Cardiovascular–Kidney–Metabolic Review Checklist

Medscape Primary Care Hacks

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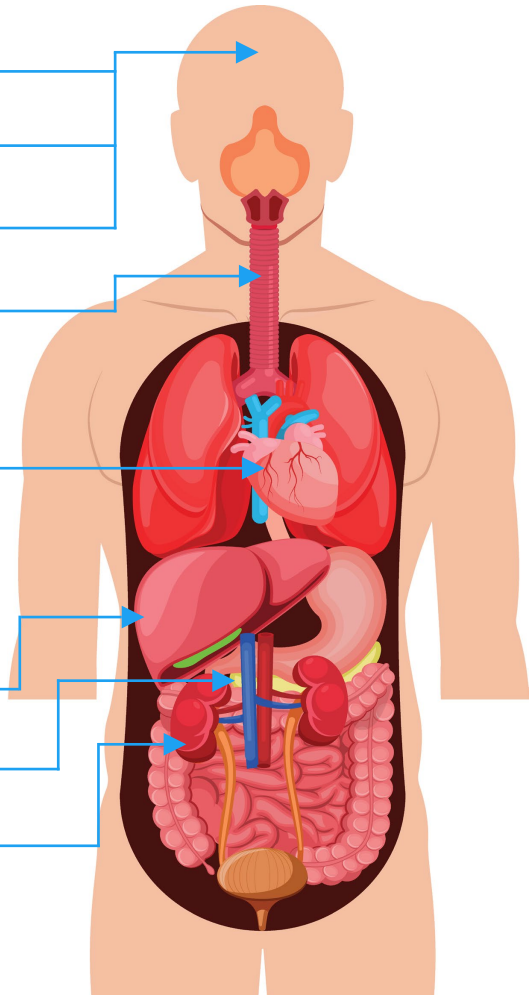
The cardiovascular, kidney, and metabolic systems are intrinsically interconnected, leading to the new description of the cardiovascular–kidney–metabolic (CKM) syndrome, also known as the CVRM syndrome.[1][2] The pleiotropic effects of many modern CKM therapies have demonstrated wide-ranging benefits in these overlapping organ systems.[1][2] However, even though the bidirectional relationship between these organ systems has long been recognised, many guidelines and clinical pathways still encourage a single-disease model of care.[1][2]

The delivery of healthcare and education needs to be reconfigured away from a disease-centred approach to a more holistic, person-centred model to improve both the quality and quantity of patients' lives and the sustainability of our healthcare systems.

This CKM checklist is a tool to facilitate an evidence-informed yet person-centred approach to patient care for all healthcare professionals.

The following areas should all be considered during shared decision-making.



Severe Mental Illness

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- Heart Failure
- Atrial Fibrillation

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Psychological Stress

Psychological stress, as well as trauma at any point in an individual's life, is associated with higher cardiometabolic risk.[1][3][4][5]

- ☐ Consider screening for anxiety and depression in people with CKM conditions; offer interventions as appropriate[1][4]
- ☐ Be aware of the overall positive relationship between time spent in **green and blue outdoor spaces** and mental health.[6]

Severe Mental Illness

SMI (including psychosis, schizophrenia, and bipolar disorder) is associated with reduced life expectancy (~15 years); this excess mortality is usually from preventable conditions such as CVD and diabetes.[7] Risk factors for CKM conditions tend to present earlier in those living with SMI than in the general population[8]

- ☐ Assess and manage CKM risk factors in people living with SMI **from the point of diagnosis**[8][9]
- ☐ Consider using the Lester positive cardiometabolic resource to guide the identification and management of CKM risk factors in people living with SMI: ***'Don't just screen—intervene'***[8]
- ☐ Consider the negative effects of different antipsychotics on CKM risk factors;[8][9] see the Lester positive cardiometabolic resource for a potential approach to monitoring side effects of antipsychotic medications.[8]

Do not forget the inverse care law: ***'The availability of good medical care tends to vary inversely with the need for it in the population served'***.[10]

Frailty and Older People

Frailty is known to worsen CV outcomes, especially in those aged >75 years with multiple long-term conditions.[2][11]

- ☐ Individualised strategies, guided by tools such as the electronic Frailty Index or the Rockwood Frailty Scale, are essential for addressing age-related decline and ensuring appropriate polypharmacy[2][12][13][14]
- ☐ Inappropriate polypharmacy can accelerate frailty progression—in one study of older men, each additional medication increased frailty risk by 27%[15]
  - careful medication review is essential for older adults, focusing on treatment optimisation and reducing unnecessary drug burden[2][14][16]
  - also see this BGS guidance on pragmatic prescribing for older people with moderate-to-severe frailty
- ☐ See the expert consensus statement on diabetes and frailty for practical methods of individualising T2D management in older adults and/or adults with frailty.[12]

Sleep Health and Airways

- ☐ Assess smoking status and offer a brief intervention to stop smoking; signpost to smoking cessation services[17]
- ☐ **Consider sleep health, which is an important and modifiable risk factor for CKM health**[18][19]
  - difficulty sleeping is one of the most common symptoms of the perimenopause and menopause;[20] refer to the BMS's *Managing sleep disturbance during the menopause transition* for specific advice
- ☐ Sleep disorders affecting quality and/or quantity of sleep (e.g. insomnia, restless legs syndrome, or OSAHS) have adverse CKM effects if not identified promptly (see also the AHA's fact sheet on sleep disorders and CV health)[18][19]
- ☐ For insomnia, consider **CBT-based sleep management programmes**,[21] e.g. Sleepful, Sleepio
- ☐ Consider **OSAHS** (using both the Epworth Sleepiness Scale and the STOP-BANG questionnaire)[22]
  - OSAHS is more prevalent in people with T2D, obesity/overweight, CVD, moderate/severe asthma, PCOS, hypothyroidism, and treatment-resistant hypertension[18][22][23][24]
  - consider OSAHS in hypertension especially, as the prevalence of OSAHS is 30–50% in hypertension, 70–85% in resistant hypertension, and >90% in refractory hypertension[24]
    - OSAHS identification and management may play an important role in treating resistant hypertension
- ☐ Be aware of possible glucocorticoid-induced hyperglycaemia and diabetes in people with asthma and/or COPD and manage appropriately.[25][26]

Overweight and Obesity

Obesity is an LTC with multiple pathophysiological aspects; prevention and treatment involve more than just 'eating less and moving more'. Like other LTCs, obesity is relapsing in nature and can lead to a range of complications, including cardiometabolic disease and 13 different types of malignancy.[27][28]

- ☐ Remember that language matters: seek permission to discuss weight, and use language that is person-first, collaborative, and nonjudgemental[29][30]
- ☐ Offer a brief intervention to people living with overweight or obesity: ASK, ASSESS, ADVISE, AGREE, and ASSIST.[29][31][32] **Be aware of weight bias and stigma**
- ☐ Consider using the 4 M's framework to assess drivers and complications of obesity—**mental, mechanical, metabolic, and monetary**[28]
- ☐ **Assess BMI and WtHR in any CKM review;**[31][33] this guide explains how best to measure waist circumference
  - the EASO and NICE now recommend WtHR (instead of waist circumference) in addition to BMI in the diagnostic process of obesity, with NICE thresholds as follows:[31][33]
    - **0.40–0.49:** healthy central adiposity (low risk of CKM syndrome)
    - **0.50–0.59:** increased central adiposity (increased risk of CKM syndrome)
    - **≥0.60:** high central adiposity (further increased risk of CKM syndrome)
  - remember to adjust BMI according to ethnicity and discuss individualised weight loss goals as appropriate[31][33]
- ☐ Consider referral to an evidence-based weight-management programme for multicomponent interventions (using a lower referral threshold for people from high-risk ethnic backgrounds)[31]
- ☐ Consider a GLP-1 RA or GLP-1/GIP RA as an adjunct to behavioural change strategies and lifestyle interventions in people living with overweight or obesity and one or more weight-related conditions (see also the Primary Care Hack on incretin therapies for obesity/overweight)[31]
  - it may be worth considering the role of CVD when making such decisions, as around two-thirds of obesity-related excess mortality is attributable to CVD[34]
  - the SELECT trial has shown that semaglutide improves CV outcomes in people living with obesity and CVD[35]
- ☐ Weight loss of 5–10% confers improvements in metabolic and mechanical complications (e.g. BP, lipids, mobility, sexual dysfunction), but weight loss of ≥10–15% can have a disease-modifying effect (e.g. on T2D, OSAHS, MASH)[23][36]
  - **BMI ≤27 kg/m² and WtHR ≤0.53 have been proposed as potential treatment targets for obesity**, as post-weight-change WtHR ≤0.53 has been strongly associated with lower risk of T2D, hypertension, and ASCVD and BMI ≤27 kg/m² with lower risk of hip/knee OA[37]
- ☐ In holistic obesity care, remember the integral roles of prevention, improvement, and resolution of obesity-related diseases, disorders, and complications inclusive of the CKM syndrome.[28][31][33]

Liver and MASLD (see also the Primary Care Hacks on LBTs and MASLD/MASH)

MASLD is primarily a metabolic disease; it is heavily influenced by lifestyle factors, and is the liver's manifestation of the MetS.[38][39]

To be diagnosed with MASLD, an individual must have **hepatic steatosis and at least one cardiometabolic risk factor**.[40]

MASLD is associated with an increased prevalence and incidence of CVD; **in MASLD, CVD is a more common cause of death than liver disease**.[41]

- ☐ European guidance recommends case-finding for MASLD with liver fibrosis in individuals with one or more of the following: T2D; abdominal obesity with one or more additional metabolic risk factors; abnormal LBT results[40]

- ☐ Assess the risk of advanced liver fibrosis using a noninvasive scoring system such as the FIB-4 score[40][42][43]
  - FIB-4 is calculated by the laboratory using age, AST, ALT, and platelets
- ☐ Consider referral for VCTE (a noninvasive ultrasound-based technique that measures liver stiffness, e.g. FibroScan®) or an equivalent second-line test, e.g. ELF or magnetic resonance elastography, if at intermediate risk for liver fibrosis; if at high risk, consider referral to Hepatology[40]
- ☐ Strongly encourage and facilitate weight loss where possible; a weight loss target of:[40]
  - ≥5% is recommended to reduce liver fat
  - 7–10% is recommended to improve liver inflammation
  - ≥10% is recommended to improve fibrosis.

Insulin Resistance, Prediabetes, and Type 2 Diabetes (see also the Primary Care Hack on prediabetes)

- ☐ **Identify those at high risk of T2D using a two-stage strategy:**[44]
  - offer a risk assessment using a validated computer-based diabetes risk-assessment tool that uses individuals' electronic health records, such as QDiabetes®-2018
  - offer an HbA_{1c} blood test to those with high risk scores (e.g. QDiabetes® score ≥10%)
  - if aged ≥25 years and of South Asian or Chinese descent with BMI >23 kg/m^2 or WtHR ≥0.5, HbA_{1c} testing can be offered without the need for a risk assessment tool
- ☐ In those at high risk of diabetes, follow the recommendations on HbA_{1c} monitoring, management, and referral in the NICE guideline[44] and Primary Care Hack on prediabetes
- ☐ **Note: prediabetes is more than just dysglycaemia:** in a 2023 prospective cohort study, reversion to normoglycaemia in prediabetes was only associated with lower mortality risk and longer life expectancy when it came with significant lifestyle change[45]
- ☐ For people living with T2D:
  - discuss the importance of 24-hour physical behaviours (the 5 S's):[23] breaking up prolonged **sitting; sweating; strengthening; sleep; stepping** (Kevin's patient-facing videos may be a useful reference)
  - review their current HbA_{1c} and trend[46]
  - consider other factors when individualising HbA_{1c} goals, e.g.: risks potentially associated with hypoglycaemia and other adverse effects; life expectancy; multiple LTCs; established vascular complications; and patient preference, resources, and support systems[46]
  - strive for remission if possible,[47] irrespective of weight.[48] **Note: weight loss of 5–10% confers metabolic improvement; weight loss of ≥10–15% can have a disease-modifying effect and lead to remission of T2D**[23]
  - review history of hypoglycaemia/hypoglycaemia awareness, DVLA adherence, and CBG monitoring where appropriate, and **consider CGM in all people with T2D on insulin**[23][49]
  - diabetic retinopathy: be aware of the possibility of worsening of pre-existing retinopathy if HbA_{1c} is rapidly lowered in those with T2D (e.g. when initiating a GLP-1 RA or GLP-1/GIP RA)[50]
  - be aware that all SGLT2is have negligible glucose-lowering effect once eGFR falls below 45 ml/min/1.73 m^2, so consider adding in an additional glucose-lowering medication such as a GLP-1 RA or GLP-1/GIP RA as required[51]
  - when considering medications, also see the Primary Care Hack *What Next After Metformin?* (parts one and two).

Lipids (see also the Primary Care Hack on lipid modification)

- ☐ To assess CV risk, use estimations of 10-year risk (e.g. QRISK®3, SCORE2, ASSIGN) but do not over-rely on them; calculations of lifetime CV risk (e.g. QRISK®3-lifetime) may offer a more holistic view[52]
- ☐ **Focus on LDL-C when managing CV risk**;[52][53] aim for levels as low as possible, as quickly as possible, for as long as possible[53][54]
- ☐ Consider tighter European LDL-C targets over the current, more lenient UK targets:[52][53]
  - **high risk (mostly primary prevention)**—≥50% LDL-C reduction from baseline; LDL-C <1.8 mmol/l
  - **very high risk (mostly secondary prevention)**—≥50% LDL-C reduction from baseline; LDL-C <1.4 mmol/l
- ☐ Encourage positive lifestyle choices, which are fundamental for CV risk management and overall CV health over and above cholesterol[52][53]
  - see Heart UK's guide on dietary assessment and tailoring dietary advice to a person's responses
- ☐ **Consider combination lipid lowering (e.g. statin + ezetimibe) as the first-line strategy in individuals at very high risk of CVD**[55]
- ☐ **Consider Lp(a) measurement at least once in every adult's lifetime**[56]—see also Heart UK's consensus statement on Lp(a)
  - Lp(a) is an independent risk factor for both CVD and calcific aortic valve stenosis;[57][58][59] elevated levels are largely genetically determined, and are not significantly affected by lifestyle choices or LLTs[53][57][58][59][60]
  - note: one in five people in the UK has raised Lp(a) levels.[57]

Blood Pressure

Hypertension is the leading modifiable risk factor for CVD-related morbidity and mortality worldwide.[13]

- ☐ Check BP in all CKM reviews, and opportunistically
- ☐ Refer to the Primary Care Hack on lifestyle changes for managing hypertension; it is possible to delay or prevent the progression of stage-1 hypertension through positive lifestyle choices alone[61]
- ☐ With guidelines disagreeing about the ideal approach to diagnosis and management of hypertension, it may be worth considering the BIHS's simplified approach:[13]
  - confirm a diagnosis of hypertension in people with sustained BP ≥135/85 mmHg despite dietary and lifestyle advice; BP should be measured with 7-day HBPM or day-time average ABPM
  - following a confirmed diagnosis of hypertension, commence BP-lowering therapy (alongside positive lifestyle choices) **irrespective of CV risk**
  - use the same BP targets, regardless of whether BP is measured in clinic, with HBPM, or with ABPM

- for **ALL** adults with confirmed hypertension, **use one consistent BP target of <130/80 mmHg** (or as low as reasonably achievable without causing unacceptable side effects) within 6 months of initiating treatment
  - exercise clinical judgement for those with frailty and/or a limited life expectancy, for whom higher BP targets may be appropriate

- ☐ Refer to the BIHS therapeutic management pathway for guidance on pharmacological management and referral[62]
- ☐ In line with ESC/ESH guidance, **consider low-dose combination therapy from initiation** (with a preference for single-pill combinations)[61]
- ☐ For **resistant hypertension**, take a practical approach to investigation and management (see also the BIHS's guidance on resistant hypertension)—i.e. verify BP measurements, support adherence to antihypertensive therapies, address modifiable causes, and exclude secondary causes,[61][63] e.g.:[63][64]
  - OSAHS (use the Epworth Sleepiness Scale and the STOP-BANG questionnaire)
  - iatrogenic causes—e.g. steroids, NSAIDs, hormone therapies (including CHC)
  - renal pathology—consider renal imaging.

Kidneys (see also the Primary Care Hack on CKD)

Prevention of ESKD matters, as it has lower survival rates than colorectal, prostate, and breast cancer.[65] Both eGFR <60 ml/min/1.73 m^2 and microalbuminuria are **independent** and **amplifying** predictors of mortality.[66]

Albuminuria reflects widespread vascular dysfunction, not just kidney damage;[67] even low-grade albuminuria (uACR ~0.8–1.1 mg/mmol) is associated with increased risk of CVD, CKD, and all-cause mortality.[67]

- ☐ Consider the presence of CKD through identification and testing of individuals at risk (key clinical risk factors include hypertension, CVD, diabetes, and obesity)[68][69]
- ☐ **Consider checking for albuminuria (with uACR) in all people with CKM syndrome, not just with CKD or diabetes**
- ☐ Establish a diagnosis of CKD by identifying low eGFR (<60 ml/min/1.73 m^2) and/or high uACR (≥30 mg/g [≥3 mg/mmol]) for >3 months, and/or structural disease (e.g. one kidney, ADPKD, horseshoe kidney)[68][69][70]
- ☐ Classify, code, and monitor CKD appropriately (for more information, refer to the section on identification in the Primary Care Hack on CKD)[68][69][70]
- ☐ Consider interventions for CKD inclusive of lifestyle and dietary modifications, lipid management, BP management, meeting HbA_{1c} targets (in DKD), and weight optimisation (for more information, refer to the section on interventions in the Primary Care Hack on CKD)[52][68][69][71][72]
  - in appropriate patients, the following drug classes may help to reduce the risk of adverse kidney **and** CV outcomes: RAASis, SGLT2is, nonsteroidal MRAs, GLP-1 RAs, and GLP-1/GIP RAs[69][73]
  - individualise HbA_{1c} targets in people with DKD[72]
- ☐ Consider referral as per NICE criteria, or if 5-year risk of requiring renal replacement therapy is >5% (measured using the four-variable KFRE).[68][69]

Heart—Atherosclerotic Cardiovascular Disease

ASCVD is inclusive of ACS, CCS (formerly known as stable CAD), coronary revascularisation, stroke, TIA, PAD, and aortic atherosclerosis (including AAA and TAA).[74][75]

- ☐ For those with any form of ASCVD, effective treatment of associated LTCs (i.e. CKM management) is recommended, inclusive of lifestyle interventions, behavioural modifications, and pharmacology[52][76][77][78][79]
- ☐ **Antiplatelets are not prescribed routinely for the primary prevention of CVD**[52]
- ☐ For **secondary prevention** of ASCVD, long-term antiplatelet therapy is recommended as follows:
  - **CCS**: aspirin 75–100 mg daily is the preferred treatment, after an initial period of DAPT[76]
  - **stable cerebrovascular disease (stroke/TIA)**: clopidogrel 75 mg daily is the preferred treatment[77]
  - **symptomatic PAD**: clopidogrel 75 mg daily is the preferred treatment[79][80]
  - note: DAPT is usually used initially after ACS/PCI/an acute cerebrovascular event, and will be directed by specialists[77][78]
- ☐ **If stable angina is suspected:** while awaiting specialist review, consider issuing a GTN spray (don't forget the 3S's—stop, sit, spray) and initiating a high-intensity statin (e.g. atorvastatin 40 mg), a beta-blocker (e.g. bisoprolol 5 mg), and aspirin (75 mg).[76][81]

Note: the STRIDE trial has shown that semaglutide improved walking capacity, QoL, and disease progression for people with T2D and PAD.[82]

Heart Failure (see also the Primary Care Hacks on HFpEF and HFrEF)

- ☐ **Prioritise the primary prevention of HF across the lifespan.**[4][83][84] This involves the early detection and treatment of CKM conditions as key targets for HF prevention[4][83][84]
- ☐ **Always consider the presence of HF**, especially in those at increased risk and/or with signs and symptoms (for further information, see the Primary Care Hacks on HFpEF and HFrEF)[84][85]
  - key risk factors include conditions characteristic of the CKM syndrome (including obesity, T2D, CKD, hypertension, and dyslipidaemia)[84][85]
  - risk of HHF and all-cause mortality are highest during the initial weeks after first presentation in the community.[86] Therefore, acting quickly in primary care really matters
- ☐ While awaiting specialist assessment for suspected HF, consider **managing congestion with diuresis** (e.g. furosemide 40–80 mg or double pre-existing dose) **and initiating an SGLT2i**
- ☐ HF management, irrespective of subtype, involves **guideline-recommended medical therapy** and treatment of associated LTCs (i.e. **CKM management**).[84][85][87]

Atrial Fibrillation

- ☐ Check pulse; if irregular, consider an ECG to identify AF[88]
- ☐ If AF confirmed:
  - establish duration and type, consider cause, and assess symptom severity[88][89]

- carry out physical examination and consider checking FBC, U&E, HbA_{1c}, lipids, TFTs, LBTs, weight, WtHR, BP, and uACR—for differential diagnosis and maximisation of holistic interventions[88][89][90]
- **conduct risk assessments for stroke using CHA_2DS_2-VASc[90][91] and for bleeding risk using ORBIT**[91]
- commence anticoagulation (with a DOAC or warfarin) if appropriate (see also the Primary Care Hack on DOACs in people with nonvalvular AF and renal impairment)[90][91]
  - be aware that those with a history of moderate-to-severe mitral stenosis or a metallic artificial heart valve require anticoagulation with warfarin[90]
- consider initiation of HR control with a cardioselective beta-blocker (e.g. 2.5 mg bisoprolol) if necessary, followed by referral to local Cardiology services for further investigations (e.g. echocardiography) as required[89]

- ☐ Note: those with AF often do not have a normal BNP[92]
- ☐ Weight loss of ≥10% is strongly recommended in those with AF and overweight or obesity;[90] weight loss of >10% is linked to a sixfold higher chance of arrhythmia-free AF survival[93]
- ☐ Physical activity can help to prevent AF incidence and recurrence, with regular aerobic exercise improving AF-related symptoms and QoL[90][94]
  - avoid excessive endurance exercise, however, which may promote AF.[90]

Inflammatory Conditions

Chronic inflammation increases CV risk by driving atherogenesis and atherosclerosis.[4][53]

Inflammatory conditions therefore increase the risk of CVD, both acutely and in the long term; notable conditions include inflammatory arthritis (e.g. rheumatoid arthritis, gout), SLE, IBD, and psoriasis (see also the Primary Care Hack on psoriasis).[4][95][96][97]

- ☐ Undertake CV risk assessment in people with long-term inflammatory conditions and consider increasing the risk estimate based on the level of disease activity[4]
- ☐ Consider an annual CKM check in all people living with a chronic inflammatory condition
- ☐ Alongside any anti-inflammatory therapies, CV risk should be treated with similar interventions in those with inflammatory conditions as in the wider high-risk population.[4]

Polycystic Ovary Syndrome

- ☐ Because women with PCOS are at increased risk of the CKM syndrome,[98][99][100] always consider the presence of PCOS in women with CKM conditions
- ☐ Use the **Rotterdam Criteria** to diagnose PCOS in women aged ≥18 years[99][101][102]
- ☐ **Educate women with PCOS at point of diagnosis** that they are at higher risk of developing certain CKM conditions (obesity, prediabetes, T2D, gestational diabetes, dyslipidaemia, hypertension, OSAHS, and CVD)[98][99][100][101][103]
- ☐ If PCOS is confirmed, consider assessment and monitoring of CKM risk factors:
  - **at point of diagnosis**, screen for obesity, dyslipidaemia, hypertension, T2D, and other possible CKM risk factors[99][101]
  - thereafter, consider **annual screening** for obesity, T2D/prediabetes, dyslipidaemia, and hypertension, **regardless of age or weight at diagnosis**[99]
- ☐ **Tailored lifestyle interventions and positive lifestyle choices**, involving dietary and physical activity strategies and modest weight loss (e.g. 5–10%), are a mainstay of the metabolic management of PCOS; these interventions can help to restore ovulatory cycles, improve metabolic risk, and aid weight management[99][101][104][105][106]
- ☐ Bear in mind that maintaining weight loss is even more difficult for women with PCOS than the general population[101]
- ☐ Consider use of:
  - **metformin** for those with PCOS and a BMI ≥25 kg/m^2 (to improve metabolic and anthropometric outcomes, as evidence suggests the effect on reproductive outcomes is modest)[99][104]
  - **a GLP-1 RA or GLP-1/GIP RA** for the management of coexisting obesity, as per general population guidelines[99][104] (note: 35–50% of women diagnosed with PCOS have overweight or obesity[104]).

Perimenopause and Menopause

- ☐ Appreciate the effects of the menopause transition on cardiometabolic risk:
  - increased risk of obesity, T2D, CVD, osteoporosis, dementia, and cancer, all of which can emerge 10–15 years after menopause onset[107][108][109]
  - increased cardiometabolic risk from menopause-related vasomotor symptoms, sleep disorders, and mood changes[107]
  - a modest rise in LDL-C and triglyceride levels (~10–15%) and a modest reduction in HDL-C levels[109]
- ☐ Recognise in the reproductive history the female-specific conditions (i.e. **hypertensive pregnancy disorders**, **gestational diabetes**, **POI**, and **early menopause**) that enhance CV risk before, during, and after the menopausal transition[109]
- ☐ **Early individualised optimisation of cardiometabolic risk factors** (i.e. hypertension, obesity, diabetes, dyslipidaemia, and smoking) is essential during menopause transition and midlife[107][108]
- ☐ **HRT:**
  - when initiated aged <60 years or within 10 years of the menopause, HRT may reduce CVD and all-cause mortality[109]
    - if initiated aged >60 years, or >10 years after the onset of menopause, CV protection is likely to be lost[110]
    - **remember that early initiation of HRT has the greatest CV benefit**[109]
  - in menopausal women at increased risk of VTE, including those with increased BMI, consider transdermal (rather than oral) HRT[111]
  - also use transdermal oestrogens for women with hypertriglyceridemia[112]
  - for advice on managing the menopause in women with established CVD, refer to BMS guidance[113]
  - for specific advice on prescribing incretin-based therapies alongside HRT, refer to the BMS tool for clinicians on HRT and incretin therapies.[114]

Erectile Dysfunction

ED is associated with subclinical CVD and is an independent predictor of future CV events.[4][115]

- ☐ Assess CKM risk in all men presenting with ED (including WtHR, lipid profile, BP, HbA_{1c}, uACR)[116]
- ☐ Review any concurrent medications (e.g. spironolactone, eplerenone, beta-blockers, and thiazide-like diuretics) to exclude a iatrogenic cause of ED.[4][116]

HIV

Because of the success of ART, many PLWH can expect a life expectancy comparable to that of the general population; therefore, **noncommunicable diseases including CVD are now leading causes of death in PLWH.**[117]

- ☐ Offer PLWH regular CKM monitoring at least annually (including WtHR, lipid profile, BP, HbA_{1c}, U&E, and uACR)[118][119]
- ☐ Consider offering all PLWH aged ≥40 years a statin for the primary prevention of CVD, irrespective of lipid profile or estimated CV risk[56]
- ☐ Check any potential drug interactions using the Liverpool drug interaction checker.

Migraine

Migraine is associated with a twofold increased risk of ischaemic stroke and 1.5-fold increased risk of IHD; stronger evidence supports this association in migraine with aura.[4]

QRISK®3 and QRISK®3-lifetime include migraine as a CV risk factor.

Note: triptans are contraindicated in patients with certain CV disorders, including IHD, previous MI or stroke, arrhythmias, mild uncontrolled or moderate-to-severe controlled hypertension, and peripheral vascular disease.

Prescribing Considerations

- ☐ Discuss medication adherence and, if necessary, explore barriers/preferences[120]
- ☐ Whenever possible, try to prescribe alternatives to drugs associated with weight gain (e.g. certain tricyclic antidepressants, corticosteroids, beta-blockers, and antipsychotics)[121]
- ☐ Provide sick-day guidance for:[122][123][124][125]
  - o people with T2D on insulin
  - o people taking other relevant medications (i.e. those covered in the SADMANS mnemonic, as well as incretin therapies and nonsteroidal MRAs); remind patients to pause these drugs during any significant intercurrent illness, and to restart them once they are eating and drinking normally and recovered from their illness
- ☐ Educate women of childbearing age that many medications (e.g. ACEis, ARBs, statins, SGLT2is, incretin therapies, nonsteroidal MRAs) are contraindicated in pregnancy, and counsel them regarding contraception[51][125][126][127][128]
- ☐ If planning pregnancy, consider prompt referral to prepregnancy services for those in certain cohorts—e.g. previous history of metabolic surgery, T1D, or T2D[127][129]
- ☐ For specific advice on prescribing incretin-based therapies alongside contraception or HRT, refer to the CoSRH guidance on contraception and incretin therapies[130] and the BMS tool for clinicians on HRT and incretin therapies[114]
- ☐ Ensure appropriate/optimal prescribing:
  - o consider de-intensifying therapy in the context of functional dependence and/or frailty,[14][16] and if medications are no longer indicated because of the health benefits of weight loss (e.g. antihypertensives)
  - o consider following the seven steps to appropriate polypharmacy to aid achievement of appropriate, realistic polypharmacy[16]
- ☐ When an SGLT2i or incretin therapy is commenced (see also the Primary Care Hacks *Extra-Glycaemic Indications of SGLT2 Inhibitors* and *What Next After Metformin? Part 2*):
  - o in T2D, consider reduction in SU or insulin dose.[51][125] If on insulin and initiating a GLP-1 RA, consider cautiously reducing insulin dose in a stepwise manner (with increased CBG monitoring) to avoid DKA[131]
  - o when introducing an SGLT2i, consider adjustment of any dose of diuretic[132]
  - o Kevin's patient-facing YouTube videos on SGLT2is may be a useful reference for patients.

AAA=abdominal aortic aneurysm; **ABPM**=ambulatory blood pressure monitoring; **ACEi**=angiotensin-converting enzyme inhibitor; **ACS**=acute coronary syndrome; **ADKDP**=autosomal dominant polycystic kidney disease; **AF**=atrial fibrillation; **AHA**=American Heart Association; **ALT**=alanine transaminase; **ARB**=angiotensin receptor blocker; **ART**=antiretroviral therapy; **ASCVD**=atherosclerotic cardiovascular disease; **AST**=aspartate transferase; **BGS**=British Geriatrics Society; **BIHS**=British and Irish Hypertension Society; **BMI**=body mass index; **BMS**=British Menopause Society; **BNP**=B-type natriuretic peptide; **BP**=blood pressure; **CAD**=coronary artery disease; **CBG**=capillary blood glucose; **CBT**=cognitive behavioural therapy; **CCS**=chronic coronary syndrome; **CGM**=continuous glucose monitoring; **CHA_2DS_2-VASc**=Congestive heart failure, Hypertension, Age ≥75 years, Diabetes, Stroke/TIA/thromboembolism, Vascular disease, Age 65–74 years, Sex category; **CHC**=combined hormonal contraceptive; **CKD**=chronic kidney disease; **CKM**=cardiovascular–kidney–metabolic; **COPD**=chronic obstructive pulmonary disease; **CoSRH**=College of Sexual and Reproductive Health; **CV**=cardiovascular; **CVD**=cardiovascular disease; **CVRM**=cardiovascular–renal–metabolic; **DAPT**=dual antiplatelet therapy; **DKA**=diabetic ketoacidosis; **DKD**=diabetic kidney disease; **DOAC**=direct oral anticoagulant; **DVLA**=Driver and Vehicle Licensing Agency; **EASO**=European Association for the Study of Obesity; **ECG**=electrocardiogram; **ED**=erectile dysfunction; **eGFR**=estimated glomerular filtration rate; **ELF**=Enhanced Liver Fibrosis; **ESC**=European Society of Cardiology; **ESH**=European Society of Hypertension; **ESKD**=end-stage kidney disease; **FBC**=full blood count; **FIB-4**=Fibrosis-4; **GIP**=glucose-dependent insulinotropic polypeptide; **GLP-1**=glucagon-like peptide-1; **GTN**=glyceryl trinitrate; **HbA_{1c}**=glycated haemoglobin; **HBPM**=home blood pressure monitoring; **HDL-C**=high-density-lipoprotein cholesterol; **HF**=heart failure; **HFpEF**=heart failure with preserved ejection fraction; **HFrEF**=heart failure with reduced ejection fraction; **HHF**=hospitalisation for heart failure; **HIV**=human immunodeficiency virus; **HR**=heart rate; **HRT**=hormone-replacement therapy; **IBD**=inflammatory bowel disease; **IHD**=ischaemic heart disease; **KFRE**=Kidney Failure Risk Equation; **LBT**=liver blood test; **LDL-C**=low-density-lipoprotein cholesterol; **LLT**=lipid-lowering therapy; **Lp(a)**=lipoprotein(a); **LTC**=long-term condition; **MASH**=metabolic dysfunction-associated steatotic hepatitis; **MASLD**=metabolic dysfunction-associated steatotic liver disease; **MetS**=metabolic syndrome; **MI**=myocardial infarction; **MRA**=mineralocorticoid receptor antagonist; **NICE**=National Institute for Health and Care Excellence; **NSAID**=nonsteroidal anti-inflammatory drug; **OA**=osteoarthritis; **ORBIT**=Outcomes Registry for Better Informed Treatment; **OSAHS**=obstructive sleep apnoea/hypopnoea syndrome; **PAD**=peripheral artery disease; **PCI**=percutaneous coronary intervention; **PCOS**=polycystic ovary syndrome; **PLWH**=people living with HIV; **POI**=premature ovarian insufficiency; **QoL**=quality of life; **RA**=receptor agonist; **RAASi**=renin–angiotensin–aldosterone system inhibitor; **SADMANS**=SUs, ACEis, Diuretics/Direct renin inhibitors, Metformin, ARBs, NSAIDs, SGLT2is; **SGLT2i**=sodium–glucose co-transporter-2 inhibitor; **SLE**=systemic lupus erythematosus; **SMI**=severe mental illness; **STOP-BANG**=Snoring, Tiredness, Observed apnoea, Pressure, BMI, Age, Neck size, Gender; **SU**=sulfonylurea; **T1D**=type 1 diabetes; **T2D**=type 2 diabetes; **TAA**=thoracic aortic aneurysm; **TFT**=thyroid function test; **TIA**=transient ischaemic attack; **uACR**=urinary albumin:creatinine ratio; **U&E**=urea and electrolytes; **VCTE**=vibration-controlled transient elastography; **VTE**=venous thromboembolism; **WtHR**=waist-to-height ratio.