**PERI-OPERATIVE OLIGURIA**

Dr.S.PonnambalaNamasivayam.MD.DA.DNB.

Professor & HOD

Dept. of Anaesthesiology

Govt. Stanley Medical College & Hospital

Chennai

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1. **Introduction:**

Perioperative oliguria (Reduction in the urine output during the perioperative period) is a common clinical presentation. Oliguria in the peri-operative period in a patient without any preexisting renal disease may be due to Acute Kidney Injury (AKI). It is usually diagnosed late and managed sub optimally. Perioperative AKI is one of the important reasons for perioperative mortality and morbidity. Up to 90% mortality rate has been reported in patients who have developed perioperative oliguria. In 50% of patients who require new, acute hemodialysis, it is due to perioperative oliguria. Early diagnosis and prompt intervention is necessary to reduce the mortality and to prevent further renal damage.

1. **Definition:**

Oliguria is strictly defined as a urine output of less than 400ml/day in an adult (urine flow rate of < 15 ml/hr). Usually, in clinical settings, a urine output of less than 0.5 ml/kg/hr (<30-40ml/hr in an adult) is considered as oliguria.

1. **Classification:**

Perioperative oliguria is traditionally classified into three types.

1. Pre renal
2. Renal/ Intra renal/ Parenchymal
3. Post renal

Pre renal oliguria is usually due to decreased renal blood flow.

Renal uremia is due to renal parenchymal damage.

Post renal oliguria is a result of urinary tract obstruction.

Although it is classified into three types, they are contributory to each other and can coexist. Renal hypoperfusion can rapidly result in parenchymal damage; Urinary obstruction can also result in parenchymal damage

1. **Etiology(Causes of oliguria):**
2. *Pre renal Oliguria:*

* Hypovolemia
* Hypotension
* Pre-existing renal disease
* Renal vascular disease
* Sepsis

Hypovolemia may be absolute or relative. *Absolute hypovolemia* may be due to acute hemorrhage, severe diarrhea, vomiting and fluid restriction. Causes of *Relative hypovolemia* include Congestive cardiac failure, sepsis, hepatic failure etc. More than 90% of cases perioperative ARF is due to hypovolemia and inadequate renal perfusion.

1. *Renal oliguria:*

* Hypoxia from pre renal causes/renal vein thrombosis
* Nephrotoxins (aminoglycosides, Amphotericin, NSAIDs,

Chemotherapeutic agents, contrast media)

* Tissue injury {Hemoglobinuria, myoglobinuria, uric acid (tumor lysis)}
* Inflammatory nephritis (glomerulonephritis, interstitial nephritis)
* Polyarteritis
* Myeloma

1. *Post renal oliguria* :

* (urinary tract obstruction at the level of renal pelvis, ureter, urinary bladder, urethra, urinary catheter)
* Bladder neck obstruction
* Blocked drainage system
* Pelvis surgery
* Prostatic enlargement
* Raised intra-abdominal pressure
* Renal/ ureteric calculi/clots
* Necrotic papillae

1. **Risk factors predisposing to perioperative oliguria:**
2. *Age*: Increase in age (>56 years) is associated with more incidence of perioperative oliguria.
3. *Gender*: Men are more prone to develop perioperative oliguria than women.
4. *Preexisting renal disease*: Most important risk factor for development of perioperative renal failure is preexisting renal insufficiency with decreased GFRand decreased renal reserve e.g. Chronic Kidney Disease (CKD).
5. *Systemic diseases (Co morbid conditions)*associated with CRF: Hypertension (esp. Reno vascular hypertension, Pregnancy induced hypertension), Perioperative Hypotension, hypovolemia, Coronary arterial disease, Congestive cardiac failure, Diabetes mellitus, Liver failure, jaundice, peripheral vascular disease, Scleroderma, SLE, Rheumatoid arthritis, Wegener’s granulomatosis, Advanced age, Sepsis, Shock.
6. *Nephrotoxic drug exposure*:

* Acetaminophen (usually with hepatotoxicity)
* ACE inhibitors (impair renal autoregulation)
* Allopurinol
* Aminoglycosides (proximal tubular necrosis)
* Amphotericin B (Glomerulo nephritis & ATN)
* Asparaginase
* Cephalosporins (esp. with aminoglycosides)
* Cimetidine, Ranitidine (interstitial nephritis)
* Cisplatin (ATN)
* Cyclosporine A, tacrolimus
* Intravenous radio contrast (oliguria within 24 hours)
* Methotrexate
* Metaclopramide (inhibits renal D2 receptors)
* Nitrosoureas
* NSAIDs (phenacetin, indomethacin, Tramadol, generic is ketorolac

tromethamine, Cox2 inhibitors)

* Penicillin, Sulphonomides (interstitial nephritis)

1. *Surgical procedures associated with ARF*:

* Biliary surgery
* Burns
* Cardiac surgery
* Genito-urinary/ Obstetric surgery
* Organ transplantation surgery
* Trauma
* Vascular surgery (esp. Supra renal aortic cross clamping)
* Prolonged surgery
* Major surgery
* Emergency surgery
* Surgery with significant third space loss
* Immunosuppressive therapy
* Intra peritoneal surgery

1. Intra operative *hypovolemia* and prolonged *hypotension* can cause ARF in normal patients and exacerbates the renal effects of all the above conditions.
2. **Pathophysiology:**

Perioperative oliguria is most often a physiologic response to hypovolemia and thus a *prerenal* response. The physiologic response to dehydration, hypovolemia and hypotension is water and sodium retention and vasoconstriction through activation of osmoreceptors, volume receptors and baroreceptor reflexes. The sympatho adrenal and renin-angiotensin systems are activated and aldosterone and ADH are released. The net effect on renal tubules is the concentration of urine due to the avid reabsorption of sodium and water.

When *prerenal oliguria* is severe or combined with other nephrotoxic injury, *intrarenal oliguria* and frank acute renal failure may ensue. Renal tubules become necrotic due to ischemic injury and lose their ability to conserve sodium and water. Physiologic, reversible prerenal states may deteriorate into frank Acute Tubular Necrosis (ATN) if the ischemic injury persists. Despite restoration of normal renal hemodynamics, urinary flow will remain low in ATN. *Prerenal states* sensitize the kidney to other nephrotoxic insults. ATN is more easily induced by nephrotoxic drugs in the dehydrated, prerenal patient.

The most common cause of perioperative ARF is ATN caused by ischemia. Although this process is commonly called “necrosis”, tubular epithelial cell loss after ischemia results from both necrosis and apoptosis.

Damaged tubular cells slough and obstruct the narrow portion of the descending part of the loop of Henle, causing the filtrate to leak back into the renal interstitium (back leak). A secondary contribution to the injury is activation of the renin angiotensin system, constricting glomerular vessels and reducing glomerular filtration. The luminal cells of the proximal convoluted tubule and medullary thick ascending limb of Henle are very active and thus most susceptible to ischemia. Nearly 90-95% of the blood flows to the cortex while the medulla receives only 5-10%, resulting in a regional PaO2 of 10 mmHg in the medulla compared to 50 mmHg in the cortex. Oxygen extraction on the other hand is much greater due to active water and salt reabsorption. This explains the ease with which medullary hypoxia can develo. After ATN is triggered by acute ischemia, a maintenance phase of 1-2 weeks usually follows when the GFR decreases markedly. During this time, renal vasomotor tone depends upon the opposing influences of nitric oxide and the very potent and long acting endogenous vasoconstrictor endothelin (ET1). Other than the degree and duration of the initial insult, the factors which determine whether renal function recovers are poorly understood. If recovery of renal function is going to occur, it is usually detectable within three weeks days of the initial injury.

**Staging of AKI:**

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| **STAGE** | **SERUM CREATININE CRITERIA** | **URINE OUTPUT CRITERIA** |
| 1 | Increase in serum creatinine  >0.3 mg/dl (≥26.4μmol/l);  or  Increase >150-200% (1.5–2x)  from baseline | <0.5 ml/kg/hr  for >6 hours |
| 2 | Increase in serum creatinine  >200-300% from baseline | <0.5ml/kg/hr  for >12 hours |
| 3 | Increase in serum creatinine >300% from baseline; or  Serum creatinine ≥ 4.0mg/dL (≥354 μmol/l) with an acute increase ≥0.5mg/dL (44 μmol/l);or  Receiving renal replacement therapy (RRT) | <0.3ml/kg/hr  for >24hrs  Or  anuria for 12 hours |

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1. **Intra operative considerations:**

*Induction of anaesthesia:* All commonly used induction agents (except ketamine) decrease systemic vascular resistance, reduce cardiac contractility & cardiac output and attenuate the normal response to hypovolemia. Hemodynamically unstable patient is at risk of cardiovascular collapse. Adequate IV access should be obtained, the patient’s intravascular volume should be adequately restored and if necessary an arterial line inserted and other invasive monitoring techniques considered. Induction dose should be reduced in patients at risk of AKI. Prior to renal excretion, induction agents undergo redistribution and biotransformation into inactive products. However, in hypovolemia there is a diversion of blood to essential organs and across the blood brain barrier, therefore effects of induction agents may be prolonged.

*Volatile anaesthetic agents* such as isoflurane and sevoflurane contain nephrotoxic fluoride, which poses a theoretical risk for AKI although there is little evidence for avoidance of these agents.

*Opioids:*AKI prolongs the action of opioids as they are renally excreted. The administration of lower doses is recommended in these patients.

*Muscle relaxants: Suxamethonium* should be avoided in AKI patients with raised potassium levels as it increases potassium efflux from muscle cells and its administration can lead to life-threatening hyperkalemia. Non depolarizing muscle relaxants (e.g. *pancuronium*, *vecuronium*) have an altered duration of action, and should be avoided.*Atracurium* and*Cisatracurium* are the muscle relaxants of choice in patients with risk for AKI as they do not depend on kidney for excretion.

*Intra-operative management:* The aim of intra-operative management in those at risk of AKI is to maintain adequate renal perfusion pressure. The following may allow optimal intra-operative care:

* Appropriate intravascular volume replacement
* Avoidance of nephrotoxic drugs
* Urinary catheter aiming for a urine output >0.5ml/kg/hr
* Maintenance of a suitable Mean Arterial Pressure (MAP) for the patient and operation
* Monitoring of central venous pressure (CVP)
* Monitoring of cardiac output
* Vasopressors (there is no evidence supporting the use of “renal dose” dopamine)
* Anticipation of anaesthetic and surgically induced hemodynamic perturbations both intra and post operatively.

Intra-operatively the neurohumoral response to surgery causes a sympathetic response, releasing vasopressin, aldosterone and cortisol in the ‘fight or flight’ response. One of the aims of this is to aid salt and water retention protecting the renal vasculature. Anaesthetic agents, ACE inhibitors and NSAIDs will alter this protective response

*The post-operative period: P*atients may remain at risk of AKI due to relative hypotension caused by ongoing 3rd space fluid loss, pharmacological causes (NSAIDs and ACEi/ARBs) and residual effects of anaesthesia. Epidural anaesthesia can cause hypotension secondary to sympathetic blockade. The risk of AKI will be more if there is inadequate intra-operative fluid replacement. Post-operative fluid therapy is guided by clinical examination, monitoring of urine output and monitoring renal function and electrolytes. 80% of patients with post op AKI respond to fluid therapy alone - *‘Optimise fluid and defend pressure’*. Patients with metabolic disturbances e.g. acidosis, hyperkalemia, uremia or fluid overload not responsive to simple measures may need Renal Replacement Therapy. NSAIDs for post-operative pain relief should be used with caution in patients with risk of AKI.

*Positive pressure ventilation* causes a decrease in renal blood flow. This is because of positive intra thoracic pressure, decreased cardiac output and reflex (hormonal) systemic response to decreased cardiac output. Adequate intra vascular blood volume will attenuate this hemodynamic changes and hormonal and renal response to positive pressure ventilation.

*Renal perfusionpressure:* In high risk patients, it is essential to maintain a mean arterial pressure of 70-80 mm of mercury to preserve adequate renal blood flow. Hemorrhage and fall in MAP from 80 to 62 mm of Hg may cause a decrease in renal blood flow by 20–30%.

*Intra-abdominal pressure:* Increase in in intra-abdominal pressure to > 18 mm of Hg causes reduced renal function. Intra-abdominal pressure may be increased in intra-abdominal bleeding, intestinal distension, peritonitis, paralytic ileus, ascites and during pneumoperitoneum in laparoscopic surgery.

*Aortic cross clamp:* when aortic cross clamp is applied, renal blood flow decreases. This happens especially during vascular surgery e.g. Supra renal aortic cross clamping/Thoracic or thoraco abdominal aortic surgery. Aortic cross clamping is associated with high incidence of acute tubular necrosis. Decreased glomerular blood flow and renal perfusion may occur after infra renal aortic cross clamp also. Prophylactic measures should be taken to preserve the renal function during aortic cross clamp procedures.

*Cardio pulmonary bypass (CPB):*Non pulsatile blood flow during CPB causes a decrease in Renal blood flow by 25% and Glomerular filtration rate by 30%. Hemolysis and perioperative hemodynamic instabilities during prolonged CPB may cause ATN. Measures to minimize the renal insult include maintaining high perfusion rate, optimal perfusion pressure, adequate oxygenation, minimizing, duration of CPB, minimizing hemolysis and following myocardial preservationstrategies.

1. **Diagnosis:**

Differentiating between pre renal/ renal/ post renal type of kidney injury may be essential in planning the management strategy.

*Pre renal oliguria* is usually associated with Dehydration, tachycardia, low pulse volume, hypotension, orthostatic hypotension and low filling pressures (CVP, PCWP).

*Post renal oliguria* usually manifests as anuria. When a patient presents with anuria post operatively, urinary tract obstruction should be suspected; Urinary catheter should be inspected, flushed and/or replaced. If the obstruction is not due to catheter malfunction, Ultra sonogram of abdomen and pelvis has to be done to identify the cause of urinary tract obstruction.

*Renal type of kidney injury* is the most difficult to diagnose and treat. It is usually diagnosed by ruling out the causes of renal and post renal type of AKI.

Diagnostic indices to differentiate between pre renal and post renal state is given in the table. The characteristic findings in a *Prerenal state* are oliguria, high urine osmolality and low urine sodium.

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| Evaluation of oliguria | | | |
|  | Urinary indices | Pre renal states | Intra renal states |
| 1 | U:P osmolality | * 1.4: 1 | 1 : 1 |
| 2 | U.P creatinine | > 50 : 1 | < 20 : 1 |
| 3 | Urine sodium (m eq/l) | < 20 | * 80 |
| 4 | Fe sodium | < 1% | * 3% |
| 5 | Creatinine clearance (Ccr)  (ml/min) | 15 – 20 | < 10 |
| 6 | Osmolality | ------- | < 400mOsm/kg H2O |
| 7 | U:P urea | * 14 | < 10 |
| 8 | Renal failure index | < 1 | * 1 |
| 9 | Free water clearance | < 20 ml/hr | * 20 ml/hr |

1. **Management:**

When a patient develops perioperative oliguria, the cause for oliguria should be identified; the type of oliguria has to be ascertained. Intervention should be initiated immediately.

Intra vascular volume status of the patient has to be noted (palpation of peripheral pulses, Measurement of BP, orthostatic hypotension,heart rate, skin colour, turgor, mucous membrane, measurement of central venous pressure.

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| Algorithmic approach to oliguria |
| Oliguria    OliguriaEvaluate Fluid Status  Hypovolemia Hypervolemia  Fluid challenge Diuretics  Oliguria persists Oliguria persists  Optimize hemodynamics Diuretics resistant  Oliguria persists  Institute AKI care bundle and referral pathway protocol |

**AKI care bundle:**

* Institute in all patients with a 1.5 x rise in creatinine or oliguria (<0.5ml/kg/hr) for 6 hours.
* This is a Medical Emergency.
* Full set of observations, circulatory assessment, treat life-threatening complications, if NEWS triggering give oxygen, begin resuscitation and contact critical care outreach team.
* Diagnose the cause(s) and treat all – STOP AKI

**S**epsis and hypo perfusion, **T**oxicity, **O**bstruction, **P**rimary renal disease

* **S**epsis and hypo perfusion:

Circulatory assessment (history, heart rate, blood pressure, JVP, capillary refill (should be < 3 sec), conscious level. Bolus fluids (e.g. 250-500ml balanced crystalloid) until volume replete with regular review of response.

Senior review.

If no response, 2 litres filling.

Stop anithypertensives, if relative hypotension.

Infection/sepsis screening (history, examination, cultures, CRP) and antibiotics if suspected If severe sepsis ‘sepsis six’ and antibiotics within 1 hour.

* **T**oxicity:

Ascertain full drug history including contrast exposures.

Avoid further nephrotoxic insults if possible

Stop ACEi/ARB

Stop NSAID

If poisoning AKI (e.g. lithium, ethylene glycol) get specialist renal and toxicology help

* **O**bstruction:

Ascertain any urological history.

High index of suspicion if malignancy

Examine or bedside scan for bladder

Consider urinary catheter.

Perform renal tract imaging (ultrasound or CT KUB) within 24 hours unless non-obstructive cause is clear.

If obstructed and infected urinary tract is suspected (pyonephrosis) imaging within 6 hours

If likely/suspected obstructed AKI refer urology.

Target time to relief of obstruction 12 hours after diagnosis and immediate if infected.

* **P**rimary renal disease:

Ascertain relevant history (e.g. autoimmune disease, myeloma, HUS/TTP)

Urine dipstick (all AKI patients).

If protein high measure PCR.

Check CK (rhabdo), CRP, FBC.

If platelets low, do blood film, bill, LDH, relics (HUS/TTP) Consider myeloma screen (Igs, Ig electrophoresis, serum free light chains, urine bench jones.

Consider renal immune screen (ANCA, anti-GBM, ANA, complement, rheumatoid factor, Igs)

If likely/suspected primary renal injury refer nephrology

* General supportive care and escalation:

Once euvolemic, give maintenance fluids (e.g. output plus 500mls), fluid chart, daily weights, regular fluid assessment.

Regular (at least 4 hourly) observations/NEWS with clear escalation plans

Review all drug dosages, consider proton pump inhibitor Consider dietetic review and nutrition Urea, electrolytes, bone and venous bicarbonate at least daily

Consider ABG

Monitor for complications, treat and escalate.

Severe AKI (AKI 3) should be discussed with nephrology and critical care regardless of cause

* Follow up:

Ensure patient/carers have adequate support and information Monitor recovery to completion and ensure adequate follow up arrangements in place

Diuretics:

1. Renal Cortical Vasodilation = Dopaminergic agents, loop diuretics

2. Prevention of Tubular Obstruction = osmotic and loop diuretics

3. Suppression of vasoconstriction = Dopaminergic agents, ANP

4. Decreased Tubular O2 consumption = Dopaminergicagents, loopdiuretics

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| Diuretic agents | | | | | |
| Class | Example | Site of action | Mechanism | FE  Na | K |
| Loop diuretics | Lasix  Bumex  Ethacrynicacid | mTAL | Inhibits Na-K-2Cl | 20-25% | **+** |
| Thiazides | Hctz  metolozone | Early distal tubule | Inhibit NaCl uptake | 5-8% | + |
| K-sparing | Triamterene  Amiloride  Spironolactone | Late distal tubule collecting ducts | Inhibit Na uptake (T,A)  Aldosterone  antagonist (S) | < 5% |  |
| Carbonic anhydrase  inhibitor | Acetylzolamide | Proximal tubule | Bicarbonate loss | < 5% | **-** |
| Osmotic diuretics | Mannitol | Entire tubule | Osmotic pressure  prevention of H2O  absorption | < 5% |  |

*Mannitol:* is an osmotic diuretic. Mannitol increases RBF secondary to release of intrarenal vasodilating prostaglandins and ANP, decreases the production of rennin and reduces endothelial cell swelling. In renal transplantation patients, mannitol reduces the incidence of postoperative renal failure in the presence of adequate volume expansion. However,mannitol can be injurious in large doses causing intrarenal vasoconstriction and subsequent ARF.

*Loop diuretics* (e.g. furosemide): cause renal vasodilation as well as increasing sodium, potassium, and urine outputand creatinine clearance. Furosemide induced dieresis without maintenance of volume expansion may be detrimental. Prophylaxis using loop diuretics may be effectiveagainst pigment nephropathies

*Dopaminergic agents (Dopamine, Dopexamine):*act on dopamine receptors,DA1 and DA2. Low dose dopamine (1-3 mcg/kg/min) is widely used in common clinical practice to prevent or treat renal dysfunction.This concept has not been confirmed; the improvement in urine output in non-shocked patients is only an expression of the diuretic effect of dopamine rather than its protective effect on renal function.The natriuretic effect of dopamine increases solute delivery to the distal tubular cells, which may increase medullary oxygen consumption and exacerbate the ischemia during hypotension. This explains why increases in RBF is not protective. It is also shown that ‘renal doses’of dopamine may increase the incidence of arrhythmias and worsen renal function.

*Fenoldopam mesylate* is a dopamine analogue.It stimulates postsynaptic peripheral DA1receptors. The potential advantages of fenoldopam over dopamine include: increase in dopaminergic potency,lack of tachyarrhythmias and ability to safely infuse through a peripheral vein. It may be beneficial in the prevention of POARF.

*Calcium channel blockers* exert direct vascular effect with preservation of renal autoregulation and enhanced recoveryof RBF, GFR and natriuresis. Calcium channel blockers have been tried successfully in the prevention of radiocontrast induced nephropathy. Critically ill patients may not tolerate high doses of these drugs which may further compromise their hemodynamic status. As of now, calcium channel blockers cannot be recommended for the preventionof renal function.

*Atrial Natriuretic Peptide(ANP)****:***is a potent endogenous renal protective hormone and diuretic. It is produced in the cardiac atria in response to volume overload. ANP acts on the renal glomeruli to increase glomerular hydrostatic pressure by dilating afferent arterioles, constricting efferent arterioles and increasing GFR.The synthetic ANP analogue *anaritide* and a renally produced natriuretic peptide *ularitide* have been tried inpreventing or improving renal failure.

*Endothelin receptor antagonists (ET antagonists):* Endothelins (ET) are potent vasoconstrictor peptides. In the kidney, ET1 causes dose dependant vasoconstriction. Cross-clamping can increase plasma ET concentrations resulting renal vasoconstriction being preventable by nifedipine. Thus either ET receptor antagonists or ET antibodies might offer protection against hypoxic renal injury.

*Prostaglandin E1,*(an endogenous renal vasodilator) and *Acetyl cysteine* (independently decrease serum creatinine without any effect on GFR) are also under trial for preventing and treating oliguria.

1. **Prevention:**

‘Preoperative AKI Risk Assessment’ should be done in patients with high risk for AKI.

Pre-operative optimization should be achieved in ward or ICU in conditions which may precipitate perioperative oliguria e.g.Pre-existing renal insufficiency, Hypertension, Diabetes mellitus, coronary arterial disease, congestive cardiac failure, hepatic failure etc.

*Hypertension* is a major contributing factor for perioperative renal failure. Patients with hypertension have a contracted blood volume. Hypertensive patients are more prone for intra operative hemodynamic instability. Blood pressure control with antihypertensives and optimization of intra vascular volume status is essential in patients with uncontrolled hypertension.

If there is risk of long-term renal insufficiency (e.g. nephrectomy in CKD) discuss with nephrology team.

Review of the patients’ medications is essential to identify whether the patient is on any *nephrotoxic drugs*. Discontinue or avoid nephrotoxic drugs if possible.

Pre-operative *volume status* has to be evaluated and optimized. Central venous cannulation and central venous pressure monitoring should be considered in risky patients. *Dehydration* has to be corrected promptly.

Peri operative *hemodynamic stability* should be maintained*. Hypotension* should be avoided.

Adequate monitoring has to be ensured intra and postoperatively

*Adequate oxygen delivery (Adequate cardiac output, Blood pressure, intra vascular volume status and hemoglobin)* has to be maintained.Blood loss has to be managed.

In patients with risk of renal failure, *contrast media* has to be avoided; or it has to be used with great caution (Adequate prehydration before administration of contrast media, prophylactic oral N-acetyl cysteine, Allowing several days for renal recovery after contrast media before elective surgery); renal tubular flow can be maintained with use of loop diuretics and mannitol. Renal vaso constriction has to be avoided by ensuring adequate volume preload, mannitol, ACE inhibitors and calcium channel blockers. Renal vaso dilatation can be achieved with dopaminergic agents, Prostaglandins and Atria Natriuretic peptides (ANP) (although there is no role for the routine use of dopamine or frusemide in perioperative AKI prevention).Oxygen demand may be decreased by loop diuretics and mild cooling. Ischemic reperfusion injury (which may occur due to release of oxygen free radicals and calcium ions) has to be has to be attenuated.

Post-operative admission to critical care unit should be planned in high risk patients.

1. **Conclusion:**

Peri operative oliguria is a common condition and is associated with high mortality and morbidity. Prompt diagnosis and appropriate management is essential for the better outcome of the patient. Patients who are at high risk of developing Acute Kidney Injury in the perioperative period should be identified and preventive strategies should be undertaken to prevent further damage to the renal function.