Chapter 6: Acute Kidney Injury

Author: Fiona Murphy
Assistant Professor & Renal Educational Facilitator, School of Nursing & Midwifery,
Trinity College Dublin

Principles of Nursing in Kidney Care (Editors: Afra Masià-Plana & Anastasia Liossatou)
Endorsed by both the EDTNA/ERCA and the ESNO (European Specialist Nurses Organisation)
Publication: 7 January 2024
Springer Link Publication

Abstract

Acute kidney injury (AKI) is a serious life-threatening clinical syndrome which is prevalent throughout primary and secondary care settings worldwide. There are many risk factors for AKI and its impact is substantial in terms of individuals concerned and the relevant healthcare settings. There are various types of AKI including hospital acquired, community acquired and more recently COVID-19 associated AKI. Nurses play a pivotal role in identifying AKI in a prompt manner along with other members of the interdisciplinary team. Both education and emotional support are vital for individuals affected by AKI. Nurses need to update their knowledge and understanding of AKI so that they can continue to maintain best practice within their clinical areas.

Learning outcomes

- To discuss the definition and stages of acute kidney injury (AKI) as well as the aetiology, pathophysiology, risk-factors and outline its epidemiology.
- To identify acute kidney disease (AKD) term and its interlinkage with AKI and chronic kidney disease (CKD).
- To discuss the initial and ongoing management of AKI including volume status, medication review, pharmacological and nutritional support throughout AKI.
- To highlight the complications of AKI to include hyperkalaemia, metabolic acidosis, pulmonary oedema, and indications for kidney replacement therapy (KRT).
- To recognise the various types of AKI such as hospital acquired AKI, community acquired AKI, drug-induced AKI, COVID-19 associated AKI, sepsis associated AKI and contrast-induced AKI.
- To address the relevance of education and emotional support for individuals with AKI and their partners/ family members as applicable.
- To outline the relevance of education needs for nurses in the management and preventative measures of AKI.

Key words: nursing, acute kidney injury, acute kidney disease, chronic kidney disease, education, prevention, sepsis, primary care, secondary care, hospital acquired AKI, community acquired AKI, COVID-19 associated AKI.

Introduction

This chapter will address the main principles of nursing care for individuals presenting with acute kidney injury (AKI). That old phrase of 'prevention is better than cure' is so apt when identifying how to manage AKI with prevention ideally the first line of defense through education and close monitoring of those who are at risk. Nurses whether working in primary or community care settings or within secondary or hospital care settings must 'Think Kidneys' in their care delivery. There must be effective ongoing communication between nurses and other members of the interdisciplinary team as without this care delivery could deteriorate. The importance of not forgetting basic nursing care is paramount within AKI care delivery. This includes nurses being mindful of risk factors of AKI, assessing clinical history and clinical presentation, close monitoring of vital signs and fluid balance, importance of good hydration, and nutrition, using both clinical judgement and sound intuition along with reporting any concerns in an expedient manner so appropriate management can be promptly delivered.

AKI is exceptionally widespread in adults who are hospitalised ensuing in 10-20% of emergency hospital admissions and coupled with major adversative outcomes. AKI is not just a major concern within secondary care or hospital care settings, but also plays a pivotal role in primary or community care settings in terms of prevention with prompt identification and management along with post AKI care. The adversative outcomes of AKI signify the reality that AKI behaves as a 'force multiplier' and enhances the seriousness of co-existing acute illness. AKI basically is an indicator of the 'individual who is unwell' who needs timely detection and treatment. Those adults with AKI signify a heterogeneous group seldomly representing with a solitary disease process. Sepsis and multi-organ failure is frequently represented with AKI. [1, 2].

Background and epidemiology of AKI

AKI is a major public health concern with its associated increased morbidity and mortality rates. However, it is probable that a considerable number of deaths correlated with AKI could be avoided. This has been identified based upon preceding studies that unnecessary deaths from AKI are known to occur because of three distinct settings. These are secondary to public health concerns such as unsanitary water, diarrhea, and widespread infections; secondly delayed or lack of detection, scarcity of access to laboratory studies, insufficient response or iatrogenic issues resulting in further insults to a deteriorating kidney. Finally insufficient dialysis support to manage hyperkalaemia, fluid overload and acidosis all of which are life threatening. [3-6]. It was established by the National Confidential Enquiry into Patient Outcome and Death within the United Kingdom (UK) in 2009 that up to 100,000 deaths in hospitals related to AKI and a quarter to a third of these deaths could potentially have been avoided. [7]. Further adverse outcomes associated with episodes of illness convoluted by AKI include increased mortality (both short and long term), cardiovascular events, rehospitalisation, development, and progression of chronic kidney disease (CKD) and reduced quality of life. [8-12].

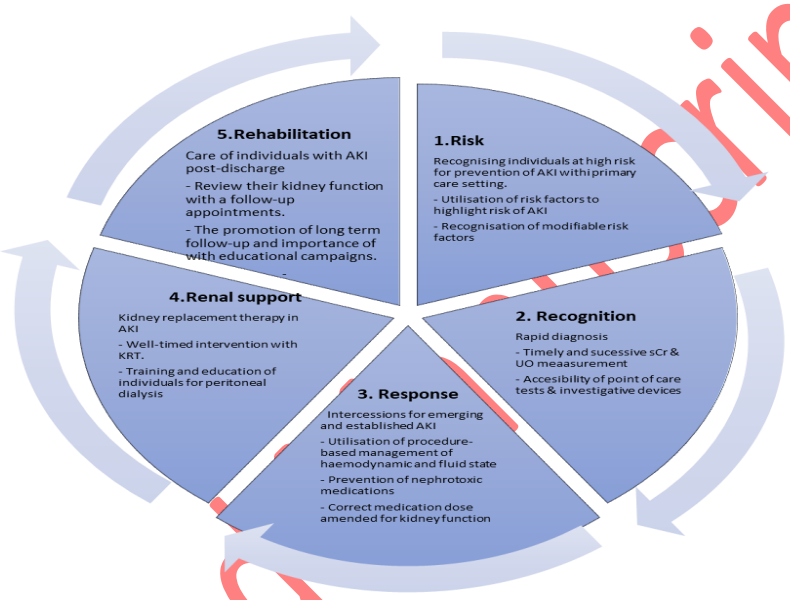
AKI can transpire with individuals within primary or community care settings with or without any acute illness [10,11,12]. It can often ensue in hospitalised patients including those in the emergency department (ED), intensive care unit (ICU), coronary care unit (CCU), high dependency unit (HDU), outpatients, theatre, oncology, maternity care, mental health services, along with surgical, medical, and care of the older person's units. One could contend that there is potentially not a clinical area where AKI could not transpire with individuals when delivering their nursing care [13]. Individuals can present with multiple comorbid conditions including older persons with complex health issues. [3, 10, 11]. It is imperative therefore that nurses and other members of the interdisciplinary team understand what AKI is, recognise the signs and symptoms, and how to manage it in a prompt effective manner so that holistic care can be delivered to individuals concerned. [13, 14].

The International Society of Nephrology (ISN) acknowledging the rising global concerns of AKI instigated in 2013 the AKI 0 by 25 initiative with the impressive aim to eliminate every preventable AKI death globally by 2025. [5]. The incidence of AKI reported as generally either community-acquired or hospital-acquired AKI. Hospital-acquired AKI occurring primarily in high-income countries, whilst in lower-income settings community-acquired AKI is more widespread. These configurations are applicable internationally to both adults and children. [15]. The incidence of AKI within community and hospital care settings has increased with an estimated excess of 13 million individuals. AKI is increasingly observed in primary care settings in persons without any acute illness, and recognition of the condition needs to heighten among primary health care professionals. [4, 11]. including nurses working within all areas of community care settings. There are limited studies that have concentrated on community-acquired AKI in low-resource settings despite knowledge of the epidemiology of AKI having increased greatly because of the standardised AKI classification system used. [6]. The Global Snapshot was an initial large epidemiological study to investigate globally the outcomes related to AKI involving data from patients from both ICU and non-ICU. It highlighted that community-acquired AKI (main causes being dehydration, infection, and sepsis) was more prevalent than hospital-acquired AKI

in low middle income countries and correlated with more severe AKI at presentation and adverse outcomes. [16].

There is a requirement to increase the awareness of AKI especially the recognition of those individuals who present with earlier stages of AKI based on the findings of this Global Snapshot study. [6]. The 0 by 25 initiative recommended the use of the 5R framework (risk, recognition, response, renal support, and rehabilitation) to address the main essentials for a sustainable basis to maintain AKI care (Figure 1). [5, 6].

Figure 1. 5R Framework. Methta et al (2015); Macedo et al (2019).



Interestingly AKI identified as an important target for the European nephrology community with a proposed model to promote clinical partnerships including the development of a pan-European network of researchers and clinicians to enhance for example clinical outcomes and promote research on AKI in Europe. [17]. 'Think Kidneys' is a nationwide program led by the National Health Service (NHS) England in collaboration with the UK Renal Registry (UKRR) with the purpose being to ensure the avoidance of harm connected to AKI is averted in all healthcare settings. This program has a wealth of online educational resources and several best practice guidance to increase knowledge and understanding for healthcare professionals written by experts throughout the UK along with use of 'Think Kidneys' AKI app which contain many relevant resources. These resources are not just for those working within a renal healthcare background but instead across spectrum of care delivery such as primary, secondary care (hospital care settings) cancer care, mental health, and residential or care homes. One best practice guidance is an example of the importance of early recognition of AKI through the AKI warning stage test results produced from electronic detection systems. This is based in biochemistry laboratories which are relayed to primary or community care settings with the purpose to make it easier to notice any changes in serum creatinine concentration. However, it is vital to treat individuals and not just the test result as it must be considered in the overall clinical circumstances. [18].

The global financial costs of AKI to health care systems remains high. [4]. In the UK it estimated that within the NHS the spending on AKI (exclusive of costs in primary care settings) anticipated to be amid £434 million and £620 million annually, which is more than the expenditure combined related to breast cancer, or lung and skin cancer. [11]. AKI is associated in the United States (USA) with an

escalation in hospitalization costs that range from $5.4 to $24.0 billion. The costliest are those individuals with AKI of sufficient severity to necessitate dialysis. Regardless of these costs, there is considerable hospital-level variation still present in the cost of AKI care in the USA. These appraisals only report the inpatient costs of AKI and overlook AKI that ensues in primary care settings and the enduring significances of an AKI episode. [19].

Definition of AKI

AKI is typified by the accrual of creatinine, urea, and other unmeasured waste products following a sudden decrease in kidney function. It was previously known as acute renal failure. However, 'injury' has replaced 'failure' to highlight the disease continuum as even discreet reductions in kidney functions are associated with adverse outcomes. AKI is multifactorial in nature. It is not just kidney failure but instead incorporates an extensive spectrum of injury to the kidneys from minor changes in kidney function to the most severe where kidney replacement therapy (KRT) or renal replacement therapy (RRT) may be essential. [3, 4, 13]. AKI can occur because of numerous insults to the kidney including decreased kidney perfusion, intrinsic kidney disease, exposure to nephrotoxins and blockage to urinary outflow. The consequences of this involve reduced clearance, volume regulation, control of metabolic balance homeostasis and distorted acid/ base and electrolyte balance. [20]. There is no universally accepted validated risk score for AKI for either primary or secondary care (hospital care settings). [21].

Staging of AKI

During the past near twenty years there has been international guideline groups that have endeavored to determine coherent definitions and staging systems for AKI. [22]. The RIFLE classification was identified in 2004 as a diagnostic standard of acute renal failure (ARF) with the intention to improve the morbidity and mortality of ARF. This classification considers three severity classes (risk of kidney dysfunction, injury to the kidney and failure of kidney function) matching to the changes in serum creatinine and/or urine output and two outcome classes (loss of kidney function and end stage kidney disease (Table 1). Although this chapter is addressing the management of adults with AKI, it is important to recognise that AKI can develop in babies, children, and teenagers with the availability of (p) RIFLE guidelines (paediatric risk, injury, failure, loss, end stage renal disease) to assist nurses and other interdisciplinary team members in both detection and care delivery. [3-4, 22-23]. The RIFLE guidelines were modified by the AKI Network (AKIN) another group of intensive care and nephrology global experts who enhanced the RIFLE criteria and renamed the classes as stages 1, 2 and 3. The AKIN classification of AKI characterised any individual needing KRT as stage 3 AKI (Table 1). [3-4, 22-24].

Table 1. Comparison of Consensus AKI Definitions (Moore et al, 2018).				
AKI Stage	**Urine Output**	**KDIGO**	**AKIN**	**RIFLE**
1	<0.5ml/kg/h for 6-12 hours	sCr to 1.5 -1.9 x baseline over 7d or ≥ 0.3mg/dL absolute increase over 48 hours	sCr to 1.5 -2.0 x baseline or ≥ 0.3mg/dL absolute sCr increase within 48hours	*Risk:* sCr to ≥ 1.5 x increase within, sustained for ≥ 24 hours
2	<0.5ml/kg/h for ≥ 12 hours	sCr to 2.0-2.9 x baseline	sCr to ≥ 3.0 x baseline	*Injury:* sCr to ≥ 2.0 x baseline
3	<0.3ml/kg/h for ≥ 24h or anuria for ≥ 12 hours	sCr to ≥ 3.0 x baseline, or sCr increase to ≥ 4.0mg/dL or initiation of KRT	sCr to ≥ 3.0 x baseline, or sCr increase to ≥ 4.0mg/dL (with increase of 0.5 mg/dL) or initiation of KRT	*Failure:* sCr to ≥ 3.0 x increase sCr increase to ≥ 4.0mg/dL (with increase of 0.5 mg/dL) or initiation of KRT
				Loss: Complete loss of kidney function for > 4weeks
				ESKD: ESKD for > 3 months

The Kidney Disease Improving Global Outcomes (KDIGO) AKI guidelines in 2012 merged the RIFLE and AKIN classifications so that there is a standardised classification of AKI for the purposes of practice, research, and public health. There is a more definitive timeframe within this definition, although staging remains based upon serum creatinine (sCr) concentration and urinary output (Table 1). [3-4, 14, 25-27].

It has been recommended that the highest stage be considered if the sCr concentration and urinary output does not relate to the same stage. Urine output is a vital kidney function consideration that highlights individuals at increased risk for adversative outcomes. However, its pathophysiological importance in the absence of excesses of oliguria or other substitutes of reduced glomerular filtration rate (GFR) is more contentious. It should be recognised that individuals who develop AKI based on the KDIGO urine output measures irrespective of whether sCr measures are available, are endangered to developing fluid overload due to the usually high obligate intake of seriously ill persons. [3]. Since the creatinine concentration is not at a stable state throughout AKI the existing eGFR equations (Cockcroft-Gault, CKD-EPI and MDRD study) cannot be applied. The use of equations has been suggested to calculate kinetic GFR when sCr concentration is actively altering, however this has not been endorsed for extensive use. The premise should be that the GFR is < 10mL/min when urine output is negligible which can occur when the individual is oligo-anuric in severe AKI. However, it has been identified that sCr concentration levels is a shortcoming of the KDIGO classification. It could be viewed as flawed biomarkers of AKI being influenced by various acute or chronic factors making it challenging to determine a precise diagnosis. The rise of sCr concentration levels does not automatically indicate AKI as it is a functional marker [3-4, 25-27].

The National Institute for Health and Care Excellence (NICE) in the UK identified that AKI in adults can be detected in line with the AKIN or KDIGO definitions by utilizing any of the subsequent criteria [10-12]. Whilst the UK Kidney Association (UKKA) clinical practice guidelines on AKI also endorsed the KDIGO system for the diagnosis and staging of AKI with the current NHS England biochemical detection algorithm being used to apply the sCr based criteria. The incidence of an active episode of AKI arising in the secondary care or hospital care setting can be deduced from regular sCr testing at 12 and 24 hours post the index value. [2]. However, it is vital to apply clinical judgement in clarifying consequences for individuals involved, including for those who may have pathological AKI and do not meet diagnostic or staging criteria. The core management of AKI is to treat the primary cause therefore irrespective of the stage of AKI, a diagnosis of AKI is only partial without efforts made to delineate its

aetiology. The degree to which additional investigations are needed will depend upon clinical judgement for example when the clinical image implies acute glomerulonephritis but not ischaemic AKI a histological confirmation may be required. [3].

Acute Kidney Disease (AKD)

It is recognised that both AKI and CKD are well defined and classified, however there remains a disparity in research, care, and guidance for those individuals with abnormalities in kidney function and/or structure who do not meet the definition of AKI or CKD. [28]. Both AKI and CKD embody common presentations for heterogeneous conditions. These presentations in principle could be caused by similar diseases that cause AKI or CKD which could be identified, evaluated, and managed before they develop same. The lack of a recognised definition and nomenclature for these presentations signifies a plausible disparity in the classification of kidney disease (KD) and disorders. This results in individuals and health care professionals lacking management recommendations that could possibly enhance care and outcomes. [29]. The KDIGO AKI guidelines proposed a working definition of acute kidney diseases and disorders shortened to acute kidney disease (AKD) to address this disparity and to complement the criteria for the continuum of clinical presentations of AKI, AKD, CKD and the lack of these presentations or what is identified as no known kidney diseases (NKD) (Table 2). [25, 28-29].

Table 2 | Functional and structural criteria for kidney diseases and disorders.
Acute kidney disease (AKD); acute kidney injury (AKI); chronic kidney disease (CKD); glomerular filtration rate (GFR); no kidney disease (NKD): serum creatinine (sCr) (Lameire et al, 2021).

	AKI	**AKD**	**CKD**	**NKD**
Duration	Within 7 days	< 3 months	> 3 months	
Functional criteria	Increase in sCr by 50% within 7 days Or Increase in SCr by 0.3mg/dl (26.5µmol/l) within 2 days Or Oliguria for > 6 hours	AKI Or GFR < 60ml/min/1.73 m^2 Or Decrease in GFR by >35% Or Increase in sCr by > 50%	GFR < 60ml/min/1.73 m^2	GFR >60ml/min/1.73 m^2
And/or		And/or	And/or	And
Structural criteria	Not defined	Marker of kidney damage (albuminuria, haematuria, or pyuria are most common)	Marker of kidney damage (albuminuria is most common)	No kidney damage

The Acute Dialysis Quality Initiative (ADQI) 16 report in 2017 suggested an alternative definition and a classification of AKD, as well as an amended definition of AKI. This report perceived in the majority of cases AKI as preceding AKD with AKI having a window of 7 days with continuation beyond this timeframe as being AKD. AKD was classified according to the previous AKI stage. [29-30]. KDIGO arranged a consensus workshop with global experts and researchers online in 2020 to investigate current data and discuss main concepts relative to AKD. The consensus reached include the maintenance of the KDIGO definition of AKD, a recommendation of a classification of KD in harmony with the KDIGO definitions of AKI and CKD along with management approaches, and priorities for research. [28-30].

Aetiology, pathophysiology, and classifications of AKI

The aetiology of AKI can be separated based on its pathophysiology into classifications of AKI. The three classifications of AKI are pre-kidney (pre-renal) AKI, intrinsic AKI, or post-kidney (post-renal) AKI. [20].

Pre-kidney AKI

This is the most common cause usually seen in about 80% of cases. [11, 25]. Pre-kidney AKI is where decreased kidney perfusion causes injury. The response of the kidneys to decreased perfusion pressure is to increase sodium and water reabsorption. In the carotid artery and aortic arch baroreceptors activate the sympathetic nervous system in reaction to the reduced blood pressure. The GFR of the kidneys is maintained within a narrow scale by both vasoconstriction in the efferent arteriole and vasodilation of the afferent arteriole. The reduced perfusion within the kidneys stimulates the activation of the renin-angiotensin-aldosterone hormonal system which is vital for the regulation of blood pressure and fluid balance. The potent vasoconstrictor angiotensin II stimulates the release of aldosterone encouraging sodium and water reabsorption at the collecting duct. The hypothalamus is stimulated by the reduced blood volume supporting the release of the antidiuretic hormone which amplifies tubular water reabsorption resulting in concentrated urine. [20]. The integrity of the kidney parenchymal tissue is preserved and the GFR is corrected quickly with the re-establishment of kidney perfusion with pre-kidney AKI. However, severe kidney hypoperfusion that is untreated could add to the development of ischaemic acute tubular necrosis. [31].

The major causes of pre-kidney AKI are intravascular volume depletion such as haemorrhage, gastrointestinal losses, renal losses (overzealous diuretic usage, osmotic diuresis, diabetes insipidus), increased insensible losses, 'third space' losses (such as crush syndrome or rhabdomyolysis, pancreatitis). Cardiac failure, systemic vasodilation, sepsis, liver failure, anaphylaxis, drugs such as antihypertensives, anaesthetics, afterload reduction and drug overdoses are all examples of decreased cardiac output which is another major origin of pre-kidney AKI. Renal vasoconstriction is another rationale for the development of pre-kidney AKI. This can encompass norepinephrine, ergotamine, hypercalcaemia, sepsis and liver disease. Pharmacological agents that severely effect autoregulation and GFR rate in particular settings are all sources of pre-kidney AKI. This includes angiotensin-converting enzyme inhibitors (ACEi) in severe renal hypoperfusion or in renal artery stenosis or nonsteroidal anti-inflammatory drugs (NSAIDs) throughout renal hypoperfusion. Pre-kidney AKI can sporadically be activated by direct renal vasoconstriction minus overt systemic hypotension as transpires with the use of a calcineurin inhibitor (cyclosporin/ tacrolimus), administration of vasoconstrictors or in the presence of hypercalcaemia. [32].

Intrinsic AKI

This is where there is direct injury to the kidneys itself. It can be beneficial to label intrinsic AKI as the following: glomerular (such as acute and rapidly progressive types of glomerulonephritis); tubular ischaemic (such as extreme pre-kidney AKI, sepsis, pancreatitis), toxic: intrinsic toxins (such as rhabdomyolysis, massive haemolysis, tumour lysis, myeloma) and extrinsic toxins (such as radio contrast and drugs), interstitial (such as autoimmune, toxic, infectious) and vascular: large vessel (such as renal artery stenosis, atheroembolic) and small vessel (such as vasculitis, polyarteritis nodosa, malignant hypertension, haemolytic uraemic syndrome and eclampsia). [33].

Acute tubular necrosis (ATN) accounts for 45% of cases of AKI. It triggered by sepsis in 19% of adults within ICU. [20]. ATN is typified by an extreme decrease in GFR. However, it is not fully understood the association between the tubular injury and the loss of glomerular function. There are three processes that are considered to highlight the diminished kidney function in ATN. Firstly, the concept of intratubular obstruction wherein after an ischaemic or nephrotoxic injury to the kidneys there is occlusion of the tubule lumen distally following tubular epithelial cells and cellular debris being sluffed from the tubular epithelium. The results of these sluffed cells and fragments shape the granular casts spotted in urine sediment. The construct of tubular back leak occurs because of the sluffing of these necrotic and apoptotic tubular epithelial cells ends. This results in denuding of the tubular basement membrane and unfettered back leak of the glomerular filtrate. These two processes result in reduced urine flow across the tubular lumen. The final process is whereby there is both vasoconstriction and

microvascular injury that ensues in diminished glomerular perfusion, directly decreasing the GFR and adding to the enlargement of the initial injury. [31].

The kidney is predisposed to drug-induced injury as drugs and metabolites are simply accrued in high easily concentrations in the kidney. Drug-induced AKI is a generic term for recently occurring kidney injury or kidney injury that has been aggravated by the drugs dispensed for diagnosis and management. It can be catergorised into the following: acute interstitial nephritis, toxic kidney injury, drug-induced indirect toxicity through decreased blood flow and abnormal electrolytes and drug-induced crystal development and urinary tract obstruction kidney injury for calculus development. [27].

Acute interstitial nephritis (AIN) is within the classification of intrinsic AKI and can be known as drug-induced AKI. It is an immune-mediated type of kidney injury that is categorised histologically by immune cells being infiltrated within the tubulointerstitium. Medications are the most prevalent cause of AIN with an estimation of > 70% of AIN monitored in high-income countries. There are over 120 drugs identified to cause AIN with most frequent offenders being antibiotics, NSAIDs, proton pump inhibitors (PPIs) and immune-checkpoint inhibitors (ICPs). It is therefore not surprising that it a relatively common cause of hospital-acquired AKI [34]. AIN may also arise due to infections (such as bacterial or viral) malignancy and immunologically mediated systemic disease such as systemic lupus erythematosus (SLE). [31].

Post-kidney (post-renal) AKI

This is injury to the kidneys because of urinary outflow obstruction. The prevalence of this classification is 5-10% of individuals with AKI primarily in older men who have prostatic problems such as benign prostatic hypertrophy or prostate cancer. These conditions can lead to partial or complete urinary obstruction. Acute urinary obstruction may also be present in young boys with congenital urethral valves. It is important to note that complete urinary tract obstruction is rare in women in the absence of surgery, malignancy, or irradiation within the pelvic region. Kidney, urinary and bladder stones, ascending urinary infection (including pyelonephritis), and urinary retention, retroperitoneal fibrosis, lymphoma, tumour, and trauma are all other causes of post-kidney AKI. [20, 33].

Clinical sequence of AKI

Individuals with AKI may have normal urine output, oliguria, or anuria [4]. The clinical sequence of AKI can separate into four stages. The initiating stage develops when the kidneys injured for example with hypovolaemia or nephrotoxic event. This is when the diagnosis is reached, and therapy established. The timeframe of this stage can last from hours to days. There is a necessity for thorough assessment and prompt intervention at this stage to avoid advancing to the later stages of AKI. It is important to be mindful here of those individuals who are at risk of developing AKI including advanced age, existing CKD, liver failure, heart failure, diabetes, previous history of AKI, hypertension, use of nephrotoxic medications, hypovolaemia, major surgery, history or risk of urological obstruction, use of iodinated contrast agents, sepsis, neurological or cognitive impairment and haematological malignancy. [11, 35-36].

The oliguric stage is the next phase which may last from five to over 15 days. The more protracted this stage the more adverse outcomes there will be for the individual involved who is especially vulnerable to bleeding and infection throughout this stage. This is because of endocrine issues where there is diminished manufacturing of erythropoietin and functional kidney changes including both reduced glomerular filtration and urine formation. The glomerular basement membrane substituted with fibrous scar tissue and the functional units of the kidneys the nephrons will be congested with inflammatory effects as renal healing begins to process. [35-36].

The diuretic stage is whereby the kidney begins to recuperate many of its absent function as it continues to heal, however this is contingent on how severe the initial kidney injury was. The urinary output begins to increase back to typical levels. However, it is important the individual is closely monitored in terms of polyuria and potential dehydration whereby they could urinate up to 3 litres daily in this stage. This can be because of either reduced fluid reabsorption by damaged renal tubules or the osmotic impact of accumulated metabolites. The individual's signs and symptoms related to their initial condition should also begin to recede within this stage. The recovery stage or convalescent phase is the final stage and can last anything from a few months to over a year. It commences when laboratory values stabilise and concludes when kidney function returns to normal. The glomerular basement membrane returned to its former structure with clinically insignificant scar tissue remaining. [35-36].

Clinical presentation of AKI

The clinical appearance of AKI is contingent on the cause and gravity of the renal insult. AKI is often regarded as a 'silent disease '. Individuals with most types of AKI lack explicit signs and symptoms initially in the development of the injury. Therefore, detection requires a high degree of suspicion in those at risk especially in those that are acutely ill. They may also present for example with symptoms only applicable to the precipitating illness (such as sepsis). [20, 38]. Those individuals with mild to moderate AKI are asymptomatic and are detected based on laboratory analysis. However, individuals with severe AKI usually present with numerous symptoms for example fatigue, nausea and vomiting, anorexia, restlessness, confusion, and fluid retention. There can be central nervous system indicators such as uraemic encephalopathy with asterixis, polyneuritis, mental status changes, muscle cramps and seizures, platelet dysfunction, and severe anaemia in serious and protracted AKI. [4].

It is important therefore to assess the individuals' medical and social history and determine their risk factors such as frailty, older age, CKD, diabetes mellitus, liver disease, heart failure and cognitive and/ or neurological impairments. Their oral intake should become established along with any gastrointestinal symptoms, manifestation of rash or joint symptoms, urinary output, and any susceptibility to urinary tract infections (UTIs) or any other urinary symptoms and/or history with same. The most common causes of AKI triggering kidney insult include sepsis, hypovolaemia and/ or hypotension and nephrotoxins. The types and dosages of medications should be reviewed such as nephrotoxic, over the counter, recreational drugs, and the use of herbal therapies. [20, 22, 37-39].

Recreational or occupational history should be established such as recent travel, tropical diseases, exposure to sewer systems and rodents. Initially there should be airway, breathing, circulation, disability, exposure (ABCDE) assessment conducted, and observations recorded using the National Early Warning Score 2 (NEWS2). There should be monitoring for any signs of sepsis and palpation of the abdomen examining for a full bladder. Since AKI is so familiar in individuals who are acutely ill the Royal College of Physicians in the UK advocates a NEWS2 score of 5 or above. This should trigger a check concerning fluid intake, urinary output, and kidney function all relative for AKI. It is important to be mindful that some individuals with AKI may not have an escalated early warning score as urinary output is not incorporated in general used scores such as NEWS2. Therefore, oliguria which is an indicator of potential AKI will not generate any escalation in the individual's score. [20, 22, 37-39]. The Irish NEWS2 identifies the medical team must be notified if an individuals' urine output is < 0.5mL/ kg/ hr when monitoring their INEWS2 which acts as an aid for nurses to use their clinical judgement and decision making. It identifies this as a small window of opportunity to recognise AKI. The use of ISBAR (identify, situation, background, assessment, and recommendation) communication tool is relevant to escalate the response protocol as required. [40].

The 'Think Kidneys' AKI best practice guidance responding to AKI warning stage test results for adults in primary care identifies the relevance of this test result and its recognition within this pertinent area of care delivery. The AKI warning stage test result regarded as a two-step process wherein stage one

detects the creatinine changes coherent with AKI produced by NHS England detection algorithm. This repeatedly highlights probable causes of AKI from laboratory information in real time and generates a test result (i.e., AKI stage 1, 2 or 3) conveyed together with the serum creatinine result. This test result is known as an 'AKI warning stage '. The second step of this process is advising this AKI result to healthcare staff which is the warning phase of this process. Those positive AKI warning stage results will transmit from the relevant laboratory system to general practice (GP) clinical systems. These systems need to ensure timely communications of test results both in and out of standard working hours in primary care services. Primary or community care teams need to determine how responsive to act on the applicable test result. This includes appropriate action to adopt using the guidance provided by 'Think Kidneys' to maintain a timely and suitable response to AKI warning stage test results. Questions such as what is the stage of AKI? How severe is the AKI with escalating severity of AKI associated with increased risk of adverse outcomes. Stage 1 being the least severe typically treated in primary care settings whilst AKI stage 3 being the most severe should treat in hospital care settings. [18, 21].

This same guidance identifies the importance of nurses and other members of the interdisciplinary team recognise and respond to adults with AKI within primary care or community care settings. Nurses working in these areas must recognise any AKI risk factors and/or clinical features instigating earlier review including fluid intake and urinary output. Are there any indications of hyperkalaemia particularly if moderate or severe? What about reduced immunity, advanced stages of CKD, history of kidney transplant or previous AKI? Is there frailty with comorbidities such as CKD, diabetes, heart failure, liver disease, neurological, cognitive impairments? These should all be reviewed. There are four principal areas here i.e., 'Think' cause, 'Think' medication, 'Think' fluids, 'Think' review. [18, 21].

The 'Think' cause addresses the following questions which must be deliberated and acted upon such as, is there a history of acute illness? Nurses need to 'Think' sepsis, 'Think' hypotension; is intrinsic AKI suspected such as vasculitis? 'Think' urinalysis; are there any signs of urinary obstruction? [18, 21]. 'Think' medications encompasses any medication that could aggravate AKI. Nurses and other members of the interdisciplinary team must be mindful of any types of medications that could accrue resulting in harm throughout AKI and/or any new medications that could cause AKI such as drug-induced AKI. There are an extensive range of common medications including NSAIDs, cardiovascular medications including antihypertensives, diuretics, digoxin, and statins (if rhabdomyolysis depicted) and some antibiotics including trimethoprim and co-trimoxazole which all need to be assessed in terms of withholding, avoiding, stopping, or reducing dosages as clinically assessed and deemed appropriate on an individual basis. [18, 21].

The concept of 'Think' fluids revolves around individual's volume status and do they have hypovolaemia? When was the last time the individual urinated? Can they increase fluid intake or is there a necessity for hospital admission for monitoring and intravenous (IV) fluid replacement? Finally, another relevant aspect here is does the individual have and/or require carer support? 'Think' review is the final principal area and addresses does the individual require acute admission? If not when will the nurse and other members of the interdisciplinary care team review? Is there a thorough handover to colleagues regarding this individual? [18, 21].

Clinical investigations of AKI

A basic metabolic profile should be conducted with creatinine used for AKI diagnosis in conjunction with the potential risk factors for AKI**.** There is a necessity to ensure that test results are regarded with a recognition of the clinical indicators in which they were derived. The importance of effective communication in terms of a thorough handover, medical records, access to significant clinical information when deriving blood tests using relevant laboratory forms can assist supporting a timely and suitable response to a test result. It is vital to distinguish AKI from a progression of CKD at initial clinical presentation. This can be challenging if there are no current comparison creatinine values. The

clinical background will assist whether an increase in serum creatinine has been sudden or transpired over a longer timeframe. False positives can occur with rises in serum creatinine such as pregnancy wherein an increase in serum creatinine may be a false positive following recent delivery; usually serum creatinine decreases throughout pregnancy. [18, 20, 25, 37].

Daily monitoring of bloods tests including urea and electrolytes are vital for individuals in hospital care settings. This is until the AKI resolves as even moderately changes in sCr levels are related to a substantial increase in mortality. The staging of AKI is determined based on sCr or urinary output whichever one provides the higher stage. Liver function tests (LFTs) will assist in diagnosing hepatorenal syndrome. Whilst careful monitoring of serum potassium is important to determine presence of hyperkalaemia which can be a severe complication of AKI. A full blood count (FBC) could indicate an infection with leukocytosis and an increased or decreased white blood count (WBC) could transpire with sepsis. Anaemia can ensue in AKI consequent to vasculitis, myeloma and/or haemolytic uraemic syndrome. Bicarbonate should be queried if it not a facet of a standard panel with decreased levels indicates metabolic acidosis. Venous blood gases should be requested for future appraisal of metabolic acidosis especially if an initial bicarbonate sample was not included with the initial bloods taken which indicated AKI. Venous blood gases can also evaluate acid/ base status which can occur in AKI because of diminished excretion of non-volatile acids. C-reactive protein should be ordered with all adults as increase levels signify infection and vasculitis. Blood cultures are requested if infection assumed. Sepsis is a widespread cause of AKI so a septic screen should instigate if sepsis deduced using a local care bundle such as sepsis six as appropriate. [18, 20, 25, 37]. Individuals with AKI are more immunocompromised; therefore, can be more at risk to infection so stringent infection control measures using standard personal protective equipment (PPE) as applicable to local policy within hospital care or primary care settings when conducting procedures and other care delivery.

Further specific bloods tests as determined by the diagnosis include serum creatine kinase which would distinctly raise in rhabdomyolysis. Examples of immunological tests including anti-nuclear antibodies (ANA) which is a wide-ranging screening check (can be normal or elevated) for variety of autoimmune diseases such as kidney dysfunction within SLE. The anti-glomerular basement membrane (anti-GBM) antibody can be raised for example with anti-GBM disease or Goodpasture disease. The therapies for which include immunomodulating drugs, corticosteroids, cytotoxic agents and/or plasma exchange. Whilst anti-neutrophil cytoplasmic antibodies (ANCA) can also be used to diagnose anti-GBM disease as well as ANCA associated vasculitis. Human immunodeficiency virus (HIV) serology is applicable concerning HIV-associated nephropathy along with nephrotoxicity of some of the drugs used to treat HIV (positive or negative). The acute hepatitis profile is used to ascertain positive serology in active Hepatitis B or C. This relates to kidney conditions, serum electrophoresis (paraprotein highlighted) and urine electrophoresis (Bence Jones protein established) with myeloma. Myeloma is a significant cause of AKI, and management includes use of corticosteroids, cytotoxic agents, plasma exchanges and KRT as required. [18, 20, 25, 37].

There are various urinary investigations that are required as soon as AKI is speculated or identified, including a urine dipstick to test for specific gravity, protein, blood, nitrites, glucose, and leucocytes. Urine PCR is conducted if protein present on urinalysis. Intrinsic AKI may be a potential cause if urinalysis is positive with proteinuria and haematuria with the lack of any apparent different cause such as injury/ insult from urinary catheterisation or a UTI. The presence of proteinuria and haematuria may suggest an active urine sediment because of glomerular disease. Individuals who present with glomerular disease have symptoms of proteinuria, haematuria, oedema, and hypertension and require timely referral to nephrology. Infection, myoglobinuria (rhabdomyolysis) and calculi are some further examples of other causes of an active urinary sediment. A urine culture is obtained if there is a presence of a UTI with urinalysis for blood, protein, or leukocytes or the individual concerned has clinical presentation of same. [18, 20, 25, 37].

The monitoring of urinary output is paramount when caring for individuals with AKI; this can be hourly if the individual has a urinary catheter (u/c) in situ, 4-hourly if they do not. However, the accuracy of urinary output is challenging if the individual is not catheterised. A diagnosis of AKI is confirmed if urine output <0.5 ml/kg/hour for at least 6 consecutive hours. There must be a careful risk assessment for using a u/c in individuals with AKI as they are not routine standard in care delivery. The pros and cons of u/c must be assessed on an individual basis. The potential benefits include urine output is difficult to quantify without using a u/c and a continual decrease in urinary output is a diagnostic indicator for verifying AKI. Hourly urinary output monitoring assists the ongoing evaluation of the individuals' outcome to their treatment. The use of u/c can assist with identification and treatment for bladder neck blockage. Should a large volume of urine discharge following u/c insertion, this does direct to an obstruction of the bladder opening. Whilst if there is only minimal residual urine following u/c insertion indicates an advanced level urinary tract obstruction or non-obstructive basis of AKI. However, the cons for a u/c include possibilities of infection, trauma, and risk of falls. The use of u/c is denoted where it is vital to manage fluid balance, wherein the individual concerned is excessively unwell to utilize a bottle or commode, if bladder neck blockage is deduced and this cannot be rapidly established via ultrasound. [18, 20, 25, 37]. Nurses must ensure strict infection control measures with catheter care using PPE, in accordance with the hospital's PPE guidance. They should monitor for any potential signs and symptoms of a UTI. This also includes educating the individual if feasible concerning rationale for using a urinary catheter and potential length of time it may be used to monitor urinary output.

Further investigations include PCR testing for Coronavirus Disease 2019 (COVID-19) and appropriate measures taken if positive in accordance with local hospital protocols. The use of a chest x-ray could demonstrate signs of infection, (fluid) pulmonary oedema, cardiomegaly, or haemorrhage such as Goodpasture's disease with pulmonary haemorrhage or ANCA-associated vasculitis. An abdominal x-ray is conducted if renal calculi is speculated. An ECG would highlight any associated changes with hyperkalaemia. The individual will have an ultrasound if pyonephrosis is inferred; if required they may need a nephrostomy within a 6-hour window owing to the threat of septic shock. The use of a renal tract ultrasound has high sensitivity usually conducted within 24 hours. It is not typically required and is only conducted if there is no apparent cause of AKI or should there be any urinary or bladder obstruction, pyelonephritis or pyonephrosis. The ultrasound may need to be repeated post 24 hours for example if there is high suggestive indicator for hydronephrosis. AKI can also distinguish from CKD occasionally using an ultrasound which can highlight small kidneys (occasionally scarred) coherent with advanced CKD. This is helpful if there are no previous creatinine levels to provide a baseline. An abdominal CT or MRI scan would be required if an ultrasound demonstrated an obstruction to highlight a mass or stone imagery. A cystoscopy may be needed to determine cause of obstruction such as bladder tumour or ureteric stenosis. The kidney biopsy may be needed to further establish the cause of AKI including types of glomerular diseases following positive serological studies. [18, 20, 22, 25, 37].

Initial management of AKI

Once diagnosed the management of AKI is mainly supportive. The Royal College of Physicians in the UK recommends the application of the STOP (sepsis, toxins, optimize blood pressure and prevent harm) AKI acronym as a reminder for health care professionals to elicit the urgent steps required for management of AKI. A local care bundle such as sepsis six (surviving sepsis campaign bundles)/ 'Think sepsis' for example should be executed within 1 hour if sepsis is deduced or verified. The sepsis screen should establish source of infection and treat same. The use of nephrotoxins should be stopped or avoided such as NSAIDs, iodinated contrast agents, aminoglycoside, and antibiotics. Both volume status and blood pressure must be optimized through the continual assessment of same. An immediate IV bolus of crystalloid should be administered if the patient is hypovolaemic. A balanced crystalloid (for example Hartmann's solution or ringer's acetate) should be selected except if hyperkalaemia is established or questioned then normal saline would be used. The use of ACEi or

angiotensin-11 receptor antagonists should be withheld as these can aggravate AKI. The use of diuretics and other antihypertensive medications should also be withheld. If the individual concerned continues to be hypotensive regardless of acceptable volume resuscitation. There should be an escalation to critical care for consideration of vasopressors. The prevention of any harm to the individual is paramount with detecting and managing revocable causes such as the relief of any urinary tract blockage, managing severe acute complications such as hyperkalaemia and metabolic acidosis. All medications must be reviewed, and dosages modified as required in accordance with the extent of kidney injury. [2, 4, 20, 25, 26, 36]. KRT commenced when these methods fail to deliver a sufficient and safe regulation of homeostasis. [26].

'Think Kidneys' national program in England recommend the use of care bundles for AKI within hospital care settings [40]. The UK Kidney Association clinical practice guideline on AKI identify the importance of a personalised approach to detect individuals at risk of AKI based on the subsequent questions. (1) what risk factors for AKI does this individual have? (2) can the healthcare team reduce this risk? (3) can the applicable healthcare team avert adding to this risk? (4) is the healthcare team able to identify AKI as early as feasible through frequent monitoring of urinary output and kidney blood tests. [2].

The 5**R**s approach in managing AKI is another algorithm that could be used to assist nurses and other members of the interdisciplinary team both in the primary care and hospital care settings. This 5**R**s approach (risk, recognition, response, renal support, and rehabilitation) is comparable to the 5R framework for sustainable basis to maintain AKI care already outlined (Figure 1). [5-6]. Individuals at risk of AKI should avoid episodes of dehydration and exposure to nephrotoxins. Recognition of AKI is contingent upon reliable clinical judgement, vigilant evaluation of urinary output and serum creatinine. Rapid response to AKI involves the STOP AKI acronym already highlighted. [38]. Individuals with severe AKI should be referred and access renal support. Finally, those adults with AKI or at risk and their partners/ family members should obtain applicable rehabilitation including information concerning the risk factors, preventative methods, therapy options and potential outcomes. [41].

Ongoing management of AKI

Volume status

It is vital to conduct an initial assessment of the adult with AKI with swift adjustment of volume depletion or volume overload particularly if related with deteriorating cardiac output. This can overturn or improve AKI. Meticulous management of fluid balance is necessary as both hypovolaemia and volume overload are related with adversative outcomes. Restoring kidney perfusion is the main factor to enhance the haemodynamic state of the individual concerned which pre-kidney AKI which is frequently a result of hypovolaemia and/or hypotension. Signs and symptoms of hypovolaemia to observe for include daily weights, capillary refill time, NEWS2 scoring, postural blood pressure, jugular venous pressure, skin turgor and dry axillae/ mucous membranes. All input and output should be recorded on fluid balance chart with early nutritional assessment (using a standardized nutritional tool) instigated. Blood tests conducted include urea and electrolytes to include bicarbonate, FBC, LFTs, bone profile including phosphate. Signs and symptoms of volume overload are less frequent at initial presentation and include respiratory rate with tachypnoea implies fluid overload and/or acidosis, peripheral and sacral oedema. The presence of crackles on auscultation of lungs suggests pulmonary oedema. [2, 10-11, 20, 25, 37, 39].

Fluid resuscitation for hypovolaemia involves administering a 500 mL IV bolus (preferably crystalloid fluid) of fluid over 15 minutes, with the use of a balanced crystalloid except hyperkalaemia is speculated or identified, normal saline (0.9% sodium chloride) should be used if hyperkalemia is confirmed. The haemodynamic status must be reassessed following the initial fluid bolus and judge whether further 250-500 mL boluses are needed as well as the individual's response to each fluid challenge using ABCDE approach, NEWS2, clinical assessments including urinary output. The care must

be escalated if there is no improvement following two fluid challenges with necessity for critical care support. It is vital to recognise when to decrease IV fluid therapy when the individual concerned is euvolaemic and haemodynamic stability reestablished. Should this not be conducted volume overload could occur hastening pulmonary oedema. This is more adversative from over-aggressive or overzealous fluid resuscitation if the individual has a history of heart failure or is oliguric/ anuric. The use of IVs must have a clear intention with a clear rationale for administering same. [2, 10, 11, 20, 25, 37, 39].

The concept of volume overload can occur in AKI due to oliguria in intrinsic or post-kidney AKI along with overzealous fluid resuscitation which is frequently found in individuals with sepsis who originally exhibited with hypovolaemic pre-kidney AKI. There is a necessity again for meticulous monitoring to ameliorate any adversative outcome such as pulmonary oedema. Volume overload may be managed by sodium restriction and prudent use of a loop diuretic such as furosemide under consultation with the nephrology team with evaluation of urinary output with said medication ceased if there is no success. Urgent KRT is specified for intractable volume overload or volume overload again following discussions with nephrology team and if there is a poor response of the loop diuretic usage for volume overload. [2, 11, 20, 25, 37, 39]. Nurses must deliver ongoing care using NEWS2, addressing signs and symptoms of volume overload, administering oxygen if and as prescribed and frequent monitoring of skin integrity including mouth care. The care delivery should be explained taking on board there may be altered mental status because of AKI such as confusion, lack of concentration and delirium. It is important therefore to reassure and provide emotional support to individuals with AKI and their partners/ family members as this can be a distressing and difficult time for all involved.

Medication review

Any medication that lowers blood pressure, circulating volume, or kidney blood flow will enhance the risk of AKI. All medications and dosages must be reviewed particularly if there are nephrotoxic medications prescribed when AKI is inferred or verified. These nephrotoxic medications must be stopped or avoided as they could impact on kidney function. There are numerous common medications that are frequently prescribed including NSAIDs, iodinated contrast agents, aminoglycoside antibiotics, ACEi, angiotensin-II receptor antagonists, diuretics, or other antihypertensive medications. Specialist pharmacist advice should be sought to reduce negative impacts (such as dose amendment, maintain a short course of therapy as possible, evaluate blood levels of the medication if possible) particularly if there are prevailing rationales to maintain the usage of a potential harmful medication. Before the individual concerned is discharged all their medications must be reviewed with a plan created to reestablish any medications that have been suspended at a suitable time with re-titration dosage as applicable in primary care or community care settings. Medications for pre-existing heart failure would be re-commenced as soon as clinically practical and optimum control of both fluid balance and blood pressure achieved through re-titration. A necessary part of the discharge planning would be ensuring procedure prepared for monitoring of serum creatinine and potassium 1 to 2 weeks post discharge. Individuals with AKI and nurses along with other members of the interdisciplinary team require suitable and effective education as to the capacity for kidney injury and dysfunction from nephrotoxic agents. [2, 18, 20, 39, 42].

Pharmacological management

There is currently no evidence to provide the use of a particular pharmacological therapy in the management treatment of AKI secondary to hypoperfusion injury and/or sepsis. There is also no evidence that using loop diuretics minimises the prevalence or severity of AKI. Loop diuretics should not administer routinely to treat AKI. They may be contemplated for treating fluid overload or oedema whilst an adult is awaiting KRT or if kidney function is improving in an adult not receiving KRT. Loop diuretics are used with adults in oliguric AKI, to expediate the management of fluid and electrolyte disturbances and minimise the necessity for KRT. Diuretics are also used to control fluid balance or oedema and allow administration of nutrition and medications. However, diuretics can also be

harmful, by unduly minimising the circulating volume and adjoining a prerenal insult, deteriorating established AKI. It is vital to appraise efficacy of diuretics to enhance outcome of adults with AKI, not solely for fluid management. It has been suggested that dopamine may possibly minimise ischaemic cell injury in adults with AKI by enhancing renal blood flow and decreasing oxygen consumption by means of inhibition of sodium transport. There have been numerous studies examining the use of dopamine in the avoidance and management of AKI which identified that there is no good evidence to promote any significant clinical advantages to adults with or at risk of AKI. Regardless of the lack of supportive data loop diuretics and dopamine are still often used inappropriately to try to determine and sustain a urinary output. [2, 3, 10, 25, 37].

Nutritional support

There are several areas that need to take into consideration when addressing nutritional support for individuals with AKI including exact metabolic disturbances allied with the kidney injury, the primary disease process, the probable nutritional deficits, and benefits during KRT, fluid retention and preservation of uraemic toxins and inflammatory markers. The overall purpose of nutritional support is to sustain nutritional status while curbing the complications of AKI. This comprises averting or reducing malnutrition especially protein energy wasting (PEW), maintaining lean body mass, preventing extra metabolic imbalances, enhancing wound healing, promoting immune function, and thereby decreasing risk of mortality. Currently there is no endorsed screening tool to detect malnutrition in adults with AKI plus they are frequently not suitable in the critical care setting. There should be an individual assessment conducted by a dietician specifically if the individual is at high risk of malnutrition and if they are receiving KRT as a treatment for AKI. [2, 43].

The dietetic classification of AKI is separated into adults with AKI in the non-catabolic state and those adults with AKI in the catabolic state. Usually, adults that are stable with pre-kidney (such as dehydration, certain medications) or post kidney AKI (such as urinary blockage) depict in a non-catabolic state. Should KRT be required it is frequently supported through standard haemodialysis (HD). These individuals primarily with stage 1 and 2 AKI can manage alone with an oral diet and as necessary, the addendum of nutritionally dense supplementary sip feeds will regularly suffice to match their needs. Should this not be the case artificial nutrition support should be instigated. Usually, individuals with intrinsic AKI (such as sepsis, trauma, and acidosis) depict in a catabolic state. They may have any stages of AKI although mainly stages 2 and 3 AKI and are prone to be treated in ICU. PEW can be a common outcome in this cohort resulting in protracted hospital stays and adversative outcomes including mortality rates. The use of artificial nutritional support ideally via the enteral route is regularly required especially if the individual concerned is intubated and sedated. Nutritionally dense feeds with or without decreased electrolyte substance are beneficial where the regulation of fluid balance and/or serum phosphate and potassium levels demonstrate challenges. There needs to be a cautious approach for adults with AKI in ICU as there can be reduced tolerance for nutritionally dense feeds. [2, 43].

Management complications of AKI

Hyperkalaemia

This is a widespread complication of AKI. The management of hyperkalaemia is reliant on the gravity and existence of muscular weakness and/or cardiac complications such as cardiac arrhythmias. Any aggravating aspects should be adjusted such as treating the AKI, suspending culprit medications such as ACEi, potassium sparing diuretics, angiotensin-II receptor antagonists and limit dietary intake of potassium type foods and fluids. [20].

The treatment of hyperkalaemia in hospital should follow a 5-step approach according to the UKKA 2020 clinical practice guidelines on acute hyperkalemia in adults. Step 1 protects the heart and advocates that IV calcium chloride or calcium gluconate at an equal dose is administered to the adult with hyperkalaemia in the existence of ECG indication of hyperkalaemia. Step 2 is shifting potassium

(K+) into cells using an insulin-glucose infusion wherein 10 units soluble insulin in 25g glucose is administered by IV infusion to treat severe hyperkalaemia (K+ ≥ 6.5 mmol/l) and moderate hyperkalaemia (K+ 6.0 – 6.4 mmol/l). It is vital in step 2 to avoid hypoglycaemia by preventively initiating an infusion of 10% glucose at 50ml/ hour for 5 hours after insulin-glucose therapy in adults with a pre-therapy blood glucose < 7.0 mmol/l (target blood glucose 4-7 mmol/l). Step 2 continues with shifting K+ into cells using salbutamol (10-20mg) as adjuvant therapy for moderate and severe hyperkalaemia. The use of IV sodium bicarbonate is not usually administered for the acute therapy of hyperkalaemia within step 2. Step 3 progresses with the use of potassium binders and removal of K+ from the body. This step recommends that sodium zirconium cyclosilicate is applied in the emergency therapy of acute life-threatening hyperkalaemia (serum K+ ≥ 6.5 mmol/l) whilst patiromer is suggested as an option to also treat this level of hyperkalaemia. Calcium resonium should not be used in the emergency management of severe hyperkalaemia but could be considered in adults with moderate hyperkalaemia. [44].

It is recommended within step 4 that serum K+ is closely monitored in all adults with moderate or severe hyperkalaemia (at least 1, 2, 4, 6 and 24 hours) to evaluate efficacy of therapy and to observe for rebound hyperkalaemia. Blood glucose concentration should also be monitored regularly for example 0, 15mins, 30mins, 60mins, 90mins, 120mins etc. up to 12 hours post administration of insulin-glucose infusion in all adults with hyperkalaemia. Individuals who are receiving HD with severe hyperkalaemia obtain urgent dialysis therapy. Should they have toxic ECG changes IV calcium salts should be administered to minimise the risk of arrhythmias even if dialysis therapy is instantly accessible. Should dialysis not be instantly accessible then standard medical therapies be used to reduce serum potassium (in those again with severe hyperkalaemia). The use of potassium binders may be contemplated throughout the interdialytic timeframe. Specialist referral to local renal or critical care team for an urgent opinion for adults with severe hyperkalaemia and potential escalation of care, if necessary, from the start or if the individual concerned fails to react to early therapy. [44].

Metabolic acidosis (pH <7.25)

This is a frequent metabolic intrusion in AKI. It transpires mainly due to reduced excretion of the usual load of metabolic acid in the context of a reduced GFR. Further circumstances that may also influence is enhanced creation of lactic acid in individuals with sepsis. It is important to recognise there will be comparative challenge to vasopressors in the occurrence of severe metabolic acidosis. IV sodium bicarbonate is a guide for use in severe metabolic acidosis (pH <7.2). However, it is important to access expert support beforehand and throughout this process as it can result in volume overload and/ or hypernatraemia. There should be consideration made to refer to ICU. IV sodium bicarbonate should only be administered if there are no signs of volume overload and if venous bicarbonate is <16 mmol/L. Ionised Ca^{2+} is slashed following swift rectification of metabolic acidosis with IV sodium bicarbonate. However, this can initiate tetany, seizures, and cardiac volatility. Before administering IV sodium bicarbonate the low ionised Ca^{2+} must be amended through a distinct IV route because of the inconsistency of bicarbonate and calcium suspensions. The individual concerned would be referred for KRT if there is no response to initial therapy with refractory acidosis (pH <7.15) or they have severe acidosis in the context of volume overload. [2, 20, 37, 39].

Pulmonary oedema

This could occur with overzealous IV fluid resuscitation in individuals who depicted hypovolaemic pre-kidney AKI. It may also ensue in some forms of AKI such as cardiac failure with AKI and renal tract obstruction with salt and water retention. Acute pulmonary oedema is an emergency management with high mortality rates the care delivery is therefore vital. The individual concerned must be sat straight in the bed and administered high-flow oxygen (15 L/ minute through a reservoir mask) and continuous positive airway pressure ventilation should be considered if accessible. IV glyceryl trinitrate should be administered with the dosage titrated upwards, planning to sustain systolic blood pressure (SBP) >95mmHg. A loop diuretic could be considered if the individual is haemodynamically

secure. This must be again conducted under specialist supervision with also senior support obtained. The warning for emergency KRT is refractory pulmonary oedema as the application of an IV-glyceryl trinitrate and a loop diuretic like furosemide could be a helpful supporting measure. However, this should not suspend advancing to decisive management with kidney support if required. [2, 25, 37, 39]. The use again of ABCDE and NEWS2 is vital here, monitoring of bloods including venous blood gases, deep breathing and coughing exercises if tolerated as well as ongoing emotional support for the individual experiencing this serious distressing complication of AKI.

Indications for kidney replacement therapy

The decision whether to commence continuous or intermittent KRT should be based on clinical presentation of the individual concerned. These extracorporeal modalities should regard as complementary therapies for AKI. The selection of KRT should be directed by clinical condition, expertise of nurses and medical team along with the accessibility of machines. [2]. The indications for dialysis in AKI are well recognised and are based primarily on the progression of refractory to diuretics, life-endangering fluid overload and critical solute imbalances attended by hyperkalaemia and metabolic acidosis. [26]. Those individuals who are haemodynamically unstable or have cerebral oedema or acute brain injury should ideally provide with continuous KRT. [2]. However, the decision as to when to commence KRT in critically ill individuals with AKI is frequently much more challenging in the absence of these vital indicators. During the last decades, this area remains contentious despite a considerable progress in intensive care medicine. It is important to recognise that while KRT is unavoidable and lifesaving in many incidences of severe AKI, it can also adversely influence the outcomes of individuals experiencing same. The process of dialysis subjects the individuals or exacerbates the previously present haemodynamic instability with detrimental intradialytic hypotension, anticoagulation-induced bleeding, vascular-access-associated bacteraemia, and inflammatory stress due to extracorporeal circuit bio-incompatibility. One of the main problematic impacts of KRT is a not completely foreseeable change in the pharmacokinetics of many therapeutic agents particularly antibiotics which presents a fundamental threat of underdosing them. [26].

The discontinuation of KRT established on one of three clinical developments: intrinsic kidney function has sufficiently recovered to sustain stresses, the condition that induced renal support has improved, or ongoing KRT is no longer coherent with goals of care. [3]. The UKKA clinical guideline on AKI also highlight that progression in the individual's clinical condition, improvement in fluid status and urinary output would warrant temporary suspension of ongoing renal support to investigate if AKI is improving. [2].

COVID-19 Associated Acute Kidney Injury (C19-AKI)

COVID-19 originated in Wuhan, China in December 2019 and is an infectious disease instigated by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). [45]. COVID-19 associated AKI (C19-AKI) is multifactorial in origin and may require admittance to ICU and the ensuing use of KRT. [26] AKI is more widespread amid adults hospitalised with COVID-19 than in adults hospitalised because of other illness. [46]. The demographic risk factors for C19-AKI are like those for individuals developing AKI from other routine causes including those over 65 years of age, CKD, diabetes mellitus, obesity, smoking, hypertension, cardiovascular disease, and adults who are already immunocompromised. Individuals with COVID-19 may present with AKI or acquire same throughout their hospitalisation with those needing admission to ICU have an increased rate of AKI as compared to those that are hospitalised on the wards. A recent systematic review highlighted that AKI commonly complicates the path of COVID-19 hospitalisations and is coupled with enhanced severity of illness, protracted period of hospitalisation, and a weak prognosis. [47]. There is a high mortality rate for adults who develop C19-AKI. It is therefore vital that there is early detection and response system in position. [2]. There is also a necessity for prompt detection of comorbidities and renal complications to enhance the outcomes of individuals with COVID-19 in view of the adverse influence of AKI. There is a requirement

for large scale research to enhance comprehension of this disease and devise clinical methods to manage C19-AKI. [47].

It was highlighted that many published reports of individuals with COVID-19 do not incorporate definitions or staging of AKI, or data concerning renal recovery or follow up. The difference between de novo AKI and AKI superimposed on pre-existing CKD is also seldom designated. The reported hospital admission rates of individuals with COVID-19 differs amid and within countries revealing diverse national and regional healthcare structures, procedures for hospitalisation and for allocating stages of care for example ICU admission. The comparisons of AKI rates centered exclusively on the number of hospitalised persons are all complicated by these aforementioned factors. [46].

The pathophysiology and processes of C19-AKI have not fully interpreted. However, it does appear to be similar with the pathophysiology of AKI in other individuals who are acutely ill [46]. The more apparent concepts are hypovolaemia, haemodynamic changes, direct cellular invasion, acute respiratory distress syndrome (ARDS) and cytokine eruption. It identified that persons with C19-AKI in their histopathological reports exhibited ATN occasionally with necrosis and failing glomerulopathy or rhabdomyolysis. It may also ally with abnormal serum electrolyte levels, proteinuria, and haematuria. [45, 48].

The management for COVID-19 and management of pre-existing conditions may all augment the threat of AKI. In individuals with COVID-19 fever and enhanced respiratory rate increase insensible fluid loss [49], highlighting further challenges in the management process. It is critical for minimising the incidence of AKI in people with COVID-19 to maintain optimal fluid status (euvolaemia) with relevant fluid resuscitation as hypovolaemia will aggravate the hypercoagulable status and organ injury encompassing C19-AKI. [49]. The emergency management of serious life-threatening hyperkalaemia should address as per local hospital guidelines and can include the use of potassium binders such as patiromer if designated. There is a necessity for a complete medication review with drug dose adjustments as required. There is a need for malnutrition screening and assessment for individuals including older adults and polymorbid individuals who are at risk of adverse outcomes and increased mortality following infection with COVID-19. Members of the interdisciplinary team should be mindful of the consequences of choice of continuous and intermittent KRT on fluid balance and pharmacokinetics. The outcome to commence KRT for AKI in COVID-19 should not be based merely on the AKI stage or kidney function but on the specific assessment of the individual concerned. KRT modality will be determined upon local accessibility, preparation, and proficiency of the interdisciplinary team. There is no available high-quality evidence recommending one acute KRT modality over another modality choice. [49].

Sepsis-Associated AKI

Sepsis-Associated AKI (SA-AKI) or sepsis-induced AKI accounts for 50% of critically ill persons in the ICU with morbidity and mortality rate high. The causes of SA-AKI are sepsis and septic shock. The initial identification and aggressive sepsis management are vital in enhancing patient outcomes and limiting the impact that sepsis can have on kidney function. The main therapy for SA-AKI is the administration of suitable antibiotics, appropriate fluid therapy, vasopressors such as norepinephrine which has been established to increase mean arterial pressure (MAP) and enhance perfusion to the kidneys, avoidance of nephrotoxic medications and the consideration of KRT as required. Nurses are usually educated to 'Think sepsis' when minor changes occur in patients with potential infection. They are frequently the frontline personnel in combating sepsis and occupy more time with individuals who are hospitalised than any other members of the interdisciplinary team. They are therefore in leading positions for early sepsis recognition. The importance of early identification including signs, and symptoms of sepsis along with standard screening protocols must be incorporated within nursing education and ongoing study days. The importance of identifying individuals within the primary or community care settings must also not be underestimated in terms of accessing assistance in a timely manner especially if there

is already some degree of kidney function. A recent systematic review identified that long-term consequences relevant to SA-AKI differ from absolute recovery to reliance on KRT with the need for uniform definitions for AKI and a necessity to highlight long-term impact to make evaluations across studies. [50-51].

Contrast-induced AKI

This was previously referred to as contrast induced nephropathy (CIN) wherein AKI developed after iodinated contrast was administered. The KDIGO international guidelines recommends the term contrast induced AKI (CI-AKI) using the KDIGO AKI definition. CI-AKI is unusual within the general population with a prevalence of only 1-2%. It usually transpires 24-48 hours of receiving iodinated contrast media and the individual concerned typically improves over the subsequent 5 days. However, the prevalence of CI-AKI is enhanced considerably in individuals with risk factors including older age, CKD, diabetic kidney disease, cardiac failure, sepsis, volume depleted states, and exposure to nephrotoxic medications. It is related to protracted hospital stays, compounded mortality, and escalated health care costs [51]. The pathophysiological basis of CI-AKI is based on the interaction of cytotoxicity and viscosity. There is no treatment for CI-AKI, prevention therefore is the best management strategy. Hydration levels should be assessed for all individuals undergoing iodine-based contrast media with safety. This is especially pertinent for those individuals at high risk of CI-AKI as an incorrect fluid balance (oral or IV) could be detrimental leading to fluid overload and cardiovascular events. Individuals in hospital care settings should be supported to drink oral fluids prior to and post when provided with a contrast agent. However, those at exceptionally elevated risk should obtain IV fluids for volume expansion when undergoing a contrast agent. As a result of a lack of evidence the administration of N-Acetylcysteine is no longer recommended although the decision for this remains with the referring medical team. There has been a diversity of other pharmacotherapies considered for individuals with CI-AKI prevention, however none are identified as noticeably beneficial. [3, 11, 52-53].

Education for individuals with AKI

It is well documented individuals with severe AKI have weaker outcomes should they not receive suitable follow up reviews including medication reviews post discharge. A service evaluation was conducted with individuals following successful implementation of a nurse-led AKI follow up clinic in the UK. Adults here were requested to complete a questionnaire to determine their perceived knowledge concerning AKI pre and after attendance at an AKI-nurse led clinic appointment. The results were overall positive and identified that individuals were more knowledgeable and deemed relatively confident in avoiding future AKI. [54].

In another recent study conducted in the USA, several individuals who developed AKI and since discharged were interviewed on their experiences and views on the care they obtained whilst in the hospital care setting. Many had underlying kidney disease and over half of the individuals interviewed had stage 3 or severe AKI. The study highlighted that despite seen by the inpatient nephrology services that most AKI ‘survivors ‘(as they were called within this study) were uninformed of their AKI and possessed limited information as to the cause. It identified that only a few individuals were educated on how to care for their AKI at home when discharged, while others wanted necessary information concerning self-management of their illness. There were issues about the use of medical jargon and terminology and confusion concerning for example the term ‘injury‘with some relating same to being ‘punctured‘ or ‘hit ‘. This study demonstrated that there is a definitive need to engage individuals in their care delivery. The use of shared and information-decision making and the use of individual empowerment throughout their hospital stay and beyond is vital when discharged into primary care settings. It is crucial to individual recovery for shared decision-making and communication among care partners, the individual concerned and family members. There remains the necessity for close monitoring on discharge from hospital possibly to rehabilitation facility as the individual recuperates

from critical illness and AKI. There is a need for interdisciplinary recovery-concentrated care to ensure sufficient complete recovery to a previous condition of health and well-being. [43, 55-56].

Emotional support for individuals with AKI

Individuals with AKI should be the core of every part of communication and care. All decisions concerning treatment whenever feasible should be communicated with individuals concerned and their partners/ families and if necessary, every member at the end-of-life care interdisciplinary team. Every communication should be delivered in simple lay language at frequent intervals, with the recognition that individuals with AKI may be traumatised. [42]. This can remain a time of great uncertainty for all involved with individuals describing themselves as anxious, stunned, shocked, and stressed concerning future damage to their kidneys and potential dialysis related pain [56]. These emotions are also common with individuals living with CKD. [57], similarly the individual with AKI must be provided with time to discuss their acute illness with assistance by nurses using open-ended questions and listening skills taking on board their clinical presentation such as uraemia, concentration levels, fatigue, and nausea. Partners/ family members should be included with potential referral by nurses for further emotional support as required including the use of kidney support groups. [13, 58].

Education for nurses focused on individuals who suffer from AKI

The importance of education for nurses and other member of the interdisciplinary team is paramount concerning AKI. This highlighted in a recent study of non-renal nurses who completed an AKI knowledge and confidence scale. The results indicated that these nurses seemed knowledgeable and confident in escalating and managing AKI. There were however a lack of knowledge and confidence in the significance of atypical physiological parameters and urinalysis and serum biochemistry. This variance in levels of knowledge and confidence in detecting and preventing AKI could infer ineffective management. It is obvious that for AKI detection, deterrence, and management on general wards within hospital care settings to be successful, education must be continual and dispensable within standard workloads. [59]. The role of the AKI specialist nurse has implemented within various NHS trusts in the UK. It developed to accelerate time from diagnosis to management of AKI, uphold accurate and suitable intervention and minimise the length of hospital stay following AKI diagnosis. Education plays a pivotal role in that of the AKI specialist nurse to not only educate nurses but also other members of the interdisciplinary team with clinical, formal, and informal ad hoc teaching sessions depending upon service needs. [60]. There needs to be educational and ongoing training days for those nurses and other members of the interdisciplinary team working in primary or community care settings including residential or care homes such as detecting and identifying older adults at risk of AKI and instigating appropriate care in an expedient manner.

Prevention of AKI

The best treatment for AKI is 'prevention' with the old phrase already highlighted of 'prevention is better than cure '. A key to primary prevention is that of good kidney health and its strong interlinkage with other comorbidities such as cardiovascular disease and diabetes. This should not be underestimated. Nurses needs to promote a healthy lifestyle including good hydration, healthy eating, smoking cessation, alcohol reduction and use of physical activity. Any potential complications such as reduced urinary output and/ or gastrointestinal symptoms should be observed and reported. It is imperative that individuals concerned, or their partners/ family members know to report any concerns/ worries initially to their primary or community care teams including their GP in a timely manner. [2, 20, 58].

The use of medications that are nephrotoxic to the kidneys should be avoided. Should a nephrotoxic drug become essential, continual evaluation is required to balance any potential damage to the kidneys such as examining therapeutic levels, reducing the duration and dosage of the applicable drug and reviewing concomitant drug administration to detect any interfaces from a nephrotoxic position. The use of recreational drugs and over the counter herbal medications should be avoided. Individuals

should be educated concerning signs and symptoms of UTIs including good personal hygiene, good hydration and again seeking further advice when concerned. Kidney, urinary and bladder stones could potentially cause post-renal AKI as already outlined. Nurses need to educate individuals who can be at risk including the importance of good kidney health. Other primary preventative measures include prevention of CI-AKI through use primarily of oral hydration especially if the individual concerned are more increased risk of CI-AKI. The prevention of perioperative AKI highlights individuals' risk factors preceding surgery such as sepsis, hypovolaemia, CKD, and individuals over ≥65 years. [2, 20, 58].

The use of secondary prevention includes nurses advising individuals on potential risk factors of AKI including what measures to undertake to avoid a further incident including involving partners/ family members as applicable. The monitoring and prognosis should be discussed. This should include immediate and long-term therapy options and promotion of self-management and support in cooperation with interdisciplinary members applicable to individuals' distinct requirements. Post discharge detailed information is necessary for those individuals who require KRT involving what planning may be required such as modality choice, amount and length of dialysis sessions, type of vascular access. Further education and awareness for the individual and their partners/ family members and/ or health care workers in residential or care homes as appropriate concerning the risk of developing future AKI specifically for those with pre-existing CKD, and as already highlighted the avoidance of dehydration and use of medications that can cause or deteriorate kidney injury. [2, 20, 58].

Conclusion

This chapter addressed AKI and the main principles of nursing care for individuals presenting with AKI. This included the relevance of early detection and identification of AKI throughout primary (community) and secondary care (hospital care) settings. It highlighted the stages of AKI and the pivotal role nurses play throughout the AKI journey for the individual concerned as safety is paramount in this often-critical care delivery. It highlighted the relevance of education and emotional support for individuals alongside their partners/ family members and the necessity for ongoing monitoring and evaluation of good kidney health and potential risk factors on their discharge. There is a possibility that individuals with AKI may need KRT on a short or longer-term basis. Nurses along with other members of the interdisciplinary team should deliver personalized care, promote self-management and shared decision making with these adults and their partners/ family members. There is a need for ongoing education and support for nurses throughout their applicable healthcare system when it comes to detection and distinguishing AKI in an expedient manner as well as promoting increased awareness of this clinical syndrome. Nurses are valuable members of the interdisciplinary team. They are usually the first point of contact for individuals with AKI. The use of care bundles, best practice guidance, as well as good intuition and clinical judgement should guide and support nurses in their care delivery along with close collaboration with members of the interdisciplinary team so that individuals with AKI will benefit by receiving the best care available and ideally the best outcomes.

Reflective questions
Review the chapter and answer the following questions. Reflect on your own clinical practice area as a nurse and apply your answers to where you work.

Use your clinical judgement as a guide and 'Think Kidneys' best practice guidance to support your answers: 'Think' cause, 'Think' medication, 'Think' fluids, 'Think' review. The 5Rs approach to managing AKI (risk, recognition, response, renal support, and rehabilitation) can also be used to guide your answers.

- Identify the risk factors for AKI with individuals you nurse in your own clinical practice area?
- What are the 3 stages of AKI?
- What are the classifications of AKI?
- Would you see any of these classifications with individuals you nurse in your own clinical practice area?
- Outline the main clinical investigations conducted to assess if an individual has AKI?
- What are the complications of AKI?
- What advice would you give to individuals with AKI on their discharge from hospital care settings into primary care or community care settings?

References

1. National Health Service and United Kingdom Renal Registry. "*Think Kidneys". Communities at Risk of developing Acute Kidney Injury.* 2018a. Available at: https://www.thinkkidneys.nhs.uk/aki/wp-content/uploads/sites/2/2018/11/Nov-18-Communities-at-risk.pdf (accessed 10th September 2022).
2. Kanagasundaram S, Ashley C, Bhojani S, Caldwell A, Ellam T, Kaur A, et al. *Clinical Practice Guidelines. Acute Kidney Injury (AKI).* 2019; UK Kidney Association. Available at: https://ukkidney.org/sites/renal.org/files/FINAL-AKI-Guideline.pdf. (accessed 5th September 2022).
3. Moore PK, Hsu RK, Liu KD. Management of acute kidney injury: Core curriculum 2018. *AJKD*. 2018; 72(1): 136-148. https://doi.org/10.1053/j.ajkd.2017.11.021.
4. Alkhunaizi AM. Acute kidney injury. In: Karker A, editor. *Aspects in Continuous Renal Replacement* Therapy. Vienna: IntechOpen; 2018. p. 5-31. http://dx.doi.org/10.5772/intechopen.80625.
5. Mehta RL, Cerdá J, Burdmann EA, Tonelli M, García-García G, Jha V, et al. International Society of Nephrology's 0by25 initiative for acute kidney injury (zero preventable deaths by 2025): a human rights case for nephrology. *Lancet*. 2015; 385(9987): 2616–43. https://doi.org/10.1016/S0140-6736(15)60126-X.
6. Macedo E, Garcia-Garcia G, Mehta RL, Rocco MV. International Society of Nephrology 0 by 25 Project: Lessons Learned. *Ann Nutr Metab.* 2019; 74 (suppl 3):45–50. https://doi.org/10.1159/000500345.
7. National Confidential Enquiry into Patient Outcome and Death. *Acute Kidney Injury: Adding Insult to Injury.* 2009. Available at: https://www.ncepod.org.uk/2009aki.html (accessed 20th November 2022).
8. Tsang JY, Murray J, Kingdon E, Tomson C, Hallas K, Campbell S, et al. Guidance for post-discharge care following acute kidney injury: an appropriateness ratings evaluation. *BJGP Open.* 2020. https://doi.org/10.3399/bjgopen20X101054.
9. See EJ, Jayasinghe K, Glassford N, et al. Long-term risk of adverse outcomes after acute kidney injury: a systematic review and meta-analysis of cohort studies using consensus definitions of exposure. *Kidney Int*. 2019; 95 (1): 160-172. https://doi.org/10.1016/j.knt.2018.08.036.
10. National Institute for Health and Care Excellence. *Acute Kidney Injury. Prevention, detection, and management up to the point of renal replacement therapy.* 2013; National Clinical Guidance Centre, Clinical guideline [CG 169]. Available at https://www.nice.org.uk/guidance/ng148/evidence/full-guideline-pdf-7019266862 (accessed 10th November 2022).
11. National Institute for Health and Care Excellence. *Acute Kidney Injury: Prevention, Detection and Management.* 2019a; NICE guideline [NG148]. Available at: https://www.nice.org.uk/guidance/ng148. (accessed 12th September 2022).
12. National Institute for Health and Care Excellence. *Acute Kidney Injury.* 2021. Clinical knowledge summaries. Available at https://cks.nice.org.uk/topics/acute-kidney-injury/. (accessed 12th September 2022).
13. Murphy F, Byrne G. The role of the nurse in the management of acute kidney injury. *Br J Nurs.* 2010; 19 (3): 146-152. https://doi-org.elib.tcd.ie/10.12968/bjon.2010.19.3.46534.
14. Byrne G, Murphy F. Acute kidney injury and its impact on the cardiac patient. *Br J Card Nurs.* 2008; 3 (9): 416-422. https://doi-org/10.12968/bjca.2008.3.9.30874.
15. Kellum JA, Romagnani P, Ashuntantang G, Ronco C, Zarbock A, Anders H-J. Acute kidney injury. *Nature Reviews Disease Primer*. 2021; 7: 52. http://doi.org/10.1038/s41572-021-00284-z.
16. Mehta RL, Burdmann EA, Cerdá J, Feehally J, Finkelstein F, García-García G, et al. Recognition and management of acute kidney injury in the International Society of Nephrology 0by25 Global Snapshot: a multinational cross-sectional study. *Lancet.* 2016; 387(10032): 2017–25.

https://doi.org/10.1016/S0140-6736(16)30240-9.
17. Vanholder R, Rondeau E, Anders H-J, Carlson N, Fliser D, Kanbay M, et al. EDTAKI: a nephrology and public policy committee platform call for more European involvement in acute kidney injury. *Nephrol Dial Transplant.* 2021; 1-9. http://doi.org/10.1093/ndt/gfab018
18. National Health Service and United Kingdom Renal Registry. *"Think Kidneys". Acute Kidney Injury Best Practice Guidance: Responding to AKI Warning Stage Test Results for Adults in Primary Care.* 2018a. Available at: https://www.thinkkidneys.nhs.uk/aki/wp-content/uploads/sites/2/2018/11/Nov18-Primary-Care-Best-Practice-Guidance.pdf (accessed 10th September 2022).
19. Silver SA, Chertow GM. The economic consequences of acute kidney injury. *Nephron.* 2017; 137(4):297-301. http://doi.org/10.1159/000475607.
20. BMJ. Acute Kidney Injury. *BMJ Best Practice.* 2020. Available at https://bestpractice.bmj.com/topics/en-gb/3000117/guidelines. (accessed 11th November 2022).
21. National Health Service and United Kingdom Renal Registry. "*Think Kidneys". Communities at Risk of developing Acute Kidney Injury.* 2018b. Available at: https://www.thinkkidneys.nhs.uk/aki/wp-content/uploads/sites/2/2018/11/Nov-18-Communities-at-risk.pdf (accessed 10th September 2022).
22. Willacy H. Acute kidney injury. 2020. Available at https://patient.info/doctor/acute-kidney-injury-pro (accessed 10th November 2022).
23. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P. Acute renal failure-Definition, outcome measures, animal models, fluid therapy and information technology needs: The Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Critical Care.* 2004; 8(4): R204-12. https://doi.org/10.1186/cc2872.
24. Mehta R, Kellum J, Shah S, Molitoris B, Ronco C, Warnock D, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Critical Care*. 2007; 11(2), 31- 39. https://doi.org/10.1186/cc5713.
25. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney Int.* 2012; Suppl. 2: 1–138. https://doi.org.10.1038/kisup.2012.6.
26. Matuszkiewicz-Rowinska J, Malyszko J. Acute kidney injury, its definition, and treatment in adults: guidelines and reality. *Pol Arch Intern Med.* 2020; 130 (12): 1074-1080. http://doi.org.10.20452/pamw.15373.
27. Hosohata K, Inada A, Oyama S, Iwanaga K. Acute kidney injury (AKI). In: Rath T, editor. *Advances in Nephropathy*. Vienna: IntechOpen; 2018. p. 173-190. http://dx.doi.org/10.5772/intechopen.79348.
28. Lamiere NH, Levin A, Kellum JA, Cheung M, Jadoul M, Winkelmayer WC, et al. Harmonizing acute and chronic kidney disease definition and classification: report of a Kidney Disease Improving Global Outcomes (KDIGO) Consensus Conference. *Kidney Int.* 2021; 100: 516-526. https://doi.org/10.1016/j.knt.2021.06.028.
29. Levey AS. Defining AKD: The Spectrum of AKI, AKD, and CKD. *Nephron.* 2022; 146:302-305. https://doi.org/10.1159/000516647
30. Chawla LS, Bellomo R, Bihorac A, Goldstein SL, Siew ED, Bagshaw SM, et al. Acute Disease Quality Initiative Workgroup 16. Acute kidney disease and renal recovery: consensus of the Acute Disease Quality Initiative (ADQI) 16 Workgroup. *Nat Rev Nephrol*. 2017; 13(4):241-257. https://doi.org/10.1038/nrneph.2017.2.
31. Palevsky PM. Epidemiology, etiology, pathophysiology, and diagnosis. In: Lerma EV, Sparks MA, Topf JM, editors. *Nephrology Secrets.* 4th Edition. Philadelphia: Elsevier; 2019. p. 45-52.
32. Clarkson MR, Magee CN, Brenner BM. *Pocket Companion to Brenner & Rector's The Kidney.* 8th Edition. Philadelphia: Saunders Elsevier; 2010.
33. Blakeley S. Acute kidney injury. In: Blakeley S, editor. *Renal Failure and Replacement Therapies*. Hampshire: Springer, Hampshire; 2008.p.19–25.

34. Perazella MA, Rosner MH. Drug-induced acute kidney injury. *CJASN*. 2022; 17: 1220-1233. https://doi.org/10.2215/cjn.11290821
35. Dainton M. Acute Kidney Injury. In: Thomas N, editor. *Renal Nursing. Care and Management of People with Kidney Disease.* 5th Edition. Oxford: Wiley Blackwell; 2019. p.105-126.
36. Terrill B. *Renal Nursing. A Guide to Practice.* Oxon: Radcliffe Medical Press; 2002.
37. Royal College of Physicians. *Acute Care Toolkit 12: Acute Kidney Injury and Intravenous Fluid Therapy.* 2015. Available at: https://www.rcplondon.ac.uk/guidelines-policy/acute-care-toolkit-12-acute-kidney-injury-and-intravenous-fluid-therapy (accessed 22nd November 2022).
38. Stirling C, Stevens K. *eGFRsupport Acute Kidney Injury-Investigations and Management.* 2021; Clinical Guideline. NHS Greater Glasgow and Clyde. Available at: https://covid19app.nhsggc.org.uk/media/2355/id-207-egfr-kidney.pdf (accessed 11th December 2022).
39. National Health Service and United Kingdom Renal Registry. "*Think Kidneys". Recommended Minimum Requirements of a Care Bundle for Patients with AKI in Hospital.* 2020; Available at: https://www.thinkkidneys.nhs.uk/aki/wp-content/uploads/sites/2/2020/03/AKI-care-bundle-2020.pdf. (accessed 8th December 2022).
40. Health Service Executive. *What's new in INEWS V2*. 2020; Irish National Early Warning Score System (INEWS). Available at https://www.hse.ie/eng/about/who/cspd/ncps/deteriorating-patient-improvement-programme/inews-patient-observation-chart.pdf (accessed 16th December 2022).
41. Centre for Reviews and Dissemination University of York. *Acute Kidney Injury: the 5R's approach*. 2015; Effectiveness Matters. Available at: https://www.thinkkidneys.nhs.uk/aki/wp-content/uploads/sites/2/2015/12/Effectiveness-Matters-Dec-2015-AKI.pdf (accessed 16th December 2022).
42. Ostermann M, Bellomo R, Burdman EA, Doi K, Endre ZH, Goldstein SL et al. Controversies in acute kidney injury: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Conference. *Kidney Int.* 2020; 98(2): 294–309. https://doi.org/10.1016/j.kint.2020.04.020.
43. National Health Service and United Kingdom Renal Registry. "*Think Kidneys". Nutritional Considerations in Adult Patients with Acute Kidney Injury.* 2021. Available at: https://www.thinkkidneys.nhs.uk/aki/wp-content/uploads/sites/2/2021/05/Think-Kidneys-Nutrition-Guide-2021-FINAL.pdf. (accessed 10th September 2022).
44. Alfonzo A, Harrison A, Baines R, Chu A, Mann S, MacRury M. *Clinical Practice Guidelines Treatment of Acute Hyperkalaemia in Adults.* 2020; UK Kidney Association. Available at: https://ukkidney.org/sites/renal.org/files/RENAL%20ASSOCIATION%20HYPERKALAEMIA%20GUIDELINE%20-%20JULY%202022%20V2_0.pdf (accessed 18th December 2022).
45. Glowacka M, Lipka S, Mlynarska E, Franczyk B, Rysz J. Acute kidney injury in COVID-19. *Int J Mol Sci.* 2021; 22:8081. https://doi.org/10.3390/ijms22158081.
46. Nadim MK, Forni LG, Mehta RL, Connor Jr MJ, Liu KD, Ostermann M, et al. COVID-19 - associated acute kidney injury: consensus report of the 25th Acute Disease Quality Initiative (ADQI) workgroup. *Nature Reviews Nephrology*. 2020; 16: 747-764. https://doi.org/10.1038/s41581-020-00356-5
47. Sabaghian T, Benham Kharazmi A, Ansari A, Omidi F, Neda Kazemi S, Hajikhani B, et al. COVID-19 and acute kidney injury: a systematic review. *Front Med*. 2022; 9: 705908. http://doi.org/10.3389/fmed.2022.705908.
48. National Institute Health and Care Excellence. *COVID-19 rapid guideline: Managing COVID-19.* 2022; NICE guideline [NG191]. Available at: https://www.nice.org.uk/guidance/ng191 (accessed 27th November 2022).
49. Murray J, Suntharalingam G, Cockwell P, Baharani J, Devonald M, Lui Forni L et al. *Renal replacement therapy for critically unwell adult patients: Guidelines for best practice and service resilience during COVID-19.* 2020; UK Kidney Association, British Renal Society, and Intensive Care Society. Available at: https://ukkidney.org/health-

professionals/guidelines/renal-replacement-therapy-critically-unwell-adult-patients (accessed 28th November 2022).
50. Setyawati T, Aditya R, Trihartini Maskoen T. Sepsis associated acute kidney injury. In: Neri V. editor. *Infections and Sepsis Development*. Vienna: IntechOpen; 2021. http://dx.doi.org/10.5772/intechopen.97609.
51. Harris PL, Umberger RA. Long term renal outcomes in adults with sepsis-induced acute kidney injury. *Dimensions of Critical Care Nursing*. 2020; 39 (5): 259-268. http://doi.10.1097/DCC0000000000000432.
52. National Institute for Health and Care Excellence. *Acute Kidney Injury: Prevention, Detection and Management. [A] Evidence reviews for preventing contrast-induced acute kidney injury.* 2019b; NICE guideline [NG148]. Available at: https://www.nice.org.uk/guidance/ng148/evidence/a-preventing-contrastinduced-acute-kidney-injury-pdf-70192655. (accessed 13th December 2022).
53. Morcos R, Kucharik M, Bansal P, Al Taii H, Manam R, Casale J et al. Contrast-induced acute kidney injury: Review and practical update. *Clin Med Insights Cardiol*. 2019; 1; 13. http://doi.10.1177/1179546819878680.
54. Bonfield B. An evaluation of patient's knowledge about acute kidney injury pre- and post-follow-up clinic. *J Kidney Care.* 2022; 7 (3): 120-126.
55. Diamantidis CJ, Burks E, Mohottige D, Riley J, Bowman C, Lunyera J et al. What do acute kidney injury survivors want to know about their condition: A qualitative study. *Kidney Med.* 2022; 4(4):100423. https://doi.org/10.1016/j.xkme.2022.100423.
56. Murphy F, Byrne G. Promoting kidney health in cardiovascular disease: part two. *Br J Card Nurs.* 2022; 17 (12) https://doi.org/10.12968/bjca.2022.0111.
57. Murphy F. Psychological perspectives. In: Thomas N, editor. *Renal Nursing: Care and Management of People with Kidney Disease.* 5th edition. Oxford: Wiley Blackwell; 2019. p. 81-104.
58. Murphy F, Byrne G. Promoting kidney health in cardiovascular disease: part one. *Br J Card Nurs.* 2022; 17 (10) https://doi.org/10.12968/bjca.2022.0083
59. Osmer C, Dunne N. Nurses' knowledge and confidence in acute kidney injury: a feasibility study. *J Kidney Care.* 2022; 7 (6): 269-278.
60. Grace A. A role that has transformed the care of patients with acute kidney injury. *Br J Nurs.* 2022; 31 (5): 296.