

Can Dexmedetomidine Prevent the Unwanted Hemolytic Events in the Glucose-6-Phosphate Dehydrogenase-deficient Person Undergoing Cardiac Surgery?

Sir,

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most frequent human enzyme deficiency, affecting at least 400 million people globally. The G6PD enzyme plays an essential role in the production of nicotinamide adenine dinucleotide phosphate (NADPH), which is an intracellular antioxidant enzyme. The production of NADPH is impaired in patients who suffer from G6PD enzyme deficiency. This can predispose erythrocytes to oxidative metabolites stress and ultimately causing hemolysis.^[1] For clinicians, the most important point to consider about G6PD deficiency is the asymptomatic nature of this abnormality, such that G6PD-deficient patients develop clinical manifestations of hemolysis only under specific situations such as infection, food (fava beans), certain drugs, diabetic ketoacidosis, neonatal age, and stressors with oxidative actions.^[2] In the list of anesthetic drugs that should be avoided are propofol, benzodiazepines, ketamine, and fentanyl.^[3] Therefore, one of the most important considerations that anesthesiologists have to contemplate in the management of G6PD deficiency patients is avoiding these stressors and triggering factors in addition to reducing the pain and stress of surgery by selecting the suitable anesthetic and analgesic medications.^[4]

Cardiac surgery is associated with a considerable increase in systemic oxidative stress and free radicals. The erythrocytes of a G6PD-deficient patient undergoing cardiac surgery are incapable of scavenging these free radicals, resulting in hemolysis; hence, G6PD deficiency can trigger the destructive effect of free radicals in such patients.^[4] Due to the production and accumulation of toxic peroxides, injection of some anesthetic and analgesic agents before, during, and after cardiac surgery can initiate the oxidation of red blood cell membrane and hemoglobin in patients with G6PD deficiency, and thus, these drugs might have the potential to evoke acute episode of hemolysis.^[5] As a result, the induction of anesthesia with oxidative agents in G6PD deficiency patients undergoing cardiac surgery multiplies the risk of hemolysis.^[6] Nevertheless, the evidence regarding the effects of currently used anesthetic and analgesic agents on G6PD enzymatic activity in patients undergoing cardiac surgery is lacking, and selecting the best anesthetic and analgesic agent in these patients is controversial.^[4,7] Consequently, this inconsistency and uncertainty concerning conflicting data may limit the use of analgesics for sedation and pain control in patients with G6PD deficiency.

Recently, some sedative agents with analgesic and anesthetic properties such as dexmedetomidine (DEX)

have shown promising effects in minimizing the potential for oxidative stress and hemolysis during before, during, and after surgery in G6PD-deficient infants.^[3] DEX, a selective alpha₂-adrenoceptor agonist, is a sedative agent with concurrent anesthetic, anti-anxiety, antioxidant, and opioid-sparing effects. It has been reported that DEX can significantly decrease the levels of inflammatory cytokines and free radicals during cardiac surgery.^[8] In addition, it has minimal effects on the respiratory and cardiovascular functions after surgery, which facilitates rapid recovery of patients. To prevent the unwanted events of hemolysis in the G6PD-deficient person undergoing cardiac surgery, it seems that DEX has potential to be a good candidate for sedation, induction, and maintenance of anesthesia, as well as for analgesia after surgery. However, owing to the dearth of evidence, it is not possible to make strong evidence-based recommendations on the use of DEX in G6PD-deficient person. As a result, future research should be aimed at providing answers to this question: Can perioperative DEX prevent the unwanted hemolytic events in the G6PD-deficient person undergoing cardiac surgery?

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Conflicts of interest

There are no conflicts of interest.

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