RPSG & KDARs Collaborative

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The Renal Patient Support Group

Laboratory Tests, Biomarkers & Investigations

Chronic Kidney Disease (CKD)

Stage 1 (CKD1):
Renal damage with normal or raised GFR → more than 90mL/min/1.73m^2

Stage 2 (CKD2):
Renal damage, the GFR normal or raised → 60 – 89mL/min/1.73m^2

Stage 3 (CKD3):
Moderately reduced GFR → 30-59mL/min/1.73m^2

Stage 4 (CKD4):
Severely reduced GFR → 15-29 mL/min/1.73m^2

Stage 5 (CKD5):
End- Stage Renal Disease (ESRD), GFR less than 15mL/min/1.73m^2

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Muhammad S, Gardner J, Gardner V (2020). Is There a Need for Healthcare Scientists and Educational Support Surrounding Chronic Kidney Disease (CKD) Laboratory Investigations and Tests? Summarizing Polls via a Patient Support Group Platform. Turk J Nephrol; 29(2): 141-52.

Creatinine

- Routine biomarker used to determine renal function
- Creatinine is a by-product of muscle breakdown and excreted by ne[hrons (filtering components of the kidneys)
- The normal range of creatinine: 60 - 120μ mmol/L
- When kidneys fail, the creatinine goes up as kidney function go down.
- Creatinine levels may be either very high or very low in early stage of a CKD diagnosis

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Ihmoda IA, Turner N (2005). “What I tell my patients about blood and urine tests”. British Journal of Renal Medicine 10(2), 15-18

estimated Glomerular Filtration rate (eGFR)

- eGFR is a clinical standard to determine estimation of renal function
- eGFR is used for the definition and staging of CKD
- eGFR equations are used to determine renal function and renal filtration biomarkers markers are used in conjunction, for example, serum creatinine or Cystatin C with other parameters

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Shlipak MG, et al. (2013). Cystatin C versus Creatinine in Determining Risk Based on Kidney Failure'. The New England Journal of Medicine 369(10), 932-943

Blood, Urea, and Nitrogen

- Urea is a molecule produced from protein excreted by the kidneys.
- The level of urea in the blood rises in kidney disease
- The amount of protein, which you eat can affects the test results
- The value can rise if you are dehydrated.
- Normal value: 2.5 – 6.6 mmol/L, according to general healthy population
- Urea is accumulated in blood in CKD.
- It is not an accurate test for kidney function, but it useful when used together with creatinine tests.

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Ihmoda IA, Turner N (2005). “What I tell my patients about blood and urine tests”. *British Journal of Renal Medicine* 10(2), 15-18

Potassium

- Potassium comes from fruit, vegetables and nuts.
- Potassium rises in people with renal failure.
- Potassium levels can rise with some drugs, and it can be dangerous when the kidneys do not function adequately.
- High potassium levels can be treated by giving glucose or insulin.
- If the potassium level is high, and kidney function is low, dialysis or a form of renal replacement therapy is necessary.
- Normal range in healthy population 3.6 – 5.0 mmol/L.

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Sodium

- Sodium comes from salt.
- It increases, when the kidneys do not function well.
- Increased sodium can cause high blood pressure, swelling and fluid on the lungs.
- Diuretic medication increase the sodium put out in urine.
- Most of the renal patients need to eat less salt in the food and many may require diuretics.
- Normal value in healthy population is 135 – 145 mmol/L
- When there is renal insufficiency, the sodium rarely changed.

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Ihmoda IA, Turner N (2005). “What I tell my patients about blood and urine tests”. British Journal of Renal Medicine 10(2), 15-18

Anaemia

A reduction of blood haemoglobin [Hb] concentration, a common problem in CKD.

It is present when blood Hb concentration is reduced to levels below that necessary to adequate tissue oxygenation.

Anaemia is investigated in patients with CKD when Hb levels fall below (in healthy population):

- 11.5g/dl in adult females,
- 13.5g/dl in adult males,
- 12.0g/dl in adult males over 70 years

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Sexton J, Vincent M (2004). Managing anaemia in renal failure. The Pharmaceutical Journal 273, 603-605

Anaemia

Anaemia is a well-known complication in chronic kidney disease and associated with progression of CKD, poor quality of life, and increase in morbidity and mortality (Ryu et al. 2017). The mechanisms of anaemia in CKD are multifactorial and include:

- Erythropoietin (EPO) deficiency from reduced renal mass,
- Iron and nutritional deficiencies
- Various pro-inflammatory mediators commonly elevated in CKD may affect the erythropoiesis in CKD
- Uraemic-induced inhibitors of erythropoiesis,
- Shortened erythrocyte survival, and disordered iron homeostasis (Ryu et al. 2017).

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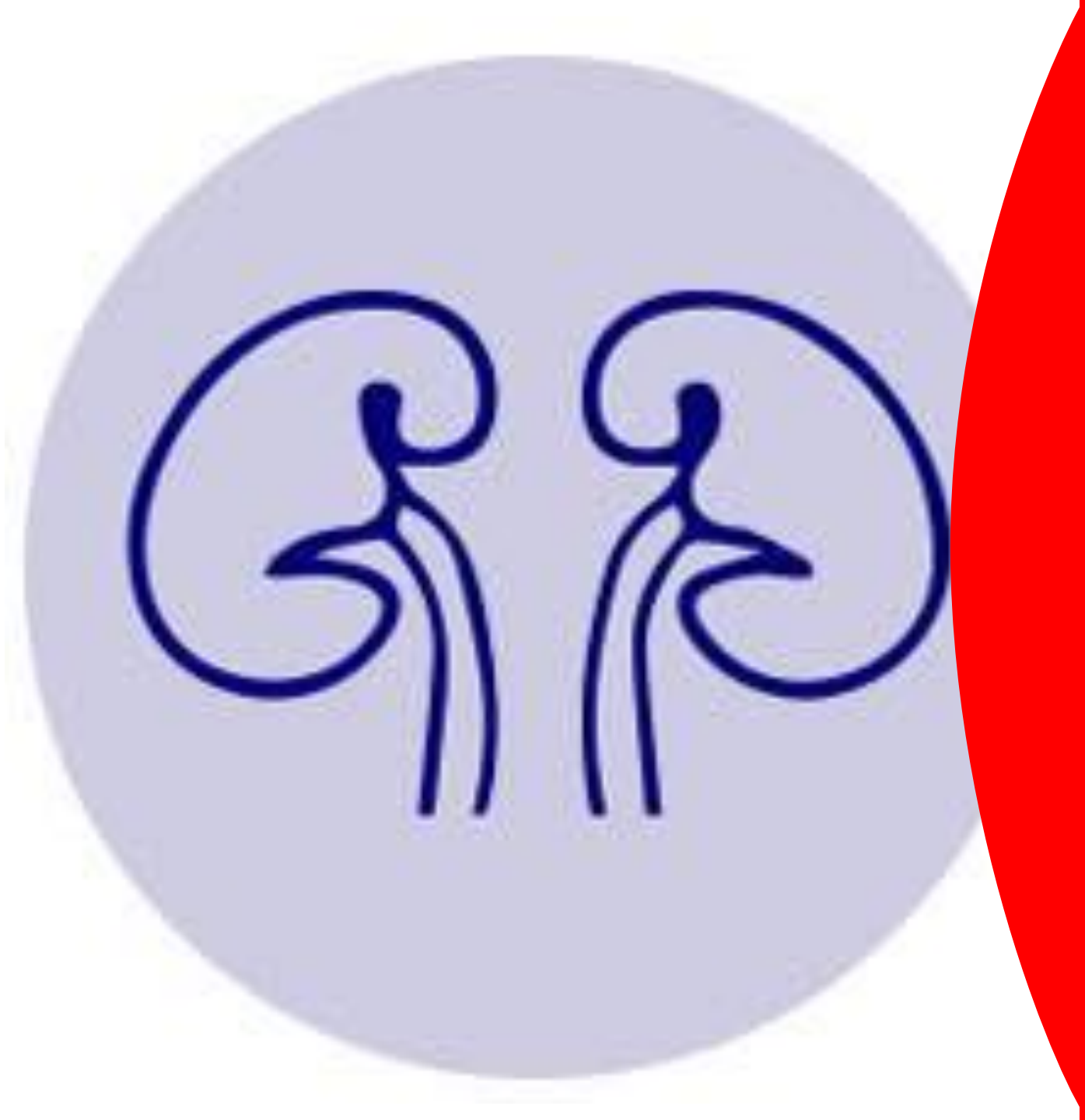
Ryu S-R, Park SK, Jung JY, Kim YH, Oh YK, Yoo TH, Sung S (2017). The Prevalence and Management of Anaemia in Chronic Kidney Disease Patients: Result from the Korean Cohort Study for Outcomes in Patients With Chronic Kidney Disease (KNOW-CKD). J Korean Med Sci; 32: 249-256.



Anaemia

Anaemia increases progressively as the estimated glomerular filtration rate (eGFR) declines to lower than 60 mL/min/1.73m2 (Ryu et al. 2017).

Anaemia is associated with poor outcomes, including higher mortality in patients with end-stage kidney disease (ESKD) and in those with non-dialysis-dependent CKD (Coyne et al. 2017).

The prevalence of anaemia is high in patients with non-dialysis CKD and increases as CKD progress, being present in approximately 42% of patients with stage 3 CKD, increasing to approximately 76% in stage 5 CKD (Coyne et al. 2017).

Non-dialysis CKD and patients on dialysis have disordered iron metabolism due to increases in hepcidin, the regulator of iron absorption and release from cells (Coyne et al. 2017).

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Coyne DW, Goldsmith D, MacDougall IC (2017). New options for the Anaemia of chronic kidney disease. Kidney International Supplements; 7: 157-163.

Ryu S-R, Park SK, Jung JY, Kim YH, Oh YK, Yoo TH, Sung S (2017). The Prevalence and Management of Anaemia in Chronic Kidney Disease Patients: Result from the Korean Cohort Study for Outcomes in Patients With Chronic Kidney Disease (KNOW-CKD). J Korean Med Sci; 32: 249-256.

Haemodialysis (HD)

- A form of renal replacement therapy whereby there is a process of removal toxins and waste from blood.
- Treatment does not completely perform all functions of renal anatomy.
- Dialysis is prompted when an individual reaches End-Stage Renal Disease (ESRD) and where filtration rate falls below 15ml/min/1.73m^2.
- This procedure is responsible for development of oxidative stress.

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Vadakedath S, Kandi V. (2017). ‘Dialysis: A Review of the Mechanisms Underlying Complications in the Management of Chronic Renal Failure’. Cureus. 9(8)

Peritoneal Dialysis (PD)

- PD involves using a peritoneal membrane as a filter inside your abdomen.
- A catheter is inserted into the peritoneal cavity.
- It is recommended for younger patients because of its flexibility.
- It can be performed at home.
- The patient must be capable to perform technique or have a carer.
- Peritonitis is a most common infection and complication of PD treatment.

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Teitelbaum I, Burkart J (2003). ‘Peritoneal dialysis’. AJKD 42(5): 1082-1096

Iron Deficiency

Iron is essential for many important biologic reactions, including oxygen transport, cellular respiration, and DNA synthesis (Babitt and Lin 2012).

Systemic iron balance is maintained by regulating dietary iron absorption and iron release from storage sites in the liver (Babitt and Lin 2012).

CKD patients have increased iron losses, estimated at 1-3g per year in haemodialysis patients, due to chronic bleeding from uraemia-associated platelet dysfunction, frequent phlebotomy, and blood trapping in the dialysis apparatus (Babitt and Lin 2012).

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Babitt JL and Lin HY (2012). Mechanisms of Anaemia in CKD. J Am Soc Nephrol; 23: 1631-1634.

ACKNOWLEDGEMENTS

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