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# IMPA NEWS

THE OFFICIAL NEWS LETTER OF THE INDEPENDENT MEDICAL PRACTITIONERS ASSOCIATION

## IMPA News

- An excellent medical update programme on "Over view of Diabetes Mellitus" By Dr. Kayathri Periasamy, Consultant Physician and “Management of Chronic Renal Failure" By Dr. Arjuna Marasinghe Consultant Nephrologist was held on Sunday 27th May 2018 at the OPA Auditorium sponsored by Novartis (A Baur & Company).

- The IMPA council has requested the Dr. A.H.A. Hazari to inform the Director General Health Services, Dr. Anil Jasinghe who is also the Chairman of the PHSRC to request all circulars released by the Ministry of Health to be forwarded to the IMPA and all medical practices registered with the PHSRC. This decision was made by the council as a letter circulated by the Ministry of Health (Family Health Bureau) on “ Fever in Pregnancy and Post-partum period” to be circulated among the medical profession was not sent to the IMPA nor to any practice registered with the PHSRC.

- The IMPA Hon. Joint Secretary, Dr. Jayantha Jayatissa has been elected at the new President of the College of General Practitioners at the AGM held on Sunday 20th May 2018. The IMPA representative in the PHSRC Dr. D.K.D. Mathew who is also a council member of the IMPA has been elected as the Vice President of the CGP. The IMPA wish them well in their new appointments. The induction of the President of the CGP was held on Saturday 9th June 2018 at the Hotel JAIC Hilton

- The IMPA was invited by the SLMA for a discussion / brainstorming session on Human Resources for Primary Care - The Private Sector Perspective with Prof Buchan on Thursday 31st May 2018 at the Lionel Memorial Auditorium of the SLMA.

- The SLMA invited 15 IMPA members to participate in the Pre-congress workshop “2nd Annual Mini Congress on Wound Care” on 1st June 2018 at the Auditorium of the College of Surgeons of Sri Lanka.

- 15 IMPA members were invited by the Ministry of Health to participate in a Symposium on “Containment of Antimicrobial Resistance” on Monday 18th June 2018 at the Waters Edge, Battaramulla. The IMPA has also been requested to nominate one member for the Panel Discussion on “Averting tomorrows nightmare by responsible use of antibiotics”

*Editor - Dr A L P de S Seneviratne*

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# MANAGEMENT OF CHRONIC KIDNEY DISEASE

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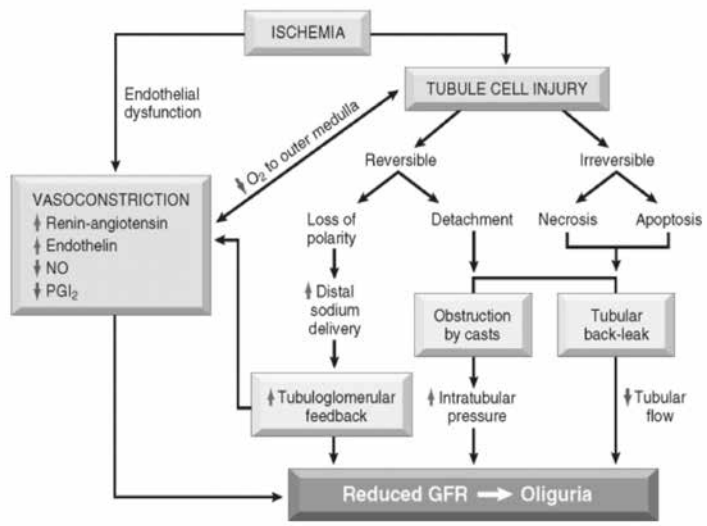
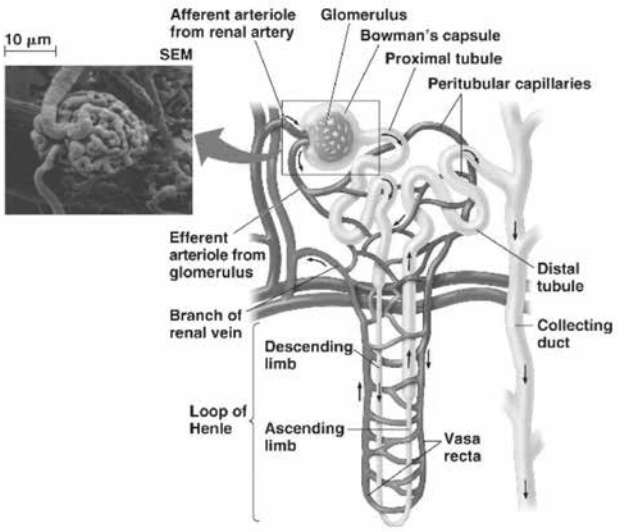
Consultant Nephrologist
CSTH Kalubowila

## Chronic Kidney Disease (CKD)
### Pathophysiology

- **Repeated injury** to kidney




**Causes of CKD include**

I. Diabetes Mellitus
II. Hypertension
III. Autoimmune Diseases
IV. UTI or Urinary Tract Infection
V. Kidney Stones
VI. Lower Urinary Tract Obstruction
VII. Systemic Infection
VIII. Drug Toxicity
IX. Neoplasia
X. **CKDu (CKD of unknown origin)**

**CKD in patients with Diabetes Mellitus (DM)**

- The prevalence of chronic kidney disease (CKD) is rising globally, and is attributed to the epidemic of type 2 diabetes mellitus
- The age-sex standardized prevalence of diabetes for Sri Lankans aged ≥ 20 years
- 10.3% (9.4-11.2%) males and 10.9% (9.7- 12.1%), females
- Diabetes prevalence was higher in the urban population compared with rural [16.4% (13.8-19.0%) vs. 8.7% (7.8-9.6%); P < 0.001].
- The projected diabetes prevalence for the year 2030 is 13.9%.

**The natural history of diabetic nephropathy**

- Type 1 DM
- The earliest - incipient nephropathy
  - appearance of low but abnormal levels (≥ 30 mg/day) of albumin in the urine

**Overt nephropathy**

- Without specific interventions, ~80% of subjects with type 1 diabetes who develop sustained microalbuminuria have their urinary albumin excretion increase at a rate of ~10–20% per year to the stage of overt nephropathy or clinical albuminuria (≥300 mg/24 h or ≥200 μg/min) over a period of 10–15 years
- with hypertension also developing along the way.

- **CKD**
  - Once overt nephropathy occurs, without specific interventions, the glomerular filtration rate (GFR) gradually falls over a period of several years at a rate that is highly variable from individual to individual (2-20 ml • min-1 • year-1).
  - ESRD develops in 50% of type 1 diabetic individuals with overt nephropathy within 10 years and in >75% by 20 years

**Albuminuria and overt nephropathy**

- A higher proportion of individuals with type 2 diabetes
- shortly after the diagnosis of their diabetes
- Without specific interventions, 20-40% of type 2 diabetic patients with microalbuminuria progress to overt nephropathy

***Cont. on page 04***

**CKD and End Stage Renal Disease (ESRD)**

- by 20 years after onset of overt nephropathy, only ~20% will have progressed to ESRD.
- Once the GFR begins to fall, the rates of fall in GFR are again highly variable from one individual to another, but overall, they may not be substantially different between patients with type 1 and patients with type 2 diabetes.
- greater risk of dying from associated coronary artery disease in the older population with type 2 diabetes may prevent many with earlier stages of nephropathy from progressing to ESRD.

**CKD of unknown aetiology (CKD-U)**

- Increase in a new form of CKD which is not attributed to DM, HT, GN or other known aetiologies observed
- Case load more in certain areas i.e. regional clustering
- Insidious which probably starts in second decade of life
- Slowly progressive and asymptomatic until very advanced
- High economical cost for patient, family & state

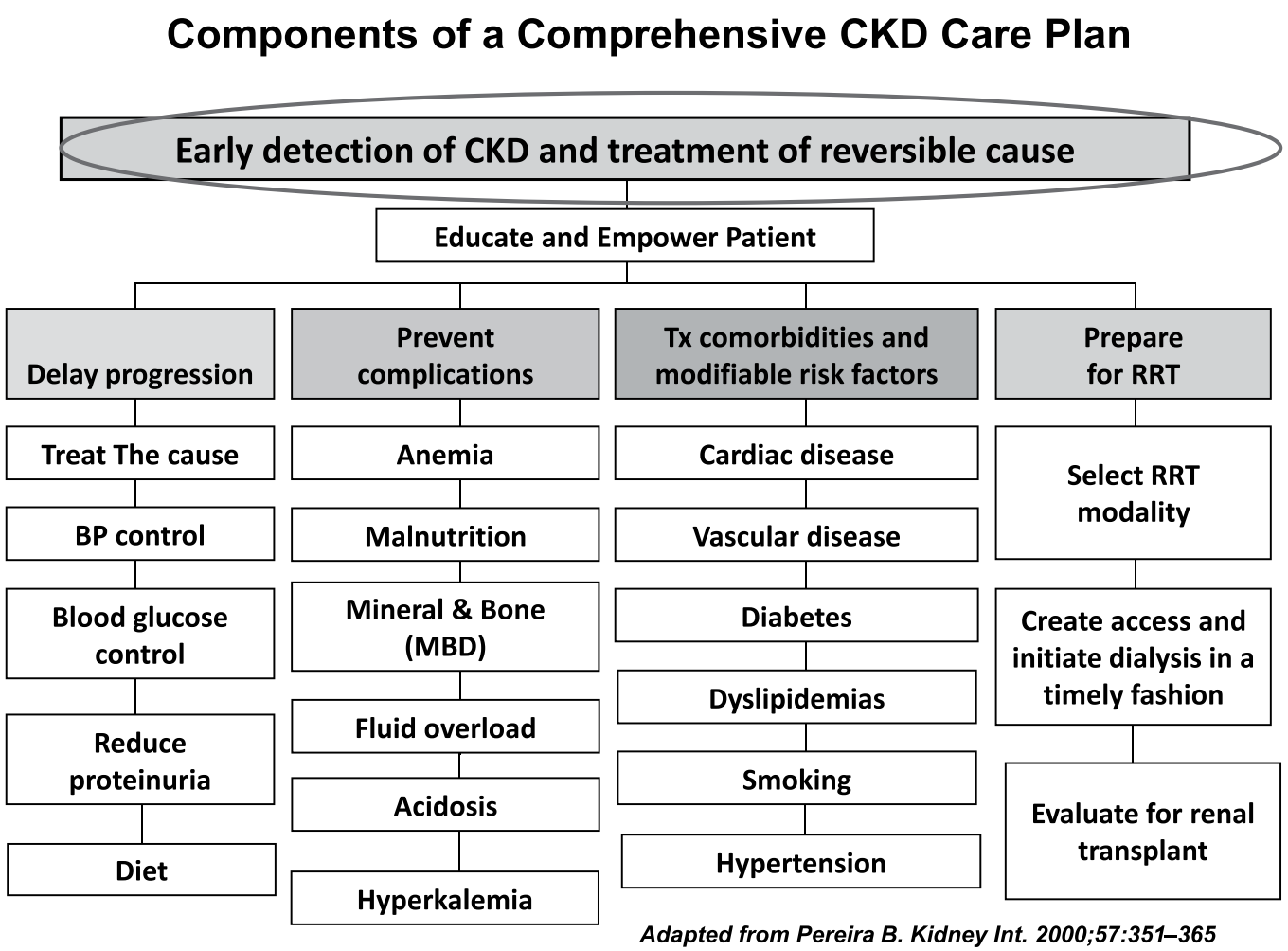
**Case Definition for CKDu**

- Urine ACR ≥ 30 mg/g on two occasions
- No past history of ureteric calculi, glomerulonephritis, pyelonephritis or snake bite
- Not on treatment for diabetes
- Normal HbA1C (< 6.5%)
- If on treatment for raised Blood pressure BP < 140/90m if not on treatment for Blood pressure, BP < 160/100

**Prevalence**

- 15.1% in Anuradhapura ( 17 GN divisions selected from 4 DS areas randomly)
- 20.6% in Polonnaruwa ( 3 GN divisions selected from 1 DS area randomly)
- 22.9% in Badulla ( 2 GN divisions selected from 1 DS area randomly)

**Management of CKD**




*Adapted from Pereira B. Kidney Int. 2000;57:351–365*

*Cont. on page 05*

Criteria for CKD (either of the following present for >3 months)

- Markers of kidney damage (one or more)
  - Albuminuria (AER ≥30 mg/24 hours; ACR ≥ 30 mg/g [>3 mg/mmol])
  - Urine sediment abnormalities
  - Electrolyte and other abnormalities due to tubular disorders
  - Abnormalities detected by histology
  - Structural abnormalities detected by imaging
  - History of kidney transplantation
- Decreased GFR <60 ml/min/1.73 m2 (GFR categories G3aG5)

**Staging of CKD(CGA)**

- Cause
  - fundamental importance in predicting the outcome of CKD and choice of cause-specific treatments
- GFR category
- Albuminuria category
  - fundamental importance in predicting the outcome of CKD and choice of cause-specific treatments

**Table 5 | GFR categories in CKD**

| GFR category | GFR (ml/min/1.73 $m^2$) | Terms |
|---|---|---|
| G1 | ≥90 | Normal or high |
| G2 | 60–89 | Mildly decreased* |
| G3a | 45–59 | Mildly to moderately decreased |
| G3b | 30–44 | Moderately to severely decreased |
| G4 | 15–29 | Severely decreased |
| G5 | <15 | Kidney failure |

**Table 6 | Albuminuria categories in CKD**

| Category | AER (mg/24 hours) | ACR (approximate equivalent) (mg/mmol) | ACR (approximate equivalent) (mg/g) | Terms |
|---|---|---|---|---|
| A1 | <30 | <3 | <30 | Normal to mildly increased |
| A2 | 30-300 | 3-30 | 30-300 | Moderately increased* |
| A3 | >300 | >30 | >300 | Severely increased** |

**Table 7 | Relationship among categories for albuminuria and proteinuria**

| Measure | Categories: Normal to mildly increased (A1) | Moderately increased (A2) | Severely increased (A3) |
|---|---|---|---|
| AER (mg/24 hours) | <30 | 30-300 | >300 |
| PER (mg/24 hours) | <150 | 150-500 | >500 |
| ACR | | | |
| (mg/mmol) | <3 | 3-30 | >30 |
| (mg/g) | <30 | 30-300 | >300 |
| PCR | | | |
| (mg/mmol) | <15 | 15-50 | >50 |
| (mg/g) | <150 | 150-500 | >500 |
| Protein reagent strip | Negative to trace | Trace to + | + or greater |

*Cont. on page 06*

**Indications for referral**

- AKI or abrupt sustained fall in GFR;
- GFR <30 ml/min/1.73 m2 (GFR categories G4-G5)
- a consistent finding of significant albuminuria (ACR >300 mg/g [>30 mg/mmol] or AER>300 mg/24 hours, approximately equivalent to PCR>500 mg/g [>50 mg/mmol] or PER >500 mg/24 hours)
- progression of CKD
- urinary red cell casts, RBC >20 per high power field sustained and not readily explained;
- CKD and hypertension refractory to treatment with 4 or more antihypertensive agents
- persistent abnormalities of serum potassium
- recurrent or extensive nephrolithiasis
- hereditary kidney disease

Diet

- Protein intake
  - Limit protein intake to 0.8 g/kg/day in adults with or without diabetes and GFR < 30 ml/min/1.73 m2
  - Avoid high protein intake >1.3g/kg/day
- Salt intake
  - lowering salt intake to <90 mmol(<2g) per day of sodium (corresponding to 5g of sodium chloride) in adults

Anaemia of CKD

- Diagnose anemia in adults
  - Male - Hb <13.0 g/dl (130 g/l)
  - Females - Hb <12.0 g/dl (<120 g/l).
- Monitor Hb
  - when clinically indicated GFR >60 ml/min/1.73 m2
  - annually in people with GFR 30–59 ml/min/1.73 m2
  - twice per year in people with GFR<30 ml/min/1.73 m2

Iron replacement

- TSAT is< 30% and ferritin is <500 ng/ml (500mg/l)
  - For adult CKD patients with anemia not on iron or ESA therapy - a trial of IV iron
  - in CKD ND patients alternatively a 1-3 month trial of oral iron therapy
- Evaluate iron status at least 3monthly
- Address all correctable causes of anaemia
- Not to initiate if Hb>10g/l
- Target Hb <11.5g/dl
- Epoetin Alfa/beta
  - 50 -150 iu/kg/week
- Darbepoetin
  - 0.45µg/Kg weekly or 0.75 µg/Kg fortnightly
- CERA
  - 0.6 µg/Kg fortnightly or 1.2µg/Kg monthly

**Renal Replacement Therapy**

- Haemodialysis
- Peritoneal Dialysis
- Renal Transplantation

Management of Chronic Kidney Disease;
Shifting the Paradigm

- Diabetes and CKD u epidemic
- Diabetic nephropathy as a leading cause of ESKD
- Treat diabetes optimally
- Delay Progression, treat complications and prepare for RRT
- Individualized treatment of ckd
  - Avoid blanket treatment regimes
- Refer early to nephrologist

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