CHAPTER 12

Community- and Hospital-Acquired Acute Kidney Injury

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OBJECTIVES

This chapter will:
1. Describe the epidemiology of community and hospital-acquired acute kidney injury.
2. Review the different causes of acute kidney injury.

Acute kidney injury (AKI) is a common and devastating problem that has an independent effect on mortality.[1] Traditionally, *acute kidney injury* has been defined as a rapid (i.e., over hours to weeks) and usually reversible decline in glomerular filtration rate.[2,3] Although AKI has been the focus of extensive clinical and basic research efforts over the last decades, more than 30 definitions had been published as of 2002.[4] Recognizing the need to have standardized definition, Kidney Disease: Improving Global Outcomes (KDIGO) recently developed a consensus AKI definition.[5]

The epidemiology of chronic kidney disease (CKD) is well characterized, and the disease has been defined by the KDIGO guidelines with stages based on estimates of glomerular filtration rate (GFR).[6] The United States Renal Data System (USRDS) provides ongoing analysis of the incidence, prevalence, treatment, morbidity, and mortality of end-stage renal disease (ESRD) based on systematic data collection. Similar national data on the epidemiology of AKI are limited.

In 2005 the International Acute Dialysis Quality Initiative (ADQI) group proposed new criteria for classifying AKI on the basis of serum creatinine and/or urine output values.[7] This classification system defines three increasingly severe levels of renal dysfunction. Patients are classified according to their *risk* of renal dysfunction, type of kidney *injury*, and extent of renal *failure* associated with two clinical outcomes: *loss* and *end-stage* renal disease (RIFLE).[7] Although serum creatinine (SCr) and urine output values may not delineate clearly the timing, nature, and pathophysiologic basis of AKI, the data obtained from the RIFLE classification suggest a strong relationship between the magnitude of renal dysfunction and adverse outcomes.[8] A collaboration of more than 25 international societies in nephrology and critical care has led to the creation of an Acute Kidney Injury Network (AKIN). This group proposed a new definition and staging system for AKI based on either an absolute or a percentage increase in SCr.[9] The AKIN criteria differ from RIFLE criteria by suggesting change of SCr within 48 hours' duration.[8,9] In 2012 the KDIGO proposed a revision of the AKI definition by including absolute increase in SCr of ≥0.3 mg/dL over 48 hours or ≥50% increase in SCr within 7 days as a

diagnostic criterion.[5] Warnock et al. have demonstrated that recognizing the pattern of SCr change is important and can help in distinguishing between community-acquired AKI versus hospital-acquired AKI. The patients who developed hospital-acquired AKI have peak SCr that follows minimum SCr and are associated with worse outcomes, whereas peak SCr preceded the minimum SCr in community-acquired AKI.[10] More recently, the concept of acute kidney disease (AKD) is being proposed, which is defined as AKI, or GFR <60 mL/min/1.73 m^2 for <3 months, or a decrease in GFR by ≥35% for <3 months, or an increase in SCr by >50% for <3 months. However, the interpretation and application of AKD criteria and its prognostic significance are yet to be determined.[11]

COMMUNITY-ACQUIRED ACUTE KIDNEY INJURY

The majority of epidemiologic studies on AKI have been published with data from developed countries (Table 12.1). A study from the United States by Kaufman et al. in 1991[12] found that community-acquired AKI was responsible for 1% of all hospital admissions and that prerenal azotemia and exacerbation of CKD were the major causes. Similarly, Feest et al.[13] published in 1993 a prospective study that looked at community-acquired AKI in England. They found that the incidence of severe AKI in the community was at least twice as high as that reported from renal unit-based studies. Another interesting observation was that 72% of the patients were older than 70 years. The survival rate at 2 years was 34%.

In a study published in 1996, the Madrid Acute Renal Failure Study Group found the incidence of AKI to be 209 per million population (pmp).[14] Importantly, they defined *renal failure* at that time as a sudden rise in serum creatinine to more than 177 μmol/L or sudden increase in SCr by more than 50% in patients in whom prior renal function was normal. Using an SCr level greater than 300 μmol/L as a definition for AKI, Khan et al.[15] reported an incidence of 602 pmp in Scotland in 1997. These results show that the incidence of community-acquired AKI has been rising over the years. These data were substantiated further by the Kaiser Permanente study, in which the incidence of

TABLE 12.1

Epidemiology of Community-Acquired Acute Kidney Injury in Developed World

COUNTRY	STUDY*	TYPE OF STUDY	STUDY PERIOD	DEFINITION OF AKI	STUDY POPULATION (MILLIONS)	INCIDENCE (PER MILLION POPULATION PER YEAR)
Israel	Eliahou et al., 1973[83]	Prospective	1965–1966 (2 years)	Unknown	2.2	52
Kuwait	Abraham et al., 1989[84]	Prospective	1984–1986	Unknown	0.4	95
United States	Kaufman et al., 1991[12]	Prospective	17 mo	Serum creatinine >2 mg/dL	1% of all hospital admissions	-
United Kingdom	McGregor et al., 1992[85]	Prospective	1986–1988		0.94	185
United Kingdom	Feest et al., 1993[13]	Prospective	2 years	Serum creatinine >500 μ mol/L	0.44	175
France	Chanard et al., 1994[86]	Prospective	1991	Requirement for dialysis		104
Spain	Liaño & Pascual, 1996[82]	Prospective	9 mo	Sudden increase in serum creatinine by >177 μmol/L or sudden increase in serum creatinine by >50% when prior renal function was normal	4.2	209
Scotland	Khan et al., 1997[15]	Retrospective	1 year	Serum creatinine >300 μ mol/L	0.5	620
England	Stevens et al., 2001[87]	Prospective	1 year	Serum creatinine >300 μmol/L	0.59	486
Canada	Bagshaw et al., 2005[88]	Prospective	3 years	New requirement for renal replacement therapy with evidence of renal dysfunction (serum creatinine ≥150 μmol/L)	1	11 per 100,000 per year
Scotland	Ali, 2007[89]	Retrospective	6 months	Rise of serum creatinine to >1.5× baseline times or drop in glomerular filtration rate of 25%	0.52	1811
United States	Hsu, 2009[16]	Retrospective	1996–2003	Rise of serum creatinine of 0.5 mg/dL for baseline ≤ 1.9 mg/dL; 1 mg/dl for baseline level 2.0–4.9 mg/dL; 1.5 mg/dL for baseline≥5 mg/dL	3.7	322.7 to 522.4 per 100,000 person-years
England	Talabani, 2014[90]	Retrospective	2009 (1 month)	Rise of serum creatinine to >1.5× baseline creatinine	0.03	-

*Superscript numbers indicate chapter references.

community-acquired AKI not requiring dialysis increased from 322.7 to 522.4 and that dialysis-requiring AKI increased from 19.5 to 29.5 per 100,000 person-years between 1996 and 2003.[16] This increase in the incidence of community-acquired AKI may be attributed to the use of more sensitive definitions, identifying the disease at an earlier stage.

Recently, Susantitaphong et al. performed a world-wide meta-analysis of 154 studies (130 adult studies and 24 studies from children; n = 3,585,911) using KDIGO-equivalent AKI definition. This meta-analysis showed pooled global incidence rates of AKI of 21.6% (95% CI, 19.3 to 24.1) in adults and 33.7% (95% CI, 26.9 to 41.3) in children.[17] However, the majority of studies included were from high-income countries (41% from Europe and 43% from North America). This meta-analysis highlighted the growing incidence of community-acquired AKI in low-income and middle-income countries, which is comparable to the developed world.[17]

Table 12.2 lists epidemiologic studies on the incidence of community-acquired AKI in the developing world. There are no reliable statistics about the true incidence of community-acquired AKI in developing countries. It is believed that the incidence is 150 per million population.[18] This incidence is based on sporadic regional reports. However, these results are probably biased because they represent only the cases encountered in large hospitals by physicians having an interest in AKI. Also, different definitions are used in different regions to assess the incidence of AKI. Moreover, the majority of AKI causes are medical, as opposed to surgical or traumatic causes observed in the developed world.

TABLE 12.2

Epidemiology of Community-Acquired Acute Kidney Injury in Developing World

COUNTRY	AUTHOR*	STUDY FEATURES	INCIDENCE
South Africa	Seedat et al., 1993 [23]	Adults study from 1986–1988	20 per million population
Saudi Arabia	Al Homrany et al., 2003[91]	26,000 adults during 2 year of observation	2.3 per 1000 admissions
Chile	Vukusich et al., 2004[92]	10 urban centers, 114 adults requiring dialysis during a 6 month period	0.31 per 1000 discharges
Nigeria	Anoche et al., 2005[93]	Children presenting to referral center	11.7 per million children per year
China	Wang et al., 2005/2007[94]	225,000 patients admitted between 1994–2003	0.54 per thousand admissions
India	Kohli et al., 2007[95]	33,301 admissions during 1 year period	6.6 per thousand admissions

*Superscript numbers indicate chapter references.

Chugh et al.[19] have shown that obstetric and hemolytic causes of AKI tend to decrease in an area or country as economic power and availability of hospitalization improve. Jayakumar and colleagues published 10-year data on AKI at a tertiary care hospital in South India.[20] The interesting findings in this paper were the younger age of patients (37.8 years) and the overwhelming majority of medical causes (87%). Obstetric causes were not significantly different from those observed in 1987, accounting for 8.9% of cases. Similar findings are seen in publications from Africa. The patients in whom AKI develops are young, and the predominant causes of AKI are infections, toxins, and herbal medications.[21–23]

In a landmark multinational observational cross-sectional study among 289 centers in 72 countries, Mehta et al. showed that the incidence of community-acquired AKI was 58% (2337/4018) in adult patients.[24] The incidence of community-acquired AKI was higher in low-income and lower-middle-income countries (77%; 889/1153) as compared with upper-middle-income (51%; 815/1605) and high-income (50%; 633/1260) countries.[24] AKI was defined using modified KDIGO AKI criteria. Dehydration was the most common cause of AKI among low-income countries (46% [526/1153] vs. 39% [492/1360] in high-income), and hypotension or shock were most common in high-income countries (45% [564/1260] vs. 38% [440/1153] in low-income).[24]

In conclusion, the ethnicity, geographic climate, culture, and socioeconomic status of a country determines the spectrum of AKI causes observed. When a developing country improves its economic situation, the spectrum moves toward that observed in developed countries.[19,23]

HOSPITAL-ACQUIRED ACUTE KIDNEY INJURY

Over the past decade, several studies have shown that the burden of hospital-acquired AKI is increasing in the developed world,[25,26] whereas there are limited data on the incidence of hospital-acquired AKI in the developing world. In one study looking at the incidence and mortality of AKI in US Medicare beneficiaries (5,403,015 discharges), Xue et al. showed that between 1992 and 2001 the incidence of AKI was 23.8 cases per 1000 discharges, with an 11% increase per year.[25] Similarly, using US Nationwide Inpatient Sample, a nationally representative sample of discharges from acute-care nonfederal hospitals, Waikar et al. demonstrated that between 1988 and 2002, the incidence of AKI increased from 61 to 288 per 100,000 population, and the incidence of AKI requiring dialysis increased from 4 to 27 per 100,000 population.[26] In both studies, AKI was determined using ICD-9-CM diagnosis codes, which may have underreported the true incidence of AKI.

Uchino et al. studied the epidemiology of AKI across 54 hospitals in 23 countries and showed that from 29,269 patients admitted in intensive care units (ICUs), 1738 (5.6%) patients developed AKI during their ICU stay.[27] The prevalence of AKI ranged from 1.4% to 25.9% across all study centers. In this study, AKI was defined as oliguria (urine output < 200 mL in 12 hours) and/or blood urea nitrogen level >84 mg/dL. In another multicenter prospective observation study among intensive care centers in both developing (5 centers) and developed (9 centers) countries, Bouchard et al. demonstrated that out of 6647 patients screened, 1275 (19.2%) developed AKI using AKIN criteria (≥0.3 mg/dL

within 48 hours).[28] Patients in developed countries had more sepsis (52.1% vs. 38.0%) and higher Acute Physiology and Chronic Health Evaluation scores (61.1±27.5 vs. 51.1±25.2) and most often suffer from AKI attributed to prerenal, septic, cardiorenal factors, or acute tubular necrosis, whereas patients in developing countries had more CKD (54.3% vs. 38.3%), and were diagnosed more often with glomerulonephritis (6.3% vs. 0.9%), and interstitial nephritis (7.0% vs. 0.6%).[28] The results from recent Global-snapshot 0by25 AKI survey showed the incidence of hospital-acquired AKI to be 39% (1429/3661) in adults and 53% (187/354) in children. The incidence of hospital-acquired AKI was higher in developed countries (64% in North America, 39% in West Europe) compared with low-income developing countries (15% in Africa, 18% South Asia, and 19% in Eastern and Central Europe).[24] In conclusion, the majority of incidence estimates of hospital-acquired AKI are obtained primarily from ICU-specific data, whereas limited data are available for hospital-acquired AKI, which develops outside ICUs.

CAUSE OF ACUTE KIDNEY INJURY

Table 12.3 lists the most common causes of AKI. In the developed world, AKI is associated with multiorgan failure, sepsis, postoperative states, use of iodinated contrast and drugs. In opposition, the spectrum of AKI causes in the developing world differs and includes infectious causes such as malaria, leptospirosis or hemolytic uremic syndrome, gastroenteritis, poisoning, and septic abortion.

CONTRAST-INDUCED ACUTE KIDNEY INJURY

It is estimated that 6000 diagnostic and 2000 therapeutic coronary catheterizations are performed per million population every year in developed countries. The proportion of procedures that require the use of iodinated contrast media has increased over time, and the population getting these procedures is older, with more comorbidities.[29] Depending on the definition used, the incidence of AKI because of the use of iodinated contrast media has been reported to be as high as 40% to 90% in high-risk groups.[30,31] The most common definition of contrast-induced acute kidney injury (CI-AKI) is a rise of SCr of 0.5 mg/dL or a 25% relative rise in SCr at 48 hours after contrast exposure.

Iodinated contrast–induced AKI is typically asymptomatic and nonoliguric.[32] Serum creatinine concentration usually rises 24 to 72 hours after exposure, peaks at 3 to 5 days, and returns to baseline in 7 to 14 days.[33,34]

Studies have shown that outpatient coronary catheterization is safe and feasible in selected groups.[35,36] Most patients undergoing the procedure have been discharged home just when their SCr would have begun to rise if they were to experience CI-AKI. Therefore it is important to ensure that the SCr level is measured within 48 to 72 hours after this procedure to detect AKI.

TABLE 12.3

Causes of Acute Kidney Injury in Developed and Developing Countries

In developed countries	Infections
	Community-acquired pneumonia
	Urosepsis
	Drugs
	Nonsteroidal antiinflammatory drugs
	Antibiotics
	Antiretroviral agents
In developing countries	Infections
	Diarrheal diseases: cholera
	Parasitic: malaria, dengue
	Drugs
	Herbs and indigenous medicines
	Industrial exposure to chemicals
	Hemolytic: snake bite
	Obstetric: septic abortion
	Earthquakes
	Crush syndrome
	Accidents

COMMUNITY-ACQUIRED PNEUMONIA AND ACUTE KIDNEY INJURY

Community-acquired pneumonia is the seventh leading cause of death overall and the most common cause of death from infectious diseases in the United States. In 2006 about 1.2 million US patients were hospitalized for treatment of community-acquired pneumonia.[37] The majority of pneumonia occurs in adults 65 years or older, and AKI is observed in 11% to 24% of patients with this condition.[38,39] In a post-hoc analysis of the Genetic and Inflammatory Markers of Sepsis (GenIMS) study, out of 1836 patients hospitalized with community-acquired pneumonia, AKI was observed in 631 patients (34%). Patient with AKI had higher mortality at hospital discharge (11 vs. 1%), at 90 days (24 vs. 10%), and at 1 year (36 vs. 20%).[40] In another multicenter study, AKI was found to be one of the three independent variables independently associated with death in patients who had community-acquired pneumonia requiring mechanical ventilation.[39]

DRUGS AND ACUTE KIDNEY INJURY

Drug-induced kidney disease is a frequent cause of renal dysfunction and accounts for approximately 19% to 26% of cases of AKI in hospitalized patients.[41] The phenotype of drug induced kidney injury includes AKI (SCr and urine output criteria), glomerular disorder (proteinuria or hematuria), tubular disorder (electrolyte imbalances), and nephrolithiasis.[42] Nonsteroidal antiinflammatory drugs (NSAIDs) are prescribed commonly as analgesics and antiinflammatory drugs in the general population. Various adverse effects of NSAIDs, such as gastrointestinal bleeding, have been studied widely and quantified, but much less is known about the renal effects of these agents in the general population. The incidence of NSAID-induced AKI in the community has been reported as 1.1 per 10,000 patient-years[43] to 2 per 100,000 patient-years.[44] The relative risk of AKI associated with NSAIDs use is 1.6 to 2.2 compared with nonusers.[45] One nested case-control study from the United Kingdom showed that NSAID users had a threefold increased risk for developing AKI than nonusers in the general population. The use of diuretics and

angiotensin-converting enzyme inhibitors were associated independently with an increased risk for AKI, and the risk was even greater with the concomitant use of NSAIDs and diuretics or calcium channel blockers.[46]

A few other epidemiologic studies have looked at the association between the use of NSAIDs and AKI in the general population.[47] A history of hypertension, diabetes, or heart failure and the use of cardiovascular drugs are risk factors for AKI.[43,47]

In another study, Schneider et al.[48] looked at the association between exposure to conventional NSAIDs and cyclooxygenase-2 inhibitors and hospitalization for AKI. They found the risk of AKI with low-dose rofecoxib comparable to that with conventional NSAIDs. For celecoxib, the risk also appeared to be dose dependent.[48] In a recent meta-analysis, pooled risk ratios of AKI among individual traditional NSAIDs were not significantly different.[49]

Herbal and nonherbal nephrotoxins are common causes of AKI in Africa and Asian countries and contribute to 18% of cases of community-acquired AKI globally.[50] Traditional medications known to be associated with AKI includes cancer bush *(Sutherlandia frutescens),* ysterhouttoppe *(Dodonaea angustifolia),* marking nut tree *(Semecarpus anacardium),* Bao Gong Teng *(Erycibe obtusifolia Benth), Opuntia megacantha,* and Alder buckthorn bark.[50]

SEPSIS AND ACUTE KIDNEY INJURY

Martin et al.[51] have studied the epidemiology of sepsis in the United States over a 22-year period (1979–2000). They found that the incidence of sepsis rose during that time from 82.7 to 240.4 cases per 100,000 population, for an annualized increase of 8.7%. AKI was observed in 15% of cases. Similarly, Angus et al.,[52] analyzing more than 6 million hospital discharge records from seven states in the United States, estimated that 751,000 cases of severe sepsis occur annually, with a mortality rate of 28.6%. AKI was observed in 22% of cases. In a multicenter, international study of 4532 adults admitted with septic shock, 64.4% of patients developed AKI (16.3% had risk, 29.4% had injury, and 18.7% had failure). Patients who developed AKI were sicker, older, more likely to be female, and had more comorbidities.[53] In another study including critically ill patients with AKI by the Program to Improve Care in Acute Renal Disease (PICARD) group, 174 (28%) of patients were septic before AKI diagnosis.[54]

HUMAN IMMUNODEFICIENCY VIRUS INFECTION AND ACUTE KIDNEY INJURY

Acute kidney injury is a common finding in people infected by human immunodeficiency virus (HIV) and is associated with advanced stages of HIV infection (i.e., CD4 cell count less than 200 per mm^3 and HIV RNA level greater than 10,000 copies/mL), hepatitis C virus coinfection, and a history of antiretroviral treatment.[55,56] A prospective analysis of 754 ambulatory HIV-infected patients reported an incidence of 5.9 cases of AKI per 100 patient-years.[57] Several drugs, including aminoglycosides, amphotericin, foscarnet, trimethoprim-sulfamethoxazole, tenofovir, indinavir, and acyclovir potentially can cause AKI in these patients.[58] Furthermore, AKI may be related to thrombotic thrombocytopenic purpura–hemolytic uremic syndrome resulting from the HIV disease process.[56]

MALARIA AND ACUTE KIDNEY INJURY

Malaria is widespread throughout the world. It affects close to 400 million people every year, most of whom live in Africa, India, Southeast Asia, and Latin America.[59] Of these, 2 to 3 million die from complications of the disease each year.[60] The case-fatality rate is increased in the presence of AKI and pulmonary complications.[61]

The incidence of AKI resulting from malaria varies between 2% and 39%, and with hyperparasitemia, can be as high as 60%.[62–64] This wide variation may be due to the local prevalence of the disease, the relative preponderance of other causes, patient referral policy, and other factors. Between 60% and 80% of patients with malarial AKI will need immediate dialysis at presentation.[63,64]

Late referral, high parasitemia, and presentation with multisystem involvement including hepatitis or acute respiratory distress, are risk factors for death from malarial AKI. The rate of mortality has been reported between 15% and 45%.[65] The prevention of malarial infection and early diagnosis are the only measures likely to decrease malarial AKI in developing countries. Early referral to centers equipped to provide renal replacement therapy, along with antimalarial therapy and support, could further reduce mortality and enhance recovery of renal function.

In tropical areas such as Thailand and Singapore, leptospirosis is the leading cause of AKI, with mortality ranging from 17% to 36%.[66] Other infectious diseases including dengue hemorrhagic fever, hanta virus, typhoid fever, shigellosis, and schistosomiasis are associated with AKI in different regions of the developing world.[67] Most cases of hemolytic uremic syndrome (HUS) in Asia and Africa develop in response to shigella dysenteriae type 1 and *Escherichia coli* is the main causative organism for HUS in North America and Europe.[68] In one such report, the incidence of AKI associated with HUS was 36%, with a very high mortality (59%).[69]

GYNECO-OBSTETRIC AND ACUTE KIDNEY INJURY

Occurrence of obstetric AKI is more common in the developing world and accounts for 10% to 30% of AKI cases.[67] In contrast, the incidence of obstetric AKI has decreased in the developed world with 1 case per 20,000 pregnancies.[70] These observed differences may be attributed to improved antenatal care and legalization of abortion in most developed countries. The main factors leading to pregnancy-related AKI include preeclampsia, septic abortions, and uterine hemorrhage. In a retrospective observation study among 4758 pregnant women from the Indian subcontinent, AKI developed in 85 cases (1 in 56 births). Preeclampsia and puerperal sepsis were responsible for AKI in 35% and 25% of cases, respectively.[71] Another study from the subcontinent reported a similar incidence of pregnancy-related AKI (36%).[72] Improved antenatal and perinatal care, with focus on sterile delivery practices and safe practices of abortion, can decrease further the incidence of obstetric AKI in the developing world.

POSTOPERATIVE ACUTE KIDNEY INJURY

AKI is a frequent postoperative complication in high-risk patients undergoing major cardiac and noncardiac surgery. The incidence of cardiac surgery related AKI varies between 8.9% and 39%.[73,74] This wide range is attributed to differences in baseline patient characteristics and type of cardiac surgery (coronary artery bypass, valvular surgery, or combined type). Demographics (age, gender), comorbidities (diabetes, peripheral vascular disease, chronic obstructive pulmonary disease, congestive heart failure, baseline renal function), and intraoperative (use of an intraaortic balloon pump, clamp-time, and on-pump surgery) risk factors are related to cardiac surgery associated AKI.[75]

Thakar et al. retrospectively examined 325,395 patients admitted to Veterans Affairs ICUs between 2001 and 2006 and found that 32% of AKI cases occurred in surgical settings (cardiac surgery 28%, orthopedic surgery 23%, abdominal surgery 20%, trauma surgery 16%, and neurologic surgery 8%).[75] The same investigators reported an incidence of AKI of 8.5% in patients who underwent gastric bypass surgery.[76] The incidence of AKI also is reported to be high in nonrenal solid organ transplant settings, especially in liver transplants.[77]

Last, AKI is also seen in burn patients, with incidence ranging from 10% to 30% and is associated with higher mortality.[78] AKI in burn patients is attributed to fluid loss, myocardial depression, inflammatory mediators, pigmenturia, and use of nephrotoxic drugs.[79]

ACUTE KIDNEY INJURY IN THE ELDERLY POPULATION

Several groups have reported a gradual increase in the mean or median age of population with AKI.[80–82] Over the next decades, demographic projections in developed countries have predicted a large increase in the proportion of patients older than 65 years. This population also has CKD owing to the structural and functional changes in the kidneys occurring with advanced age. In addition, elderly persons are more likely to consume medications and to have comorbidities.

CONCLUSION

In population studies, the incidence of hospital-acquired AKI has risen in the last two decades. This rise appears to be multifactorial, being due to improved survival of patients with diabetes mellitus and ischemic heart disease, a growing elderly population, and better care of high-risk surgical and intensive care patients. There is still uncertainty regarding the true incidence of community-acquired AKI in the developing world. AKI in the developed world is encountered in patients more commonly with multiple organ failure, whereas infections, toxins, and obstetric complications are the main causes of AKI in the developing world.

Key Points

1. The incidence of acute kidney injury is rising worldwide and is more likely to be associated with other organ failure.
2. The increase in incidence of acute kidney injury may be attributed to increased awareness and improvement in the diagnostic capabilities, use of sensitive definitions, and increased comorbidities.
3. The spectrum of acute kidney injury has evolved over the years and is different in developing and developed countries.
4. Sepsis and shock are predominant causes of acute kidney injury in the developed world while diarrhea, infections, and obstetric complications are common causes of acute kidney injury in the developing world.
5. The incidence of community versus hospital-acquired acute kidney injury is not well characterized in the developing world.
6. Various factors in the management of acute kidney injury influence its course and duration and contribute to the differences seen in the developing and developed world.

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