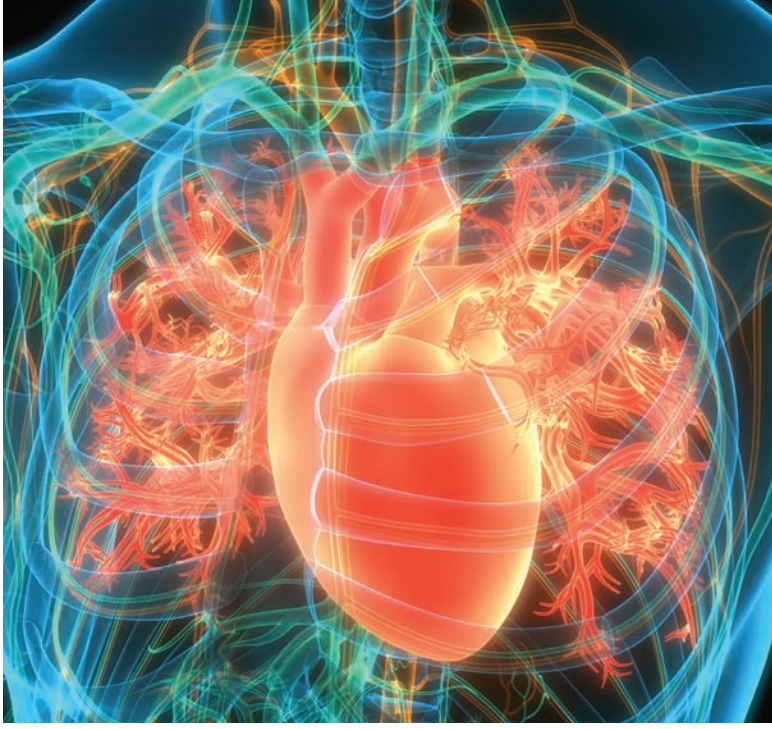
Managing CVD risk in type 1 diabetes

Optimising outcomes



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Cardiovascular disease (CVD) risk is increased in people with type 1 diabetes compared with those without diabetes. CVD risk recognition, regular patient education and multiple risk factor care together reduces CVD risk. GPs are well placed to provide CVD risk factor management for people with T1D and to co-ordinate their ongoing care.

Key points

- **Cardiovascular disease (CVD) is increased several-fold in people with type 1 diabetes (T1D) relative to their peers without diabetes, and this risk is particularly high in those with microvascular complications or with early-onset (under 10 years of age) T1D.**
- **Managing multiple traditional risk factors is key for both CVD prevention and risk reduction of other chronic complications of diabetes.**
- **Achieving glycated haemoglobin (HbA_{1c}) targets alone is not sufficient to prevent CVD. Optimising other aspects of glycaemia, insulin delivery modality and other risk factors for CVD is also desirable.**
- **Early and ongoing education and dialogue for people with T1D are key to CVD risk reduction.**
- **A team-based approach involving GP care and shared care with other physicians (e.g. endocrinologist, cardiologist) and allied healthcare professionals as needed is ideal.**

An estimated 135,000 Australians have type 1 diabetes (T1D), two-thirds of whom are aged 40 years or above and one-third 60 years or above, hence most are at an age when cardiovascular disease (CVD) may become clinically manifest.[1] Postmortem studies show that in Western societies atherosclerosis starts in youth, even in those without diabetes.[2] Other studies have shown that carotid intima-media thickness, a surrogate marker of atherosclerosis, is increased in youth with, versus those without, T1D, even after just one year of having diabetes.[3] CVD is a leading cause of morbidity and mortality in T1D.[4] Based on the T1D Index, in Australia relative to the general population, an estimated 22 years of healthy life years are lost by people with T1D, and an estimated 18,000 people would otherwise be alive today had they not died prematurely due to T1D and its complications.[5]

Although recent trends show improved long-term survival for people with T1D, they still have substantial risk of cardiovascular morbidity and mortality.[4,6] Recent population-based studies show a four- to 10-fold higher CVD risk in people with T1D compared with matched counterparts without diabetes.[7] Therefore, early recognition and intervention to comprehensively address risk factors and subclinical and clinical CVD are of vital importance.

There is substantial overlap in CVD risk factor care in those living

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with T1D, those living with type 2 diabetes (T2D) and the general population. The GP is well placed to provide CVD risk factor management for people with T1D and to co-ordinate their care. Here, we review the drivers of and mitigation of CVD in people with T1D, with a focus on primary prevention. Secondary prevention of CVD also includes aggressive risk factor management.

Characteristics and drivers of atherosclerosis in type 1 diabetes

Atherosclerosis in people with diabetes, including T1D, differs from that in people without diabetes, although the responses to medical treatments are similar. In people with T1D, atherosclerosis starts earlier, progresses faster and extends more distally in arteries, often reducing the potential for interventions such as bypass surgery and stenting and, therefore, placing great emphasis on medical therapies. There is more inflammation and more unstable (lipid-rich, inflamed, thin fibrous cap) plaques in people with diabetes compared with those without.[8] People with T1D are also more likely to have a procoagulant and antifibrinolytic state, which promotes blood clotting and often triggers an acute CVD event.[8]

Accelerated atherosclerosis in people with diabetes is multifactorial. Traditional risk factors remain important – i.e. age, diabetes per se (including diabetes duration and various aspects of glycaemia, discussed later), adiposity, smoking, dyslipidaemia, hypertension, family history and genetics. Novel risk factors include renal dysfunction, sedentary lifestyle, insulin resistance, peripheral hyperinsulinaemia (due to subcutaneous insulin delivery), qualitative changes in lipoproteins (e.g. glycation and oxidation), inflammation, oxidative stress and endothelial dysfunction.

In those affected, CVD starts in youth and progresses silently for many years until an acute event occurs. The 2025 American Diabetes Association (ADA) guidelines do not recommend screening (e.g. by coronary artery calcium [CAC] scoring or angiography) for silent or subclinical disease, as it does not improve clinical outcomes provided the multiple traditional risk factors are treated aggressively.[9] However, in the general and CVD population, showing clinicians and patients image-based evidence of vascular damage has been associated with greater likelihood of starting or continuing risk factor care, such as lipid-lowering drugs.[10] In Australia, screening for retinal and renal diabetes complications is recommended and subsidised but screening for subclinical CVD is not. However, as clinicians, we should have a low threshold for suspicion, particularly as CVD in people with diabetes may manifest atypically.

Once CVD is clinically evident it should be treated aggressively, but we must be mindful that the first clinical evidence of CVD may be sudden cardiac death.[11] CVD in people with T1D may also be silent or present with atypical symptoms.[11] For people without clinically evident CVD (primary prevention), the use of CVD risk calculators, ideally ones developed for people with T1D, should be used to guide clinician and patient dialogue and treatment decisions. Using calculators that have not been developed from, or validated in, people with T1D usually underestimate risk.

CVD risk factor assessment and management

Much evidence in the general and T2D populations supports that managing multiple risk factors reduces CVD.[12] However, there are few T1D-specific trials, and they are unlikely to be conducted given the high CVD risk of adults with T1D and the compelling indirect evidence base supporting recommendations for multiple risk factor management. Furthermore, if hard rather than surrogate clinical events are used, trials would take many years to conduct. Pleasingly, subgroup analyses of trials show similar benefits of some treatments, such as lipid-lowering therapy for people with T1D or T2D and in the general population. For example, for every 1 mmol/L reduction in LDL-cholesterol (LDL-C) there is a 21% risk reduction of a CVD event and 9% lower risk of five-year cardiac death.[13] Large real-world T1D observational studies are of interest.

CVD risk calculators for type 1 diabetes

Six CVD risk calculators for people with T1D are summarised in Table 1. Included are the definition of CVD, risk period and included risk factors. Importantly, the comprehensive COSMO App (https://antranduy.shinyapps.io/cosmo-t1d/) assumes some background risk factor levels.

Risk factor targets and suggested levels

Recommended risk factor targets are summarised in Table 2, and further details on some of these are discussed below.

Glucose management

The T1D Diabetes Complications and Control Trial (DCCT; n = 1 441), with over 30 years' and ongoing follow up, showed a 31% increased CVD risk for every 1% increase in HbA_{1c} levels and that lowering HbA_{1c} was associated with reduced risk of CVD and microvascular complications and with long-term metabolic memory.[14] Intensive diabetes therapy (basal bolus or insulin pump) is now routine for T1D care, and for adults with T1D a general target of less than 7.0% (53 mmol/mol) is recommended.[15] For children and adolescents with T1D, the recommended target HbA_{1c} is at least 6.5% (48 mmol/mol) for those with access to diabetes technology such as insulin pumps, and at least 7.0% (53 mmol/mol) for those without.[16]

Other glucose-related metrics associated with increased CVD risk in people with diabetes include hypoglycaemia and greater glucose and HbA_{1c} variability.[15] Even mild hypoglycaemia in people with T1D is associated with increased inflammation, oxidative stress and vasoconstriction, which can last a week.[17] Severe hypoglycaemia (blood glucose <2.0 mmol/L) in T2D trials has been associated with increased cardiovascular mortality.[18] Hypoglycaemia prolongs the QT-interval and is proarrhythmogenic, and a likely cause of the 'dead-in-bed' syndrome (sudden unexpected overnight death) in people with T1D.[19] Hence, avoiding hypoglycaemia is a key consideration in T1D care.

Glucose variability is also an independent CVD risk factor in diabetes.[20] Although no study has directly investigated glucose variability and CVD events in T1D, glycaemic variability has been associated with increased CAC scores in men with T1D.[21] In people with T2D HbA_{1c}

Table 1. CVD risk calculators for people with type 1 diabetes

Name and website	Definition of CVD	Time period	Risk factors
Steno T1 (CVD) Risk Engine https://steno.shinyapps.io/T1RiskEngine/	A composite of fatal and nonfatal events of ischaemic heart disease, ischaemic stroke, heart failure and peripheral artery disease	5- and 10-year CVD risk Note: also calculates 5-year ESKD risk	Previous CVD, age, sex, diabetes duration, SBP, LDL-C, HbA_{1c}, albuminuria, eGFR, smoking, regular exercise
QRISK3 risk calculator https://www.qrisk.org/index.php	Stroke, transient ischaemic attack, myocardial infarction or angina	10-year CVD risk	Age, sex, smoking status, diabetes status, CVD family history, CKD, AF, BP drugs, migraine history, history of RA/SLE, severe mental illness, atypical antipsychotic use, steroid use, diagnosis of ED, cholesterol/HDL-C ratio, SBP, BMI
MSD Cardiovascular Risk Assessment in T1D https://www.msdmanuals.com/professional/multimedia/clinical-calculator/5-year-risk-of-cardiovascular-disease-in-type-i-diabetes	Fatal or nonfatal myocardial infarction or stroke	5-year CVD risk	Age, diabetes duration, total cholesterol, HDL-C, HbA_{1c}, SBP, smoking status, microalbuminuria, prior CVD event
Diabetes Epidemiology Group (DEG) CVD Risk Prediction T1D https://diabepi.shinyapps.io/cvdrisk/	Any hospital admission or death due to myocardial infarction, unstable angina, stroke, transient ischaemic attack peripheral vascular disease; or any coronary, carotid or peripheral artery revascularisations; or major amputation procedures; or any death due to these conditions; or acute coronary heart disease	10-year CVD risk	Diabetes duration, age, sex, smoking status, HbA_{1c}, eGFR, total cholesterol/HDL-C, BMI, SBP, albuminuria, retinopathy status, hypertension, dysplipidaemia, AF
LIFE-T1D https://dom-pubs.onlinelibrary.wiley.com/doi/10.1111/dom.15531 (under supporting information)	Coronary heart disease, cerebrovascular disease or peripheral artery disease	10-year CVD risk	Age, T1D onset, smoking status, BMI, SBP, HbA_{1c}, eGFR, non-HDL-C, albuminuria, retinopathy
COSMO-T1D https://antranduy.shinyapps.io/cosmo-t1d/	Gives separate risk for: fatal and nonfatal myocardial infarction, fatal and nonfatal stroke, heart failure, peripheral vascular disease, severe hypoglycaemia and hyperglycaemia (i.e. events leading to hospitalisation), amputation, ESKD, percutaneous coronary intervention and coronary artery bypass graft	Lifetime risk of previous noted categories rather than CVD as one outcome	Age, sex, smoking status, HbA_{1c}, BMI, SBP, triglycerides, HDL-C, LDL-C, eGFR
ESC CVD Risk Calculation App SCORE2-Diabetes calculator https://www.escardio.org/Education/Practice-Tools/CVD-prevention-toolbox/esc-cvd-risk-calculation-app#	Nonfatal myocardial infarction, stroke or any CVD mortality	10-year CVD risk	Age, smoking, SBP, total cholesterol, HDL-C, age at diabetes diagnosis, HbA_{1c}, eGFR

Abbreviations: AF = atrial fibrillation; BMI = body mass index; BP = blood pressure; CKD = chronic kidney disease; CVD = cardiovascular disease; ED = erectile dysfunction; eGFR = estimated glomerular filtration rate; ESKD = end-stage kidney disease; HbA_{1c} = glycated haemoglobin; HDL-C = high-density lipoprotein-cholesterol; LDL-C = low-density lipoprotein-cholesterol; RA = rheumatoid arthritis; SBP = systolic blood pressure; SLE = systemic lupus erythematosus; T1D = type 1 diabetes.

variability has also been associated with higher CVD risk.[22,23] With continuous glucose monitoring (CGM), we have better means to assess glucose variability as reflected by the coefficient of variation, with a treatment goal of less than 36.0%. The CGM-related treatment goal for time-in-range (3.9 to 10.0 mmol/L) is greater than 70% and for time in hypoglycaemia (<3.9 mmol/L) is less than 4.0%.[24] CGMs are now subsidised for all people in Australia with T1D, and multiple T1D studies show improved HbA_{1c} and CGM metrics.[25] CGM uptake by

Table 2. Risk factor targets in type 1 diabetes[9]

Risk factor	Screening test	Target	Actions to be considered
Hyperglycaemia	HbA_{1c} test CGM time-in-range (3.9–10 mmol/L)	≤7.0% (≤53 mmol/mol) or as individualised >70%	Increase glucose monitoring, review insulin doses and timing, review carbohydrate counting
Glucose variability	CGM glucose variability Serial HbA_{1c} SD or CV	<36.0% Nil recommended	Optimise insulin dosing
Hypertension	Blood pressure measurement	<130/80 mmHg	Lifestyle changes (e.g. weight loss, low-salt diet, smoking cessation), BP drugs
Dyslipidaemia	Lipid profile	**As per ADA 2025 guidelines[9]** **Primary prevention** • Adults – Age 20–39 years and additional CVD risk factors: consider statin* – Age 40–75 years and no other CVD risk factors: start statin – Age 40–75 years and other CVD risk factors: reduce LDL-C by ≥50% of baseline and goal of <1.8 mmol/L – Age >75 years: continue statin or consider starting a statin • Youth – If LDL-C >3.4 mmol/L consider a statin **Secondary prevention** • Reduce LDL-C by ≥50% of baseline and goal of <1.4 mmol/L	Optimise glycaemia, lifestyle changes, plant sterol foods, lipid-lowering drugs – e.g. statin
Albuminuria	Urine ACR	<3 mg/mmol	Optimise glycaemia, consider commencing ACEi/ARB or SGLT-2 inhibitor
Smoking	History	Smoking cessation	Encourage smoking cessation by both behavioural measures and pharmacotherapy
Thrombosis prevention	None	**Primary prevention** • Only for individuals at high CVD risk **Secondary prevention** • All individuals with known CVD	Aspirin therapy

Abbreviations: ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BP = blood pressure; CGM = continuous glucose monitoring; CV = coefficient of variation; CVD = cardiovascular disease; HbA_{1c} = glycated haemoglobin; LDL-C = low-density lipoprotein-cholesterol; SD = standard deviation; SGLT-2 = sodium-glucose cotransporter-2; urine ACR = urine albumin-to-creatinine ratio.
* Statins should not be used in pregnant or breastfeeding women.

people in Australia with T1D is estimated to be about 80%.

Insulin pump use, either alone or linked with CGM (using an automated insulin delivery system), is also associated with better HbA_{1c} and glucose metrics, and, as shown by the Swedish Diabetes Registry data, associated with major reductions in CVD death (hazard ratio, 0.58), even for the same mean HbA_{1c} levels.[26] Putative mechanisms include reduced exposure to elevated glucose levels, less hypoglycaemia, lower glucose and HbA_{1c} variability, the (roughly 20-30%) lower total daily insulin dose, and lower inflammation and oxidative stress. Although CGM systems are subsidised, insulin pumps are not, with only about 20% of people in Australia with T1D using pumps, which are usually funded via private health insurance.[27] Advocacy for government funding of pumps for all with T1D who desire them is ongoing.[27]

Dyslipidaemia

Dyslipidaemia is a major and very treatable risk factor, and in T1D is associated with CVD, independent of hyperglycaemia.[28,29] Unlike in people with T2D, hypertriglyceridaemia and low HDL-cholesterol (HDL-C) levels are not common, partly due to the hyperinsulinaemia of treated T1D promoting (triglyceride-rich) very-low-density lipoprotein catabolism. The major lipid involved is usually LDL-C, which, even if levels are normal, has diabetes-related qualitative changes that greatly enhance its atherogenicity.[30] Despite this, dyslipidaemia remains often undertreated in people with T1D, likely due to clinician and patient factors.[31]

LDL-C targets for people with diabetes predominantly come from evidence in trials involving people with T2D or meta-analyses of

International lipid management guidelines in diabetes: current recommendations

ADA: Cardiovascular disease and risk management: standards of care in diabetes – 2025[9]

Primary prevention

- 20–39 years of age
  - Consider statin therapy if there are additional CVD risk factors
- 40–75 years of age and no other CVD risk factors
  - Use moderate-intensity statin therapy*
- 40–75 years of age and ≥1 CVD risk factor
  - Use high-intensity statin therapy,† with an aim to reduce LDL-C by ≥50% of baseline and goal of <1.8 mmol/L
- >75 years of age
  - Reasonable to continue current statin therapy, or commence moderate-intensity statin after discussion of benefits and risks

Secondary prevention

- Use high-intensity statin therapy,† with an aim to reduce LDL-C by ≥50% of baseline and goal of <1.4 mmol/L

ESC/EAS: 2023 guidelines for the management of cardiovascular disease in patients with diabetes[34]

Primary prevention

- Moderate CV risk
  - Aim for LDL-C target of <2.6 mmol/L
- High CV risk
  - Reduce LDL-C by ≥50% of baseline and goal of <1.8 mmol/L
- Very high CV risk
  - Reduce LDL-C by ≥50% of baseline and goal of <1.4 mmol/L

Secondary prevention

- Aim for LDL-C target of <1.4 mmol/L

IPSAD: 2022 clinical practice consensus guidelines in children and adolescents[33]

- LDL-C >2.6 mmol/L
  - Dietary and lifestyle interventions
- LDL-C >3.4 mmol/L
  - Consider statin therapy

Abbreviations: ADA = American Diabetes Association; CV = cardiovascular; CVD = cardiovascular disease; ESC/EAS = European Society of Cardiology/European Atherosclerosis Society; IPSAD = International Society for Paediatric and Adolescent Diabetes; LDL-C = low-density lipoprotein-cholesterol.

* Moderate intensity statin therapy: atorvastatin 10-20 mg, rosuvastatin 5-10 mg, simvastatin 20-40 mg, pravastatin 40-80 mg, fluvastatin 80 mg.

† High intensity statin therapy: atorvastatin 40-80 mg, rosuvastatin 20-40 mg.

trials including people with T1D.[13,32] Because age and T1D are major CVD risk factors, most guidelines suggest the use of lipid-lowering drugs, irrespective of lipid levels for adults with T1D aged 40 years or above.[9] Even in children and adolescents, recent International Society for Paediatric and Adolescent Diabetes (IPSAD) guidelines recommend considering lipid-lowering therapy for youth with T1D with an LDL-C level greater than 3.4 mmol/L.[33] Generally for CVD prevention, 'lower LDL-C is better' and best is even lower LDL-C and for longer. The Box summarises recent guidelines favouring more aggressive LDL-C targets in T1D.[9,33,34]

Along with lifestyle measures, lipid-lowering medications are often needed to meet recommended targets and can be added in a stepwise

Table 3. Currently available lipid-lowering drugs in Australia[35]

Drug class	Mode of action	Available drugs on PBS	PBS criteria relevant to T1D	Lipid-lowering effects	Examples of potential side effects
Predominantly LDL-C lowering					
Statin	HMG-CoA reductase inhibitor	Once daily orally: • atorvastatin 10–80 mg • fluvastatin 80 mg • pravastatin 10–80 mg • rosuvastatin 5–40 mg • simvastatin 10–80 mg	Existent CVD or high CVD risk – e.g. diabetes + microalbuminuria; or 60 years or older; or Aboriginal or Torres Strait Islander people; or TC >5.5 mmol/L	↓ LDL-C 25–55% ↓ TGs 10–20%	Elevated LFTs, myalgia or myositis or CK rise, GI upset, increased risk of type 2 diabetes (if no diabetes)
Ezetimibe	NPC1L1 (cholesterol absorption) inhibitor	10 mg oral daily	Monotherapy or combination therapy with rosuvastatin or atorvastatin or simvastatin	↓ LDL-C 15–25%	GI upset, elevated liver enzymes, dizziness
Colestyramine (cholestyramine)	Bile acid sequestrant	4 g oral daily	PBS restriction: primary hypercholesterolaemia with GP Management Plan/Team Care Arrangement	↓ LDL-C 15–25% TGs may ↑	GI upset, impaired absorption of fat-soluble vitamins and of some drugs
Evolocumab	PCSK9 monoclonal antibody	140 mg SC injection every 2 weeks	PBS authority: • homozygous FH • heterozygous FH • non-FH with symptomatic CVD, high CV risk and LDL-C >1.8 mmol/L on maximal tolerated statin + ezetimibe	↓ LDL-C 50–60% ↓ Lp(a) 20–30%	Injection site reactions, flu-like symptoms, nasopharyngitis, back pain
Inclisiran	PCSK9 small interfering RNA	284 mg SC injection, repeat at 3 months then 6-monthly	PBS authority: • heterozygous FH • non-FH with symptomatic CVD, high CV risk and LDL-C >1.8 mmol/L on maximal tolerated statin + ezetimibe	↓ LDL-C 50% ↓ Lp(a) 20–30%	Injection site reactions, bronchitis/ nasopharyngitis
Predominantly TG lowering					
Fibrates	PPAR alpha agonist	• Fenofibrate 145 mg oral daily (or 96 mg or 48 mg as per reduced kidney function) • Gemfibrozil 600 mg oral twice daily	Existent CVD; or high CVD risk – e.g. diabetes + microalbuminuria; or 60 years or older; or Aboriginal or Torres Strait Islander people; or TC >5.5 mmol/L	↓ TGs 25–50% ↑ HDL-C 10–30%	GI symptoms, gallstones, elevated LFTs, elevated CK, higher risk of myopathy if used with statin (less if fenofibrate)
Icosapent ethyl	TG reduction, plaque stabilisation related to increase in omega 3 fatty acid levels	2 g oral twice daily	PBS authority: • established CVD + on statin + LDL-C 1.0–2.6 mmol/L + fasting triglycerides 1.7–5.6 mmol/L	↓ TGs 20%	Atrial fibrillation, GI upset, bleeding

Abbreviations: CK = creatinine kinase; CV = cardiovascular; CVD = cardiovascular disease; FH = familial hypercholesterolaemia; GI = gastrointestinal; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density. lipoprotein cholesterol; LFTs = liver function tests; Lp(a) = lipoprotein(a); NPC1L1 = Neimann-Pick C1-like 1 protein; PCSK9 = proprotein convertase subtilisin/kexin type 9; PPAR alpha = peroxisome proliferator-activated receptor alpha; SC = subcutaneous; T1D = type 1 diabetes; TC = total cholesterol; TG = triglyceride; ↑ = increases ↓ = reduces.

Table 4. Interventions to reach blood pressure target[9]

Initial BP	Intervention
>120/80 mmHg	Lifestyle intervention, including weight loss, reducing dietary sodium, smoking cessation and increased physical activity
≥130/80 mmHg and <150/90 mmHg	Lifestyle interventions + • Recommend commencing one BP agent • If albuminuria or coronary artery disease present, commence ACEi or ARB • If no albuminuria or coronary artery disease present, commence ACEi/ARB or CCB or diuretic
≥150/90 mmHg	Lifestyle interventions + • Recommend commencing two BP agents • If albuminuria or coronary artery disease present, commence ACEi/ARB plus CCB or diuretic • If no albuminuria or coronary artery disease present, commence two of ACEi/ARB, CCB and diuretic
If still not meeting a BP target of <130/80 mmHg on two agents, add a third agent from medications classes above Finally, consider adding an MRA if not meeting target on three medications and refer patient to specialist for investigation of secondary causes of hypertension	

Abbreviations: ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BP = blood pressure; CCB = calcium channel blocker; MRA = mineralocorticoid receptor antagonist.

manner. Table 3 summarises currently available lipid-lowering drugs in Australia.[35] The most commonly used LDL-C-lowering medications for CVD prevention or treatment in adults with T1D are a statin and ezetimibe, which can be used in combination, and a proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor can be used if needed.

Hypertension

Hypertension is more common in people with T1D than in those without.[15] Screening should be routine, including blood pressure (BP) measurement at each clinic visit, at the pharmacy, at home or with 24-hour BP monitoring. An elevated BP without a previous diagnosis of hypertension should be confirmed on multiple readings on separate days.[9] Again, there is an absence of high-quality data on BP targets in T1D, and so current targets mirror that of people with T2DM.[9] Although BP targets should be individualised to take into account potential adverse effects of medications, general BP targets are less than 130/80 mmHg. Table 4 summarises ADA-recommended BP targets and medications.[9]

Thrombosis prevention

Aspirin use is strongly recommended for secondary prevention in people with diabetes and CVD.[9] The evidence for aspirin use in primary prevention for people with diabetes is less clear. Aspirin appears to have a modest effect on CVD events, increasing with underlying CVD risk.[9] Its use does, however, come with increased risk of bleeding, often gastrointestinal bleeding. Therefore, current ADA guidelines only recommend the use of aspirin as primary prevention for CVD in people with diabetes at high CVD risk and only after shared decision making with patients regarding the benefits and risk of aspirin use. It has been suggested that people with diabetes have a relative 'aspirin resistance' due to platelet dysfunction, and higher doses may be necessary.[36] More research is needed before higher than usual doses of antiplatelet drugs are recommended for people with T1D.

Adjunct treatments for risk factor management

T2D drugs may also be cardioprotective in people with T1D. There has been a rise in the use of glucagon-like peptide-1 (GLP-1) receptor agonists and sodium-glucose cotransporter-2 (SGLT-2) inhibitors in people with T2D given their glucose lowering (in diabetes) and weight-loss effects and significant cardiorenal benefits. Neither has regulatory approval currently for use in people with T1D, mainly due to the risk of diabetic ketoacidosis (DKA).

Some GLP-1 receptor agonist T1D trials have shown moderate benefit in HbA_{1c} levels and weight without increased incidence of severe hypoglycaemia or DKA.[37] Further efficacy and safety studies are needed. SGLT-2 inhibitors have been linked with increased risk of genitourinary infections and DKA in people with T1D.[38,39] Continuous ketone monitoring may have a role in early ketosis detection; however, such monitors are not yet clinically available. Given their potential benefits, there may be a role for these medications in T1D, but currently we do not recommend their use in all people with T1D. Although recognising the existence of off-label use, we advise considerable caution and prescription of these agents should only be implemented in conjunction with a diabetes specialist after a detailed discussion with the patient and provision of appropriate education to minimise the DKA risk.

Conclusion

A multimodal approach needs to be taken to reduce CVD risk in people with T1D. Using T1D-validated CVD risk calculators can help quantify risk, discussions with patients and treatment decisions. Glucose management to reduce hyperglycaemia, glucose variability and hypoglycaemia is important. Likewise, management of lipid levels, blood pressure and weight and supporting nonsmoking are also key. **ET**

References

A list of references is included in the online version of this article (www.endocrinologytoday.com.au).

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