CKM Guidance

Cardiovascular-Kidney-Metabolic Disease

Preventing Adverse Outcomes in Cardiovascular Kidney Metabolic Conditions

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Please make sure to periodically check for updated content.

Introduction:

The Cardiac, Renal, Diabetes and Stroke National Clinical Networks in conjunction with the Long-Term Conditions and Planning, Funding and Outcome Teams in Te Whatu Ora have developed national consensus best-practice guidance on optimising Cardiovascular-Kidney-Metabolic (CKM) health and reducing associated risk factors in adults.

The prevalence of CKM disease continues to increase and is a major cause of morbidity and mortality in Aotearoa New Zealand. Importantly, CKM disease also creates significant disparities for Māori, Pacific and Indo-Asian populations which have not improved over the past 20 years. This has provided the catalyst for Aotearoa New Zealand to develop holistic and people centric consensus guidance which aims to improve outcomes for all people with CKM disease and to eliminate current disparities and achieve equity. This kaupapa is an extension of the 2018 New Zealand Cardiovascular Disease Risk Assessment and Management for Primary Care guidelines.

The intent is to have a concise and pragmatic resource for all health professionals working with people who have CKM disease, particularly in community care. The guidance will link into appropriate specialist society detailed guidelines and Health Pathways wherever needed and possible. Our guidance will be updated as evidence and practice evolve. New sections will also be added with time including management of metabolic dysfunction-associated steatotic liver disease, obstructive sleep apnoea and more detailed guidance on interventions for weight loss.

We have included suggestions on the likely best medications to use based on efficacy, tolerance and adherence for prescribers if needed. Tips to improve access and alternatives are provided when these medications are not funded or available. As per all aspects of the guidance, all suggestions are recommendations only and clinical judgement and individualisation of care remain paramount. This link summarises the changes in the guidance from previous care.

Instructions:

The guidance is separated into the multiple sections.

Clicking on the yellow highlighted text will take you to the relevant section of the guidance within this document.

Clicking on a pink highlighted abbreviation will take you to the relevant abbreviation within the abbreviations section of this document.

Clicking on a blue link will open relevant external guidance in a new window for more detailed information.

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Abbreviations

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1. Commitment to equity in CKM care

Te Tiriti O Waitangi and our Shared Obligation

Te Tiriti O Waitangi and our Shared Obligation

- Our clinical guidance is grounded in the articles and principles of Te Tiriti o Waitangi, which form the foundations of Pae Ora – healthy futures for Māori. We acknowledge the full text of Te Tiriti and the Ritenga Māori Declaration, which affirm Māori rights to tino rangatiratanga, equity, and the protection of cultural identity and mātauranga Māori
- Te Tiriti establishes obligations for the entire health system, including how care is designed, delivered, and experienced by Māori. These obligations are active and enduring, and must shape every aspect of our response to CKM conditions, from prevention to treatment to long-term support.

Equity in CKM conditions

Equity in CKM conditions

- While Te Tiriti provides the foundation for Māori health, equity is a broader commitment that extends to all groups who experience disadvantage in health outcomes.
- CKM conditions drive some of the deepest and most entrenched health inequities in Aotearoa, particularly for Māori, Pacific, and Indian populations and the intersection with rurality, disability and co-existence of mental health conditions. These groups experience earlier onset, more severe complications, and higher mortality

from CKM conditions. They are also less likely to receive timely, evidence-based, and culturally safe care.

- These inequities are not caused by individual choices or cultural behaviours, but stem from:
    - The historical and ongoing impacts of colonialism as it applies to Māori
    - Institutional racism and implicit bias
    - Unequal access to high-quality, culturally safe healthcare
    - Chronic underfunding of Māori- and Pacific-led models of care

- We also recognise the health needs and strengths of other populations who face structural barriers, including refugees and those experiencing socioeconomic hardship or disability.
- To change these outcomes, **our guidance must be implemented with intentionality, accountability, and resourcing** — especially to support Māori and Pacific-led solutions that are by, with, and for communities.
- **We can all achieve equity in CKM care by**:
    - Reflecting and challenging how personal, institutional and systemic racism shapes our practice
    - Create safe environments where Māori and Pacific whānau feel safe, respected, and understood: Prioritise whanaungatanga and mana-enhancing approaches.
    - Support whānau-centred models of care, including kaupapa Māori and Pacific providers where available. Enable shared decision-making that honours lived realities, cultural values, and whānau strengths.
    - Address social determinants of health wherever possible such as housing, kai/food security, and income, which shape CKM conditions and their management.
    - Challenging system constraints and continuing to advocate for access and funding to diagnostics, treatments (e.g. GLP1Ra, SGLT2i, CGMs, nsMRAs, PCSK9Is, etc.), and support services to reduce disparities.

Why equity matters

Why equity matters:

- Achieving equity is not an aspirational marker of clinical excellence — it is the ethical foundation of quality care.
- Every clinical interaction is an opportunity to either uphold or underpin equity.
- We encourage you to use this guidance not only as a technical resource, but as a platform for transformative practice that challenges disparities and improves outcomes for all those most affected with CKM conditions.

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2. Outline, Screening and General Treatment of CKM conditions

Outline of CKM conditions

Outline of CKM conditions

- CKM conditions now affect at least one third of adults in Aotearoa New Zealand and combined are the most common cause of death, particularly from cardiovascular disease.
- Common CKM conditions include:
    - Cardiovascular conditions:
        - Hypertension
        - Coronary artery disease
        - Cerebrovascular disease
        - Peripheral arterial disease
        - Heart failure
        - Atrial fibrillation
    - Chronic kidney disease
    - Metabolic conditions:
        - Obesity
        - Diabetes
        - Dyslipidaemia
        - Gout
        - Metabolic dysfunction-associated steatotic liver disease (MASLD) – previously non-alcoholic fatty liver disease (NAFLD)
- CKM conditions may occur in isolation, but it is important to consider them together because:
    - People with one CKM condition often have other CKM conditions due to shared risk factors and pathophysiology
    - Whānau are not just one body part and management of all CKM conditions is important in optimising health and reducing cardiovascular risk
    - Management of each CKM condition is often influenced by other CKM conditions present

- Management of one CKM condition can help prevent and manage other CKM conditions
- This enables delivery of co-ordinated and integrated care

Screening for CKM conditions

Screening for CKM conditions

- Early identification of CKM conditions are important to maximise the chance of remission and to prevent and delay complications and other CKM conditions.
- Screening for CKM conditions now occurs at the CKM risk assessment (CKMRA). The CKMRA replaces and expands from the traditional CV risk assessment to include screening for excess adiposity, kidney disease, gout and OSA. The first CKMRA is performed earlier in higher-risk groups which include:
  - Māori, Pacific, Indo-Asian and other non-European ethnicities
  - Significant mental illness
  - Long term glucocorticoid and/or antipsychotic use
  - First-degree family history of CKM conditions at < 40 years of age
  - Unemployment and low family income
  - Kai unavailability/insecurity
  - Tobacco smoking
  - Excessive alcohol intake
  - Prediabetes (HbA1c 42 – 47 mmol/mol)
  - History of preeclampsia or gestational diabetes
  - Chronic inflammatory conditions e.g. autoimmune inflammatory disease
  - Clinical features of insulin resistance e.g. acanthosis nigricans, PCOS etc.
  - Chronic dental and/or periodontal disease
  - Sleep disorders
  - Post transplant
- Opportunistic screening of CKM conditions remains important because many people will have contact with the health system with related presentations (e.g. recurrent infections with diabetes) or incidental findings (e.g. obesity at immunisations) years before their first or next CKMRA is due.

General Treatment of CKM conditions

General Treatment of CKM conditions

- Best management of CKM conditions is important in achieving equitable outcomes, prolonging healthy years of life and reducing early death from cardiovascular and renal disease.
- Early identification and ongoing appropriately intensive management of CKM conditions to treatment targets is essential to reduce CV events and organ-specific complications. Therefore, a CKM risk assessment (CKMRA) should be performed after diagnosing any CKM condition, as these conditions often coexist and best management depends on the presence of other CKM conditions, whether the CKM condition is high risk, and the calculated 5-year cardiovascular risk.
- High risk CKM conditions include ANY of:
    - CV disease including asymptomatic coronary or carotid disease (includes CT calcium score > 300)
    - Diabetes with any microvascular or macrovascular complication(s)
    - LDLc ≥ 4.9 mmol/L and/or familial hypercholesterolaemia
    - UACR ≥ 30 mg/mmol
    - eGFR < 45 mL/min
    - UACR 3 – 29 mg/mmol AND eGFR 45 – 59 mL/min
- CV risk is now re-stratified as:
    - High CV risk → 5 year CV risk ≥ 10%
    - Moderate CV risk → 5 year CV risk 5 - 9.9%
    - Low CV risk → 5 year CV risk < 5%
- **Treatment of CKM conditions should be offered when benefits are likely to outweigh risks based on best available evidence. Treatment decisions should be made through shared decision-making** with the person and their whānau, considering capacity to benefit, potential adverse effects, personal values, treatment alternatives, comorbidities, and life expectancy. Whilst absolute benefit is greatest in high risk CKM conditions, individuals deemed 'low or moderate risk' often still gain net treatment benefit, particularly if young and/or if marked abnormalities e.g. significantly elevated blood pressure.
- High risk CKM conditions include ANY of:
    - CV disease including asymptomatic coronary or carotid disease (includes CT calcium score > 300)
    - Diabetes with any microvascular or macrovascular complication(s)
    - LDLc ≥ 4.9 mmol/L and/or familial hypercholesterolaemia
    - UACR ≥ 30 mg/mmol
    - eGFR < 45 mL/min
    - UACR 3 – 29 mg/mmol AND eGFR 45 – 59 mL/min
- **Healthy living interventions are the cornerstone of management of CKM conditions regardless of body weight.** Six key areas should be optimised:

1. Education and support
2. Holistic care
3. Healthy eating
4. Physical activity
5. Healthy sleep
6. Interventions for weight loss if excess adiposity → important because excess adiposity is often the primary driver of CKM conditions.

- Despite best attempts at healthy living, pharmacotherapy is often required to reach treatment targets. Specific recommendations for best pharmacotherapy for each CKM condition are discussed in each section of the guidance.
    - **NB:** There are currently marked disparities in best practice prescribing for people with CKM conditions, notably, less prescribing according to need for Māori and Pacific communities. Māori and Pacific people with CKM conditions have loudly expressed that these disparities can only be reduced by never assuming whether the individual will take medication or not and ensuring best practice is always offered regardless of funding. Failure to do so:
    - Undermines basic tikanga concepts such as rangatiratanga, manaakitanga and kaitiakitanga
    - Is a breach of Te Tiriti o Waitangi
    - Ignores evidence that best practice provides the greatest chance to achieve:
        - Equitable outcomes in CKM conditions
        - Standardised care with reduced regional variation
        - Increased learning, efficiency and accountability in the health system
        - Lowest cost of care to the health system
        - BP and lipid lowering medications are now some of the most cost-effective interventions in health

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3. CKM Risk Assessment (CKMRA)

Role of the CKMRA

Role of the CKMRA

The CKMRA replaces and expands from the traditional CV risk assessment to include screening for excess adiposity, kidney disease, gout and OSA to enable early intervention, more accurate calculation of CV risk and best management based on overall risk. The CKMRA should include:

- Seated +/- standing blood pressure to screen for high blood pressure
    - Measure sitting/standing or ideally lying/standing BP if any concerns over postural hypotension e.g. postural symptoms, frail, elderly etc.
- HbA1c +/- fasting glucose to screen for diabetes
    - A fasting glucose is typically only required if measurement of HbA1c may be unreliable due to:
        - Any haemoglobinopathy e.g. thalassaemia, sickle cell etc.
        - Altered red cell turnover e.g. bleeding, haemolysis, pregnancy
        - Post blood transfusion
- eGFR and Urinary ACR to screen for chronic kidney disease
- Non-fasting lipid studies to screen for dyslipidaemia
- Waist circumference, height and weight to screen for excess adiposity
- Smoking and alcohol history
- Gout history → serum urate if history suggestive
- Epworth Sleep score if history suggestive of OSA
- Calculation of CV risk using CKM-RAM calculator
    - CKM-RAM is an updated version of the PREDICT calculator to more accurately calculate CV risk by including significant mental illness, chronic kidney disease and gout

Timing of the first CKMRA

Timing of the first CKMRA

- A CKMRA should be performed whenever a CKM condition is diagnosed even if < 30 years of age including ANY of:
    - HbA1c ≥ 48 mmol/mol
    - Blood pressure ≥ 130/80 mmHg
    - LDLc ≥ 4.9 mmol/L
    - Chronic kidney disease (UACR > 3 mg/mmol and/or eGFR < 60 mL/min)
    - Obesity
- Otherwise the first CKMRA should be performed at the following ages:

Population subgroup	Men	Women
Māori, Pacific peoples or Indo-Asian peoples	30 years	40 years
Individuals with risk factors for CKM conditions*	35 years	45 years
Individuals without risk factors for CKM conditions	45 years	55 years
People with severe mental illness	25 years	25 years

*Risk factors for CKM conditions include:

- First-degree family history of CKM conditions at < 40 years of age
- Unemployment and low family income
- Kai unavailability/insecurity
- Tobacco smoking
- Excessive alcohol intake
- Prediabetes (HbA1c 42 – 47 mmol/mol)
- History of preeclampsia or gestational diabetes
- Chronic inflammatory conditions e.g. autoimmune inflammatory disease
- Clinical features of insulin resistance e.g. acanthosis nigricans, PCOS etc.
- Long term glucocorticoid and/or antipsychotic use
- Chronic dental and/or periodontal disease
- Sleep disorders
- Post transplant

Stratifcation of CV and CKM risk

Stratification of CV and CKM risk

- CV risk is now stratified as:
    - High CV risk → 5 year CV risk ≥ 10%
    - Moderate CV risk → 5 year CV risk 5 – 9.9%
    - Low CV risk → 5 year CV risk < 5 %
    - **NB:** 10 year CV risk is approximately 2.5 times greater than 5 year CV risk and may be more useful in shared decision making with younger people

        - E.g. 5 year CV risk of 5% equates to a 12.5% or 1 in 8 chance of a CV event within the next 10 years

- CKM conditions may be high-risk independent of the calculated CV risk and include ANY of the following:
    - CV disease including asymptomatic coronary or carotid disease (includes CT calcium score > 300)
    - Diabetes with any microvascular or macrovascular complication(s)
    - LDLc ≥ 4.9 mmol/L and/or familial hypercholesterolaemia
    - UACR ≥ 30 mg/mmol
    - eGFR < 45 mL/min
    - UACR 3 – 29 mg/mmol AND eGFR 45 – 59 mL/min

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4. Healthy living interventions

Education and Support

Education and support

- Adequate education and support is essential in empowering all individuals and whānau to self-manage their CKM condition(s) to achieve best outcomes.
- Providing evidence-based advice that applies to people living in Aotearoa New Zealand is paramount.
- Identifying and addressing social determinants of health and barriers to self-management are important in CKM conditions. The Wellbeing Wheel can be a useful tool to discover the greatest priorities and urgent needs of people with CKM conditions.
- Utilise specialists within the primary care multidisciplinary team to optimise health outcomes. In addition to the GP and nursing team, many practices have access within the practice, PHO or community clinicians including:
    - Dietitian (diet/nutrition and prescribing thereof)
    - Health Improvement Practitioner (to support mental/psychological wellbeing/adherence)
    - Pharmacist (medicines and prescribing)
    - Podiatrist (feet)
    - Psychologist (to support mental/psychological wellbeing)

    - Social worker (social support and wellbeing)
    - Support from the unregulated workforce is also important:
    - Health Coach (to support physical wellbeing and behaviour change)
    - Kaiāwhina (to support navigation of the healthcare system)

- Continuity of care is also important, where people and their care teams collaborate to achieve a shared goal. Continuity of care is associated with increased adherence, less acute care, reduced hospital admissions, and better mental health
- Utilise locally available courses and programmes that are relevant and evidence-based to improve health outcomes, e.g. Green Prescription, Diabetes Self-Management Education (DSME), Cardiovascular Rehabilitation/Self-Management, etc.

Holistic care

Holistic care

- Smoking cessation remains critical and should be offered yearly if smoking tobacco
    - There is increasing evidence that vaping may increase the risk of CV disease, lung disease and heart failure → so vaping should likely only be seen as an interim measure for stopping smoking
- Discuss reduction of alcohol intake and other recreational drugs as required
    - There is no safe alcohol limit in CV health.
    - Risk of all recreational drugs including marijuana and methamphetamine are much greater in people with CKM conditions.
    - Reduction in kava intake can aid CKM management
- Ensure vaccination status is up to date given greater adverse effects of communicable diseases in people with CKM conditions
- Ensure cancer screening as per national recommendations is up to date given greater risk of solid cancers, particularly in obesity and type 2 diabetes
- Screen for depression and treat as required as high risk of depression in CKM conditions
    - Scores ≥ 3 should prompt further screening with PHQ-9 or other tools with referral to psychology and pharmacological treatment as required.
- Screen for diabetes distress if known diabetes
    - Scores ≥ 3 on DDS2 highlights need to fully evaluate diabetes distress and consider support as appropriate
- Contraception and pregnancy advice should be discussed in women of childbearing age

- Optimise treatment of non-CKM conditions that increase CV risk – these include but are not limited to:
    - Dental and periodontal disease
    - Mental Health Disorders e.g. depression and anxiety
    - Chronic inflammatory conditions e.g. rheumatoid arthritis, SLE, inflammatory bowel disease, psoriasis, HIV/AIDS etc.
    - Respiratory and sleep disorders e.g. COPD, asthma and OSA
    - Endocrine disorders e.g. PCOS, thyroid disease, hypogonadism etc.

Healthy eating

Healthy eating

- Healthy eating is essential for all people with CKM conditions, irrespective of body weight.
- Social determinants of health and the unequal distribution of obesogenic environments are major risk factors for the development of CKM conditions, particularly for Māori and Pacific communities. Screen for household food insecurity, assess knowledge on how to achieve a healthy, balanced diet on a budget and utilise social workers, kaiāwhina or health navigators input as required.
    - Screen for household food security with these 2 questions using the scale ‘often true’ or ‘sometimes true’ (vs. ‘never true’). Often true should be referred to a social worker +/- kaiāwhina for support
    - Within the past 12 months we worried whether our food would run out before we got money to buy more
    - Within the past 12 months the food we bought just didn’t last and we didn’t have money to get more
- Strongly consider referral to a dietitian to improve nutrition-related biomarkers and/or if requiring personalised nutrition advice
- General principles of healthy eating in CKM conditions align to guidance on healthy eating for all New Zealanders. People with or at risk of CKM conditions should be encouraged to enjoy a variety of nutritious foods every day, including:
    - Plenty of vegetables (5 serves/day) and whole fruit (2 serves/day) - fresh, seasonal, frozen, low-salt or low-sugar canned options are acceptable
    - Good quality grain foods, mostly whole grains, wholemeal grains and those naturally high in fibre
    - Some reduced-fat milk and milk products, or calcium fortified plant-based alternatives
    - Some protein foods (2.5 – 3 serves/day) - legumes, unsalted nuts, seeds, fish and other kai moana (seafood), eggs, poultry and/or red meat with the fat removed.
        - Promote plant-based protein e.g. legumes and nuts, and limit cooked red meat to less than 500g per week/person

- Avoiding calorie dense and nutrient-poor food and drinks as much as possible e.g. ultra processed foods including packaged snacks, processed meats, sugary drinks etc.
- People with or at risk of CKM conditions should be encouraged to choose and prepare foods and drinks:
  - With unsaturated fats and oils (e.g. canola, rice bran, avocado, olive, plant-based margarines) instead of saturated fats and oils (e.g. coconut, lard, butter)
  - Low in salt (sodium)
  - With little or no free sugar
  - That are mostly wholefoods, or less processed
  - Make water their first choice over juices, energy and sports drinks or other carbonated drinks

- “Following a diet” might be interpreted as a time-bound activity, but these general principles of a balanced, healthy eating pattern with small changes made over time are more sustainable long term. Evidence-based eating patterns to support people living with CKM conditions include:
  - Mediterranean diet (whole, plant-based foods, moderate reduced fat dairy, moderate animal protein, high Ω3 & Ω9, high fibre)
  - Dietary Approaches to Stop Hypertension – DASH (adapted Mediterranean with low sodium, high calcium, potassium & magnesium)
  - Portfolio diet (adapted Mediterranean, increased plant sterols, unsalted nuts, soy protein and soluble fibre)

- Specific recommendations for healthy eating in each CKM condition are discussed in the relevant section

Physical activity

Physical activity

- Any increase in physical activity is beneficial. Current recommendations for non-pregnant adults include:
  - 150 minutes of moderate or 75 minutes vigorous aerobic exercise per week spread over ≥ 3 days each week with ≤ 2 consecutive days without exercise
  - ≥ 2 sessions of resistance exercise at low to moderate intensity per week
  - Sitting for < 30 minutes at a time
  - **NB:** The intensity and duration of the exercise may need to be reduced due to comorbidities such as heart disease and previous stroke etc.
    - Ensure safety including adequate footwear if increased foot risk e.g. peripheral arterial disease.

- Some of these recommendations may not be immediately realistic for many people with CKM conditions. Resistant exercise can be an effective form of exercise for people with high body weight, but support should

be provided to increase physical activity and movement as much as possible, given the significant benefits:

- Explain that any physical activity is better than being sedentary
- Physical activity and movement can take on many forms rather than ‘exercise’.
  - Housework, gardening, dance, walking around shops, taking the stairs and mowing the lawns etc. are effective and sometimes overlooked forms of physical activity.
- A 5 – 6 minute brisk walk a day is associated with an additional 4 years of life
- Adding 500 steps per day is associated with up to 10% reductions in mortality
- Moving briskly doing everyday activity is associated with up to 50% reductions in CVD
- Stretching reduces blood pressure and glucose levels

Healthy sleep

Healthy sleep

- Sleep disorders are common in people with CKM conditions and are associated with weight gain, above-target glucose levels, high blood pressure, arrhythmias and cardiovascular disease.
- Obstructive sleep apnoea (OSA) is the most common sleep disorder in people with CKM conditions. Treatment of OSA significantly reduces sleep-related respiratory events with associated improvements in HbA1c and blood pressure.
- Discuss healthy sleep patterns and good sleep hygiene with all people with CKM conditions
  - Optimal length of overnight sleep for beneficial effects on body weight and CKM conditions appears to be 6 – 8 hours every day.
  - Unfortunately, ‘catch-up’ sleep does not fully reverse the deleterious effects of insufficient sleep duration across the week → beware of the risk of CKM conditions in shift workers.
  - Screen for OSA and other sleep disorders when appropriate for all people with CKM conditions

Interventions for weight loss

Interventions for weight loss

- Interventions for weight loss should be considered in all people with CKM conditions who are overweight or obese. These are discussed in detail here
- Pharmacotherapy and bariatric surgery for weight loss can be considered if failure to reach medical targets for weight loss with nutritional strategies alone.
  - 5% total body weight loss significantly improves the majority of metabolic parameters including glucose levels, BP, and lipid profile. This may allow for a reduction in medications, but typically, greater weight loss is required for remission of CKM conditions.

- At least 10-15% total body weight loss is typically required to achieve remission of:
  - Type 2 diabetes
  - OSA
  - Hypertension
- At least 15-20% reduction in total body weight loss is typically required to achieve remission of:
  - Metabolic dysfunction-associated steatotic liver disease
  - Heart failure with preserved ejection fraction

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5. Management of obesity

Definition of overweight, obesity and excess adiposity

Introduction

The Aotearoa New Zealand CKM Guidance Group has identified an urgent need for new, detailed national guidance on obesity and weight management. This section provides interim guidance until that new national guidance is available.

Definition of overweight, obesity and excess adiposity

- Definitions of overweight and obesity were traditionally defined by BMI or waist circumference (WC) by sex and by ethnicity

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- Although BMI is an effective screening tool, there is now a new international consensus definition of obesity due to the limitations of BMI identifying excess adiposity at the individual level when the BMI is < 40 kg/m^2. The new international definition of excess adiposity and obesity is:
  - BMI > 40 kg/m^2 OR
  - BMI > ethnicity-specific threshold AND ≥ 1 anthropometric criteria OR
    - > 35 kg/m^2 in Pacific Peoples
    - > 32 kg/m^2 in Māori
    - > 30 kg/m^2 in Europeans
    - > 25 kg/m^2 in Indo-Asian Peoples

        - NB: Threshold is based on predominant ethnicity

    - ≥ 2 anthropometric measures regardless of BMI
    - Anthropometric features of increased risk are:
        - Waist:height ratio > 0.5
            - Likely most pragmatic as easily performed and not ethnicity- or sex-specific
        - Waist:hip ratio > 0.86 in women and > 1 in men
        - Waist circumference:
            - Non Indo-Asian ethnicity > 88 cm in women; >102 cm in men
            - Indo-Asian ethnicity > 80 cm in women; > 90 cm in men
        - Body fat percentage by DEXA or bioimpedance > 30% for men and > 42% for women

- Excess adiposity is now also defined as either being preclinical or clinical obesity, to identify those greatest at risk and to enable targeted and prioritised interventions.
    - Preclinical obesity is defined as excess adiposity with preserved function of other tissues and organs.
        - The term healthy obesity should no longer be used given the high lifelong risk of developing clinical obesity
    - Clinical obesity is defined as excess adiposity impacting the function of tissues, organs and the entire individual.
        - Impacted function may be due to CKM and/or non-CKM conditions
            - CKM conditions include:
                - Cardiovascular disease
                - Chronic kidney disease
                - Elevated blood pressure and hypertension
                - Type 2 diabetes
                - Dyslipidaemia
                - Gout
                - Metabolic dysfunction-associated steatotic liver disease

        - Non CKM conditions include:
            - Joint pain and osteoarthritis
            - Reduced age-adjusted mobility
            - Lymphoedema
            - Idiopathic intracranial hypertension (or raised intracranial pressure without space-occupying brain lesion)
            - Obstructive sleep apnoea

Overview of interventions for weight loss

Overview of interventions for weight loss

- An evidence-based, personalised weight loss plan is strongly recommended for all people with clinical obesity.
    - Conversations about weight loss MUST be conducted with a positive, culturally safe and non-judgmental approach.
    - The 5A's framework of Ask, Advise, Assess, Agree, Arrange may be helpful
    - The focus should be on weight loss for health reasons with medical targets rather than societal targets for weight loss or a specific ‘ideal weight’.
        - 5% total body weight loss significantly improves the majority of metabolic parameters including glucose levels, BP, and lipid profile. This may allow for a reduction in medications, but typically, greater weight loss is required for remission of CKM conditions.
        - At least 10-15% total body weight loss is typically required to achieve remission of:
            - Type 2 diabetes
            - OSA
            - Hypertension
        - At least 15-20% reduction in total body weight loss is typically required to achieve remission of:
            - Metabolic dysfunction-associated steatotic liver disease
            - Heart failure with preserved ejection fraction
- Ongoing care is essential because sustained weight loss can be difficult, given that obesity is typically a lifelong remitting and relapsing disease. Twin studies have shown that genetics account for 50 -70% of the

variation in body weight and body weight is vigorously defended by increased appetite and reduced mitochondrial energy expenditure through no fault of the person. Ongoing supportive care includes:

    - Whānau-inclusive strategies, which are likely more effective over the longer term and benefit the whole whānau.
    - Dietitian-led nutrition plan with a health coach supporting the patient to implement the plan under dietitian guidance.
    - Where dietitian care is unavailable, ensure basic screening for the frequency of sugary drinks, alcohol, takeaway foods, processed snacks, and sweets, and the ability/motivation to shift to a routine of home-prepared nutritionally balanced meals.
    - Psychology input if any concerns of disordered eating or depression.
    - Screen for household food insecurity, assess knowledge on how to achieve a healthy, balanced diet on a budget and utilise social workers, kaiāwhina or health navigators input.
        - Screen for household food security with these 2 questions using the scale ‘often true’ or ‘sometimes true’ (vs. ‘never true’). Often true should be referred to a social worker +/- kaiāwhina for support
        - Within the past 12 months we worried whether our food would run out before we got money to buy more
        - Within the past 12 months the food we bought just didn’t last and we didn’t have money to get more
    - Refer to local weight loss programmes if evidence-based and available.

- Healthy lifestyle interventions alone are often sufficient, but additional strategies for weight loss should be considered for all with preclinical or clinical obesity as appropriate, particularly people with CKM conditions who are overweight. These strategies include:
    - Specific nutritional strategies for weight loss
    - Pharmacotherapy for weight loss
    - Bariatric surgery

Nutritional strategies for weight loss

Nutritional strategies for weight loss

- There is currently no conclusive evidence that any specific nutrition strategy is superior to any other for long-term weight management.
- Pragmatically, the best nutrition strategy for weight loss is the one that works and people can maintain, is nutritionally adequate, reduces the risk of as many obesity-related conditions as possible and is sustainable.
- In general long-term nutrition needs to optimise fibre, low saturated fat, and include wholegrains, alongside fruit and vegetables.

- Current evidence suggests the following strategies are effective in achieving long-term weight loss and reductions in CKM conditions
    - Intensive very-low-energy-diet programs with ongoing support to achieve a healthy, balanced diet long term e.g. DiRECT style intervention
        - The DiRECT Trial intervention was a very low energy total meal replacement for 12-20 weeks followed by step wise food reintroduction with ongoing support to sustain a healthy, balanced diet.
    - Mediterranean diet
    - Dietary approach to stop hypertension (DASH)
    - Plant-based diets
    - High fibre low fat diets
    - Low carbohydrate diets → carbohydrates 20 - 45% of total energy expenditure (~130g carbohydrates per day).
        - Not recommended if on empagliflozin due to risk of ketoacidosis
- Other strategies that appear safe and effective to achieve weight loss in the short-term and awaiting long term data include:
    - Very low carbohydrate or ketogenic diets → carbohydrates < 20% of total energy expenditure (~ 50g of carbohydrates per day)
        - Requires monitoring of CV risk long term due to increased dyslipidaemia
        - Not recommended if on empagliflozin due to risk of ketoacidosis
    - Intermittent fasting
- Dietary approach should be an informed shared decision determined by health risk biomarkers, medications and related conditions (e.g. presence of CKD or dyslipidaemia), personal preference, cultural acceptability, tolerability, affordability and nutritional adequacy.
- Particular care needs to be taken to ensure adequate nutrition in children, pregnancy, breastfeeding and the elderly or anyone at risk of sarcopenia
    - Remember being mildly overweight is protective in the elderly
    - Adequate protein intake and physical activity is important in maintaining muscle mass
    - Utilise dietitian resources for these high-risk groups

Pharmacotherapy for weight loss

Pharmacotherapy for weight loss

- Pharmacotherapy should be considered if weight loss targets are not reached by nutritional strategies alone AND either BMI > 30 kg/m^2 or BMI > 27 kg/m^2 with at least one obesity-related condition.
- Unfortunately all pharmacotherapy registered for weight loss is not funded in Aotearoa New Zealand but options include:
    - Phentermine (Duromine)
        - Least expensive agent at ~ $80/month
        - Sympathetic side effects may limit use particularly if CV disease
            - Do not use phentermine if CV disease, arrhythmias, untreated hypertension or thyrotoxicosis, substance abuse, pregnancy, breastfeeding or children
        - Phentermine 15 mg daily appears best dose as similar efficacy with less adverse effects than higher doses.
        - Common misconceptions are that phentermine is addictive and can only be used for up to 3 months, which are untrue.
    - Orlistat (Xenical)
        - Cost is ~ $120/month but not used commonly due to GI adverse effects
            - Best to use orlistat with a low fat diet with doses of 120 mg with main meals
    - Buproprion and naltrexone (Contrave)
        - Cost of Contrave ~ $250/month
        - Contrave tablets contain 8 mg of naltrexone and 90 mg of bupropion and titrate based on adverse effects
            - Typically start at 1 tablet per day and increase by 1 tablet per week up to 2 tablets twice daily or maximal tolerated dose.
            - Slow down dose increases adverse effects e.g. nausea, dizziness, headache
            - GI adverse effects typically dissipate within 2-3 week
        - Can be useful to help low mood, or if smoking cessation desired
        - Do not use in pregnancy, breastfeeding, children, uncontrolled hypertension, history of seizures, bipolar disorder, MAOI use or withdrawal of alcohol or benzodiazepines etc.
    - GLP1 receptor agonists (GLP1Ra)
        - Likely the most effective pharmacological treatment for weight loss.

- All current GLP1Ra in NZ are subcutaneous injections.
- Utilise funded liraglutide (Victoza) or dulaglutide (Trulicity) if the person has type 2 diabetes as GLP1Ra are expensive to self-fund.
- Liraglutide (Saxenda) was the principal GLP1Ra used for weight loss in NZ until the arrival of newer GLP1Ra, but is still available:
    - Cost is ~ $480 - $500/month
    - Start at 0.6 mg daily and increase dose by 0.6 mg per day each week to 3 mg daily or maximal tolerated dose
    - Titration can be slowed if adverse effects occur
    - Need to prescribe with BD fine 4 mm or 5 mm needles
- Semaglutide (Wegovy) and tirzepatide (Mounjaro) are now available and are weekly injections that typically lead to greater weight loss and CV protection than liraglutide. Semaglutide is also registered for CV risk reduction alone in those with a BMI ≥ 27 kg/m^2
    - Cost of semaglutide is ~ $370 - $500/month (often variation between pharmacies)
        - Start semaglutide at 0.25 mg weekly and increase every 4 weeks to 0.5mg weekly, 1 mg weekly, 1.7 mg weekly and 2.4 mg weekly or maximal tolerated dose
        - Titration can be slowed if adverse effects occur
    - Cost of tirzepatide is ~ $430 - $900/month (cost is dose-dependent and often variation between pharmacies)
        - Start tirzepatide at 2.5 mg weekly and increase every 4 weeks to 5 mg weekly, 7.5 mg weekly, 10 mg weekly, 12.5 mg weekly, and 15 mg weekly or maximal tolerated dose
        - Titration can be slowed if adverse effects occur
- Ensuring adequate protein and nutrient intake is important if people are prescribed GLP1Ra. People with obesity are already at risk of malnutrition if they consume an energy-dense, nutrient-poor diet. GLP1Ra may worsen this by reducing appetite, which can lead to nutrient deficiencies, loss of lean body mass and bone density.
    - Consider Dietitian referral for advice on nutritionally adequate intake to avoid deficiencies
    - Consider screening and monitoring of muscle wasting and nutritional deficiencies including protein, vitamins A, C, D, E, B12, folate, thiamine, iron, calcium, magnesium and zinc
- Discuss exercise and strength training as important to maintain muscle and bone mass

        - Provide advice with all GLP1Ra on how to reduce adverse effects:
            - Ensure adequate hydration
            - To stop eating when feeling full
            - Eat smaller meals and avoid alcohol, fatty and spicy foods
            - Slow down dose increases if GI adverse effects occur
            - GI adverse effects typically dissipate within 2-3 weeks
            - Doses of sulfonylureas and insulin may need to be reduced to avoid hypoglycaemia
            - Consider halving sulfonylureas and reducing total daily doses of insulin by approximately 20%, particularly if baseline HbA1c < 64 mmol/mol
            - Do not use in pregnancy, breastfeeding , children < 10 years of age, significant GI disease or medullary thyroid cancer
            - GLP1Ra should be stopped once eGFR < 15 mL/min

- The traditional approach with pharmacotherapy is to treat for 3 months to determine if ‘responder’, which is defined as ≥ 5% total body weight loss in this time period.
    - If ‘non-responder’ → consider different pharmacotherapy for weight loss
    - If ‘responder’ → aim to continue treatment until weight loss plateaus. Can then consider:
        - Dose reduction or cessation, but advise weight regain is common and refer to dietitian for review of nutrition plan
        - Continuing current dose to ensure weight stability
            - All pharmacotherapy above safe for at least 3 years
        - Switching to or adding in alternative pharmacotherapy agent
            - Do not use phentermine and bupropion in combination

- Some medications have been used ‘off-label’ for weight loss, particular if cost is an issue:
    - Metformin typically only leads to a maximum average of 2 kg weight loss.
    - Topiramate may be useful if history of migraines or epilepsy
        - Start at 25 – 50 mg daily and can increase to 100 mg daily

        - Often used in combination with phentermine internationally
        - Beware of teratogenic effects

    - Bupropion ± naltrexone may be prescribed individually but the efficacy and safety for weight loss in not known
    - Empagliflozin is not recommended for weight loss unless treating underlying type 2 diabetes, renal disease or heart failure.

Bariatric surgery for weight loss

Bariatric surgery

- Bariatric surgery continues to be an important treatment for weight loss, particularly if targets are not reached with nutritional strategies and pharmacotherapy.
- Access to funded bariatric surgery is limited and varies regionally. Consider if BMI > 40 kg/m^2 or BMI > 35 kg/m^2 with obesity-related conditions:
    - Roux en Y bypass and sleeve gastrectomy appear most effective procedures for weight loss
    - Appropriate patient selection continues to be critical and early referral to a dietitian strongly recommended
        - NB: Upper BMI limit of < 55 - 65 kg/m^2 for bariatric surgery varies across Aotearoa → check with local centre.
- Post-operative care is important after bariatric surgery to reduce complications and to ensure adequate nutrition.
    - Many people are now travelling overseas for bariatric surgery and have no post-operative plan. Please click here if post-operative advice required.

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6. Management of elevated blood pressure and hypertension

Definition of elevated BP and hypertension and how to measure BP

- Treatment of high blood pressure (BP) is likely the most easily modifiable risk factor in preventing CV and renal sequelae in CKM disease so BP should be measured at every practical opportunity
- An individualised approach is recommended taking into account competing risks and preferences and explored through shared decision making

- **Recommended thresholds for treatment of BP depend whether other CKM disease features are present:**
    - **BP ≥ 140/90 mmHg → prompt confirmation and treat with lifestyle advice + pharmacotherapy in all if appropriate**
        - Ideally elevated BP should be confirmed as soon as possible on validated home or ambulatory BP monitoring. Home monitoring is best done in a structured approach e.g. morning and evening BP measurements at rest for 1 week. If home or ambulatory BP monitoring not available then recommend measurement of BP in clinic with the following:
            - Measure BP after 5 mins seated comfortably in a quiet environment with a validated device with an appropriate cuff size with the cuff at the level of the heart.
            - Measure BP 3 times 1-2 mins apart and average the last 2 readings
            - Often useful to measure BP at both arms and to assess whether postural drop at 1st visit to aid monitoring at future visits.
    - **BP 130 – 139/80 - 89 mmHg → prompt confirmation and treat with lifestyle advice + pharmacotherapy if 5 year CV risk ≥ 10% OR ANY of the following:**
        - Ideally elevated BP should be confirmed as soon as possible on validated home or ambulatory BP monitoring. Home monitoring is best done in a structured approach e.g. morning and evening BP measurements at rest for 1 week. If home or ambulatory BP monitoring not available then recommend measurement of BP in clinic with the following:
            - Measure BP after 5 mins seated comfortably in a quiet environment with a validated device with an appropriate cuff size with the cuff at the level of the heart.
            - Measure BP 3 times 1-2 mins apart and average the last 2 readings
            - Often useful to measure BP at both arms and to assess whether postural drop at 1st visit to aid monitoring at future visits.
        - CVD including ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, atrial fibrillation and heart failure
        - Diabetes with any microvascular or macrovascular complication
        - Familial hypercholesterolaemia
        - Chronic kidney disease (eGFR <60mL/min and/or UACR >3 mg/mmol)
        - NB: Gout, MASLD and OSA all independently increase CV risk and events, but are not currently included in the PREDICT CV risk calculator. Therefore, CV risk is likely underestimated in these conditions and treatment of BP 130-139/80-89 mmHg should still be considered in these conditions if no other indications, particularly if 5 year CV risk 4-5%.

    - **BP 130 - 138/80 - 89 mmHg and 5 year CV risk 5 - < 10% → lifestyle advice + treat underlying condition + consider pharmacotherapy IF ANY of the following:**
        - Gout or auto-immune inflammatory disease
        - MASLD
        - OSA
        - Severe mental illness particularly with antipsychotic use
        - Previous gestational diabetes and/or preeclampsia
        - Direct family history of CVD < 40 years of age
        - Cardiac calcium score ≥ 100
    - **BP 130 - 138/80 - 89 mmHg and 5 year CV risk < 5% → lifestyle advice alone**

- **Target systolic BP is 120 – 129 mmHg in most individuals**
    - A systolic BP < 120 mmHg is not concerning if well tolerated and likely preferable if young or heart failure
    - **Relaxed BP targets to the lowest reasonably and safely achievable BP are appropriate if any of the following:**
        - Frailty and/or limited life expectancy
        - Age ≥ 85 years
        - Symptomatic postural hypotension
            - Measure lying/standing or sitting/standing BP if high risk of postural hypotension and treat to standing BP if > 10 mmHg postural drop.
        - Intolerant of antihypertensives
    - Unlike other features of CKM, treating BP has no ‘metabolic memory or legacy effect’. Therefore, BP targets and treatment should only be relaxed when clinically appropriate
    - Diastolic BP targets are now no longer first-line BP targets due to evidence for the greater benefit of controlling systolic BP. Although it is desirable to have a diastolic BP < 80 mmHg, a systolic BP 120 – 129 mmHg is the primary target.

- Treatment consists of both non-pharmacological and pharmacological management and should ideally be based on several ‘out of office’ or home BP monitoring at rest as BP levels in clinic are typically ≥ 5 mmHg higher. Home monitoring is best done in a structured approach e.g. morning and evening BP measurements at rest for 1 week.

    - If out of office BP monitoring not available then measure BP after 5 mins seated comfortably in a quiet environment with a validated device with an appropriate cuff size with the cuff at the level of the heart. Ideally BP should be measured 3 times 1-2 mins apart with the average of the last 2 readings used.

- Non-pharmacological management is always important and is discussed in detail in lifestyle management. Key points include:
    - Smoking and alcohol cessation
        - Increasing evidence that vaping cessation is also beneficial
    - A low-salt diet with fruit and leafy vegetables to ensure adequate potassium intake
    - Increased physical activity and movement
    - Aiming for > 5% total body weight loss if overweight

- First line pharmacotherapy for lowering BP are ACE inhibitors (ACEi) or angiotensin receptor blockers (ARB), calcium channel blockers (CCB) and thiazide diuretics (TD). Typically, these medications are more effective and better tolerated in low doses in combination, but may be used alone. Cardioselective beta blockers (β-blocker) may be used if ischaemic heart disease, heart failure or atrial fibrillation. Guidance on likely best agents in each to use based on efficacy and tolerance is below.
- Best choice of ACEi, ARB, CCB or TD is dependent on whether renal disease predominates and presence of other comorbidities:
    - Chronic kidney disease is present (eGFR <60 mLmin and/or UACR > 3 mg/mmol):
        - Strongly consider ACEi or ARB if no hypotension and check BP in 1 month
        - Titrate ACEi or ARB to maximal tolerated dose
        - Check BP monthly until to target – if BP remains above target add CCB or TD
        - If BP still above target then add other (e.g. TD if CCB previously added)
    - No renal or CV disease:
        - Strongly consider low dose ACEi or ARB and CCB in combination
        - Check BP in 1 month → if above target increase dose of combination agents
        - Check BP monthly until to target - if BP remains above target then add TD
    - Gout:

        - Consider ACEi or ARB as first-line options. (Note that losartan and dihydropyridine CCBs may serve as helpful adjuncts due to their small uricosuric effect, but they should not replace urate-lowering therapy and the focus should be on the most effective anti-hypertensive)
        - Beware TD typically increase uric acid levels
    - Ischaemic heart disease, heart failure or atrial fibrillation:
        - β-blockers may be added at any stage. May need to decrease CCB and/or TD if hypotension
        - In heart failure Entresto (Sacubitril/Valsartan) is preferred renin-angiotensin system inhibitor (rather than ACEi or ARB) but need to meet special authority criteria for funding. Use spironolactone or eplerenone and empagliflozin rather than TD or CCB if risk of hypotension.
            - Current special authority criteria for Entresto requires the following:
                - NHYA/WHO functional II-IV AND
                - LVEF ≤ 35% OR echocardiogram is not reasonably practical AND
                - Other standard treatments of heart failure are optimised
            - Maximise doses of Entresto, β-blocker and spironolactone before addition of other agents.
                - Remember to monitor potassium when using Entresto or spironolactone as per ACEi/ARB below

- Causes of secondary hypertension should be considered if young and/or persistent hypertension despite adherence to ≥ 3 antihypertensive agents
    - Obstructive sleep apnoea is likely the most common cause of secondary hypertension in CKM and is often missed
    - Consider other investigations as appropriate to exclude renal disease, pregnancy, and endocrine conditions such as primary hyperaldosteronism, Cushing’s syndrome, phaeochromocytoma, thyrotoxicosis and acromegaly.
    - Consider referral to secondary care to discuss addition of other antihypertensives as required
        - Spironolactone likely preferable if heart failure
            - Caution advised if low eGFR and/or baseline hyperkalaemia
        - Alpha blockers likely preferable if prostatic disease

- Likely preferred agents in each of blood pressure lowering medications available in Aotearoa NZ:

- ACEi and ARB:
    - Ramipril typically preferred ACEi but perindopril useful alternative. Quinapril also useful alternative if combination with TD desirable but often requires twice daily dosing.
        - Usual dose range of ramipril is 2.5 mg – 10 mg daily but may start 1.25 mg daily if elderly and/or risk of hypotension
        - Usual dose range of perindopril is 2 mg - 8 mg daily
        - Usual dose range of quinapril is 2.5 mg – 20 mg once or twice daily. Combination with hydrochlorothiazide available at 10 mg and 20 mg doses of quinapril.
    - Candesartan typically preferred ARB but losartan useful alternative if combination with TD desirable.
        - Usual dose range of candesartan is 8 mg – 32 mg daily but may start at 4 mg daily if elderly and/or risk of hypotension
        - Usual dose range of losartan is 50 mg – 100 mg daily but may start at 12.5 mg - 25 mg daily if elderly and/or risk of hypotension. Combination with hydrochlorothiazide available at 50 mg losartan dose.
    - Check creatinine + electrolytes 2-4 weeks after starting or dose change to ensure no hyperkalaemia or deterioration of renal function
        - K+ < 5.5 mmol/L and up to 30% decrease in creatinine requires no dose change
        - Hyperkalaemia is often spurious due to haemolysis during the delay from collection until analysis in the lab - consider repeating to ensure real.
- CCB:
    - Amlodipine and felodipine preferred with usual dose range for both 2.5 mg – 5 mg daily
    - 10 mg daily doses associated with greater risk of adverse effects for often little benefit
    - Useful if elevated diastolic BP as vasodilators
- TD:
    - Chlorthalidone is typically preferred TD but bendroflumethazide useful alternative.
    - Usual dose of chlorthalidone: is 12.5 mg – 25 mg daily and usual dose of bendroflumethazide is 2.5 mg - 5 mg daily. Higher doses typically lead to little additional BP lowering but greatly increase adverse effects.

        - Hydrochlorothiazide is a weak BP lowering agent but may be useful if combination tablets preferable
        - Check eGFR + electrolytes 1-2 weeks after starting to ensure no adverse effects
    - β-blockers – Metoprolol controlled release (CR) or bisoprolol preferred for ischaemic heart disease and rate control
        - Carvedilol likely preferable if congestive heart failure

- ***The guidance in this section is predominantly based on the 2024 European Society of Cardiology Guidelines for the management of elevated blood pressure and hypertension, which can be accessed at www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Elevated-Blood-Pressure-and-Hypertension***

Treatment approach to elevated BP and hypertension

Treatment approach to elevated BP and hypertension

- All with a confirmed BP ≥ 130/80 mmHg should have a CKM risk assessment and causes of secondary hypertension considered particularly if young without risk factors or family history of hypertension. Interventions to optimise healthy living should also be offered to all with specific advice including:
    - Smoking and alcohol cessation. Complete cessation of vaping also likely beneficial
        - There is no safe alcohol limit for cardiovascular health
    - Low-salt intake (< 2 g of sodium or < 5 g of salt per day) and ideally 5-7 serves of vegetables and 2 fruit per day to ensure adequate potassium intake
        - Avoiding processed foods and adding salt to food important
        - Mediterranean diet and Dietary Approaches to Stop Hypertension (DASH) are eating patterns with best long-term evidence for reducing BP and CV risk.
            - These eating patterns emphasise fruits, vegetables, whole grains, low-fat dairy and lean proteins.
    - Avoiding medications that increase BP e.g. NSAIDs.
    - Increased physical activity and movement
    - Interventions for weight loss if overweight or obese aiming for > 5% total body weight loss
- BP lowering therapy is recommended in addition to healthy living interventions:
    - Immediately if BP ≥ 160/100 mmHg

    - If BP persistently ≥ 140/90 mmHg
    - If BP persistently 130 – 139/80 – 89 mmHg and ANY of the following:
        - 5 year CV risk ≥ 10%
        - CV disease including asymptomatic coronary or carotid disease (includes CT calcium score > 300)
        - CKD (UACR > 3 mg/mmol and/or eGFR < 60 mL/min/1.73m^2)
        - Diabetes with microvascular and/or macrovascular complications
        - LDLc ≥ 4.9 mmol/L and/or known familial hypercholesterolaemia

- BP lowering therapy should still be strongly considered if BP persistently 130 – 139/80 – 89 mmHg and 5 year CV risk 5 – 9.9% → particularly if additional risk factors for CVD
    - Additional risk factors for CVD include:
        - < 50 years of age
        - Direct family history of CV disease at < 50 years of age
        - Cardiac calcium score 100 - 300
        - Previous gestational diabetes and/or preeclampsia
        - MASLD

- BP lowering therapy is typically not required if BP 130 – 139/80 – 89 mmHg and 5 year CV risk < 5%, but healthy living interventions remain important.

Treatment targets

- A systolic BP < 130 mmHg is the treatment target for most
    - **NB:** A systolic BP < 120 mmHg should be considered if hypertension-induced organ effects including left ventricular hypertrophy, aortic root dilatation and/or CKD.

- **Relaxed BP targets to the lowest reasonably and safely achievable BP are appropriate if any of the following:**
    - Frailty and/or limited life expectancy
    - Age ≥ 85 years
    - Symptomatic postural hypotension
        - Treat to standing BP if > 10 mmHg postural drop.

- Intolerant of BP lowering medications

Secondary hypertension

Secondary hypertension

- Most people with hypertension have primary or essential hypertension where there is no identifiable cause for their high blood pressure. However, 5 - 10% of adults with hypertension have secondary hypertension where there is an underlying and potentially correctable cause of their high blood pressure.
- Secondary hypertension should be considered at the outset of confirmed hypertension, particularly if young without risk factors or a family history of hypertension. Secondary hypertension should also be considered if persistent hypertension despite adherence to ≥ 3 blood pressure lowering therapies
- Causes of secondary hypertension include:
  - Obstructive sleep apnoea
  - Renovascular disease and renal parenchymal disease
  - Medications e.g. oral contraceptive pill, NSAIDs, steroids, cyclosporin etc.
  - Recreational drug use e.g. amphetamine, cocaine, excess alcohol intake
  - Endocrine disorders e.g. primary hyperaldosteronism, Cushing’s disease, phaeochromocytoma, thyroid disease and acromegaly
  - Pregnancy
  - Coarctation of the aorta
- History and examination often provide guide to which investigations should be performed to confirm or exclude causes of secondary hypertension.

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* Primary hyperaldosteronism is a common cause of secondary hypertension in adults and the aldosterone:renin ratio is best performed before starting BP lowering medications.

Choice of BP lowering therapy

Choice of blood pressure lowering therapy

- First line BP lowering medications are ACE inhibitors (ACEi) or angiotensin receptor blockers (ARB), calcium channel blockers (CCB) and thiazide diuretics (TD).
  - **Typically these medications are more effective and better tolerated in low doses in combination**.
  - Cardioselective beta blockers (β-blocker) may be used if ischaemic heart disease, heart failure or atrial fibrillation.

- Best choice of ACEi, ARB, CCB or TD is dependent on whether albuminuria (UACR > 3 mg/mmol) or other comorbidities are present:
    - Albuminuria (UACR > 3 mg/mmol):
        - Start ACEi or ARB if no hypotension and titrate to maximal tolerated dose
        - If BP remains above target add CCB or TD
        - If BP still above target then add other (e.g. TD if CCB previously added)
    - No albuminuria present (UACR < 3 mg/mmol):
        - Strongly consider low dose ACEi or ARB and CCB in combination
        - If BP above target increase dose of combination agents
        - If BP remains above target then add TD
    - Gout:
        - Losartan and CCBs may be helpful due to their small uricosuric effect, but they should not replace urate-lowering therapy and the focus should be on the best BP-lowering agent
        - Beware TD typically increase uric acid levels
        - Serum urate will rise with heart, kidney and respiratory failure
    - Ischaemic heart disease, heart failure or atrial fibrillation:
        - Cardioselective β-blockers may be added at any stage. May need to decrease CCB and/or TD if risk of hypotension
        - Entresto (Sacubitril/Valsartan) is the preferred neprilysin/renin-angiotensin system inhibitor in heart failure rather than other ARB or ACEi, but requires special authority funding.
            - Current special authority criteria for Entresto requires the following:
                - NHYA/WHO functional II-IV AND
                - LVEF ≤ 35% OR echocardiogram is not reasonably practical AND
                - Other standard treatments of heart failure are optimised
        - Empagliflozin and spironolactone or eplerenone are also preferred in heart failure rather than TD or CCB if risk of hypotension.
- When starting BP lowering therapy ensure up date sick day management advice and contraception if applicable e.g. for ACEi/ARB

- Check serum creatinine 1-2 weeks after a dose change for either ACEi/ARB or TD to ensure no significant derangement
    - If K^+ < 6 mmol/L and < 30% decrease in eGFR then no change required
    - If K^+ ≥ 6 mmol/L **urgently review**
        - Exclude spurious hyperkalaemia due to dietary intake, haemolysis and/or delayed processing, or medication effect e.g. trimethoprim.
        - K^+ ≥ 6.5 mmol/L is a potential medical emergency
        - If K^+ is 6 - 6.5 mmol/L and K^+ rise is < 30% aim to reduce K^+ by:
            - Decreasing other K^+ increasing medications, especially NSAIDs, trimethoprim and β-blockers if appropriate
                - β-blockers have greater K+ retention effects than RAS inhibition but should not generally be stopped abruptly
            - Reducing dietary potassium intake
            - Consider frusemide if volume overload or refractory hypertension
            - Consider oral sodium bicarbonate if metabolic acidosis
            - If K^+ is 6 - 6.5 mmol/L but K^+ rise is ≥ 30% withhold ACEi or ARB and other K^+ elevating medications
            - Recheck K^+ in 1-2 days and reintroduce ACEi or ARB as soon as K^+ normalises.
            - Titrate ACEi or ARB to maximal dose based on K^+ levels
    - If > 30% decrease in eGFR withhold ACEi or ARB and review:
        - Assess for other causes of acute kidney injury particularly medications e.g. diuretics, NSAIDs
        - Correct volume depletion
        - Recheck eGFR and ensure person is well hydrated before the test. Restart ACEi or ARB if eGFR close to baseline
        - If appears ACEi or ARB-induced then discuss with renal team whether restart ACEi/ARB and consider renal artery stenosis
- Consider causes of secondary hypertension if BP still above target despite adherence to ≥ 3 BP lowering therapies

Recommended agents in each class of BP lowering therapy

Choice of agent in each of BP lowering medications

The likely preferred agents in each of blood pressure lowering medications based on efficacy, tolerance and availability in Aotearoa NZ are:

- ACEi and ARB:
    - Ramipril typically preferred ACEi but perindopril useful alternative. Quinapril also useful alternative if combination with TD desirable but often requires twice daily dosing.
        - Usual dose range of ramipril is 2.5 mg – 10 mg daily but may start 1.25 mg daily if elderly and/or risk of hypotension
        - Usual dose range of perindopril is 2 mg - 8 mg daily
        - Usual dose range of quinapril is 2.5 mg – 20 mg once or twice daily.
        - Combination with hydrochlorothiazide available at 10 mg and 20 mg doses of quinapril.
    - Candesartan typically preferred ARB
        - Usual dose range of candesartan is 8 mg – 32 mg daily but may start at 4 mg daily if elderly and/or risk of hypotension
        - Usual dose range of losartan is 50 mg – 100 mg daily but may start at 12.5 mg - 25 mg daily if elderly and/or risk of hypotension.
        - Combination with hydrochlorothiazide available at 16 and 32 mg doses of candesartan and 50 mg dose of losartan
- CCB:
    - Amlodipine and felodipine preferred with usual dose range for both 2.5 mg – 5 mg daily
    - 10 mg daily doses are associated with greater risk of adverse effects for often little additional benefit
    - Useful if elevated diastolic BP due to vasodilatory effect
- TD:
    - Usual dose of chlorthalidone: is 12.5 mg – 25 mg daily and usual dose of bendroflumethazide is 2.5 mg - 5 mg daily. Higher doses typically lead to little additional BP lowering but greatly increase adverse effects.
    - Chlorthalidone has a significantly longer half-life (lasting up to 72 hours) compared to other TDs. This may be helpful and more efficacious, but the prolonged duration of action also means a sustained effect on renal sodium and water excretion, and should be balanced against the increased likelihood of cumulative electrolyte disturbances over time.
    - Hydrochlorothiazide is a weak BP lowering agent but may be useful if combination tablets preferable
- β-blockers:

- Metoprolol controlled release (CR) or bisoprolol preferred for ischaemic heart disease and rate control
- Carvedilol likely preferable if congestive heart failure

Other key practice points in managing elevated BP and hypertension

Other key practice points in managing elevated BP and hypertension

- Management of high blood pressure should not occur in isolation. Ensure up to date CKM risk assessment with smoking cessation and management of other CKM conditions as required.
- Measure BP at least 6 – 12 monthly once treatment optimised to ensure targets are still met
- Consider urgent referral if BP ≥ 180/110 mmHg AND pregnant OR signs of malignant hypertension
  - Signs of malignant hypertension include:
    - Severe headache
    - Altered vision
    - Papillioedema, retinal haemorrhages and exudates
    - Altered mental state
    - Acute kidney injury with proteinuria and haematuria
- Consider non-urgent referral if BP above target despite adherence to ≥ 3 BP lowering therapies and/or any concerns over potential secondary hypertension.

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7. Management of dyslipidaemia

Definition of dyslipidaemia and familial hypercholesterolaemia

Definition of dyslipidaemia and familial hypercholesterolaemia

- Dyslipidaemia is defined as abnormal levels of circulating lipids, primarily increased LDL cholesterol (LDLc), increased triglycerides (TG) and/or low HDL cholesterol. At present, LDLc is the major treatment target for reducing cardiovascular disease.
- Most cases of significantly raised LDLc (≥ 5 mmol/L) are due to polygenic risk or *secondary causes*. However, **familial hypercholesterolaemia (FH) should be considered if LDLc ≥ 5 mmol/L and no secondary cause evident**, particularly if LDLc > 6.5 mmol/L, xanthomas, or family history of significantly raised LDLc and/or early CVD. If FH suspected, calculate the Dutch Lipid Clinic Network (DLCN) Score. DLCN scores ≥ 6 should be discussed with lipid specialist to proceed with gene testing for FH.

- Secondary causes of high LDLc include:
  - Ketogenic diet
  - Nephrotic syndrome
  - Chronic kidney disease
  - Cholestatic liver disease
  - Type 2 diabetes
  - Hypothyroidism
  - Obesity
  - Anorexia nervosa
  - Post menopause
  - Pregnancy
  - Medications including corticosteroids, antipsychotics, retinoids, protease inhibitors etc.

- Specialist consultation is recommended if FH is confirmed for genetic counselling and cascade testing, particularly if FH is suspected or confirmed in children.
  - FH should be considered in children if LDLc > 3.5 mmol/L
  - FH should be aggressively managed and lipid lowering therapy is recommended from 8 – 10 years of age.

Treatment approach to dyslipidaemia

Treatment approach to dyslipidaemia

- All with dyslipidaemia should have a CKM risk assessment and interventions to optimise healthy living. Specific advice for dyslipidaemia includes:
  - Avoid trans fats and reduce saturated fats and oils (e.g. coconut, lard, butter).
    - Limit processed and deep-fried foods.
    - Unsaturated fats and oils (e.g. canola, rice bran, avocado, olive, plant-based margarines) may be useful alternatives.
  - Increase intake of fruits, vegetables and wholegrain foods which bind excess cholesterol e.g. oats, barley, quinoa, ground flaxseed and chia seeds
  - Specific dietary interventions to reduce TG levels include:

        - Reduced free sugar intake e.g. avoiding sugary drinks and lollies etc.
        - Reduced alcohol intake
        - Eating foods and/or supplements containing omega-3 fats regularly
    - Physical activity and movement can increase HDLc and reduce LDLc and TG
    - Interventions for weight loss if overweight or obese aiming for > 5% total body weight loss

- Lipid lowering therapy is strongly recommended in addition to healthy living interventions aiming for LDLc < 1.4 mmol/L and > 50% reduction in LDLc from baseline if ANY of the below:
    - LDLc ≥ 4.9 mmol/L and/or known familial hypercholesterolaemia
    - CV disease including asymptomatic coronary or carotid disease (includes CT calcium score > 300)
    - Diabetes with any microvascular or macrovascular complication(s)
    - UACR ≥ 30 mg/mmol
    - eGFR < 45 mL/min
    - UACR 3 – 29 mg/mmol AND eGFR 45 – 59 mL/min
    - 5 year CV risk ≥ 10%

- Lipid lowering therapy is recommended in addition to healthy living interventions if 5 year CV risk 5 – 9.9% aiming for target LDLc < 1.8 mmol/L and > 50% reduction in LDLc from baseline.
- Lipid lowering therapy should also be considered if 5 year CV risk 3 – 4.9% and additional risk factors for CVD. Target LDLc is < 1.8 mmol/L.
    - Risk factors to consider lipid lowering therapy include:
        - < 50 years of age
        - Direct family history of CV disease at < 50 years of age
        - Cardiac calcium score 100 - 300
        - Previous gestational diabetes and/or preeclampsia
        - MASLD

Choice of lipid lowering therapy

Choice of lipid lowering therapy

- First line lipid lowering medications are atorvastatin and rosuvastatin.

    - Rosuvastatin is typically preferred if previous CV event but mismatch with special authority criteria
        - Current special authority requires previous failures of atorvastatin or simvastatin to reach LDLc target unless Māori or Pacific ethnicity
        - Maximise rosuvastatin use in Māori and Pacific peoples given funded and their high CV risk
        - Other ethnicities may choose to self-fund rosuvastatin without trialling atorvastatin as cost is currently around $12 per week

- Usual dose range is atorvastatin is 10 – 80 mg daily and rosuvastatin 5 – 40 mg daily
    - Consider starting rosuvastatin 5 mg daily if South East Asian ancestry due to a common genetic polymorphism that increases rosuvastatin levels
    - Maximum dose of rosuvastatin is 10 mg daily if eGFR < 30 mL/min
    - At least 40 mg atorvastatin or 10 mg rosuvastatin daily is typically required to achieve a 50% reduction in LDLc

- Statins have low and potentially no teratogenicity - so statins can be used in women of child-bearing age without fear.
    - Current advice is to stop statins in pregnancy and breastfeeding, but discuss with secondary care if known cardiovascular disease and/or familial hypercholesterolaemia as it may be safest to continue statin.

- Measure non-fasting LDLc at least 3 monthly and titrate statin to reach LDLc target or maximal tolerated dose
    - 90% of LDLc lowering evident within 2 weeks so do not need to wait 3 months before titrating
    - Repeat testing may be barrier to optimising treatment so consider starting statin at or rapidly titrating statin to estimated dose to reach LDLc target

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- Switch atorvastatin to rosuvastatin if LDLc is above target on 80 mg daily or maximal tolerated dose
    - Consider trialling pravastatin if intolerant of low dose atorvastatin and rosuvastatin

- Consider ezetimibe 10 mg daily if LDLc above target despite maximal tolerated dose of statin
    - Ezetimibe no longer requires special authority approval

- Consider evolocumab (Repatha) or inclisiran if LDLc still above target but require SC injection (2-4 weekly or 6 monthly), are not funded and are expensive (particularly evolocumab). They will reduce LDL on average by a further 50% from baseline.

- Measure non-fasting LDLc 6 - 12 monthly once treatment optimised to ensure targets are still met.

Adverse effects of statins

Adverse effects of statins

Statins are typically well tolerated with most adverse effects only minor:

- Many reported adverse effects of statins may be due to the nocebo effect, particularly fatigue, muscle aches and memory loss, particularly as statins protect against and do not cause dementia.
- Serious adverse effects such as rhabdomyolysis and hepatotoxicity are extremely rare with statins other than simvastatin. Doses of simvastatin > 40 mg daily are no longer recommended.
- Only 50% of reported mild adverse effects recur on re-trialling the same statin or a different statin. Mild transient elevation of liver enzymes is common and does not require cessation. Exclude other causes.
- Statin-induced myopathy occurs in < 5% of people and predominantly affects the shoulder and hip girdle rather than generalised muscle pain.
- The benefits of statins almost always outweigh the rare deleterious effects on hyperglycaemia in people with prediabetes or diabetes and should not prevent treatment.

Treatment of persistent hypertriglyceridaemia

Treatment of persistent hypertriglyceridaemia

- Hypertriglyceridaemia is independently associated with increased CV risk but fibrates do not reduce CV events or total mortality in people treated with statins.
- Statins continue to be first line agents to reduce CV risk in hypertriglyceridaemia and healthy living interventions remain important aiming for a TG level < 1.7 mmol/L in secondary prevention and < 5 mmol/L in primary prevention.
    - Avoid trans fats and reduce saturated fats and oils (e.g. coconut, lard, butter).
        - Limit processed and deep-fried foods
        - Unsaturated fats and oils (e.g. canola, rice bran, avocado, olive, plant-based margarines) may be useful alternatives.
    - Increase intake of fruits, vegetables and wholegrain foods which bind excess cholesterol e.g. oats, barley, quinoa, ground flaxseed and chia seeds
    - Specific dietary interventions to reduce TG levels include:
        - Reduced free sugar intake e.g. avoiding sugary drinks and lollies etc.
        - Reduced alcohol intake
        - Eating foods and/or supplements containing omega-3 fats regularly
    - Physical activity and movement can increase HDLc and reduce LDLc and TG

- Interventions for weight loss if overweight or obese aiming for > 5% total body weight loss

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8. Management of hyperglycaemia in type 2 diabetes

Overview and diagnosis of type 2 diabetes

Management of hyperglycaemia in type 2 diabetes

- This section provides a summary of recent changes in the management of type 2 diabetes, focusing on management relevant to CKM conditions only. Detailed guidance on the screening for diabetes, differentiating between the types of diabetes, and management of type 2 diabetes and its complications can be found here.
- Confirming the correct type of diabetes remains important for whānau with diabetes to access and receive best care.

Diagnosis of type 2 diabetes

- The diagnostic criteria for diabetes and prediabetes in Aotearoa New Zealand now aligns with the rest of the world:
    - Diabetes
        - HbA1c ≥ 48 mmol/mol OR
        - Fasting glucose ≥ 7 mmol/L OR
        - 2 hour glucose > 11 mmol/L on a 75 g glucose tolerance test OR
        - Random blood glucose > 11 mmol/L if symptoms of diabetes
    - Prediabetes
        - HbA1c 42 – 47 mmol/mol OR
        - Fasting glucose 6.1 – 6.9 mmol/L OR
        - 2 hour glucose 7.8 – 11 mmol/L on a 75 g glucose tolerance test
- The diagnosis of diabetes still requires two confirmatory tests unless the initial HbA1c is > 53 mmol/mol. If required, the 2nd confirmatory test should be done as soon as possible.

Treatment targets in type 2 diabetes

Treatment targets in type 2 diabetes

- HbA1c is the most common glycaemic treatment target in type 2 diabetes because continuous glucose monitoring is currently not funded for type 2 diabetes in Aotearoa
    - Target HbA1c for most is < 53 mmol/mol
    - Target HbA1c < 48 mmol/mol preferred in young adults and pre-pregnancy
    - Targets should always be balanced against risk of hypoglycaemia → **only insulin and sulphonylureas can cause significant hypoglycaemia**
    - **Target HbA1c 55 – 70 mmol/mol may be suitable if high risk of hypoglycaemia or tight glycaemia is not required e.g. life expectancy limited by other conditions**

- **Continuous glucose monitoring (CGM) can also be useful in type 2 diabetes, especially in high-risk groups, but is not currently funded.**

- High-risk groups with type 2 diabetes who will likely benefit from CGM:
    - Treatment with insulin and/or sulfonylureas
    - Youth and young adults
    - Pregnancy
    - On dialysis
    - Physical and/or cognitive impairment that prevents monitoring blood glucose levels

- Tips to increase access to CGM in type 2 diabetes include:
    - Discuss self-funding intermittent use of CGM
    - Using the disability allowance to fund CGM if able
    - Utilise free trials of CGM

- Important CGM targets include:
    - Time in range (TIR; % glucose levels 3.9 – 10 mmol/L) > 70%
        - Target TIR is > 80% if target HbA1c < 48 mmol/mol
    - Time below range (TBR; % glucose levels < 3.9 mmol/L) < 4% → only relevant if on insulin or sulfonylureas
    - Glucose management indicator (GMI) < 53 mmol/mol
        - Target GMI is < 48 mmol/mol if target HbA1c < 48 mmol/mol

Best choice of glucose lowering therapies

Best choice of glucose lowering therapies in type 2 diabetes

Management of type 2 diabetes is now focused on preventing, delaying and reducing the progression of CV and renal disease and aiding weight loss if appropriate rather than just lowering glucose levels. Clinical inertia by health care professionals is still the greatest barrier to people with diabetes reaching their treatment targets. Comprehensive guidance on all aspects of the management of type 2 diabetes and how to reduce treatment inertia can be found here. **Key concepts in best choice of glucose lowering therapies includes:**

- Healthy living interventions are first line management and are important at all stages of type 2 diabetes → they are discussed in detail here
    - 10-15% total body weight loss (TBWL) is typically required to achieve remission of type 2 diabetes if increased adiposity, but even 5% TWBL will significantly improve glucose levels
- Metformin should be started at diagnosis if eGFR > 15 mL/min regardless of HbA1c
    - Metformin is often best tolerated starting at 250 – 500 mg with largest meal
    - Titrate metformin to 1 g twice daily or maximal tolerated dose
    - Metformin in combination tablets (e.g. Jardiamet, Galvumet) seems to be better tolerated than metformin alone
    - Doses of metformin need to be reduced once eGFR < 45 mL/min
    - GFR 30 – 44 mL/min → maximum metformin dose is 1 g daily
    - eGFR – 15 – 29 mL/min → maximum metformin dose is 500 mg daily
    - eGFR < 15 mL/min → stop metformin
- **If chronic kidney disease (UACR > 3 mg/mmol OR eGFR < 60 mL/min), heart failure, CVD OR high CV risk (5 year CV risk ≥ 10%) add empagliflozin OR a GLP1 receptor agonist (GLP1Ra) regardless of HbA1c**
    - Empagliflozin is typically preferred if heart failure or CKD predominate
        - Start 10 mg daily alone or in combination with metformin
        - Can increase to 25 mg daily if HbA1c remains above target
        - Glucose-lowering effects of empagliflozin reduce once eGFR < 30 mL/min but CV and renal protection persist
        - Empagliflozin can be started if eGFR > 20 mL/min and should only be stopped if adverse effects occur or dialysis is started.
        - Sick day advice and tips to reduce adverse effects should be provided
        - Withhold empagliflozin in acute illness and 3 days before (including day of) major surgery, bowel prep or low carb diet. Restart when well and eating and drinking normal.

        - Doses of sulfonylureas may need to be reduced by 50% and doses of insulin by approximately 20% to avoid hypoglycaemia when starting empagliflozin – typically only required if baseline HbA1c < 64 mmol/mol.
        - Discuss importance of genital hygiene and reporting changes or concerns
        - Do not use in pregnancy, breastfeeding or children < 10 years of age
        - Do not use in type 1 diabetes, significant alcohol intake, previous diabetic ketoacidosis (DKA) or low carbohydrate diets without specialist advice
        - If symptoms of DKA (e.g. nausea, vomiting, abdominal pain etc.) need to present to GP practice or A+E urgently to ensure blood ketones are < 1.5 mmol/L. DKA needs to be excluded if ketones > 1.5 mmol/L

    - GLP1Ra likely preferred if greater reduction in HbA1c and/or weight desired
        - Currently two funded injectable GLP1Ra for type 2 diabetes:
            - Dulaglutide 1.5 mg weekly → can increase to 3 mg and 4.5 mg weekly if HbA1c remains above target but beware potential for short supply
            - Liraglutide 0.6 mg daily → titrate to 1.8 mg daily or maximal tolerated dose
                - Need to prescribe BD fine 4 mm needles for injecting
        - Semaglutide is a more potent GLP1Ra and is well tolerated, but is not funded and costs approximately $480 – 500 per month
            - Start 0.25 mg weekly and increase the dose every 4 weeks to 0.5 mg weekly then 1 mg weekly then 1.7 mg weekly then 2.4 mg weekly or maximal tolerated dose
        - Discuss strategies on how to reduce adverse effects
            - Ensure adequate hydration and stop eating when feeling full
            - Eat smaller meals and avoid alcohol, fatty and spicy foods
            - Slow down dose increases if GI adverse effects
            - GI adverse effects typically dissipate within 2-3 weeks
            - Doses of sulfonylureas may need to be reduced by 50% and doses of insulin by approximately 20% to avoid hypoglycaemia when starting GLPRa – typically only required if baseline HbA1c < 64 mmol/mol
            - Do not use in pregnancy, breastfeeding or children < 10 years
                - GLP1Ra should be stopped once eGFR < 15 mL/min

- Dual empagliflozin/GLP1Ra therapy is typically preferred if HbA1c remains above target on either agent alone. **There is a mismatch between best practice and the special authority criteria**, which states the patient must have heart failure (empagliflozin) or an HbA1c > 53 mmol/mol (both empagliflozin and GLP1Ra) if no heart failure.
    - Dual therapy can only be fully funded with GLP1Ra under the diabetes special authority and empagliflozin under the heart failure special authority.
    - Self-funding of these agents should be offered but are expensive (approximately $85 per month for empagliflozin and minimum $250 per month for GLP1Ra). Tips to increase access include:
        - Ensuring empagliflozin is funded under the heart failure criteria if applicable
        - Funding the GLP1Ra under the diabetes special authority due to the much greater cost
        - Utilising the disability allowance to cover the cost of empagliflozin if able
        - Prescribing half a 25 mg tablet of empagliflozin or 1 tablet of empagliflozin 12.5 mg with metformin to halve the cost to approximately $43 per month – please note this is off-label.
        - Checking the cost between pharmacies because there continues to be wide variation
- Pioglitazone is likely the next best agent if HbA1c remains above target if no contraindications
    - History of bladder cancer
    - High risk of fractures – especially if known osteoporosis
    - Peripheral oedema e.g. uncontrolled heart failure
    - Pioglitazone now appears safe in macular oedema but best to withhold if severe macular oedema undergoing treatment
    - Pregnancy or breastfeeding
    - Start 15 mg daily → it may take up to 16 weeks before the full effects on HbA1c are seen but can titrate up to 45 mg daily as required

- **If no renal or CV disease and 5 year CV risk < 10% then treatment is added (not switched) if HbA1c is above target:**
    - If weight loss desired → empagliflozin and/or GLP1Ra preferred. Consider acarbose if HbA1c still above target but beware of adverse GI effects.
    - If weight loss not desired → consider vildagliptin (typically weight neutral and redundant if on GLP1Ra) and pioglitazone (may cause minimal weight gain)
        - Usual dose of vildagliptin is 50 mg twice daily either alone or in combination with metformin.
        - Maximum dose is 50 mg daily once eGFR < 50 mL/min

- **If HbA1c remains above target then consider sulfonylureas and insulin but beware risks of hypoglycaemia and weight gain**. These risks can be reduced by:
    - Using sulfonylureas first as the risks with sulfonylureas are minimal
    - Reinforcing healthy eating and dietitian input
    - Regular monitoring of glucose levels and consider CGM
    - Maximising other glucose lowering therapies so only lowest doses of sulfonylureas and insulin are required.
    - Ensuring all patients have an up to date sick day management plan
    - Reducing doses of sulfonylureas and insulin with declining renal function
    - Adding in prandial insulin rather than increasing basal insulin if the HbA1c is above target once doses of basal insulin reach 0.5 units/kg per day
    - Reducing doses of sulfonylureas by ≥ 50% and insulin by ≥ 20% if episodes of hypoglycaemia are occurring

Other important practice points for managing type 2 diabetes

Other important practice points in managing type 2 diabetes

- Intervening early in type 2 diabetes is important to increase chances of remission, slow progression of diabetes and prevent long term complications
    - Chances of remission of type 2 diabetes greatly decrease > 6 years post diagnosis
    - Diabetes continues to be the most common cause of visual loss, amputation, dialysis and renal transplant in Aotearoa New Zealand despite end-stage complications being largely preventable.
- Addressing inequities are critical in type 2 diabetes
    - Prevalence of type 2 diabetes is 1.4 to 3 times higher in Māori, Pacific peoples, and people from Indo-Asia.
    - Complications of diabetes are up to 3 to 5 times higher in these populations than Pākehā with diabetes.
    - Consider cultural supports early to address any barriers to diagnosis and management.
    - Aim for whānau-based care if possible and integrate care with Rongoā Māori practitioners if whānau wish
- Ideally HbA1c should be measured 3 monthly if above target with escalation of therapy as required. Once to target, HbA1c should ideally be measured 6 monthly as type 2 diabetes is a progressive disease.
- Smoking cessation and optimising management of blood pressure and dyslipidaemia are likely at least as important as optimising glucose levels in preventing, delaying and slowing complications of diabetes.

- The annual diabetes review continues to be important opportunity to optimise care including acting on results from the following:
    - Consideration whether treatment targets are appropriate if change in clinical circumstances
    - Performing a neurovascular examination of the feet and ensuring foot cares if increased risk
    - Ensuring retinal photoscreening is being performed at least 2-3 yearly
    - Screening for depression → the PHQ-2 score can be a useful screening tool
        - Scores ≥ 3 should prompt further screening with PHQ-9 or other tools
    - Screening for diabetes distress → the DDS2 score is a useful screening tool
        - Scores ≥ 3 highlight need to fully evaluate diabetes distress and consider support as appropriate
    - Calculating CV risk using the CKM risk assessment and management (CKM-RAM) calculator
    - Ensuring vaccinations and screening for malignancy is up to date
    - Screening for dental and periodontal disease
    - Discussing contraception and planning pregnancy in women and men of child bearing age
        - Glycaemia to target is also important in potential fathers
    - **NB:** The annual diabetes review does not have to be performed all in one appointment and different components may be split across the year
- Development of either a microvascular diabetic complication (e.g. diabetic eye, kidney or foot disease) or macrovascular diabetic complication (e.g. coronary artery disease) requires management as per high risk CKM conditions and high CV risk to prevent progression of the complication(s). These treatment targets include:
    - Adding empagliflozin or GLP1Ra regardless of HbA1c
    - ACEi or ARB if CKD or heart failure and no concerns over hypotension
    - Systolic BP 120 – 129 mmHg or lowest reasonably safely achievable BP
    - LDLc < 1.4 mmol/L
    - Serum urate < 0.36 mmol/L if gout (< 0.3 mmol/L if tophi)
    - Aspirin if previous CV event
        - NB: The risks of aspirin appear to outweigh the benefits for primary prevention of CV events in people with diabetes
- Neuropathic pain is common in people with diabetes and can usually be successfully treated

- Mild pain → paracetamol
- Moderate to severe pain → low dose tricycylic e.g. nortriptyline 10 mg nocte
  - Can titrate nortriptyline and add pregabalin or gabapentin as required
  - Pregabalin is typically more effective than gabapentin with less adverse effects in diabetic neuropathy
  - Carbamazepine can be added in severe cases
- Topical capsaicin 0.075% may be useful for localised neuropathic pain
- Diabetic neuropathic pain is typically not responsive to NSAIDs or opiates
- Supportive footwear and early involvement of podiatry if foot disease is important

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9. Management of chronic kidney disease

Definition of chronic kidney disease and overview of management

Definition of chronic kidney disease (CKD)

- CKD is defined as renal impairment (eGFR <60mL/min) AND/OR persistent albuminuria (2 out of 3 UACR >3 mg/mmol) for more than 3 months.
  - Albuminuria indicates endothelial inflammation.
  - Renal impairment indicates renal damage or scarring.
  - Both are independent risk factors for CV disease irrespective of each other with and without diabetes.

Overview of management of CKD

- This guidance focusses on the management of albuminuria and renal impairment relevant to CKM conditions. Detailed guidance on the staging, investigation and management of chronic kidney disease (CKD) can be found here. This is important in identifying the cause of the CKD, which is critical in best management.
- All people diagnosed with CKD should have a CKM risk assessment to help guide management. Best management of CKD includes 7 key areas to reduce progression of CKD and adverse sequelae:
  1. Healthy living interventions
  2. Renin-Angiotensin system (RAS) inhibitors and blood pressure (BP) lowering therapy
  3. SGLT2 inhibitors

4. GLP1 receptor agonists
5. Mineralocorticoid receptor antagonists
6. Lipid lowering therapy and antiplatelet therapy
7. Other key practice points in managing CKD

NB: Starting this standard of care management as soon as possible in CKD is important because it achieves at least 7 more years of health free of significant kidney disease.

Healthy living interventions in chronic kidney disease

Healthy living interventions in CKD

- Healthy living interventions in CKD are similar to those for all CKM conditions , particularly in reducing hyperglycaemia and BP to slow progression of CKD. Important specific advice to CKD includes:
    - Low sodium intake (< 2 g of sodium or < 5 g of salt per day) especially if high BP.
        - Avoiding processed foods and adding salt to food important.
    - A low potassium diet should not be recommended routinely unless persistent hyperkalaemia (K^+ > 6 mmol/L).
    - Specialist renal dietitian advice is recommended if eGFR < 45 mL/min/1.73m^2 if ANY of the following:
        - Persistent hyperkalaemia despite addressing other causes e.g. medications, constipation etc.
        - Consideration of very low calorie or low carbohydrate diets
        - Very high protein intake can be harmful and may lead to kidney hyperfiltration and glomerular injury
        - Concerns over malnutrition or electrolyte disturbances

Renin - Angiotensin system inhibitors and BP lowering therapy in chronic kidney disease

Renin - Angiotensin system (RAS) inhibitors and blood pressure (BP) lowering therapy

- Treatment regimen with either an ACE inhibitor (ACEi) or angiotensin receptor blocker (ARB) is dependent on whether albuminuria is present (UACR > 3 mg/mmol). Calcium channel blockers (CCB) and thiazide diuretics (TD) may also be required if the BP is above target. The target BP for most with CKD is a systolic BP < 120 mmHg.
    - **Relaxed BP targets to the lowest reasonably and safely achievable BP are appropriate if any of the following:**

        - Frailty and/or limited life expectancy
        - Age ≥ 85 years
        - Symptomatic postural hypotension → treat to standing BP if > 10 mmHg postural drop.
        - Intolerant of BP lowering medications

    - **Albuminuria present**:
        - Start ACEi OR ARB if no concerns over hypotension → titrate to maximal tolerated dose
            - Add a CCB or TD if BP > target
            - If BP still above target add other e.g. TD if CCB used previously

    - **No albuminuria but BP above target**
        - Start low dose ACEi OR ARB with CCB in combination → if BP above target increase dose of combination agents
        - If BP remains above target then add TD

- When starting or changing the dose of ACEi or ARB it is important to:
    - Ensure up to date sick day management advice and contraception if applicable
    - Measure creatinine and serum potassium 1-2 weeks after dose change
        - K^+ < 6 mmol/L and < 30% decrease in eGFR requires no change
        - If K^+ ≥ 6 mmol/L urgently review
            - Exclude spurious hyperkalaemia due to dietary intake, haemolysis and/or delayed processing, or medication effect e.g. trimethoprim.
            - K^+ ≥ 6.5 mmol/L is a potential medical emergency
            - If K^+ is 6 - 6.5 mmol/L and K^+ rise is < 30% aim to reduce K^+ by:
                - Decreasing other K^+ increasing medications, especially NSAIDs, trimethoprim and β-blockers if appropriate
                    - β-blockers have greater K+ retention effects than RAS inhibition but should not generally be stopped abruptly

        - Reducing dietary potassium intake
        - Consider frusemide if volume overload or refractory hypertension
        - Consider oral sodium bicarbonate if metabolic acidosis

    - If K^+ is 6 - 6.5 mmol/L but K^+ rise is ≥ 30% withhold ACEi or ARB and other K^+ elevating medications
        - Recheck K^+ in 1-2 days and reintroduce ACEi or ARB as soon as K^+ normalises
        - Titrate ACEi or ARB to maximal dose based on K^+ levels

    - If > 30% decrease in eGFR withhold ACEi or ARB and review
        - Assess for other causes of acute kidney injury particularly medications e.g. diuretics, NSAIDs
        - Correct volume depletion
        - Recheck eGFR and ensure person is well hydrated before the test. Restart ACEi or ARB if eGFR close to baseline
        - If appears ACEi or ARB-induced then discuss with renal team whether restart ACEi/ARB and consider renal artery stenosis

NB: ACEi, ARB, CCB, and TD are discussed in detail in management of elevated blood pressure and hypertension

SGLT2 inhibitors in chronic kidney disease

SGLT2 inhibitors in chronic kidney disease

- Start SGLT2 inhibitor, e.g. empagliflozin 10 mg daily if ANY of the below AND eGFR > 20 mL/min:
    - Type 2 diabetes with UACR > 3 mg/mmol
    - Heart failure
    - UACR > 20 mg/mmol
    - eGFR 20 – 44 mL/min at any level of albuminuria
    - **NB:** Empagliflozin is only funded at present under special authority if type 2 diabetes and/or reduced ejection heart failure is present. Although currently expensive at ~$85 per month, self-funding should be offered including tips to increase access
        - Utilising the disability allowance to cover the cost of empagliflozin if able

        - Prescribing half the 25 mg tablet of empagliflozin or 1 tablet of empagliflozin 12.5 mg with metformin (Jardiamet) if type 2 diabetes to halve the cost to approximately $43 per month – please note this is off-label.
        - Checking the cost between pharmacies because there continues to be wide variation

- The dose of empagliflozin can be increased to 25 mg daily if type 2 diabetes AND the HbA1c remains above target
    - Beware glucose-lowering effects of empagliflozin are eGFR < 30 mL/min
- Empagliflozin should only be stopped if adverse effects occur or dialysis is started.
    - A transient decrease in eGFR is normal when starting empagliflozin
- Sick day advice AND tips to reduce adverse effects should be provided for all on empagliflozin:
    - Withhold empagliflozin in acute illness and 3 days before (including day of) major surgery, bowel prep or low carb diet. Restart when well and eating and drinking normal.
    - Doses of sulfonylureas may need to be reduced by 50% and doses of insulin by approximately 20% to avoid hypoglycaemia when starting empagliflozin → typically only required when baseline HbA1c < 64 mmol/mol.
    - Discuss importance of genital hygiene particularly for individuals who may have difficulty accessing the genital area (e.g., due to body habitus or limited mobility). Warn people to stop if there is any hint of pain or redness.
    - Do not use in pregnancy, breastfeeding or children < 10 years of age
    - Do not use in type 1 diabetes, significant alcohol intake, previous diabetic ketoacidosis (DKA) or low carbohydrate diets without specialist advice
    - If symptoms of DKA (e.g. nausea, vomiting, abdominal pain etc.) need to present to GP practice or A+E urgently to ensure blood ketones are < 1.5 mmol/L. DKA needs to be excluded if ketones > 1.5 mmol/L.
    - Beware glucose levels may be normal or only mildly elevated in DKA with empagliflozin

GLP1 receptor agonists in chronic kidney disease

GLP1 receptor agonists (GLP1Ra) in chronic kidney disease

- GLP1Ra should be considered in CKD if weight loss is desirable (i.e. if overweight or obese) and/or if type 2 diabetes and the HbA1c is above target despite metformin and empagliflozin. GLP1Ra are not currently recommended if the eGFR is < 15 mL/min/1.73m^2

    - Please click here for more information on using GLP1Ra in type 2 diabetes (dulaglutide and liraglutide currently funded but would need to self-fund empagliflozin)
    - Please click here for more information on using GLP1Ra for weight loss (no GLP1Ra are currently funded without diabetes)

- Post hoc analyses suggest that semaglutide may reduce the progression of kidney and cardiovascular disease in those with a BMI < 27 kg/m^2 and without diabetes. Further dedicated studies are underway to confirm safety and efficacy in this population. At present semaglutide (Wegovy) is only registered for those with a BMI > 27 kg/m^2 in Aotearoa New Zealand.

Mineralocorticoid receptor antagonists in chronic kidney disease

Mineralocorticoid receptor antagonists (MRAs)

- Non-steroidal MRAs such as finerenone are a pillar of management CKD internationally, but are currently not available in Aotearoa New Zealand.
- Older MRAs such as spironolactone or eplerenone may still be used to treat concomitant heart failure or refractory hypertension despite ACEi or ARB, CCB and TD. However, the use of spironolactone or eplerenone in CKD should only be with specialist input due to the risk of hyperkalaemia.

Lipid lowering therapy and antiplatelet therapy in chronic kidney disease

Lipid lowering therapy and antiplatelet therapy in CKD

- Start lipid lowering therapy if eGFR > 15 mL/min aiming for LDLc < 1.4 mmol/L and > 50% reduction in LDLc from baseline if ANY of the below:
    - UACR ≥ 30 mg/mmol
    - eGFR < 45 mL/min
    - UACR 3 – 29 mg/mmol AND eGFR 45 – 59 mL/min
    - LDLc ≥ 4.9 mmol/L and/or known familial hypercholesterolaemia
    - CV disease including asymptomatic coronary or carotid disease (includes CT calcium score > 300)
    - Diabetes with any microvascular or macrovascular complication(s) → includes diabetic kidney disease
    - 5 year CV risk ≥ 10%
- Lipid lowering therapy is also recommended if none of the above and 5 year CV risk 5 – 9.9% aiming for target LDLc < 1.8 mmol/L and > 50% reduction in LDLc from baseline.
- Lipid lowering therapy should be considered if none of the above and 5 year CV risk 3 – 4.9% and additional risk factors for CVD. Target LDLc is < 1.8 mmol/L.
    - Risk factors to consider lipid lowering therapy include:

        - < 50 years of age
        - Direct family history of CV disease at < 50 years of age
        - Cardiac calcium score 100 - 300
        - Previous gestational diabetes and/or preeclampsia
        - MASLD

- Lipid lowering therapy is discussed in detail in management of dyslipidaemia
- Aspirin is important for secondary prevention of CV events in CKD. However, the balance of benefits and risks for aspirin for primary prevention in CKD is unknown.

Other key practice points in managing chronic kidney disease

Other key practice points in managing chronic kidney disease

- Addressing inequities are important in CKD
    - Incidence of CKD is 1.3 to 3 times higher in Māori, Pacific peoples, and people from Indo-Asia. Advanced CKD incidence is 3 to 5 times higher in these populations.
    - Consider cultural supports early to address any barriers to diagnosis and management.
    - Aim for whānau-based care if possible and integrate care with Rongoā Māori practitioners if whānau wish
- Ensuring adequate coding of CKD to enable best care
    - Coding of CKD is missing in 60 – 95% of cases internationally and is associated with:
        - Faster rates of CKD progression and reduced access to best care
        - Increased end stage kidney disease and major adverse CV events
- Optimising management of hyperglycaemia and gout in CKD is important to reduce the progression of CKD and CV risk
    - Healthy living interventions, metformin AND empagliflozin +/- GLP1Ra are best management of all people with CKD and T2D
    - Please click here for more information on these interventions and other treatment options if the HbA1c remains above target
- Medication adjustment is often required in CKD

    - Please click here for more information on medications that require dose adjustment or cessation in CKD

- Provide vaccinations as per the Immunisation Advisory Centre guidance in pre-dialysis, dialysis and pre- and post-kidney transplant
- People with CKD are at high risk of an acute kidney injury so it is important to:
    - Avoid nephrotoxic medications if possible e.g. NSAIDs
    - Ensure adequate hydration and sick day management plan
- Strongly consider renal advice if ANY of the following:
    - eGFR < 30 mL/min/1.73m^2
        - Formal review may not be required if eGFR stable, UACR < 30 mg/mmol or if the individual is frail.
    - eGFR 30 - 45 mL/min/1.73m^2 and UACR > 30 mg/mmol if known diabetes
        - Formal review may not be required if eGFR stable, UACR < 30 mg/mmol or if the individual is frail.
    - eGFR < 60 mL/min/1.73m^2 and declining by > 10mL/min/1.73m^2 in the last 12 months AND/OR UACR > 70 mg/mmol on ≥ 2 occasions as at high risk of progressive CKD.
    - If family history of, or known intrinsic kidney disease e.g. Polycystic Kidney Disease
    - **NB:** It may be prudent to recheck/confirm eGFR before referral if the decrease in eGFR may be an anomaly, ensuring adequate hydration before the repeat test.
- Depression is common in CKD and should be screened for and treated as required
    - The PHQ-2 score can be a useful screening tool
- People with advanced kidney disease (eGFR < 15 mL/min) usually have symptoms that significantly impact quality of life.
    - Treatment involves consideration of kidney replacement therapy (dialysis or kidney transplantation) and symptom management.
    - Consider advanced care plan (ACP) and palliative care input.
    - Examine feet and consider podiatry input as high risk of active foot disease

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10. Management of gout

Management of gout

Management of gout

- Gout is common in people with CKM conditions and is an independent risk factor for CV and renal disease. Effective treatment of gout is important in preventing gout flares and reducing CV and renal sequelae.
- Detailed guidance on managing gout can be found here including ensuring the correct diagnosis and management of acute gout flares. The guidance below is a brief synopsis of the management of gout relevant to CKM conditions:
    - Start urate lowering pharmacotherapy if any of the following:
        - Recurrent gout flares – especially > 1 flare per year
        - Gouty tophi or chronic gouty arthritis
        - Evidence of joint damage due to gout
        - Urate lowering pharmacotherapy should also be considered if ANY of the following:
            - ≥ 1 episode of gout with onset of gout at a young age or strong family history of gout (common in Māori and Pacific populations)
            - ≥ 1 episode of gout and serum urate > 0.6 mmol/L
            - ≥ 1 episode of gout and impending significant weight loss e.g. bariatric surgery, total diet replacement strategies etc.
            - ≥ 1 episode of gout and eGFR < 60 mL/min
    - Allopurinol is first line agent aiming for serum urate levels < 0.36 mmol/L or < 0.3 mmol/L if tophi.
        - Initial dosing ONLY is based on renal function:
            - Initial dosing ONLY is based on renal function:
                - eGFR > 60 mL/min → 100 mg allopurinol daily
                - eGFR 30 – 60 mL/min → 50 mg allopurinol daily
                - eGFR < 30 mL/min → 50 mg allopurinol on alternate days
        - Consider HLA-B*5801 screening in high-risk Asian populations before starting allopurinol to reduce risk of severe hypersensitivity reactions

        - All people of Chinese or Thai descent
        - All Korean people with eGFR < 60 mL/min
    - Ask people to stop allopurinol and to contact the practice if they develop a skin rash
    - Increase dose of allopurinol by 50 – 100 mg every 4 weeks until urate to target
    - Typically recommended cover colchicine when starting allopurinol to prevent gout flares.
        - Consider reducing dose in renal impairment and beware of drug to drug interactions
        - Discuss adverse effects, particularly GI adverse effects and to stop if significant effects occur
        - Ensure safe storage and to keep out of the reach of others as overdose can be fatal as no reversal agent
            - Consider requesting child-proof packaging on prescription if appropriate
        - Continue colchicine for 3-6 months after target serum urate is achieved
    - Once at target → measure serum urate 6 - 12 monthly to ensure still to target
    - Reassure that once target urate reached it may take > 12 months for gout flares to stop and years for tophi to dissolve
- Consider probenecid or febuxostat if intolerant of allopurinol or failure of allopurinol despite ensuring adherence.
- Gout is almost always caused by genetic variants and is typically more severe with a younger age of onset in Māori and Pacific Peoples. Recommended lifestyle interventions may be helpful, particularly if dietary triggers, but should not delay starting urate lowering pharmacotherapy or dominate the consultation.
    - Weight loss if overweight
    - Drinking 2 litres of water per day if no concerns over fluid overload
    - Avoiding excess alcohol
    - Reducing high sugar food and drinks
    - Eating regular meals as gout can be triggered by both fasting and overeating
    - Avoid foods that have previously triggered their gout flares (if any).
        - Once urate levels are to target, previous dietary triggers are often well tolerated.

    - Acute flares of gout may still occur. If so:
        - Prednisone or colchicine are typically preferred in CKM conditions with normal precautions
        - Consider back pocket prescription in case a further gout flare occur
        - Ensure serum urate to target but beware that urate levels may be normal in 50% of acute flares
    - It is important to ensure people with gout should have an up to date CKM risk assessment and optimised management of other CKM conditions to reduce their CV risk.

- **NB:** There continues to be no conclusive evidence supporting urate lowering treatment of asymptomatic hyperuricaemia

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11. Sick day management in CKM conditions

Sick day management in CKM conditions

Sick day management in CKM conditions

- Intercurrent illness can cause significant morbidity and mortality in people with CKM conditions, particularly at risk populations. Therefore, it is important everyone with CKM conditions have a management plan for sick days with basic advice on how to stay well and what to do with their medications.
    - Populations with CKM conditions at risk of adverse events from intercurrent illness:
        - Heart failure and other cardiac disease
        - Chronic kidney disease → especially if on dialysis
        - Diabetes → especially if type 1 diabetes or treated with insulin or sulfonylureas and/or corticosteroids e.g. prednisone
        - Cognitive impairment
        - Frailty and/or elderly → especially if living alone
        - History of falls
        - Post transplant or other immunodeficiency states
        - Chronic corticosteroid use
        - Significant liver disease
        - Previous adverse sick day events e.g. acute kidney injury

- Basic advice to stay well includes:
    - Notifying somebody that they are unwell
    - Avoiding strenuous activity
    - Avoiding NSAIDs particularly if CKD, ACEi/ARB and/or thiazide use
    - Ensuring they stay hydrated e.g. 1 standard glass of water per hour
        - May need to adjust if on dialysis or heart failure etc.
    - To continue eating as per normal or light meals if not tolerated → especially if on insulin
    - To monitor glucose levels 3-4 times per day or immediately if symptoms of hypoglycaemia or hyperglycaemia if known diabetes
        - People with type 1 diabetes, pancreatogenic diabetes or previous DKA will also need to check ketone levels
    - When to contact an ambulance and when to contact the practice
        - Consider use of personal medical alarm and/or alerts if high-risk particularly if living alone, feeling unsafe or reduced access to phone.

- Advice on whether to reduce or stop medications depends on the medication and type of illness, particularly if at risk of:
    - Hypovolaemia from fever, vomiting and/or diarrhoea
    - Hyperglycaemia if underlying diabetes, prediabetes or steroid treatment
    - Hypoglycaemia if reduced oral intake on insulin and/or sulfonylureas
    - Ketoacidosis if on empagliflozin or if insulin-deficient diabetes

- Common medications used to treat CKM medications that need to be stopped on sick days include:
    - Empagliflozin → stop in all acute illnesses including at least 3 days before (includes day of) an elective procedure, low carb diet or bowel preparation.
    - Diuretics → typically need to stop if at risk of hypovolaema
    - BP lowering therapy → typically need to stop if at risk of hypovolaemia, particularly ACEi and ARB
    - Metformin, vildagliptin and GLP1Ra → stop in GI illness
    - Sulfonylureas → likely need to reduce or omit dose if reduced oral intake
    - Meal time insulin → likely need to reduce or omit dose if reduced oral intake
    - Basal and premixed insulin → important to continue but reduce dose by ~ 30%

        - People on large doses of insulin may need 40-50% reductions

- It is important that patients have a plan when to restart their medications because prolonged cessation of their medications may lead to deterioration of their CKM condition e.g. increasing glucose levels, decompensated heart failure etc.
    - General rule is to restart medications 48 hours after feeling better and eating and drinking normally.
    - May need to restart medications earlier if clinically required

- Beware of medications such as opioids, lithium and gabapentinoids that are renally cleared and may accumulate causing significant adverse effects.
- Managing glucose levels during sick days can be difficult due to the stress response of illness, the need to withhold glucose lowering therapies, altered oral intake, reduced physical activity and medication use e.g. corticosteroids. Insulin is often required to control resulting hyperglycaemia.
    - Detailed guidance on management of sick days in people with diabetes including use of correction insulin can be found here

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12. Consideration of women with CKM conditions in child-bearing years and in pregnancy

Consideration of women with CKM conditions in child-bearing years and in pregnancy

Consideration of women with CKM conditions in child-bearing years and in pregnancy

This guidance on preventing adverse outcomes in CKM conditions does not cover the care of women in pregnancy. CKM conditions and their management can increase risks during pregnancy so the aim is to ideally optimise management before pregnancy to achieve the best outcomes for the mother and her baby/babies. Important points to consider are:

- Women with significant CKM conditions who are considering pregnancy should be offered a Preconception Consultation with a Maternal Fetal Medicine or Obstetric Medicine Specialist.
- Some medications used to treat CKM conditions are contraindicated in pregnancy and can lead to harmful outcomes for the mother and/or developing fetus. Women of childbearing age treated with these medications should be made aware that:
    - Contraception should be considered if pregnancy is not desired
    - Their medication should be changed once they become pregnant, or switched to an alternative medication that is safer for pregnancy.

    - Medication should typically never be stopped without an alternative discussed or commenced.
    - They contact their medical team as soon as possible if pregnancy confirmed

- Women with CKM conditions should be referred early in the first trimester for care under their regional Secondary or Tertiary Obstetrics Services. If a Preconception Consultation has not occurred then contact the local Obstetric team urgently for advice if concerns over medications or other aspects of care.
    - Stopping medications pre-emptively in significant CKM conditions can cause more harm than benefit. It is important these decisions are made in conjunction with the Obstetric team, particularly as many medications can be safely taken throughout pregnancy.

- Contraception options may be limited in women with CKM conditions, but are often safer than pregnancy. Contraception should be discussed in all women of child bearing age when pregnancy is not desired. Detailed guidance on contraception and its use in CKM and other conditions in Aotearoa New Zealand can be found here

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13. Symptom management and future planning beyond disease modification

Symptom management

Symptom management

- People with CKM conditions often have a high burden of physical and psychosocial symptoms. These should be routinely and actively screened for, identified and managed by the clinical team. Useful screening tools include the PHQ-2 score for depression and the DDS2 score for diabetes distress.
- People with advanced kidney disease (eGFR < 15 mL/min) typically have symptoms that significantly impact quality of life → please click here for advice on symptom management.
- Neuropathic pain is common in people with diabetes and can usually be successfully treated
    - Mild pain → paracetamol
    - Moderate to severe pain → low dose tricycylic e.g. nortriptyline 10 mg nocte
        - Can titrate nortriptyline and add pregabalin or gabapentin as required
        - Pregabalin is typically more effective than gabapentin with less adverse effects in diabetic neuropathy
        - Carbamazepine can be added in severe cases
    - Topical capsaicin 0.075% may be useful for localised neuropathic pain

Future planning beyond disease modification

Future disease planning beyond disease modification

- Despite best efforts, CKM conditions, often through cardiovascular and chronic kidney disease, will progress to increased morbidity and mortality. Early identification of palliative care needs and addressing these needs is the role of all health care practitioners working with CKM conditions
- People with advanced stages of CKM conditions have:
    - a high burden of physical symptoms
    - increased risk of hospitalisations
    - increased risk of sudden death
    - increased needs in the social, psychological and spiritual domains of Te Whare Tapa Whā
- Future planning of people's wishes in case of deterioration should be considered early, alongside ongoing management. To identify people whose health is deteriorating, the SPICT tool can be used. The Surprise Question; *'Would I be surprised if this patient were to die in the next year?'* can provide a prompt for action.
- Advance Care Planning (ACP), Shared Future Goals of Care and Serious Illness
- Conversation Guides are available to aid health care professionals.
- For people with complex needs, referral to specialist palliative care services should be considered early.

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Abbreviations:

ACE
Angiotensin-Converting Enzyme

ACEi
Angiotensin Converting Enzyme Inhibitors

ARB
Angiotensin Receptor Blocker

β-Blocker
Beta Blocker

BP
Blood Pressure

CCB
Calcium Channel Blocker

CKM
Cardiovascular-Kidney-Metabolic

CV
Cardiovascular

eGFR
Estimated Glomerular Filtration Rate

LVEF
Left Ventricular Ejection Fraction

NHYA
New York Heart Association

TD
Thiazide Diuretic

UACR
Urinary Albumin:Creatinine Ratio

WHO
World Health Organisation

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