Perioperative Management of Acute Renal Failure

Dr. Sekar Michael, **M.D.,D.A.,Dip NB**

Prof & HOD, Anaesthesiology and Pain Services,

Karpagam Faculty of Medical Sciences & Research,Coimbatore

Introduction:

Acute Renal Failure (ARF) in surgical patients is a major challenge for the Anaesthesiologists since it definitely increases the morbidity and mortality. The task is to recognize, diagnose, assess and prevent further deterioration of the renal status during the perioperative period.

Definition:

ARF can be described as a sudden sustained fall in glomerular filtration rate (GFR) associated with accumulation of metabolic waste products and water. It could be a major postoperative complication in surgical patients with a quoted incidence of 10–23%. In addition to the severity of physiological insult, predisposing factors likeexisting comorbidity, hypovolemia and sepsiswill also determine the outcome. Despite improvements in recognition and management, the mortality remains high.

Renal Physiology:

The main function of the kidneys is to maintain the fluid and electrolyte balance. In addition to this it excretes metabolic waste products, control the vascular tone and regulate haemopoiesis and bone metabolism. The renal blood flow is not homogenous and in fact the metabolically active medulla receives only one tenth of the total renal blood flow. Kidneys receive almost 20% of the cardiac output and the blood flow is auto regulated at a mean arterial pressure of 50 – 150 mm of Hg in a normal individual. The blood flow to the glomerulus is regulated through the afferent and efferent sphincters which adjust the glomerular filtration pressure. As a large amount of fluid is filtered in the Bowman's capsule, both sodium chloride (NaCl) and water are reabsorbed to the interstitium through adenosine triphosphate pump (ATP) and passive movement. Urine and plasma osmolality are regulated by the feedback mechanism of the loop of Henle.

Patho-Physiology:

Kidneys tolerate hypo perfusion and ischemia to a certain extent. But repeated severe insults lead to acute kidney injury and the renal function declines. The most common cause during surgery is ischemia-reperfusion injury which leads to necrosis and apoptosis. Acute kidney injury may results from pre renal azotaemia as well as intra renal acute tubular necrosis. Pre renal azotaemia is commonly due to physiological response to hypovolemia whereas post renal azotaemia is often caused by an obstruction. As a result of hypovolemia interstitial concentration of NaCl increases which in turn increases the reabsorption of water and urine output decreases. In acute renal failure renal function deteriorates over hours or days and once ARF is establishedthere is no intervention that has proven beneficial to expedite the recovery of renal function. It is essential to avoid further renal insults and support impaired physiological systems to prevent progression to chronic renal failure.

Recognition & Diagnosis:

A thorough history with clinical examination and appropriate investigations may help us to diagnose ARF and estimate the level of injury. A few pre-existing systemic illness have to be taken in to consideration in cases of undiagnosed acute renal failure. The factors associated with the development of AKI are listed in table 1.

Table 1

Factors predisposes acute kidney injury

Age
Hypertension
Diabetes Mellitus
Chronic obstructive pulmonary disease
Left ventricular failure
Chronic kidney disease
Sepsis
Peripheral vascular disease
Cerebrovascular disease
Ascites

The lack of sensitive markers of renal injury hampers the diagnosis very often. Though the novel biomarkers are emerging out in recent times they are still under trials and left us with the golden markers which are listed in table 2.

Table 2

Markers	Facts
Serum creatinine	It is insensitive and slow to increase. It is not directly proportional to the GFR initially. It may be showing false negativeresults in the immediate post op period due to dilution. But an increased levels definitely indicates renal failure.
Urine output	It may not be a reliable marker. Adequate urine output usually is usually associated with adequate renal function. Anuria may be a sign of severe renal injury in non-obstructive disease.
Fractional excretion of sodium (FeNa)	Helps us to differentiate pre renal azotaemia from acute tubular necrosis. A FeNa less than 1% is consistent with pre-renal azotaemia

Serum CystatinC , a protein which is produced by all nucleated cells is filtered by kidneys completely and not reabsorbed. Since it is independent of age, sex, muscle mass it reflects GFR better than serum creatinine but further studies may be required to validate its accuracy.

Assessment of Renal Function:

Once the diagnosis is established it is very essential to assess the level of injury and the status of renal function. The RIFLE criteria were established by the Acute Dialysis QualityInitiative (ADQI) Group to assess the level of renal injury. The other criteria were established by Acute Kidney Injury Network, a consensus panelinvolving national and international societies in nephrology and critical care.

Table 3

Acute Kidney Injury Network Criteria			**RIFLE Criteria**		
	Creatinine/ GFR	**Urine Output**		**Creatinine/ GFR**	**Urine Output**
Stage 1	Increased Cr 0.3mg/dL or Cr 150% Baseline	UOP<0.5 mL/kg/h for>6h	Risk (R)	Increased Cr 1.5_or GFR decreased <25%	UOP <0.5mL/ kg/h for >6h
Stage 2	Cr 200%-300%	UOP<0.5 mL/kg/h for >12h	Injury (I)	Increased Cr 2_or GFR decreased	UOP <0.5mL/ kg/h

	Baseline			<50%	for >12h
Stage 3	Cr>300% of baseline or >4mg/dL with 0.5mg/ dL acute increase	UOP<0.3 mL/kg/h for 24 h or anuria for 12h	Failure (F)	Increased Cr 3_or GFR decreased <75% or Cr >4mg/ dL with 0.5mg/dL acute increase	UOP <0.3mL/kg/ h for 24h or anuria for 12 h
			Loss(L)	Persistent ARF >4wk	
			ESKD (E)	Persistent loss >3months	

Pre Anaesthetic Preparation:

Pre anaesthetic work up has to be done if time permits. The volume status, acid-base derangements and electrolyte abnormality has to be checked and treated appropriately. In volume repleteoliguric patients consider reducing fluid intake to keep up with output, but remember an allowance for insensible losses. In patients with fluid overload, loop diuretics may have a limited role. It may be helpful in in responsive patients in preventing fluid overload. We should remember that acute renal failure may not be an isolated renal problem and physiologically unstable patients may deteriorate further.Vasopressors and inotropes may be used to maintain an adequate perfusion pressure.

Efforts have to be taken to diagnose and treat appropriately to avoid unnecessary complications. A few conditions and marks are given in table 4.

Table 4

Conditions	Remarks
Septic shock	Hypotension and maldistribution of blood flow jeopardise the renal blood flow. Medullary hypoxia may result in acute tubular necrosis and enhances the renal failure
Cardiogenic shock	Results in hypo perfusion and aggravate the renal insult and injury. A proper inotrope support may reduce the damage.
Nephrotoxic renal injury	Requires judicial fluid administration to maintain renal perfusion and prevent further insult.
Uraemic pericarditis	Is a consequence of coagulopathy, need preoperative

	echocardiography to know the cardiac status.

Symptomatic patients may need an urgent dialysis to stabilise before subjecting for anaesthesia and surgery. The indications are definite signs of fluid overload, hyperkalaemia, severe maetabolic acidosis and symptomatic uraemia.

Anaesthetic Management:

The goals in management of AKI include preservation of existing renal function as well as prevention of acute complications like hyperkalemia, acidosis and volume overload. There is no anaesthetic technique which is better than the other and each one has got its own merits and demerits. Care should be taken to maintain normovolemia and normotension to avoid decreases in renal perfusion. In general, all volatile anaesthetic agents decrease GFR either by reducing systemic vascular resistance or by depressing myocardium.

Table 5

Volatile agents	**Side effects**
Isoflurane	Decreases systemic vascular resistance (SVR)
Halothane	Decreases the cardiac output
Sevoflurane	Fluoride toxicity, decreases SVR
Desflurane	Inorganic fluoride toxicity can’t be ruled out

The induction agents can be titrated to avoid sudden decrease in blood pressure. The ideal intravenous fluid will be Ringer lactate and half normal saline. Noxious stimulation due to surgical incision may induce catecholamine which in turn affects the GFR. A short acting analgesic like fentanyl has lesser impact on respiratory or cardio-vascular system and considered to be good analgesics in these cases. Cis-atracurium and atracurium are the best choices for relaxation as they do not rely on renal function. Positive pressure ventilation can decrease cardiac output, renal blood flow and GFR. Moreover it can alter the carbondi-oxide levels which may alter the deranged acid-base status. The use of regional anaesthesia may be beneficial due to its attenuation of catecholamine release. Epidural anaesthesia technique seldom alters the blood pressure and GFR as long as normovolaemia is maintained. Avoiding intraoperative renal insults and maintaining normovolaemia, adequate cardiac output and renal perfusion pressure are the best interventions to

prevent postoperative AKI and are more important than the choice of a specific anaesthesia technique.

Post Op Follow up:

A good intra operative management poses fewer problems in the post-operative period. Patients who receive general anaesthesia can be continued to be on ventilator if requires. An unsettled acid-base status, marked electrolyte imbalance or additional risk factors like cardiac failure may be vulnerable in the post-operative period. ARF due to obstructive pathology may recover much faster once the obstruction is relieved but those who have medical renal problem need time to recover. The immediate post-operative period has to be monitored vigorously and a time to time survey of the renal function and vitals will be helpful.

Conclusion:

Surgical patients with known or unknown acute renal failure are having high mortality and morbidity inspite of careful management. But efforts have to be taken to improve the outcome and reduce mortality. An understanding of the patho-physiology and medical management of acute renal failure will be handy for the anaesthesiologist to take care of those who suffer from acute renal failure.

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