MODERN APPLIED MEDICAL RESEARCH (MAMR) 1(4): 13-17
ISSN:2582-9181

Incidence and Prevalence of Acute Kidney Injury In Critically Ill Patients

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REVIEW-ARTICLE

Received: **03-04-2021**
Accepted: **10-04-2021**
Published: **11-04-2021**

Abstract: Many patients with AKI have a mixed aetiology as the presence of co-factors as sepsis, ischaemia and nephrotoxicity often co-exist and complicate recognition and treatment. This syndrome is more common among patients without critical illness and it is essential that health care professionals, particularly those without specialisation in renal disorders, detect it easily.

Keywords: Acute kidney injury - Critical Patients -Acute pain - VAS score

Introduction.

At the beginning of the twentieth century, ARF, then named Acute Bright's disease, was described in William Osler's Textbook for Medicine (1909), to be "as a consequence of toxic agents, pregnancy, burns, trauma or operations on the kidneys". During the First World War the syndrome was named war nephritis, and was reported in several publications. The syndrome was forgotten until the Second World War, when Bywaters and Beall published their classical paper on crush syndrome. [1-2]

Acute Kidney Injury (AKI) is the term that has recently replaced the term ARF. AKI is defined as an abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and impairment (loss of function). It is a syndrome that rarely has a sole and distinct pathophysiology. [3]

Many patients with AKI have a mixed aetiology as the presence of co-factors as sepsis, ischaemia and nephrotoxicity often co-exist and complicate recognition and treatment. This syndrome is more common among patients without critical illness and it is essential that health care professionals, particularly those without specialisation in renal disorders, detect it easily. [4-5]

Acute tubular necrosis (ATN) was the term that was used to describe this clinical entity, because of histological evidence for patchy necrosis of renal tubules at autopsy. For many years in clinical practice, the terms ATN and ARF were used interchangeably. [6]

Homer W. Smith who is credited for the introduction of the term *acute renal failure*, in a chapter on *Acute renal failure related to traumatic injuries* in his 1951 textbook *The kidney-structure and Function in Health and Disease*. Until recently, a precise biochemical definition for ARF was missing. [7]

The aim of this manuscript was to assess the incidence and privilence of acute kidney injury in critically ill patients.

Epidemiology:

In urban areas of developing countries, main causes of AKI are hospital acquired (renal ischaemia, sepsis and nephrotoxic drugs) while in rural areas it is more commonly a consequence of community acquired disease (diarrhoea, dehydration, infectious diseases, animal venoms etc.). Under-reporting of AKI especially in developing countries is also a major problem that relates with the true knowledge of its impact in many parts of the world. [8-9]

In developed countries the prevalence of AKI is increasing. In hospital inpatients it is estimated to occur up to 15% and is more common in critically ill patients, in whom its prevalence is estimated to be up to 60%. Community AKI is usually uncommon although a recent study estimated its incidence at 4.3% among all hospital admissions.
[10]

Multiple studies have shown that AKI in the elderly (usually defined as older than 65 years) is increasingly common and that there is an age-dependent relationship between AKI and older age. This may be due to mixed aetiology of anatomic and physiologic changes in the aging kidney and in part to various comorbidities as: hypertension, cardiovascular disease, chronic kidney disease (CKD) - that may require procedures and/or medications that act as kidney stressors and alter renal haemodynamics or are nephrotoxic. [11-12]

1(4): 13-17
ISSN:2582-9181

Table 1.
Risk, Injury, Failure, Loss of kidney function and End-stage kidney disease (RIFLE) classification

Class	GFR	UO
Risk	↑ SCr × 1.5 or ↓ GFR >25%	<0.5 mL/kg/h × 6 h
Injury	↑ SCr × 2 or ↓ GFR >50%	<0.5 mL/kg/h × 12 h
Failure	↑ SCr × 3 or ↓ GFR >75% or if baseline SCr ≥353.6 µmol/L(≥4 mg/dL) ↑ SCr>44.2 µmol/L(>0.5 mg/dL)	<0.3 mL/kg/h × 24 h or anuria × 12 h
Loss of kidney function	Complete loss of kidney function >4 weeks	Anuria
End-stage kidney disease	Complete loss of kidney function >3 months	Anuria

GFR (glomerular filtration rate) ; UOP (urine output) ; SCr (serum Creatinine)

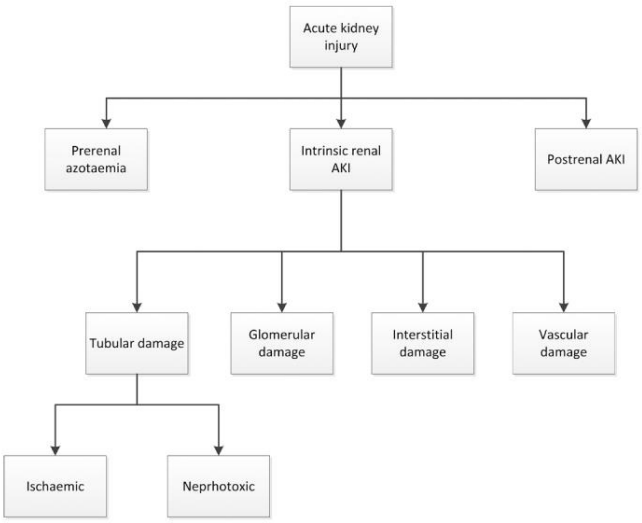
Table 2.

The AKIN classification/staging system of acute kidney injury

Stage	SCr	UOP
1	↑ SCr ≥26.5 µmol/L (≥0.3 mg/dL) or ↑SCr ≥150 a 200% (1.5 a 2×)	<0.5 mL/kg/h (>6 h)
2	↑ SCr>200 a 300% (>2 a 3×)	<0.5 mL/kg/h (>12 h)
3	↑ SCr>300% (>3×) or if baseline SCr ≥353.6 µmol/L (≥4 mg/dL) ↑SCr ≥44.2 µmol/L (≥0.5 mg/dL)	<0.3 mL/kg/h (24 h) oranuria (12 h)

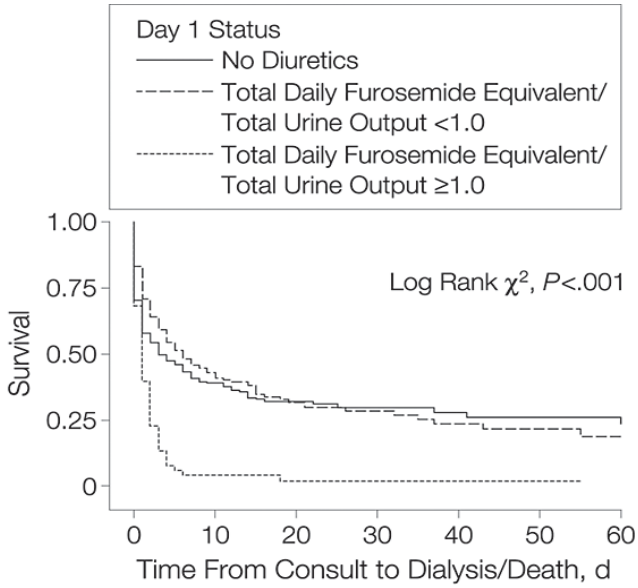
SCr (serum Creatinine) – UOP (urine output) - Stage 3 also includes patients requiring RRT independent of the stage (defined by SCr and/or UOP) they are in at the moment they initiate RRT.

Figure 1; Classifiation and causes of AKI



During an ischaemic insult all segments of the nephrons can be affected but proximal tubular cells are the most commonly injured. In addition, the nephron's natural function is to filter, concentrate and reabsorb many substances from tubular lumen, and the concentration of these substances may reach toxic levels for the surrounding epithelial cells. A detailed description of the sequence of events and the cellular changes during ischaemic AKI can be found elsewhere.

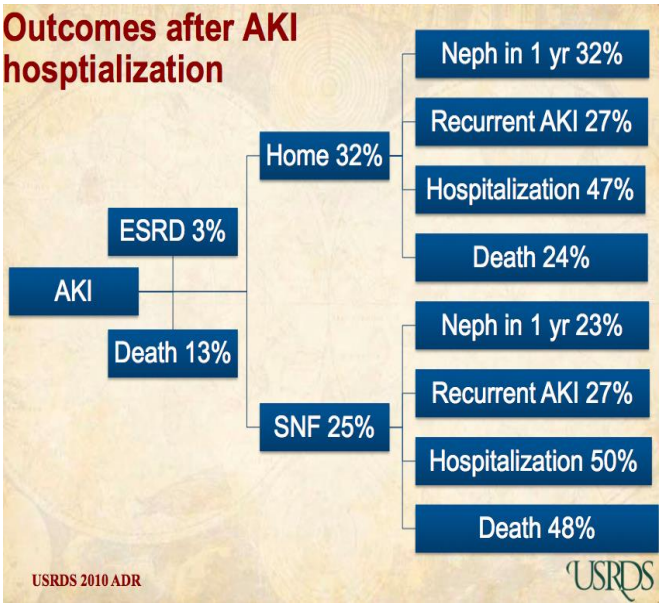
Figure 2 Time to Death or Dialysis from Day of Consultation in Intensive Care Unit Groups are stratified by day 1 status.



1(4): 13-17
ISSN:2582-9181

For those patients who were diuretic resistant (furosemide equivalent per milliliter ratio ≥1.0), the No. at risk for days 1, 2, 3, and 5 were 35, 19, 10, and 3, respectively. Analysis includes 411 of the 416 patients who survived at least 7 days after nephrology consultation in the intensive care unit. Data are excluded for 5 patients who died at an unknown time.

Figure 3; Outcomes of AKI



Approximately 5% to 6% of patients with AKI require renal replacement therapy and the mortality rate in this popu- lation that requires renal replacement therapy is approximately 50% to 70%. AKI also significantly increases length of hospital stay and is associated with a significant increase in the risk for chronic kidney disease and end-stage renal disease.

Discussion

Recently, studies also have identified that even small changes in serum creatinine are associated with significant increases in mortality. Furthermore, AKI survivors are still at high risk for long-term adverse outcomes such as chronic kidney disease, end-stage renal disease, and premature death, even if the serum creatinine level returns to normal. [13]

The importance of RIFLE (Table 1) criteria is that they move beyond ARF. The term “acute kidney injury/impairment” has been proposed to encompass the “entire spectrum of the syndrome from minor changes in markers of renal function to requirement for renal replacement therapy (RRT)”. [14]

The concept of AKI, as defined by RIFLE, creates a new paradigm. AKI encompasses ATN and ARF as well as other, less severe conditions. It includes patients without actual damage to the kidney but with functional impairment relative to physiologic demand. Including such patients in the classification of AKI is conceptually attractive because these are precisely the patients that may benefit from early intervention. The RIFLE criteria have also been modified for use in the paediatric setting. [15]

Pickering et al., 2011 showed that there was a mismatch between increases in sCr concentration and decreases in GFR (estimated with MDRD or Cockroft-Gault formulae) in the descriptions of Risk and Failure severity grades. A 1.5-fold increase in sCr corresponds to a one-third decrease (not 25%) in GFR and a three-fold increase corresponds to a two-third decrease in GFR (not 75%). [16]

If the GFR is not directly measured but estimated by a formula then results might be also different depending on the formula used. With the MDRD formula a 1.5-fold increase in sCr corresponds to a 37% decrease in GFR, and a three-fold increase in sCr to a 72% decrease in GFR. [16]

The limitations of RIFLE classification include, the endogenous production and serum release of Cr are variable, and it is influenced by multiple factors, namely age, gender, diet, and muscle mass. 10 to 40% of Cr elimination is performed by tubular secretion and this mechanism is amplified as the GFR diminishes, thus, overestimating renal function in AKI patients. [18]

The pathophysiology of AKI (Figure 1) is multi-factorial and complex. The most common cause of AKI is ischemia, which can occur for a number of causes. Physiological compensatory accommodations occur, in response to the reduction in blood flow can compensate to a certain degree, but when delivery of oxygen and metabolic substrates becomes inadequate, the resulting cellular injury leads to organ dysfunction. The kidney is highly susceptible to injury related to ischaemia, resulting in vasoconstriction, endothelial injury, and activation inflammatory processes. [19]

Following the reduction in effective kidney perfusion, the epithelial cells are unable to maintain adequate intracellular ATP for essential processes. This ATP-depletion leads to cell injury and if it is severe enough can lead to cell death by necrosis or apoptosis. During an ischaemic insult all segments of the nephrons can be affected but proximal tubular cells are the most commonly injured. In addition, the nephron’s natural function is to filter, concentrate and reabsorb many substances from tubular lumen, and the concentration of these substances may reach toxic levels for the surrounding epithelial cells. A detailed description of the sequence of events and the cellular changes during ischaemic AKI can be found elsewhere. [20-21]

The aim of work is to prevent complications and allow the

kidneys time to heal. Treatments that help prevent complications include: Treatments to balance the amount of fluids in the blood. If acute kidney failure is caused by a lack of fluids in the blood, the doctor may recommend intravenous (IV) fluids. In other cases, acute kidney failure may cause to have too much fluid, leading to swelling in your arms and legs. In these cases, the doctor may recommend medications (diuretics) to cause the body to expel extra fluids. [22-23]
Dialysis aims to remove toxins from the blood. If toxins build up in the blood, It may be needed temporary hemodialysis — often referred to simply as dialysis — to help remove toxins and excess fluids from your body while your kidneys heal. Dialysis may also help remove excess potassium from the body. During dialysis, a machine pumps blood out of your body through an artificial kidney (dialyzer) that filters out waste. The blood is then returned to your body. [24]

Conclusion: the frequency post-operative AKI (abnormal KFT or decreased uop , 25% of all studied patients) and incidence of AKI in these patients was 48.1% versus 10.4% in patients with normal post-operative KFT. Hypotension is strongly associated with increased postoperative complication as AKI.

Abbreviation:

AKI: acute kidney injury,

ATN: acute tubular necrosis,

CKD: chronic kidney disease,

CK: creatine kinase,

ECG: Electrocardiogram,

eGFR: estimated glomerular filteration rate,

IOH: intraoperative hypotension,

MAP: mean arterial blood pressure,

CVP: central venous pressure

Aim: To detect incidence and prevalence of AKI in critically ill patients.

Results: 25.2% of critically ill patients developed AKI, 19.9% of them required dialysis, which was 22.1% in patients with change to chronic renal failure, (4.9%) who developed complications were died during stay in ICU.

Conclusion: incidence of AKI 48.1% versus 10.4% in patients with normal post-operative KFT. Hypotension is strongly associated with increased postoperative complication as AKI.

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