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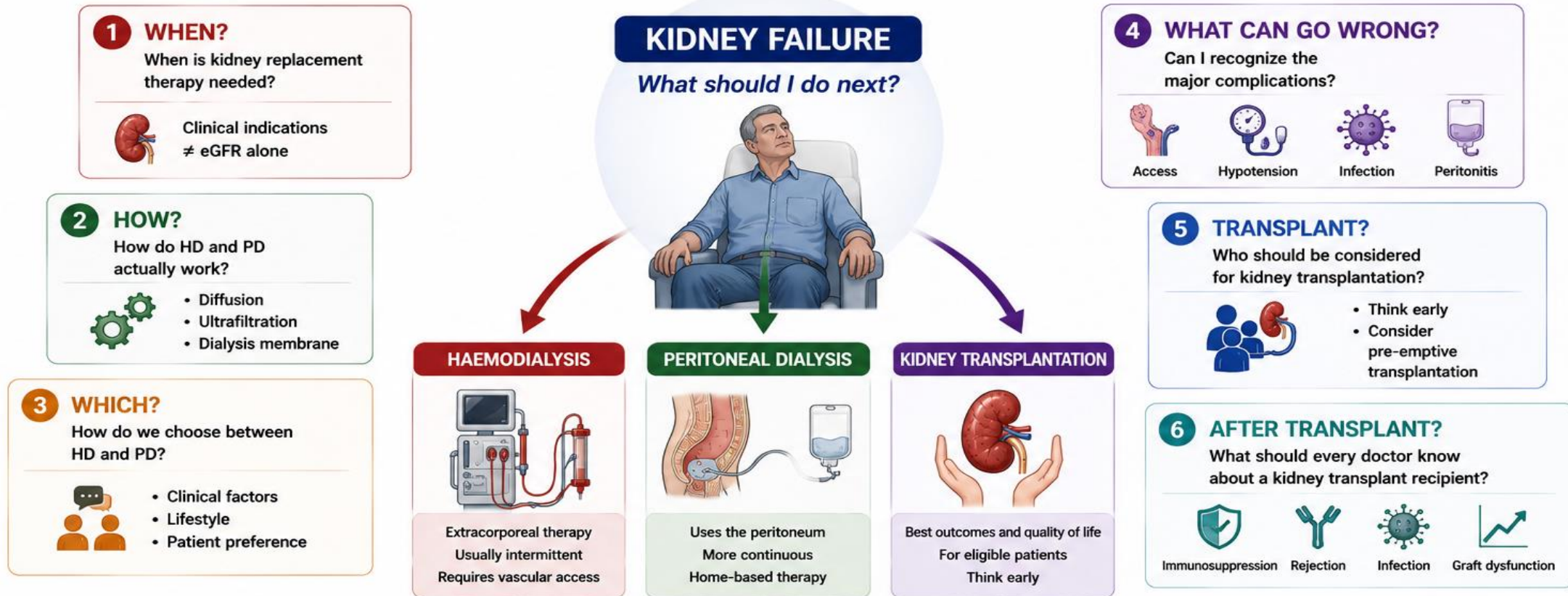
**Kidney Replacement Therapy and Kidney Transplantation
Haemodialysis • Peritoneal Dialysis • Kidney Transplantation**

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What Should You Know After This Lecture?

By the end of this session, you should be able to make the first clinical decisions in a patient approaching kidney failure.



TODAY'S ROADMAP



The goal is not to memorize dialysis techniques — it is to understand how to choose the right strategy for the right patient.



A 62-year-old patient with CKD G5

eGFR 8 mL/min/1.73 m²

K⁺ 4.8 mmol/L

HCO₃⁻ 21 mmol/L

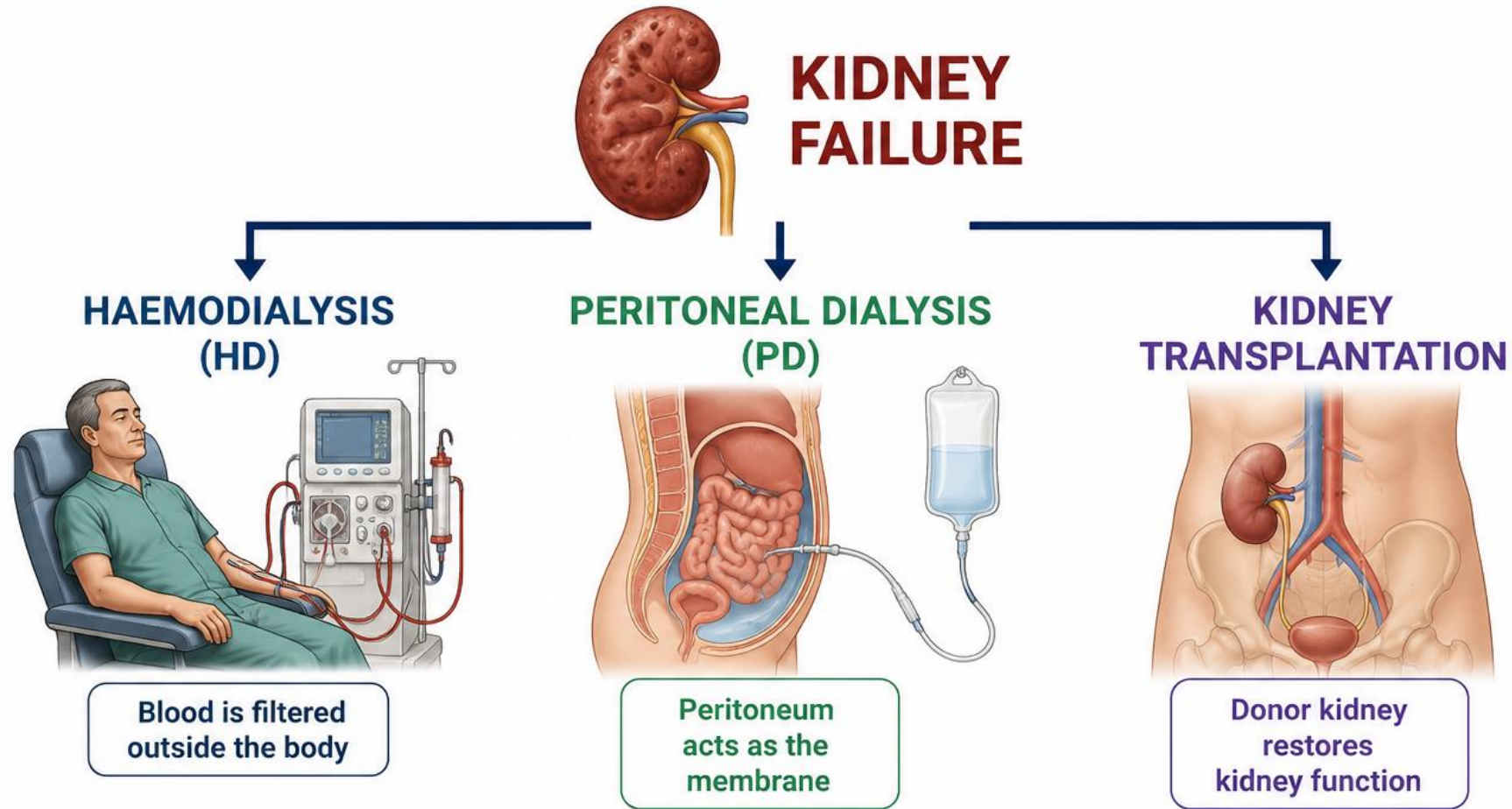
mild ankle oedema

no uraemic symptoms

Does this patient need dialysis today?



Kidney Replacement Therapy (KRT)



Dialysis replaces selected kidney functions.
Transplantation provides the most complete kidney replacement.



The choice of KRT should be individualized and planned before kidney failure whenever possible.

When Should We Start Dialysis?



Our patient (Slide 1):

- eGFR 8 mL/min/1.73 m²
- K⁺ 4.8 mmol/L
- HCO₃⁻ 21 mmol/L
- Mild ankle oedema
- No uraemic symptoms

→ Dialysis today?
NOT NECESSARILY.

eGFR 8 mL/min/1.73 m²

≠

automatic indication for dialysis



We treat the patient – not the eGFR



Uraemic symptoms

encephalopathy,
pericarditis,
nausea, pruritus,
severe symptoms



Volume status

uncontrolled
fluid overload,
pulmonary oedema,
refractory to diuretics



Electrolytes

refractory
abnormalities,
especially
hyperkalaemia



Acid–base balance

severe metabolic
acidosis, refractory
to medical treatment



Nutrition

progressive
deterioration,
malnutrition,
cachexia



Dialysis may NOT be needed yet

Low eGFR + Clinically stable +
Complications controlled medically



Dialysis should be considered



Symptoms or complications
that **cannot** be adequately controlled medically



There is no single eGFR threshold for starting dialysis.

Emergency Dialysis – Remember **AEIOU**



Think about **urgent Kidney Replacement Therapy** when one or more of the following are present.

A

ACIDOSIS



Severe metabolic acidosis

refractory to medical treatment

- especially with haemodynamic compromise or other indications for KRT

E

ELECTROLYTES



Severe / refractory hyperkalaemia

$K^+ \uparrow$ + ECG changes = emergency

- other severe electrolyte abnormalities refractory to medical therapy

I

INTOXICATIONS



Selected dialysable toxins

- Lithium
- Methanol
- Ethylene glycol
- Salicylates*



*depending on clinical severity and toxicological indications

O

OVERLOAD



Refractory pulmonary oedema

despite appropriate medical therapy

- severe fluid overload causing hypoxia or haemodynamic instability

U

URAEMIA



Severe uraemic complications

Encephalopathy
Pericarditis

- bleeding, intractable nausea, pruritus, or other severe uraemic manifestations



AEIOU = think about urgent Kidney Replacement Therapy



The decision is clinical – not every abnormal laboratory value requires immediate dialysis.

Consider severity, symptoms, response to treatment and whether dialysis can correct the problem.

UREMIA

CLINICAL MANIFESTATIONS



GENERAL SYMPTOMS

- Fatigue, weakness
- Loss of appetite
- Weight loss
- Itching
- Sleep disturbances
- Decreased concentration, confusion
- Restless legs syndrome

PLEURITIS (UREMIC PLEURITIS)



- Chest pain
- Pleuritic pain
- Dyspnea
- Pleural effusion

PERICARDITIS (UREMIC PERICARDITIS)



- Chest pain
- Pericardial friction rub
- Pericardial effusion
- Risk of tamponade

NAUSEA, VOMITING, ANOREXIA



- Nausea
- Vomiting
- Loss of appetite
- Metallic taste

SKIN CHANGES



- Dry, itchy skin
- Uremic frost (white crystalline deposits)
- Hyperpigmentation

UREMIA – SIGNS AND SYMPTOMS

EDEMA, HYPERTENSION



- Edema (peripheral, periorbital)
- Fluid overload
- Hypertension

HEMATOLOGIC MANIFESTATIONS



- Anemia
- Easy bruising
- Bleeding tendency
- Platelet dysfunction

NEUROLOGIC MANIFESTATIONS



- Headache
- Drowsiness
- Confusion
- Seizures
- Peripheral neuropathy
- Asterixis (in severe cases)

CARDIOVASCULAR MANIFESTATIONS



- Left ventricular hypertrophy
- Heart failure
- Arrhythmias
- Increased risk of atherosclerosis

EKG CHANGES



- Peaked T waves
- Prolonged PR interval
- Widened QRS complex (in severe hyperkalemia)
- Flattened or absent P waves
- Risk of arrhythmias and asystole

OTHER POSSIBLE MANIFESTATIONS



Renal
osteodystrophy



Endocrine
disturbances
(e.g., secondary
hyperparathyroidism)



Immunodeficiency –
increased susceptibility
to infections



Sexual dysfunction,
amenorrhea,
infertility

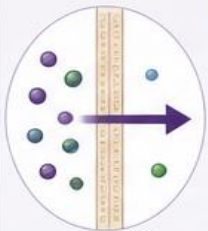
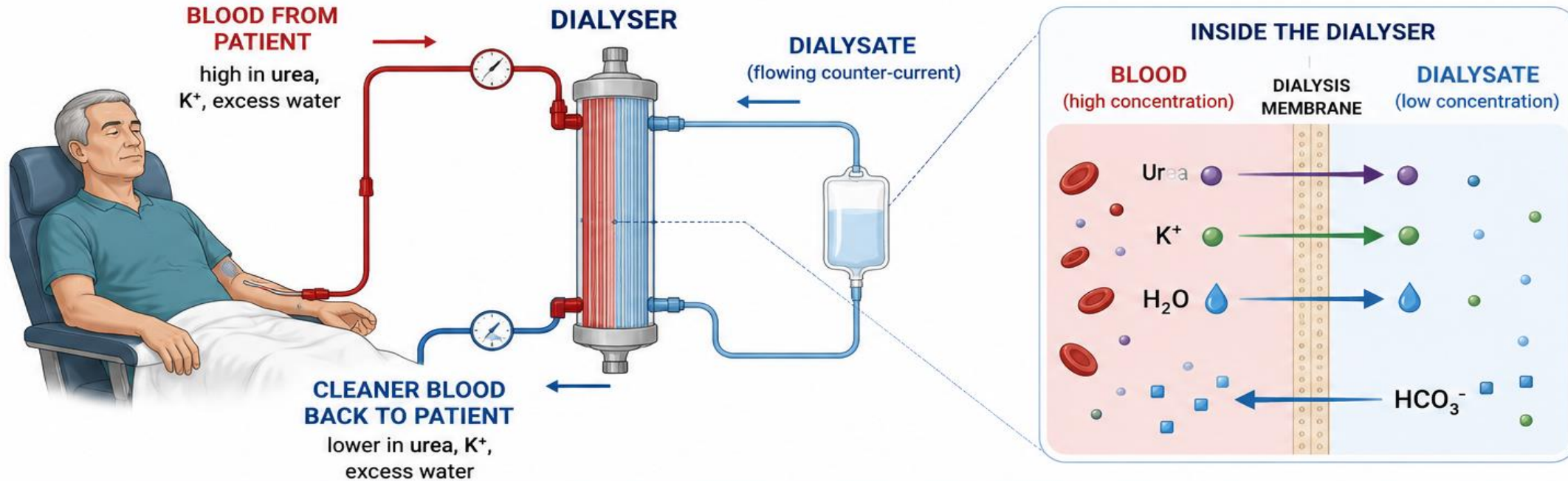


Uremic
retinopathy



Uremic
fetor (bad
breath)

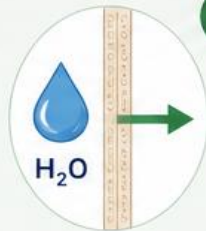
Haemodialysis – How Does It Work?



1 DIFFUSION

Solutes move down a concentration gradient.

Removes: urea, K^+ , creatinine, phosphate and other small solutes



2 ULTRAFILTRATION

Pressure gradient removes excess water.

Removes: excess water (fluid overload)



3 DIALYSATE

Dialysate composition corrects electrolyte and acid-base balance.

Examples: $K^+ \downarrow$ $HCO_3^- \uparrow$ (acidosis correction)



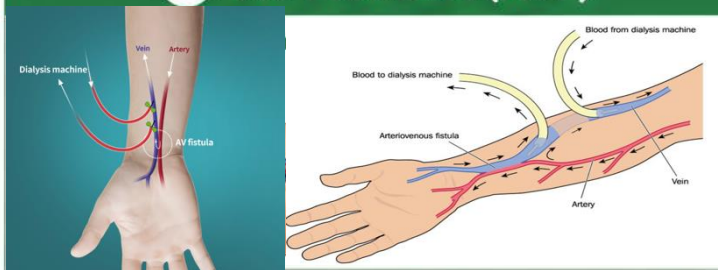
Haemodialysis removes solutes and excess water – but it **does not** replace **all** kidney functions.

Blood → Dialyser → Blood

VASCULAR ACCESS FOR HAEMODIALYSIS

Haemodialysis requires reliable access with sufficient blood flow

1 AV FISTULA (AVF)



- ✓ Durable
- ✓ Lowest infection risk
- ✓ Excellent long-term access when appropriate

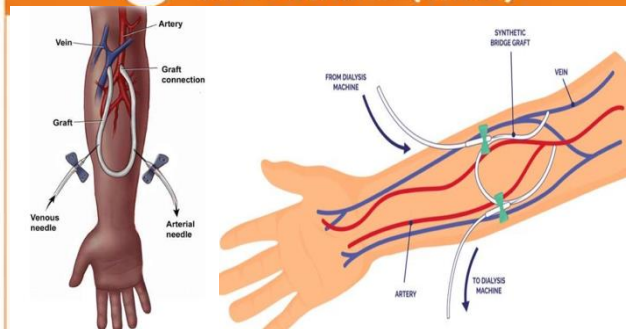


Needs time to mature

Needle placement (mature fistula)



2 AV GRAFT (AVG)

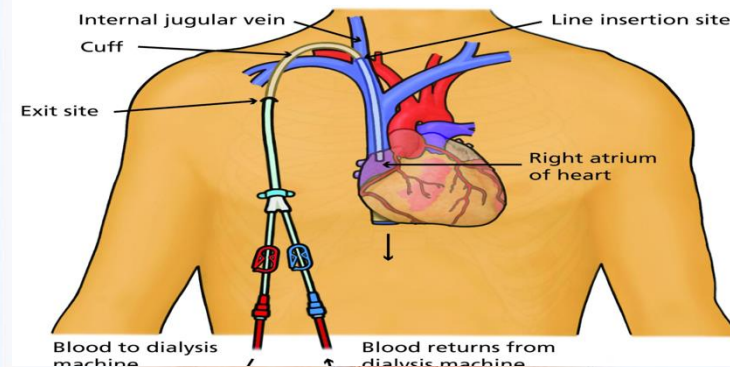


- ✓ Earlier use than conventional AVF
- ⚠ More thrombosis
- ⚠ More infections than AVF

Typical appearance



3 CENTRAL VENOUS CATHETER (CVC)



- ✓ Ready to use immediately
- ⚠ Infection
- ⚠ Thrombosis
- ⚠ Central venous stenosis



Temporary or bridge access in many patients

EXAMINE THE FISTULA



LOOK

Assess the access appearance



FEEL

Palpate for THRILL



LISTEN

Auscultate for BRUIT



No thrill? → suspect access dysfunction / thrombosis

⚠ PROTECT THE FISTULA ARM ⚠



NO BLOOD PRESSURE MEASUREMENT



NO VENEPUNCTURE / BLOOD SAMPLING



NO PERIPHERAL IV CANNULA

No BP • No venepuncture • No IV cannula



Vascular access is the haemodialysis patient's **lifeline**.



AVF / AVG / CVC



Adequate blood flow



Effective haemodialysis

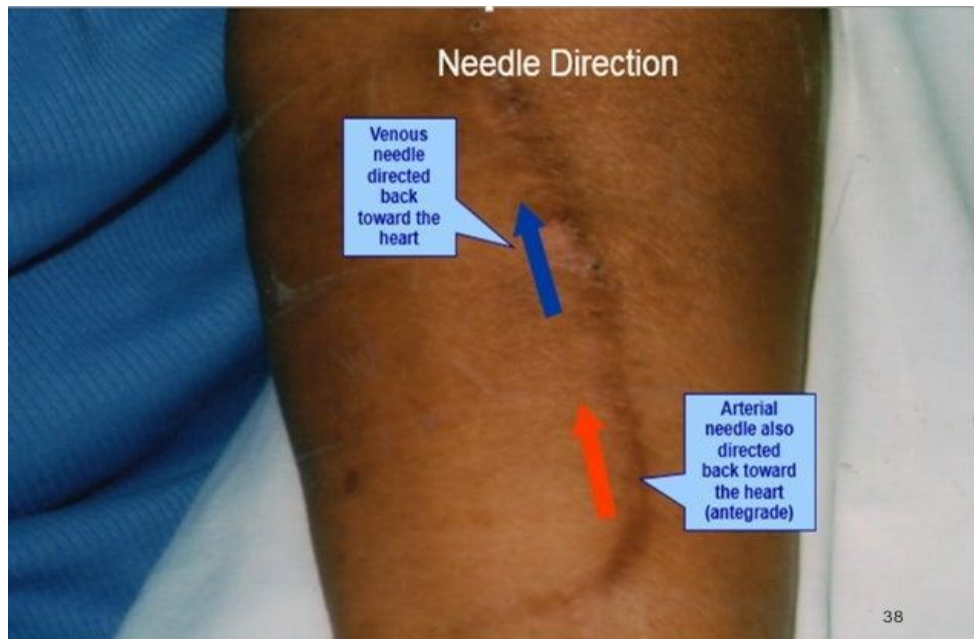


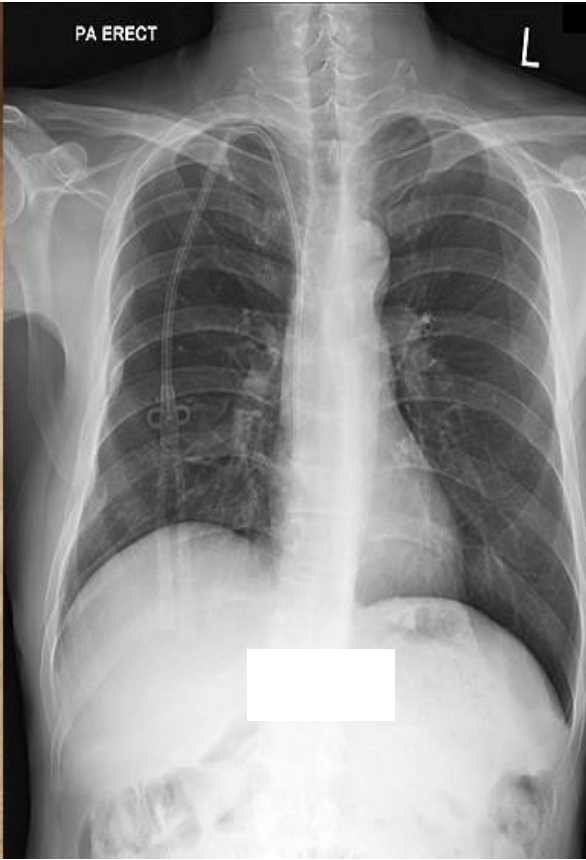
Better outcomes

Access flow
 ≥ 600 mL/min

Vein diameter
 ≥ 6 mm

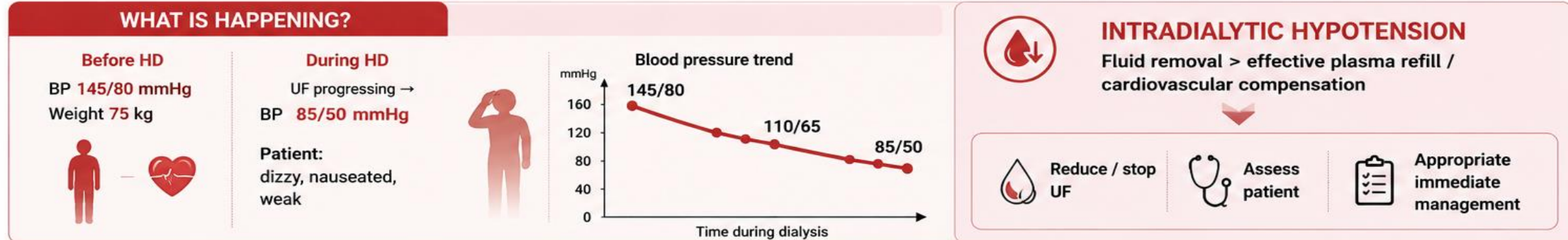
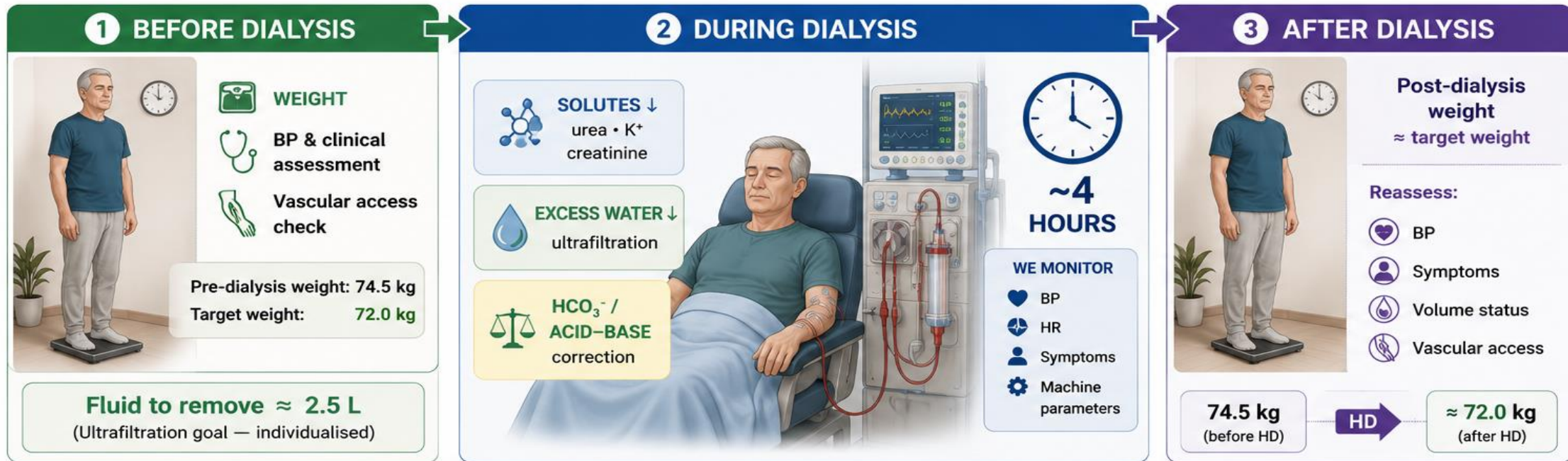
Depth below skin
 ≤ 6 mm





A TYPICAL HAEMODIALYSIS SESSION

Usually ~4 hours • 3 times per week — individualized prescription



Haemodialysis is a balance:
remove enough fluid — but not too much, **too fast**.

Remove excess water + Remove solutes + Correct acid-base = Better outcomes

Haemodialysis — What Can Go Wrong?

Haemodialysis is life-sustaining, but it is not physiologically equivalent to normal kidney function.



DURING / IMMEDIATELY AFTER HD



INTRADIALYTIC HYPOTENSION

Most common acute complication

Dizziness • nausea
weakness • cramps

→ Excessive / rapid UF,
impaired cardiovascular
compensation



ARRHYTHMIAS

Rapid electrolyte
and volume shifts



MUSCLE CRAMPS

Often associated
with volume
removal /
hypotension



RARE BUT SERIOUS

Chest pain
dyspnoea
altered consciousness

→ **STOP & ASSESS**



LONG-TERM / ACCESS RELATED



INFECTION

Especially catheter-
related bloodstream
infection

Fever + chills during HD
→ think access infection



ACCESS THROMBOSIS / DYSFUNCTION

No thrill?

→ **URGENT ASSESSMENT**



CARDIOVASCULAR BURDEN

Hypertension • LVH •
heart failure • arrhythmias



COMPLICATIONS OF KIDNEY FAILURE CONTINUE

Anaemia • CKD-MBD

Dialysis does not replace
endocrine kidney function.



A DIALYSIS PATIENT SUDDENLY DETERIORATES — THINK:



VOLUME

Too much or
too little?



ELECTROLYTES

Especially
potassium



ACCESS

Infection?
Thrombosis?



INFECTION

Fever, rigors,
sepsis?



CARDIAC

Ischaemia?
Arrhythmia?
Heart failure?



KEY TAKE-HOME MESSAGES

Monitor the patient continuously during HD.

- ✓ Always examine the vascular access.
- ✓ Recognize complications early and act promptly.
- ✓ Dialysis replaces excretory function — not all endocrine kidney functions.



The goal is **safe** and **effective** dialysis, tailored to each patient.



Adequate
dialysis



Euvoalaemia



Fewer
complications



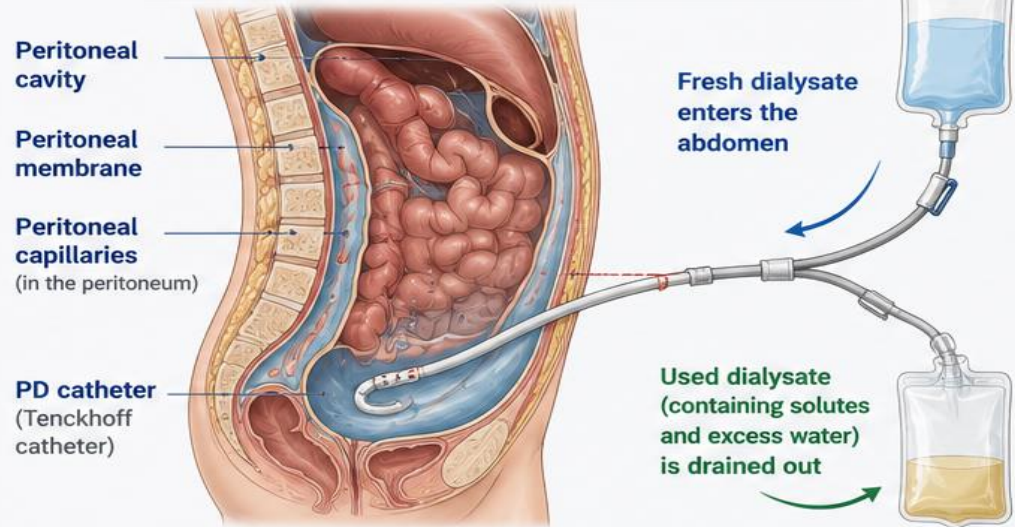
Better
quality of life



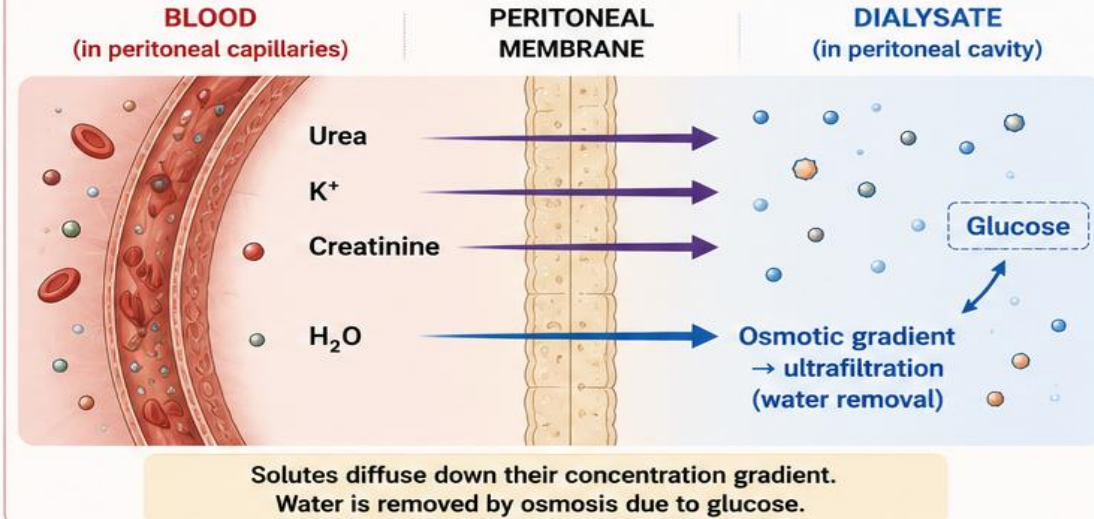
Peritoneal Dialysis – Using the Patient's Own Membrane

The peritoneum acts as a natural semipermeable dialysis membrane

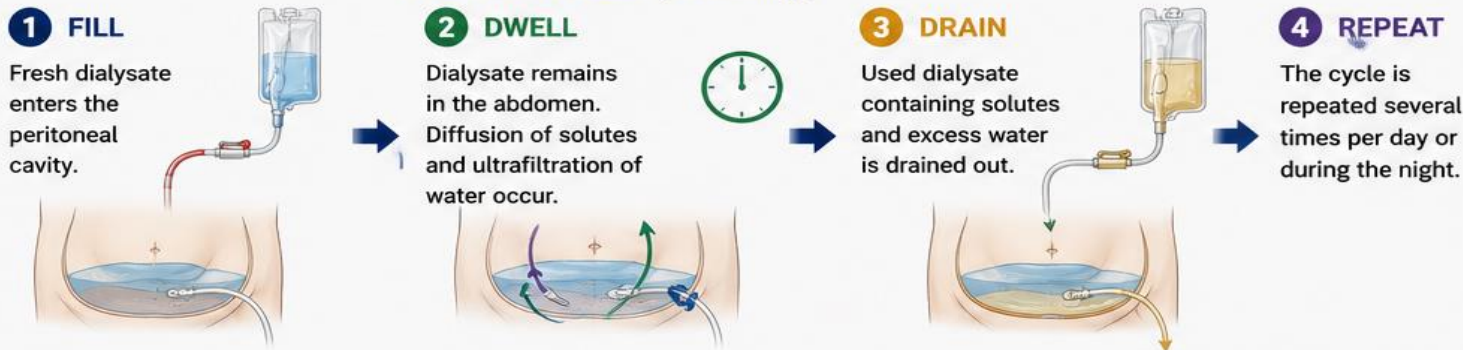
WHAT IS PERITONEAL DIALYSIS?



HOW DOES IT WORK?

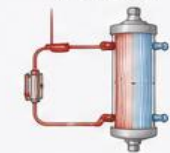


THE PD CYCLE (an exchange)



HD vs PD

HAEMODIALYSIS
Artificial membrane (dialyser)



Blood leaves the body

PERITONEAL DIALYSIS
Patient's peritoneal membrane



Blood stays inside the body

VS



PD = **continuous exchange** of solutes and water across the peritoneal membrane.
No extracorporeal blood circuit is required.



KEY POINT

In HD we bring the blood to an artificial membrane; in PD we bring the dialysis fluid to the patient's natural membrane.

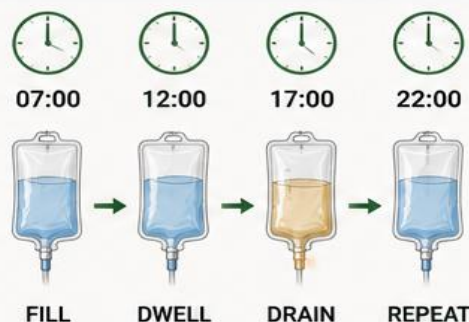
Peritoneal Dialysis at Home — CAPD vs APD

Same principle • Different way of performing the exchanges



CAPD

Continuous Ambulatory Peritoneal Dialysis



Manual exchanges

FILL → DWELL → DRAIN

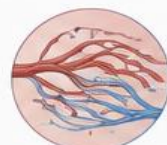
Usually several exchanges during the day

- ✓ No machine required
- ✓ Patient remains mobile during dwell
- ⚠ Manual exchanges several times/day



Mobile throughout the day

SAME DIALYSIS PRINCIPLE



Peritoneal membrane



Diffusion + osmosis



Solute + water removal



APD

Automated Peritoneal Dialysis



- ✓ Automated exchanges
- ✓ Usually performed overnight
- ✓ Greater daytime freedom
- ⚠ Requires a cyclor and appropriate training



Sleep well, dialysis works while you rest

WHY IS PD DIFFERENT?



HOME-BASED

Treatment is performed by the patient at home



CONTINUOUS

More continuous removal of solutes and water



PATIENT-LED

Education, training and engagement are essential



Residual kidney function matters

PD often allows good preservation of residual kidney function, particularly early in therapy.

KEY TAKE-HOME MESSAGES

- CAPD = manual exchanges during the day
- APD = automated exchanges, usually overnight
- Same dialysis principle, different approach
- Prescription is individualized to the patient, lifestyle and peritoneal membrane characteristics



PD = continuous exchange of solutes and water across the peritoneal membrane

No extracorporeal blood circuit is required

PD Peritonitis — A Complication You Must Recognize

Think early. Diagnose promptly. Treat without delay.



A 54-year-old patient on PD presents with:

- ⚡ Abdominal pain
- 🗑️ Cloudy dialysis effluent
- 🤢 Nausea
- 🌡️ ± Fever

NORMAL EFFLUENT



VS.

CLOUDY EFFLUENT



⚠️ **PERITONITIS?**



Cloudy effluent should be considered peritonitis until excluded.

1 RECOGNIZE

Think PD-associated peritonitis



Typical presentation:

- Cloudy effluent
- Abdominal pain
- ± Fever / nausea

2 SAMPLE



Send PD effluent for:

- ✓ Cell count + differential
- ✓ Gram stain
- ✓ Culture

Do this before antibiotics whenever possible — but do not unnecessarily delay treatment.

Diagnosis is supported by elevated effluent WBC with neutrophil predominance.

3 TREAT PROMPTLY

Empirical antibiotics

Cover Gram-positive + Gram-negative organisms

Adjust to culture results



Intraperitoneal route is generally preferred when feasible.

WHERE CAN THE INFECTION COME FROM?



Touch contamination (during exchanges)



Exit-site / tunnel infection (spread to peritoneal cavity)



Enteric / other causes (translocation, etc.)



Strict aseptic technique is essential for prevention.

ALWAYS EXAMINE THE CATHETER



- ✓ Exit-site erythema?
- ✓ Discharge?
- ✓ Tunnel tenderness?

Catheter-related infection may coexist with peritonitis.

WHY IS IT IMPORTANT?



Can require hospitalization



Risk of sepsis



Can damage peritoneal membrane



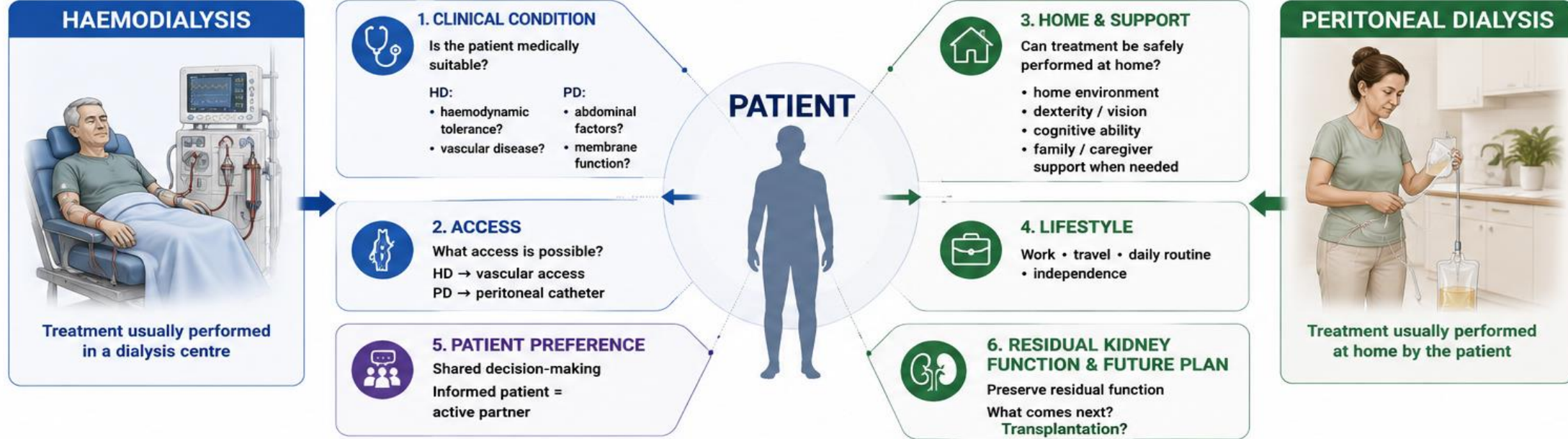
May lead to catheter loss or loss of PD as a treatment option



PD + CLOUDY EFFLUENT → THINK PERITONITIS → SAMPLE → TREAT PROMPTLY

Haemodialysis or Peritoneal Dialysis — How Do We Choose?

The best dialysis modality is the one that fits the patient — medically and personally.



Do not choose a dialysis machine. **Build a kidney failure life-plan.** | Our goal: the best possible outcomes and quality of life.

Kidney Transplantation — The Preferred KRT for Eligible Patients

A functioning kidney transplant provides the most complete form of kidney replacement therapy

WHY TRANSPLANT?



SURVIVAL

Better long-term survival for appropriate candidates



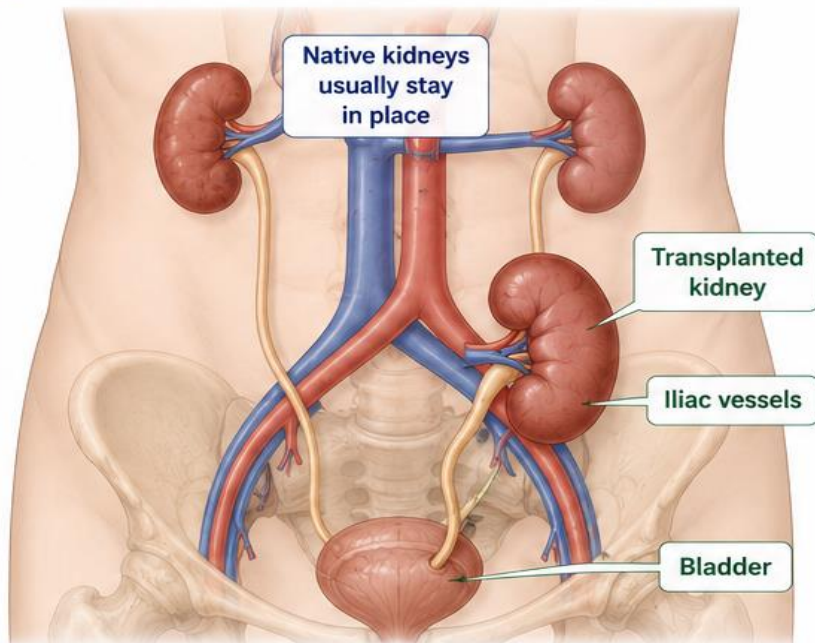
QUALITY OF LIFE

Greater independence
Fewer dialysis-related restrictions



KIDNEY FUNCTION

More complete replacement of kidney function
Excretory + regulatory + endocrine functions



WHERE DOES THE KIDNEY COME FROM?



LIVING DONOR

Related or unrelated

- ✓ Planned procedure
- ✓ Shorter waiting time
- ✓ Possibility of pre-emptive transplantation



DECEASED DONOR

Kidney allocated from a deceased donor

- ✓ Major source of donor kidneys
- ⌚ Requires waiting-list allocation



PATHWAYS TO TRANSPLANTATION

PATH A

Traditional pathway

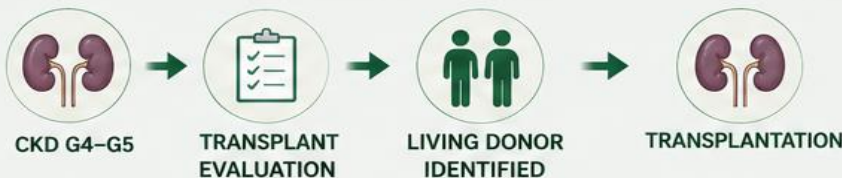


PATH B

★ PRE-EMPTIVE TRANSPLANTATION

Transplantation before dialysis is needed.

Pre-emptive transplantation pathway



When feasible, this is an excellent strategy for an eligible patient.

IMPORTANT

Transplantation is not suitable for every patient.

Eligibility requires careful assessment of:

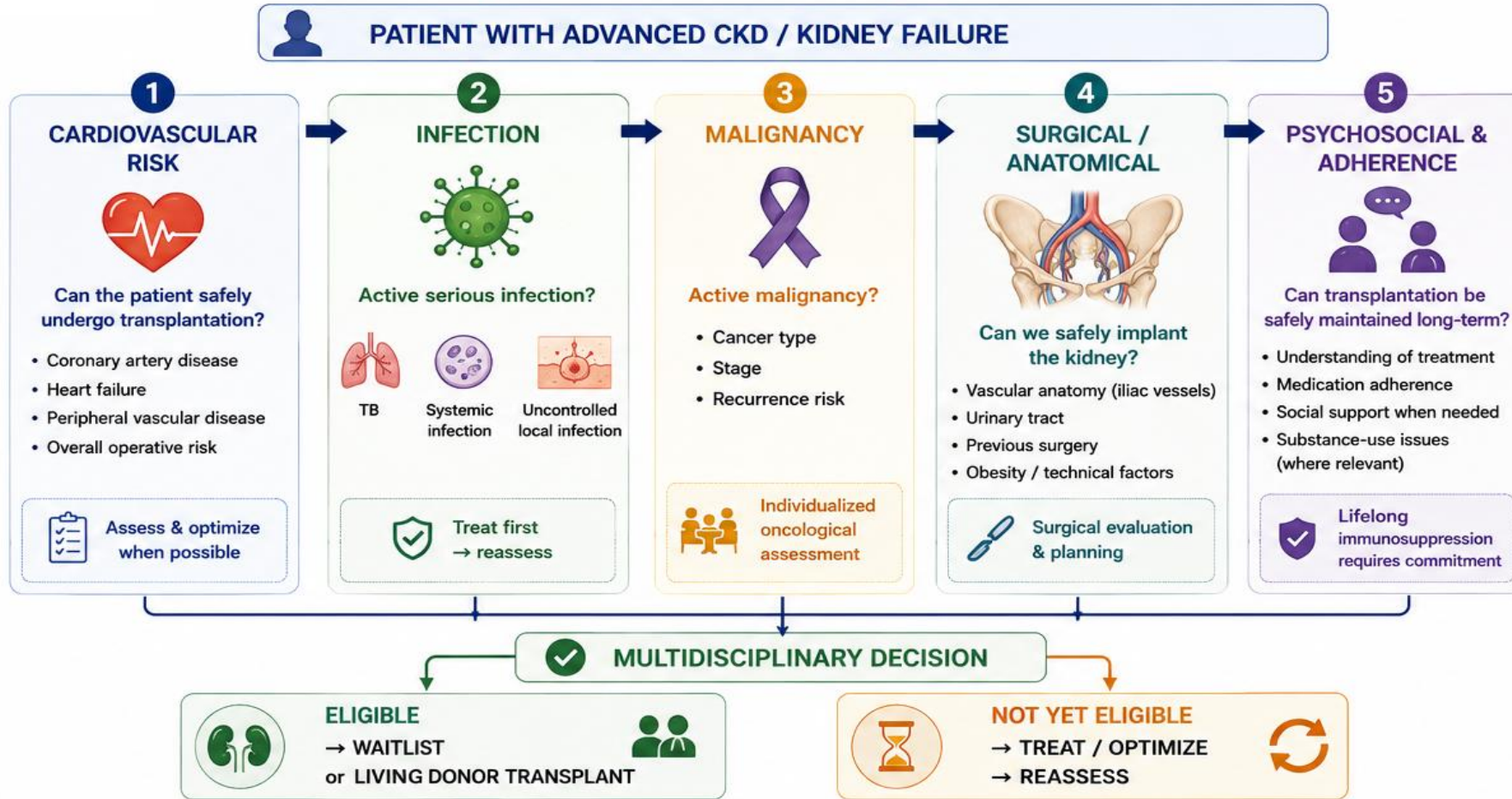
- ♥ Cardiovascular risk
- 🦠 Infection
- 🩸 Malignancy
- 🔪 Surgical / anatomical risk
- 🧠 Adherence / psychosocial factors



For an eligible patient, **think transplantation early** — not only after dialysis has started.

Who Can Receive a Kidney Transplant?

Transplant evaluation = balancing expected benefit against individual risk



IMPORTANT PRINCIPLE



Age alone is NOT the decision.

Consider biological age, frailty, comorbidity and expected benefit.

THE TEAM APPROACH



Nephrologist



Cardiologist



Infectious disease specialist



Oncologist



Surgeon / Urologist



Psychologist / Psychiatrist



Transplant coordinator



KEY POINT

Transplantation is not suitable for every patient.

Eligibility requires careful assessment of risks and expected benefit.

CONTRAINDICATION ≠ ALWAYS PERMANENT



Problem identified



Treat / optimize (modify risk factors)



Reassess patient



Transplantation may become possible

Examples

- Active infection → treat → reassess
- Poorly controlled cardiovascular disease → optimize → reassess
- Smoking → cessation → reassess

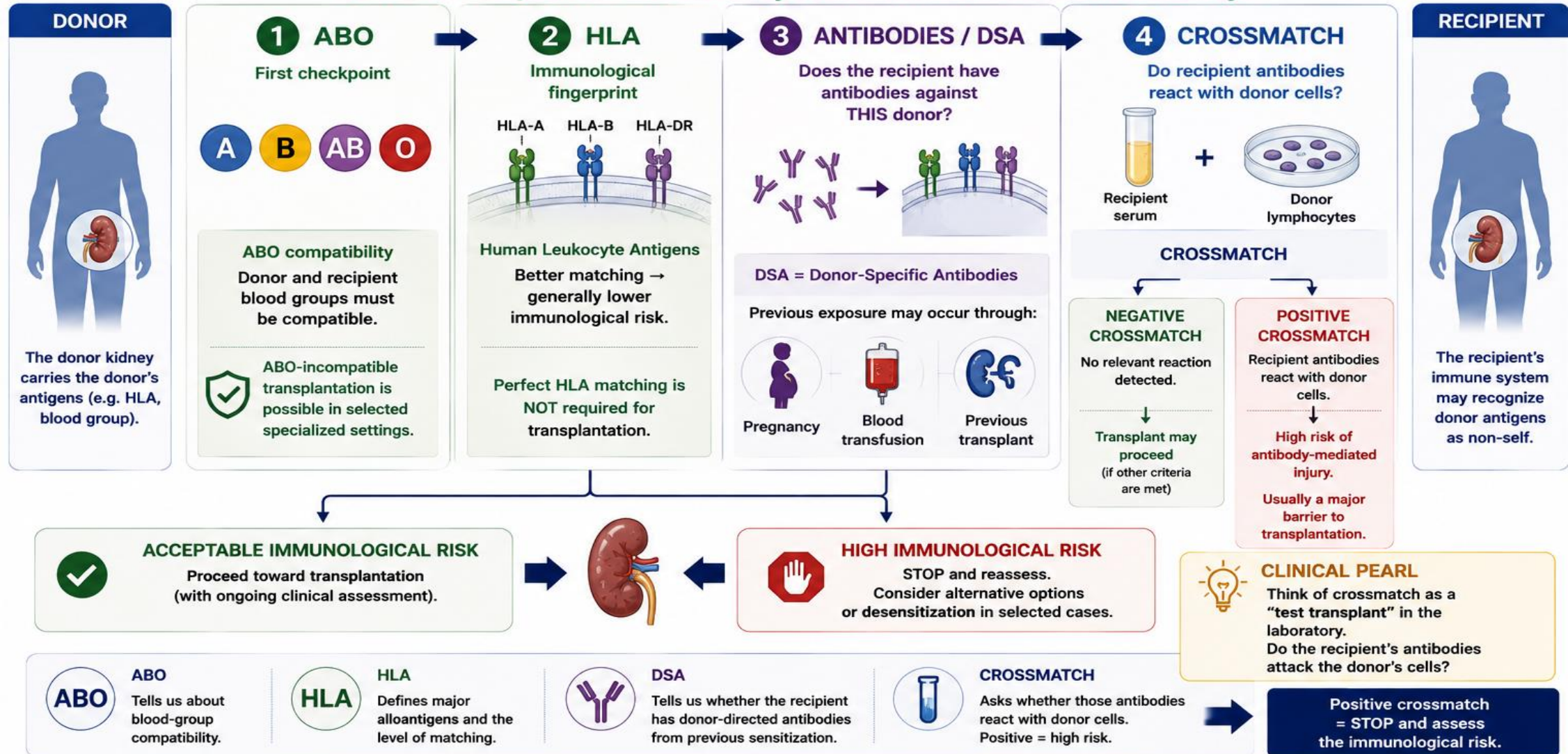


The question is not simply “Can we transplant?” — but “Will transplantation provide **meaningful benefit** at acceptable risk?”



Kidney Transplantation — Will This Kidney Match?

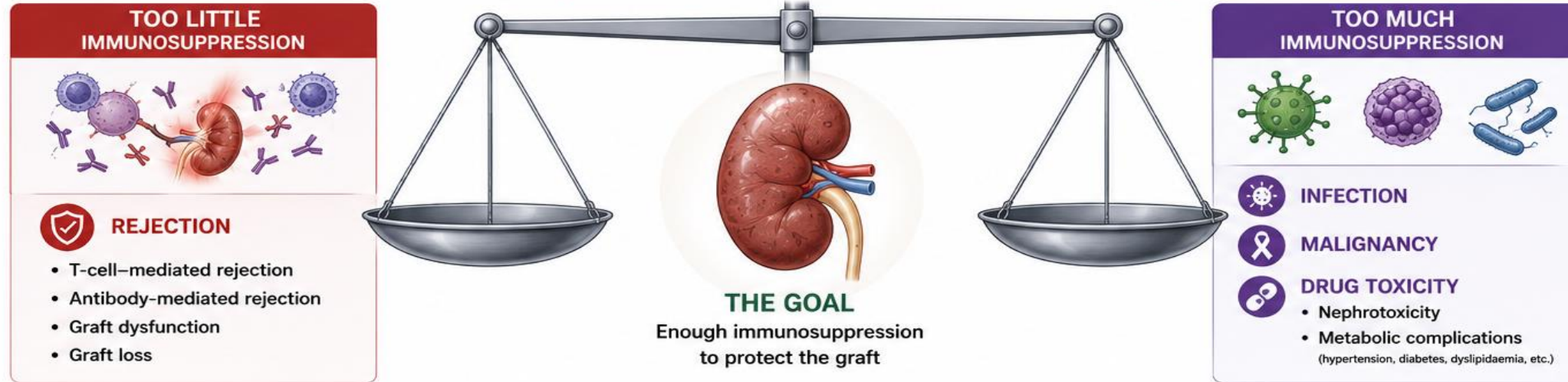
Before transplantation, we ask one essential question:
Will the recipient's immune system attack this donor kidney?



Only after both **CLINICAL** and **IMMUNOLOGICAL** evaluation can we safely proceed toward transplantation.

Immunosuppression After Kidney Transplantation – Finding the Balance

Prevent rejection — without excessive immunosuppression



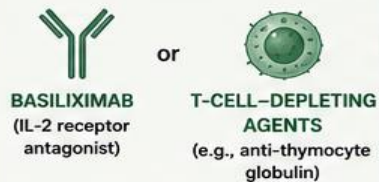
IMMUNOSUPPRESSIVE THERAPY OVER TIME

TRANSPLANTATION



1. INDUCTION

Strong immunosuppression around transplantation



Reduces early risk of acute rejection

2. MAINTENANCE

Long-term therapy — usually triple combination

TACROLIMUS

Calcineurin inhibitor



MYCOPHENOLATE

Antimetabolite



CORTICOSTEROID

(Prednisone)



Regimens are individualized according to:
immunological risk • time after transplantation • adverse effects



CLINICAL PEARL — TACROLIMUS

Narrow therapeutic window



MONITOR: TROUGH LEVEL

Major adverse effects:

- Nephrotoxicity
- Neurotoxicity
- Hyperkalaemia
- Hypertension
- Diabetes mellitus
- Tremor

WHY DO WE COMBINE SEVERAL DRUGS?



- ✓ Different drugs act on different immune pathways.
- ✓ Better rejection prevention with lower doses of individual drugs.

SIMPLE CONCEPT



Immune activation against graft



Immunosuppression blocks overreaction



Graft protected and functions

LIFELONG THERAPY



- Never stop immunosuppression without medical supervision.
- Adherence is essential for graft survival.



Transplant medicine is a continuous balance:

REJECTION



IMMUNOSUPPRESSION

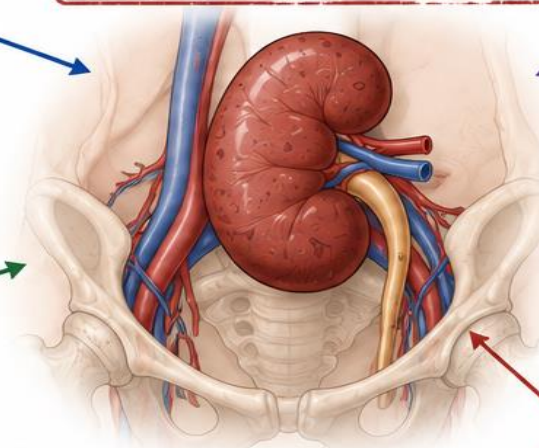


INFECTION • MALIGNANCY • TOXICITY

The Creatinine Is Rising — What Is Happening to the Graft?

Kidney transplant recipient: creatinine 1.2 → 2.4 mg/dL

ISING CREATININE ≠ REJECTION



Think broadly.
Investigate systematically.

1 HAEMODYNAMIC / PRE-RENAL



- Volume depletion
- Hypotension
- Drug-related haemodynamic effects

Is the graft being adequately perfused?

2 TACROLIMUS TOXICITY



- Check tacrolimus trough level
- Nephrotoxicity
- ± hyperkalaemia
- ± tremor

Any new drugs or interactions?

3 INFECTION



- UTI / pyelonephritis
- BK polyomavirus

Immunosuppressed patients may have atypical presentations

4 OBSTRUCTION / VASCULAR PROBLEM



- Hydronephrosis?
- Renal artery / vein problem?

Ultrasound ± Doppler

5 REJECTION



- T-cell-mediated
- Antibody-mediated
- DSA?

Biopsy when indicated

BEFORE YOU SAY “REJECTION”...



Is the patient dehydrated?



Any diarrhoea or vomiting?



Any new medication?



Tacrolimus level?



Fever / urinary symptoms?



Urine output?

DIAGNOSTIC APPROACH — STEP BY STEP

1 HISTORY + EXAMINATION



Symptoms, fluid status, BP, urine output, medications, adherence

2 BASIC LABS + URINE



Creatinine, urea, electrolytes, FBC, urinalysis, culture

3 TACROLIMUS LEVEL



Check trough level and review drugs for interactions

4 GRAFT US ± DOPPLER



Assess hydronephrosis, vascular flow and perigraft collections

5 DSA / VIRAL TESTING AS INDICATED



DSA (antibody-mediated risk), BK virus, CMV, EBV, others if needed

6 KIDNEY ALLOGRAFT BIOPSY IF NEEDED



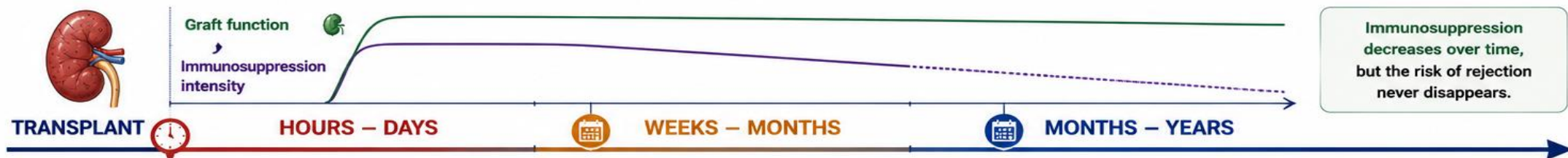
Definitive diagnosis (rejection, other pathology, CNI toxicity)



A rise in creatinine is a **sign of graft dysfunction** — not a diagnosis.

After Kidney Transplantation — What Can Go Wrong, and When?

Time after transplantation helps narrow the differential diagnosis



1 HOURS – DAYS

Think: SURGERY & EARLY GRAFT FUNCTION



Delayed graft function
Kidney does not function immediately



Vascular complications
Renal artery / vein thrombosis



Urinary complications
Leak • obstruction



Bleeding / fluid problems



Ultrasound + Doppler is often an early key investigation



CLINICAL PEARL

Sudden anuria after transplantation = urgent assessment

2 WEEKS – MONTHS

Think: IMMUNITY • INFECTION • DRUGS



REJECTION

- T-cell mediated
- Antibody mediated



OPPORTUNISTIC INFECTIONS

CMV • BK polyomavirus • others

High immunosuppression = higher infection risk



IMMUNOSUPPRESSIVE TOXICITY

- Tacrolimus nephrotoxicity
- Metabolic adverse effects (e.g., diabetes, hypertension, dyslipidaemia)



REMEMBER

Symptoms may be subtle or atypical in immunosuppressed patients.

3 MONTHS – YEARS

Think: LONG-TERM CONSEQUENCES



CARDIOVASCULAR DISEASE

Major long-term risk



MALIGNANCY

Skin cancer • PTLD • other cancers



CHRONIC GRAFT DYSFUNCTION

Multiple possible causes



RECURRENT / DE NOVO KIDNEY DISEASE

Original kidney disease may recur in the graft



LIFELONG FOLLOW-UP

Regular monitoring is essential for graft survival and patient health.



CLINICAL THINKING

FIRST QUESTION:

“When was the transplant?”



DAY 1–3

Anuria / oliguria?
→ think vascular / obstruction



WEEK 2–8

Fever, ↑ creatinine?
→ think infection, rejection, drugs



MONTH 3–6

Rising creatinine?
→ infection? toxicity? rejection?



YEAR 1+

Declining graft function?
→ broad differential: chronic injury, recurrent disease, malignancy, CVD



TIME HELPS — BUT DOES NOT MAKE THE DIAGNOSIS.
Complications overlap. Always evaluate the individual patient.



Consider the whole clinical picture



Investigate systematically



Treat the cause, not just the numbers



Understanding timing after transplantation is essential for **early recognition**, appropriate **investigation** and **better outcomes**.

One Patient — Three Kidney Replacement Strategies

What would you recommend?



CASE

45-year-old man with progressive CKD G5

eGFR	7 mL/min/1.73 m ²
K ⁺	4.8 mmol/L
HCO ₃ ⁻	20 mmol/L
BP	135/80 mmHg
Symptoms	Mild fatigue
Volume status	Euvolaemic
Urine output	Preserved

No urgent indication for dialysis

Potential transplant candidate • Wife interested in living kidney donation

OPTION A

HAEMODIALYSIS



- Requires vascular access
- Usually intermittent
- Centre-based in many patients

Does he need HD today?
→ NO

OPTION B

PERITONEAL DIALYSIS



- Home-based
- More continuous
- Requires PD catheter + training

Could this be an appropriate option?
→ YES — if dialysis becomes necessary

OPTION C

KIDNEY TRANSPLANTATION



Evaluate NOW

Potential living donor

★ PRE-EMPTIVE TRANSPLANTATION?

Potentially before dialysis is ever required

✗ OLD QUESTION

"Which dialysis should we start?"



CKD G5
eGFR ~7
No urgent dialysis need



TRANSPLANT
EVALUATION
NOW
Assess eligibility,
optimize health



LIVING DONOR
EVALUATION
In parallel
with recipient



EDUCATION
ABOUT HD & PD
Shared decision-
making



PREPARE ACCESS
IF/WHEN
APPROPRIATE
Fistula for HD
or PD catheter



PRE-EMPTIVE KT_x
IF FEASIBLE
Best option
when possible

OR



PD / HD
if dialysis
becomes
necessary

LATER
TRANSPLANTATION
Continue evaluation
and re-assessment

✓ NEW QUESTION What is this patient's kidney failure life-plan?

1



eGFR alone does not
determine dialysis initiation.
Treat the patient, not the number.

2



Plan kidney replacement
therapy before an emergency
occurs.
Preparation improves outcomes.

3



Think transplantation early.
Evaluate for transplant and consider
pre-emptive transplantation when
possible.



KEY MESSAGE

Choose the best strategy for
the right patient at the right time.



Do not wait for kidney failure to become an emergency. **PLAN EARLY** • **EDUCATE** • **SHARE THE DECISION** • **THINK TRANSPLANT**

KIDNEY LIFE PLAN — THINK BEYOND THE NEXT TREATMENT



KTx
Kidney transplant



PD
Peritoneal dialysis

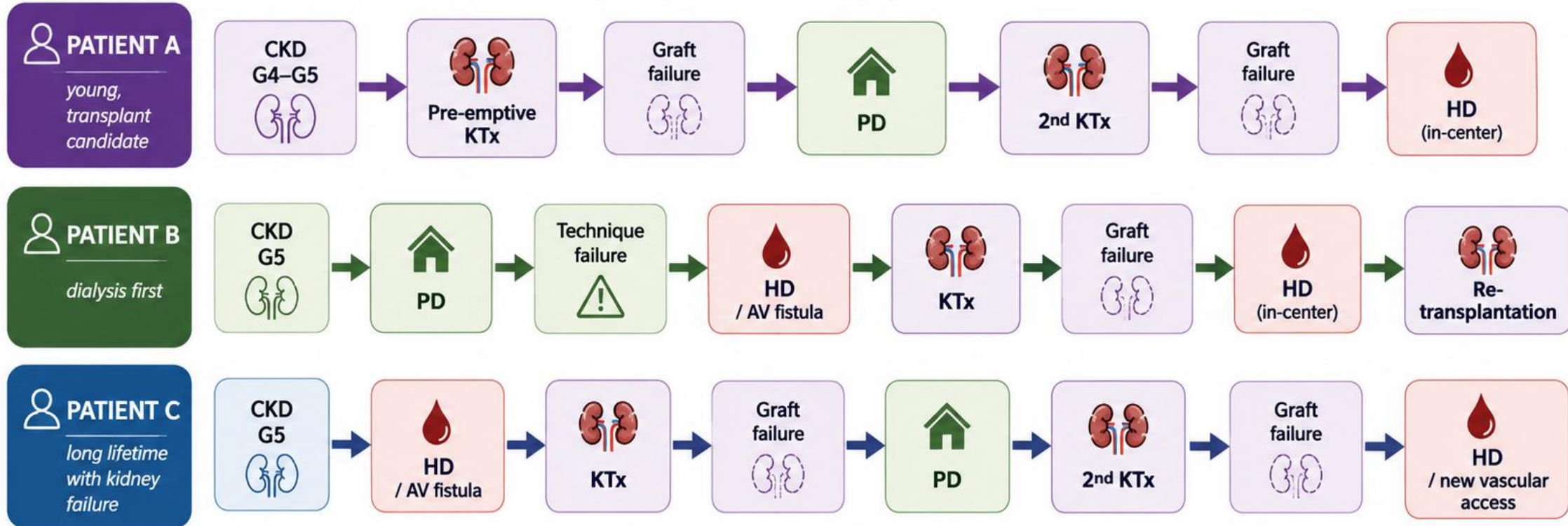


HD
Hemodialysis
(in-center)



AV Access
Vascular access
(fistula / graft)

Patient's lifetime →



Kidney failure is lifelong. No kidney replacement therapy lasts forever.

Plan today while preserving tomorrow's options.



Plan ahead
for the whole lifetime

Preserve vascular access
and future treatment options

Each therapy may be needed
more than once

Individualize the path,
not just the next step

QUESTIONS?

Thank you for your attention!

Kidney health • Life • Shared decisions



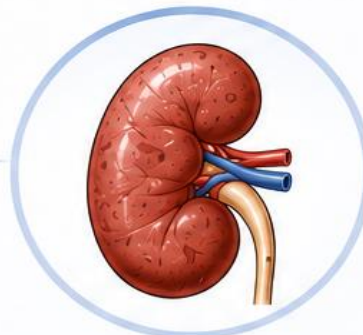
HAEMODIALYSIS

Diffusion removes solutes
Ultrafiltration removes water



PERITONEAL DIALYSIS

The peritoneum
is the dialysis membrane
Diffusion + osmotic ultrafiltration



**RIGHT PATIENT
RIGHT THERAPY
RIGHT TIME**



KIDNEY TRANSPLANTATION

Best outcomes and quality of life
For eligible patients,
think transplant early



SHARED DECISION-MAKING

Plan together
Educate
Respect preferences

THE ESSENCE OF KIDNEY REPLACEMENT THERAPY



1. PLAN EARLY

Prepare access
and options
before the need arises.



2. UNDERSTAND THE BASICS

Know how dialysis works
and when to act.



3. CONSIDER THE WHOLE PATIENT

Medical, lifestyle,
psychological
and social factors.



4. PREVENT AND MONITOR

Complications can be
prevented and outcomes
improved.



5. THINK TRANSPLANT EARLY

Especially pre-emptively
when possible.



6. SHARE THE DECISION

The patient is our partner
in every step.

“

The best kidney replacement therapy is the **right strategy**
for the **right patient** at the **right time**.

