The role of the nurse in the management of acute kidney injury

Fiona Murphy, Gobnait Byrne

Abstract

Acute kidney injury can occur with patients within the hospital and within the community setting. The term acute kidney injury replaces the previous recognized term acute renal failure in the literature and uses two internationally recognized classification systems. The causes of acute kidney injury remain the same, pre renal failure, intra renal (intrinsic), or post renal failure. The nursing care of acute kidney injury is challenging and multifaceted as the patient can be critically ill and requires constant monitoring. It is therefore vital that nurses understand what acute kidney injury is, and the management of it in order to deliver holistic care to the patient concerned.

Key words: Acute kidney injury ■ Acute renal failure ■ Critical Care ■ Diagnosis

Interestingly, there has never been a time that humankind has not endured acute kidney injury (AKI), largely as a result of our past ancestors' pursuit for survival as both hunters and gatherers, and the trauma they sustained in doing so (Eknoyan, 2008).

Today, AKI can occur in patients both within the hospital and the community setting, including accident and emergency, intensive care, coronary care, along with surgical, medical, and care of the elderly units. In fact, one could argue that there is probably not a clinical area that exists where AKI could not potentially develop in patients. It is therefore vital that *all* nurses possess an understanding of what AKI is, and how to manage it, in order to deliver holistic care to the patient concerned.

The nursing care of AKI is challenging and multifaceted. The patient may be critically ill and will require constant monitoring as the clinical situation can alter rapidly (Walsh and Crumbie, 2007). This article is aimed at both students and staff nurses who may be unfamiliar with AKI and its impact on nursing care delivery. Nurses should be familiar with the anatomy and physiology of the renal system in order to comprehend AKI.

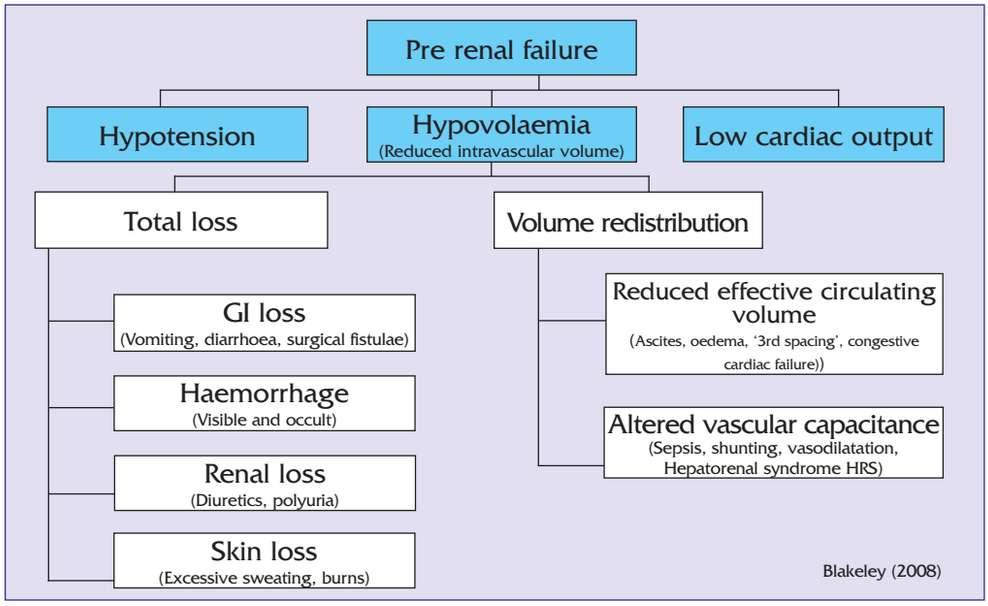
It is important to recognize the disparities between AKI and chronic kidney disease (CKD): the latter usually involves a slower, insidious disease which has a strong interrelationship with both cardiovascular disease and diabetes (Murphy et al, 2008a; b; Murphy and Byrme 2009a; b). AKI refers to a loss in kidney function, which can occur over a number of hours to days. This subsequently causes a build up of nitrogenous waste products, along with a disturbance of volume, electrolyte and acid-base homeostasis (Vijayan, 2008a).

The term 'acute kidney injury' replaces the term 'acute renal failure' in the literature (Davenport et al, 2008). The term 'kidney' is more familiar to the general public than the term 'renal'. The CKD classification was updated in 2002 and the term 'kidney' is also used in this classification system (NKF-K/DOQI, 2002).

AKI addresses the important fact that an increase in serum creatinine does not necessarily mean failure of the kidneys, but a dysfunction that may or may not lead to failure (Vijayan, 2008a). The progression of AKI increases the mortality connected with any primary disease. It develops in 5% of all hospitalized patients, with around 20–60% of patients that develop AKI requiring dialysis treatment. Around 50 to 60% of patients that develop AKI will revert to most, if not all, of their kidney function. The highest cause of mortality in patients with AKI is infection which accounts for 75% of deaths followed by cardiorespiratory complications (Biel et al, 2008). Vijayan (2008a) identifies AKI as a disease of the hospitalized patient, with prevalence altering from 5% in the overall hospital patients to 25% in patients in intensive care.

The Acute Dialysis Quality Initiative (ADQI), a panel of international specialists in both nephrology and intensive care, developed the RIFLE classification system for AKI in 2004. The RIFLE system addresses three grades of severity i.e. Risk, Injury and Failure along with two outcomes – Loss and End Stage Kidney Disease (see *Table 1*).

Figure 1. Pre Renal causes of AKI.



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This classification system was developed due to a lack of a universally recognized definition for Acute Renal Failure (ARF) Faubel et al, 2009). The stages of renal dysfunction can be distinguished by changes in serum creatinine, glomerular filtration rate (GFR) or urine output (Blakeley, 2008). The RIFLE criteria highlights the perception that renal dysfunction is not only regarded as being important when it reaches the point of failure, but also is a spectrum that varies from initial risk to long term failure. This classification system facilitates the early diagnosis of AKI. This system should permit recognition of patients that are susceptible to acquiring AKI as well as those patients with established AKI (Faubel et al, 2009).

The Acute Kidney Injury Network (AKIN), another group of global specialists in both nephrology and intensive care medicine, modified the RIFLE criteria, renaming the stages as stage one, two and three. The AKIN criteria categorizes any patient requiring renal replacement therapy as stage 3 AKI (Mehta et al, 2007) (see *Table 2*). The UK Renal Association published guidelines on AKI in 2008 and has adopted the AKIN criteria (Davenport et al, 2008).

Table 1. The RIFLE Classification for AKI

Class	GFR Criteria	Urinary Output Criteria
R Risk	Increased Serum Creatinine x 1.5 baseline Or GFR decreases ≥ 25%	< 0.5 mL/kg/hr for 6 hours
I Injury	Increased Serum Creatinine x 2 baseline Or GFR decreases ≥ 50%	< 0.5 mL/kg/hr for 12 hours
F Failure	Increased Serum Creatinine x 3 baseline Or GFR decreases ≥ 75% Or Serum Creatinine ≥ 354 µmol/L with an Acute rise of at least 44 µmol/L	< 0.3 mL/kg/hr for 24 hours or Anuria for 12 hours
L Loss	Persistent Acute Renal Failure= Complete loss of kidney function > 4 weeks	
E ESKD	End-Stage Kidney Disease >3 months	

(Bellomo et al, 2004; Bagshaw et al, 2008)

Table 2. The Acute Kidney Injury Network Diagnostic Criteria for AKI

AKIN stage	Serum Creatinine criteria (SCr)	Urine output criteria
1	Increase in SCr ≥ 26.4 µmol/L or Increase in SCr ≥ 150-200% (1.5-2 fold) from baseline	< 0.5 mL/kg/hr for > 6 hr
2	Increase in SCr > 200 - 300 % (>2-3 fold)from baseline	< 0.5 mL/kg/hr for >12 hr
3	Increase in SCr > 300 % (>3 fold) from baseline or SCr ≥ 354 µmol/L with an acute rise of ≥ 44 µmol/L in ≤ 24 hr or Initiated on Renal Replacement Therapy (irrespective of stage at time of initiation)	< 0.3 mL/kg/hr for 24 hr or Anuria for 12 hr

(Mehta et al, 2007; Davenport et al, 2008).

Causes of acute kidney injury

The terminology may have changed for AKI, but the causes have not and can still be categorized into pre renal, intra renal (intrinsic) or post renal (Davenport et al, 2008; Vijayan, 2008a). The kidney is a very vascular organ, acquiring about 25% of cardiac output. It autoregulates to maintain a continuous renal blood flow (RBF) through a mean arterial pressure range of 65 to 180mmHg (Blakeley, 2008; Byrne and Murphy, 2008). Pre renal failure is an embellished physiological reaction to renal hypoperfusion. RBF is maintained by the stimulus of renin-angiotensin-aldosterone system, which helps regulate the blood volume. When the renal perfusion is re-established, the renal function should recover. There are various conditions that can lead to hypoperfusion, all of which result in pre renal failure (see *Figure 1*).

A continued decline in perfusion, however, can result in intrinsic or intra renal failure. (Blakeley, 2008). This occurs as a consequence of factors that result in injury to the kidney itself or nearby vasculature. These factors are glomerular, vascular, interstitial and tubular (see *Figure 2*) (Ashley, 2008; Blakeley, 2008). The vascular disorders are classified according to the size of the vessels affected. These vessel sizes also determine the signs and symptoms along with providing a method of categorizing these diseases. Glomerular disorders can occur as a result of diverse, typically immune-mediated insults within the glomeruli of the kidney (Ashley, 2008). These disorders are classified by urine findings such as large proteinuria and minimal haematuria, which is prevalent in nephrotic glomerular disorders and haematuria and proteinuria prevalent in glomerulonephritis. Interestingly, patients with recognized glomerulonephritis may develop AKI; indeed this disorder can frequently present as AKI (Faubel et al, 2009). There is a glossary of terms to assist the reader, especially regarding some of the causes of intrinsic or intra renal failure (*Table 3*).

Interstitial intra renal or intrinsic renal failure involves the interstitial part of the kidney and any disturbance to this vastly interdependent structural design can cause renal failure. Another common name for this type of AKI is acute interstitial nephritis (AIN). AIN results from marked oedema within the interstitial space caused by the penetration of the renal interstitium with lymphocytes and/ or eosinophils inflammatory cells. Drug hypersensitivity most commonly occurs as a result of AIN, with in excess of 100 drugs implicated in drug induced AIN (see *Table 4*). AIN may also occur as a result of bacterial infections such as staphylococcus, streptococcus and tuberculosis or viral infections such as cytomegalovirus and The Epstein Barr virus (Faubel et al, 2009).

Tubular damage to the kidney is known as Acute Tubular Necrosis (ATN) and is caused by ischemic or nephrotoxic changes. McIntyre (2008) identifies ATN as a clinical syndrome. Ischemic ATN develops after prolonged pre renal failure, and is a result of decreased blood flow to the kidneys, which can occur after problems like septic or haemorrhagic shock.

The causes of nephrotoxic ATN are multifactorial, with some of the common causes including nephrotoxic drugs like aminoglycoside antibiotics, radiocontrast dye, ethylene glycol (antifreeze), and anaesthetics. Other causes of

Figure 2. Intra Renal (intrinsic) causes of AKI.

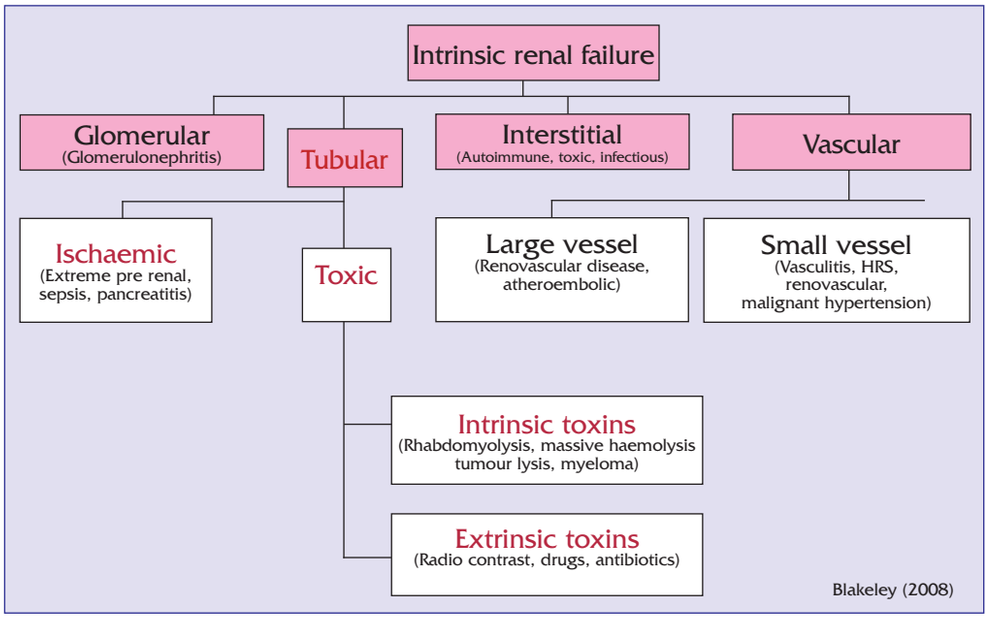


Table 3. Glossary of Terms

Systemic Lupus Erythematosus (SLE)	This is a chronic systemic inflammatory disease of the connective tissue which affects many organs and systems including the kidneys, musculoskeletal, cardiovascular and the skin (Schira, 2008)
Nephrotic Glomerular Disorders	These are characterized by large proteinuria (> 3g in 24 hrs) and minimal haematuria. These disorders are unusually connected with AKI. They may occur in Minimal Change Disease as a result of volume depletion (Faubel et al, 2009)
Nephritic Glomerular Disorders (Glomerulonephritis)	These disorders are characterized by haematuria and proteinuria (usually 1 to 2g in 24 hrs) and involves an inflammatory process that mainly affects the glomerular capillaries of the kidneys (Faubel et al, 2009, Schira, 2008)
Goodpasture's Syndrome	This is an autoimmune disease caused by Rapidly Progressive Glomerulonephritis. Some individuals present with renal involvement (Goodpasture's disease) whilst others present with both nephritis and pulmonary haemorrhage which is known as Goodpasture's syndrome (Johnson and Tisher, 2009)
Rhabdomyolysis	This is necrosis of skeletal muscle and can arise as a result of direct traumatic or non traumatic injury. Some causes include muscle overexertion, muscle compression, and drug abuse. When muscle cells are injured, myoglobin is released into the circulation which can be nephrotoxic to the kidneys (Biel et al, 2008).
Multiple Myeloma	This is a malignancy of plasma cells resulting in an over development of monoclonal immunoglobulin; the so named M protein. Myeloma kidney pertains to intrinsic or intra renal AKI that occurs as the filtered light chain part of the M-protein (the Bence Jones protein) wields both obstructive and toxic damage to the renal tubules (Sambandam, 2008)
Wegener Granulomatosis	This is a primary systemic vasculitis which is uncommon. It mainly affects the small and medium-sized arteries of the kidneys and respiratory tract (Shaw Bichier, 2009)
Acute Poststreptococcal Glomerulonephritis	This is classified as a primary glomerular disorder. The inception of which is 1-4 weeks following beta-haemolytic streptococcal throat or skin infection (Schira, 2008).
Haemolytic Uremic Syndrome	This is a systemic disease. The patient can present with AKI, thrombocytopenia, haemolysis as a result of verotoxin from Escherichia coli 015: H7 gastrointestinal infection (Thurman and Wiseman, 2009).

nephrotoxic ATN include endogenous toxins such as myoglobin (rhabdomyolysis) and haemoglobin for example, as a result of an incompatible blood transfusion (Blakeley, 2008; Faubel, 2009).

The final classification, or cause of AKI is post renal failure, which can occur anywhere between the renal tubules and the urethral outlet resulting in urine flow obstruction. There are both intrinsic and extrinsic factors that can cause this obstruction (see *Figure 3*) (Blakeley, 2008; Pratt and Nouri, 2009).

Assessment

Most of the main organs are affected by AKI, and nurses must be able to assess how the disease affects the patient holistically (Walsh and Crumbie, 2007). The assessment process is therefore vital, and involves obtaining an accurate and comprehensive history of the patient, including whether or not he/she has AKI or CKD (see *Table 5*). Their urinary pattern must be established, as it is essential to identify how much urine has been passed, along with investigating any recent changes in the intake/output, and any difference in the patient's weight.

The inception of the urinary symptoms may also indicate a temporal sign to the length of the illness. It must be established whether there is any history of haematuria, dysuria or pyruia, or if the patient has any urgency, dribbling, and incontinence which is particularly prevalent in the older person, but could also be an indicator of prostatic disease (Vijayan, 2008a). The volume status must be assessed. For example, with a patient who has had surgery, it is vital to check the intra and postoperative haemodynamic records in both the nursing and medical notes.

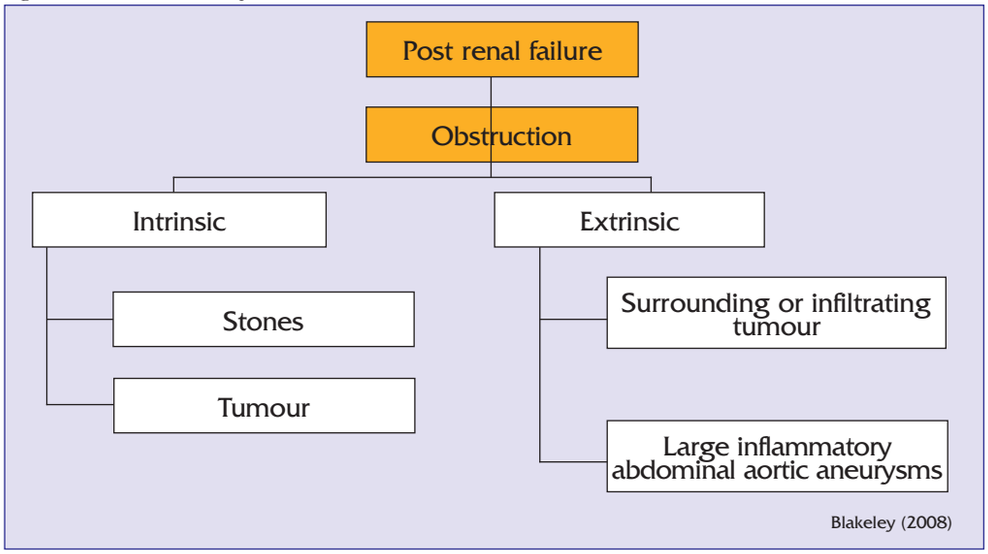
It is also important to check for any episodes of alterations in blood pressure. Is there a history of dizziness or orthostatic volatility? This may indicate intravascular reduction, while the presence of oedema, weight gain and periorbital swelling may point towards fluid retention. All causes of fluid loss must be identified, such as haemorrhage, polyuria, vomiting and diarrhoea, which can also result in excessive insensitive losses as they all influence volume depletion (Vijayan, 2008a). The issue of medications must be addressed. Has the patient been taking any herbal products, over-the-counter medications, and/or health and food supplements? It is important to recognize that some herbal products such as mutong and fangchi contain aristolochic acid which can result in AIN (Biel et al, 2008). Other medication issues must also be considered, like whether or not the patient is taking/has been taking administered drugs that are potentially nephrotoxic, such as NSAIDs, ACE inhibitors or aminoglycosides, or been exposed

Table 4. Some of the drugs commonly associated with AIN

- Antibiotics (e.g. methicillin, cephalosporins, sulfonamides, rifampicin, erythromycin, and ciprofloxacin)
- Diuretics (e.g. furosemide, thiazides, chlorthalidone)
- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Anticonvulsant drugs (e.g. phenytoin, carbamazepine)
- Allopurinol

(Faubel et al, 2009)

Figure 3. Post Renal causes of AKI.



Blakeley (2008)

to intravenous contrast media (nephrotoxic) as part of angiographic or radiological investigations in hospital. A number of medications may precipitate or aggravate urinary retention, including tricyclic antidepressants (Vijayan, 2008a). The patient must be assessed for any signs and symptoms of infections as these can lead to AKI. The infection legionella, for example, can directly cause AIN. Severe infections resulting in sepsis may also cause hypovolaemia, a pre renal cause of AKI.

Other etiological factors that must be explored including any symptoms of systemic diseases, such as systemic lupus erythematosus (SLE) including skin rashes, arthritis, cough, hair loss and oral ulcers. This is important as symptoms such as haemoptyis, cough and sinusitis could suggest the diseases Goodpastures syndrome or Wegener granulomatosis, while a recent sore throat or significant skin infections could indicate acute poststreptoccocal glomerulonephritis.

The disorder rhabdomyolysis may be highlighted by symptoms such as muscle pain, tenderness and swelling, while recent gastroenteritis may point towards Escherichia coli-associated haemolytic uremic syndrome. The patient must also be assessed for any history of chronic liver disease, including cirrhosis, along with anaemia or bone pain and hypercalcaemia, which could indicate multiple myeloma. It is important to assess the patient's cardiac status, including history of dysrhythmias, hyperkalaemia and co-existent atherosclerotic vascular disease that typically includes the renal arteries, especially in the older person (O'Callaghan, 2009; Hilton, 2007; Vijayan, 2008a).

Investigations

There are various investigations that the patient with AKI needs to have. (see *Table 6*). These investigations should be individualized depending on the patient's clinical circumstances. It is not necessary, for example, for a patient to have a barrage of immunological investigations if s/he has a urinary tract obstruction, i.e. post renal AKI. However, these investigations may be warranted if the diagnosis is unclear, or there may be a renal inflammatory disorder in the presence of proteinuria and/ or haematuria (Hilton, 2007). There is increasing awareness in the development of newer serum and urine biomarkers for early diagnosis of AKI. Within 24–48 hours after injury, serum creatinine normally rises, but by this time the proverbial horse has bolted. It is proposed that biomarkers that can distinguish renal injury within hours, just as troponin does for myocardial infarction, could assist greatly in both the initial diagnosis and management of AKI (Vijayan, 2008a).

Waiker et al (2008) assert that any future research concerning biomarkers of AKI should consider important consequences of the actual patients' injury, for example whether there is a need for dialysis, the length of hospital stay and whether or not they correspond to changes in serum creatinine or other markers of GFR

Management

There are three phases within the clinical course of AKI (see *Table 7*) (Pratt and Nouri, 2009). The aims of the treatment for AKI include the correcting of the primary disorder (which can mean removing nephrotoxic insults, for example, discontinuing the use of applicable drugs such as NSAIDs, or removing the obstruction in post renal failure) along with the correction of fluid and electrolyte disorders, the prevention of infection, the maintenance of optimal nutrition, the treatment of systemic effects of uraemia and the provision of education and support to the patient and family (Biel et al, 2008). Renal replacement therapy (RRT) may be initiated if the above treatment principles are ineffective. RRTs are discussed further later in the article.

Fluid management

The nurse plays a pivotal role in the assessment of the patient's volume status to determine if he/she is hypovolaemic or hypervolaemic (Byrne and Murphy, 2008). Not all patients in shock are hypovolaemic. For example, the patient experiencing cardiogenic shock can be negatively affected by fluid challenges (Ashley, 2008). The treatment must be individually tailored to the patient in order to address the precise presentation of AKI (Biel et al, 2008)

Volume resuscitation is vital in the management of pre renal AKI, with the first choice usually being crystalloids (e.g normal saline), along with colloids or blood as necessary later. The goal is to restore renal perfusion, reduce ischemic time and prevent the development of intrinsic renal failure (Biel et al, 2008). It can be challenging to re-establish effective renal perfusion in patients with volume redistribution from the intravascular space (for example capillary leak, low serum albumin, congestive heart failure) without hastening indicative volume overload or pulmonary oedema. In these circumstances, use of a packed red cell infusion along with invasive monitoring, such as central venous pressure monitoring, should be considered (Pratt and Nouri, 2009).

There are many clinical features that a patient with oliguric will present with (see *Table 8*). The

Table 5. Differences between AKI and CKD

	Acute Kidney Injury	Chronic Kidney Disease
History	Short duration History of acute illness	Long duration History of kidney disease and/ or causative co-morbidity
Examination	Acutely ill May have hypotension, fluid overload, metabolic acidosis,	Better toleration of biochemical abnormalities Anaemia, cachexia, grey discolouration of skin
Creatinine	Rapidly increasing values	History indicating derangement Stable serial measurements
Calcium	Usually normal	Low in untreated chronic disease
Haemoglobin	May be normal or low	Due to erythropoietin deficiency- chronic normocytic anaemia
Renal ultrasound	Often normal When the cause is acute obstruction- hydronephrosis	Scars from reflux nephropathy Renovascular disease- asymmetry Large kidneys in • Diabetes, • Amyloidosis, • Chronic Hydropnephrosis • Polycystic Kidney disease

Adapted from Hall (2008)

Table 6. Investigations for AKI

- Urinalysis- Dipstick for blood and/ or protein, Cultures, Microscopy for cells, casts, crystals
- Biochemistry- Serial urea, creatinine, electrolytes, Blood gas analysis, Serum bicarbonate, Creatine kinase, Myoglobinura, C-reactive protein, Serum immunoglobulin, Serum protein electrophoresis, Bence Jones proteinuria
- Haematology- Full blood count, Blood film, Coagulation studies
- Immunology – Anti-nuclear antibody (ANA), Anti-double-stranded DNA antibodies, Antineutrophil cytoplasmic antibody (ANCA), Antiproteinase 3 (PR3) antibodies, Antimyeloperoxidase (MPO) antibodies, Complement levels, Antiglomerular basement membrane antibodies, Antistreptolysin 0 and anti-DNAse B titres.
- Virology- Hepatitis B and C, HIV
- Radiology- Renal ultrasound, Angiography or Ultrasonographic Doppler studies or radio-isotope methods, CT, MRI
- Renal Biopsy

(Hilton, 2007; Holcombe and Kern Feeley, 2009; O' Callaghan, 2009)

patient must be assessed for signs and symptoms of fluid overload, including pitting oedema, peri-orbital, sacral, and peripheral oedema, and rapid pulse with auscultation of the heart (potentially indicating the presence of a third or fourth heart sound or murmur secondary to volume overload (Adams and Snyder, 2009).

Other presentation features can include rapid dyspnoea, jugular venous distention, increased central venous pressure, weight gain, moist tongue and hypertension. Vital signs must be monitored frequently as the clinical situation presents. There is a need for a strict intake and output, with daily fluid intake being 500 mL, plus the amount equal to the urinary output of the previous day (Walsh and Crumbie, 2007). When administering fluid boluses, the cardiovascular response should be monitored in reaction to the increased intravascular volume, and an increase in both the blood pressure and central venous pressure would be expected (Biel et al, 2008). Anti-hypertensives must be administered and any potential side effects monitored if the fluid overload cannot be managed by adjusting the fluid balance (O'Callaghan, 2009).

The UK Renal Association does not advocate the use of loop diuretics like frusemide in the deterrence and treatment of AKI (Davenport et al. 2008). Faubel et al (2009) asserts that there is no data which promotes the use of high dose diuretic therapy in established ATN, However, frusemide and other loop diuretics are often utilized in oliguric AKI in an attempt to alter it to non-oliguric AKI.

Metabolic acidosis management

Metabolic acidosis can occur as renal failure due to the inability of the nephrons to secrete and expel hydrogen ions and reabsorb bicarbonate ions.

Within the critically-ill patient, metabolic acidosis may be intensified due to coexisting conditions such as diabetic ketoacidosis or lactic acidosis which can subsequently increase the discharge of intracellular acids into the circulation as a result of the high catabolic state that present with these conditions. Clinical features of metabolic acidiosis include nausea and vomiting, Kussmaul respirations (deep and rapid respirations), hyperkalaemia, tachycardia, distorted mental status. In the situation of severe metabolioc acidiosis, hypotension and bradycardia can develop due to myocardial depression and vasodilation, along with a sudden depression of consciousness level leading to a comatose state. Arterial blood gases should be monitored frequently, along with constant measuring of the patients' oxygen saturation level with a pulse oximetry and oxygen therapy through the use of a face mask or nasal specs as applicable.

Depending upon the clinical manifestations, mechanical ventilation may be required. The patient must be assessed for signs and symptoms of pulmonary distress. The physiotherapist must be involved in the care conducting chest percussion, airway suctioning, incentive spirometer, turning the patient in the bed and if feasible mobilising out of bed to a chair (Holcome and Kern Feeley (2009). A refractory metabolic acidosis with a pH < 7.1 would be an indicator to commence RRT (Davenport et al, 2008).

Electrolyte management

Patients' electrolytes, including sodium and potassium levels, fluid balance and weight must be monitored to determine fluid requirements (Walsh and Crumbie, 2007). The early recognition and management of hyperkalaemia is vital. The function of the cardiac system can be impaired by retention of potassium and fluid, or by hypertension, which can be associated with renal parenchymal disease. ECG monitoring should be conducted, and may include tall-tented T waves, ST segment depression, prolonged P-R interval, and broadening of the QSR complex with eventual ventricular fibrillation and cardiac standstill (Walsh and Crumbie, 2007). If hyperkalaemia develops, an intravenous infusion of insulin and dextrose can be prescribed as a bolus dose, or by slow infusion. It is very important to monitor blood sugars when administering these infusions. Potassium should be restricted in the patient's diet and potassium-sparing diuretics should be

eliminated. However, RRT should be initiated if the patient has a refractory hyperkalaemia > 6.5 mmol/litre (Davenport et al, 2008).

When the patient enters the diuretic phase of AKI, it is important that the nurse continually assesses the fluid volume and electrolyte balance. This diuresis of (as much as 6000 to 8000 mL in 24 hours) is accompanied by significant losses of potassium, sodium and water as a result of the renal tubules being unable to regulate the volume and composition of urine. During this diuresis fluid intake needs to increased to account for the fluid volume lost. The patient should be constantly monitored for signs of dehydration, like loss of skin elasticity and dry mouth.

Immune system management

Haematological manifestations can occur in AKI, exposing the patient to bleeding tendencies, including reduction in clotting factors, and platelet synthesis. This may cause haematemesis, malaena and anaemia. There is impairment in the immune system, and the patient will be prone to developing infections such as wound, urinary, pneumonia and sepsis, which can be a major cause of mortality in AKI.

The patient must be assessed for signs and symptoms of infection, like pyrexia and laboratory indicators (for example raised ESR and CRP, blood and urine cultures as applicable). The multidisciplinary team must conduct frequent and meticulous hand hygiene before dealing with the patient. Hospital visitors and invasive procedures should be reduced wherever possible (Holcombe and Kern Feeley, 2009).

Nutritional management

Uraemia causes must be addressed, for example, by administering anti-emetic medications in cases of nausea and vomiting. A renal dietician will work with the rest of the team, especially the nurse, regarding an individualized diet prescription that considers the patient's catabolic rate (Biel et al, 2008). Protein calorie malnutrition has been implicated as one of the contributing factors in the high mortality rate seen in AKI. Nitrogen balance is exceptionally negative, especially in AKI this is linked with sepsis, post surgery and multiorgan dysfunction syndrome. This negative nitrogen balance is contributed by various renal factors such as uraemia, acidosis, inadequate protein intake and parathyroid hormone abnormalities. Supplemental nutritional support with enteral versus parenteral nutrition may enhance nutritional status, reduce infections and sepsis along with producing a better survival rate in critically ill patients. Therefore, enteral feeding is the preferred method of nutritional support (Faubel et al, 2009).

Table 7. Clinical Course of AKI

- Initial Phase: This is the time frame between the exposure to an insult and a reduction in renal function when renal damage can be potentially reversed.
- Maintenance Phase: This period can last from days to weeks or sporadically, up to 2 months, during which renal damage cannot be reversed. During this phase, patients can be anuric, oliguric or nonoliguric.
- Recovery Phase: This phase is marked by a return of serum BUN and creatinine toward normal ranges. The patients may experience a polyuric phase which can result in fluid and electrolyte abnormalities. Therefore the recovery function may be incomplete.

(Pratt and Nouri, 2009)

Table 8. Clinical Features – Oliguric phase

- Fluid Volume Overload: pulmonary and peripheral oedema, dyspnoea, hypertension.
- Blood Chemistry effected: urea, creatinine, sodium, calcium, phosphate, hyperkalaemia
- Uraemic state: headache, apathy, hiccups, nausea, alterations in mental state
- Metabolic Acidosis
- Risk of Infection
- GIT- anorexia, nausea, vomiting, hiccoughs, gastritis-GIT bleeding
- Anaemia and Platelet Dysfunction- bruising, bleeding

Personal care management

Issues such as mouth care are imperative. The patients' tongue can become coated with saliva excretion, along with dry mucosa. The lips becomes regularly encrusted. The symptoms of uraemia such as urea breakdown in saliva can cause the patient to experience dysgeusia (a distortion of the sense of taste), which can result in a metallic taste in the mouth and a peculiar smell to the breath. The patient may be prone to intractable hiccups as a result of metabolic derangements. This can be managed by dialysis (Lee and Bosch, 2007). Mouth lesions can lead to further complications, like respiratory infections. The nurse may encourage the patient to rinse out their mouth with ice cold mouthwashes or water rather than lukewarm solutions, and suggest applying petroleum jelly to the lips (Walsh and Crumbie, 2007). Skin care continues with regular washing and close monitoring of the skin's condition, along with regular pressure-area care, especially if the patient is oedematous.

Patient education management

The patient with AKI will need emotional support from the nurse and members of the multidisciplinary team. This time is extremely challenging, not just for the patient, but for the family as well. The patient's knowledge and understanding of AKI must be comprehensively assessed, and the course of treatment and long-term outlook should be discussed. The family must be involved and they, along with the patient, should be given the opportunity to have the various procedures and treatments explained to them. Issues arising from the patient's physical status, such as the effect of uraemia on concentration levels, fatigue, and nausea must all be taken on board when explaining their care.

Open-ended questions, and being listened to may enable the patient to discuss potentially difficult thoughts and feelings regarding this acute illness and open up a space to freely discuss concerns. The nurse should also be in a position to refer the patient and family on for further psychological support if necessary.

Prevention of AKI

There are various mechanisms that should be considered in order to prevent AKI. A patient's risk of AKI should be identified based on their pre-existing medical conditions, the type of surgery they are undergoing (cardiovascular, poses a particularly high risk) and planned peri-operative use of nephrotoxic substances. The pre-operative hypovolaemia should be corrected by maintaining intraoperative euvolaemia and stable haemodynamics using support from invasive haemodynamic monitoring as needed. Intravenous hydration, combined with acetylcysteine should be utilized for the prevention of radiocontast nephropathy. The agent acetylcysteine is a known antioxidant and is effective in decreasing the incidence of contrast nephropathy after intravenous administration of radiocontrast media in patients with chronic renal deficiency.

Other areas to note in the prevention of AKI include the avoidance of hypoxia. There should be a close observation of nephrotoxic drugs and prescribing two nephrotoxic drugs together must be avoided. The levels of these

drugs must be monitored. It should be assumed that postoperative oliguria is hypovolaemic in nature until it is established otherwise (Jarnberg, 2004).

Renal replacement therapy in AKI

As identified, the foundation of the management of AKI is prevention, but factors that activate RRT include metabolic acidosis, hyperkalaemia, uraemia and volume overload. The available dialysis modalities are haemodialysis (HD), continuous renal replacement therapy (CRRT), sustained low-efficiency dialysis (SLED) or peritoneal dialysis (PD). The choice depends on a number of circumstances, including which therapies are on offer at the patient's hospital, their haemodynamic state, present co-morbid conditions, and the physicians preference. It is important to recognize that not all patients will choose to receive RRT. This must be assessed on a case-by-case basis, and the ramifications of not receiving this treatment must be thoroughly explained to both patient and family. Patients with sepsis or hepatic failure may see possible benefits with continuous therapies.

In spite of the overall safety of these dialysis modalities, complications and adverse problems can occur. This requires detailed attention, and in some instances, frequent laboratory measurements to anticipate and avoid their incidence (Vijayan 2008b).

Conclusion

AKI is multifactorial in nature. The RIFLE classification and the Acute Kidney Injury Network Diagnostic Criteria are two internationally recognized systems for classifying AKI, which should help the multidisciplinary team to manage the care of a patient with this critical disorder. There are three causes of AKI which are categorized into pre renal, intra renal or intrinsic, and post renal failure. It is important to recognize that not all AKI is reversible. The prognosis of pre renal and post renal failure is comparatively good if the precipitating injury is corrected. Intrinis or intra renal causes of AKI have a poorer diagnosis with the mortality being 38% among hospitalized patients, and 79% among patients in the intensive care unit. The modality figures advance with age, co-morbidities, sepsis, oliguria and multiorgan failure (Pratt and Nouri, 2009).

Prevention of AKI is paramount. It can occur in both hospital and community settings, and nurses should be vigilant and able to identify individuals who are at greater risk of developing AKI. At-risk groups include older people, those with multiple co-morbidities, people with pre-existing renal disease, post surgical patients (especially cardiovascular), post-trauma patients who suffered major blood loss and muscle damage, those who have been exposed to nephrotoxic insults such as nephrotoxic medications, and patients with multiple organ dysfunction (Biel et al, 2008). The nurse must provide holistic care when managing the patient with AKI, continually monitoring the patient's progress, correcting fluid and electrolytes, treating systemic effects of uraemia, maintaining optimal nutrition, and preventing infection, along with the constant provision of information and support to the patient and family members.

This care is challenging and requires the provision of ongoing education for nurses and continual collaboration with the multidisciplinary team to effectively mange the patient presenting with AKI. BJN

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KEY POINTS

- AKI is prevalent both in the hospital and community setting.
- There are two international recognised classifications of AKI namely RIFLE and AKIN.
- The causes of AKI are pre renal, intra renal (intrinsic) and post renal.
- The main nursing priority in the management of AKI is to identify the aetiology and to treat same as expediently as possible.