Renal Disease and Dentistry

by Dr David Lim

21 Jul 2026 (Tuesday) 9-10

Acknowledgement of Country

We acknowledge the Traditional Custodians of this land.
We acknowledge the waterways, the land, the sky and
all who inhabit this place, Country.
We acknowledge their ancestors, and Elders and
recognise those who continue to protect and promote
Aboriginal and Torres Strait Islander cultures.
We acknowledge the past and stand together
for our future.

IMPT!

Learning Objectives

1. Discuss the specific characteristics and dental needs of individuals with kidney disease

2. Perform a risk assessment and make appropriate treatment adaptations to ensure safe and effective dental care.

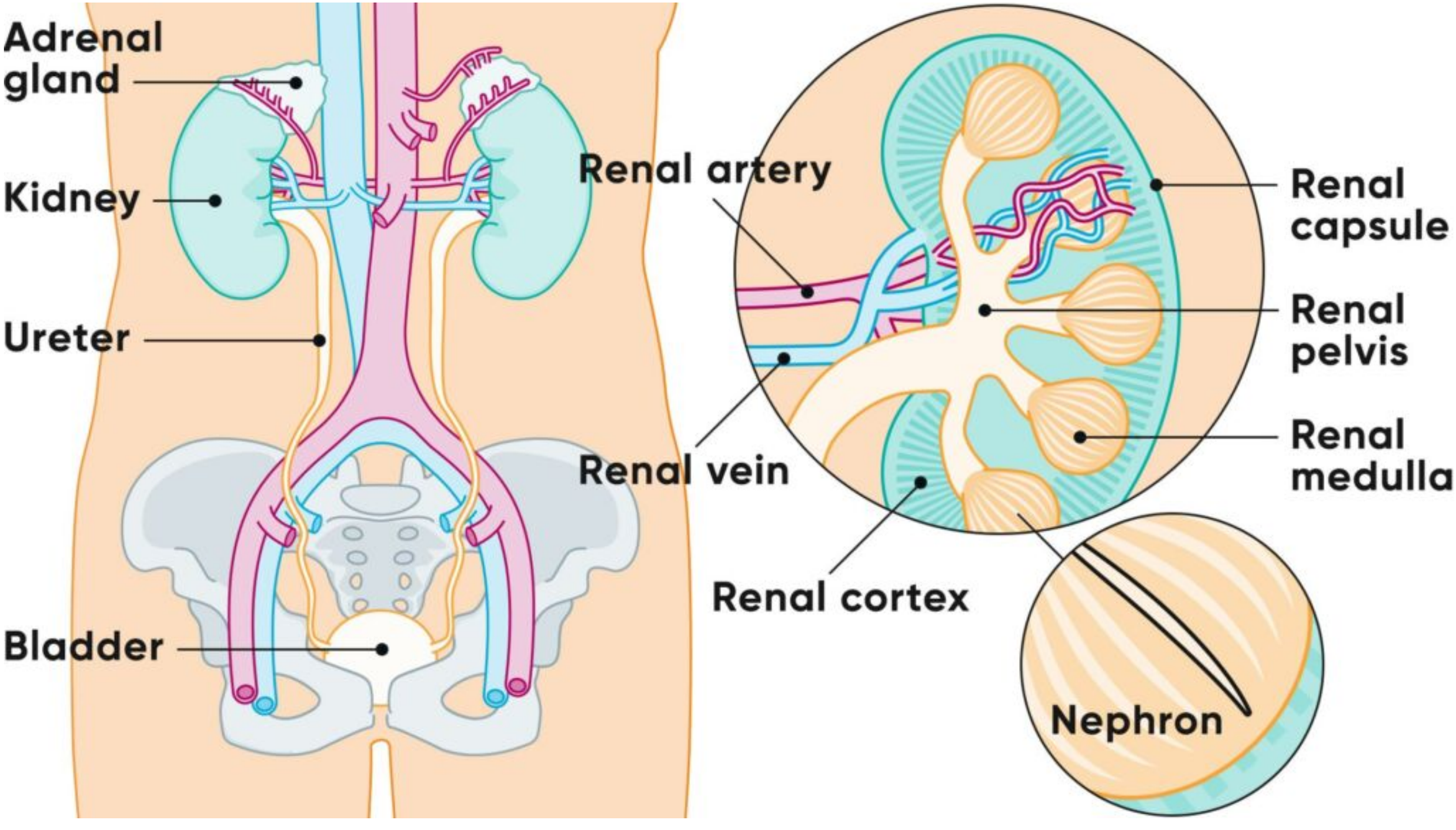
WHAT IS IT ABOUT XXX DISEASE/CONDITION?

- Definition
- Aetiology
- Epidemiology
- General Manifestations/Clinical Features
- General Management/Treatment
- Oral Manifestations
- Oral Management = Risk Assessment and ACCESS acronym

ADC: 5.1	iADH: 5A, 5B, 5C, 6Ci	5.1	Scientific and Clinical Knowledge	apply the social, cultural, biological, biomedical, physical and behavioural sciences in relation to oral health care provision and disease prevention

https://www.usz.ch/en/organs/kidney/

Recap on Structures



Renal physiology

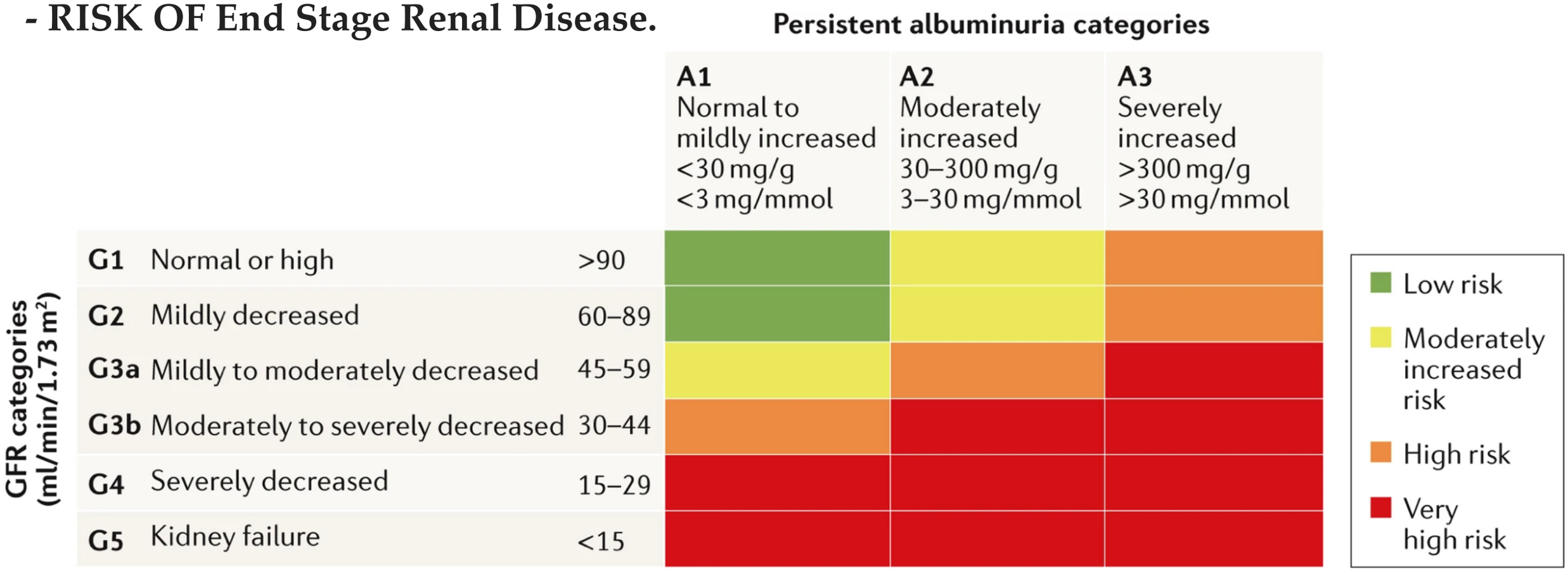
- **Maintain homeostasis by**
    - FLUID-ELECTROLYTE BALANCE - balance Na+ electrolytes, regulate fluid
    - ACID-BASE BALANCE: excrete hydrogen ions and regenerating HCO3-
    - EXCRETE WASTE: Urea, drugs
- **Endocrine function:**
    - Renin,
    - Erythropoietin (stimulate formation of Haemoglobin)
    - Calcitrol (active form of Vitamin D, regulating Ca2+ balance and others)
    - Others (minute amount) e.g. thrombopoietin

3 common Renal Diseases

- Chronic Kidney Disease - caused mainly by HBP and DM
    - Treated with adapted diet/fluids, controlling BP and Blood glucose (diabetes), dialysis, or renal transplant
- Pyelonephritis-affects mainly women due to shorter urethra. Fever, vomiting, back pain
- Kidney stones (nephrolithiasis, urolithiasis/urethral stone, cystolithiasis/bladder stone) cause severe pain (renal colic)
    - Passed out in urine, or dissolved with medication, or removed with surgery.

Kidney Disease Improving Global Outcomes (KDIGO) 2D matrix classification of CKD.

- RISK OF End Stage Renal Disease.



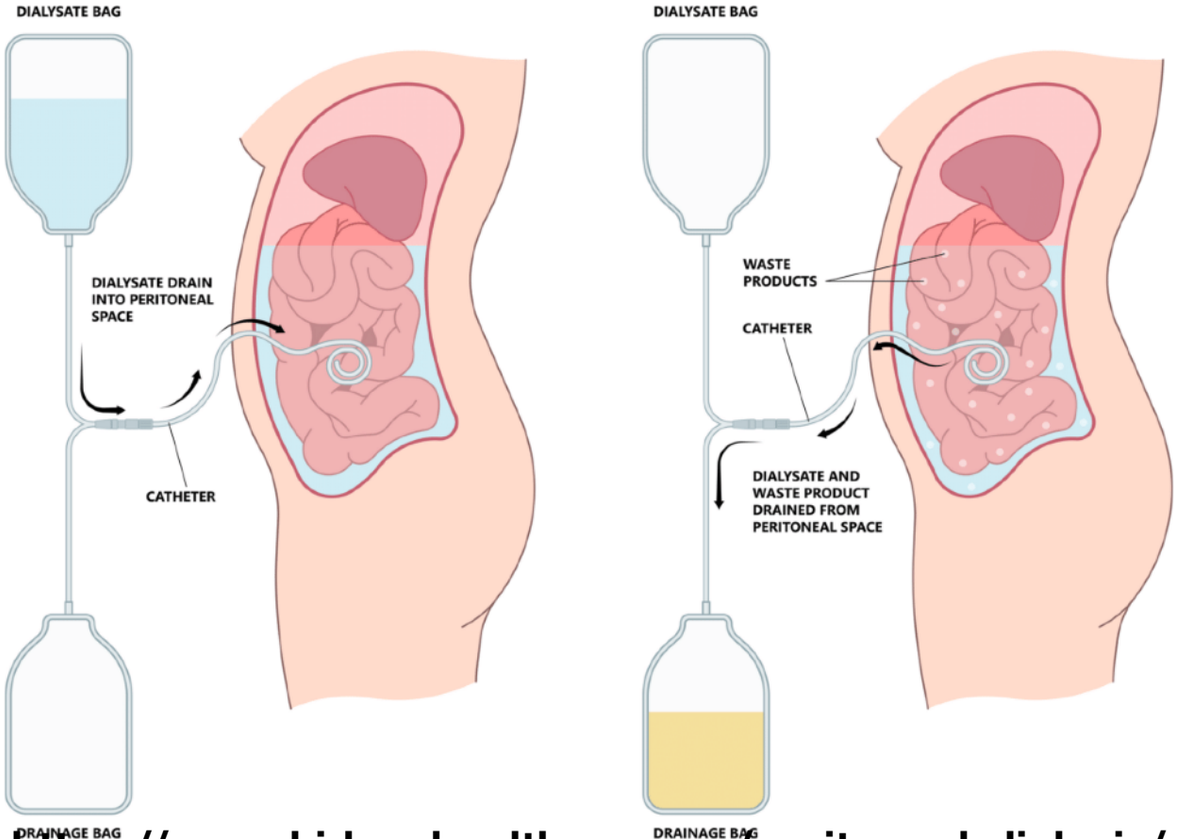
Measuring the exact GFR is a complex, invasive procedure. Instead, doctors use an estimate (eGFR) calculated through a simple formula. [1]

- **The Blood Test:** The lab measures the level of waste products in your blood, primarily **creatinine** (a byproduct of muscle breakdown) or occasionally **cystatin C**. Healthy kidneys filter these wastes out into your urine. If kidney function declines, wastes build up in the bloodstream. [1, 2, 3, 4]
- **The Calculation:** The creatinine level is plugged into a mathematical formula. To ensure accuracy, the calculation also accounts for your **age** and gender

TYPES OF DIALYSIS

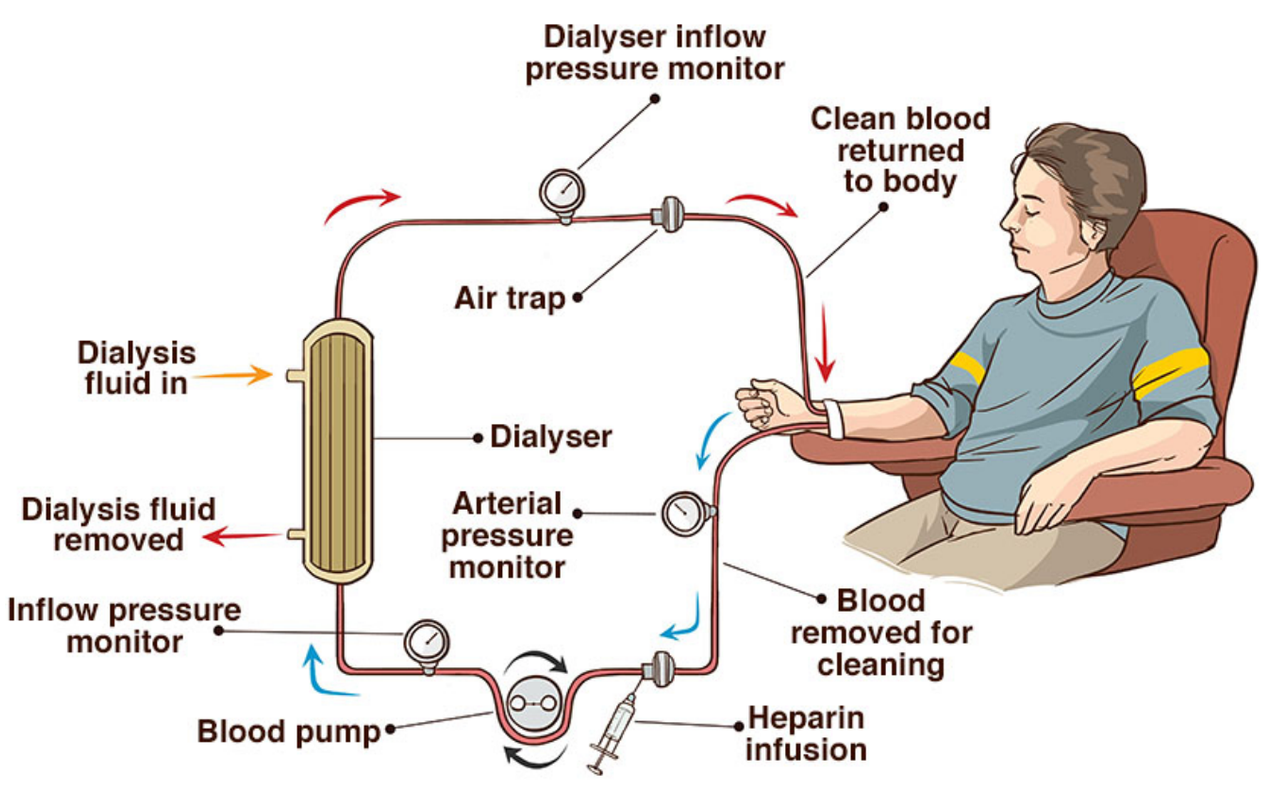
- Haemodialysis - AV fistula on arm, blood drawn into a machine, filtered, and returned. Done <u>usually</u> in a clinic, with anticoagulation (usually LMW Heparin)
- Peritoneal dialysis - catheter inserted into peritoneal cavity where dialysis fluid is introduced. This cleanses waste and fluid is replaced. Repeated several times a day or during sleep, done at home
- Continuous renal replacement therapy (in ICU)

PERITONEAL DIALYSIS



https://www.kidneyhealth.com.sg/peritoneal-dialysis/

Haemodialysis



https://www.healthdirect.gov.au/dialysis

CKD Patient Case

- A 49 y.o. male underwent hemodialysis for End Stage Kidney/ Renal Disease (ESRD). He was referred by a nephrologist for an oral assessment to eliminate potential oral foci of infection prior to planned kidney transplant.

CKD Patient Case

MH:

- CKD-ESRD, Hyperuricaemia
- HTN, HLD
- Osteopenia (renal osteodystrophy)
- Fe deficiency anaemia

MED: HD 3 days a week for 5 years (heparin), Nifedipine, Irbesartan, Furosemide, Cal acetate Vitamin D3, Iron, Atorvastatin, Allopurinol.

1. The patient reports halitosis despite brushing TDS. There is also a metallic taste that improves after HD. What do you think is the reason?
2. The patient has multiple decays and feels tired easily. How are these related or not to CKD?
3. When prescribing analgesics, what medications should be avoided?

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1. High salivary urea levels decomposing to ammonia. Xerostomia additionally contribute to dysguesia (abnormal taste)

The patient has multiple decays and feels tired easily. How are these related or not to CKD?

2. Saliva pH should be alkaline, protecting against decay. Tiredness due to low erythropoietin, and shorted Hb survival due to uraemia. Plus Fe Deficiency

When prescribing analgesics, what medications should be avoided?

3. Ibuprofen, diclofenac, Tramadol are contraindicated in ESRD. In late stage CKD, amoxicillin can be given at half dose

Transplant case

MH:

- History of Renal failure due to bilateral polycystic kidney disease
- Rt kidney transplant 9 years ago
- HTN, Osteopenia
- Absolute polycythaemia (Dx 3 years ago)
- Fe deficiency anaemia

MED: Mycophenolate mofentil, Tacrolimus, Prednisolone (5mg OD). Irbesartan, Trimethoprim/ sulfamethoxazole

1. Why are the patient taking 3 immunosuppressants?
2. What dental risks should we note for these immune suppressants?
3. Will you consider antibiotics prophylaxis when conducting multiple extractions?

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Why are the patient taking 3 immunosuppressants?

1. Reduce risk of T-cell alloimmune rejection

What dental risks should we note for these immune suppressants?

2. Risk of myelosuppression, poor healing, infections, MRONJ (e.g. sirolimus), steroid cover, gingival hyperplasia (cyclosporin etc), candida.
    2.1. The patient might be on long-term prophylactic antiviral (HSV, VZV), anti fungal, or antibiotics

Will you consider antibiotics prophylaxis when conducting multiple extractions?

3. The is a significantly increased risk of post-extraction infection. Some transplant centres might indicate AB cover but evidence is limited. Case-by-case consult please.

Consider graft-v-host disease also.

CKD and dentistry

Oral Findings

- Brittle bone during extractions - inability to convert 25-OH Vitamin D to 1,25-dihydorxyl Vitamin D (active form), phosphate retention and calcium loss
- Osteitis fibrosa cystica (brown tumor, below) from secondary hyperparathyroidism
- Transplant-related
- Risk of bleeding (thrombopoeitin produced by kidney, heparin)

Other considerations

- Immunosuppressant effects
- Dental scheduling with dialysis (heparin, tiredness)
- Prescription and drug elimination considerations



Brown tumors are giant cell focal lesion that arises as a result of abnormal bone metabolism in patients with hyperparathyroidism (HPT). The lesions localize in areas of extensive bone resorption, which is replaced by fibrovascular tissue and giant cells with abundant deposits hemorrhage and hemosiderin.

Shetty AD, Namitha J, James L. Brown tumor of mandible in association with primary hyperparathyroidism: a case report. *J Int Oral Health*. 2015;7(2):50-52.