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The Meaning and Management of Perioperative Oliguria

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Summary Statement:

Perioperative oliguria is an alarm signal. The initial assessment includes closer patient monitoring, evaluation of volemic status, risk-benefit of fluid challenge or furosemide stress test, and investigation of possible perioperative complications.

Urine output is often considered a critical target in patients undergoing surgery and is the primary trigger for fluid boluses. Low urine output or oliguria meeting criteria for acute kidney injury (AKI) have been associated with complications including increased length of hospital stay, costs, and mortality.[1,2]

The scope of this review is to discuss the determinants and consequences of perioperative oliguria and guide physicians' decision-making from the operating room to the intensive care unit (ICU) or the ward on how to manage oliguria in surgical patients. We will discuss the factors regulating urine output, propose a pragmatic approach to oliguria management during and after surgery, and describe how a search for perioperative complications (*e.g.*, sepsis, bleeding, pulmonary embolism, tamponade) should be considered a priority when facing a patient with persistent postoperative oliguria.[1–7]

Perioperative Oliguria: Definition and Association with Prognosis

Post-operative Oliguria

The definition of acute kidney injury (AKI) based on Kidney Disease Improving Global Outcomes (KDIGO) criteria includes a urine output lower than 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for more than 6 hours (Table 1).[3,8,9]

The risk associated with oliguria varies however with the duration and degree of oliguria. In this line, a threshold of 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for more than 6 hours may be too liberal

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with regards to the association with mortality.[9] A cohort study including over 15,500 patients from both medical and surgical ICUs reported that thresholds of urine output < 0.2 $ml \cdot kg^{-1} \cdot h^{-1}$ over 3 to 6 hours and 0.3 $ml \cdot kg^{-1} \cdot h^{-1}$ for 12 and 24 h had stronger associations with 90-day mortality compared to < 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for 6 hours.[9] In another study including critically ill patients, the risk of death increased with urine output < 0.3 $ml \cdot kg^{-1} \cdot h^{-1}$ for 3 hours.[2] More prolonged periods of oliguria were associated with higher risk of death.[2,7,10] To summarize, while the current definition of AKI requires a urine output below 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for more than 6 hours, this definition might lack specificity for kidney damage and a threshold < 0.3 $ml \cdot kg^{-1} \cdot h^{-1}$ over 3 hours is associated with worse outcomes (Table 1).[2,7,9,10]

Intraoperative Oliguria

Evidence regarding the association between intraoperative urine output and outcomes is scarce. A few studies have associated intraoperative oliguria with post-operative AKI. Most studies of intra-operative oliguria have not found significant association with mortality.[1,4,11,12] Among 3,560 patients undergoing abdominal surgery, the inclusion of intraoperative urine output < 0.3 $ml \cdot kg^{-1} \cdot h^{-1} \geq 1$ hour in a statistical model to predict postoperative AKI (defined by KDIGO criteria, based on increase in serum creatinine of ≥ 26.5 $mmol.liter^{-1}$ within 48 h or ≥1.5 times baseline within 7 days after surgery) led to an improvement in the risk stratification (net reclassification improvement 0.159, 95% CI: 0.049 to 0.270, p=0.005).[11] In a meta-analysis, including 17,148 patients undergoing non-cardiac surgery, intraoperative oliguria increased postoperative AKI risk (urine output < 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$) (odds ratio[OR]: 1.74, 95% CI: 1.36 to2.23, p < 0.0001).[12] In the RELIEF trial, intraoperative oliguria (urine output < 0.5 $ml \cdot kg^{-1} \cdot h^{-1} \geq 1$ hour) was associated with postoperative AKI (risk ratio: 1.38, 95% CI: 1.14 to 1.44, p < 0.001).[1] However, the positive predictive value of intraoperative oliguria was low (25.5%), and 74% of the patients with postoperative AKI did not present intraoperative oliguria.[1] Another single-center retrospective study including 165 patients undergoing elective abdominal surgery showed no association between intraoperative oliguria (urine output < 0.5 $ml \cdot kg^{-1} \cdot h^{-1} \geq 1$ hour) and post-operative AKI (p=0.772).[4] Of note, 20% of the patients presented intraoperative oliguria.[4] Both studies defined oliguria based on at least one-hour registry and the effects of profound and/or persistent oliguria were not studied in detail.[1,4] Altogether, even though transient intraoperative oliguria defined as a urine output < 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for at least 1 hour was associated with an increased risk of postoperative AKI, the predictive value of intraoperative oliguria for post-operative AKI was low.

Pathophysiology of Perioperative Oliguria

Factors outside the kidney (pre-renal and post-renal)

Several perioperative factors can impact urine output without direct injury to the kidneys. Pain, stress, and hypotension can trigger sympathetic nervous system activation and hormonal responses (*e.g.*, antidiuretic hormone release, renin-angiotensin-aldosterone system activation, suppression of atrial natriuretic peptide release) that lead to antidiuresis and antinatriuresis.[1,2,4,5] The release of cortisol in response to surgical stress or exogenous

administration of steroids (*e.g.*, dexamethasone for postoperative nausea and vomiting) can increase sodium and water retention, as volatile anesthetics.[12,13]

Surgery and anesthesia can also alter renal perfusion via systemic hemodynamics. Anesthesia-induced vasodilation, bleeding, and hypovolemia can decrease venous return, cardiac output, and oxygen transport.[14,15] Positive pressure ventilation can affect venous return, reducing cardiac output and decreasing renal perfusion pressure, renal plasma flow, glomerular filtration rate, and urine output.[16,17]

Renal veins can also be externally compressed by intra-abdominal hypertension such as may occur from ascites, intestinal obstruction, or laparoscopic surgery. Ischemia-reperfusion can lead to an elevation in renal interstitial pressure and consequently to renal microvasculature compression, as the renal capsule is non-distensible – an entity named “renal compartment syndrome”.[18]

Post-renal causes should always be excluded, including urinary catheter obstruction. Other causes of post-renal AKI such as ureteral obstruction are more likely to occur in pelvic or urologic procedures.[14,19]

Intrinsic etiologies of perioperative acute kidney injury

Even though easy to apprehend from an educational standpoint, the classical dichotomy between pre-renal and intrinsic causes of AKI can be difficult to apply in practice. First pre-renal factors (*e.g.*, hypovolemia) and intrinsic causes of AKI (*e.g.*, sepsis) can co-exist. Second, venous congestion (*e.g.*, due to cardiac failure, tamponade, or increased intraabdominal pressure) can decrease kidney perfusion and mimic “pre-renal” azotemia (anti-natriuresis, low fractional excretion of urea) despite requiring opposite treatments. In critically ill patients with oliguria, urine sodium concentration, and fractional excretion are not predictive of fluid responsiveness.[5] The diagnosis of pre-renal azotemia is therefore often made retrospectively after rapid resolution of oliguria after correcting hemodynamic contributors. Of note, biomarkers of AKI (e.g., neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, combination of tissue inhibitor of metalloproteinases 2, and insulin-like growth factor binding protein 7) can detect early ongoing kidney damage.[8,14,20–22] Elevated biomarkers of AKI suggest that “pre-renal AKI” is not the sole contributor of AKI and is associated with worse outcomes, but a detailed review of their performance is beyond the scope of this manuscript.[23,24]

Among intrinsic causes of AKI, several causes have a higher likelihood in the perioperative setting such as acute tubular injury secondary to prolonged ischemia, macrovascular complications (e.g., arterial embolism, arterial thrombosis or dissection, renal infarction), microvascular complications (e.g., thrombotic microangiopathy or cholesterol emboli) (Table 2)[14,19] Cholesterol emboli can lead to post-operative AKI (with a few days delay) are mostly present in vascular and cardiac surgery, aortic cross-clamping, with intra-aortic balloon counterpulsation devices, and in patients with atherosclerosis.[8,21] Sepsis is another important cause of AKI.[14,25] Sepsis leads to intrarenal microcirculatory defects, regional and systemic inflammatory cell infiltration, and apoptosis-promoting AKI.[12,19] Patients with surgical site infection or pneumonia have increased odds of developing AKI (OR: 10.5 [95%

CI: 3.8 to 29.3; p < .001] and OR: 11.8 [95% CI: 7.7 to 18.3; p < .001] respectively).[25] In rare cases, interstitial nephritis can occur in the perioperative setting after certain medication exposures (e.g., NSAID, antibiotics, anticonvulsants, proton pump inhibitors).[19]

Pragmatic Approach to Oliguria Management

Is my patient in need of close monitoring?

As mentioned above, persistent oliguria in the post-operative setting should be considered a red flag and prompt further assessment and monitoring (*e.g.*, closer urine output and serum creatinine monitoring, potential admission to an ICU, and a search for the etiology). Preoperatory scores and models that include patients' characteristics have been developed to estimate the risk for postoperative severe AKI but overall have low accuracy.[26,27] Some populations, such as patients with chronic kidney disease, diabetes, or end-stage liver disease, have higher baseline risk for post-operative AKI and should undergo closer monitoring.[14] Liver disease patients undergoing liver transplant have a 20% to 50% incidence of AKI.[20]

Cardiac, vascular, and intra-abdominal surgeries also present a higher risk for post-operative AKI.[8,22,28] The incidence of AKI in cardiac and vascular surgery are between 20% to 70%, where prolonged cardiopulmonary bypass and aortic cross-clamping are the main contributors.[8,21,28] Cardiac surgery patients are also at risk of cardiac tamponade for which oliguria can be an inaugural sign.[29]

Decision-making in Perioperative Oliguria

Is my patient in need of fluid administration?

Oliguria is poorly predictive for fluid responsiveness (*i.e.*, an increase in cardiac output after fluid administration).[8] A prospective multicenter observational study evaluating the response to fluid challenge among oliguric ICU patients (urine output < 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for 3 consecutive hours) divided fluid responsiveness into cardiac (increase in stroke volume >15 % after fluid challenge) or renal (urine output > 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for 3 consecutive hours after fluid challenge).[7] In the majority of patients (72%) cardiac output did not improve with fluids and only half of the patients increased urine output with an area under the receiver operating characteristic curve (AUC) for predicting renal fluid responsiveness of 0.65 (95% CI: 0.53 to 0.70).[7] The RESPONSE trial included 130 oliguric ICU patients (urine output < 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ for > 2 hours) who were randomized to receive fluid bolus or not.[30] The patients who received fluid bolus had higher cumulative fluid balance at 6 hours compared to the control group but no difference in urine output. Occurrence of creatinine-based AKI was similar in both groups (fluid bolus group: 59.7% *vs* control group: 58.7%).[30] The trial did not provide information on changes in cardiac output.[30]

A "pre-test" probability of fluid responsiveness based on the clinical scenario is important to consider (Figure 1 and Figure 2). Intraoperative oliguria in a patient with active bleeding, extensive open abdominal surgery, diarrhea, or small bowel obstruction is more likely to be fluid responsive. In the RELIEF trial, restrictive fluid administration during abdominal surgery (open and laparoscopic) was found to lead to more episodes of postoperative AKI.[32]

The median fluid balance after 24 hours of surgery was 3.092 L among the liberal group *vs* 1.380 L from the restrictive group (p <0.001). Patients in the restrictive group developed AKI more frequently (8.6% vs 5.0% in the liberal group, hazard ratio: 1.71, 95% CI: 1.29 to 2.27, p <0.001).[32] Similar results were described in a multicenter retrospective cohort evaluating the trends in fluid therapy among patients undergoing elective abdominal surgery.[33] From 2015 to 2019, fewer patients received fluids > 10 $ml \cdot kg^{-1} \cdot h^{-1}$, and the incidence of AKI increased.[33] These results (although not specifically focusing on oliguric patients) show that restrictive fluid administration was associated with more episodes of postoperative AKI.

Predictive tools assessing fluid responsiveness can also be used in the operating room to evaluate the likelihood of an increase in stroke volume and cardiac output with a fluid bolus. These tools are mostly used to predict preload responsiveness and include pulse pressure variation, stroke volume variation, inferior vena cava diameter variation, superior vena cava diameter variation (requires transesophageal Doppler), and end-expiratory occlusion test, among others. The impossibility of using the majority of these methods in patients with spontaneous breathing (*e.g.*, pulse pressure variation, stroke volume) and the requirement for a precise cardiac output monitoring limits their clinical use.[31] Furthermore, the results from these tools have gray zones for which the predicted response to fluid is uncertain. Finally, available monitoring tools can vary widely depending on the unit (*i.e.*, ICU *vs* surgical ward). Assessment of peripheral perfusion by physical exam (*i.e.*, mottling, capillary refill time) can guide the decision for potential fluid administration.

Venous excess ultrasound score (VExUS), a new ultrasound-guided systematic assessment of inferior vena cava, hepatic, portal, and intra-renal veins has been proposed to evaluate venous congestion. The VExUS score quantifies venous congestion by evaluating for 1) increased inferior vena cava size and reduced collapsibility, 2) hepatic vein flow abnormalities, 3) pulsatile portal flow, and 4) intermittent interruptions of intra-renal venous flow. The predictive value of the VExUS score for AKI appears higher than its individual components.[34] Signs of venous congestion should discourage additional fluids and prompt consideration of diuretics.

Overall, a fluid bolus is most likely to correct oliguria if 1) the clinical scenario is compatible with a decrease in intravascular volume (*e.g.*, large “insensible” losses such as diarrheas, low intakes, recent treatment with diuretics with large diuresis, bleeding), and 2) other indicators of fluid responsiveness exist. On the other hand, a fluid bolus is unlikely to improve urine output (and more likely to lead to harmful consequences) when 1) no other indication of fluid responsiveness is present, 2) previous fluid challenges did not result in urine output improvement, and 3) the patient is at high risk of poor tolerance of fluids (*i.e.*, elevated central venous pressure [CVP], congestive heart failure, signs of congestion on VExUS, ARDS with positive fluid balance).[22,34] Repeated fluid boluses or high-volume maintenance fluids in a patient with persistent oliguria (*e.g.*, due to acute tubular injury) are unlikely to correct oliguria and may put the patient at higher risk of fluid overload and its complications, such as pulmonary edema, decompensated heart failure or increased intraabdominal pressure.[22,35] Fluid overload can precipitate the need for renal replacement therapy.[36]

If I give fluid, which one?

Among crystalloids, buffered solutions (*i.e.,* Ringer lactate, Plasmalyte®) are associated with a lower risk of metabolic acidosis when compared with normal saline (NaCl 0.9%).[37] High concentration of chloride (*i.e.,* with NaCl 0.9%) has also been associated with antidiuresis.[37] One hypothesis is that chloride causes vasoconstriction of the afferent glomerular arteriole through activation of tubulo-glomerular feedback, which decreases glomerular hydrostatic pressure. Compared with normal saline, buffered solutions have also been associated with decreased incidence of major adverse kidney events (including death, need for dialysis, and absence of renal recovery).[33,38,39] A Bayesian meta-analysis including critically ill patients concluded that balanced solutions have a 89.5% posterior probability of reduced mortality compared to saline solutions.[38] Recently, in a trial of patients receiving deceased donor kidney transplants, the use of buffered crystalloid solutions was associated with lower rates of delayed graft dysfunction when compared to saline (adjusted risk difference 10,1% [95% CI: 3.5 to 16.6]).[37] In summary, based on the evidence, buffered crystalloid solutions should be preferred in the majority of surgical patients. Saline should be restricted to patients with hypochloremia.

Colloids are often used in the perioperative setting for more complex cases (*e.g.,* liver transplant, prolonged surgery). Hydroxyethyl starches are contraindicated in patients with AKI and critically ill patients as their use can increase AKI and death.[38] No well-powered randomized trials are available to assess the impact of albumin on postoperative outcomes. However, in a retrospective multicenter observational study, intraoperative albumin was associated with an increase in risk of postoperative complications including AKI, pulmonary complications, and death among patients undergoing major noncardiac surgery.[40] In the SAFE trial, 4% albumin was associated with an increased risk of death among trauma brain injury patients, likely resulting from the hypoosmolarity of the solution compared to plasma.[41] Blood products are indicated in case of acute significant bleeding and to restore renal oxygenation.[6,15] The American Society of Anesthesiology (ASA) guidelines recommend red blood cells should be given unit by unit followed by routine reevaluation.[42] In most stable situations, a hemoglobin threshold of 7 g/dl is appropriate.

Is my patient in need of diuretics?

The use of diuretics is considered in patients with signs of fluid overload or venous congestion (such as CVP > 12 mmHg or on ultrasound). The association between elevated CVP and worse renal function has been reported in heart failure and in critical illness.[22,43,44] However, CVP may not predict the risk of worsening renal function after diuretic administration.[44]

A furosemide-stress test can be used in euvolemic and hypervolemic patients to predict the risk of AKI progression. Intravenous furosemide is given at a dose of 1 mg/kg for patients not using diuretics routinely and 1.5 mg/kg for those with regular use of diuretics.[45] Urine output of at least 200 ml after 2 hours is considered a positive response, and the absence of diuresis is a predictor for worsening of AKI and the need for renal replacement therapy.[45]

Among patients with stage 1 or 2 AKI, the furosemide-stress test predicts progression to stage 3 AKI (AUC: 0.87, p = 0.001) with sensitivity of 87.1% and specificity of 84.2%.[46]

Oliguria may represent the inaugural presentation of a complication

An abrupt and persistent decrease in urine output in the postoperative period should raise suspicion for a postoperative complication such as decompensated heart failure, pulmonary embolism, or cardiac tamponade (*i.e.*, in post-cardiac surgery settings)[22,29,47] or be an early sign of sepsis.

Additionally, intraabdominal hypertension should be ruled out, especially in abdominal or aortic surgery cases (*e.g.*, concern for peritonitis or mesenteric ischemia). Abnormal intraabdominal pressure is considered when higher than 12 mmHg, and abdominal compartment syndrome is defined when intraabdominal pressure is sustained higher than 20 mmHg with organ failure.[34] Finally, oliguria after specific surgery (*e.g.*, vascular surgery, urologic procedure) should raise concerns for vascular complications (*e.g.*, cholesterol emboli, renal artery dissection, arterial thrombi) or urinary obstruction. When oliguria does not reverse, an intrarenal cause of AKI should be suspected, and a thorough full work-up should be initiated to identify potentially treatable causes. A nephrology consult is to be considered.

Is my patient in need of kidney replacement therapy?

The main indications for renal replacement therapy include persistent metabolic disorders (*e.g.*, hyperkalemia, acidosis) and fluid overload not responsive to medical treatment. Oliguria alone nor an increase in serum creatinine should be indications for renal replacement therapy *per se*.

The STARRT-AKI trial included patients with KDIGO stage 2 or 3 and no emergency indications for renal replacement therapy comparing two strategies: the accelerated group (renal replacement therapy was initiated in the first 12 hours after randomization) and the standard group (renal replacement therapy was encouraged to be initiated after fulfilling specific criteria: potassium> 6 mmol/L, pH≤7.20, bicarbonate ≤ 12 mmol/L, severe respiratory failure or renal failure ≥ 72 hours).[48] There was no difference in 90-day mortality between the groups (relative risk: 1.00; 95% CI 0.93 to 1.09, p=0.92) and the accelerated group had higher dependence on renal replacement therapy at 90 days (relative risk: 1.74, 95% CI 1.24 to 2.43, and more adverse events (p< 0.001).[48] The results were consistent in the subgroup of surgical patients (32% of the patients, OR: 1.2, 95% IC: 0.91 to 1.59).[48] These results aligned with the AKIKI trial, but no details on the surgical population were reported in that trial.[49,50]

Conclusions

Perioperative oliguria can be a useful alarm signal for potential complications but should not be interpreted as an automatic indication for fluid administration. The decision to give fluids should integrate the clinical context, indicators of fluid responsiveness, risk of developing fluid overload, and previous responses to fluid challenge. Diuretics are indicated in patients

with signs of congestion or fluid overload and can help identify patients who are likely to progress to more severe AKI. Closer monitoring may be the only intervention indicated in some cases.

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Abbreviations

AKI	acute kidney injury
ARDS	acute respiratory distress syndrome
ASA	American Society of Anesthesiology
AUC	area under the receiver operating characteristic curve
CABG	coronary artery bypass graft
CI	confidence interval
COPD	chronic obstructive pulmonary disease
CVP	central venous pressure
ICU	intensive care unit
KDIGO	Kidney Disease Improvement Global Outcomes
LVEF	left ventricle ejection fraction
MAP	mean arterial pressure
NSAID	nonsteroidal anti-inflammatory drug
OR	odds ratio
SBP	systolic blood pressure
SV	stroke volume
VExUS	venous excess ultrasound score

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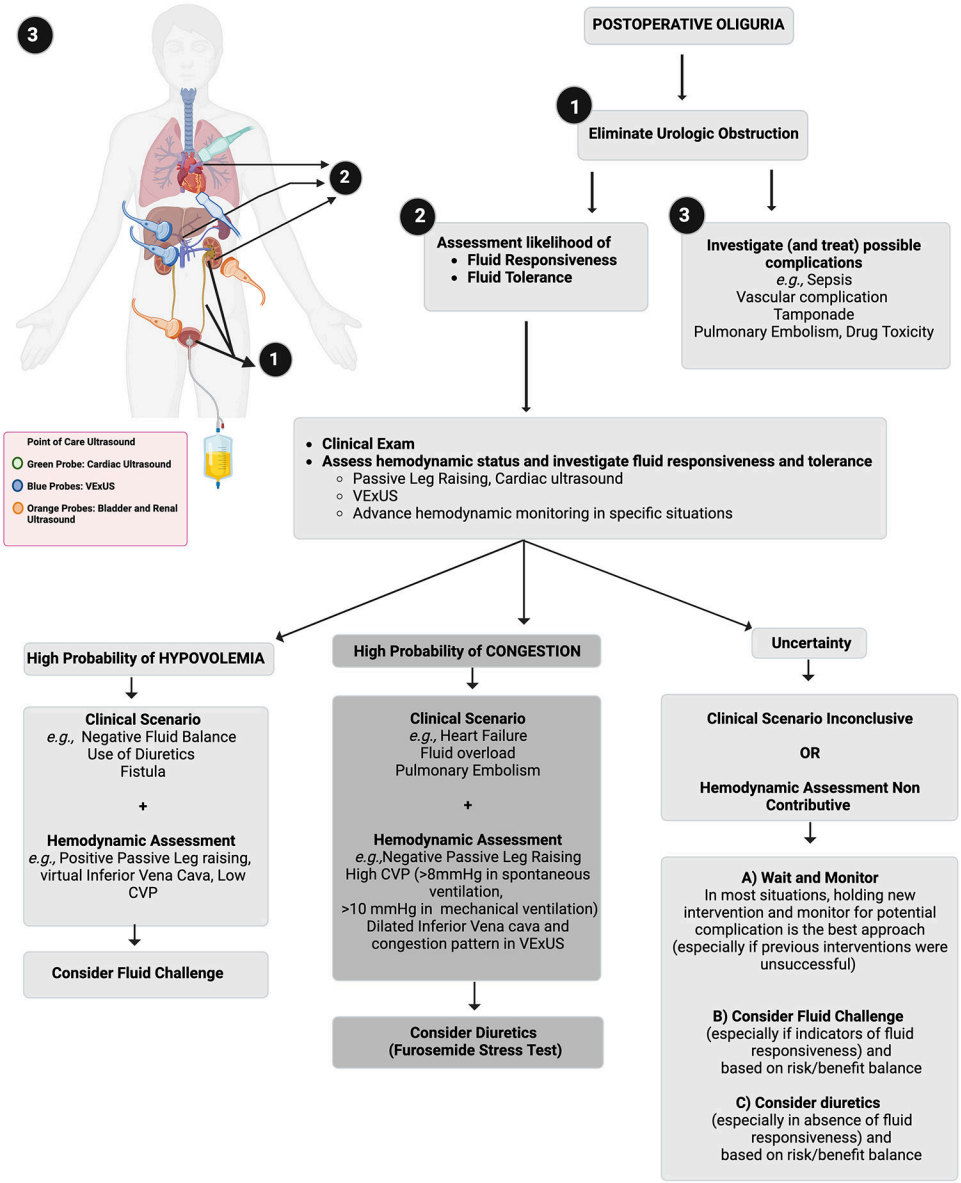

Figure 1.

Proposal algorithm in perioperative oliguria.

(CVP: central venous pressure, VExUS: venous excess ultrasound score).

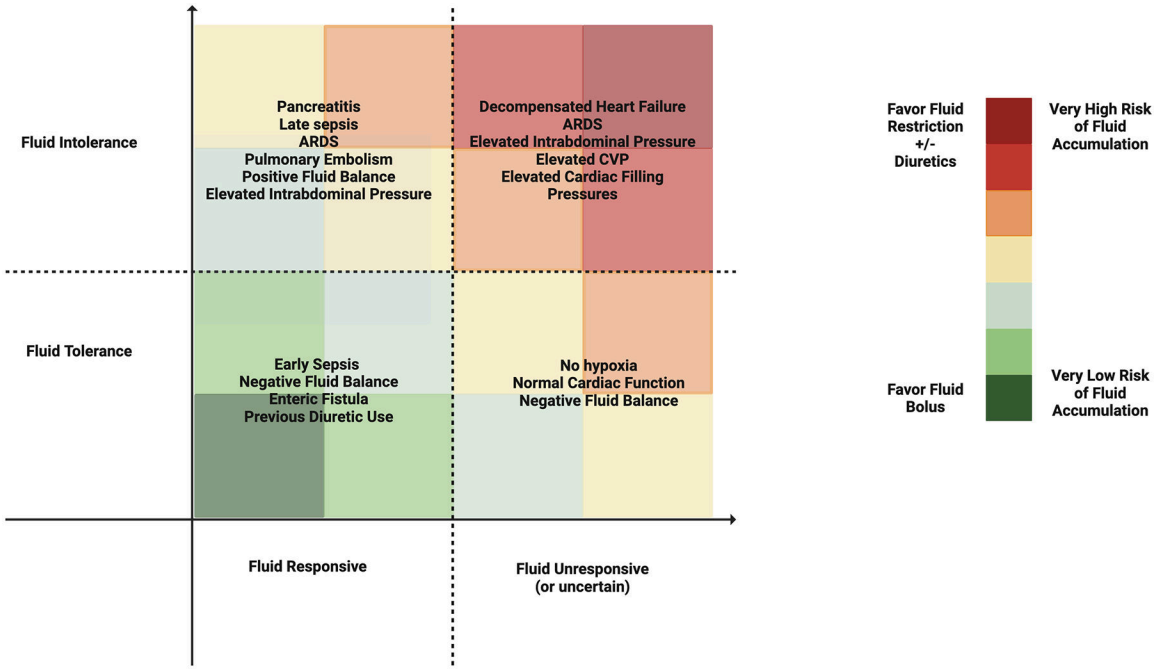

Figure 2.

Risk-benefit balance estimation for fluid management in perioperative oliguria. (adapted from[31]) (ARDS: acute respiratory distress syndrome, CVP: central venous pressure)

Table 1.

Key concepts in Acute Kidney Injury and Oliguria

Key concepts	
Acute Kidney Injury by KDIGO definition	
Stage	Urine Output
1	< 0.5 $mL.kg^{-1}.h^{-1}$ for 6 to12 hours
2	< 0.5 $mL.kg^{-1}.h^{-1}$ for > 12 hours
3	< 0.3 $mL.kg^{-1}.h^{-1}$ for > 24 hours OR Anuria for > 12 hours
Oliguria definition	
Liberal	≤ 0.5 $mL.kg^{-1}.h^{-1}$ for > 6 hours
Conservative	≤ 0.3 $mL.kg^{-1}.h^{-1}$ for > 3 hours

Table 2.

Causes of Post-operative Oliguria

Causes of Post-operative Oliguria	Diagnostic Clues	Examples of Clinical Scenarios
Acute tubular injury	Oliguria unresponsive to fluid challenge Muddy brown casts on urine sediment Recent prolonged hypotension or nephrotoxin exposure (*e.g.*, NSAID)	Surgical stress vascular or cardiac surgery, aortic clamping Hemodynamic instability Cardiopulmonary bypass Sepsis
Sepsis	Hyperdynamic state (high cardiac output) Fever or hypothermia, infection, Persistent Leukocytosis or Leucopenia	Surgical Site Infection Pneumonia Central-line associated blood stream infection
Hypovolemia	Low cardiac output VExUS – hypovolemia pattern Fluid responsiveness	Hemorrhage Recent diuretic use Diarrhea High output ostomies Low fluid intakes
Cardiac failure & Venous congestion	Cardiac dysfunction on echocardiogram Elevated CVP Elevated Brain natriuretic peptide VExUS – venous congestion pattern	Left or right ventricular failure Pulmonary embolism Tamponade
Urinary Obstruction	Imaging Bladder & renal ultrasound. Renal ultrasound if suspicion for ureteral obstruction	Urologic, Gynecologic, Colorectal surgeries Ureter injury Urinary catheter misplacement or obstruction Recent opioid use

In this Table, we listed clinical scenario and diagnostic orientation for main post-operative causes of oliguria. Note that no diagnostic criteria are fully sensitive or specific. (CVP: central venous pressure, VExUS: venous excess ultrasound score, NSAIDs: Nonsteroidal anti-inflammatory drug).