COMPLICATION DURING HD

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Common Complications during Dialysis

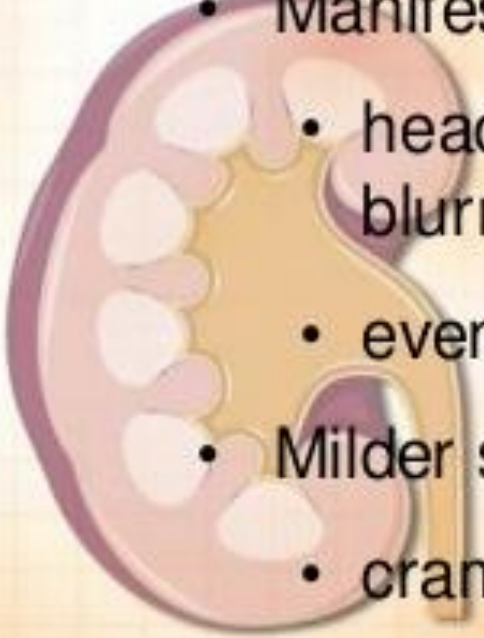
Hypotension	25-60% of treatment sessions
Cardiac arrhythmias	5-60% (usually asymptomatic)
Cramps	5-25%
Nausea & vomiting	5-15%
Headache	5-10%
Back pain	2-5%
Chest pain	2-5%
Itching	1-5%
Fever	1%

Less Common Complications

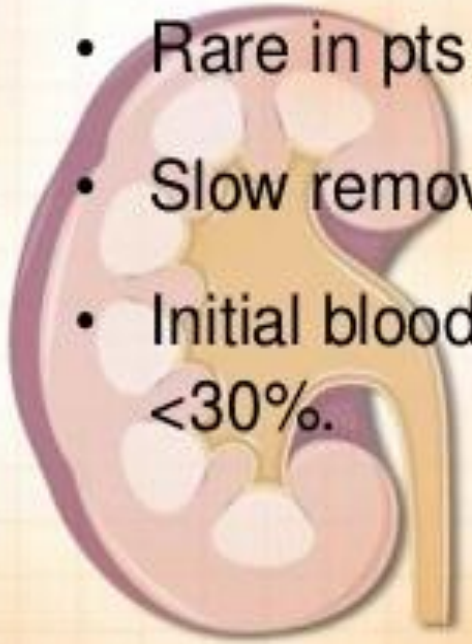
- Air embolism
- Seizures
- Hemolysis
- Severe dialysis disequilibrium
- 1st use syndromes
- Acute urticaria
- Cardiac tamponade

Dialysis Disequilibrium Syndrome

- Occur in ARF or CRF if blood urea levels are reduced too fast, in those with very marked uremia, or pre-existing alterations in mental state
- Manifestations:
  - headache, nausea, disorientation, restlessness, blurred vision, asterixis, fits, coma
  - even death, occurring during or after dialysis
- Milder symptoms :
  - cramps, nausea, dizziness



- Probably caused by cerebral edema due to osmotic influx of water into the brain after removal of urea by dialysis, before equilibration across cell membranes occurs.
- Cerebral acidosis may play a role
- Rare in pts initially dialyzed for 2 h at low BFR
- Slow removal of urea minimizes risk
- Initial blood urea reduction during a 1st dialysis should be <30%.



Prophylactic **phenytoin**

- Used in pts at highest risk (severe uremia, abnormal mental state)
- 1000 mg loading dose, then 300 mg/day

- Symptoms are self-limiting over a few hrs.
- Pts with severe fitting can be treated with IV **mannitol** (10-15 g) or **hypertonic saline** (5 ml of 23%).

Hypotension

- Occurs in 15-50% of HD sessions
- Can be episodic or persistent (less common)
- More frequent in pts with lower body mass, cardiac disease
- Intradialytic hypotension forms part of a vicious cycle maintaining HPN & fluid overload
  - The response to a drop in BP is frequently to infuse NS or hypertonic saline, leaving the pt hypertensive, necessitating further use of antihypertensive agents, which in turn worsen intradialytic hypotension by inhibiting appropriate reflexes (tachycardia, vasoconstriction)
- Other s/sx: N&V, cramps, yawning

Common Causes of Hypotension

Patient-specific causes
Diabetes
Autonomic neuropathy
Reduced cardiac reserve (especially LVH & diastolic dysfunction)
Arrhythmias
Poor nutritional state
High wt gain
Ingestion of food during HD (increased splanchnic venous pooling)
Antihypertensive agents impairing cardiac stability & reflexes
Septicemia
Release of adenosine during organ ischemia (e.g. induced by hypotension; adenosine is a vasodilator & inhibits norepinephrine release)

Common Causes of Hypotension

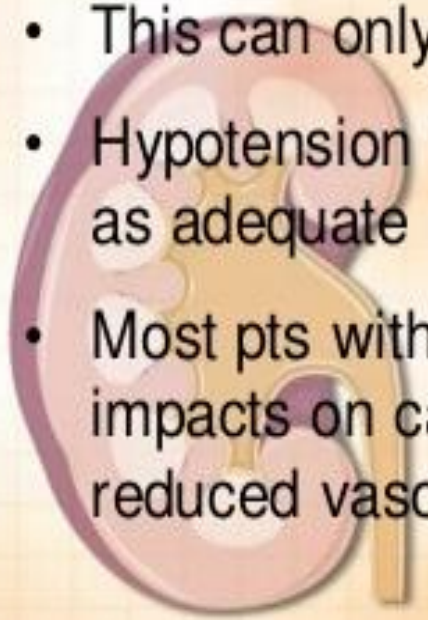
Treatment-specific Causes
Rapid fluid removal (high UF rate)
Antihypertensive agent use
Rapid reduction in plasma osmolality (leading to water movement from the vascular into interstitial compartment)
Warm dialysate
Low sodium dialysate
Low dialysate osmolarity
Use of acetate as buffer (a vasodilator)
Bioincompatibility

Less Common Causes of Hypotension

- Pericardial effusion or tamponade
- Reactions to dialysis membranes
- Increased dialysate magnesium
- GI bleeding
- Disconnection of blood lines
- MI
- Hemolysis
- Air embolism

Hypotension from excessive UF

- During UF, as water is removed from the vascular compartment, blood volume is maintained by movement of water from tissues (refilling).
- This can only occur until the true dry wt is achieved.
- Hypotension will be induced if UF is too rapid or excessive, as adequate refilling will not occur
- Most pts with ESRD also have diastolic dysfunction, which impacts on cardiac output especially under conditions of reduced vascular filling



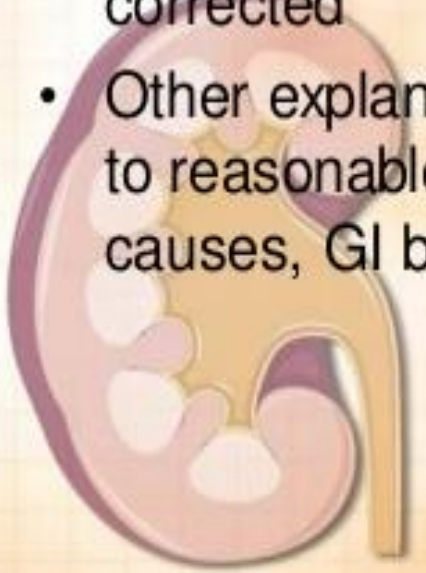
Factors contributing to uncontrolled UF

- HD machines without volumetric control of UF can lead to rapid fluctuations in UF rates
- Pts who drink too much between HD sessions have excessive (unachievable) UF requirements during the HD
  - This is usually driven by salt consumption (often hidden in foods).
  - Salt intake of 0.5 g/day will lead to a 1.5 kg wt gain on average in a 70 kg anuric pt
  - Wt gain should be 1 kg/day
- Excess BFR is a rare cause of hypotension
- Too low EDW will lead to hypotension. This occurs especially during recovery from an acute illness when lost muscle wt is being recovered.

Management of Hypotension

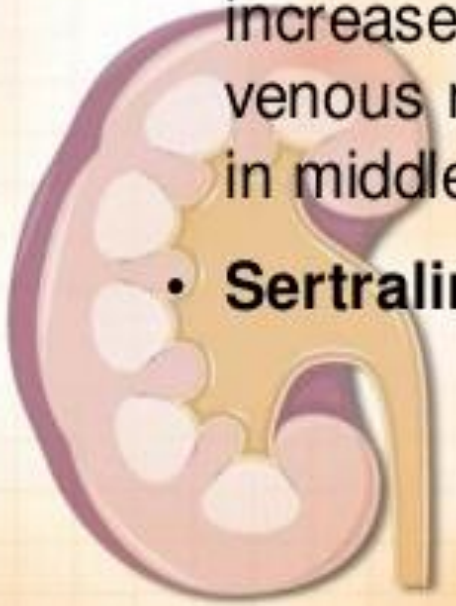
- Episodes of hypotension are uncomfortable & distressing for pts
- Hypotension lead to morbidity, contribute to cardiovascular mortality.
- Immediate management requires volume resuscitation:
  - place pt head-down
  - administer 100 ml bolus of NS (some units use 10 ml of 23% saline, 30 ml 7.5% saline, 50 ml 20% mannitol or albumin solutions)
  - reduce UF rate to zero
  - if BP does not normalize rapidly, further saline may be given

- Hypertonic saline may increase thirst, prevent achievement of dry wt, worsen fluid overload
- Albumin is very expensive
- In most cases hypotension is due to excess UF, rapidly corrected
- Other explanations should be sought if BP does not respond to reasonable saline replenishment, especially cardiac causes, GI bleeding, sepsis



- If hypotension occurs repeatedly, review:
  - Dry wt (too low?)
  - Use of short-acting antihypertensive agents pre-HD (give drugs post HD; doesn't apply to most modern long-acting agents)
  - UF rate
  - Wt gains between sessions (counsel pt about salt, limit wt gain to 1 kg/day, look for hidden fluids (soup, salt intake)
  - Dialysate sodium (keep above plasma sodium)
  - Use bicarbonate not acetate dialysate
  - Lower dialysate temperature to 34-36°C (makes some pts feel uncomfortable)
  - Increase Hb
  - Avoid food intake during HD

- If all else fails the following drug treatments can sometimes help:
  - **Carnitine** 20 mg/kg/treatment IV
  - **Midodrine** 2.5-30 mg 30 min before dialysis; increases peripheral vascular resistance, increases venous return & cardiac output; can give second dose in middle of session;
  - **Sertraline** 50-100 mg/day PO



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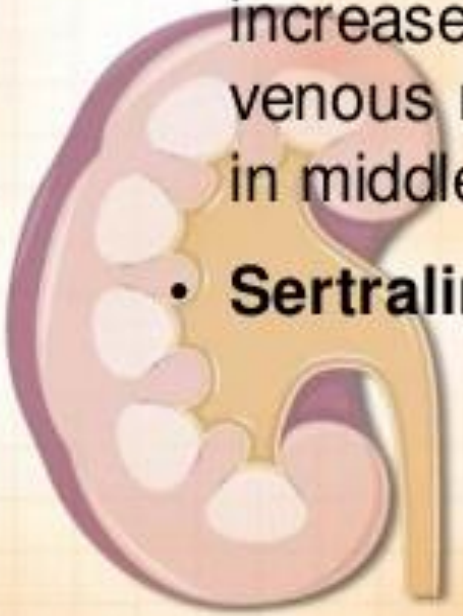
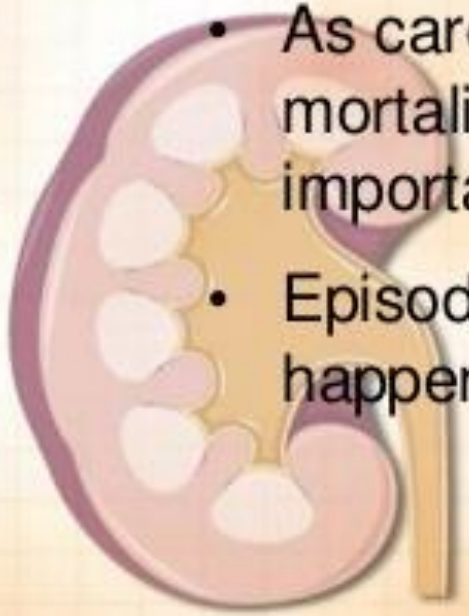


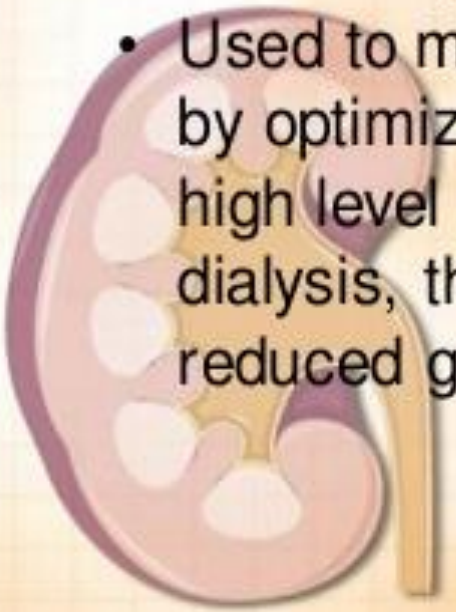
Prevention of hypotension

- Hypotension is unpleasant for pts, makes achievement of euvolemia difficult, leads to persistent fluid overload & HPN
- As cardiovascular disease is the major cause of mortality in ESRD, controlling HPN is of paramount importance.
- Episodic intradialytic hypotension prevents this happening.

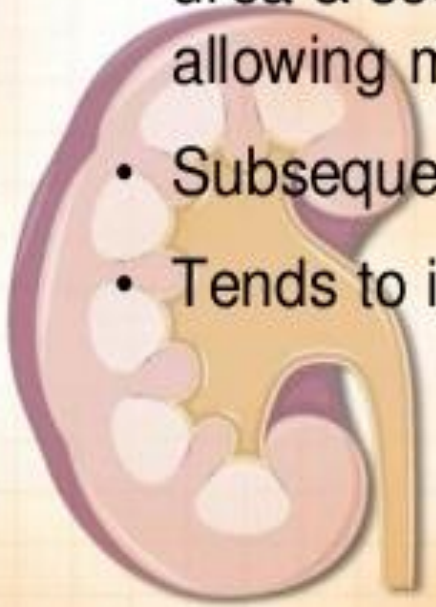


- Time on dialysis
  - Slower, longer HD often cures episodic hypotension but is not favored by pts.
  - Pts need educating.

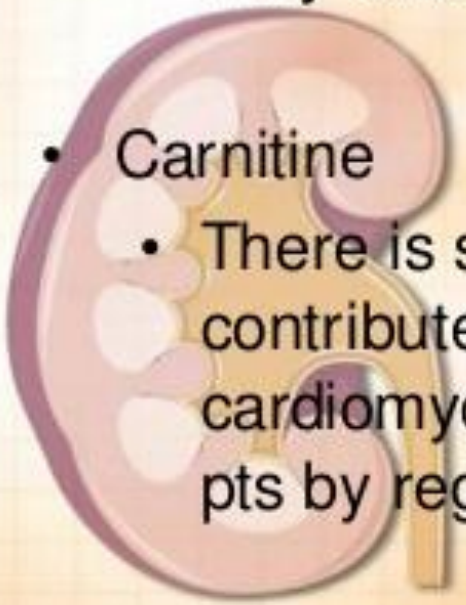
- Sodium ramping or profiling
  - Used to minimize symptoms of hypotension & cramping by optimizing vascular refilling-dialysate sodium is set at a high level (e.g. 155 mmol/l) during the 1st hr (or 2) of dialysis, then either stepped downwards in intervals or reduced gradually over the next 3 h



- Sequential UF & isovolemic dialysis
  - Helps some pts achieve dry wt without hypotension
  - Less effective than sodium profiling
  - UF is performed initially without dialysis during the first hr or 2 of a session, ensuring fluid loss occurs while plasma urea & sodium concentrations may be highest, and allowing most rapid refilling of the vascular compartment
  - Subsequent dialysis is performed with minimal further UF
  - Tends to increase HD session time



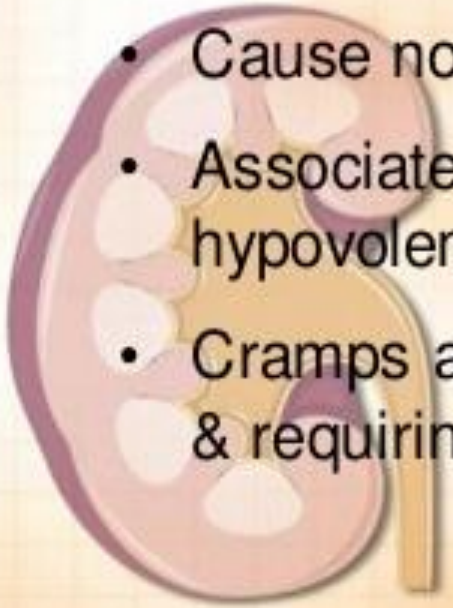
- Temperature modelling
  - The pt's temperature is kept 0.5°C below normal by reducing the dialysate temperature
  - This essentially leads to cutaneous vasoconstriction, which helps maintain BP during dialysis
  - Very effective in preventing hypotension



- Carnitine
  - There is some evidence that carnitine deficiency may contribute to hypotension on dialysis (muscle fatigue, cardiomyopathy, anemia), and may be helped in some pts by regular IV carnitine therapy

Cramps

- Occur in up to 90% of HD treatments, mainly towards the end of dialysis
- A significant cause for early termination & underdialysis
- Cause not entirely clear
- Associated with hyponatremia, hypotension, hypovolemia, hypoxia, carnitine deficiency.
- Cramps are increased in pts using low sodium dialysate & requiring increased UF

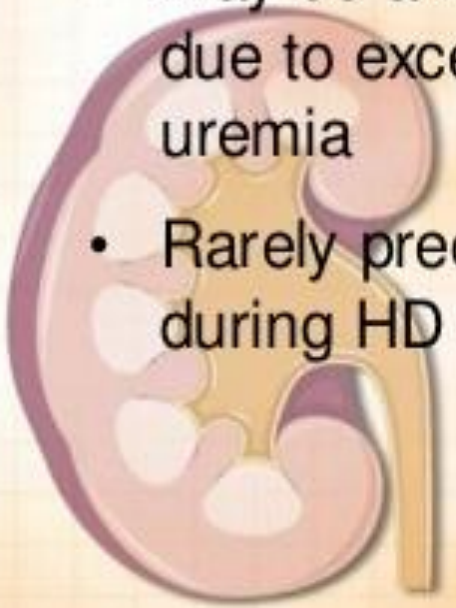


Management of Cramps

- Minimize interdialytic wt gain & need for excessive UF, prevent dialysis hypotension, higher sodium dialysate, or sodium profiling.
- IV saline (normal or hypertonic); IV **50% dextrose** are very effective (but saline will contribute to HPN & volume overload)
- Local massage offers some relief
- **Carnitine** supplementation & **quinine sulphate** may help some pts. Quinine is best used 2 h before dialysis. **Vitamin E** (400iu)
- Some pts respond to **diazepam**, **carbamazepine**, **amitriptyline**, **phenytoin**, or **alcohol**

Nausea, Vomiting, Headache

- Common
- Usually associated with hypotension
- May be a minor manifestation of disequilibrium syndrome due to excess urea removal in pts with persistent marked uremia
- Rarely precipitated by caffeine or alcohol withdrawal during HD

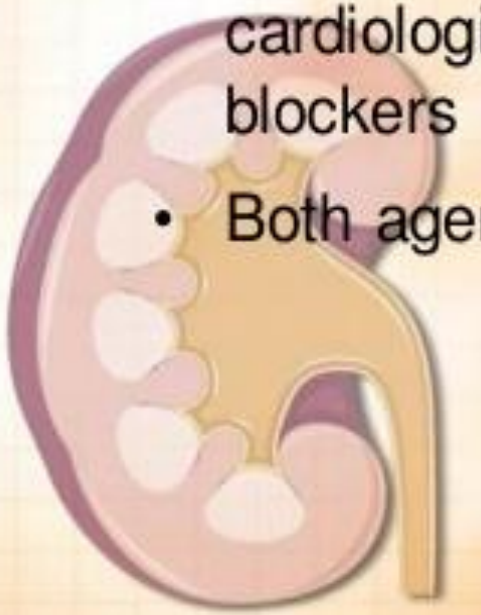


Management of N&V, headache

- Treat & prevent hypotension
- **Antiemetics** & **paracetamol** may help if not precipitated by hypotension
- Reduction of BFR (by 25-30%) during 1st hr of HD sometimes useful (but overall dialysis time must be lengthened to maintain dose of dialysis)
- Use bicarbonate rather than acetate dialysis

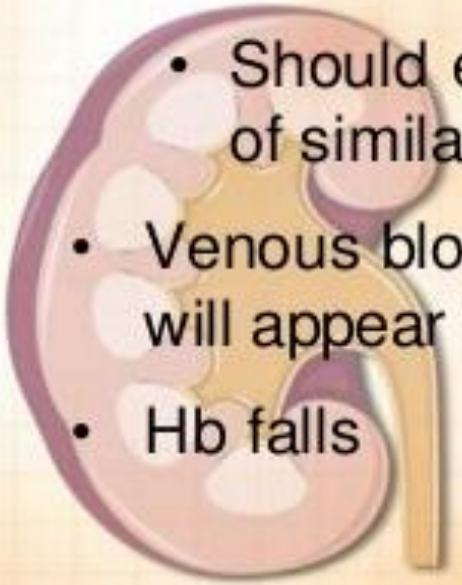
Chest Pain

- Commonly caused by angina, but also by hypotension, DDS, hemolysis, air embolism
- Recurrent angina during HD should be investigated cardiologically, can be treated with nitrates or B^2-blockers
- Both agents may cause hypotension



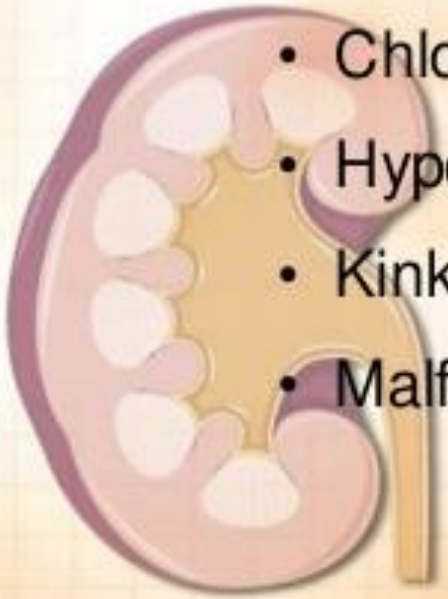
Hemolysis

- Severe hemolysis is rare
- S/sx: chest pain, abdominal or back pain, chest tightness, headache, nausea, malaise
- Life threatening hyperkalemia can occur if unrecognized
  - Should especially be considered if several pts complain of similar symptoms simultaneously
- Venous blood may develop a darker appearance, plasma will appear pink in clotted or spun blood samples
- Hb falls



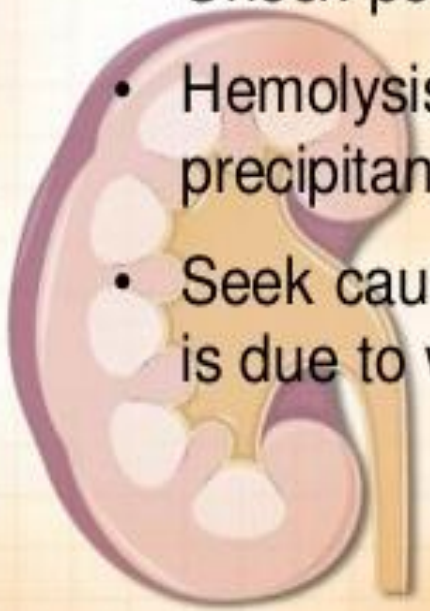
Causes of Hemolysis

- Overheating of dialysate
- Contamination with bleach, formaldehyde, or peroxide from water purification or reprocessing
- Chloramine, nitrates, or copper from water supply
- Hypotonic dialysate
- Kinks in blood tubing
- Malfunctioning blood pump



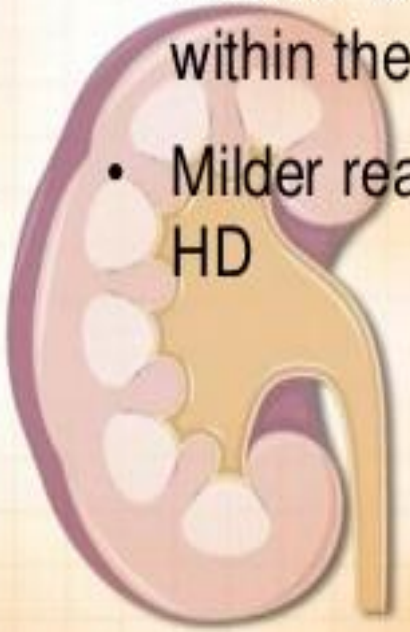
Management of Hemolysis

- Stop blood pump immediately; clamp lines
- Risk of severe hyperkalemia
- Check potassium & Hb
- Hemolysis may continue for several hrs after removal of precipitant
- Seek cause urgently, as multiple pts may be affected if it is due to water or a central dialysate problem



Dialyzer Reactions

- AKA: first use syndromes
- Can occur with reused dialyzers
- Severe anaphylactic reactions (type A) typically occurring within the first few min of HD (but can occur up to 30 min)
- Milder reactions (type B) occur min to hrs after starting HD



	Type A Anaphylactic	Type B Mild
Incidence	Rare (max 5/100 000 dialyses)	Common (3-5/100 dialyses)
Onset	Usually 1st 5 min	Up to 30 min 30-60 min
Symptoms	Moderate-severe	Mild
	Anaphylaxis	Chest pain, back pain
	Itching, urticaria, cough, abdominal cramps, dyspnea, burning, collapse, death	
Causes	•Ethylene oxide (previously common, now rare; pts often have IgE anti-ethylene oxide antibodies) •ACE inhibitors & AN69 membranes (activation of bradykinin system by membrane amplified by ACEI •Bacterial contamination of dialysate in high flux dialysis •Reused dialyzers (bacterial contamination, endotoxin, unknown causes) •Heparin allergy (rare)	•Unknown •Complement activation

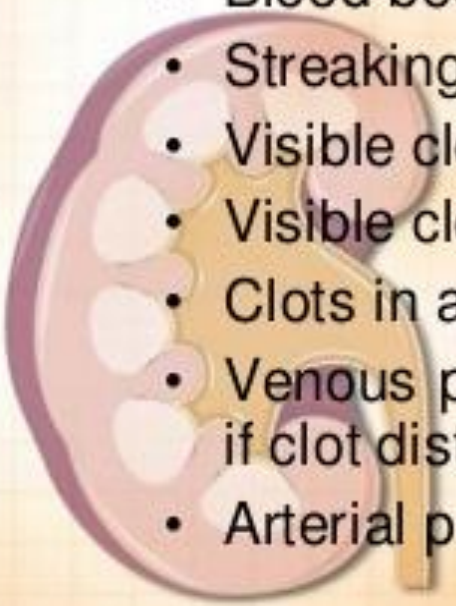
	Type A Anaphylactic	Type B Mild
Treatment	Stop dialysis immediately	Exclude other causes of chest pain
	Clamp lines & discard	Supportive
	CPR if necessary	O2
	IV antihistamines, steroids, adrenaline (SC/IM) if severe	Continue HD
Outcome	Can be fatal Seek cause	Symptoms usually resolve after 30-60 min
Prevention	•Seek cause •Avoid ethylene oxide sterilized dialyzers •Rinse all dialyzers well, with increased volumes •Stop ACE inhibitors, especially if using AN69 or PAN membrane •Change membrane type •If occurs with different dialyzers on several occasions, try heparin-free dialysis	•Change from cellulose to modified cellulose or synthetic membrane •Reusing dialyzers may help

Blood clotting during HD

- Contact between blood & the various plastic surfaces within the extracorporeal circuit initiates platelet adherence, activation of the intrinsic clotting pathway, thrombosis.
- Clotting is promoted by slow BFR, high Hb, high UF rate, intradialytic BT, or parenteral nutrition containing lipid.
- Most HD sessions require anticoagulation, usually with heparin.
  - Low MW heparin, prostacyclin, regional anticoagulation with citrate, heparin-protamine
- Heparin-free dialysis is possible

Clotting during dialysis

- Can contribute to anemia & necessitate BT (with risk of HLA sensitization).
- Leads to underdialysis
- Features:
  - Blood becomes very dark in circuit
  - Streaking in dialyzer
  - Visible clots in bubble trap
  - Visible clots in venous lines
  - Clots in arterial end of dialyzer (not just small strands)
  - Venous pressure will drop if clot forming in dialyzer, or rise if clot distal to monitor
  - Arterial pressure may rise

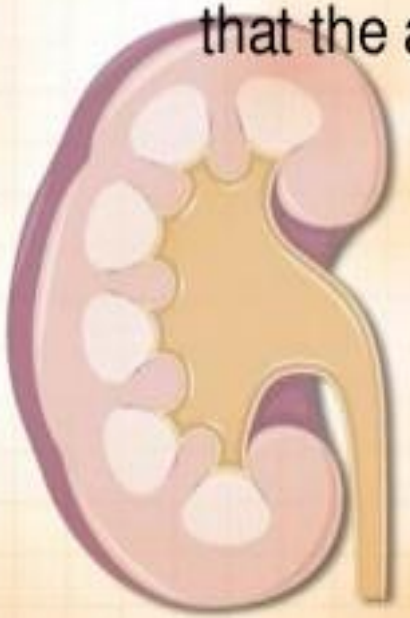


Dialysis Encephalopathy

- Aluminum toxicity
- Occurs as a result of aluminum in the water sources used in the dialysate, ingestion of aluminum-containing antacids (phosphate binders)
- Assessment:
  - Progressive neurological impairment
  - Mental cloudiness
  - Speech disturbance
  - Dementia
  - Muscle incoordination
  - Bone pain
  - Seizures

Management of Dialysis Encephalopathy

- Monitor for s/sx
- Notify the physician if dialysis encephalopathy occur
- Administer aluminum-chelating agents as prescribed so that the aluminum is freed up & dialyzed from the body



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