HYPERTENSION

INTRODUCTION

- Hypertension is a risk factor for chronic kidney disease (CKD) that increases in prevalence as CKD progresses, and it is nearly universal in patients with kidney failure.
- The hemodialysis (HD) procedure itself induces hemodynamic changes throughout the course of treatment that warrant routine pre-, post-, and intradialytic blood pressure (BP) monitoring for safety purposes.
- Hypertension is present in approximately 30%–50% of the general population, but its prevalence is disproportionately higher in patients with CKD and greater with each incremental stage of CKD

PATHOPHYSIOLOGY OF HYPERTENSION IN HEMODIALYSIS PATIENTS

- Among patients with kidney failure, nearly 90% have hypertension (defined by BP > 140/90 mm Hg or use of an antihypertensive drug) with little difference in distribution between varying demographics based on age, sex, race, or ethnicity
- ECV overload is a critical factor in hypertension in HD patients

TABLE 44.1 Mechanisms Contributing to Hypertension In Hemodialysis Patients

- Excessive sympathetic nervous system activity
- Excessive activity of the renin-angiotensin aldosterone system
- Endothelial cell dysfunction (Imbalance between endothelin-1, asymmetric dimethylarginine, and nitric oxide)
- Vascular stiffness
- Oxidative stress
- Exogenous pressor effects (erythropoetin stimulating agents)
- Dialyzability of antihypertensive drugs
- Extracellular volume overload

FACTORS NOT RELATED TO EXTRACELLULAR VOLUME OVERLOAD

- Sympathetic nervous system hyperactivity
- Increased activity of the renin-angiotensin system (RAS): Failing kidneys can still secrete renin even in the context of impaired glomerular filtration rate. Inhibitors of the RAS system are first-line agents in the general population
- ET-1 is released from endothelial cells, while ADMA is an endogenous inhibitor of nitric oxide synthase and can interfere with nitric oxide-induced vasodilatation

FACTORS NOT RELATED TO EXTRACELLULAR VOLUME OVERLOAD

- Erythropoietin stimulating agents may contribute to hypertension by increasing release of ET-1.
- The combined calcium-phosphorus product has been associated with higher mean arterial pressure in HD patients
- In addition to consideration of the kidney clearance of medications that may vary, one must consider the dialyzability of the individual drugs, which can even vary within the same drug class.

EXTRACELLULAR VOLUME OVERLOAD

- These patients have lost ability to excrete sodium and water
- They can develop chronic extracellular overload if the appropriate target weight is either not identified correctly or is not able to be achieved due to limitations of fluid removal during dialysis
- The initial increase in BP from acute ECV expansion is likely due to increased cardiac output, but persistence of the volume expanded state can ultimately sustain high BP through a delayed increase in vascular resistance

Extracellular Volume Overload

- Using either biomarker such as brain natriuretic peptide or bioimpedance spectroscopy to define ECV overload, there is evidence that patients who are more volume overloaded have higher mortality risk compared to patients who are more euvolemic

BLOOD PRESSURE TARGETS IN HEMODIALYSIS PATIENTS

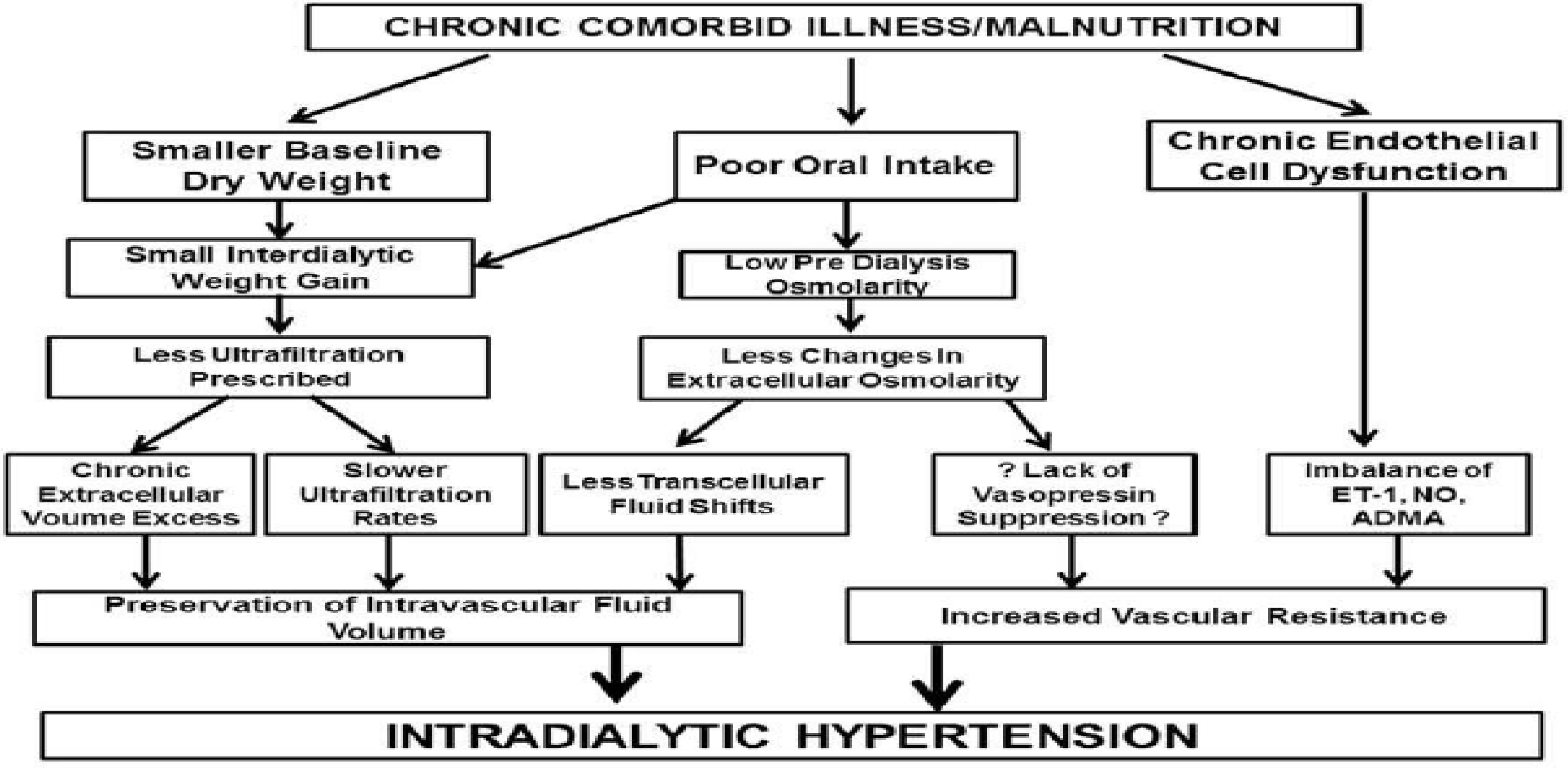
- Management strategies for patients with and without advanced CKD are not exactly the same.
- The current recommendation from the American Heart Association for patients with CKD is to target a BP of 130/80 mm Hg
- The current existing guidelines from the Kidney Disease Outcomes Quality Initiative recommend targeting a pre dialysis BP of 140/90 mm Hg or a post dialysis BP of 130/80 mm Hg

- Several large epidemiologic studies show a U-shaped curve for the association between *change* in BP from pre to post-HD and mortality, demonstrating a poor prognosis from large decreases in BP or *any increase* in BP from pre- to post-HD

INTRADIALYTIC Hypertension

- 76% of dialysis patients are on anti-hypertensive medications.
- Only close to 25% of patients have controlled BP.
- Intradialytic HTN is observed in at least 10% of the maintenance hemodialysis patients.(range of 5-15%)
- Increase in MAP of 15mmHg during or immediately after HD.
- Increase SBP of 10mmHg from pre-to post dialysis

proposed pathophysiology of intradialytic hypertension



PATHOPHYSIOLOGY OF INTRADIALYTIC HYPERTENSION

- Pt factors

•Renin-angiotensin-aldosterone system over-activity

•Sympathetic nervous system over-activity

- Endothelial cell dysfunction
- •Increasing Endothelin-1
- •Decreasing Nitric oxide

PATHOPHYSIOLOGY OF INTRADIALYTIC HYPERTENSION

- HD treatment factors
- •Increasing dialysate sodium
- •Increasing dialysate calcium
- •Increasing plasma osmolality
- •Erythropoietin stimulating agents
- •Removal of antihypertensives(atenolol , metoprolol and ACE inhibitors)
- Volume/fluid factors
- •Increasing weight gain
- •Inaccurate target weight

PATIENT CHARACTERISTICS

- Lower dry weight
- Smaller body mass index
- Lower interdialytic weight gain
- Lower Hb, serum albumin, serum creatinine, serum Ca ,serum phosphorus, serum ferritin and TIBC level
- Tend to be on more anti HTN meds.
- older age
- Non ambulatory status
- male gender

Treatment Options

ble 1 | Potential strategies for the treatment of intradialytic hypertension

otential strategy	Potential methods
duce volume erload	Increase ultrafiltration Reduce cardiac output Restrict dietary salt
ntrol electrolyte anges	Ensure an adequate intradialytic sodium balance Reduce dialysate calcium concentration
duce sympathetic eractivity	Administer angiotensin-converting-enzyme inhibitors Administer angiotensin II receptor blockers Administer direct renin inhibitors Administer adrenergic receptor blockers (α-blockers and β-blockers) Start patient on daily dialysis Increase duration of dialysis
hibit the nin–angiotensin– dosterone system	Administer angiotensin-converting-enzyme inhibitors Administer angiotensin II receptor blockers Administer direct renin inhibitors
aluate concurrent erapies	Consider whether the patient's antihypertensive drugs might be being removed by dialysis

INTRADIALYTIC Hypotension

- Increased mortality
- Access thrombosis
- Under-dialysis and volume overload
- Organs ischemia (MI , CVA and ischemic bowel)
- Increase nursing intervention

PATIENTS AT RISK OF IDH

- 65 years or older age
- DM
- *Patients with CVD:*
  - LVH and diastolic dysfunction with or without CHF
  - LV systolic dysfunction and CHF
  - *Valvular heart disease*
  - Pericardial disease (constrictive pericarditis or pericardial effusion)

PATIENTS AT RISK OF IDH

- *Poor nutritional status and hypoalbuminemia*
- Hyperphosphatemia
- *Uremic neuropathy or autonomic dysfunction due to other causes*
- *Severe anemia*
- *Patients requiring high volume ultrafiltration; more than expected interdialytic weight gain*
- *Patients with pre dialysis SBP of <100 mm Hg*

SAFE ULTRAFILTRATION RATE?

- HEMO Study data: Data from 1846 patients.

•Compared by Uf rates:

–up to 10 ml/h/kg

•70kg patient, 4 hrs = <2.8 Liter target

–10–13 ml/h/kg

•70kg patient, 4 hrs = 2.8 ~3.64 Liter target

•Higher risk of CHF without mortality risk increase seen from this point

–over 13 ml/h/kg:

•70kg patient, 4 hrs = >3.64 Liter target

•Increased risk of mortality

DIALYSATE NA SHOULD BE REGULATED BASED ON SERUM NA:

- **Hyponatremia:**

➢**If Na > 130: Dialysate Na: 140+(140-predialysis Na)**

➢**If Na<130: Dialysate Na: PredialysisNa + 15-20**

- **Hypernatremia:**

➢**Dialysate Na: PredialysisNa-2 mmol**

DIALYSIS TEMPERATURE

- Cool dialysate temperature dialysis (35–36°C) or iso thermic treatments by blood temperature controlled feedback should be prescribed in patients with frequent episodes of IDH (Evidence level I).
- With cool temperature dialysis, dialysate temperature should be gradually reduced in steps of 0.5°C from 36.5°C until symptoms are controlled (Opinion).
- Dialysate temperatures <35°C should not be used (Opinion).

CHOOSING THE RIGHT UF PROFILES

- A profile that begins with the highest UF that can be tolerated by the patient which then decreases to a minimum will work for patients:

•With large weight gains between treatments

•Who become hypotensive late in treatment

•Who cramp late or at the end of treatment