Acute Kidney Dysfunction in the ICU
Upon completion of this module you should:

- Be able to define acute kidney dysfunction and sub-classify it into its main forms.
- Understand the clinical consequences of acute kidney dysfunction.
- Be able to list common risk factors for acute kidney dysfunction.
- Be able to identify which agents are likely to be useful and which agents are likely to ineffective or harmful in the prevention and treatment of acute kidney dysfunction.
Outline

- Epidemiology and Definitions
- Etiology/Diagnosis
- Outcome
- Prevention
- Treatment
Acute kidney dysfunction (AKD) is characterized by abrupt and sustained decline in glomerular filtration rate which leads to accumulation of urea and other toxins in the blood.

- Glomerular filtration rate = rate of transfer of protein free plasma filtrate (ultrafiltration) across the walls of the glomerular capillaries.
- In its most severe form AKD is referred to as acute renal failure.

Until recently, no standard criteria existed for diagnosis and classification of AKD. A recent international, interdisciplinary consensus panel has classified AKD according to a change from baseline serum creatinine or urine output (RIFLE criteria).

- RIFLE = Risk, Injury, Failure, Loss, ESKD
- ESKD = End-stage kidney disease
### RIFLE Criteria for Acute Kidney Dysfunction

<table>
<thead>
<tr>
<th>Category</th>
<th>GFR Criteria*</th>
<th>Urine Output Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Risk</strong></td>
<td>Increased creatinine x 1.5 or GFR decrease &gt;25%</td>
<td>UO &lt; .5 ml/kg/h x 6 hrs</td>
</tr>
<tr>
<td><strong>Injury</strong></td>
<td>Increased creatinine x 2 or GFR decrease &gt;50%</td>
<td>UO &lt; .5 ml/kg/h x 12 hrs</td>
</tr>
<tr>
<td><strong>Failure</strong></td>
<td>Increase creatinine x 3 or GFR dec &gt;75% or creatinine ≥4 mg/dl (Acute rise of ≥0.5 mg/dl)</td>
<td>UO &lt; .3 ml/kg/h x 24 hrs or anuria x 12 hrs</td>
</tr>
<tr>
<td><strong>Loss</strong></td>
<td>Persistent AKD** = complete loss of renal function &gt; 4 weeks</td>
<td><strong>ESRD</strong> = End Stage Renal Disease</td>
</tr>
<tr>
<td><strong>ESRD</strong></td>
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</tbody>
</table>

**High Sensitivity**

**High Specificity**

*www.ADQI.net*

AKD is classified according to the worst grade for each domain (creatinine or urine output). If baseline serum creatinine is abnormal, a smaller relative increase is required to reach “failure.”

<table>
<thead>
<tr>
<th>Baseline</th>
<th>0.5 (44)</th>
<th>1.0 (88)</th>
<th>1.5 (133)</th>
<th>2.0 (177)</th>
<th>2.5 (221)</th>
<th>3.0 (265)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk</td>
<td>0.75 (66)</td>
<td>1.5 (133)</td>
<td>2.3 (200)</td>
<td>3.0 (265)</td>
<td>3.8 (332)</td>
<td>---</td>
</tr>
<tr>
<td>Injury</td>
<td>1.0 (88)</td>
<td>2.0 (177)</td>
<td>3.0 (265)</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Failure</td>
<td>1.5 (133)</td>
<td>3.0 (265)</td>
<td>4.0 (350)</td>
<td>4.0 (350)</td>
<td>4.0 (350)</td>
<td>4.0 (350)</td>
</tr>
</tbody>
</table>

Creatinine is expressed in mg/dL and (mcmol/L).

Epidemiology of AKD

The prevalence of AKD among patients in the intensive care unit is not known.

- As many as 70\% of critically ill patients experience some degree of AKD.

Approximately 5\% of patients in the ICU receive renal replacement therapy (e.g., hemofiltration, hemodialysis).

- Hospital mortality in this group is 40 - 80\%.
Risk Factors for AKD

- Hypovolemia
- Hypotension
- Sepsis
  • Frequently as part of multiple organ failure
- Pre-existing renal, hepatic, or cardiac dysfunction
- Diabetes mellitus
- Exposure to nephrotoxins
  • Aminoglycosides, amphotericin, immunosuppressive agents, nonsteroidal anti-inflammatory drugs, angiotensin converting enzyme inhibitors, intravenous contrast media

*Two or more risk factors are usually present.*
Types of Acute Kidney Dysfunction

- **Pre-renal (40 - 80%)**
  - renal artery disease
  - systemic hypotension
  - Dehydration

- **Intra-renal (10 - 50%)**
  - acute tubular necrosis
  - interstitial nephritis

- **Post-renal (< 10%)**
  - obstruction

Significant overlap
# Types of Kidney Dysfunction

Biochemical indices useful to distinguish a pre-renal from a renal ARF episode

<table>
<thead>
<tr>
<th></th>
<th>pre-renal</th>
<th>renal</th>
</tr>
</thead>
<tbody>
<tr>
<td>osm u (mOsm/kg)</td>
<td>&gt; 500</td>
<td>&lt; 400</td>
</tr>
<tr>
<td>Na u (mmol/L or meq/L)</td>
<td>&lt; 20</td>
<td>&gt; 40</td>
</tr>
<tr>
<td>BUN/s creatinine</td>
<td>&gt; 20</td>
<td>&lt; 10</td>
</tr>
<tr>
<td>u/s creatinine</td>
<td>&gt; 40</td>
<td>&lt; 20</td>
</tr>
<tr>
<td>u/s osmolality</td>
<td>&gt; 1.5</td>
<td>&gt; 1</td>
</tr>
<tr>
<td>FeNa (%)*</td>
<td>&lt; 1</td>
<td>&gt; 2</td>
</tr>
</tbody>
</table>

* \((\text{u Na} / \text{s Na}) / (\text{u creat} / \text{s creat})\) \times 100

u for urinary, s for serum, Fe = fractional excretion
Etiology of (intra-renal) AKD and Typical* Urinalysis Findings

- **Acute Tubular Necrosis (ATN)** (~ 90% of AKD cases)
  - urine sediment benign, mild proteinuria/hematuria
  - muddy-brown casts

- **Allergic Interstitial Nephritis**
  - urine eosinophils
  - variable urine sediment, proteinuria and hematuria

- **Rhabdomyolysis**
  - brown urine, dip stick (+) blood but RBC (-) by microscopy
  - myoglobin (+)

- **Glomerulonephritis**
  - marked proteinuria
  - RBC casts (highly specific)

* urinalysis is often non-diagnostic
Cellular Injury and Repair in acute tubular necrosis (ATN)

Proliferation And Redifferentiation

Normal Tubular Cells

Injury

Propagation Inflammation

Injured Cells

Recovery (rapid)

De-Differentiated Cells

Apoptotic Cells

Recovery (slow)

Necrotic * Cells

Exfoliation Into the Urine

* very few necrotic cells are observed from patients with ATN
Presence of AKD is Strongly Associated with Hospital Mortality

After adjusting for differences in comorbidity, AKD was associated with a 5.5 times greater chance of death compared to matched controls without AKD.

Need for Renal Replacement Therapy (RRT) is Strongly Associated with Hospital Mortality

Metnitz et al. Intens Care Med. 2002
Metnitz et al. Intens Care Med. 2002
Prevention of AKD
Goals of therapy are to prevent AKD or need for RRT

**Effective**
- Hydration
- Prevent hypotension
- Avoid nephrotoxins

**Unknown**
- N-acetylcysteine
- Sodium Bicarbonate
- Prophylactic Hemofiltration

**Ineffective/harmful**
- Diuretics
- Dopamine
- Other renal vasoactive drugs
  - DA-1 agonists
  - PDE inhibitors
  - Ca++ blockers
  - Adenosine antagonists
  - Natriuretic peptides

Maintain hydration (Isotonic IVF)

Reducing risk from nephrotoxins
- Single vs. multiple daily doses of aminoglycosides
- Lipid complex vs. standard amphotericin
- Iso-osmotic vs. standard or “low” osmolality radiocontrast media

Maintain “perfusion pressure” (? Optimal)

<table>
<thead>
<tr>
<th>ATN Type</th>
<th>Prevention</th>
<th>Evidence Level</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radiocontrast ATN</td>
<td>no</td>
<td>Level I</td>
<td></td>
</tr>
<tr>
<td>Ischemic ATN</td>
<td>no</td>
<td>Level I *</td>
<td>No data in humans</td>
</tr>
<tr>
<td>Vascular surgery</td>
<td>no</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other settings</td>
<td>(?)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* diuretics were begun after surgery

Dopamine is not Effective

Dopamine is not Effective

- 328 patients in 23 ICUs
- Dopamine 2 µg/kg/min vs. placebo
- Peak serum creatinine: 245 ± 144 vs. 249 ± 147 µmol/L
- # with ARF: 56 vs. 56
- # needing RRT: 35 vs. 40
- ICU LOS: 13 vs. 14 days
- # of deaths: 69 vs. 66

Dopamine Can Increase Urine Output by Various Mechanisms

- Direct renal vasodilatation (DA-1 receptors)
- Increased cardiac output (β-receptors)
- Increased renal perfusion pressure (α-receptors)
- Inhibition of Na-K ATPase at the tubular epithelial cell level resulting in natriuresis

Risks of “Low-dose” Dopamine

- Bowel mucosal ischemia
- Digital necrosis
- Pro-arrhythmic
- Hypo-pituitarism
- Immune suppression
Other Vasoactive Agents

- DA-1 Agonists
  - Dopexamine
  - Fenoldapam

- Natriuretic Peptides
  - Atrial natriuretic peptide
  - Urodilatin
  - B-type natriuretic peptide

- Adenosine Antagonists
  - Theophylline
  - Pentoxifylline
  - Rolipram

- Calcium Antagonists
  - Nifedipine
  - Diltiazem
DA-1 Agonists: Fenoldapam

- Pure DA-1 effect (no $\alpha$ or $\beta$)
- Potent anti-hypertensive

Five published clinical trials ($n = 28, 31, 45, 80$ and $315$)
    - For prevention of contrast nephropathy
    - No differences between either group in any outcome
Adenosine Antagonists: Theophylline

- Adenosine decreases renal blood flow (tubular glomerular feedback)
- Contrast Nephropathy
- Four RCTs to date (n = 39, 58, 80, 100)
  - ¾ studies: hydration status is unclear
  - One study (n = 80) hydration was well defined and no difference between treatment and control
- CABG patients (n = 56)
  - No difference
Atrial Natriuretic Peptide

- Contrast Nephropathy (n = 247, 3-doses)
  - No effect

- ATN (n = 504)
  - No overall effect
  - Harm to non-oligurics; benefit in oliguria

- Oliguric ARF (n = 220)
  - No effect; hypotension

- Urodilatin (ANP analog - no hypotension)
  - No benefit
An expensive diuretic?
The next “renal dose dopamine”?
No data!!
Should be avoided in AKD given results with other natriuretic peptides.
N-acetylcysteine (NAC)

- 83 patients with chronic renal insufficiency (mean crt 2.4)
- CT scans, low-osmolal contrast agent
- N-acetylcysteine (600 mg p.o. BID) with saline hydration or placebo and saline hydration.
- Control patients: 21% (9/42) had an increase in crt > 0.5 at 48 h vs. 2% of NAC pts (P= 0.01).
- The mean crts: NAC: decreased from 2.5 +/- 1.3 to 2.1 +/- 1.3 (P < 0.001), placebo: increased

**Figure 1.** Serum Creatinine Concentrations before and 48 Hours after the Administration of Contrast Agent to Patients with Chronic Renal Insufficiency.

Mean (±SD) concentrations for the acetylcysteine group (41 patients) and for the control group (42 patients) are indicated by squares and vertical lines. To convert values for serum creatinine to micromoles per liter, multiply by 88.4.
NAC reduces the risk of AKD (increased creatinine) by 50%.

Does NAC prevent AKD or just decrease Serum creatinine?


- Healthy volunteers given NAC showed a fall in Scrt without any change in cystatin C
- NAC increases creatinine kinase activity
- Increases tubular secretion of creatinine?
Does isotonic sodium bicarbonate work better than isotonic sodium chloride solution for prevention of AKD after radiocontrast?


- n = 114, hydration alone vs. hydration plus hemofiltration
- > 25% rise in Scrt: 5% vs. 50% P < 0.001
- Need for acute RRT post-procedure: 3% vs. 25% P < 0.001
- In-hospital mortality: 2% vs. 14% P = 0.02

Results not consistent with hemodialysis studies

- Awaiting conformation
So-called “low osmolality” radio-contrast

- Iohexol: 700 - 800 mOsm
- Iodixanol: 200 - 300 mOsm (iso-osmolar)

Incidence of AKD was 3% (iodixanol) compared with 26% (iohexol) \( (p = 0.002) \).

Treatment of AKD

Goals of therapy are to prevent death, reduce complications, hasten/permit renal recovery

Effective
- Hemodialysis
- Biocompatible membranes
- More dialysis

Ineffective/harmful
- Diuretics *
- Dopamine

Unknown
- CRRT vs. IHD
- Earlier dialysis

* Diuretics are never a treatment for oliguria but are sometimes required for management of volume overload.

Cumulative Survival vs. Ultrafiltration Rate

![Graph showing cumulative survival vs. ultrafiltration rate]

- Group 1 (n=146) (Uf = 20 ml/h/Kg): 41%
- Group 2 (n=139) (Uf = 35 ml/h/Kg): 57%
- Group 3 (n=140) (Uf = 45 ml/h/Kg): 58%

*p < 0.001* for Group 2 vs. Group 1 and Group 3.
*p n.s.* for Group 3 vs. Group 1.

Cumulative Proportion Survival

Survival vs. Dialysis Dose In Intermittent Hemodialysis

Membrane Biocompatibility

<table>
<thead>
<tr>
<th>1st Author</th>
<th>N</th>
<th>Year</th>
</tr>
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<tbody>
<tr>
<td>Schiffl</td>
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</tr>
<tr>
<td>Kurtal</td>
<td>57</td>
<td>1995</td>
</tr>
<tr>
<td>Assouad *</td>
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<td>1996</td>
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<tr>
<td>Neveu</td>
<td>166</td>
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<tr>
<td>Albright *</td>
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<td>1998</td>
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<td>Himmelfarb</td>
<td>153</td>
<td>1998</td>
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<tr>
<td>Jorres</td>
<td>160</td>
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</tr>
<tr>
<td>Gastaldello</td>
<td>159</td>
<td>2000</td>
</tr>
<tr>
<td>Combined</td>
<td>857</td>
<td></td>
</tr>
<tr>
<td>Excluding</td>
<td>698</td>
<td></td>
</tr>
</tbody>
</table>

* - Abstracts

Odds Ratios for Survival

Continuous vs. Intermittent RRT

Insufficient evidence from published studies to determine which therapy is best.

However, CRRT appears to be superior under most sets of assumptions.

CRRT v IHD

• Most recent study (NEJM May 2008)
  – Multicenter, RCT of CRRT v IHD v SLED
  – Also compared “low dose” v “moderate dose” CRRT (20 cc/kg v 35 cc/kg)
  – No difference in renal recovery or mortality
  – BUT:
    • Actual dialysis dose delivered was variable
    • 35cc/kg may not be enough. > 40cc/kg?
Diuretics: Effects on outcome (small RCTs)

- 66 patients randomized to receive furosemide (1.5 - 6.0 mg/kg)
- No significant differences in recovery or need for HD.


- 58 patients randomized to single dose (1g) vs. continued dosing of furosemide (3g/day).
- Oliguria was reversed in 2/30 vs. 24/28.
- No differences in mortality, renal recovery, or need for RRT.
- Permanent deafness in one patient.

## Treatment: Diuretics

**Diuretics: Effects on outcome (large observational studies)**

- 4-center, retrospective analysis of patients referred for nephrology consults (1989 - 1995; n = 552)
  - With adjustments for co-variates and propensity score, diuretic use was associated with:
    - Significantly increased risk of death or non-recovery of renal function (odds ratio 1.77; 95% CI 1.14 - 2.76)
  

- 52-center, prospective inception cohort of ICU patients (n = 1743)
  - No differences in mortality, or renal recovery, even after adjustment for the same co-variates and propensity score
    - Odds ratio 1.22 (p = 0.15)
  - However, no benefit associated with diuretics either!

Conclusions

- AKD is a common ICU syndrome.
  - As many as 70% of ICU patients develop AKD.
  - Approximately 5% of ICU patients receive RRT.

- AKD in the critically ill carries a very high mortality, and current treatment is disappointing.

- Inflammation likely plays a significant role in the development of AKD.
For Prevention of AKD in the ICU:

- Avoid nephrotoxins, hypotension, and dehydration.
  - Grades B - D for various options
- Don’t use diuretics, dopamine, or other vasoactive drugs.
  - Grade A +
- N-acetylcysteine + fluids for high-risk patients undergoing radio-contrast studies.
  - Grade A -
- Consider bicarbonate-based fluids for prevention of radio-contrast induced AKD.
  - Grade C
Conclusions/Recommendations

For Treatment of AKD in the ICU

- Avoid further injury from nephrotoxins, hypotension, and dehydration.
  - Grades B - D for various options
- Don’t use dopamine or other vasoactive drugs.
  - Grade A +
- Avoid diuretics.
  - Grade D
- Use biocompatible membranes.
  - Avoid cuprophane (Grade A -)
  - Avoid all cellulosic membranes (Grade C)
- Use 35 ml/kg/min for CRRT and possibly daily dialysis for IRRT.
  - Grade C
- Use CRRT?
  - Grade D
Hepatorenal Syndrome
  • profound renal vasoconstriction
  • low RBF and low GFR
  • marked Na and water retention
  • “pre-renal” urine chemistries
  • bland pathology and urine sediment
  • Type I (rapid renal failure) and Type II (diuretic-resistant ascites)
Hepatorenal Syndrome

Management
- low Na diet and diuretics
- paracentesis
- shunt
- liver transplant
- aquaretic agents (? effectiveness)
  - AVP - V2 receptor antagonists
  - selective kappa-opioid agonists
- vasopressors
Pathophysiology of HRS

HRS = arterial hypotension, very low SVRI, very high renin, NE and ADH, and vasoconstriction in non-splanchnic arterial vascular territories, including the kidneys, the brain, and the muscle and skin.

Splanchnic circulation: marked arterial vasodilation = impairment in circulatory function and the homeostatic activation of the endogenous vasoconstrictor systems.

Drug treatment (limited efficacy data)

- Ornipressin, terlipressin, and vasopressin
- Midodrine (an oral alpha-agonist)


Case 1

A.B. is a 53-year-old male with a past medical history of “poorly controlled” hypertension (taking an ACE inhibitor and a Ca++ channel blocker). He weighs 80 kg and presents with a two-day history of fever and cough, and his chest radiograph shows an RLL infiltrate. His BP on admission is 88/54, and he is given IV fluids (saline) and antibiotics (ampicillin sulbactam).

His admission labs show a serum creatinine of 1.5 mg/dL (133 mcmol/L) and his BUN is 42. Six months ago, his serum creatinine was 1.2. Over the next six hours his urine output is 20 - 30 ml/hr. He is given 2L of 0.9% saline and 500 ml of 5% hetastarch. His BP improves to 110/60 and his pulse decreases from 128 to 109. He is admitted to the ward and you are called by his nurse for continued low urine output.
Case 1 (cont.)

The patient’s UO has been < 0.5ml/kg/hr for more than 6 hours. This may indicate AKD (“risk” category for urine output by RIFLE criteria), but it might just as easily represent inadequate circulating blood volume or (much less likely) an obstructive uropathy.

You place a Foley catheter and there is only 20 ml of urine. While this does not rule out obstructive uropathy, it makes it very unlikely. Additional testing (e.g., renal ultrasound) might be indicated if there is still a diagnostic question but pre-renal or intra-renal disease is far more likely.

You send the urine for electrolytes and this reveals a uNa of 10 mmol/L, uCr of 50 mg/dL, and you calculate a fractional excretion (FE) of Na of 0.5%. These results are consistent with pre-renal disease but urine studies are not themselves diagnostic.

Examination of the urine reveals no WBCs or casts. These findings make interstitial or glomerulular nephritis very unlikely. The absence of muddy brown casts do not exclude the diagnosis of ATN.
You also send a repeat BUN and serum creatinine which are 40 and 1.8 mg/dL. The ratio of BUN/creatinine > 20 is consistent with (but not diagnostic of) pre-renal disease.

You decide to give additional fluid (1L 0.9% saline) over the next hour, but the urine output remains low and the BP decreases to 90/55.

You now need to establish the etiology of the persistent hypotension. Possibilities include: hypovolemic (even though the patient has received 3.5 L of fluid), septic (distributive), cardiogenic, and obstructive. Options for determining the etiology range from noninvasive (e.g., echocardiography) to invasive (e.g., pulmonary arterial catheterization). No technique is completely failsafe but if cardiac output is increased, the diagnosis must be distributive.
You determine that the cardiac output is increased and you also measure an arterial lactate (2.7) and mixed venous oxygen saturation (72%). You also determine that the central venous pressure is 14 mm Hg. These findings make hypovolemia unlikely.

At this point, even though the mean arterial pressure is 62 mm Hg, you are concerned that the patient’s BP is too low and that he may not have adequate perfusion pressure for his organs (including the kidneys). This is a significant concern, especially in a chronic hypertensive. Atherosclerotic disease is likely and a decreased blood pressure may result in insufficient flow. The slight elevation in the arterial lactate also suggests this diagnosis.

This scenario is further supported by this combination of urine chemistries (pre-renal) and systemic hemodynamics (hyperdynamic). You decide to increase the mean arterial pressure to 70 mm Hg using norepinephrine.
The patient is given activated protein C and his adrenal axis is evaluated using a short ACTH stimulation test (his response is normal).

Over the course of the next 12 hours, you maintain his mean arterial pressure > 70 mm Hg with 0.02 – 0.04 mcg/kg/min of norepinephrine. His urine output gradually increases, and his central venous pressure falls to 8 mm Hg. You administer additional fluids (lactated Ringers this time to avoid giving additional saline, which may cause acidosis) and continue supportive care.

The next day, the patient’s Crt increases to 2.2 (BUN falls to 32). Repeat urine electrolytes show an Na of 35 and the FeNa is 1.8. Muddy brown casts appear in the urine. The next day the serum creatinine decreases to 2.0 and his blood pressure improves. You discontinue the norepinephrine and by the next day he is requiring antihypertensive therapy. He makes a complete recovery.
C.D. is a 64-year-old female with a history of hypertension, 3-vessel coronary artery disease, and poor left ventricular function (ejection fraction: 20%). She weighs 80 kg and undergoes coronary arterial revascularization. The surgery is uneventful but she requires fluids and vasoactive medications (epinephrine and dobutamine) to come off of cardiopulmonary bypass.

Her initial postoperative care is unremarkable except that she a borderline urine output 30 - 40 ml/hr and her blood pressure is very labile.

Her admission labs (drawn 24 hours before surgery) showed a serum creatinine of 1.5 mg/dL (133 mcmol/L). Over the first 24 hours after surgery, she makes 200 mL of urine. Her serum creatinine increases to 2.0 mg/dL (177 mcmol/L). She is maintained on vasoactive medications but is weaned from mechanical ventilation and extubated. Her cardiac function remains poor but cardiac index is 2.2 on epinephrine and dobutamine. She has not received any nephrotoxic agents. Urine chemistries and microscopy are consistent with a diagnosis of ATN.
The following day her serum creatinine increases to 3.0 mg/dL (266 mcmol/L) and her BUN increases to 65 mg/dL. She has made 300 mL of urine in the last 24 hours, and her total fluid intake has exceeded all output by 11L since the surgery. Her weight is now 90 kg and she has edema on physical exam.

Furosemide is administered but she does not respond. The next day the creatinine is 4.0 mg/dl and she is started on continuous veno-venous hemofiltration at an ultrafiltration rate of 35 ml/kg/hr based on her admission weight. Initially 100 mL of fluid are removed per hour and this is increased to 150 mL/h, but her blood pressure becomes unstable, and the removal rate is returned to 100.

Over the course of the next five days 8L of fluid are removed, and her heart function improves such that all vasoactive medications are discontinued. She is converted to intermittent dialysis and is discharged from the ICU.

A week later renal function gradually recovers, and one month later her serum creatinine has returned to baseline.