

# Management in Chronic Kidney Disease

Dr.Nadia Asadi  
Nephrologist



# Topic Outline

## **IV** TREATMENT

### A. SLOWING THE PROGRESSION OF CKD

1. Reducing Intraglomerular Hypertension and Proteinuria

### B. SLOWING PROGRESSION OF DIABETIC RENAL DISEASE

1. Control of Blood Glucose
2. Control of Blood Pressure and Proteinuria
3. Protein Restriction

### C. MANAGING OTHER COMPLICATIONS OF CHRONIC KIDNEY DISEASE

1. Medication Dose Adjustment
2. Preparation for Renal Replacement Therapy
3. Patient Education



# Primary prevention

HbA1c <7%

blood pressure target of <140/90 mmHg

tobacco cessation

BMI <27 to prevent the development of CKD

## Screening

all individuals with diabetes and hypertension aged <50 years

all of those aged >50 years

family history of kidney disease



## Secondary prevention

- blood pressure of  $<140/90$  mmHg with ACE inhibitors or angiotensin receptor-blocking agents
- lower blood pressure goal in those with proteinuria of  $>500$  mg per 24 hours
- Protein restriction should not be recommended until late stage 4 or 5 disease
- Aspirin use has also been beneficial for cardioprotection in those with CKD



# SLOWING PROGRESSION OF DIABETIC RENAL DISEASE

## Control of Blood Pressure and Proteinuria

### ■ albuminuria

- a strong predictor of cardiovascular events
- and nephropathy

### ■ Microalbumin testing

- At least ANNUALLY



# MANAGING OTHER COMPLICATIONS OF CHRONIC KIDNEY DISEASE

## 1. Medication Dose Adjustment

- loading dose – no dose adjustment
- >70% **excretion** is by a nonrenal route – no adjustment
- NSAIDs** should be avoided
- Nephrotoxic medical imaging **radiocontrast agents and gadolinium** should be avoided



# CKD progression

- Steps to identify progressive CKD
  - obtain a minimum of three eGFR over not less than 90 days
  - in new cases of reduced eGFR, repeat within 2 weeks to exclude acute deterioration of GFR
- CKD progression is either a decline in eGFR:
  - of  $> 5$  ml/min/1.73 m<sup>2</sup> within 1 year
  - or  $> 10$  ml/min/1.73 m<sup>2</sup> within 5 years



Any acceleration in the rate of decline should prompt a search for superimposed acute or subacute processes that may be reversible

1. ECFV depletion,
2. uncontrolled hypertension,
3. urinary tract infection,
4. new obstructive uropathy,
5. exposure to nephrotoxic agents
6. and reactivation or flare of the original
7. disease, such as lupus or vasculitis



# Monitoring

the rate of progression of CKD serially starting in stage 3a/3b disease screened for anaemia and bone mineral disorders at least every 6 to 12 months :haemoglobin, calcium, phosphorus, and intact parathyroid hormone (PTH).

stage 4 disease, haemoglobin, calcium, phosphorus should be monitored every 3 to 6 months and intact PTH every 6 to 12 months.

stage 5 CKD, anaemia should be evaluated with a monthly haemoglobin, and bone mineral disease with a calcium and phosphorus every 1 to 3 months and an intact PTH every 3 to 6 months.

Lipids should be checked annually for all patients with CKD



# Other recommendations

- Offer a renal ultrasound to all people with CKD who:
  - have progressive CKD
  - have visible or persistent invisible haematuria
  - have symptoms of urinary tract obstruction
  - have a family history of polycystic kidney disease and are aged over 20
  - have stage 4 or 5 CKD
  - are considered by a nephrologist to require a renal biopsy



# Referral criteria

- Refer the following people with CKD for discussion or specialist assessment:
  - stage 4 and 5 CKD (with or without diabetes)
  - higher levels of proteinuria
  - proteinuria together with haematuria
  - rapidly declining eGFR
  - poorly controlled hypertension
  - people with rare or genetic causes of CKD
  - suspected renal artery stenosis



# Modalities

- \* Peritoneal dialysis
  - \* Intermittent hemodialysis
  - \* Hemofiltration
  - \* Continuous renal replacement therapy
- 
- \* Decision of modality determined by catabolic rate, hemodynamic stability, and whether primary goal is fluid or solute removal



# Hemodialysis Access

- \* Acute dialysis catheter (vascular catheter)
- \* Cuffed, tunneled dialysis catheter (Permcath)
- \* Arteriovenous graft
- \* Arteriovenous fistula



# HEMODIALYSIS

## **ABSOLUTE INDICATIONS:**

- Uremic pericarditis or pleuritis
- Uremic encephalopathy

## **Common indications:**

1. Declining nutritional status
2. Persistent or difficult to treat volume overload
3. Fatigue and malaise
4. Mild cognitive impairment
5. Refractory acidosis, hyperkalemia, and hyperphosphatemia

**TABLE**  
**2.3**

**Complications That May Prompt Initiation of Kidney Replacement Therapy<sup>a</sup>**

Intractable extracellular volume overload and/or hypertension  
Hyperkalemia refractory to dietary restriction and pharmacologic treatment  
Metabolic acidosis refractory to bicarbonate treatment  
Hyperphosphatemia refractory to dietary counseling and to treatment with phosphorus binders  
Anemia refractory to erythropoietin and iron treatment  
Otherwise unexplained decline in functioning or well-being  
Recent weight loss or deterioration of nutritional status, especially if accompanied by nausea, vomiting, or other evidence of gastroduodenitis

**Urgent Indications**

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Neurologic dysfunction (e.g., neuropathy, encephalopathy, psychiatric disturbance)  
Pleuritis or pericarditis without other explanation  
Bleeding diathesis manifested by prolonged bleeding time

# Goals of Dialysis

- \* Solute clearance
  - \* Diffusive transport (based on countercurrent flow of blood and dialysate)
  - \* Convective transport (solvent drag with ultrafiltration)
- \* Fluid removal



# Principles of dialysis

- \* **Dialysis** = diffusion = passive movement of solutes across a semi-permeable membrane down concentration gradient
  - \* Good for small molecules
- \* **(Ultra)filtration** = convection = solute + fluid removal across semi-permeable membrane down a pressure gradient (solvent drag)
  - \* Better for removal of fluid and medium-size molecules

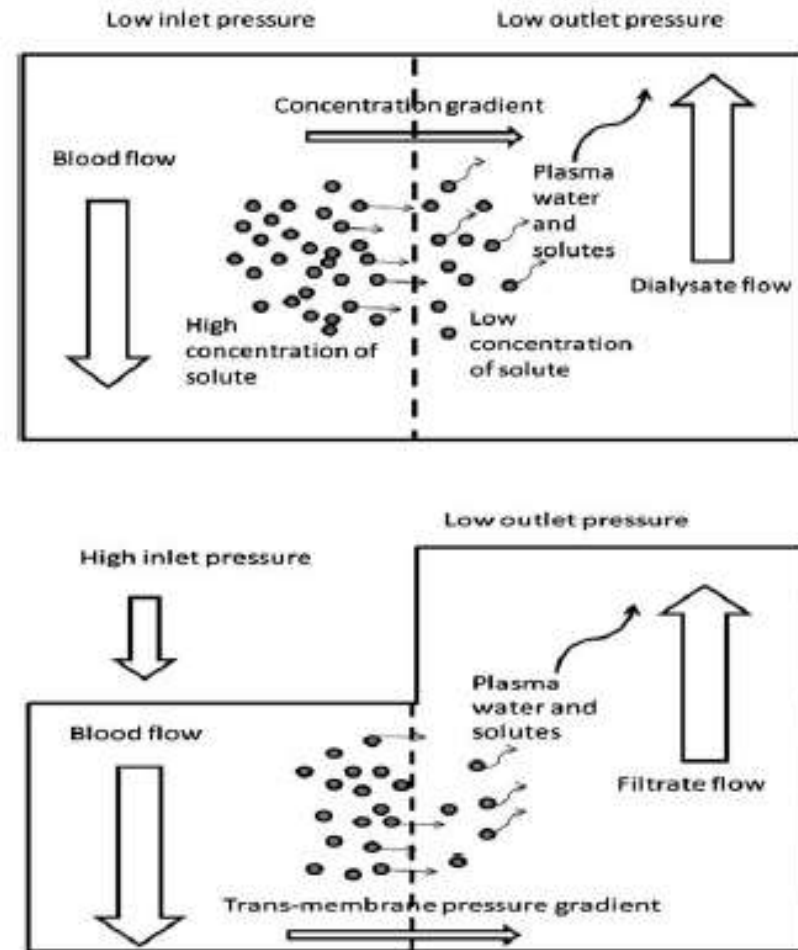


Figure 1 Principles of dialysis (top panel) and filtration (lower panel).

# Complications of Hemodialysis

1. Intradialytic Hypotension
2. Nausea and vomiting
3. Muscle cramp
4. Itching
5. Headache
6. Dialyzer reaction
7. Dialysis dysequilibrium
8. Hemolysis
9. Intradialytic hypoxemia
10. Chest pain and back pain
11. Air embolism

# \* Hypotension

- \* most common
- \* particularly diabetes.
- \* **Factors including:** excessive ultrafiltration with inadequate compensatory vascular filling, impaired vasoactive or autonomic responses, osmolar shifts, overzealous use of antihypertensive agents, and reduced cardiac reserve.

# \* management of hypotension

- \* discontinuing ultrafiltration
- \* administration of 100–250 mL of isotonic saline or 10 mL of 3% saturated hypertonic saline, or salt-poor albumin

# \* Hypotension prevented

- \* the dry weight
- \* ultrafiltration modeling
- \* sequential ultrafiltration
- \* cooling of the dialysate
- \* Avoiding heavy meals during dialysis
- \* Midodrine, an oral selective  $\alpha_1$  adrenergic agent

## \* Anaphylactoid reactions,

- \* first use
- \* most frequently with the bioincompatible cellulosic-containing membranes
- \* . Dialyzer reactions  
two types, A and B
- \* **Type A reactions**
- \* are attributed to an IgE-mediated intermediate hypersensitivity reaction to ethylene oxide

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- \* This reaction typically occurs soon after the initiation of a treatment (within the first few minutes) and can progress to full-blown anaphylaxis if the therapy is not promptly discontinued
  - \* **Treatment** steroids or epinephrine

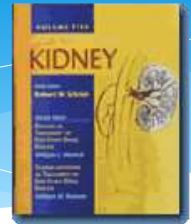


## \* type B reaction

nonspecific chest and back pain

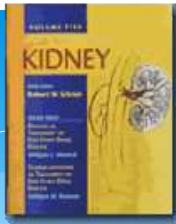
- \* complement activation and cytokine release. several minutes into the dialysis run and typically resolve over time with continued dialysis

# Why to start with PD ?

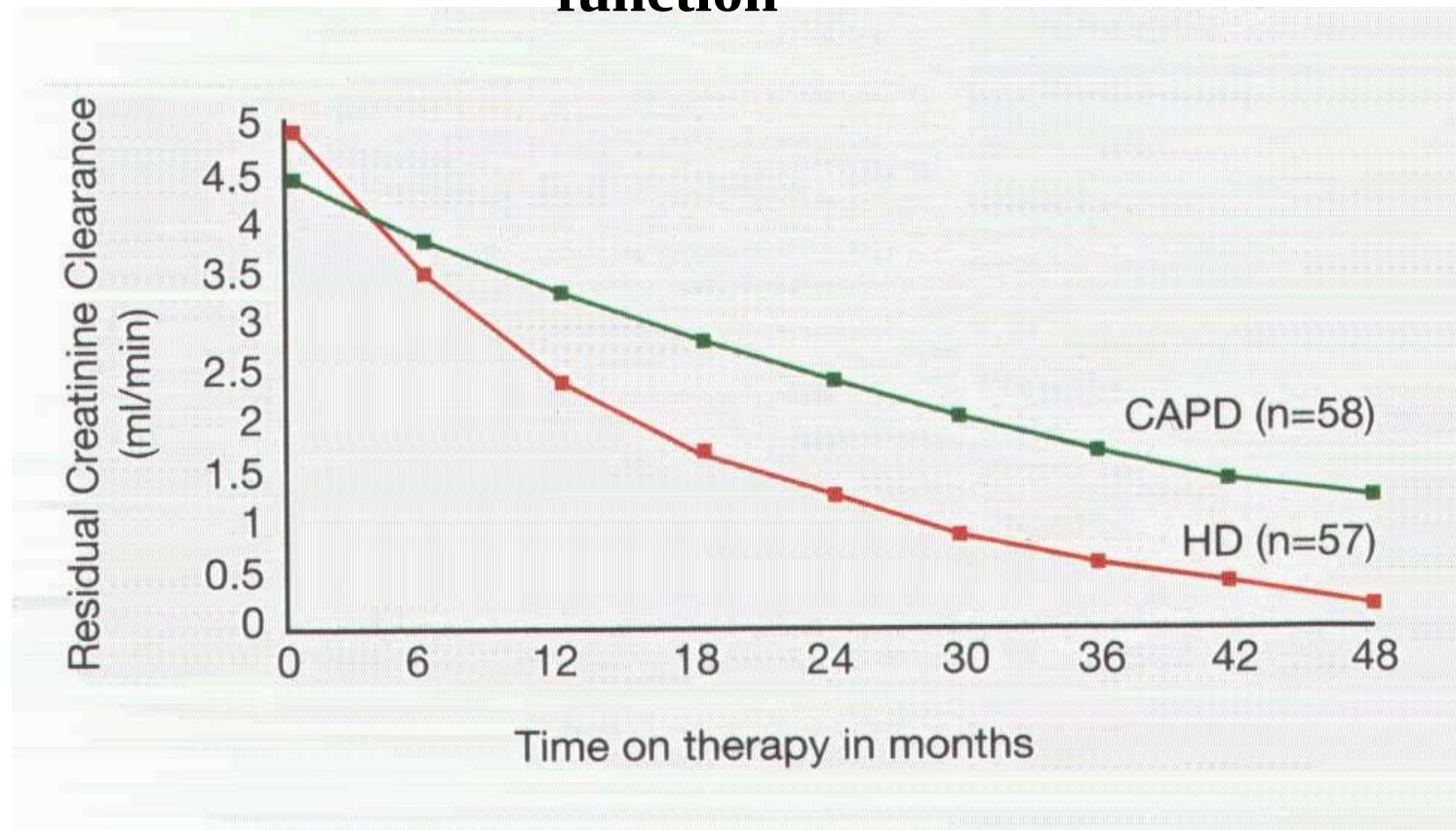


- **clinical outcomes comparable to HD, no difference in 2 year and 5 year mortality vs. HD (study NECOSAD)**
- **saves vascular access**
- **preferred for children (APD)**
- **modality choice is a lifestyle issue**

# Why to start with PD ?

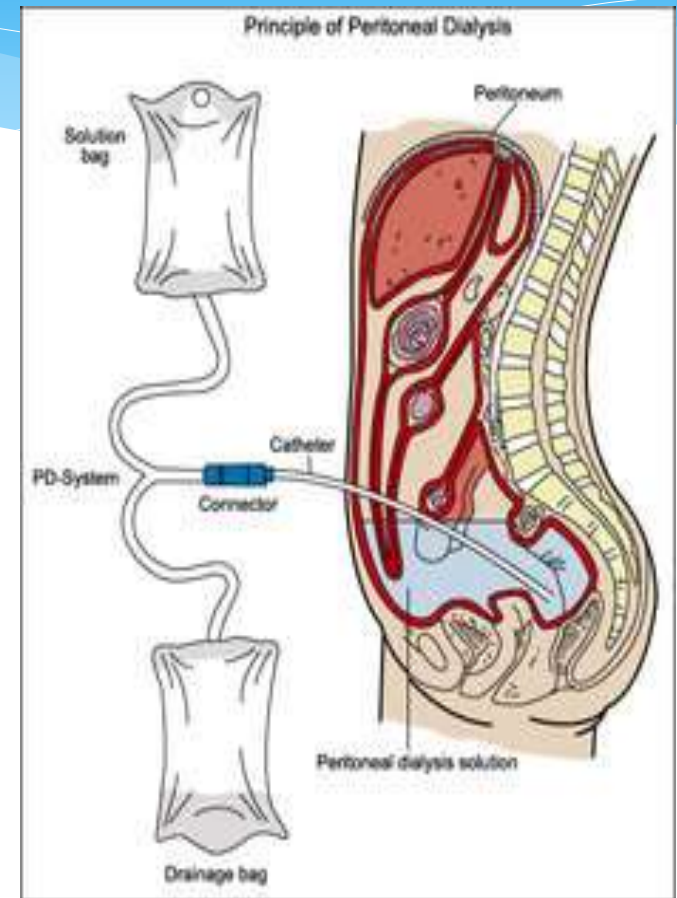


## 1. better maintenance of residual renal function

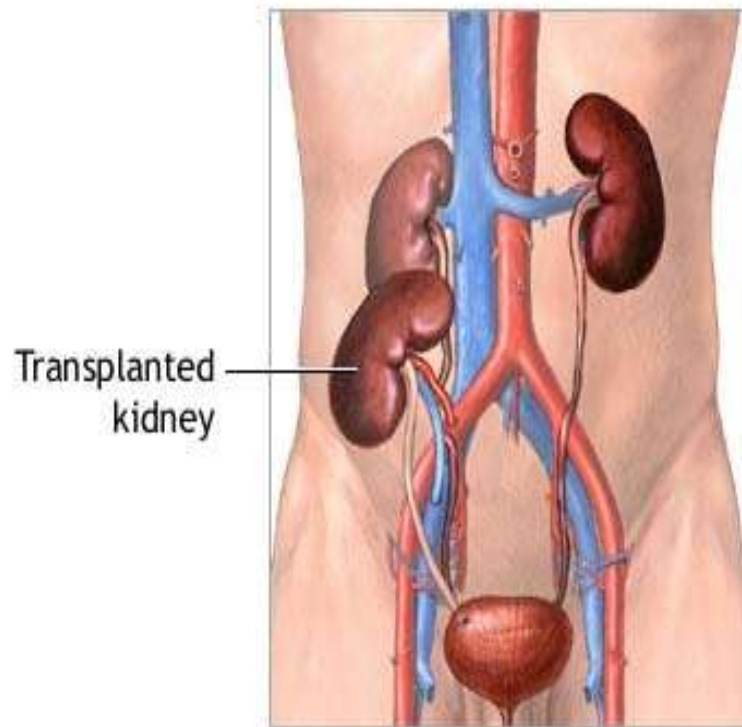


# How it Works (PD)

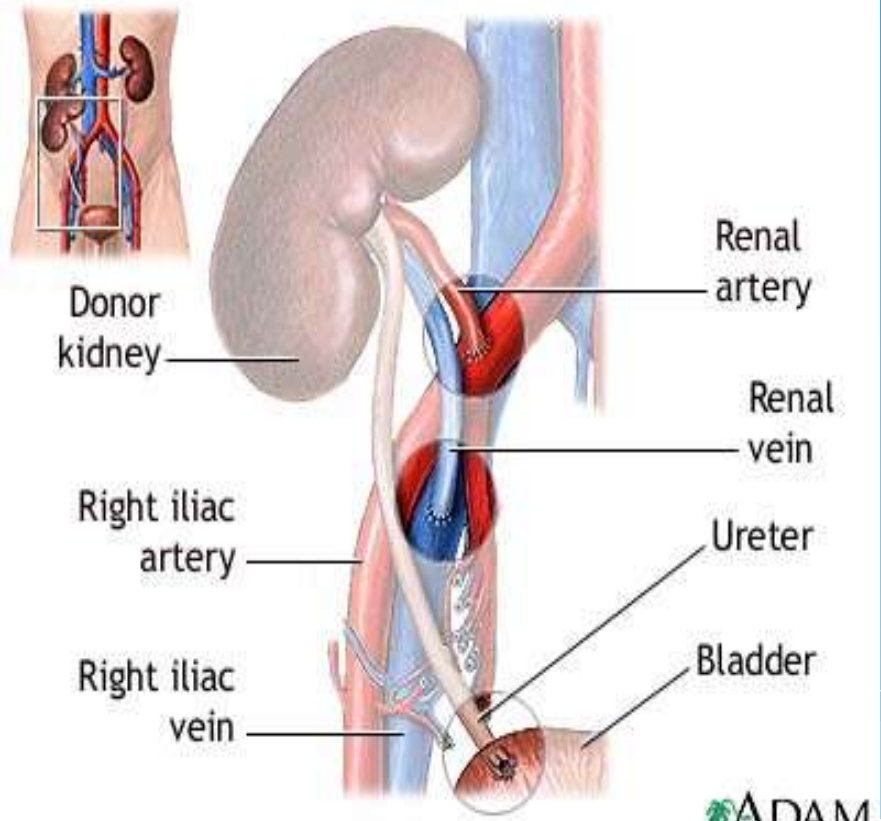
- \* Peritoneal Dialysis
  - \* Creation of access site
    - \* Permanent Peritoneal Catheter
  - \* Dialysate drawn into abdominal cavity
  - \* Peritoneum acts as filter
    - \* Waste stored in dialysate
    - \* Dextrose level determines ultrafiltration rate
  - \* 2 types: CAPD, CCPD
  - \* Dialysate drained from cavity



# Anatomy of Renal Transplantation



ADAM.



ADAM.

**TABLE 337-4 THE MOST COMMON OPPORTUNISTIC INFECTIONS IN RENAL TRANSPLANT RECIPIENTS**

Peritransplant (<1 month)

Wound infections

Herpesvirus

Oral candidiasis

Urinary tract infection

Early (1–6 months)

*Pneumocystis jiroveci*

Cytomegalovirus

*Legionella*

*Listeria*

Hepatitis B

Hepatitis C

Late (>6 months)

*Aspergillus*

*Nocardia*

BK virus (polyoma)

Herpes zoster

Hepatitis B

Hepatitis C

**THANK YOU**

