

## The EM Educator Series

### The EM Educator Series: The Sick ESRD Patient

Author: Alex Koyfman, MD (@EMHighAK) // Edited by: Brit Long, MD (@long\_brit) and Manpreet Singh, MD (@MprizzleER)

#### Case 1:

A 61-year-old male with history of ESRD presents with chest pain during his dialysis session. His session was stopped, and EMS was called. He is currently hemodynamically stable.

#### Case 2:

A 55-year-old female is brought in for altered mental status. She has a history of ESRD. On exam, she is hypotensive, with GCS 10.

#### Questions for Learners:

1. What neurologic complications can occur in patients with ESRD?
2. What cardiopulmonary complications can occur in patients with ESRD?
3. What infectious complications can occur in patients with ESRD?
4. What should you consider in obtaining vascular access in patients with ESRD?
5. What are complications with dialysis fistulas?
6. What are considerations in treatment of hyperkalemia in this population?
7. How can labs be affected by ESRD? How about troponin?
8. What medication considerations are recommended in these patients?

#### Suggested Resources:

- Articles:
  - emDocs Cases - [Sick ESRD Patient](#)
  - emDocs – [Management of the Sick Dialysis Patient](#)
  - REBEL EM – [ECG changes with hyperkalemia](#)
  - emDocs – [Troponin in ESRD and CKD](#)
  - emDocs – [Sepsis Comorbidities Management Strategies](#)
  - emDocs – [Managing Fistula Complications](#)
  - emDocs Cases – [Updates in Management of Hyperkalemia](#)
- PMID:
  - [American Journal of Emergency Medicine](#)

## Answers for Learners:

### 1. What neurologic complications can occur in patients with ESRD?

A variety of neurologic complications are common. One of the most common is uremic encephalopathy. A specific level of BUN or uremia is not reliable for diagnosis, and uremic encephalopathy is a diagnosis of exclusion. A variety of other conditions must be considered before latching onto this diagnosis. For the altered patient, these conditions include intracerebral hemorrhage (ICH) or ischemic stroke. Rapid blood glucose assessment, BMP, CBC, TSH, ECG, and Head CT noncontrast should be obtained.

1. **Uremic encephalopathy** – Due to over 70 toxins and neurotransmitter imbalance, with increase in those missing dialysis. Primarily affects those with GFR < 15 mL/min. Older patients and those with comorbidities typically demonstrate more severe symptoms. Asterixis may be seen. Early signs consist of mood swings, weakness, and irritability, transitioning into tremor, asterixis, lethargy, and even coma. Neurologic deficits may change during the disease course. Treatment includes dialysis, though neurologic changes may persist for several days after dialysis.
2. **Dialysis Disequilibrium Syndrome (DDS)** – Due to serum osmolality changes in HDS, resulting in diffusion of water into the CNS. Risks include first HDS treatment, BUN > 60 mmol/L (175 mg/dL), CKD, acidosis. Treatment includes slowing or stopping HDS. Symptoms usually resolve after several hours. Symptomatic treatment warranted for nausea and vomiting. If seizing, provide a hypertonic solution (mannitol or 5ml 10-20% NaCl).
3. **ICH/ischemic stroke** – ESRD patients are at high risk for stroke, and if stroke occurs, ESRD is a poor prognostic marker. Subdural hematoma occurs 10-20X more frequently. Altered mental status is more common in patients with ICH, and patients may have focal deficit. Ischemic stroke is also more common in this population, with worse outcomes. CT head, ECG, coagulation panel, CBC, RFP are needed. Thrombolytics can be provided for ischemic stroke if contraindications are not present. If ICH is present, reversal of anticoagulation may be needed, along with neurosurgical consultation.

### 2. What cardiopulmonary complications can occur in patients with ESRD?

**Cardiopulmonary:** These patients are at significant risk for cardiopulmonary complications, with cardiovascular disease accounting for over 40% of mortality. Electrolyte abnormalities, LVH, hypercholesterolemia, and atherosclerosis predispose to increased risk of ACS, and ESRD patients are also at increased risk for severe arrhythmias and heart failure, with many patients receiving an implantable cardiac device.

1. **Pericarditis/Effusion/Tamponade** – Pericarditis classically presents with fever, sharp chest pain that radiates to the trapezius, chest pain that worsens in supine position, and pericardial friction rub (more common in uremic pericarditis) on exam. However, this rub is not always present. Patients may present with dyspnea, and pericardial effusion may occur in 20% of patients. Fluid from uremic pericardial effusion is typically sterile with fibrin. BUN is often greater than 60 mg/dL, and the ECG may not reveal the normal stages of pericarditis. Chest X-ray may show enlarged cardiac size if the effusion is longstanding. Ultrasound (US) is an important measure for these patients to evaluate for evidence of tamponade. Poor prognostic findings warranting admission include hemodynamic instability, WBC > 13x 10<sup>9</sup>/L, fever, effusion > 2 cm, poor social situation, and poor compliance with dialysis. Patients who are hypotensive require immediate assessment for tamponade, with IV fluid bolus to enhance preload. Pericardiocentesis with IV

fluid is required in the peri-arrest state. For patients with pericarditis who may be appropriate for discharge with no effusion, acetaminophen should be considered, with dialysis.

2. **ACS** – This accounts for the highest percentage of deaths in these patients. Patients may not present typically with chest pain, but nausea, shortness of breath, and weakness, and the ECG at baseline may demonstrate LVH or other abnormalities. Troponin may be elevated at baseline, but a change from baseline or elevation (by 20%) on repeat assessment is suggestive of NSTEMI.<sup>4,7,9,33-38</sup> These patients should be given aspirin and standard ACS medications. No dose adjustment is needed for aspirin, clopidogrel, unfractionated heparin, or thrombolytics, but lovenox dosing does require adjustment. If STEMI is present, PCI or thrombolytics is needed.
3. **Pulmonary Edema** – Fluid overload is the most frequent cause of dyspnea in ESRD and is most commonly due to missed HDS session. However, ACS or CHF may also result in overload. Rapid IV access, ECG, monitor, and oxygen are required. US of the heart and lungs can be beneficial (cardiac function, presence of B lines, IVC assessment). Immediate resuscitation is needed, with nitroglycerin (SL or IV) and noninvasive positive pressure ventilation if in respiratory distress. HDS is required, and phlebotomy can function as a bridge to HDS if other measures fail.
4. **Air Embolism** – Venous air embolism is the most common form and may occur during access placement, removal, or during HDS session. Air typically travels to the right side of the heart, lodging in the pulmonary circulation, but arterial embolism may occur in the setting of R to L shunt. These patients present with sudden respiratory distress, chest pain, hypotension, neurologic abnormalities, livedo reticularis, crepitus over involved vessels, and a mill wheel murmur (churning sound heard throughout the entire cardiac cycle). ECG, CK, CBC, renal function, VBG, US, and chest X-ray are needed. Thrombocytopenia and elevated CK may be found, and chest X-ray may show focal atelectasis, oligemia, or pulmonary artery enlargement. Rapid assessment of the ABCs with high flow oxygen is needed. The patient should be placed in the left lateral decubitus or Trendelenburg position for venous air embolism or supine position for arterial embolism. The catheter should be clamped and HDS discontinued. Aspiration of air from the RV is present in case report literature. Hyperbaric oxygen therapy can be used for end organ dysfunction.

### 3. What infectious complications can occur in patients with ESRD?

**Infectious:** Patients with severe renal disease are at high risk for infection, with catheter based infections the most common cause of bacteremia and sepsis (followed by lower respiratory tract infection). As renal function decreases, the risk of infection drastically rises due to decreased T-cell activation and decreased leukocyte function. The typical infections like pneumonia and cellulitis may occur, but interestingly these patients are at high risk for *Clostridium difficile* infection, recurrence, and mortality. UTI may also develop, even without urine production. Catheter-related infections possess an incidence of 5.5 episodes per 1000 catheter days. Up to 1/3 are due to Gram-positive cocci, with 20% polymicrobial. The risk of infection due to MRSA is 100 times greater in ESRD patients on HDS, with catheter at higher risk than fistula (8X). Patients with sepsis from suspected access site infection warrant broad-spectrum coverage: vancomycin at normal loading dose (20 mg/kg IV) AND third generation cephalosporin such as ceftazidime 2 g IV or gentamicin 1-2 mg/kg IV (not exceeding 100mg). Cultures should be obtained from the suspected site and a separate peripheral site. Infections in other body locations from an access site are considered metastatic infections, such as endocarditis.

What about the hypotensive septic patient? How much fluid should you provide? The Surviving Sepsis Campaign recommends 30 mL/kg in sepsis. Fluid assessment in ESRD patients is not easy, but these

patients in sepsis are intravascularly depleted. Unfortunately, literature suggests patients with ESRD and dialysis dependency do not receive the recommended 30 mL/kg (commonly less than 1L total in 6 hours). Patients who do receive 30 mL/kg or greater do not demonstrate adverse events associated with IV fluid resuscitation. These patients should receive IV fluids with repeat assessments.

#### 4. What should you consider in obtaining vascular access in patients with ESRD?

Avoid peripheral IVs on the same side of the maturing or future hemodialysis access site. However, if the patient has the potential to be hemodynamically unstable, it is paramount to obtain vascular access quickly.

#### 5. What are complications with dialysis fistulas?

**Vascular Access:** In the U.S., close to 400,000 patients with chronic kidney disease use HDS, and approximately 25% of these patients are hospitalized annually due to access site complications, dependent of the type access. If HDS is to be used, a fistula is the optimal form for access. ESRD patients are at high risk for bleeding due to uremia (vWF factor disruption) and normocytic normochromic anemia. Coagulation studies are usually normal, and stage of ESRD does not correlate with rates of hemorrhage. Physicians should assess access site color, swelling, warmth, bruit, and thrill, as well as distal extremity neurovascular status.

1. **Bleeding** – Hemorrhage is multifactorial, with risks including platelet function (uremia) and anticoagulation (heparin used during HDS). Other risks include infection, stenosis, and aneurysm. Heparin's half-life approximates 1.5-2 hours. These patients require immediate assessment of hemodynamic status, with rapid control of the bleeding site. IV access, CBC, type and screen, renal function panel, and coagulation panel are recommended. Start with direct pressure to the site for 5-10 minutes. A single suture is useful for linear tear across the access site. Topical hemostatic agents are useful in combination with direct, focal pressure (rather than a large, bulky dressing). To improve platelet function, desmopressin 0.3 mcg/kg IV should be used, which can decrease hemorrhage in 50% of patients. If anticoagulation from heparin is a concern, 1 mg of protamine IV per 100 units heparin is recommended. HDS corrects uremic platelet dysfunction in up to 85% of patients. Further strategies include cryoprecipitate (to replace fibrinogen), and estrogen 25 mg IV. If these measures fail, a tourniquet can be applied.
2. **Aneurysm/Pseudoaneurysm** – These commonly occur due to frequent cannulation, which weakens the vessel wall. Incidence ranges from 5-6%, and stenosis drastically increases the risk. Aneurysms present with pain, motor/sensory dysfunction, skin erosion, and hemorrhage, while pseudoaneurysm is more likely to present with infection or bleeding. Doppler US and vascular surgery consultation are needed, with operative repair required.
3. **Thrombosis/Stenosis** – This complication is the most common problem in HDS access sites, with over 75% of patients with grafts experiencing this complication within the first year of placement. Thrombosis/stenosis also increases the risk of aneurysm and pseudoaneurysm. Patients may have extremity edema, increased vein formation, and change in the bruit/thrill. Pain and absence of bruit/thrill are common with thrombosis. Doppler US and vascular surgery consultation are needed. Treatment (thrombolysis versus surgical thrombectomy with or without angioplasty) is not required emergently, but should occur within 24-48 hours.
4. **Infection** – We covered access site infection briefly above, but this section will give you a little more of a deep dive. Infection is most common within the first 6 months of placement and is

more common in central venous access sites compared to AV fistula (2-5% rate over fistula lifetime). Close to 10% of grafts will undergo infection, with infection most commonly from *Staph aureus*, *Staph epidermidis*, and gram-negative bacteria. This is the most common cause of graft loss. Evaluate for erythema, induration, and warmth of the access site; however, due to altered immune function, patients may not demonstrate these findings but rather systemic symptoms such as myalgias and fever. Close to 10% of patients will experience an infection at another site (endocarditis, osteomyelitis, epidural abscess, etc.). Cultures should be obtained from the site and a separate peripheral site, with the catheter blood culture often showing a 4-fold higher colony count. Doppler US of the graft, CBC, lactate, renal function, and cultures are required with admission. Vancomycin (20mg/kg) and gentamycin (100 mg) or ceftazidime (2 g) IV are recommended.

**6. What are considerations in treatment of hyperkalemia in this population?**

Effect	Agent	Dose	Onset	Duration
Membrane Stabilization	Calcium Gluconate (10%)	10mL IV over 10 min	Immediate	30 – 60 minutes
	Hypertonic (3%) Normal Saline	50mL IV push	Immediate	Unknown
Shifters	Insulin (Short Acting)	10 units IV push with 25 – 40 g dextrose (50% solution)	20 minute	4 – 6 hours
	Albuterol	10 – 20 mg in 4 mL of Normal Saline nebulized over 10 minutes	30 minute	2 hours
Excreters	Furosemide	40 – 80 mg IV x1	15 minute	2 -3 hours
	Sodium Bicarbonate	150mmol/L IV at variable rate	Hours	Duration of Infusion
	Sodium Polystyrene Sulfonate	15 – 30 g in 15 – 30 mL (70% sorbitol orally)	> 2 hours	4 – 6 hours
Definitive	Hemodialysis	-----	Immediate	3 hours

Via REBEL EM

- 1 g Calcium Chloride (CaCl) = 3 g Calcium Gluconate:** Bryan Hayes (@PharmERToxGuy) had a great post on [Academic Life in EM](#) on myths associated with different calcium regimens.
- Hypertonic (3%) Saline:** Should be restricted to patients with hyponatremia and concurrent hyperkalemia. Its effects have not been established in eunatremic patients.
- Insulin:** 10 Units of regular insulin + dextrose (25 g as 50% solution) lowers plasma potassium by about 0.6 mmol/L. Be careful as a single bolus of 25 g of dextrose has been shown to be inadequate ion prevention of hypoglycemia at 60 minutes.
  - Consider lowering amount of insulin or checking blood sugar closely in ESRD patients.

4. **Beta Agonists:** 10 mg and 20 mg of inhaled albuterol decreased serum potassium by 0.6 mmol/L and 1 mmol/L, respectively. Mild tachycardia is most common side effect.
5. **Bicarbonate:** Bolus injection of sodium bicarbonate has not been studied. The dogma of bolus bicarbonate was derived from studies using a prolonged (4 – 6 hr) infusion of bicarbonate which does not reduce plasma potassium in patients with dialysis-dependent renal failure. Infusion sodium bicarbonate over 4 – 6 hours may have some benefit in excretion of serum potassium in the setting of metabolic acidosis.
6. **Exchange Resin:** The onset of action is very long (2 hours to onset, and 6 ours to maximum effect) and there have been case reports of patients developing colonic necrosis. Also there have been studies that show no statistical benefit at 4, 8, and 12 hours [6].
7. **Hemodialysis:** Definitive treatment of hyperkalemia. Watch out for rebound hyperkalemia which can occur in 30% of patients an hour after dialysis.

## 7. How can labs be affected by ESRD? How about troponin?

- Electrolyte dearrangements, including potassium, magnesium, and phosphorous, as well as BUN in ESRD patients.
- Platelet dysfunction is a functional issue rather than a laboratory finding.
- Anemia of chronic disease
- CKD/ESRD patients are **high risk patients for ACS/CAD.**
  - An elevated troponin is **not meaningless in these patients and confers higher risk of cardiovascular morbidity.**
  - **Always trend troponins and get consultants involved** early as indicated.
  - Consider **other causes of elevated troponins in this patient population.**

## 8. What medication considerations are recommended in these patients?

ESRD patients typically have some degree of anemia due to a **deficiency in erythropoietin** produced by the kidney. **If blood transfusion is required, it should occur during dialysis** to correct for the extra volume as well as potassium changes.

If analgesia is required, nonsteroidal anti-inflammatory agents should be avoided. **Opioids such as morphine and hydromorphone should be started at a dose reduced by approximately 50%.** Most antibiotics are efficacious and may require dosage adjustment, but **nitrofurantoin should be avoided.** If the ESRD patient **requires contrast for radiographic evaluation, the ED physician can order the necessary studies without concern for renal injury.**

If a decision is made to intubate a HD patient, **succinylcholine should be avoided** if the potassium level is not yet known. Succinylcholine causes potassium efflux after binding to skeletal nicotinic receptors, thus causing a rise in potassium. While this rise in potassium is usually inconsequential, this rise from succinylcholine can cause dysrhythmia in patients with hyperkalemia. A previous emDocs article talks about this further, as well as some other advantages to using rocuronium as your RSI paralytic of choice.