rather than actual body weight. (The target injection model has not been formally validated in obesity and uses different weight values in the calculations). Although there is no clear association between obesity and the development of PRIS, the dose of sedation should be calculated based on lean body mass. A case was reported in which propofol administration based on actual body weight was associated with his PRIS, possibly exacerbated by a 'relative overdose' of propofol. Steroids are known to contribute to the development of severe myopathy, possibly by inducing enzymes that cause direct muscle damage.

CONCLUSION

The rhabdomyolysis seen in PRIS may occur as a result of similar mechanisms, and there is certainly a link between steroid therapy and the development of PRIS. There is an association between the use of vasopressors and the development of PRIS. Propofol clearance is thought to be increased by increased levels of endogenous catecholamines observed in patients with brain injury or hyperactive circulation associated with sepsis or systemic inflammatory response syndrome. This is also thought to occur with high-dose inotropic or vasopressor injections. Increased clearance may reduce the therapeutic efficacy of propofol, resulting in the need to increase the dose of propofol administered. It is not clear if this is causal. PRIS-induced acidosis may impair vasomotor tone, resulting in increased vasopressors requirement.

Received:	31-January-2023	**Manuscript No:**	IPJICC-23-16094
Editor assigned:	02-February-2023	**PreQC No:**	IPJICC-23-16094 (PQ)
Reviewed:	16-February-2023	**QC No:**	IPJICC-23-16094
Revised:	21-February-2023	**Manuscript No:**	IPJICC-23-16094 (R)
Published:	28-February-2023	**DOI:**	10.35248/2471-8505.23.9.010

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Citation Schroeppel TJ (2023) The Incidence of Propofol Infusion Syndrome in Critically Ill Patients. J Intensive Crit Care. 9:010.

ACKNOWLEDGEMENT

None.

CONFLICT OF INTEREST

The author declares there is no conflict of interest.