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Editors
Pradip Kumar Bhattacharya
Srinivas Samavedam
Sheila Nainan Myatra
Bharat G Jagiasi
Sachin Gupta

Volume 1: General Principles in Critical Care

Foreword
Dhruva Chaudhry

JAYPEE

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CHAPTER

2.3 Approach to Oliguria

Kanwalpreet Sodhi, Manu Gupta

INTRODUCTION

Oliguria, i.e., "little urine", described as the very first "biomarker" of acute kidney injury (AKI), in 100–200 AD by Epheseus, is common in critically ill patients.[1,2] Urine output abnormalities can vary from anuria to polyuria as shown in **Table 1**.

Varying definitions of oliguria, as described in **Table 2**, with differing cutoff values and time periods ranging from 1 to 24 hours, make it difficult to establish its true prevalence.[3,4] One of the earliest definitions of oliguria was given by Acute Dialysis Quality Initiative as urine output (UO) <0.3 mL/kg/h for at least 24 hours (www.ADQI.net). Recently, oliguria has been defined as UO of < 0.5 mL/kg/h for ≥6 hours, a threshold that has been adopted by various guidelines and AKI classification systems including, RIFLE, AKIN, and KDIGO.[1,3,5] But this threshold has been proposed for patients with fluid intake >3 liters/day, limiting universal application. Moreover, oliguria can be context-specific, for instance, malnourished patients may excrete the daily solute load with lower urine volumes. Body composition should also be a factor under consideration with muscle mass excreting higher nitrogenous waste in comparison to the adipose tissue. Thereby, it is recommended to use lean body mass-based estimations in obese patients to avoid over diagnosis of oliguria.

An observational cohort study, involving 15,500 intensive care units (ICU) patients, reported that thresholds of UO <0.2 mL/kg/h over 3–6 hours and 0.3 mL/kg/h for 12–24 hours is more predictive of 90-day mortality compared to <0.5 mL/kg/h for 6 hours.[6] Another study reported increased mortality in critically ill patients with UO <0.3 mL/kg/h for 3 hours.[7] The current definition of oliguria (UO <0.5 mL/kg/h for more than 6 hours, or <400 mL/day) lacks specificity for kidney damage, but despite the emergence of newer biomarkers, this definition remains a sensitive indicator of AKI in the critically ill patients.[8]

Recent evidence suggests that nearly 30–50% patients experience an episode of oliguria during ICU stay, fulfilling the criteria for oliguric AKI.[4,9] AKI-EPI study found the prevalence of AKI in critically ill patients to be approximately 57%, with oliguria as a key diagnostic criterion.[10] While transient oliguria in ICU patients is often benign, episodes lasting over 48 hours markedly elevate AKI risk, RRT need, and mortality.[11] Oliguria is associated with increased mortality, even without a rise in serum creatinine.[4]

Thereby, prompt identification, evaluation, and management of oliguria becomes imperative. This chapter explores the clinical approach to an oliguric patient in ICU, highlighting the possible etiologies, diagnostic approach, and management strategies, integrating current evidence and clinical guidelines. The approach is based on estimations of urine volume <0.5 mL/kg/h for >6 hours.

TABLE 1: Definitions of urine output abnormalities.

Condition	*Urine output*
Anuria	<100 mL/day or <0.05 mL/kg/h
Oliguria	<400 mL/day or <0.5 mL/kg/h
Nonoliguric AKI	>400 mL/day with rising creatinine
Polyuria	>3,000 mL/day

(AKI: acute kidney injury)

TABLE 2: Definitions of oliguria in adults.

Criterion	*Urine output*	*Duration*
Consensus (KDIGO/AKIN/RIFLE)	<0.5 mL/kg/h	≥6 hours
Daily volume-based	<400 mL	24 hours
Severe oliguria (peritrauma/perioperative)	<0.2 mL/kg/h	3–6 hours

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ETIOLOGY AND PATHOGENESIS OF OLIGURIA

Oliguria is usually a result of either reduced glomerular filtration rate (GFR) or a mechanical obstruction to urine outflow. The causes can be decreased intravascular volume leading to reduced renal perfusion, decreased renal perfusion with normal intravascular volume as in distributive shock (sepsis), renal vascular insufficiency, for example, microvascular vasoconstriction caused by ACE inhibitors, acute tubular necrosis (ATN), or mechanical urinary tract obstruction.[12] Based on the underlying pathophysiology, oliguria can be broadly categorized into prerenal, intrinsic renal, and postrenal **(Table 3)**.

Oliguria can represent either a normal physiological response or reflect an underlying pathological process as shown in **Flowchart 1**.

Physiological oliguria may result from hypovolemia due to excessive fasting or severe exercise leading to activation of renin–angiotensin–aldosterone system (RAAS) and sympathetic stimulation along with vasopressin release causing an antidiuresis response.[1] Usually physiological oliguria is reversible and do not increase the risk for subsequent kidney injury. Transient oliguria may occur in postoperative periods, associated with vasopressin-release and sympathetic activation due to pain or nausea.

In *pathological oliguria*, various pathophysiological pathways contribute to the development of oliguria:

- *Pre-renal*: Hypoperfusion leads to reduced GFR, which is reversible with volume resuscitation.
- *Intrinsic:* Prolonged ischemia or toxins directly damage the renal parenchyma, especially the renal tubules.
- *Post-renal:* Outflow obstruction increases hydrostatic pressure, reducing GFR.

The neurohormonal pathways influence kidney function through activation of the sympathetic nervous system leading to elevated activity of the RAAS, higher circulating levels of vasopressin, and activation of the tubule-glomerular feedback system (TGF), resulting in systemic vasoconstriction, reduced renal filtration as well as water and sodium retention.[13]

In contrast to reversible prerenal oliguria, oliguria/AKI from intrinsic kidney injury due to reduced GFR, tubular obstruction, or solute back-leak is usually irreversible. In sepsis-associated AKI (SA-AKI), systemic vasodilation and macro- and microcirculatory shifts reduce perfusion in renal regions, causing oliguria despite increased RBF. Immune and inflammatory responses further impair renal function.[14]

TABLE 3: Etiologic classification of oliguria.

Category	*Pathophysiologic mechanism*	*Common ICU-related causes*
Prerenal	Decreased intravascular volume	Hypovolemia, trauma, hemorrhage, surgery, burns, vomiting, diarrhea, osmotic diuresis
	Vasodilation	Sepsis, anaphylaxis, anesthesia, vasodilatory drugs
	Inhibition of renal autoregulation	Angiotensin converting enzyme (ACE) inhibitors
	Renal artery (RA) occlusion	RA thrombosis, RA atherosclerosis, dissecting aneurysm
	Heart failure	Myocardial ischemia/ infarction, congestive heart failure, pulmonary embolism, cardiac tamponade, mechanical ventilation
Intrinsic/ renal	Acute tubular necrosis (ATN)	Ischemic, nephrotoxic antibiotics, radiocontrast agents, heavy metals, solvents, crush injury, rhabdomyolysis, oxalate, uric acid
	Interstitial nephritis	Drugs, infection, cancer
	Acute glomerulonephritis	Small vessel vasculitis, scleroderma, systemic lupus erythematosus, malignant hypertension, microscopic polyarteritis, toxemia of pregnancy, rapidly progressive glomerulonephritis
Postrenal	Upper urinary tract obstruction	Unilateral/bilateral ureteral obstruction or ureteral injury
	Lower urinary tract obstruction	Catheter obstruction, neurogenic bladder, bladder outlet obstruction, urethral injury

CLINICAL WORKUP

The clinical evaluation of oliguria aims to pinpoint its cause and any contributing conditions. It involves a focused history including current and past medications, physical examination, laboratory tests, and bedside POCUS to detect obstruction and assess volume status. Hemodynamic monitoring further refines the diagnosis, guiding targeted treatment of the underlying issue.

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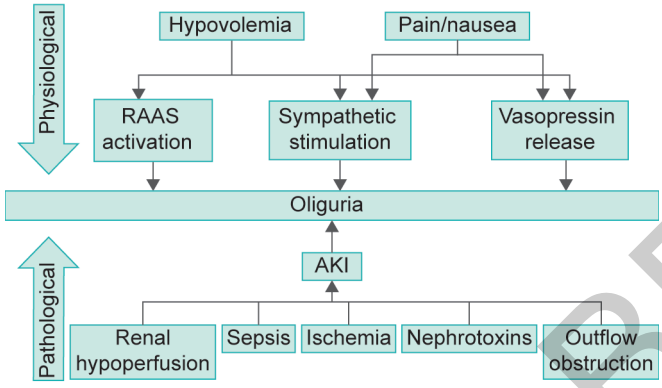
History Taking

In ICU patients with oliguria, history should focus on identifying causes such as recent fluid losses (bleeding, vomiting, diarrhea, and surgical drains) and pre-existing conditions such as diabetes, hypertension, heart failure, or chronic kidney disease. It is important to ask about recent illness, surgery, and medications, especially those that may harm the kidneys. Pay close attention to nephrotoxic drugs including chronic NSAIDs, ACE inhibitors, mannitol, aminoglycosides, vancomycin, polymyxins, amphotericin B, and contrast agents. Also inquire about any recent exposure to toxins or infections.

Flowchart 1: Pathophysiology of oliguria.[1]



Physical Examination

On physical examination, poor volume status (dehydration) might be indicated by loss of skin turgor, dry mucosa, hypotension, tachycardia, or changes in mental status. Whereas, tachypnea, edema, raised JVP, shortness of breath, crackles or wheeze, might indicate fluid overload. Fever with tachycardia, hypotension or altered mental status may indicate SA-AKI. Flank pain/tenderness or palpable bladder might suggest obstruction.

Effective fluid management in AKI is vital, as both hypovolemia and hypervolemia harm renal perfusion. Low RBF reduces GFR, while overload dilutes blood, impairs oxygen delivery, and worsens AKI. Moreover, accurately assessing fluid responsiveness (FR) in oliguric critically ill patients remains challenging.

Assessment of Fluid Responsiveness

Static measurements have limited accuracy and poor predictive value. Dynamic measures, which rely on heart–lung interactions, are preferred for assessing FR; however, these too have limitations.[15,16] **Tables 4 and 5** summarize the static and dynamic measures of FR.

TABLE 4: Static measures of fluid responsiveness.

	Advantages	*Limitations*
Central venous pressure (CVP)	• Most commonly used • Extreme values informative about hypovolemia and fluid overload or fluid tolerance	• Invasive • Catheter-related complications • Not reliable to assess FR in right heart pathologies. e.g., *tricuspid regurgitation, cor-pulmonale, and cardiac tamponade*
Pulmonary artery catheter	• Gold standard • Comprehensive data on fluid status, including PAWP, CO, and mixed venous oxygen saturation (SvO_2)	• Invasive • Catheter-related complications
Left ventricular end-diastolic volume (LVEDV)	• TTE guided • Noninvasive • Additional assessment of cardiac status	• Operator dependent • Poor window in obese and ventilated • Affected by changes in intrathoracic and abdominal pressure
IVC diameter	• Estimates RAP – IVC diameter <21 mm with >50% inspiratory collapsibility → Normal RAP (0–5 mm Hg) – IVC diameter >21 mm with <50% collapsibility → Elevated RAP (10–20 mm Hg)[17] • Static index, but if >2 cm → evaluation of organ congestion by VexUS[18,19]	• Limitations of CVP apply to IVC diameter • Affected by changes in intrathoracic pressure, respiration, and IAH • Operator dependent • Aorta can be mistaken for the IVC • cIVC value >40% can be useful in predicting FR but <40% does not exclude FR due to multiple confounding factors[20] • RAP between 5–10 mm Hg is considered "Gray" zone[17]

(CO: cardiac output; FR: fluid responsiveness; IAH: intra-abdominal hypertension; IVC: inferior vena cava; PCWP: pulmonary capillary wedge pressure; RAP: right atrial pressure; TTE: transthoracic echocardiography; VexUS: venous excess ultrasound)

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TABLE 5: Dynamic measures of FR.

	Advantages	*Disadvantages*	*Diagnostic threshold for FR*
PPV and SVV	• Well validated • Automatically measured • Gives additional information of continuous CO and CI • Does not require any maneuver	• *Not useful with:* – Spontaneous breathing (FP) – Arrhythmias (FP) – Mechanical ventilation with low tidal volume (FN) – Increased abdominal pressure (FP) – Open thorax (FN)	>15%
Tidal volume challenge	• Simple bedside test • Does not require measurement of CO or SV • Can be performed using invasive or noninvasive arterial pressure waveform analysis	• Require mechanically ventilated patients • Not reliable in patients with: – Spontaneous breathing efforts – Cardiac arrhythmias – Open chest – IAH	$\Delta PPV_{(6-8)}$ >3.5% [ΔPPV_{6-8} = difference between the PPV at 6 mL/kg (PPV_6) and at 8 mL/kg (PPV_8)][21]
Pleth variability index (PVI)	• Noninvasive • Bedside • Useful for monitoring trends over time[22]	• Does not provide a direct measure of CO • Less reliable in critically ill patients with:[23] – Poor peripheral perfusion – Arrhythmias	>13–14%
End-expiratory occlusion test (EEOT)	• Simple, quick, and bedside • Validated in low Vt ventilation • Non-invasive	• Requires real-time, precise measurement of CO or SV as the effects of the EEOT diminish rapidly after resuming ventilation • Not feasible in patients who cannot tolerate a 15-second expiratory hold (e.g., with spontaneous interruptions or ventilator dyssynchrony)	• Various cutoff: – *VTI by TTE, EEOT:* Increase ≥10% – *Combined with end-inspiratory occlusion (EEO+EIO):* Increase ≥13% – *PVI:* Increase ≥2.5% – *PPV:* Increase ≥1–3.5% – ≥5% increase in CO during the hold suggests FR[24]
Passive leg raising (PLR)	• Well validated • Works regardless of breathing activity, cardiac rhythm, Vt, and lung compliance • No actual fluid administration required • Fully reversible [25]	• Requires real-time and accurate measurement of CO or SV via pulse contour analysis or echocardiography • False-negative in IAH • Tight venous compression stockings • Contraindicated in patients with intracranial hypertension • Challenging to perform during surgery	• *CO:* Increase ≥10% • *VTI:* Increase ≥10% • *End-tidal CO_2:* Increase ≥5% • *Perfusion index:* Increase ≥9% • *Decrease in PPV or SVV:* ≥1–4 points • *Capillary refill time:* Decrease ≥27%[25]
Mini fluid challenge test	• Simple and quick • Repeatable • No fluid burden • *Applicable regardless of:* Respiratory pattern, cardiac rhythm, tidal volume, lung compliance, and intra-abdominal pressure (IAP)	• Requires real-time and precise measurement of CO or SV by sensitive monitors • Arterial pressure changes alone are insufficient to assess response • Involves fluid infusion, and repeated boluses may lead to volume overload	• *CO:* Increase ≥5% • *VTI via TTE:* Increase ≥10%

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	Advantages	*Disadvantages*	*Diagnostic threshold for FR*
Assessment of IVC variability: IVC collapsibility index (cIVC) IVC distensibility index (dIVC)	• *cIVC:* Dynamic measure of FR in spontaneously breathing patients [Collapsibility IVC = *(IVC max – IVC min)/IVC max) × 100*] • *dIVC:* In mechanically ventilated patients to assess FR. dIVC is the dynamic parameter of FR[26] [*Distensibility IVC = (IVC max – IVC min)/ IVC min) × 100*]	• Complex interplay between heart, volume status, and respiratory mechanics[27] • Aorta can be mistaken for the IVC • <40% does not exclude FR due to multiple confounding factors[20]	• cIVC >40%[28] • *dIVC:* – >18% (using IVC min as denominator) – >12% if using mean IVC diameter as denominator) **(Figs. 1 and 2)**
Left ventricular outflow tract velocity–time integral (LVOT VTI)	• Reproducible and reliable • Standard echocardiographic protocol • Reliable surrogate for CO	• Not accurate in • Aortic regurgitation and subaortic obstruction • Arrhythmias	LVOT VTI size of >10% between inspiration and expiration SVV >14%, **(Figs. 3 to 5)**[29]
SVC collapsibility index (SVC CI)	• Useful in mechanically ventilated patients • Reliable in irregular heart rhythms • Independent of afterload	Operator dependent	SVC CI > 36%

(CI: cardiac index; CO: cardiac output; FN: false negative; FP: false positive; FR: fluid responsiveness; IAH: intra-abdominal hypertension; IVC: inferior vena cava; PPV: pulse pressure variation; SV: stroke volume; SCV: superior vena cava; SVV: stroke volume variation; ; Vt: tidal volume)

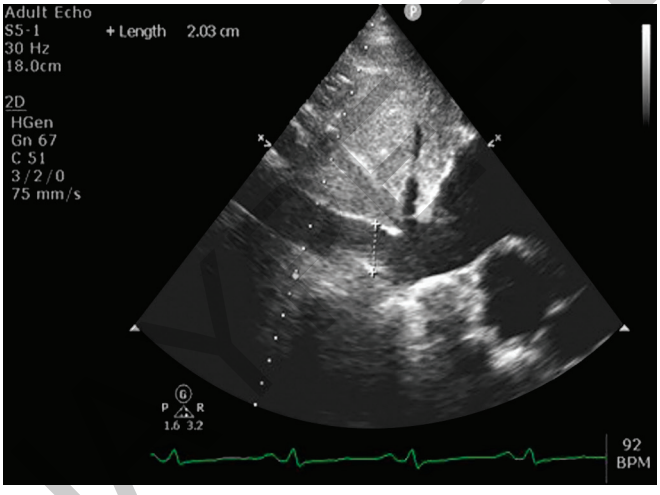

Fig. 1: IVC in subcostal view on TTE. It is measured perpendicular to long axis of IVC, 1 cm distal to junction of IVC–hepatic vein or 2 cm distal to right atrium–IVC junction at the end of expiration.

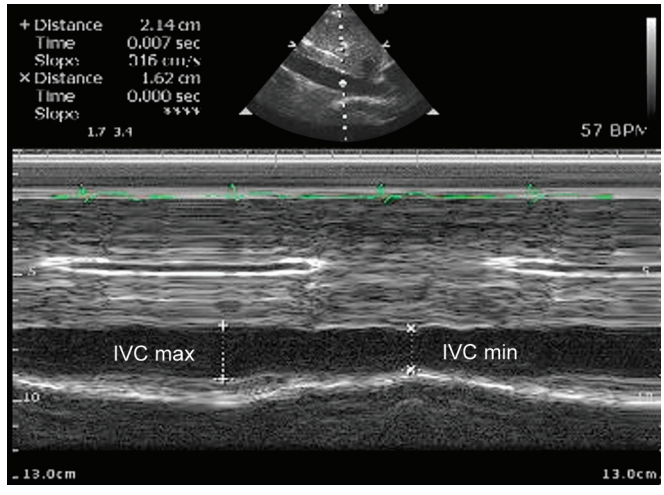

Fig. 2: IVC variability on M mode.

Fluid administration should be tailored to dynamic responsiveness, instead of static measures or routine boluses. Studies report a 50% risk of fluid overload among patients receiving empirical fluid boluses, with positive fluid balance increasing mortality, especially in septic shock.[30] Fluid therapy should be guided by the clinical stage of AKI, degree of hypovolemia, and FR. Guided fluid boluses during resuscitation and maintenance fluids during optimization, following SOSD (salvage, optimization, stabilization, de-escalation) or

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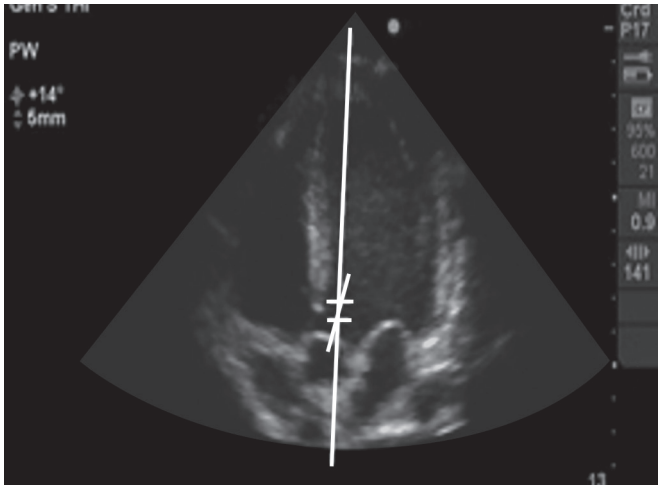

Fig. 3: Apical five-chamber view showing measurement of LVOT VTI by placing PWD curser in LVOT.

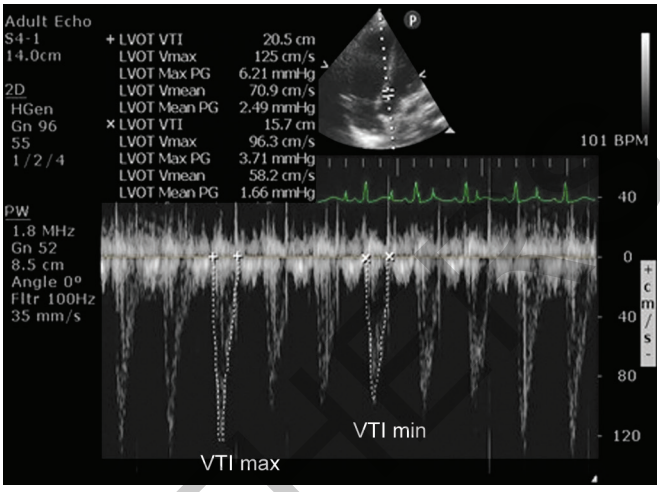

Fig. 5: LVOT VTI variations at apical five-chamber view by reducing sweep speed to see several VTI envelops. VTI max and VTI min is calculated to get VTI variations.

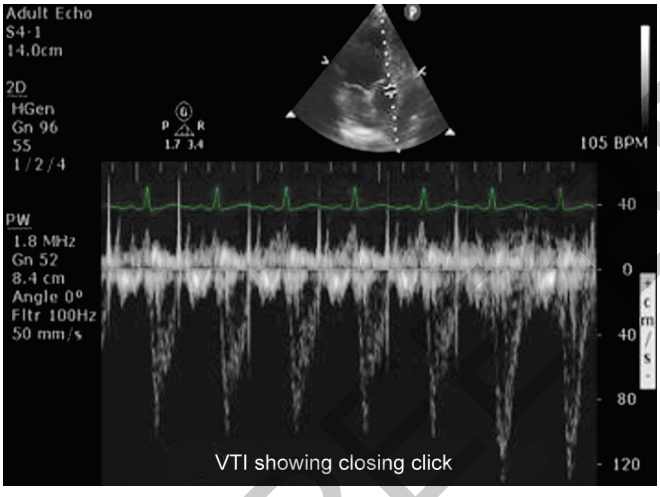

Fig. 4: LVOT VTI. Each VTI envelope is followed by closing click confirming PWD gate is in LVOT.

ROSE (resuscitation, optimization, stabilization, and de-escalation) improve kidney function.[31,32] However, determining the appropriate fluid dose in oliguric patient remains a significant clinical challenge. One must predict FR before actual fluid challenge to avoid volume overload. Studies have shown conflicting results on comparing liberal versus conservative fluid strategy.[26,33-35] Fluids should therefore be individualized, administered in small aliquots at regular intervals, and guided by clinical response and the phase of resuscitation. The approach should be adjusted according to the SOSD or ROSE model.[31]

Fluid Response-based Action Plan

The fluid response-based action plan is as follows:

- *Hypovolemia, hypotension, and fluid responsive:* Administer small aliquots of crystalloids and reassess the clinical and hemodynamic response.
- *Hypovolemia and hypotension resolve:* Discontinue fluid therapy.
- *Hypotension persists:* Reassess fluid responsiveness.
- *If patient is not fluid responsive:* Avoid fluid administration to prevent volume overload.
- *If signs of fluid accumulation are present:* Initiate de-escalation or consider active fluid removal.

Laboratory Investigations

Table 6 shows laboratory parameters that can be utilized to differentiate the causes of oliguria.[36-44]

- *Urinary indices:* Urinary specific gravity (SG) and osmolality are key measures of renal concentrating or diluting capacity. Elevated SG or osmolality indicates dehydration, congestive heart failure, SIADH, or glycosuria; diminished values occur in ATN, diabetes insipidus, or fluid overload.[37] Urine pH (normal = 4.5–8) is an indicator of renal tubular function and acidic urine may result from dehydration, ketoacidosis, or drug induced AKI, while alkaline urine can be caused by old urine samples, recurrent vomiting, or renal tubular acidosis.[38] Quantification of urinary protein is pivotal for identification of intrinsic renal injury and risk stratification. Semiquantitative dipstick

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TABLE 6: Laboratory differentiation of oliguria etiologies.

Parameter	*Normal value*	*Prerenal*	*Intrinsic renal*	*Postrenal*
Urine analysis		Minimal findings with few hyaline or fine granular casts and low protein, blood, or RBCs	• *Significant hematuria and proteinuria; broad, brown, granular/epithelial casts:* Acute tubular necrosis • *Red blood cell casts:* Acute glomerulonephritis • *White blood cells (especially eosinophils) and white cell casts:* Acute interstitial nephritis • *Positive heme with absent erythrocytes:* Hemolysis or rhabdomyolysis	• *Hematuria:* Calculi, trauma, and malignancy • Crystals
Urine sodium (mEq/L)	20	<20	>40	Variable
Urine specific gravity	1.002–1.035	>1.035	<1.005	
Urine osmolality (mOsm/kg)	275–900	>500	<350	Variable
Urine:Plasma osmolality	1:1–3	>1.4:1	1: l	
Urine:Plasma creatinine	100:1	>50:1	<20:1	
FENa (%)	<1	<1	>2	Variable
FEUrea (%)	< 35–40	<35	>50	
BUN:Creatinine	10–20:1	>20:1	~10–15:1	~10–15:1
Creatinine clearance C_{CR} (mL/min)	100–120	15–20	<10	

screening can flag abnormal proteinuria, but definitive assessment via "urine protein–creatinine ratio" or "albumin–creatinine ratio" distinguishes glomerular from tubular protein losses and predicts progression of AKI.[38]

- *Fractional excretion of sodium (FENa)* determined by comparing plasma and urine sodium concentrations, using the formula {%FENa = [(U/P)Na]/[(U/P)Cr] × 100}, aids in distinguishing between prerenal azotemia and ATN. A FENa >2% suggests ATN, while a FENa <1% is indicative of prerenal conditions. FENa may be falsely elevated in ketonuria or glucosuria.[39]
- *Fractional excretion of urea (FEUrea)* is considered more reliable in patients receiving diuretics. A FEUrea exceeding 50% generally supports a diagnosis of ATN, whereas a FEUrea below 35% is consistent with prerenal causes.[40]
- *Blood urea nitrogen (BUN) and serum creatinine:* Rising BUN and creatinine signify azotemia. BUN/creatinine ratio assists in differentiating prerenal from intrinsic renal injury, though it is less reliable in critically ill patients. Timely and repeated measurements guide accurate diagnosis, risk stratification, and management in oliguria-associated AKI, although factors like age, muscle mass, and hydration remain a caveat in creatinine measurement.[41-43]
- *Serum electrolytes:* Serum sodium monitoring is essential as oliguria disrupts fluid and sodium balance, potentially leading to hyponatremia—often due to increased sodium excretion or inappropriate ADH secretion—or hypernatremia, typically caused by insufficient water intake or excessive loss. To pinpoint the underlying cause, evaluate serum sodium together with urine sodium, osmolality, and volume status, which helps differentiate between hypovolemia, SIADH, renal pathology, and other etiologies.[44] Renal dysfunction also disrupts the potassium balance, causing acute hyperkalemia and increased cardiovascular risk.[44]
- *Acid-base disorders:* Acid–base disturbances vary with the underlying cause and duration of oliguria, affecting prognosis and management strategies.[45,46]
- *Complete blood count:* It can help identify anemia (chronic kidney disease and bleeding), leukocytosis (infection and inflammation), and thrombocytopenia (possible DIC in severe sepsis). Schistocytes or

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abnormal platelets suggest intrinsic renal impairment (hemolytic uremic syndrome and thrombotic microangiopathies).[47]

- *Imaging studies:* Imaging plays a crucial role in identifying the etiology and guiding management. Renal ultrasonography is the primary noninvasive, readily available imaging modality in the initial workup of oliguria, highly effective at assessing kidney size, structure, and the presence of hydronephrosis, which may indicate urinary tract obstruction.[1,48] When ultrasound suggests obstruction or reveals a retroperitoneal or pelvic mass, computed tomography (CT) and magnetic resonance imaging (MRI) provide detailed anatomical information. CT urography is particularly valuable for visualizing stones, tumors, or retroperitoneal fibrosis, which can compromise urinary outflow and cause oliguria.[1] Imaging can highlight other extrarenal pathologies associated systemic diseases as contributors to reduced urine output such as ascites, hepatic disorders, mesenteric lymphadenitis, bowel wall thickening, etc. Chest imaging may be needed to evaluate pulmonary edema; echocardiography can illuminate cardiac causes such as left ventricular thrombus leading to reduced renal perfusion and subsequent oliguria.

MANAGEMENT

Management of oliguria should be etiology-specific and timely to prevent progression to severe AKI. Oliguric patient should be managed in a systematic manner based upon the history, clinical findings, and investigations **(Box 1)**.

The key questions that need to be answered while managing oliguria include: What is the fluid status? Does my patient need intravenous (IV) fluids? Does my patient need diuretics? Do I need to modify my drug prescription? **Flowchart 2** shows an algorithmic approach to oliguria based upon the volume status.

The basic approach to oliguria involves:

- *Hemodynamic management:* It begins with the use of IV fluids. Administer serial fluid challenges guided by vital signs and dynamic FR measures. Standard hemodynamic management should be targeted to maximize renal blood flow with maintenance of adequate preload, judicious use of inotropic/vasopressor support to target a mean arterial blood pressure (MAP) of 65 mm Hg, and afterload reduction with vasodilator/inodilator agents.[48,49] Hourly urine output and cumulative fluid balance should be monitored to avoid volume overload.
- *When to stop fluid therapy?* Malbrain et al. found that positive fluid balance is independently associated with increased mortality.[50] The concept "fluid accumulation syndrome" has highlighted the detrimental impact of fluid overload on end-organ function, especially the kidneys. Various tools are available to assess fluid overload, but POCUS is a promising modality to identify organ congestion objectively by venous excess ultrasound score (VExUS) and extended VexUS.[18] Once AKI sets in, ongoing fluid accumulation is a known risk factor for progression to severe AKI and nonrecovery of renal function. Thus, fluid therapy should be de-escalated or stopped provided patient is no longer FR, hemodynamically stable, or signs of fluid overload appear. "Fluid creep", i.e., the cumulative burden from maintenance fluids and drug diluents, may contribute to additional sodium, chloride, and volume.[51] Cardio–abdominal–renal syndrome (CARS) is a complex interplay between elevated intra-abdominal pressure (IAP) and impaired renal perfusion, leading to worsening kidney function. Tailored medical therapy aimed to reduce IAP has improved in renal outcomes and may reduce the need of RRT.[52]
- *Deciding about diuretics:* Fluid removal strategies should be based on renal function, UO, electrolyte

BOX 1: Management of oliguria.

General measures
- Optimize perfusion (MAP > 65 mm Hg)
- Avoid nephrotoxic agents
- Monitor for fluid overload

Management by etiology
- Prerenal AKI
  - Fluid resuscitation (prefer balanced crystalloids)
  - Vasopressors if hypotensive after fluid challenge (target MAP ≥ 65 mm Hg)
  - Treat underlying cause (e.g., sepsis with antibiotics)
- Intrinsic AKI
  - Discontinue nephrotoxins
  - Manage underlying conditions (e.g., glomerulonephritis)
  - Consider early nephrology consult
  - Supportive care and possible renal replacement therapy (RRT)
- Postrenal AKI
  - Bladder catheterization
  - Imaging to locate obstruction
  - Urology referral for surgical causes

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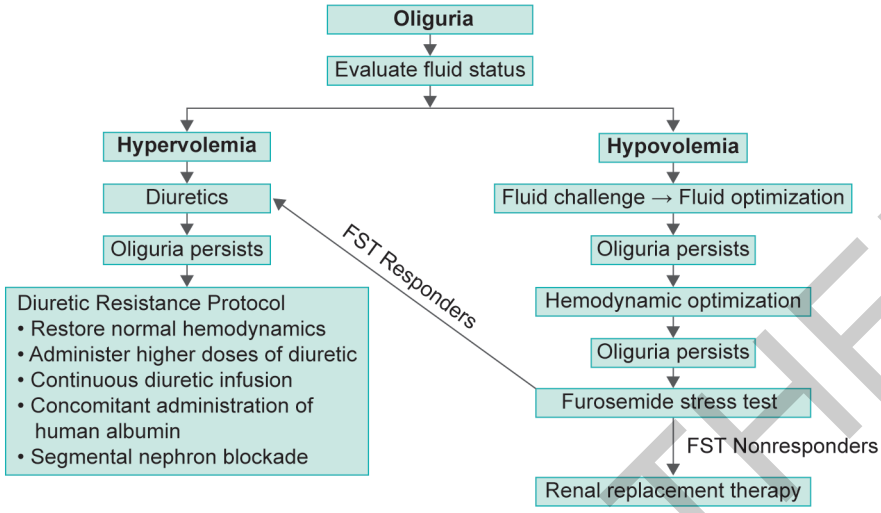
Flowchart 2: Algorithmic approach to oliguria based upon the volume status.



status, diuretic responsiveness, and fluid accumulation. Once the patient is euvolemic, diuretics can be administered based on furosemide stress test (FST). A bolus of 1 mg/Kg furosemide (to naïve patient) or 1.5 mg/Kg (to patients already received furosemide) is given. If UO is > 200 mL over 2 hours, the patient is considered FST positive, and diuretics can be initiated. If not, the patient is considered FST negative, and it is imperative not to give diuretics. Initially described in 1973 by Baek et al., FST has been widely used as a functional tool to assess renal tubular integrity in AKI with variable sensitivity and specificity.[53,54] Diuretics are first-line agents for fluid removal in patients with residual renal function and mild-to-moderate overload but should not be used to prevent or reverse AKI or to treat oliguria without proper volume assessment. The use of continuous infusion versus bolus administration of loop diuretics remains debated. Evidence shows continuous infusion may improve diuresis and fluid balance but offers no significant advantage in key outcomes such as mortality, length of hospital stay, or adverse events compared to bolus dosing.[55-58]

Diuretic nonresponsiveness is defined as a furosemide equivalent dose-to-UO ratio of 1 mg/mL/day, for example, 200 mg of furosemide yielding only 200 mL/day UO, meets this threshold. In such cases, consider RRT.[59]

- *Renal replacement therapy:* In FST nonresponders, RRT can be considered in cases of overt fluid overload, pulmonary edema, dyselectrolytemia, diuretic resistance, or to prevent further kidney injury, although oliguria alone should not be an indication per se. Role of Active Deresuscitation After Resuscitation-2 (RADAR-2) in 2022 reported that conservative fluid strategy reduces cumulative fluid balance compared with usual care.[60] However, the optimal fluid removal rate remains individualized. Fluid removal should be stopped once the therapeutic goal is achieved or signs of hypoperfusion develop.
  - *Fluid management during RRT:* It is crucial to maintain hemodynamic stability while preventing hypotension during RRT. Currently, there is no clear consensus on the optimal safe ultrafiltration (UF) rate during RRT, with studies showing the value of PLR in predicting inability to tolerate subsequent fluid removal during RRT.[61] Murugan et al. reported that higher net ultrafiltration (NUF) rates (>1.75 mL/kg/h) are associated with increased short-term mortality in critically ill patients compared to NUF <1.01 mL/kg/h.[62]
- Certain novel *biomarkers,* such as NGAL and cystatin C, can help differentiate between functional oliguria due to factors other than kidney damage and AKI and might help in risk stratification of oliguria for poor outcomes.[63,64]
- *Correct electrolytes and acid–base balance.*
- All potential *nephrotoxic medications should be discontinued* at the first symptom of oliguria.

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CONCLUSION

Oliguria can be an alarm signal for potential worsening of kidney function but should not be interpreted as an automatic indication for fluid administration. The decision to give fluids or deresuscitate should integrate the clinical context, laboratory parameters, dynamic indicators of fluid responsiveness, risk of developing fluid overload, and response to FST. Closer monitoring may be the only intervention indicated, and the algorithmic approach entailed in the chapter may serve as a bedside aid in clinical management of oliguria.

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