Individualizing the Management of Intradialytic Hypotension

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Outline
- What is Intradiaytic Hypotension?
- Understanding the risks of hypotension during dialysis
- Factors surrounding blood pressure during dialysis
- Managing hypotension during dialysis
- Take home points…

Intradialytic Hypotension (IDH)
A decrease in blood pressure during dialysis with symptoms that include:
- Abdominal discomfort
- Yawning/sighing
- Nausea/vomiting
- Muscle cramps
- Restlessness/anxiety
- Dizziness or fainting
- SBP < 100 mmHg
- DBP < 40 mmHg
- Drop in SBP > 20 mmHg
- Drop in MAP below 65

Subgroups most likely to have IDH include:
- Diabetic
- CVD
- Poor nutritional status
- Uremic neuropathy
- Autonomic dysfunction
- Severe anemia
- Age ≥65
- Predialysis systolic blood pressure <100 mm Hg

Incidence of ID Hypotension
- Most frequent acute complication during hemodialysis
- Occurs in 15-50% hemodialysis treatments
- Affecting delivery of prescribed dialysis, leads to interdialytic hypertension, fluid overload
- Alienating patients from caretakers - impairs well-being of patients, negatively affects their quality of life

Acute Complications Related to Hypotension
- Cardiovascular and cerebral ischemic events
- Vascular access thrombosis
- Arrhythmias
- Seizures

Long-term effects of IDH
- Volume overload due to limited ultrafiltration and use of fluid boluses
- Left ventricular hyperplasia (thickening of the heart wall)
- Inter-dialytic (between treatment) hypertension
- Increased morbidity, Poorer survival
  - Elderly hemodialysis patients
  - Existing cardiovascular compromise.
Step 1: Understanding Blood Pressure
…the pressure of the blood in the circulatory system, often measured for diagnosis since it is closely related to the force and rate of the heartbeat and the diameter and elasticity of the arterial walls.

Step 2: Assessing Blood Pressure
Volume Assessment
- Hypertension pre / post
- Respiratory Status
- Edema
- Dietary Pattern Changes
- Recent Hospitalizations

Pre-Dialysis / Early Treatment
Medication Regimen: To hold or not to hold
Heart Failure: Significant fluid overload, low BP
Warm Dialysate: Uremia- decrease core temperature

Mid Run
Solute Gradient Changes: Hemoconcentration vs Diffusion, K+ / Ca+ shift
Decreased Compensatory Mechanism: Autonomic dysfunction
Oxygen Saturation Level: Desaturation during HD, effective increased ‘anemia’during HD

Late Run
Dry Weight Assessment: Weight gain?
Osmotic Gradient: Plasma refill slows, UF may not!, K+ / Ca++ shifts continue
Decreased Compensatory Mechanism: Autonomic dysfunction
Oxygen Saturation Level: Desaturation during HD

Orthostatic Hypotension
Sitting to Standing Blood Pressure
- Rapid movement of blood from core organs (thorax, brain) to lower body results in a sudden drop in blood pressure with symptoms of TIA
- 20-25% fall in cardiac output = decrease of >20 mm Hg in SBP or DBP upon standing
- Decrease in venous return – autonomic

Cycle of Hypotension in Congestive Heart Failure
Congestive Heart Failure = High weight gains, low BP, low HR, DOE, edema
- Increased blood volume
- Overstretched myocardium
- Increased wall tension
- Increased cardiac O2 demand
- Decreased cardiac output
- Hypotension
- Decrease UFR
- Saline Bolus
- Increased blood volume
Step 3: Interventions to Consider:
- Medication and Blood Pressure
- Treatment Management Tools
- Patient Clinical Factors

**Medication Regimen**
Weight gains between dialysis treatments is a bigger factor than medications.
Studies have shown greater fluid removal during hemodialysis was associated with more severe blood pressure drops.
Neither number of antihypertensive medications nor dialyzability of medications, nor individual types of medications showed an association with blood pressure variability during dialysis.
Blood pressure instability during dialysis is associated with greater dialytic fluid removal and rate, as well as older age patients and how long a patient had been on dialysis (vintage).

**To hold or not to hold… “Rules of thumb”**
*Continue / Hold less likely*

**Cardio-protective drugs**
- Coreg, Atenolol, Inderal, Tenormin, Lopressor, etc

**Calcium Channel Blockers (if used as anti-arrhythmic)**
- Increase filling / increases cardiac output, controls diastolic dysfunction
- Verapamil, Diltiazem, Norvasc, Adalat

*Usually safe to hold*

‘Afterload’ reduction drugs –
- Clonidine, Hydralazine, Minoxidil

**ACE Inhibitors**
- Lisinopril, Lotensin, Vasotec

**Nitrates**
- Lower blood pressure due to systemic vasodilation
- Reduces ‘preload’, as does dialysis

**Complications with Hypertension in the Dialysis Patient**
Cardiovascular disease is the leading cause of death, especially in the first year of treatment.
Hypertension is the single most important predictor of coronary artery disease in dialysis patients, even more so than cigarette smoking and hypertriglyceridemia.
Highest risk for death from cardiovascular disease was in patients with the lowest and with the highest levels of blood pressure.
No matter what the cause or degree of the kidney disease, high blood pressure can increase damage to the kidneys.
People with kidney disease should keep their blood pressure below 140/90

**Antihypertensives:**
Nocturnal dosing of once daily antihypertensive medications is favored to control the nocturnal increase in blood pressure observed in many hemodialysis patients and to minimize the risk of intradialytic hypotension.
Most people require two or more medications to control their blood pressure.
Various types of anti-hypertensive drugs

**ACE inhibitors** (the “prls”) decrease the production of the enzyme angiotensin II, allowing the blood vessels to enlarge or dilate, thus reducing blood pressure.

ACE-I may cause dry cough, higher potassium levels, and increase EPO requirements.

**Angiotension Receptors Blockers (ARB)** have effects similar to ACE by blocking the binding of angiotensin II.

**Beta Blockers:** (the “alol’s) Preferred antihypertensive agent in patients with recent acute coronary syndrome. Do not significantly increase the incidence of intradialytic hypotension or hyperkalemia.

**Calcium channel blockers (CCBs)** (the “pines”) block the movement of calcium into muscle cells which reduces the pumping action (heart contraction) and relaxes the muscle cells surrounding the arteries to reduce blood pressure. Often effective in patients with volume overload without increasing the risk of intradialytic hypotension.

**Alpha-blockers** relax the muscles and lower blood pressure.

**Alpha-beta blockers** work the same way but also slow the heartbeat.

**Vasodilators** are muscle relaxants that work directly on the muscles surrounding the arteries.

**Central Alpha Agonists** - Inhibits transmission to nerves outside of the brain that innervate muscle cells of the heart and blood vessels, heart rate and blood pressure are reduced.

**Direct Renin Inhibitor**  The kidneys produce the hormone ‘renin’ when they detect low blood pressure. Alikiren (Tekturna) blocks the effects of renin and angiotensin so that blood pressure does not increase.

**Diuretics** are among the oldest known medications for treating high blood pressure. They work to promote the removal of salt and water (fluid) from the body.

**Midodrine to increase BP**

- Maintains the central blood volume (CBV) and cardiac output, and a marginal increase in peripheral vascular resistance (PVR)
- A single dose of midodrine (5 mg) administered 30 minutes before the dialysis session was associated with an improvement in intradialytic and postdialytic systolic and diastolic blood pressures and MAP, compared to dialysis sessions without the use of Midodrine
- Midodrine is effectively cleared by HD and its half-life is reduced to 1.4 hours by HD
- Has minimal cardiac and central nervous system effects, and it does not cross the blood-brain barrier.
- Should be used cautiously in patients with CHF and in those using beta-blockers, digoxin and some CCBs.

On to tools we can consider....

**Dialysate Temperature**

- Relevant parameter for cardiovascular stability
- Decreased core temperature in uremic patients
- Countercurrent warm dialysate acts as heat source resulting in vasodilation
Understanding Plasma Refill Rate

- News flash! We can *only* remove fluid from the vascular compartment.
- Movement of fluid is essential in maintaining blood pressure during the dialysis treatment.
- Different factors in patients can affect this movement.

**Ultrafiltration Profiling**
- Tapers UFR to accommodate individual plasma refill rate (PRR).
- Can be used alone or in conjunction with Sodium Modeling.
- Segments represent period of time on treatment, helps to correlate appropriate profile.

**Sequential Ultrafiltration (DUF, IUF)**
- No dialysate flow.
- Solute movement by “drag” only.
- Extends treatment time, not considered dialysis.
- BP assisted by:
  - Decrease in temperature of extracorporeal blood.
  - Maintains a high solute gradient.

**Electrolyte Shifts: Effect on BP**

**Low-Calcium Dialysate**
- Associated with decreased LV contractility, corresponding decrease in blood pressure.
- Significant intradialytic decrease in blood pressure in both healthy and cardiac-compromised HD patients and patients with decreased LV ejection fraction.

**Low potassium symptoms**
- Weakness, tiredness, or cramping in arm or leg muscles.
- Tingling or numbness.
- Nausea or vomiting.
- Abdominal cramping, bloating.
- Constipation.
- Palpitations (feeling your heart beat irregularly).
Sodium Modeling
- Compensates for decreasing osmolality in blood
- Helps to maintain plasma refill rate
- Assists with electrolyte balance as patient nears end of treatment
- Various programs: Step, Linear, Exponential
- Contraindicated with high pre-dialysis BP
- Concerns related to high interdialytic weight gains with increased thirst
On HD, Na+ equilibrated within about 20-30 minutes
Na+ equilibrated in body within a few hours

Additional Osmotic Factors
- Albumin
- Hemoglobin

Oxygen Saturation: Effect on BP
Hypoxia is dialysis-induced
Patients co-morbidities:
- Peripheral vascular disease
- Anemia
- COPD

Tissue Ischemia
- Release of adenosine
- Blocks norepinephrine
- Causes vasodilation
- Hypotension

Symptoms of Hypoxia
SOB- Tachycardia – Hypotension – Cramping – Tachypneia – Cyanosis - Dizziness – Nausea - Blurred vision - Confusion

Other Considerations
- Septicemia
- Recent hospitalizations - reassessment
- Medication changes
- Contributing laboratory values
- Unstable cardiovascular status
- Unstable respiratory disease
- Dialyzer reaction

Approaches to Intradialytic Hypotension
Using patient’s own saline
- Positioning
- Ace wraps
Enhancing Plasma Refill Rate

- UF modeling
- Sodium modeling
- Oxygen therapy
- ACE wraps
- On-line monitoring

Adjusting Treatment

- Sequential ultrafiltration
- Dialysate temperature
- Extend treatment time
- Consider post electrolytes
- Post-prandial factor
- Weight-based (less than or equal to 4% of total body weight) ultrafiltration

Strong coffee to drink during the last hour of dialysis!
Caffeine blocks adenosine, which can cause vasodilatation

Take home points….

- Intradialytic Hypotension is way too common for the damage it can cause, both acutely and long term, often taken for granted as “routine”
- Most important is to individually assess blood pressure
  - Ongoing assessment skills: DW eval, recent weight trends, edema, SOB
  - When does it hit them?
  - Recent changes: hospital, appetite improved, labs?
  - Comorbidities?
  - Reviewing medications: what to hold or NOT
- What tools can you apply to THIS patient?
  - Positioning?
  - How can you enhance their PRR? Oxygen, UFP, Na+ Model
  - Adjusting treatment application: DUF, extending Tx time, Weight based UF, Temperature

*Remember, YOU can be the one to positively affect the well-being of your patients, and ultimately, their quality of life*