evidence is lacking regarding the efficacy of stress management interventions on the primary prevention of ASCVD and diabetes.

3. Medical and Pharmacological Therapy

Risk assessment and evaluation

3.1 In individuals identified as having metabolic risk, we recommend global assessment of 10-year risk for either CHD or ASCVD to guide the use of medical or pharmacological therapy. (1|⊕⊕⊕◯)

Technical remark: Global risk assessment includes the use of one of the established cardiovascular risk equations. Elevated LDL is indicative of cardiovascular risk.

Evidence

Several methodologies have been developed to assess 10-year cardiovascular risk status (115–117). The Framingham Heart Study CVD 10-year risk calculator (Framingham risk calculator) is a sex-specific algorithm that predicts the 10-year risk of having any cardiovascular event. The PROCAM risk algorithm calculates the risk of MI based on the 10-year follow-up of the PROCAM study. The Systematic Coronary Risk Evaluation (SCORE) risk charts, the European cardiovascular disease risk assessment model, are based on the results of 12 European cohort studies. These cardiovascular risk charts are based on sex, age, total cholesterol, systolic BP, and smoking status and are used to predict death due to MI. More recently, as part of two ACC/AHA guidelines that addressed assessment and treatment of cardiovascular risk (30, 118), a pooled cohort equation approach was developed (119, 120).

In comparison studies of the various guidelines, several reports have found that the pooled cohort equation offers better discrimination and net benefit regarding cardiovascular risk assessment. Although this method carries a risk for overestimation of risk, it was identified as the best available methodology to date by the US Preventive Service Task Force (121–124). Collectively, these risk calculation methods use easy-to-collect clinical parameters, such as age, use of cigarettes, BP, and serum lipid levels. Other risk calculators that are less widely used have also been published. The United Kingdom Prospective Diabetes Study risk engine has been developed with validated ASCVD risk estimates for people with T2DM (125, 126), although the population with previously diagnosed diabetes is outside the framework of the primary prevention population considered in this guideline. As other authors have published findings comparing the different algorithms, this Writing Committee made no attempt to undertake such comparisons among different population groups (127, 128). The Writing Committee recommends that 10-year risk for CHD be assessed for individuals using published algorithms that best pertain to the individuals from a particular population group. Clinical judgment or national or regional recommendations can be used for making these assessments.

3.2 In individuals with LDL-C ≥190 mg/dL (4.9 mmol/L) or TGLs ≥500 mg/dL (<5.6 mmol/L), we recommend that, before considering the diagnosis of primary hyperlipidemia, practitioners should rule out secondary causes of hyperlipidemia. If a secondary cause can be excluded, primary hyperlipidemia should be suspected. (1|⊕⊕⊕◯)

Technical remark: Examples of secondary causes of hyperlipidemia include: untreated hypothyroidism, nephrotic syndrome, renal failure, cholestasis, acute pancreatitis, pregnancy, polycystic ovarian disease, excess alcohol use, treatment with estrogens/oral contraceptives, antipsychotic agents, glucocorticoids, cyclosporine, protease inhibitors, retinoids, and beta blockers.

Evidence

Elevated values of LDL-C (≥190 mg/dL) or TGLs (≥500 mg/dL) are likely to be due to primary hyperlipidemia from a genetic cause (known or unknown). However, before considering the diagnosis of primary hyperlipidemia, secondary causes of hyperlipidemia need to be ruled out. Because secondary hyperlipidemia is not rare, evaluation of underlying conditions that could be causing or exacerbating dyslipidemias is necessary before initiating or intensifying treatment as recommended in current guidelines (129–131). Secondary hyperlipidemia can occur as an isolated cholesterol elevation, an isolated TGL elevation, or as a combined pattern. Increased LDL-C levels (>190 mg/dL), high TGL levels (>500 mg/dL), xanthomas, a strong family history of hyperlipidemia, or a lack of an expected response to maximal therapeutic doses of lipid-lowering agents indicate primary hyperlipidemia. Secondary hyperlipidemia may result from different metabolic and endocrine conditions, liver or kidney disease, HIV, drug therapy, or dietary factors. As dyslipidemia may be an early indication, recognizing an underlying condition may alter subsequent treatment decisions. Furthermore, some dyslipidemias can appear to be refractory to drug treatment in the presence of an ongoing unrecognized secondary cause. For example,