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PROPOFOL-RELATED INFUSION SYNDROME: A REVIEW OF THE CLINICAL MANIFESTATIONS AND THE MANAGING APPROACH

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ABSTRACT

Introduction and purpose: Propofol, widely used as an anesthetic and sedative, has seen a significant rise in its application, particularly in intensive care units, over the past decade. In recent years, numerous reports have highlighted the emergence of a propofol-related infusion syndrome (PRIS) in critically ill adults and, more commonly, children receiving continuous propofol infusion for sedation or anesthesia. Propofol-related infusion syndrome (PRI) is typically characterized by progressive metabolic acidosis, hemodynamic instability, and bradyarrhythmias that may remain unresponsive to aggressive pharmacological intervention. This paper provides a comprehensive review of the literature on PRIS, examining its clinical manifestations, proposed pathophysiological mechanisms, and potential strategies for managing the syndrome should it arise in clinical practice. The authors want to emphasize a remarkable role of careful evaluation of patients treating with propofol.
Materials and methods: A thorough search of electronic databases, including PubMed and Google Scholar, was conducted using relevant keywords such as „Propofol-related infusion syndrome", „Propofol", „Propofol infusion", „Propofol infusion syndrome". All retrieved papers were evaluated for relevance based on their titles, abstracts, and full-text content. We used AI tools to grammatically correct text.

KEYWORDS

Propofol, Propofol- Related Infusion Syndrome, Propofol Infusion, Pediatrics

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Introduction.

Propofol is a widely used intravenous anesthetic drug known for its rapid onset estimated as 15-30 seconds and duration of action which is ca. 5-10minutes [1, 3]. It is commonly used for induction and maintenance of general anesthesia. Propofol easily penetrates the blood–brain barrier, leading to a swift onset of unconsciousness—sometimes occurring within a single pass through the circulatory system [3]. The rate of induction is influenced by patient-specific factors, with cardiac output being particularly significant, as well as by the infusion rate. Due to propofol's pharmacokinetic it is convenient to use it for intubated patients in intensive care units. The half-life of elimination is estimated to be between 30 and 60 minutes [17]. However, its duration of clinical effects is much shorter because of its distribution into peripheral tissues. It is recommended to use lower doses in elderly patients due to reduced clearance and volume of distribution [17]. The liver is crucial in metabolising propofol thus any decrease in liver blood flow effects efficiency of propofol clearance(3). However, according to studies pharmacokinetic does not differ significantly between patients with kidney or liver failure and those who do not suffer from these conditions [17]. When propofol is used for intravenous sedation, a single dose of it typically wears off in 5-10 minutes. It enables faster awaking after stopping the infusion which typically happen in 10-15 minutes [17]. There are several forms of administrating propofol. It can be given as a bolus or as an infusion or combination of both. The drug is administered as a milky-color lipid emulsion.

The mechanism of action is primarily based on effecting GABA-receptors. Propofol enhances the activity of GABA binding especially to the GABA-A receptors and slowing the channel-closing

time. Chloride ions flow into the neuron leading to its hyperpolaryzation [4]. It inhibits the nervous cell's ability to transmit signals which is associated with sedation, loss of consciousness and anesthesia.

Propofol may also have some inhibitory effects on excitatory neurotransmitters, such as glutamate. By dampening excitatory signals, propofol helps to further depress central nervous system activity, contributing to its anesthetic effects. Besides depressing central nervous system it also cause cardiovascular depression presenting as a hypotension and bradycardia.

Propofol has been also shown to have neuroproptective properties potentially by reducing oxidative stress and inflammation [17].

Propofol has been shown to offer neuroprotective benefits, particularly in intensive care settings for patients with head trauma. Research indicates that in cases of traumatic brain injury, continuous propofol infusion can help lower or stabilize intracranial pressure (ICP). It also supports cerebral autoregulation and promotes a surplus of cerebral blood flow relative to oxygen demand, contributing to its protective effect on brain tissue. In neurosurgical procedures like craniotomy, especially in patients with elevated ICP, propofol has been associated with a significant drop in ICP levels. It is also widely used for sedation in the ICU due to its fast onset and predictable recovery profile, making it a reliable option in various clinical situations such as mechanical ventilation, status epilepticus, and delirium prevention.

Nowadays propofol is very commonly used drug. As any medicament, administering it can cause many side effects. The most common one is associated with the infusion of propofol pain or irritation at the site of injection. Sometimes it is followed by occurring redness or swelling. High doses or rapid administration of propofol can cause hypotension. Sensitive patients can present bradycardia [1, 3, 9]. Quite common is also respiratory depression manifesting as apnea and shallow breathing [3]. Much rarely anaphylaxis and seizures can be observed. It is possible for patient to develop hyperlipidemia after long-term use of propofol due to propofol lipid-based structure. Some patients can suffer from gastrointestinal discomfort, such as nausea or vomiting, or headache after awaking from anesthesia. Prolonged using of propofol can lead to phenomenon described as propofol-related infusion syndrome (PRIS) which will be the topic of this article. Due to many potential side effects, propofol should be administered only by experienced or well-trained healthcare professionals. It is also important to monitor vital signs during applying propofol and after the anesthesia wears off.

Discussion

Propofol-related infusion syndrome is a consequence of prolonged administration of propofol for sedation. It occurs mainly in pediatric population but can also regard adults. Representative for PRIS is developing severe bradycardia and cardiac arrhythmias, rhabdomyolysis considered as late- onset manifestation of PRIS often followed by renal failure, liver dysfunction and hypotension in some cases leading to shock [1].

Abnormalities that can be noticed in laboratory tests are high anion gap metabolic acidosis, hypertriglycerihdemia, lipaemia and hyperkalaemia [4, 5]. Retrospective study conducted by Wai Kin et al. estimated that incidence of PRIS is 2.9% and mortality rate is 36.8% [4].

If PRIS is not recognize early, it can lead to multi-organ failure and in consequence death. Therefore propofol infusions should always be followed by blood test and monitoring vital signs. There are some critical factors which extremely increase risk of PRIS. The most important one is high dose or long-term administration of propofol. It is recommended not to give more than 4 mg/kg/hour or for longer than 48 hours to avoid PRIS [8, 16, 19]. Studies showed that it is better to switch to alternative drugs if needed [1, 6].

As there was written the most endangered group is younger age population. It is more common for children to experienced neurodvelopmental damage followed by behavioral changes due to PRIS. Furthermore PRIS is fatal more frequently in patients under 18 years old [11]. Studies showed that there is no link between the sex of the patient and frequency of developing [10] In Pepperman and Macrae's retrospective study on one hundred and six patients it was observed that 78% of metabolic acidosis incidents were noticed in children under 3 years old [11, 15].

One of the greater predisposing factors for PRIS is ongoing critical illness such as sepsis, pancreatitis or multiple trauma [18]. These conditions are associated with neuroendocrine stress response: increasing catecholamines and glucocorticoids levels. These hormones are responsible for the regulation of the enzyme activity of lipase which promotes the conversion of triglycerides into glycerol and free fatty acids [1, 6]. This reaction is signifiant for the pathophysiology of PRIS what will be explained in the next section of this article.

Another major risk factor is the usage of corticosteroids. It is proved that they activate the ubiquitin-proteasome pathway what leads to muscle rapture. Furthermore some sources report that corticosteroids alter gene transcription, which lowers mitochondrial energy output by changing the mitochondrial pathways and functions. These reactions are crucial for the development of PRIS [2, 6].

Long-term exposure to propofol can lead to mitochondrial respiratory chain defects. In consequence of that patients with congenital metabolic defects are in a group of risk for developing PRIS [1].

The pathophysiology of Propofol Infusion Syndrome (PRIS) remains complex and controversial, with several mechanisms proposed to explain its development. The syndrome is primarily linked to mitochondrial dysfunction and energy imbalance, driven by the effects of propofol on mitochondrial processes. One of the leading theories suggests that propofol interferes with the electron transport chain (ETC), particularly at complexes I and IV, cytochrome C, and acylcarnitine transferase within the mitochondria. This disruption hinders oxidative phosphorylation, a critical process for ATP production. Under normal conditions, electrons from reduced coenzymes NADH and succinate generated by the citric acid cycle are transferred through the ETC to generate a proton gradient across the inner mitochondrial membrane, driving ATP production via ATP synthase [6]. Propofol impairs this process by blocking key components of the ETC, lowering mitochondrial energy production. This results in reduced ATP synthesis, cellular hypoxia, and the accumulation of metabolic byproducts, including acidosis [1, 7, 20].

Propofol affects fatty acid metabolism in several ways. Propofol infusion increases free fatty acid (FFA) levels by promoting lipolysis in adipose tissues. FFAs are used as an energy source during periods of metabolic stress, but propofol disrupts their utilization. FFAs are transported into mitochondria for beta-oxidation, which generates acetyl-CoA for the citric acid cycle. However, propofol impairs the transport of long-chain fatty acids by inhibiting carnitine palmitoyltransferase I (CPT-I), a critical enzyme for moving fatty acids into the mitochondrial matrix [6, 9]. This inhibition leads to an accumulation of long-chain fatty acids in the mitochondria, exacerbating dysfunction in the respiratory chain and contributing to cellular hypoxia and metabolic acidosis [1, 6, 7]. Additionally, propofol is thought to act as an uncoupling agent, interfering with the coupling of electron transport and ATP synthesis. This process, referred to as mitochondrial uncoupling, leads to energy wastage and further depletion of ATP reserves, aggravating the energy crisis in cells [6]. Another critical factor is the accumulation of intermediate fatty acid metabolites, such as acylcarnitines, which further impair mitochondrial function. These metabolites are formed due to incomplete fatty acid oxidation [16]. Their accumulation, alongside the inhibition of ATP

production, creates a vicious cycle of metabolic dysfunction [9]. The buildup of these intermediates also contributes to the production of reactive oxygen species (ROS), which can damage cellular structures and exacerbate oxidative stress [1, 6, 8]. The mitochondrial dysfunction and energy imbalance induced by propofol particularly affect muscle and cardiac cells. In skeletal muscle, impaired fatty acid oxidation and ATP production lead to muscle necrosis and rhabdomyolysis, a hallmark feature of PRIS [5, 8]. This muscle injury can release myoglobin into the bloodstream, leading to kidney damage. Similarly, the heart is vulnerable to energy depletion, leading to arrhythmias and other cardiovascular disturbances [1, 2].

In certain cases, genetic predisposition may exacerbate the development of PRIS. Some patients with underlying mitochondrial defects may be more susceptible to mitochondrial dysfunction under stress, such as during surgery or severe illness. These individuals may develop PRIS even after relatively low doses or short-term administration of propofol [9].

In summary, the pathophysiology of PRIS involves a combination of mitochondrial dysfunction, impaired fatty acid metabolism, and energy imbalance. Propofol's effects on the electron transport chain and fatty acid oxidation pathways lead to decreased ATP production, cellular hypoxia, metabolic acidosis, and widespread organ damage, particularly in muscles and the heart. While the exact mechanism is still debated, mitochondrial dysfunction—especially involving the respiratory chain—appears to be a central factor in the development of PRIS.

Ichikawa et al. tried to determine a correlation between plasma propofol concentration and lactic acidosis. In their case report the plasma concentration was over 12 times higher then the predicted measurement calculated on the basis of Marsh model. It is suggested that this result was a consequence of delayed propofol clearance. [20].

Clinical manifestations

The onset of PRIS is often insidious which symptoms gradually worsening over hours or days. At first the cardiovascular system is involved. Propofol antagonizes beta-adrenoreceptors and calcium channels leading to increasing sympathetic tone and affecting in bradycardia, arrhythmias or even asystole [1, 4, 7]. Furhermore FFAs crucial in PRIS mechanism of action have pro-arrhythmic effect what enhances its cardiovascular impact. There are many possible cardiovascular presentations such as hypotension, ventricular tachycardia, ventricular arrhythmia, right bundle branch block, atrial fibrillation, cardiogenic shock, brugada-like syndrome, elevated ST segment or widening of QRS complex in ECG [5, 9, 16].

High doses of lipid deposition of propofol and FFAs can cause hepatic changes such as steatosis and liver failure. In laboratory tests it can be observed elevated levels of liver enzymes (ALT, AST) and gamma-glutamylo-transferase (GGT), hiperlipidemia, hipertriglicerydemia. Sometimes the liver failure can be consequence of cardiovascular disorders [1, 2, 4, 5, 10]. The most common renal component of PRIS is renal failure and acute kidney injury [16, 20].

It is suggested that rhabdomyolysis occurring in PRIS is a consequence of decreased oxygen supply and secondary to that anaerobic metabolism. In laboratory tests it can be observed myoglobinuria and increased serum creatinine level [7, 16]. Guitton et al. in their study described an epileptic focus localized in occipital lobe in electroencephalography (9).

In severe cases PRIS can lead to multi-system organ failure manifesting as enlarged liver, oliguria, anuria or even be fatal [7, 8, 12]. Autopsy finding include hepatic steatosis, myoglobin casts in the renal tubules and rhabdomyolysis or peripheral and cardiac muscles. In muscle biopsy it was observed focal necrosis, basophilic fibers and histocytes and decreased complex IV activity [11].

Management and treatment

The most important aspect in PRIS management is preventing its occuring. Propofol should be administered as short as possible. Patients receiving this medicine should be also monitored especially for metabolic acidosis, kalcium level creatine kinase level or developing arrhythmias or progressing heart failure. If the test results are abnormal, the dose should be reduced or the medication should be discontinued [5]. If patient requires prolonged sedation it was suggested to consider altering the medicine [1, 6, 10, 13]. Some authors suggested a dextrose infusion [13, 14, 15]. Preventing or treating the propofol infusion related syndrome involves restoring peripheral oxygen delivery. Besides monitoring laboratory parameters it is suggested, especially for smaller patients, to use ultrasound to assess aortic flow, computerized pulse waveform analysis, or impedance-based cardiac output monitors in combination with echocardiography to assess hemodynamics [15]. When lipaemia occurred the potential link between increased lipids level and impaired liver function should be considered. Administering carbohydrates can help maintain lipid metabolism, provided the liver is functioning properly. An important part is treating primary diseases such as hypoperfusion states, sepsis, hypermetabolic states, hypoxia. D. Dauwe et al. suggested ECMO as a temporary cardiac support in PRIS-induced cardiogenic shock or arrest (19). Based on case reports from Levin et al. PRIS symptoms can be managed by therapeutic plasma exchange [22, 23].

Conclusions

Propofol is now one of the most commonly used drug in anesthesia and intensive care units. Its efectiveness is remarkable due to its rapid onset and short-acting nature. Moreover its safety profile is really favorable. However prolonged administration, use in children or in critical ill patients creates risk of propofol-related infusion syndrome. Which is rare but potentially fatal complication. Therefore clinicians should use propofol carefully and provide proper monitoring during the treatment. Intensivists must be alert to recognize PRIS symptoms with dispatch to implement appropriate management which involves prompt discontinuation of propofol, supportive care, and treatment of underlying conditions. However it should be entered into consideration that symptoms of PRIS are unspecific and could appear in plenty of constellations thus its recognition is quite challenging. Further research is needed to fully understand the molecular basis of PRIS and to develop standardized protocols for prevention, early detection, and effective treatment.

Authors' contribution:

Conceptualization: JM, MM Methodology: GŁ
Software: KD, MM Check: JK, MB
Formal analysis: PK, KD Investigation: Resources: PK, JK Data curation: AN, KM
Writing- rough preparation JZ
Writing - review and editing: GŁ, JZ Visualization: AN, JM
Supervision: KD, KM Project administration:

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