Renal complication in HIV infection?

How to avoid how to manage?

Wisit prasithsirikul, MD
Bamrasnaradura infectious disease institute
Renal Disorders in patient with HIV infection

1. Coincidental renal disorders
2. Unrelated renal disease in HIV infected pt
3. Specific glomerular syndrome
4. HIV infection in pt receiving RRT
Screening Algorithm for HIV-associated disorders

Quantitative evaluation of the risk of kidney disease

- Family history
- Ethnicity
- CD4 cell count
- Viral load
- Previous use of nephrotoxic drugs
- Co morbidity (diabetes mellitus, hypertension, HCV infection)

Examination at time of initial HIV diagnosis

- Urine analysis (for proteinuria)
- Serum creatinine (Determination of Clearance or GFR)

Anomalous values

- Degree > 1+ proteinuria with Urine stick
- Clearance or GFR < 60 ml/min per 1.73 m2

No anomalous values

- Assessment of proteinuria by protein/creatinine ratio in spontaneous urine
- Ultrasound scan
- Referral to nephrologist if necessary

Patients with no risk factors for renal disease should be clinically monitored

Patients at risk of chronic renal disease should be examined yearly
Estimates of GFR: Cockcroft-Gault and MDRD (modified diet in renal disease) Formula

- CG = (140-age) x weight x (0.85 if F) x (Pcr) x 72

- MDRD = 186 x Pcr(mg/dl)^-1.154 x age (yr) + 0.203 x (1.212 if black) x (0.742 if female) x (1.233 if chinese)

Cockcroft DW and Gault MH Nephron 1976; 16:31-41
Automatic report of eGFR and UPCR

<table>
<thead>
<tr>
<th>Test Name</th>
<th>Result</th>
<th>Reference Range</th>
<th>Unit</th>
<th>Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>BUN</td>
<td>22</td>
<td>6-20</td>
<td>mg/dL</td>
<td>Urease</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1.7</td>
<td>0.7-1.2</td>
<td>mg/dL</td>
<td>Kinetic</td>
</tr>
<tr>
<td>eGFR</td>
<td>46.77</td>
<td></td>
<td>mL/min</td>
<td></td>
</tr>
</tbody>
</table>

Interpretation: eGFR = Moderate decrease in eGFR

<table>
<thead>
<tr>
<th>Test Name</th>
<th>Result</th>
<th>Reference Range</th>
<th>Unit</th>
<th>Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine Protein</td>
<td>104.1</td>
<td>&lt;12</td>
<td>mg/dL</td>
<td>Biuret</td>
</tr>
<tr>
<td>Urine Creatinine</td>
<td>282.6</td>
<td>39-259</td>
<td>mg/dL</td>
<td>Kinetic</td>
</tr>
<tr>
<td>Urine Protein-Creatinine Ratio</td>
<td>0.37</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Comment:

Requested By: น.ส.พิริมภู ม่วงกุล  
Requested Date & Time: 27/07/2009 09:08:41  
Requested Date & Time: 27/07/2009 10:26:52  
Requested Date & Time: 27/07/2009 10:27:36

Reported By: น.ส.พิริมภู ม่วงกุล  
Reported Date & Time: 27/07/2009 10:26:52  
Reported Date & Time: 27/07/2009 10:27:36

Approved By: นางมุสรา นพพัฒน์ปานมี     
Approved Date & Time: 27/07/2009 10:27:36

Wisit Prasithsirikul, MD
Bamrasnaradura Institute
# Chronic Kidney Disease Classification

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
<th>GFR (mL/min/1.73 m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Kidney damage with normal or increased GFR</td>
<td>≥ 90</td>
</tr>
<tr>
<td>2</td>
<td>Kidney damage with mildly decreased GFR</td>
<td>60-89*</td>
</tr>
<tr>
<td>3</td>
<td>Moderately decreased GFR</td>
<td>30-59</td>
</tr>
<tr>
<td>4</td>
<td>Severely decreased GFR</td>
<td>15-29</td>
</tr>
<tr>
<td>5</td>
<td>Kidney failure</td>
<td>&lt; 15 or dialysis</td>
</tr>
</tbody>
</table>

* May be normal for age

Chronic kidney disease is defined as either kidney damage or GFR < 60 mL/min/1.73 m² for ≥ 3 months

[kidney.org/professionals/kdoqi/guidelines_ckd/p4_class_g1.htm](http://kidney.org/professionals/kdoqi/guidelines_ckd/p4_class_g1.htm)
Renal consultation in HAART Era

1. ARF
2. TDF Nephropathy
3. IDV Nephropathy
4. Lipid lowering ass side effect
5. Anti HT/HAART drug interaction
6. Proteinuria
7. CKD and RRT
Acute Renal Failure

- Pre-renal causes
  - Glomerular disease
    - Inflammation (glomerulonephritis)
    - Thrombosis
  - Tubular injury
    - Ischaemia
  - Interstitial nephritis
    - Toxins
- Intrinsic renal causes
- Post-renal causes
  - Vascular disease
    - Inflammation (vasculitis)
    - Occlusion (thrombosis or embolism)

HIVAN – Pathologic findings

- Tubular microcystic dilatation
- Interstitial leukocyte infiltration
- Interstitial fibrosis
- FSGS
- 1986 AJKD: Weiss coins the term “glomerular collapse”
A,B. tubulo-reticular structure

C. Confronting cylindrical cisternae
HAART and HIVAN Incidence
12-Year Cohort Study

- Risk of HIVAN low in patients without AIDS
- **NO HIVAN when HAART used without AIDS occurrence**
- Lower HIVAN associated with NRTI and HAART use compared with no ART in patients with AIDS (p < 0.001 for trend)

Numbers in bars represent point estimates for HIV-associated nephropathy incidence in cases per 1000 person-years. Brackets above bars represent upper limits of 95% confidence intervals.

HIVAN Treatment

- No controlled randomized trials
- HAART
- Glucocorticoid therapy
- ACE-i/ARB
- Dialysis
- Transplant

Hopkins Nephrology HIV Cohort
ARV Treatment of HIVAN:
Dialysis Free Survival Estimates
Atta et al., Nephrol Dial Transpl, 2006

Dialysis-free Survival

ARV Treatment (n=26)

No ARV (n=10)

P = (0.025)
HIV-1 CD IgA nephropathy
HIV Ag + polyclonalAb

Kidney

Immune complex

Blood

idiotypic Ab

Renal disease

HIV Immune Complex
### Potential Causes of Chronic Kidney Disease in HIV-infected Individuals and Reported Associated Conditions

<table>
<thead>
<tr>
<th>Condition</th>
<th>Associations</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV-associated nephropathy</td>
<td></td>
</tr>
<tr>
<td>HIV-immune complex disease</td>
<td></td>
</tr>
<tr>
<td>Immune complex-mediated GN</td>
<td></td>
</tr>
<tr>
<td>IgA nephritis</td>
<td>HBV, HCV, syphilis, SLE</td>
</tr>
<tr>
<td>Postinfectious GN</td>
<td>HBV, HCV, and/or mixed cryoglobulinemia</td>
</tr>
<tr>
<td>Membranous nephritis</td>
<td>HBV, HCV, syphilis, SLE</td>
</tr>
<tr>
<td>Membranoproliferative GN</td>
<td>HBV, HCV, and/or mixed cryoglobulinemia</td>
</tr>
<tr>
<td>Mesangial proliferative GN</td>
<td>HCV</td>
</tr>
<tr>
<td>Fibrillary or immunotactoid GN</td>
<td>HCV</td>
</tr>
<tr>
<td>Mixed inflammatory or sclerotic variant</td>
<td></td>
</tr>
<tr>
<td>Lupus-like nephritis</td>
<td>Drugs, cytomegalovirus, EBV,BK virus,</td>
</tr>
<tr>
<td></td>
<td><em>Cryptococcus neoformans</em> tuberculosis, adenovirus</td>
</tr>
<tr>
<td>Interstitial nephritis</td>
<td></td>
</tr>
<tr>
<td>Thrombotic microangiopathies</td>
<td></td>
</tr>
<tr>
<td>Minimal change glomerulonephritis</td>
<td></td>
</tr>
<tr>
<td>Diabetic nephropathy</td>
<td></td>
</tr>
</tbody>
</table>
What is the Dx of ARF in HIV (by KBx)
ARF severe enough, not thought to be associated with prerenal causes or ATN

Criteria
1. Scr ↑ 1.2 → 2.0 mg/dL
2. normal or increase kidney size

N = 60
1. HUS 35%
2. ATN (ischemic-toxic/rhabdomyolysis) 26%
3. Obstructive renal failure (extrinsic / drug induced 2° paraprotein precipitation) 17%
4. HIVAN 15%
5. AIN 2%
6. Various glomerulonephritis 4%
**Dx of ARF in HIV (by KBx) - ambulatory type in Bamrasnaradura Institute yr 2007-2008**

- **N = 10**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIN</td>
<td>4</td>
</tr>
<tr>
<td>TDF Nephrotoxic</td>
<td>2</td>
</tr>
<tr>
<td>Post infectious GN</td>
<td>1</td>
</tr>
<tr>
<td>IDV Nephrotoxic</td>
<td>1</td>
</tr>
<tr>
<td>Ig AN</td>
<td>1</td>
</tr>
<tr>
<td>FSGS</td>
<td>1</td>
</tr>
</tbody>
</table>
### Renal Bx 152 HIV-infected pt

<table>
<thead>
<tr>
<th>Renal Pathology</th>
<th>Count (Percentage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIVAN</td>
<td>53 (34.9%)</td>
</tr>
<tr>
<td>FSGS</td>
<td>34</td>
</tr>
<tr>
<td>AIN</td>
<td>12</td>
</tr>
<tr>
<td>DN</td>
<td>8</td>
</tr>
<tr>
<td>MPGN</td>
<td>8</td>
</tr>
<tr>
<td>Post IDGN</td>
<td>7</td>
</tr>
<tr>
<td>HT nephrosclerosis</td>
<td>5</td>
</tr>
<tr>
<td>MN</td>
<td>5</td>
</tr>
<tr>
<td>IgAN</td>
<td>3</td>
</tr>
</tbody>
</table>

### Dominant renal pathology in 89 MHBB kidney tissue donors

<table>
<thead>
<tr>
<th>Renal Pathology</th>
<th>Count (Percentage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterionephrosclerosis</td>
<td>15 (16.8%)</td>
</tr>
<tr>
<td>HIVAN</td>
<td>13 (14.6%)</td>
</tr>
<tr>
<td>Glomerulonephritis</td>
<td>8 (8.9%)</td>
</tr>
<tr>
<td>Chronic Pyelonephritis</td>
<td>7</td>
</tr>
<tr>
<td>AIN</td>
<td>5</td>
</tr>
<tr>
<td>DN</td>
<td>3</td>
</tr>
</tbody>
</table>

---

John Hopkins, Baltimore, USA,  
Mean HIV RNA 123,388, Black 91%  
(Berliner, Am J Nephrol 2008)  

Mount Sinai, New York, USA,  
HIV RNA < 400 (24/80, 30%) Black 48.3%  
(Wyatt CM, Kidney Int 2009)
ART and Long term side effect

Relevant issues:
- ART/OI
- Lactic acidosis
- Neuropathy
- Anemia
- Hepatitis
- DM, HT
- Dyslipidemia
- Liposystrophy
- end organ damage
  - CHD, CKD, CVD
HIV related Renal Disease in Thailand year 2006 - 2008

Bamras ART cases ~ 6000+
Renal Disease cases ~ 30+

incidence 5 cases per 1000 person-year
The Spectrum of renal lesions among HIV-infected patients in Thailand’s HAART Era

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Paisit Paueksakon, MD²
Somchai Eiam-ong, MD³

¹Bamrasnaradura Institute,
²Phramongkutkloa College of Medicine, ³King Chulalongkorn Memorial Hospital
1. Adult age $\geq$ 18 yr with evidence of HIV infection
2. HIV RNA < 400 copies/mL
3. 24 hr Urine protein > 0.3 g/day or UPCR > 0.3 g/g Cr
4. Receiving CART

1. Contraindication to perform kidney biopsy
2. Inadequate renal tissue for pathological interpretation
Result (1)

Renal pathology (LM/IF 29/29, LM/IF/EM 21/29)

- **HIV specific lesion**
  - IgM: 6
  - IgA: 8
  - MN: 2
  - FSGS: 6
  - TDF Nephrotoxic: 3
  - AIN: 2

- **HIV non-related lesion**
  - post ID GN: 1

- **Immune cpx GN**
  - lupus-like GN: 1

- **Renal lesion related to HIV specific Rx**
  - DN: 3

- **Metabolic ass with ART**
  - 3

- **Other Renal lesion related**
  - 5
HIV Immune complex kidney disease

: The emerging finding in an old disease

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S. Eiam-ong³, K. Tungsanga³
V. Sitprija⁴

¹Bamrasnaradura Institute, ²Phramongkutkloa College of Medicine
³Chulalongkorn University Hospital, ⁴Queen Saovabha Memorial Institute
# Patients Characteristic (1)

<table>
<thead>
<tr>
<th>Patients</th>
<th>Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (Male : Female)</td>
<td>21 : 6</td>
</tr>
<tr>
<td>Risk factors (Hetero,Homo,IDU)</td>
<td>24 : 1 : 2</td>
</tr>
<tr>
<td>Proteinuria present post HIV infection</td>
<td>24/27</td>
</tr>
<tr>
<td>Proteinuria present post CART</td>
<td>20/27</td>
</tr>
<tr>
<td>Present with Cr&gt; 2 mg/dL</td>
<td>5/27</td>
</tr>
<tr>
<td>Mean age +SD (yr)</td>
<td>48.6 + 12.6</td>
</tr>
<tr>
<td>Mean time of HIV infection +SD (yr)</td>
<td>8.9 + 3.7</td>
</tr>
<tr>
<td>Mean time of CART +SD (mo)</td>
<td>73.2 + 37.0</td>
</tr>
<tr>
<td>Median CD4 (cells/ml)</td>
<td>404.2</td>
</tr>
<tr>
<td>Median time from proteinuria to KBX +SD (mo)</td>
<td>20.5</td>
</tr>
<tr>
<td>Mean 24-hr urine protein +SD (g/day)</td>
<td>2.1 + 1.8</td>
</tr>
</tbody>
</table>
Result(1)

Renal pathology (LM/IF 27/27, LM/IF/EM 21/27)

- IgAN
- Post IDGN
- MN
- IgMN
- FSGS
- Lupus like

- IgAN: 5
- MN: 4
- Post IDGN: 10
- IgMN: 1
- FSGS: 6
- Lupus like: 1
Result(3) Clinical Characteristic

Various type of Immune complex GN with common finding

1. Asian race, Heterosexual risk group
2. Proteinuria present post CART
3. Long duration of HIV infection, all receiving CART
4. Long duration of CART with relatively high CD4, HIV RNA < 400
5. Relatively slow progression (Long duration of proteinuria before KBX, median time 20.5 mo)
6. Relatively low level proteinuria, mean = 2.1 g/day
7. Relatively high percentage of T2DM (37%), HT (55.6%)
Conclusions

- A noticeable number of HIVICK cases has emerged and documented in Thailand HAART era

- The Paradoxical phenomena to HIVAN in term of the onset of disease was described

- The existence of a specific link between HIV and various types of HIVICK remains speculative

- The immunosuppressive treatment regimen should be used with caution between risk of renal progression and drug side effect
Glomerular disease in setting of HIV with HBV, HCV co-infection

membranous GN
membranoproliferative GN
cryoglobulinemic GN

: relatively rare
NAP database – TDF and IDV usage 2008/2009

Wisit Prasithsirikul, MD
Bamrasnaradura Institute
# Renal Effects of Commonly Used Medications in HIV-Infected Persons

<table>
<thead>
<tr>
<th>Drug</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atazanavir</td>
<td>Atazanavir-containing renal calculi; acute interstitial nephritis (hypersensitivity-like reaction; one case); renal stones resolve with discontinuation</td>
</tr>
<tr>
<td>Indinavir</td>
<td>Nephrolithiasis; crystalluria; pyuria; rates of nephrolithiasis are higher if RTV-boosted; most cases resolve with discontinuation</td>
</tr>
<tr>
<td>Tenofovir</td>
<td>Proximal renal tubule toxicity; Fanconi syndrome; may be concentration-dependent; risk may be increased with PIs; improves with discontinuation</td>
</tr>
</tbody>
</table>

Non-ARVs with nephrotoxic potential: amphotericin B, acyclovir, adefovir, cidofovir, foscarnet, pentamidine, sulfonamides
TENOFOVIR

- Tenofovir closely related to adefovir
- Adefovir is a well described nephrotoxin
- Tenofovir freely filtered; also secreted by proximal tubule
- Nephrotoxicity vigilance in clinical trials – no nephrotoxicity reported
Renal Transporters and Tenofovir
Fanconi Syndrome

Hypophosphatemia, acidosis, glycosuria, aminoaciduria, hypokalemia = FANCONI SYNDROME
TDF Associated With Increased Risk for Proximal Renal Tubulopathy (PRT)

- Cross-sectional analysis of Swiss HIV Cohort Study (N = 1202)
- PRT = pathological status of ≥ 3 of the following 4 measures: fractional excretion (FE) of phosphate or uric acid, protein/creatinine ratio in urine, euglycemic glucosuria
  - Incidence of PRT highest in patients receiving TDF plus a PI (vs no TDF, no PI): OR: 7.1 (95% CI: 2.5-19.8; P < .001)

Tenofovir induced mitochondria injury

A. Tubular cell necrosis, loss of brush border
B. Abnormal mitochondria enlarged in shape, broken distorted cristae
TDF nephrotoxicity

Grade 1 normal structure
2 tubular changes
3 tubulo-interstitial inflammation, cell infiltration
4 tubulo-interstitial fibrosis
5 Glomerulosclerosis, tubulo-interstitial fibrosis
Grade 1 TDF : normal structure
Grade 2 TDF : tubular epithelial degeneration
Grade 3 TDF case 1: tubular epithelial damage focal necrosis, focal dense interstitial inflammation.
Grade 3 TDF : case 2
Grade 3 TDF: case 3 ATN, tubulorrhexis, fibrinoid changes of glomeruli, interstitial edema
Grade4 TDF

case1
Grade 4 TDF case 2

Tubuloepithelial degeneration, focal necrosis, focal regeneration

Patchy tubulointerstitial fibrosis
Grade 5 TDF

Case 1
Grade 5 TDF :case 2
Quiz
## Screening for tenofovir renal toxicity

<table>
<thead>
<tr>
<th>Screening</th>
<th>Frequency</th>
<th>Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) eGFR (aMDRD)</td>
<td>Prior to starting tenofovir, after 2-4 and 12 weeks; then every 3-6 months</td>
<td>Consider stopping tenofovir if:</td>
</tr>
<tr>
<td>b) serum phosphate</td>
<td></td>
<td>• Confirmed significant hypophosphatemia of renal origin and no other cause</td>
</tr>
<tr>
<td>c) urine dipstick analysis</td>
<td></td>
<td>• Progressive decline in eGFR and no other cause</td>
</tr>
<tr>
<td><strong>Measure UP/C</strong></td>
<td></td>
<td>• Confirmed proximal renal tubulopathy / Renal Fanconi syndrome</td>
</tr>
<tr>
<td>• decline in eGFR (deterioration &gt;10ml/min compared to pre-tenofovir level &amp; eGFR&lt;90 ml/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Confirmed hypophosphatemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• If urine dipstick proteinuria &gt;1+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test Name</td>
<td>Result</td>
<td>Reference Range</td>
</tr>
<tr>
<td>-----------------</td>
<td>--------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1.0</td>
<td>0.7-1.2</td>
</tr>
<tr>
<td>eGFR</td>
<td>87.10</td>
<td></td>
</tr>
<tr>
<td>Phosphorus</td>
<td>2.8</td>
<td>2.7-4.5</td>
</tr>
<tr>
<td>Urine Phosphorus</td>
<td>29.3</td>
<td>LO</td>
</tr>
<tr>
<td>Fe P</td>
<td>13.8</td>
<td>if &gt; 20% then sugge%</td>
</tr>
<tr>
<td>Tmp/GFR</td>
<td>2.4</td>
<td>LO</td>
</tr>
</tbody>
</table>

Interpretation: eGFR = Kidney damage with mild decreased GFR (Stage 2)

Comment:

Requested By Doctor: รักิทธิศักดิ์ ประทีปศักดิ์กุล
Requested Date & Time: 17/05/2010 08:26:00

Reported By: น.ส.สายฝน หอมเดื่อขวัญ
Reported Date & Time: 17/05/2010 09:29:24

Approved By: น.ส.สายฝน หอมเดื่อขวัญ
Approved Date Time: 17/05/2010 09:29:26

Page 1/1
Kidney biopsy complication
Renal calculi in which ATV content was 40-50% by weight; ATV is excreted as unchanged drug in the urine; the solubility of ATV is increased in an acidic environment.

Indinavir crystal
IDV pseudo-renal abscess
Indinavir induced Ureteropathy
Flank pain and ARF
Retrograde pyelography
Case 2
## ARV Dosing in Renal Insufficiency

<table>
<thead>
<tr>
<th>ARV Class</th>
<th>Recommendation for Severe Renal Insufficiency (&lt; 30 mL/min)</th>
</tr>
</thead>
</table>
| Entry inhibitors     | T20: no dose adjustment  
                      MVC: use with caution if with a CYP inhibitor and CLcr < 50 mL/min |
| Protease Inhibitors  | No dose adjustment necessary                               |
| Nonnucleoside RT     | No dose adjustment necessary  
                      Caution: TDF/FTC/EFV not recommended if CLcr < 50 mL/min |
| Integrase inhibitors | No dose adjustment necessary                               |
| Nucleoside RT        | All require dose adjustment if CLcr < 30 mL/min  
                      FDCs of ZDV/3TC ± ABC and TDF/FTC/EFV not recommended if CLcr < 50 mL/min  
                      TDF/FTC (FDC) not recommended if CLcr < 30 mL/min |
# Protease Inhibitor Elimination

<table>
<thead>
<tr>
<th>PI</th>
<th>Elimination</th>
<th>Renal Insufficiency (&lt; 30 mL/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>APV</td>
<td>75% of dose accounted for as metabolite in feces</td>
<td>Has not been studied.</td>
</tr>
<tr>
<td>ATV</td>
<td>79% of a dose is recovered in feces; 13% of the ATV dose is recovered in the urine, 7% is unchanged ATV.</td>
<td>AUC ratio in renal failure (&lt;30 mL/min)/Control = 1.19</td>
</tr>
<tr>
<td>DRV/r</td>
<td>80% of dose is recovered in feces; unchanged DRV accounted for 41% in feces and 8% in urine.</td>
<td>Has not been studied. Not different if CLcr &gt; 30 mL/min.</td>
</tr>
<tr>
<td>LPV/r</td>
<td>83% of a dose is recovered in feces; unchanged LPV accounted for 20% of dose in feces; less than 3% of unchanged drug in urine</td>
<td>Has not been studied.</td>
</tr>
</tbody>
</table>
Antiretroviral Drug Dosing Information

- www.aidsinfo.nih.gov
  DHHS Guidelines for use of antiretroviral agents and updated drug interaction tables
- www.hivinsite.com
  Updated drug database with references
- Micromedex
  Comprehensive drug database; subscription required
- Guidelines for the Management of Chronic Kidney Disease in HIV-Infected Patients
  Recommendations of the HIV Medicine Association of the Infectious Diseases Society of America
Unawareness of practice for

1. Screening kidney disease
2. Toxicity monitoring
3. CKD prevention and management
4. ESRD management conflict
# Kidney disease: diagnosis, prevention and management

<table>
<thead>
<tr>
<th>Proteinuria / microhaematuria</th>
<th>eGFR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≥60 ml/min</td>
</tr>
<tr>
<td><strong>UP/C &lt;0.5 or UA/C &lt;0.3</strong></td>
<td>Regular Follow-up</td>
</tr>
<tr>
<td><strong>UP/C 0.5-1 or UA/C 0.3-0.7</strong></td>
<td></td>
</tr>
<tr>
<td>- haematuria</td>
<td>• Check risk factors for CKD and nephrotoxic medication</td>
</tr>
</tbody>
</table>
| + haematuria                 | • Perform renal ultrasound  
          • If haematuria present with any level of proteinuria refer to nephrologist; otherwise consider referral | • Perform renal ultrasound  
          • Refer to nephrologist |
| **UP/C <1 or UA/C <0.7**     |               |             |            |
### Management of nephropathy in HIV-positive patients

<table>
<thead>
<tr>
<th>Prevention of progressive renal disease</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Antiretroviral therapy</td>
<td>Start ART immediately where HIV-associated nephropathy (HIVAN) or HIV immune complex disease strongly suspected. Renal biopsy to confirm histological diagnosis recommended</td>
</tr>
<tr>
<td>2. Start ACE inhibitors or angiotensin-II receptor antagonists if:</td>
<td>Monitor eGFR and K+ level closely on starting treatment or increasing dose</td>
</tr>
<tr>
<td>a) Hypertension, and/or</td>
<td>a) Blood pressure target; &lt;130/80 mmHg</td>
</tr>
<tr>
<td>b) Proteinuria</td>
<td></td>
</tr>
<tr>
<td>3. General measures:</td>
<td>CKD and proteinuria are independent risk factors for CVD</td>
</tr>
<tr>
<td>a) Avoid nephrotoxic drugs</td>
<td></td>
</tr>
<tr>
<td>b) Life style measures (smoking, weight, diet)</td>
<td></td>
</tr>
<tr>
<td>c) Treat dyslipidaemia and diabetes</td>
<td></td>
</tr>
<tr>
<td>d) Adjust drug dosages where necessary</td>
<td></td>
</tr>
</tbody>
</table>
Management regimen

1. Assess volume status, resuscitate
2. Identify E’lyte, acid-base disorder and treat
3. Look for and avoid nephrotoxins (drugs)
4. Hx of current and recent use of traditional medicine
5. Exclude infection
6. Identify underlying cause of renal dysfunction
7. Check UPCR, UA, Renal US
8. KBx if uncertain of cause, slow resolution, proteinuria
9. Short course of dialysis (with favourable outcome) if meet criteria to dialysis
To the AIDS Doctors:
Renal Disease is a Major Issue

• ARF common in HIV+ patients
• Widespread use of tenofovir, and other nephrotoxic drugs has increased the renal issues for AIDS doctors.
• Renal guidelines for CKD appear in the ID literature (Clin Inf Dis 2005).
• Evidence for Global crisis emerging in renal disease