
“Endocrine and Metabolic Diseases Biomarkers From Diagnostic to Therapy”

Obesity related insulin elevation causes lowered steroid-hormone-binding globulin (SHBG) level. Decreased SHBG leads to increased free estradiol and androgens and these hormones are then available to stimulate the growth of estrogen and androgen receptor expressing breast and prostate cancers.

D-11 BIOCHEMISTRY OF METABOLIC SYNDROME AND EXPERIMENTAL ANIMAL MODELS

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Increases in metabolic syndrome incidence depending on dietary habits are observed in recent years. Role of fructose containing drinks in the increase of metabolic syndrome incidence is inevitable. For this reason fructose induced metabolic syndrome models are generated in order to reveal the underlying mechanisms of metabolic syndrome and to develop treatment models. Hence, I will try to express the metabolic syndrome models and the formation of biochemicals of metabolic syndromes in the light of current knowledge in this presentation.

D-12 THE BIOMARKERS OF OSTEOPOROSIS AND METABOLIC BONE DISEASE

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Osteoporosis is a chronic disease and increases the risk of fragility fractures which is a very important social and economic problem in many countries of the world. Early diagnosis of reduced bone mass or osteoporosis and fracture prevention treatment is important. An ideal examination of diagnosis and follow-up method of osteoporosis should indicate both loss of bone mass and fracture risk. Biochemical markers of bone turnover are non-invasive and efficient tool to evaluate bone diseases. Osteoelastic and osteoclastic ratio of bone matrix can be detected by either measuring leading active enzymes of bone forming and resorbing cells or measuring bone matrix components which released to circulation during bone remodelling. These markers have advantages in bone cycle such as being unexpensive, non-invasive, reusable and to be able to show bone cell activity. But the disadvantages of these markers are inequality of sensitivity and specificity and lack of fully research of some markers. As a result, in this speech we plan to make an assessment about bone biomarkers which is used to measure drug efficacy only to help bone mineral density in studies, is seen in articles about bone, is measured almost only for research, doesn’t exist in routine laboratory tests and is reached only by some academic personnel.

D-13 LABORATORY OVERVIEW OF BONE MARKERS

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Bone remodeling is characterized by temporal and spatial coupling of bone formation and resorption that is necessary for normal bone structure maintenance and skeletal growth. A wide range of biochemical markers provide information on bone cells known as bone turnover markers (BTM) which can be divided as markers of bone resorption and formation. The measurement of BTM can reflect either enzymatic activities characteristic of the bone-forming (alkaline phosphatase), or resorbing cells or bone matrix components released into circulation during resorption (collagen type I telopeptides). Although different assays for many markers have been adapted to automated biochemical analyzers making them rapid and cost-effective in clinical laboratories, none of the currently available bone markers have shown to be advantageous over others with regard to their clinical utility. The recent report of Joint Working Group of International Foundation of Osteoporosis (IOF) and International Federation of Clinical Chemistry on Standardization of Bone Turnover Markers recommend; one bone formation marker (serum PINP) and one bone resorption marker (serum CTx) to be measured by standardized assays for the prediction of fracture risk and monitoring of osteoporosis treatment in adults. To addresses the limitations of variability IOF and National Bone Health Alliance have implemented different complimentary activities around the harmonization and the use of all BTMs. However all those traditional BTMs have been used for years to decide the fracture risk prediction and largely for treatment monitoring that show earlier changes following the beginning of treatment allowing useful measurements to be observed about 1 to 3 months. Nowadays there has been a new approach which bases on our understanding of bone metabolism. Related with that peristin, cathepsin-K, sclerostin, dkk3-opostin, RANKL, FGF-23/lothio/ostecalcin, soggino-1-phosphate and microRNAs are considered as new biomarkers. Also the clinical use of those biochemical markers has not been fully established, their relationship with fracture risk has still have question marks and their use as treatment monitoring tools needs to be studied. Why we are working on them, as all those new mentioned markers can tell us about the osteocyte activities and distinguish the bone compartments that they might be helpful for exploring the physiological and pathological links between the bone and other organs, and to monitor systemic diseases.

D-14 EFFECT OF EXERCISE ON LABORATORY RESULTS IN OSTEOPOROSIS

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Osteoporosis is an osteometabolic disease which increases the risk of fracture and impairs bone quality and microarchitecture of the bone tissue is characterized by the loss of bone mass. Although osteoporosis has different classifications based on the age of a person, localization of the disorder, bone tissue involved, etiology and histological appearance, it is increasingly becoming widespread in both females and males. Although sex hormones are often reported to be effective in the development of osteoporosis, menopause and lack of physical activity are also important risk factors. Physical activity affects the bone structure by mechanical forces directly, and by hormonal factors indirectly. Mechanical forces lead to the preservation of bone mass during a physical activity by activating ground reaction forces with the contractile activity of the muscles. It is important to maintain bone health through appropriate exercise approaches, in which the type, density, frequency, and length of the activity are adjusted. Exercise activates osteocytes which lead to the release of growth factors and cytokines, resulting in increased osteoblastic activity. The measurement of bone biomarkers is important in identifying the responses of bone cells to exercise. Serum bone alkaline phosphatase (B-ALP) and serum osteocalcin indicate new syntheses in bone. After appropriate exercise, biochemical markers increase significantly. Long-term moderate intensity exercise affects the production of osteoclastogenic and antiosteoclastogenic cytokines by promoting the formation of peripheral blood mononuclear cells. Exercise exerts its effect by changing the balance between osteoclastogenic cytokines and antiosteoclastogenic cytokines. With the formation of hematopoietic cells around the bone, similar changes occur at the micro level. These affect osteoclasts which promote bone resorption, osteoblasts which are responsible for bone formation and autogenesis by producing bone-loader signals. Bone biomarkers indicate that exercise plays an important role in bone health.

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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. 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 / kevin 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 are >200 mg/dL and HDL-C concentrations <40 mg/dL. Micronal 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 geniic disorder needs to be inquired. Measurement of small dense LDL-C (sLDL-C), lipoprotein a [Lp(a)], lipoprotein-associated 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, α-sitosterol and cholesterol can be observed in Familial Combined Hyperlipidemia, Phytosterolemia and Cerebrotendinous Xanthomatosis, respectively. Assessment of apoprotein (apo) A-I in HDL particles by gel electrophoresis is important to determine CVD risk. HDL-C and Lp(a) levels are useful in HDL-C deficiency (HDL-C <20 mg/dL) due to apoA-I deficiency and Tangier disease. 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 obesity, type 2 diabetes mellitus and coronary artery diseases. 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 recomended in the prevention and treatment of the chronic inflammatur diseases such as dyslipidemia. American Association of Clinical Endocrinologist recommended that exercise programs should include at least 30 minutes of moderates intensity physical activity 4to 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 medication in dyslipidemia.