**D-15 HYPERLIPIDEMIA**

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Hyperlipidemia is a primary, major risk factor for ASCVD and may even be a prerequisite for ASCVD. Epidemiologic data also suggests that hypercholesterolemia and perhaps coronary atherosclerosis are risk factors for ischemic cerebrovascular accident (CVA), (TG) and low-density lipoprotein cholesterol (LDL-C) and a decreased concentration of high-density lipoprotein cholesterol (HDL-C), as an important risk factor for peripheral vascular disease, CVA, and ASCVD. A comprehensive strategy to control lipid levels and address associated metabolic abnormalities and modifiable risk factors is recommended primarily for lifestyle changes (Physical Activity, Medical Nutrition Therapy and Smoking Cessation) and patient education with pharmacotherapy as needed to achieve evidence-based targets. Statin therapy is recommended as the primary pharmacologic agent to achieve target LDL-C goals on the basis of morbidity and mortality outcome trials. In individuals within high-risk and very high-risk categories, further lowering of LDL-C beyond established targets with statistical results in additional ASCVD event reduction and may be considered. Combination therapy of lipid-lowering agents should be considered when the LDL-C / non-HDL-C level is markedly increased and monotherapy (usually with a statin) does not achieve the therapeutic goal. Ezetimibe and propionate convertase subtilisin / kinase type 9 (PCSK9) inhibitors (alirocumab, evolocumab) can be used in combination with statins to reduce LDL-C and ASCVD risk. In statin-intolerant patients Ezetimibe or PCSK9 inhibitors can be used as monotherapy. Fibrates should be used to treat severe hypertriglyceridemia (TG> 500 mg/dL). Fibrates may improve ASCVD outcomes only in primary and secondary prevention when TG concentrations ≥200 mg/dl and HDL-C concentrations <40 mg/dl. Microsomal Transfer Triglyceride Protein (MTP) inhibitor (lomitapide) and anti-sense Apolipoprotein B oligonucleotide ( mipomersen-subQ injection) are other treatment options for homozygous familial hypercholesterolemia.

**D-16 LABORATORY APPROACH TO DYSLIPIDEMIA**

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Standard lipid analysis includes measurement of fasting plasma or serum total cholesterol (TC), triglyceride (TG), low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C). Non–HDL-C (TC–HDL-C) reflects the amount of total atherogenic particles in plasma. The 2017 Turkish Endocrinology and Metabolism Association guidelines for diagnosis and treatment of metabolic lipid disorders indicate that non–HDL-C is a better predictor than LDL-C when determining cardiovascular risk in high TG patients with Diabetes, Metabolic Syndrome and Chronic Kidney Disease. Measurement of nonfasting non–HDL-C is useful. When nonfasting non–HDL-C concentrations are greater than 220 mg/dL or triglyceride levels are higher than 500 mg/dL a possible underlying genetic disorder needs to be inquired. Measurement of small dense LDL-C (sdLDL-C), lipoprotein a (Lp(a)), lipoprotein-associates phospholipase A2 (Lp-PLA2), LDL/HDL particle number and concentration can add significant information to the standard lipid profile regarding cardiovascular disease (CVD) risk. Lp(a) increase, combined hyperlipidemia, and dyslipidemia can be seen in familial lipid disorders associated with premature CVD. Plasma fatty acid analysis is beneficial to assess adequacy of omega-3 fatty acid intake and gain information on excess levels of circulating saturated and trans fatty acids. Plasma sterol measurements can be helpful for the diagnosis of diseases associated with very low density lipoprotein cholesterol (VLDL-C) levels over 50 mg/dL and/or LDL-C levels above 160 mg/dL. Elevated levels of lathosterol, β-sitosterol and cholesterol can be observed in Familial Combined Hyperlipidemia, Phytosterolemia and Cerebrotendinous Xanthomatosis, respectively. Assessment of apolipoprotein (apo) A-I in HDL particles by gel electrophoresis is important to determine CVD risk. HDL-C is a better predictor than LDL-C for ASCVD. Measurement of apoB is valuable for the diagnosis of abetalipoproteinemia and hypobetalipoproteinemia. Lechitin cholesteryl acyltransferase (LCAT), hepatic lipase and cholesteryl ester transfer protein (CETP) deficiency can be verified by evaluating associated protein levels and activity. Definitive diagnosis of dyslipidemias caused by genetic disorders and causes of markedly elevated triglycerides (> 1,000 mg/dl) requires next generation DNA sequencing of the appropriate and relevant genes. This can provide a molecular diagnosis to formulate optimal therapy strategies.

**D-17 EXERCISE AND BIOMARKERS IN DYSLIPIDEMIA**

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Cardiovascular diseases are the leading cause of mortality continues to be all over the world. Dyslipidemia is found in some diseases such as diabetes mellitus type 2, and elevated triglycerides. Dyslipidemia is an important cardiovascular risk factor and modifiable with life style management. Both primary prevention and secondary prevention with exercise training has been shown to decrease the development of cardiovascular events. The risk scoring of patients is one of the most important points in the treatment of dyslipidemia. Individuals must be separated according to risk scores for cardiovascular diseases and dyslipidemia approach should be planned accordingly this risk scoring. Many research demonstrated that aerobic exercise combined with weight loss significantly reduces blood cholesterol, low density lipoprotein cholesterol(LDL-C), very low density lipoprotein cholesterol (VLDL-C), and triglycerides(TG) while improving high density lipoprotein cholesterol(HDL-C). Aerobic and resistance exercise training shown to decrease in non HDL-C independent of changes in body weight. The benefit effects of single session of aerobic exercise are observed for postprandial lipemia. Acute and chronic exercise trainings have been pointed as important management to counteracts both dyslipidemia symptoms and systemic inflammation. Physical activity has been recommended in the prevention and treatment of the chronic inflammatory diseases such as dyslipidemia. American Association of Clinical Endocrinologist recommended that exercise programs should include at least 30 minutes of moderate intensity physical activity 4 to 6 times weekly, with an expenditure of at least 200kcal/day. Exercise training is cost effective tool and can cause fewer side effects than isolated medicine in dyslipidemia.

**D-18 GENERATIONS ARE DIFFERENT, WHAT ABOUT MOTIVATION?**

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Motivation is a phenomenon that can only be understood by interpreting the behavior of people and motivation factors can be common in generations. Groups that share birth date and important moments at critical developmental stages are called “generations”. Generations which categorised differently in the literature today mostly classified as Silent, Baby Boomer and Millennials including X and Y generations. The most important things that motivate loyal and consistent Silent generation who desire for stability and foreseeing career steps are assurance and status. The motivating things for baby boomer workers, born into post-war social turmoil and problematic with authority are money, senior position and individual development. X Generation tries to achieve more business / private life balance in career management, In their motivations, training and conferences will help them build business relationships; group work rather than individual work, the opportunities offered in addition to salaries are influential. The Millennium Generations includes young people who are