Medical Management of Chronic Kidney Disease

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Chronic Kidney Disease (CKD) Definition

Evidence of structural or functional kidney abnormality (urinalysis, imaging study or histology) that persists for 3 months with or without decreased GFR

Or

Decreased GFR with or without structural damage to the kidney
CKD Disease Classification

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
<th>GFR (mL/min/1.73 m²)</th>
<th>Prevalence* 2004</th>
<th>Prevalence* 2010</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Kidney damage with normal or ↑ GFR</td>
<td>≥90</td>
<td>6,490</td>
<td>10,137</td>
</tr>
<tr>
<td>2</td>
<td>Mild ↓ GFR</td>
<td>60 - 89</td>
<td>5,830</td>
<td>9,067</td>
</tr>
<tr>
<td>3</td>
<td>Moderate ↓ GFR</td>
<td>30 - 59</td>
<td>8,100</td>
<td>13,058</td>
</tr>
<tr>
<td>4</td>
<td>Severe ↓ GFR</td>
<td>15 - 29</td>
<td>428</td>
<td>687</td>
</tr>
<tr>
<td>5</td>
<td>Kidney failure</td>
<td>&lt;15 or dialysis</td>
<td>330</td>
<td>616</td>
</tr>
</tbody>
</table>

* (1000s), numbers obtained from NKF 2003, Stage 3 & 4 growth rate is 7%, Stage 4 growth rate is 10% obtained from NKF 2003

- NKF projects ESRD patient numbers will double by 2010
- NKF estimates 20 million people in the US are at risk for developing CKD
- Medicare spends nearly $18 billion on Stage 5 per year
- The U.S. has one of the highest dialysis mortality rates of ~25%
# Progression of Kidney Disease

## Initial injury to the kidney:

<table>
<thead>
<tr>
<th>Diabetes Mellitus</th>
<th>Cystic Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>Glomerulonephritis</td>
</tr>
<tr>
<td>Nephrolithiasis</td>
<td>Obstructive uropathy</td>
</tr>
</tbody>
</table>

- Progression in CKD is largely due to secondary factors sometimes unrelated to activity of disease.

- Variable progression to ESRD.
Diseases leading to ESRD

US 2004: 472,099 patients were receiving treatment for ESRD according to the US Renal Data System.

USRDS 2006 Annual Report
Factors Contributing to Progression of CKD
Intraglomerular hypertension

Glomerular hypertrophy

Adaptive Hyperfiltration

Stage 1

Stage 2

Glomerulosclerosis

Tubulointerstitial Disease

Metabolic Acidosis

Hyperlipidemia

Secondary Focal Segmental Glomerulosclerosis: histologic manifestation of hemodynamically mediated renal injury
Glomerular hypertension and hypertrophy induce glomerular injury

1. Direct endothelial cell damage
2. Detachment of glomerular epithelial cells from glomerular capillary wall.
3. Deposit of large molecules Ig or complements in denuded areas
4. Narrowing of capillary lumen
5. Mesangial cells increase cytokine production
NORMAL

Epithelial cell - Podocyte

Mesangium

ENDOTHELIUM

GBM

INJURY

Epithelial cell

Mesangium

Endothelium

GBM

RBC
Factors Contributing to Progression of CKD

**Lesson learned from MDRD:**

- Glomerular hypertension
- Greater Proteinuria
- Higher Blood Pressure
- Black Race
- Lower serum HDL
- Lower levels of serum transferrin
Proteinuria and CKD Progression

- Mesangial toxicity
- Tubular Overload and hyperplasia
- Toxicity from filters compounds
- Induction of pro inflammatory cytokines
- Podocyte apoptosis may worsen proteinuria.
Progression of renal disease increases with increasing urine protein excretion

Table 3. Adjusted Relative Risk for Kidney Disease Progression by Urine Protein Excretion during Follow-up*

| Urine Protein Excretion‡ | Patients§ | Visits§ | Events | Adjusted Relative Risk (95% CI)||
|--------------------------|-----------|---------|--------|---------------------------------|
| g/d                      |           |         |        |                                 |
| <0.50                    | 1022      | 9708    | 52     | 1.00                            |
| 0.5-0.9                  | 699       | 3340    | 35     | 0.96 (0.62-1.49)                |
| 1.0-1.4                  | 616       | 2249    | 23     | 0.89 (0.54-1.47)                |
| 1.5-1.9                  | 548       | 1712    | 26     | 1.21 (0.74-1.96)                |
| 2.0-2.9                  | 629       | 2316    | 48     | 1.67 (1.09-2.54)                |
| 3.0-3.9                  | 423       | 1280    | 38     | 2.25 (1.43-3.53)                |
| 4.0-4.9                  | 320       | 737     | 29     | 3.43 (2.09-5.64)                |
| 5.0-5.9                  | 194       | 476     | 20     | 3.41 (1.91-6.06)                |
| ≥6.0                     | 234       | 792     | 40     | 4.77 (2.92-7.81)                |
| Total                    | 4605      | 22,640  | 311    |                                 |
Risk for progression of kidney disease increases with increasing systolic BP

<table>
<thead>
<tr>
<th>Systolic Blood Pressure†</th>
<th>Patients‡</th>
<th>Visits§</th>
<th>Events</th>
<th>Adjusted Relative Risk (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;110 mm Hg</td>
<td>253</td>
<td>947</td>
<td>10</td>
<td>2.48 (1.07-5.77)</td>
</tr>
<tr>
<td>110-119</td>
<td>548</td>
<td>1976</td>
<td>12</td>
<td>1.00</td>
</tr>
<tr>
<td>120-129 (JNC normal)</td>
<td>959</td>
<td>3746</td>
<td>32</td>
<td>1.23 (0.63-2.40)</td>
</tr>
<tr>
<td>130-139 (JNC high-normal)</td>
<td>1220</td>
<td>4506</td>
<td>59</td>
<td>1.83 (0.97-3.44)</td>
</tr>
<tr>
<td>140-159 (JNC stage 1 hypertension)</td>
<td>1501</td>
<td>7369</td>
<td>113</td>
<td>2.08 (1.13-3.86)</td>
</tr>
<tr>
<td>≥160 (JNC stage 2 and 3 hypertension)</td>
<td>1088</td>
<td>4066</td>
<td>85</td>
<td>3.14 (1.64-5.99)</td>
</tr>
</tbody>
</table>

Total                      | 5569      | 22,610  | 311    |
Hypertensive patients with SBP between 120 to 130 lose 2 ml/min/yr of their GFR. Progress to ESRD in 20 yrs.

Hypertensive patients with SBP >150 lose 8 ml/min/yr of GFR. Progress to ESRD in 5 yrs.
Patients with HBP and proteinuria have higher risk of renal disease progression.

Figure. Relative risk for kidney disease progression based on current level of systolic blood pressure and current urine protein excretion.
### Other factors contributing to progression of CKD

<table>
<thead>
<tr>
<th>Factor</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angiotensin II</td>
<td>Generate fibrosis through stimulus of TGFβ and epidermal growth factor.</td>
</tr>
<tr>
<td>Phosphate retention</td>
<td>Mesangial cell proliferation after LDL receptors in mesangial cells activated. Increases Macrophage chemo-attractant factors, ROS.</td>
</tr>
<tr>
<td>Aldosterone</td>
<td>Mineralocorticoid stimulus leads to vascular remodeling and renal fibrosis.</td>
</tr>
<tr>
<td>Metabolic Acidosis</td>
<td>Remaining nephrons excrete more acid. Increased ammonia generation activates complements leading to tubulointerstitial damage.</td>
</tr>
</tbody>
</table>
Management of Chronic Kidney Disease

I. Treatment of reversible causes of renal dysfunction

II. Preventing or slowing progression of disease

III. Treatment of complications of renal dysfunction

IV. Identification and adequate preparation of the patient in whom renal replacement therapy (RRT) will be required
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Reversible causes of renal dysfunction

- **Renal hypoperfusion**
  - Hypovolemia
  - Hypotension
  - Drugs lowering GFR
    - NSAID’s
    - ACE

- **Nephrotoxic drugs**
  - Aminoglycosides
  - NSAID’s
  - Contrast material

- **Urinary Tract obstruction**

- Cimetidine, and trimethoprim interfere with creatinine secretion.
- Cefoxitin and flucytosine interfere with assay used to measure serum creatinine.
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Slowing the rate of progression

• Blood Pressure control slows progression of kidney disease particularly in patients with significant proteinuria.

• Evidence in diabetic nephropathy and non-diabetic nephropathy that administration of ACE-I or ARB slows the progression of CKD with the greatest benefit in patients with higher degrees of proteinuria.

• The benefit is to be greatest if begun before a great deal of scarring has begun.

• Combination therapy with an ACE-I and ARB gives additive antiproteinuric effect.
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MDRD: Aggressive BP control preserves renal function in proteinuric patients

MDRD: Tight BP control in patients with GFR 25-55 ml/min and non-diabetic proteinuria (> 3g) led to decrease in GFR decline.

Group A: Patients with GFR 25-55 ml/min

MDRD: Tight BP control in patients with GFR 13-24 ml/min and non diabetic proteinuria (> 1g) led to less decline in GFR

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ACE-I reduces likelihood of doubling PCreat by 50% in proteinuric DM-I patients

Benazepril trial: ACE-I reduce risk for disease progression by 53%

REIN: GFR stabilized in continued ramipril use
REIN: Greater kidney survival in long term ACE use

Figure 2: Mean GFR decline during the REIN core and follow-up study in patients continued on or switched to ramipril

Figure 3: Kidney survival in patients continued on or switched to ramipril during the whole (core and follow-up) study period
Slowing the rate of progression

• Blood Pressure control slows progression of kidney disease particularly in patients with significant proteinuria.

• Evidence in diabetic nephropathy and non-diabetic nephropathy that administration of ACE-I or ARB slows the progression of CKD with the greatest benefit in patients with higher degrees of proteinuria.

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• Combination therapy with an ACE-I and ARB gives additive antiproteinuric effect.
COOPERATE: Combined ACE and ARB decreases risk of kidney disease progression

Figure 2: Proportion of patients reaching endpoint

Number at risk

<table>
<thead>
<tr>
<th></th>
<th>Losartan</th>
<th>6</th>
<th>12</th>
<th>18</th>
<th>24</th>
<th>30</th>
<th>36</th>
</tr>
</thead>
<tbody>
<tr>
<td>Losartan</td>
<td>89</td>
<td>88</td>
<td>84</td>
<td>79</td>
<td>65</td>
<td>59</td>
<td>47</td>
</tr>
<tr>
<td>Trandolapril</td>
<td>86</td>
<td>85</td>
<td>83</td>
<td>75</td>
<td>72</td>
<td>63</td>
<td>58</td>
</tr>
<tr>
<td>Combination</td>
<td>88</td>
<td>87</td>
<td>86</td>
<td>83</td>
<td>76</td>
<td>73</td>
<td>67</td>
</tr>
</tbody>
</table>

COOPERATE Lancet 2003; 361: 117-124
COOPERATE: There is an added benefit to dual blockade in decreasing proteinuria

Figure 4: Median urinary protein excretion by treatment group

Table 3: Effect of baseline daily urinary protein excretion rate on efficacy of combination treatment on combined primary endpoint

Baseline daily urinary protein excretion rate (g/day)

<table>
<thead>
<tr>
<th></th>
<th>Hazard ratio (95% CI)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>0.34 (0.19–2.68)</td>
<td>0.031</td>
</tr>
<tr>
<td>Baseline daily urinary protein excretion rate (g/day)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>0.69 (0.22–2.28)</td>
<td>0.049</td>
</tr>
<tr>
<td>1 to &gt;3</td>
<td>0.33 (0.19–0.74)</td>
<td>0.029</td>
</tr>
<tr>
<td>≥3</td>
<td>0.40 (0.21–0.84)</td>
<td>0.033</td>
</tr>
</tbody>
</table>

Figure 3: Blood pressure by treatment group

COOPERATE Lancet 2003; 361: 117-124
Slowing Progression in Diabetic Nephropathy
Natural history of type 1 diabetic nephropathy

<table>
<thead>
<tr>
<th>Stage</th>
<th>Pre</th>
<th>Incipient</th>
<th>Overt</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Functional</strong></td>
<td>GFR ↑ (25–50%)</td>
<td>Microalbuminuria, hypertension</td>
<td>Proteinuria, nephrotic syndrome, GFR ↓</td>
</tr>
<tr>
<td><strong>Structural</strong></td>
<td>Renal hypertrophy</td>
<td>Mesangial expansion,</td>
<td>Mesangial nodules (Kimmelstiel–Wilson</td>
</tr>
<tr>
<td></td>
<td></td>
<td>glomerular basement membrane</td>
<td>lesions)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>thickening, arteriolar</td>
<td>Tubulointerstitial fibrosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>hyalinosis</td>
<td></td>
</tr>
</tbody>
</table>
Control of blood pressure retards progression of type 1 diabetic nephropathy

- ΔGFR = 0.94 mL/min per month
- ΔGFR = 0.29 mL/min per month
- ΔGFR = 0.10 mL/min per month

Commencing antihypertensive therapy

Glomerular filtration rate (GFR) (mL/min per 1.73 m²)

Years
-2 -1 0 1 2 3 4 5 6
Tight glycemic control with aggressive insulin therapy and effect on Diabetic Nephropathy: Kumamoto Study

- Normoglycemia was obtained in the patients receiving intensive insulin therapy by the third month.
- This tight control was sustained throughout the remainder of the study period.

Y. Ohkubo et al. /Diabetes Research and Clinical Practice 28 (1995) 103-117
Tight glycemic control with aggressive insulin therapy and prevention of nephropathy: Kumamoto Study

A. In patients without retinopathy and/or UAE < 30 mg/24hrs the percentage of patients who developed nephropathy after 6 years was lower in the multiple insulin injection group.

B. In patients with simple retinopathy and/or microalbuminuria < 300 mg/24h the percentage of patients who developed nephropathy after 6 years was lower in the multiple insulin injection group.
Intensive glycemic control by multiple insulin injections reduced the average risk of worsening nephropathy by 70% during the entire study period.
Other strategies to slow progression: Protein Restriction

- MDRD largest trial to evaluate protein restriction effect on disease progression.
- Randomized to 1.1 g/kg or 0.7 g/kg of protein per day.
- Little overall benefit with low protein diet.
  - Low protein: greater fall in GFR in first 4 mo, then slower progression

Recommendation: A reasonable regimen consists of rigorous blood pressure control and the intake of 0.8 to 1.0 g/kg of high biologic value protein per day.
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IV. Identification and adequate preparation of the patient in whom renal replacement therapy (RRT) will be required
| Volume Overload | Decreased ability to respond to rapid infusion of Na+ → overload.  
**Tx.** Na+ restriction and diuretic therapy |
| Hyperkalemia | Develops in oliguric patients, high K+ diet, increased tissue breakdown or hypoaldo.  
Could be due to ACE-I or ARB.  
**Tx.** low K+ diet and loop diuretic.  
Consider low dose Kayexalate if persists. |
| Metabolic Acidosis | Bone buffering of H+ can release Ca++ and PO4= from bone - worsens bone disease.  
Uremic acidosis can increase skeletal muscle breakdown and diminish albumin synthesis → lean body mass loss.  
**Tx.** Alkali therapy sodium bicarbonate 0.5-1meq/kg/d. Avoid sodium citrate |
Hyperphosphataemia and Pathogenesis of SHPT

↓ Renal Function

↓ Intestinal Ca++ absorption

Hypocalcaemia

Altered Parathyroid Gland Function

Altered 1,25(OH)2D Metabolism

Phosphate retention

Hyperphosphataemia

Hyperparathyroidism & Renal Osteodystrophy
CKD Progression

Progressive loss of 1,25D

- Lower limit of 1,25D

1,25D pg/mL

GFR, mL/min/1.73m²

Stage 1  Stage 2  Stage 3  Stage 4  Stage 5
105  95  85  75  65  55  45  35  25  15

PTH pg/mL

- 1,25D
- PTH
Bone and mineral issues are prevalent in CKD


- **High Phosphorus**
  - Stage 3 and 4, >4.6 mg/dL
  - Stage 5 >5.5 mg/dL

- **Abnormal PTH**
  - Stage 3, >70 pg/mL
  - Stage 4, >110 pg/mL
  - Stage 5 >300
# K/DOQI Guidelines on Bone and Mineral Metabolism

<table>
<thead>
<tr>
<th></th>
<th>CKD Stage 3</th>
<th>CKD Stage 4</th>
<th>CKD Stage 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phosphorus (mg/dL)</td>
<td>2.7-4.6</td>
<td>2.7-4.6</td>
<td>3.5-5.5</td>
</tr>
<tr>
<td>Calcium (mg/dL)</td>
<td>“Normal”</td>
<td>“Normal”</td>
<td>8.4-9.5; Hypercalcemia &gt;10.2</td>
</tr>
<tr>
<td>Intact PTH (pg/mL)</td>
<td>35-70</td>
<td>70-110</td>
<td>150-300*</td>
</tr>
</tbody>
</table>

*Evidence*
# K/DOQI Guidelines on Anemia Management

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosis of Anemia</td>
<td>Hb &lt;13.5 in males</td>
</tr>
<tr>
<td></td>
<td>Hb&lt; 12 in females</td>
</tr>
<tr>
<td>Target Hb</td>
<td>≥ 11 g/dL</td>
</tr>
<tr>
<td></td>
<td>Caution with Hb &gt; 13 g/dL</td>
</tr>
<tr>
<td>Target TSAT and ferritin</td>
<td>TSAT &gt; 20%</td>
</tr>
<tr>
<td></td>
<td>Ferritin &gt; 200 in HDD-CKD</td>
</tr>
<tr>
<td></td>
<td>Ferritin &gt; 100 in non HDD-CKD</td>
</tr>
<tr>
<td></td>
<td>Ferritin &gt; 500 not recommended</td>
</tr>
</tbody>
</table>
CREATE

- CKD not on dialysis
- Epoetin beta
- Hgb 10.5-11.5 vs 13-15
- Increased rate of progression of CKD
- No significant difference in CV outcomes

CHOIR

- Hgb 11.3 g/dL vs 13.5 g/dL
- CKD not on dialysis
- Stopped early because of excess CV adverse outcomes in higher Hb group
- No QOL benefit in higher Hb group

CHOIR and CREATE Impartial
Conclusions

“Taken together, these two studies suggest caution in the full correction of anemia in patients with chronic kidney disease … Although we need more information about the ideal target level and should consider the present guidelines incomplete, it seems wisest to refrain from COMPLETE correction of anemia in patients with chronic kidney disease.”

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I. Treatment of reversible causes of renal dysfunction

II. Preventing or slowing progression of disease

III. Treatment of complications of renal dysfunction

IV. Identification and adequate preparation of the patient in whom renal replacement therapy (RRT) will be required
Preparation for and Initiation of Renal Replacement Therapy

- Early referral of patients with CKD to nephrologist.
  - GFR < 60 ml/min
  - Male S Cr > 1.5 mg/dl
  - Female S Cr > 1.2 mg/dl

- Apply strategies to slow progression of disease.

- Once GFR drops < 30 ml/min educate patient with regards to RRT

- Offer counseling with multi-disciplinary team
  - Dietitian
  - Nurses
  - Social Worker
Absolute clinical indications to initiate RRT

- Pericarditis
- Fluid overload or pulmonary edema refractory to diuretics
- Accelerated Hypertension
- Progressive uremic encephalopathy or neuropathy
- Bleeding diathesis
- Persistent nausea and vomiting
- Plasma Creat > 12 mg/dl or BUN > 100 mg/dl
- 2006 K/DOQI GFR < 15 mL/min/1.73m2 or higher GFR but patient with declining health due to loss of kidney function.
Intermittent vs. Continuous

Advantages of Continuous Tx:
1. Less ECF shift
2. Less Hypotension
3. Less treatment-associated renal injury
4. Adequate removal of toxins
Renal Replacement Therapy (RRT)

CKD Stage V: ESRD

1. Hemodialysis
2. Peritoneal Dialysis
3. Kidney transplant
Hemodialysis

- Intermittent treatment offered 4 hours three times a week.
- In center therapy
- Requirements
  - Arterio-venous access
  - Filter
  - Nurses
Always Avoid Subclavian Catheters: Have 40% Superior Vena Cava Stenosis after first implanted.
• 2006 K/DOQI guidelines recommend that a fistula be placed at least 6 months prior to the anticipated start of hemodialysis.

• End to side vein to artery anastomosis of cephalic vein and radial artery. (20 yr life)

• Evaluate non maturing AVF
AV Graft: polytetrafluoroethylene (PTFE)

- Usually mature in 2 weeks.
- 2006 K/DOQI guidelines recommend that a synthetic graft be placed at least 3-6 weeks prior to anticipated start of dialysis.
Tunneled catheter: should be short duration until permanent access achieved
HEMODIALYSIS
1. Movement of solutes from area of higher concentration to area of lower concentration
2. Dialysate used to create concentration gradient across a semi-permeable membrane
3. Dialysis uses a semi-permeable membrane for selected diffusion

1. Movement of solutes with water flow, SOLVENT DRAG.
2. Certain membrane materials display adsorptive characteristics
   - Surface adsorption to the membrane
   - Bulk adsorption within the membrane when molecules can permeate it
Peritoneal Dialysis

- Parietal Peritoneum
- Peritoneal Cavity
- Catheter
- Visceral Peritoneum
Advantages of Peritoneal Dialysis

• Independent lifestyle: work and travel
  – More flexible holidays and travel
  – Higher employment rates

• Different diet: less K restriction, less protein restriction, less volume restriction

• Treatment of choice for infants and young children
Advantages of Peritoneal Dialysis

• Similar survival to HD, with superior survival in the first 2-3 years

• Considerable reductions in peritonitis and catheter related infections

• Adequate solute clearance

• Better tolerated hemodinamically, ideal for patients with heart disease
Advantages of Peritoneal Dialysis

• Better BP and fluid control in the first few years of dialysis

• Better preservation of residual renal function versus HD

• Higher hemoglobin levels, less rhUePO

• Better outcomes after transplant
Advantages of Peritoneal Dialysis

- Ability to expand patient numbers in a dialysis center with limited need for resources and major capital investments.

- Lower staff to patient ratio than center HD

- Less costly than center HD
Disadvantages

- Higher technique failure in PD compared to HD.
- Low rate of achieving long term PD due to changing membrane.
- Survival rate at 10 years after initiating PD is 20%.
- Reasons for therapy change to HD (see pie graph).

Disadvantages

• Hands on, individual therapy

• Protein loss make it a poor choice for malnourished patients
Transplant

- Evaluation of potential recipient and living donor if available.
- Timely referral
Conclusion Slow Progression of Disease

- Aggressive BP and proteinuria control
- Protein excretion 500-1000 mg
- BP reduction 130/80 mmHg
- Lower BP in patients with >1g proteinuria
- Add ARB to patient on ACE
- Add Ace to pt on ARB
- Protein intake 0.8-1.0 g/kg/d
- Treat complication of CKD
Conclusion

I. Treatment of reversible causes of renal dysfunction

II. Preventing or slowing progression of disease

III. Treatment of complications of renal dysfunction

IV. Identification and adequate preparation of the patient in whom renal replacement therapy (RRT) will be required
Questions?