

## Specificities of perioperative acute kidney injury

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The incidence of perioperative acute kidney injury (AKI) is highly variable, depending on the type of surgery, on the type of patient and on the definition of AKI. The risk of postoperative renal dysfunction is high after cardiac surgery (30-40%), severe abdominal surgery (20-35%), thoracovascular surgery (15-20%), and emergent surgery. Postoperative AKI is associated with an increased mortality and morbidity (especially cardiovascular complications). The pathophysiology of perioperative AKI is complex and associates hemodynamic and non-hemodynamic factors (nephrotoxic drugs, systemic inflammation and obstruction).

Prevention of perioperative AKI consists essentially to make an early diagnosis, to optimize the hemodynamic parameters and to avoid the exposition to nephrotoxic drugs. Therefore, the first step is to identify patients at risk of AKI. Major risk factors are arterial hypertension, previous chronic renal disease, diabetes, chronic vascular disease and sepsis. The precise place of renal biomarkers aiming to predict the risk of perioperative AKI requires more clinical evaluation.

The only curative treatment of perioperative AKI is based on the causal therapy (such as sepsis, hemorrhage or shock) as soon as possible. The first non-specific treatment is to maintain the hemodynamic parameters aiming to avoid arterial hypotension, hypovolemia and positive water balance. Therefore, it is strongly recommended to use a perioperative hemodynamic monitoring, regardless the device, to achieve these goals in patients or surgery at risks. Such a treatment is based firstly on fluid resuscitation followed by the administration of vasoactive drug (noradrenaline) if necessary. It is now admitted that neither "restrictive" nor "liberal" fluid infusion can be recommended. The goal of fluid resuscitation is to reach "the optimal hemodynamic status" allowing to maintain arterial pressure and normovolemia while avoiding water congestion. Balanced crystalloids must be preferred in case of large volume of fluid resuscitation. The second treatment consists to avoid, if possible, the administration of nephrotoxic agents. The risk of contrast iodinated induced nephropathy in the perioperative period and in critically ill patients has been overestimated for a long time. Recent data showed that this risk is limited due to a lesser nephrotoxicity of the contrast medium and the lower volume administered. On the other hand, the nephrotoxic effect of non-steroidal anti-inflammatory agents and of renin-angiotensin blockers seems to be not negligible in surgical patients. Despite the absence of real increased mortality, all data confirms that these drugs increase the incidence of perioperative arterial hypotensive episodes.

In conclusion, the incidence of perioperative AKI is frequent. Its prevention is based first on the identification of risk factors. The major efficient preventive strategy aims to reduce all episodes of renal injury such as hypotension, hypovolemia and administration of nephrotoxic drugs when possible.

### References

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