Perioperative Renal Protection during Cardiac Surgery: A Choice between Dopamine and Dexmedetomidine

The physiological mechanism in the body tries to maintain sufficient pressure to perfuse tissues for their cellular functions. The decrease in perfusion pressure may affect the physiological processes in the cells. It is observed that a perfusion pressure lower than 50, 60, or 70 mmHg affects brain, heart, and kidney, respectively. So, with reduction in this pressure, it is the kidney which gets easily affected. There are many chances of hemodynamic instability and decrease in perfusion pressure during cardiac surgery. Moreover, cardiac surgery involves the use of cardiopulmonary bypass machine and patient is exposed to renal hypo perfusion, which is predisposing risk factor to renal failure. This leads to derangement of kidney function. Renal injuries are common after cardiac surgeries and this contributes to increase in morbidity and mortality.[1] Therefore, since beginning of cardiac surgery, cardiac anesthesiologists are worried about renal derangement. One of the main objectives of a cardiac anesthesiologist is to protect kidney during cardiac surgery to decrease morbidity and mortality. Many investigators have tried various maneuvers to protect kidney during surgery. It includes pharmacological or nonpharmacological procedures. Pharmacological interventions include use of dopamine, diuretics, calcium channel blockers, angiotensin-converting enzymes inhibitors, N-acetyl cysteine, atrial natriuretic peptide, sodium bicarbonate, antioxidants, and erythropoietin, whereas nonpharmacological procedures such as maintaining high mean arterial pressure, good hemoglobin, adequate oxygen delivery, keeping normal hydration, and avoiding renal toxic drugs have been found to be beneficial. Dopamine has enjoyed the legacy to be a favorable drug to increase urine output in cardiac patients for a long time. Recently, dexmedetomidine is investigated for renal protection.[2]

Dopamine exerts its effects on D1 and D2 receptors resulting in renal vasodilatation and natriuretic effects augmenting renal blood flow and diuresis. Previously, many physicians preferred it as first-line vasopressor in hypotensive patients. They believed that dopamine with its beta and alpha receptors effects may be useful. With dopamine use, urine output increases. It seems that once the patient has adequate urine output, he has good cardiac output too. However, the use of dopamine is found detrimental. However, dopamine does not improve renal function. It impairs mucosal blood flow by aggravating reduced gastric motility, worsens splanchnic oxygenation, impairs gastrointestinal function, impairs endocrine/immunological systems, suppresses the secretion and functions of anterior pituitary hormones, and blunts the ventilation drive.[3] It induces central hypothyroidism, aggravates catabolism, and induces renal failure in patients. Recent studies indicate that there are no benefits of dopamine in preventing renal failure.[4]

The dexmedetomidine is alpha 2 adrenergic agonist and has anti-inflammatory and sympatholytic actions. It is used for sedation. It provides a semi-arousable and cooperative patient. It is an opioid sparing drug and so there is no risk of respiratory depression. It preserves the antioxidant enzyme levels and reduces the toxic oxidant metabolite levels. The use of dexmedetomidine is found with decreased proinflammatory cytokines, higher creatinine clearance, and lower serum cystatin C.[5] It has been found to provide protection from oxidative injury caused by ionizing radiation.[6] It reduces the cell death and decreases the release of plasma high mobility group protein B1 which signals to provide renal protection.[7] Its use has been found with lower reoperation rate, neurological injury, decreased hospital stay, and decreased mortality in cardiac surgery patients.[8] It decreases ischemic-reperfusion injury (I/R)[9] and improves renal function. However, there is a contradictory study also. The histological examination of kidneys of rats subjected to an experimental I/R model revealed that dexmedetomidine did not protect kidneys from I/R.[10]

More studies are required to come to the conclusion and we hope that in the near future, we will find out some definite maneuver to provide more protection to kidney and that way we will be able to decrease morbidity and mortality in our patients. More studies with higher methodological qualities are required to make a definite conclusion.

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Quick Response Code:

Website: www.annals.in

DOI: 10.4103/aca.ACA_151_17

How to cite this article: Rawat RS, Maashani SMA. Perioperative renal protection during cardiac surgery: A choice between dopamine and dexmedetomidine. Ann Card Anaesth 2018;21:4-5.