The impact of AKI on the critically ill patient: What it is, how to recognize, and how to intervene

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Gambro, Inc.
Role of the kidney
FUNCTIONS:
- Water & Waste Removal
- Electrolyte Regulation
- Hormone Function
- Acid/Base Regulations

PROBLEMS:
- Fluid Overload with Elevated Wastes (urea & creatinine)
- Electrolyte Imbalances
- Changes in Hormone Levels affecting:
  - Blood Pressure
  - Red Blood Cell Production
  - Uptake of Calcium
- Disturbance of Acid/Base Balance
What do the kidneys do?

• Contribute to homeostasis through excretory or secretory functions

• Filter the blood to:
  • Remove excess fluid
  • Remove waste products
  • Regulate ion concentrations in the blood
  • Regulate acid-base (pH) balance

• Release hormones to:
  • Promote calcium absorption (calcitriol)
  • Promote red blood cell synthesis (erythropoietin)
  • Regulate systolic blood pressure (renin)
The normal kidneys contribute to homeostasis through excretion or secretion, as appropriate. In AKI, the patient could experience:

1. Fluid overload
2. Acid-base imbalances
3. Hematologic dysfunction
4. All of the above
What happens when the kidney does not do what it supposed to?

- Fluid overload
  - Pulmonary edema, pleural effusion
  - Skin breakdown and delayed wound healing
  - Atrial distension
  - Gut-mucosal edema
- Acid-base and electrolyte imbalances
- Hematologic dysfunction
  - Anemia
  - Platelet dysfunction
- Central nervous system
  - Encephalopathy
Since 2004, the term Acute Kidney Injury (AKI) has been replacing Acute Renal Failure (ARF). Why was the terminology changed from ARF to AKI?

1. “Kidney” is a more familiar term
2. Injury indicates dysfunction
3. It’s good to change things up every once in a while
Acute Renal Failure (ARF) vs. Acute Kidney Injury (AKI)

- More than 30 definitions of ARF can be found in the literature (Bellomo, Kellum, & Ronco, 2007)
- ARF is often secondary to a kidney insult that contributes to a change in function or structure to the organ
  - AKI was proposed as an umbrella term to encompass the gamut of ARF (Mehta, et al., 2007)
The current definition of Acute Kidney Injury (AKI) is an abrupt and sustained reduction in kidney function marked by:

1. A rise in serum urea and reduction in urine output.
2. A rise in serum creatinine and reduction in urine output.
3. A rise in serum potassium and reduction in urine output.
Acute Kidney Injury

- Acute Kidney Injury Network (AKIN) defines AKI as:
  - Abrupt and sustained reduction in kidney function marked by
    - Rise in serum creatinine
    - Reduced urine output (defined as <0.5 mL/kg/hr for more than 6 hours)
  - Rapid time course (less than 48 hours)
- Approximately 6% of all ICU patients develop some degree of AKI (Uchino, et al., 2005)
Acute Kidney Injury

• Diagnosis
  • based on clinical history (e.g., decreased urine output, hypotension, prostate enlargement)
  • characteristic laboratory findings (e.g., elevated urea and creatinine)

• The epidemiology of AKI is related to the insults causative agent, specifically:
  • Prerenal – decrease in effective blood flow to kidney
  • Intrinsic – damage to the glomeruli, renal tubules, or interstitium
  • Postrenal - consequence of urinary tract obstruction
The most common causes of AKI in the ICU include sepsis, hypovolemia, low cardiac output, major surgery, and:

1. Medications
2. Diabetic nephropathy
3. Polycystic kidney disease
4. 1 and 3
## Common causes of AKI in the ICU

<table>
<thead>
<tr>
<th>Five most common causes</th>
<th>Other common causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Sepsis (most common)</td>
<td>- Hepatorenal syndrome</td>
</tr>
<tr>
<td>- Major surgery</td>
<td>- Trauma</td>
</tr>
<tr>
<td>- Low cardiac output</td>
<td>- Cardiopulmonary bypass</td>
</tr>
<tr>
<td>- Hypovolemia</td>
<td>- Abdominal compartment syndrome</td>
</tr>
<tr>
<td>- Medications</td>
<td>- Rhabdomyolysis</td>
</tr>
<tr>
<td></td>
<td>- Obstruction</td>
</tr>
</tbody>
</table>

Common nephrotoxins that cause AKI in ICU patients

Exogenous
- Medications
  - NSAIDS
  - Antimicrobials
  - Chemotherapeutic agents
- Radiocontrast dye
- Ingestions

Endogenous
- Rhabdomyolysis
- Hemolysis (HUS/TTP)
- Tumour lysis syndrome

RIFLE criteria: Timely diagnosis of AKI in the ICU
Timely diagnosis of AKI in the ICU

- Traditionally diagnosis was reliant upon convention biomarkers used for renal function: serum creatinine (sCr) and urea (Bagshaw & Gibney, 2008)
- AKIN determined that diagnosis and classification of AKI can be based on the existing RIFLE (Mehta, et al., 2007)
- RIFLE was developed to achieve a standard definition and approach to classifying AKI
  - Validated through numerous studies (Kellum, 2008)
Modified RIFLE criteria

<table>
<thead>
<tr>
<th>GFR criteria</th>
<th>Urine output criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk</td>
<td></td>
</tr>
<tr>
<td>Increased creatinine $\times 1.5$ or GFR decrease $&gt;25%$</td>
<td>UO $&lt;0.5\ \text{ml kg}^{-1}\ \text{h}^{-1}$ $\times 6$ h</td>
</tr>
<tr>
<td>Injury</td>
<td></td>
</tr>
<tr>
<td>Increased creatinine $\times 2$ or GFR decrease $&gt;50%$</td>
<td>UO $&lt;0.5\ \text{ml kg}^{-1}\ \text{h}^{-1}$ $\times 12$ h</td>
</tr>
<tr>
<td>Failure</td>
<td></td>
</tr>
<tr>
<td>Increased creatinine $\times 3$ or GFR decrease $\geq 75%$ or creatinine $\geq 350\ \mu\text{mol/L}$ (with acute rise of $\geq 44\ \mu\text{mol/L}$)</td>
<td>UO $&lt;0.3\ \text{ml kg}^{-1}\ \text{h}^{-1}$ $\times 24$ h or anuria $\times 12$ h</td>
</tr>
<tr>
<td>Loss</td>
<td>Persistent ARF = complete loss of renal function $&gt;4$ weeks</td>
</tr>
<tr>
<td>ESRD</td>
<td>End-stage renal disease</td>
</tr>
</tbody>
</table>

- Provided the first interdisciplinary and international standardized definition for AKI
- Define stages of AKI
  - Identifies point of transition from normal compensatory function to diseased organ
- Stratify stage of AKI with mortality risk

In the past 24 hours the following changes have occurred:
- sCr is up x 1.5 from baseline (89 to 148)
- Urine output < 0.3 mL/kg/h

Based on RIFLE, what stage of AKI would the patient be in?

1. Risk
2. Injury
3. Failure
Patient would be in FAILURE stage as per the RIFLE

“The three severity grades are defined on the basis of the changes in serum creatinine or urine output, in which the worst of each criterion is used” (Kellum, 2008, p. S143).

AKI Biomarkers
AKI Biomarkers

• Three features must be considered when interpreting AKI biomarkers:
  • Patient age
  • If timing of the insult is known
  • If the underlying disease that led to AKI is a primary kidney disease

Goldstein, 2010

• Biomarkers to be considered are serum and urinary
  • Rapid and reliable increase in response to injury
  • Highly sensitive and specific to AKI
  • Applicable across different populations
  • Etiologic specificity

### Biomarkers – AMI versus AKI

<table>
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<tr>
<th>Period</th>
<th>Acute Myocardial Infarction</th>
<th>Acute Kidney Injury</th>
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<td>LDH</td>
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<td></td>
</tr>
<tr>
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<td>CPK-MB</td>
<td></td>
</tr>
<tr>
<td>1990s</td>
<td>Troponin-T</td>
<td></td>
</tr>
<tr>
<td>2000s</td>
<td>Troponin I</td>
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- Structural injury biomarkers
- Early effective therapies
- Reduction in mortality

# Biomarkers – AMI versus AKI

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<td>Serum creatinine</td>
</tr>
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- **Structural injury biomarkers**: LDH, CPK, CPK-MB, Troponin-T, Troponin I
- **Functional biomarker**: Serum creatinine
- **Early effective therapies**
- **Supportive care**
- **Reduction in mortality**
- **High mortality**

AKI Biomarkers - Serum creatinine (sCr)

• Predominant marker used for more than 50 years, with documented limitations
  • American Society of Nephrology acknowledged sCr in diagnosis of AKI held “inherent deficiencies” (McIlroy, et al., 2010, p. 998)
• Is a functional marker (Waikar, Betensky, & Boventre, 2009)
• Fluid accumulation will dilute sCr resulting in underestimation of AKI, delay of diagnosis, particularly in patients without oliguria (Mehta, 2009)
• Numerous non-renal factors influence sCr concentration (Parikh & Devarajan, 2008)
• May be a poor reflection of function as a large amount of renal mass can be lost without significant changes in sCr (Coca & Parikh, 2008)
AKI Biomarkers – What else is there?

• ABC
  
  Anything
  
  But
  
  Creatinine

• Neutrophil gelatinase-associated lipocalin (NGAL)

Need early structural biomarkers for timely recognition and treatment of AKI

AKI Biomarkers - Neutrophil gelatinase-associated lipocalin (NGAL)

- Found in low concentrations in kidney tubules (Parikh & Devarajan, 2008)
- Early protein highly overproduced after ischemic, nephrotoxic injury or inflammation (Coca & Parikh, 2008; Goldstein, 2010)
- Elevated urine NGAL levels post-operatively highly predictive of AKI (Pickering & Endre, 2009)
- With CKD, NGAL will normally be elevated
  - In cases of acute-on-chronic renal failure and AKI, levels will be very high
- In cases of fluid overload with normal or low sCr – decreased urine output with + NGAL indicates need to start supportive therapy

Case Study

- 57 year old male patient
- No significant past medical history
- Admitted via emergency with sepsis (Pneumonia) and rhabdomyolysis (found lying in bed unknown time frame)
  - Hemodynamically unstable
  - Receiving vasopressor therapy
  - Receiving invasive mechanical ventilation
  - ++ fluid balance
  - Preadmission weight 100 kg
The table below outlines the fluid intake, 24 hour urine output, serum creatinine, serum hematocrit, and cumulative 24 hour fluid balance over three days.

<table>
<thead>
<tr>
<th></th>
<th>Day one</th>
<th>Day two</th>
<th>Day three</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fluid intake</strong></td>
<td>8L crystalloids &amp; meds in ED</td>
<td>8L crystalloids &amp; meds</td>
<td>4L crystalloids &amp; meds, and 2 units PRBC</td>
</tr>
<tr>
<td><strong>24 hour urine output</strong></td>
<td>700mL (from time of foley insertion in ED)</td>
<td>500mL</td>
<td>270mL</td>
</tr>
<tr>
<td><strong>Serum creatinine</strong></td>
<td>120</td>
<td>98</td>
<td>193</td>
</tr>
<tr>
<td><strong>Serum hematocrit</strong></td>
<td>0.32</td>
<td>0.25</td>
<td>0.31</td>
</tr>
<tr>
<td><strong>Cumulative 24h fluid balance</strong></td>
<td>7,300mL</td>
<td>14,800mL</td>
<td>19,130mL</td>
</tr>
</tbody>
</table>

Intensivist says in rounds
“Let’s re-evaluate in morning. We may start RRT”
Is serum creatinine a reliable biomarker of kidney function?

1. Yes
2. No
3. Je ne sais pas
American Society of Nephrology acknowledges that creatinine used in the diagnosis of AKI holds “inherent deficiencies” (McIlroy, et al., 2010, p. 998)

- Fluid accumulation will dilute sCr resulting in underestimation of AKI severity and delaying diagnosis of AKI, particularly in patients without oliguria (Mehta, 2009).
- sCR is insensitive to small but significant changes in GFR. GFR may decrease up to 50% before any significant increase in sCr (McIlroy, et al., 2010). In other words, the horse is already out of the gate!
- Numerous non-renal factors influence sCr concentration: body weight, race, age, gender, muscle mass, total body volume, drugs, protein intake, hydration status (McIlroy, et al., 2010; Parikh & Devarajan, 2008)
AKI treatment modalities for critically ill patients
Can fluid resuscitation contribute to worse outcomes in a critically ill patient with AKI?

1. Yes
2. No
AKI treatment modalities for critically ill patients – Fluid resuscitation

• Once the cornerstone for preservation of function in the septic kidney (Bagshaw, 2006)
  • While fundamental in initial resuscitation of a critically ill patient, “a threshold may exist beyond which, after acute resuscitation, additional fluid therapy may cause harm” (Bagshaw, Brophy, Cruz, and Ronco, 2008, p. 169)

• Used to reestablish or maintain renal blood flow (Payen, et al., 2008)

• Key component to hemodynamic stability and organ perfusion; however, with AKI there is impaired free fluid removal, which contributes to fluid overload (Mehta, 2009)

• Means of treating fluid overload traditionally includes diuretics (Payen, et al., 2008)
The ideal way to treat fluid accumulation in a critically ill patient in the risk stage of AKI would be to use diuretics – AKI has not progressed too far

1. Yes

2. No
AKI treatment modalities for critically ill patients - Pharmacological

- Pharmacological intervention, to either prevent or treat AKI, has been inconclusive (Uchino, 2006)
- In AKI, diuretics may have little influence on outcome
  - RRT is more beneficial on patient outcomes in the face of multiple organ dysfunction/failure, including AKI (Payen, et al., 2008)
Renal replacement therapy (RRT) in ICU includes:

1. Intermittent Hemodialysis (IHD)
2. Sustained Low Efficiency Dialysis (SLED)
3. Continuous Renal Replacement Therapy (CRRT)
4. All of the above
IHD, SLED and CRRT are most commonly used in the ICU and each have a place. What we need to keep in mind is that they are different tools that do different jobs. We need to assign the correct tool (e.g., hemodynamic instability = CRRT)
Intermittent hemodialysis (IHD)

- Also referred to as Intermittent Renal Replacement Therapy (IRRT)
- Principle is to remove large amount of fluid and solutes with each treatment and allow for reaccumulation
- Primarily diffusive clearance with ultrafiltration
  - Efficient small molecule removal (e.g., urea, creatinine, electrolytes)
- Treatments are usually 4 hours in length, alternate days
Intermittent hemodialysis (IHD)

**Advantages**
- High clearances and rapid correction for small solutes (i.e. K⁺)
- Patient’s mobility
- Convenience
- Safety
- Cost

**Disadvantages**
- Hemodynamic tolerance
- Dialysis disequilibrium syndrome
- Fluid shifts
- Does not provide a steady-state control over azotemia, acid-base balance and fluid volume
- Not for large molecule clearance
- Microbiological dialysate safety
- Cost

SLED and SLED-D

• Primarily diffusive clearance with ultrafiltration
  • Efficient in removing small molecules (e.g., urea, creatinine, electrolytes)
• Treatments are usually:
  • 8-12 hours in length
  • Blood flow and dialysate rates are traditionally 50% of those used in IHD
  • Treatment may be on alternate days (SLED) or daily (SLEDD)
• Does not provide a steady-state control over a patient’s azotemia, acid-base balance and fluid volume
Continuous Renal Replacement Therapy (CRRT)

- Diffusive and convective clearance with ultrafiltration
- Provides a slow, gentle treatment of AKI and fluid overload
- Removes large amounts of fluid and waste products over time
  - Slow fluid removal permits for intravascular refilling
  - Generally well tolerated by hemodynamically unstable patients
CRRT

Advantages
- Hemodynamic tolerance
- Control of azotemia, acid-base balance and fluid volume easily achieved and maintained
- Low osmolality variations
- Works upwards of 24h/day
- Middle and large molecule clearance
- Sterile solutions
- Cost

Disadvantages
- Anticoagulation and bleeding risk
- Low patient mobility/immobility
- One monitor per patient for extended time period
- Workload
- Cost

IHD/SLEDD vs. CRRT – the facts

• CRRT is often better tolerated by critically ill patients with AKI in the setting of sepsis, hemodynamic instability and hypercatabolic states
• In critically ill patients, CRRT
  • delivers greater solute clearance
  • can achieve greater volume removal per day
• Both IHD and CRRT have a place in the ICU
  • Different tools for different jobs

Fluid overload monitoring and impact on the critically ill patient
The SOAP Study

Sepsis Occurrence in Acutely ill Patients

• Prospective, cohort, multi-center observational study
• To better define the incidence of sepsis and the characteristics of critically ill patients in European intensive care units
• Among 3147 patients:
  • 1177 (37.4%) had sepsis
  • Lung was the most common site of infection (68%), abdomen (22%)
• Patients with sepsis have more severe organ dysfunction and higher mortality rates
• A positive fluid balance was among the strongest prognostic factors for death

Fluid Balance as a Biomarker

Bagshaw, Brophy, Cruz & Ronco (2008)

• “We must consider early RRT in order to counterbalance fluid accumulation, particularly in those with oliguria or AKI.”

• “Timing is crucial, and RRT should ideally be initiated as early and safely as possible.”

• All critically ill patients should have an estimate of baseline ‘dry’ weight and determination daily and cumulative fluid load and balance.

Fluid Balance and AKI: the missing link for predicting adverse outcomes?

Mehta (2009)

- Fluid balance is a significant outcome predictor in critically ill patients, both with and without AKI
- Fluid accumulation will dilute serum creatinine resulting in under estimation of AKI severity
- Fluid balance has emerged as an important player in ICU management and might well be the factor that determines the balance between life and death

Does timing of treatment affect outcome?
Timing of treatment

- Patient should be considered for RRT when patient has
  - An acute fall of GFR and
  - Developed, or is at risk of, clinically significant imbalance/toxicity or volume overload
- Across the board the question remains when should we start?
  - Instead of saying do we have to do RRT today change that to is there a reason why we shouldn’t do RRT today?
- Less is known about when to stop RRT

Goals of RRT
Goals of RRT

• Shortened ICU length of stay
• Decreased mortality in critically ill patients who develop AKI in ICU
• Correction of fluid overload
• Renal recovery
Positive fluid balance and ARF

• Data extracted from the SOAP study
• Aim of the study:
  • Determine how fluid management influences mortality in critically ill patients with ARF
• In patients with ARF, mean daily fluid balance were significantly more positive among non-survivors then survivors

Mortality in relation to AKI RIFLE stage

<table>
<thead>
<tr>
<th>(% of N)</th>
<th>All N = 5383</th>
<th>Non AKI n = 1766 (32.1%)</th>
<th>Risk n = 670 (12.4%)</th>
<th>Injury n = 1436 (26.7%)</th>
<th>Failure n = 1511 (28.1%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RRT # (% of n)</td>
<td>219 (4.1%)</td>
<td>1 (0.1%)</td>
<td>0 (0%)</td>
<td>4 (0.3%)</td>
<td>214 (14.2%)</td>
</tr>
<tr>
<td>Mortality # (% of n)</td>
<td>717 (13.3%)</td>
<td>97 (5.5%)</td>
<td>59 (8.8%)</td>
<td>163 (11.4%)</td>
<td>398 (26.3%)</td>
</tr>
</tbody>
</table>

R.E.N.A.L. study end point - Mortality

44.7% 90-day mortality
Correction of fluid overload
Fluid accumulation over time: CRRT vs. IHD

Which RRT modality would potentially have higher fluid removal over the course of 7 days?

1. IHD – average 2L/treatment (4h treatment, 3 treatments/week)

2. CRRT – average 130mL/h (treating 21h/day, 7 days/week)

<table>
<thead>
<tr>
<th></th>
<th>IHD</th>
<th>CRRT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Net UF (L/treatment or mL/h)</td>
<td>2.1</td>
<td>130</td>
</tr>
<tr>
<td>Treatment duration (h)</td>
<td>4.0</td>
<td>21.0</td>
</tr>
<tr>
<td>Treatments/week</td>
<td>3</td>
<td>Daily</td>
</tr>
<tr>
<td>Cumulative UF (L/week)</td>
<td>??</td>
<td>??</td>
</tr>
</tbody>
</table>
Cumulative volume removal in ATN study*

<table>
<thead>
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<tbody>
<tr>
<td>Net UF (L/treatment or mL/h)</td>
<td>2.1</td>
<td>1.6</td>
<td>130</td>
</tr>
<tr>
<td>Treatment duration (h)</td>
<td>4.0</td>
<td>8.3</td>
<td>21.0</td>
</tr>
<tr>
<td>Treatments/week</td>
<td>3</td>
<td>3</td>
<td>Daily</td>
</tr>
<tr>
<td>Cumulative UF (L/week)</td>
<td>6.3</td>
<td>4.8</td>
<td>19.1</td>
</tr>
</tbody>
</table>

* Calculated for less intensive dose groups

Hemodynamically unstable patients are usually more tolerant of CRRT than IHD because:

1. Less incidence of hypotension than with IHD
2. The therapy more closely mimics the native kidney
3. Slow fluid removal permits for vascular refilling
4. All of the above
ATN: Frequency of hypotension in IHD group

<table>
<thead>
<tr>
<th>Hypotension during intermittent hemodialysis (IHD)*</th>
<th>Intensive Management Strategy</th>
<th>Less-Intensive Management Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of IHD treatments</td>
<td>3241</td>
<td>1836</td>
</tr>
<tr>
<td>IHD treatments reported as complicated by hypotension Number (%)</td>
<td>601 (18.5)</td>
<td>347(18.9)</td>
</tr>
<tr>
<td>Hypotension requiring initiation of vasopressor support Number (%)</td>
<td>55 (1.7)</td>
<td>27 (1.5)</td>
</tr>
<tr>
<td>Hypotension requiring discontinuation of treatments Number (%)</td>
<td>51 (1.6)</td>
<td>35 (1.9)</td>
</tr>
<tr>
<td>Hypotension requiring other intervention Number (%)</td>
<td>495 (15.3)</td>
<td>285 (15.5)</td>
</tr>
</tbody>
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Renal Recovery
Renal Recovery

CVVH dose outcome study (Ronco, et al., 2000)
- Secondary outcome was recovery of renal function 15 days after CRRT had been stopped.
- Three treatment groups: 20, 35, and 45 mL/kg/h
- 95%, 92%, and 90% of survivors in groups 1, 2, and 3, respectively, had full recovery of renal function

BEST Kidney Study (Uchino, et al., 2005)
- Beginning and Ending Supporting Therapy for the Kidney
- 13% of patients who developed ARF in ICU were dialysis-dependent at discharge
Renal Recovery

R.E.N.A.L. Trial (2009)

• Recovery of baseline renal function
• Among surviving patients, found to be 94% at 90-days
• Outcomes were related to early initiation of therapy
  • Mean time 50 hours or 2.1 days following ICU admission
• Important to note these results were achieved despite the inclusion of stage 4 chronic kidney disease patients

<table>
<thead>
<tr>
<th>Renal Recovery (%)</th>
<th>Treatment Initiation (days after ICU admission)</th>
<th>Inclusion of CKD patients</th>
<th>CRRT as Initial Therapy (% of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RENAL</td>
<td>94%</td>
<td>2.1</td>
<td>100%</td>
</tr>
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</table>

94% Renal Recovery
Day 10

• Weight is now 103 kg
• 16L of fluid removed over 6 days (130mL/h x 21h/day)
• Has now passed two consecutive spontaneous breathing trial (SBT)
• Vasopressors have been discontinued
Early initiation of RRT can reduce ICU length of stay and mortality

1. True
2. False
Characteristics of patients with ARF, stratified by time of initiation of RRT

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Early RRT n=213</th>
<th>Late RRT n=65</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICU stay, days</td>
<td>6.1 (2.5-14.8)</td>
<td>12.2 (8.0-26.5)</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>ICU mortality, number (%)</td>
<td>84 (39.4)</td>
<td>40 (61.5)</td>
<td>&lt; 0.01</td>
</tr>
</tbody>
</table>

Early = within two days of ICU admission

When RRT was initiated to correct fluid balance, the ICU LOS was reduced by 50% and mortality was ~20% less in the early group.

What is the expected outcome of this patient, related to his fluid balance at the time of initiation of RRT (19.1 L positive fluid balance at day 4)

1. Longer LOS
2. Increased risk of mortality
3. Increased probability of dialysis dependence at time of discharge
4. All of the above
5. None of the above
Early AKI recognition & timely intervention has been shown to:

Payen, et al. (2008) - reduced ICU LOS & mortality

RENAL (2009) - reduced mortality & improved renal recovery

• Showed starting therapy sooner (within 50 hours or 2.1 days of ICU admission) resulted in lower mortality and 94% renal recovery

Reduced ICU LOS & improved renal recovery = health care savings

• In the US direct costs of a septic patient in ICU $19,500 to $32,800USD with average LOS 19.6 days (Hodgin & Moss, 2008)

• Annual cost to the Canadian health care system to care for patients with severe sepsis, extrapolated from this, $36.4 to $72.9 million CAD

• The US Renal Data System’s Annual Report states in 2007, it cost $4,341 to $9,760 per month ($52,092 to $117,120USD annually) to provide IHD to a patient
Thank you for your attention

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References


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