PARTNERSHIP IN CRITICAL CARE DEPT. WAIKATO

CRITICAL CARE WAIKATO HOSPITAL

WORKBOOK

NAME: _____

Te Hanga Whaioranga Mō Te Iwi
Building Healthy Communities

Waikato District Health Board

Section two

Types of renal failure

Introduction
This module will not discuss patient assessment of the renal system as such. Basic understanding of renal waste products, biochemical markers for renal failure, fluid balance management and history taking will be assumed. However, further information on this is available in chapter 29 of the required text.

Similarly, as renal failure may result in or occur in conjunction with a number of presentations of **acid-base disturbances**, please refer to table 29.6, p631 of the course text.

Approximately 66% of patients treated in intensive care units will experience **acute kidney injury (AKI),** also referred to as **acute renal failure (ARF).** Of those patients

treated with some form of renal replacement therapy there is a mortality rate of 50-60% (Morton & Fontaine, 2013).

2.1 Causes of acute kidney injury

To aid diagnosis and management, AKI is divided into 3 categories:

1. **Prerenal;**

- Characterised by any physiological event that results in renal hypoperfusion, e.g. hypovolaemia, cardiovascular failure, sepsis

2. **Intrarenal;**

- Damage to the renal parenchyma – categorised by compartment, e.g. glomerular, vascular, interstitial and tubular:
    - Glomerular causes – include immune complex-mediated causes, and diseases causing vasculitis
    - Interstitial causes – include allergic interstitial nephritis, caused by pharmacological reactions, and infectious processes such as pyleonephritis
    - Vascular causes – e.g. malignant hypertension, artheroembolic disease or haemolytic-uraemic syndrome.
    - Tubular causes – acute obstruction, or acute tubular necrosis.

3. **Post renal;**

- Any obstruction in the flow of urine from the collecting ducts to the external urethra.
- Accounts for approx 10% of hospital cases.

Renal perfusion

The kidneys have an **autoregulation** function that helps maintain glomerular filtration rate (GFR) at a constant rate despite a wide range of mean arterial pressures. However, when renal perfusion is significantly reduced, this capacity is overwhelmed, and GFR decreases.

Some drugs such as **ACE inhibitors and NSAIDs** can also interfere with the kidney's autoregulatory function.

It is important to understand that increasing the blood pressure does not necessarily result in an increased renal perfusion. **Inotropes** such as noradrenaline may cause further reduction in renal blood flow, as they may constrict renal arteries.

Ischaemic acute tubular necrosis results from prolonged hypoperfusion, so pre-renal causes can become intra-renal problems, and may become tubular problems as necrotic cells slough off and can block the tubules.

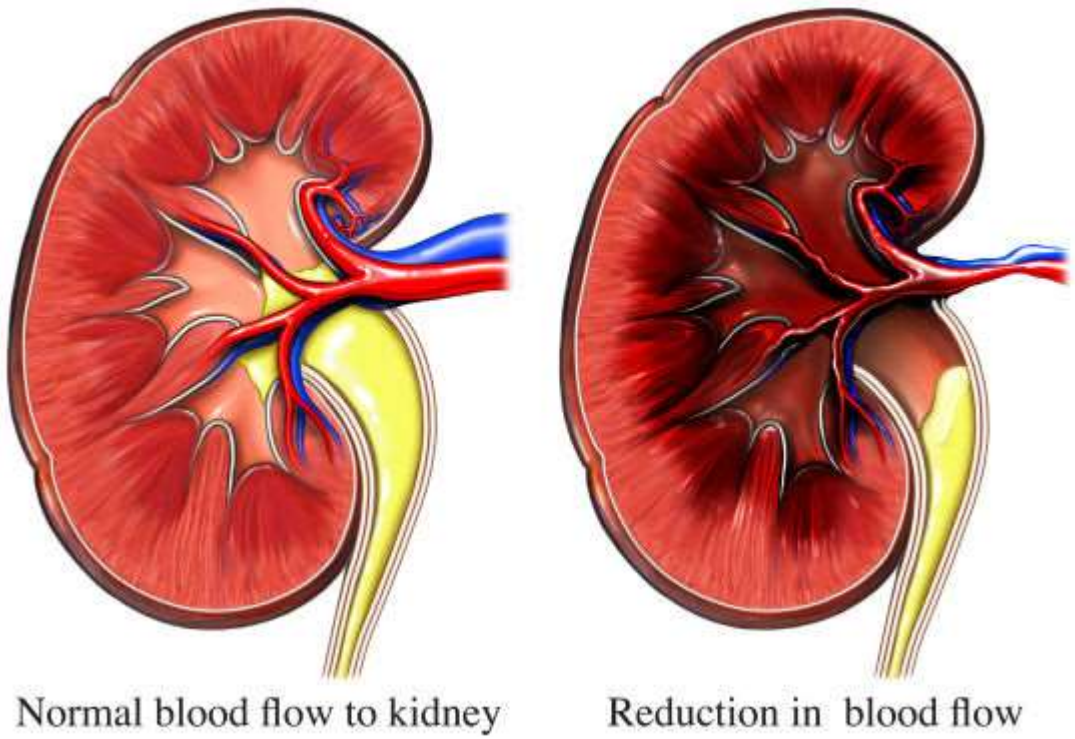

Fig.5 Renal ischaemia, (Radius Medical Illustration, 2013).

Other causes of necrosis may be the toxicity of certain drugs or chemicals. These are many and varied, but aminoglycoside antibiotics and radiocontrast dyes are commonly observed causes. The toxicity of many agents is dose-dependent, so it is important to monitor the therapeutic range of at-risk medications.

Diabetic patients are at increased risk of contrast-induced toxicity. Fluid loading at the time of contrast administration may minimize the risk.

Obstructive (post renal) problems result in increased retrograde (backward) pressure through the collecting system. This will slow the rate of tubular fluid flow and decrease GFR. Reabsorption of sodium, water, and urea is increased as a result. Further dilation of the collecting system tubules will result in compression and damage to nephrons.

A single functioning kidney is enough to maintain homeostasis, so blockage must be to both kidneys to cause a severe renal failure problem. Patients however may present with other symptoms of unwellness, related to the inflammatory response or infection.

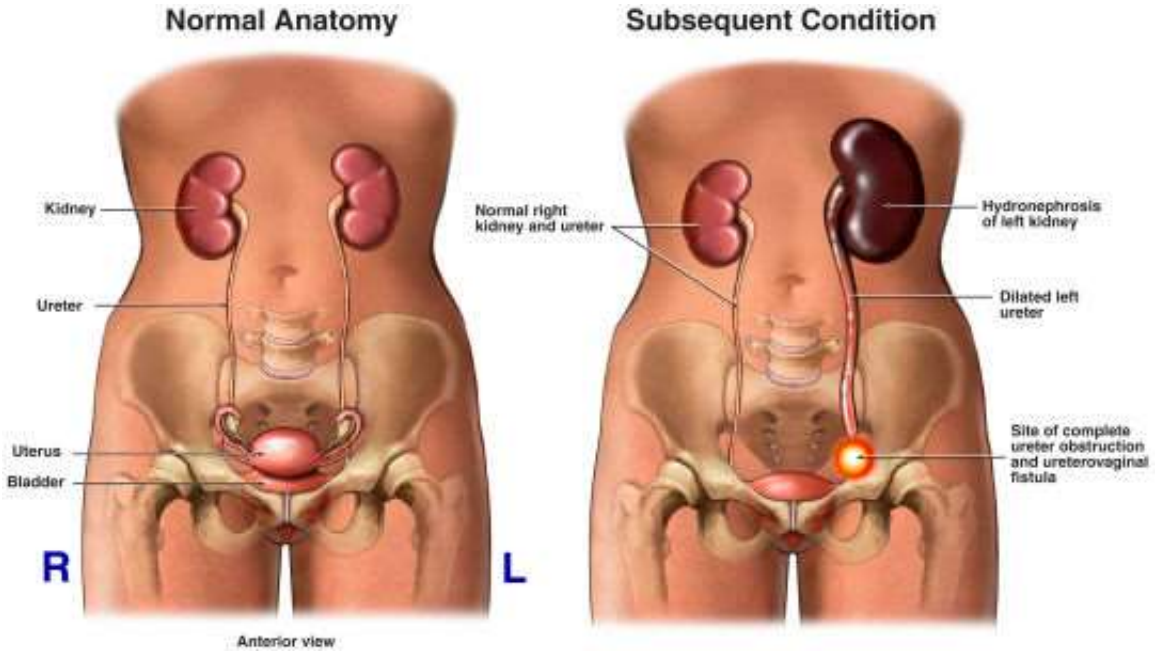

Fig 6. Obstruction example (Radius Medical Illustration, 2013).

2.2 Diagnosis of AKI

Laboratory assessment is critical to diagnosis, including serum and urinary values.

Important blood values are creatinine and urea levels, but there are may other plasma indicators (Morton & Fontaine, 2012, p.672).

(See Table 31.2, p.671, Morton & Fontaine, 2013.)

This information is further summarised in the table below:

Table 1. Differential diagnosis using lab results

Laboratory test	Prerenal ARF	Intrinsic renal failure
Creatinine urine/plasma ratio	>40	<20
BUN/creatinine ratio	↑BUN/creatinine	
UNa+ and UOsm	UNa+<20 UOsm >500	UNa+>40 UOsm<350
Fractional excretion of sodium	<1%	>2-3%
Urine specific gravity	>1.025	1.001-1.010

(Gambro, 2004, p34)

As well as urine and Na+ osmolarity, other urine tests such as specific gravity and microscopic sedimentary studies can give clues to diagnosis (see table 2). (p.33 Gambro).

Possible results from the microscopic examination of urine sediments

Urine sediment component	Normal values/field	Clinical significance
Bacteria	0	Urinary tract infection / contamination
Broad casts	0	Formation occurs in the collecting tubules, serious kidney disorder
Epithelial (renal) casts	0-2	Tubular degeneration
Fatty casts	0	Nephrotic syndrome
Granular or waxy casts	Granular: 0-2 Waxy: 0	Renal parenchymal disease
Hyaline casts	0-2	Acidic urine, high salt content
Red cell casts	0-3	Acute glomerulonephritis
White cell casts	0-4	Pyelonephritis
Epithelial cells squamous	Squamous cells are common	Normal or contamination
Renal cells	0-2	Tubular damage
Erythrocytes	0	Most renal disorders, strenuous exercise
Fat bodies	0	Nephrotic syndrome
Leucocytes	0	Most renal disorders, urinary tract infection, pyelonephritis

Table 2. Urine sediment studies (Gambro, 2004)

Chronic kidney disease (CKD) is not discussed in depth in this module, though it is important to realise the effect of pre-existing kidney disease in the context of acute admissions to ICU. Though some distinctions exist in the management of chronic versus acute renal problems, many of the clinical complications encountered are the same.

Some points of difference that may be observed in the treatment of acute failure are –

- During early stages of pre-renal failure problems, aggressive fluid administration may be indicated to address hypoperfusion issues.
- Diuretics may be utilised to increase urinary flow and overcome conditions of fluid overload, or prevent tubular obstruction. (Morton & Fontaine, 2013).

2.3 Systemic complications of renal failure

(summarised from Morton & Fontaine, 2013)

Cardiovascular alterations in renal failure

- Hypertension and hyperkalaemia are common complications of renal disease and AKI.
- **Hypertension** is the result of retention of water and sodium, over-activation of the sympathetic nervous system, and stimulation of the renin-angiotensin-aldosterone system.
- **Hyperkalaemia** is the result of reduced GFR, and consequent decreased ability to excrete potassium.
- In the ICU setting this renal impairment is compounded by catabolic states, acidosis and cellular injury, administration of potassium-containing medications, and blood transfusions – all potentially elevating potassium levels to critical states
- Fatal dysrythmias can result from hyperkalaemia and hypocalcaemia.
- **Uraemic pericarditis**, resulting from increased permeability of pericardial membranes to fluid is a complication more commonly associated with chronic renal failure

Pulmonary complications

- More commonly associated with chronic kidney disease (CKD), but can be associated with the oliguric phase of AKI. – e.g. pulmonary oedema
- Pulmonary infections can be associated with acute renal failure

Gastrointestinal complications

- GI bleeding is associated with blood clotting abnormalities and anti-coagulation used in dialysis
- Constipation can be a problem induced by decreased bulk and fluid in diets, and administration of iron supplements and calcium binders.
- Diarrhoea may occur as a result of irritation from uraemia.

Neuromuscular complications

- Alterations observed in chronic renal failure may include sleep disturbances, changes in cognitive process, lethargy, muscle irritability, and peripheral neuropathies including restless leg and burning feet syndrome.
- May result from electrolyte imbalances, uraemic toxin effects on motor and sensory nerves, and sleep disturbances
- In acute renal failure electrolyte disturbances such as hyperkalemia, hyponatremia, hypocalcaemia, and hyperphosphatemia can cause significant systemic effects. These may include; muscle irritability, tremors, muscle cramps, seizures, tetany, hallucinations, confusion, weakness, hyporeflexia, and parasthesia. In addition, fluid overload, associated with hyponatraemia can cause cerebral oedema (Gambro, 2004).

Haematological complications

- Uraemic states can lead to impaired platelet aggregation and clotting alterations
- Immunocompromised states also present as a result of the effect of uraemia on white cell function.
- Severe anaemia can result from decreased production of erythropoietin (90% made by kidney) which stimulates red cell production. This anaemia can be

compounded by decreased red blood cell (RBC) survival time and any bleeding issues.

Additionally, alterations in other body systems such as integumentary and skeletal may be observed in CKD, but infrequently in AKI.

Required reading

Morton, P and Fontaine, D. (2013). *Critical care nursing: a holistic approach*. 10th ed. Lippincott, Williams & Wilkins: Philadelphia. Read Chapter 31

Additional reading

Gambro (2004). Acute Renal Failure: Renal Intensive care Self Directed Module. Version 1, Gambro Lundia AB.

References

Morton, P and Fontaine, D. (2013). *Critical care nursing: a holistic approach*. 10th ed. Lippincott, Williams & Wilkins: Philadelphia.

Gambro (2004). Acute Renal Failure: Renal Intensive care Self Directed Module. Version 1, Gambro Lundia AB.