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Role of Dexmedotomedine in Obestetic and Gynecology

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Abstract:

Dexmedetomidine is a new generation highly selective α2-adrenergic receptor (α2-AR) agonist that is associated with sedative and analgesic sparing effects, reduced delirium and agitation, perioperative sympatholysis, cardiovascular stabilizing effects, and preservation of respiratory function. The aim of this review is to present the most recent topics regarding the advantages in using dexmedetomidine in clinical anesthesia and intensive care, while discussing the controversial issues of its harmful effects.

Keywords: dexmedetomidine, intensive care unit sedation, α2-adrenergic receptor agonist, vagomimetic action, anxiolytic, analgesic, sympatholytic

Introduction

α2-adrenergic receptor (α2-AR) agonists have been successfully used in several clinical settings in view of diverse actions which include sedation, analgesia, anxiolysis, perioperative sympatholysis, cardiovascular stabilizing effects, reduced anesthetic requirements, and preservation of respiratory function. Dexmedetomidine is a relatively new drug approved at the end of 1999 by the Food and Drug Administration (FDA) for humans use for short-term sedation and analgesia (<24 hours) in the intensive care unit (ICU). Dexmedetomidine is a useful sedative agent with analgesic properties, hemodynamic stability and ability to recover respiratory function in mechanically ventilated patients facilitating early weaning. Besides being a new modality of sedation and analgesia in ICU patient management, it has been studied in several other perioperative settings, which will be discussed. α2-AR agonists produce clinical effects after binding to G-Protein-coupled α2-AR, of which there are three subtypes (α2A, α2B, and α2C) with each having different physiological functions and pharmacological activities. These receptor subtypes are found ubiquitously in the central, peripheral, and autonomic nervous systems, as well as in vital organs and blood vessels.

Dexmedetomidine is 8 to 10 times more selective towards α2-AR than clonidine. Locus ceruleus of the brain stem is the principal site for the sedative action and spinal cord is the principal site for the analgesic action, both acting through α2A-AR. In the heart, the dominant action of α2-AR agonists is a decrease in tachycardia (through blocking cardioaccelerator nerve) and bradycardia via α2A-AR (through a vagomimetic action). In the peripheral vasculature, there is sympatholysis-mediated vasodilatation and smooth muscle cells receptor-mediated vasoconstriction. The mechanism for the antishivering and diuretic actions has yet to be established firmly. The responses to activation of the receptors in other areas include decreased salivation, decreased secretion, and decreased bowel motility in the gastrointestinal tract; contraction of vascular and other smooth muscle; inhibition of renin release, increased glomerular filtration, and increased secretion of sodium and water in the kidney; decreased intraocular pressure; and decreased insulin release from the pancreas. Combining all these effects, dexmedetomidine avoids some of the side effects of multiagent therapies.

Dexmedetomidine undergoes almost complete biotransformation through direct N-glucuronidation and cytochrome P-450 (CYP 2A6)-mediated aliphatic hydroxylation to inactive metabolites. Metabolites are excreted in the urine (about 95%) and in the feces (4%). Dose adjustments are required in patients with hepatic failure because of lower rate of metabolism.

Dexmedetomidine is used as an adjuvant for premedication, especially in patients susceptible to preoperative and perioperative stress because of its sedative, anxiolytic, analgesic, sympatholytic, and stable hemodynamic profile. Dexmedetomidine decreases oxygen consumption in intraoperative period (up to 8%) and in postoperative period (up to 17%). Premedication dose is 0.33 to 0.67 mg/kg IV given 15 minutes before surgery (this dose minimizes side effects of hypotension and bradycardia). Dexmedetomidine attenuates hemodynamic stress response to intubation and extubation by sympatholysis. In view of absent respiratory depression, it can be continued at extubation period unlike other drugs. Dexmedetomidine potentiates anesthetic effect of all the anesthetic agents irrespective of the mode of administration (intravenous, inhalation, regional block). Intraoperative administration of dexmedetomidine in lower concentrations has reduced the requirement of other anesthetic agents; fewer interventions to treat tachycardia; and a reduction in the incidence of myocardial ischemia. However, side effects like bradycardia and hypotension are limitations to its use necessitating need for pharmacological rescue therapy. These effects may be attributed to the combined properties of volatile anesthetics such as vasodilatation and myocardial depression. Dexmedetomidine administered in high concentrations may cause systemic and pulmonary hypertension because of direct peripheral vascular effects or may compromise myocardial function and blood pressure.

Locoregional analgesia

Highly lipophilic nature of dexmedetomidine allows rapid absorption into the cerebrospinal fluid and binding to α2-AR of spinal cord for its analgesic action. It prolongs the duration of both sensory and motor blockade induced by local anesthetics irrespective of the route of administration (e.g., epidural, caudal, or spinal). Dexmedetomidine though enhances both central and peripheral neural blockade by local anesthetics, however, the peripheral neural blockade is due to its binding to α2A-AR. Dexmedetomidine has been successfully used in intravenous regional anesthesia (IVRA), brachial plexus block, and intraarticularly. Addition of 0.5 μg/kg dexmedetomidine to lidocaine for IVRA improves quality of anesthesia and improves intraoperative-postoperative analgesia without causing side effects. Dexmedetomidine added to levobupivacaine for axillary brachial plexus block shortens the onset time and prolongs the duration of the block and postoperative analgesia.

Intraarticular dexmedetomidine in patients undergoing arthroscopic knee surgery improves the quality and duration of postoperative analgesia._Dexmedetomidine activates α2-AR in the spinal cord reducing transmission of nociceptive signals like substance P. It has significant opioid sparing effect and is useful in intractable neuropathic pain.

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