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ADULT DYSLIPIDIMIA

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Abstract: A significant worldwide health issue, adult dyslipidemia is defined by abnormal serum lipid levels, such as elevated low-density lipoprotein cholesterol (LDL-C), elevated triglycerides, or decreased highdensity lipoprotein cholesterol (HDL-C). It is commonly acknowledged as one of the most potent modifiable risk factors for atherosclerotic cardiovascular disease (ASCVD), which continues to be the primary cause of morbidity and mortality globally. Numerous research, such as those by Grundy et al. (2019) and Zhang et al. (2020), have shown that adult dyslipidemia is highly prevalent and on the rise, especially in nations where sedentary lifestyles, fast urbanization, and dietary changes are happening. Unhealthy eating habits, physical obesity, smoking, excessive alcohol use, genetic predispositions such familial inactivity, hypercholesterolemia, and metabolic diseases like diabetes mellitus are important risk factors. Ross's pathophysiological evidence

Index Terms - Component, formatting, style, styling, insert.

I. Introduction

Dyslipidemia is a medical condition characterized by abnormal concentrations of lipids in the blood, including elevated levels of low-density lipoprotein cholesterol (LDL-C), total cholesterol, and triglycerides (TG), as well as reduced levels of high-density lipoprotein cholesterol (HDL-C) (Grundy et al., 2019). It is a critical modifiable risk factor for atherosclerotic cardiovascular disease (ASCVD), which includes coronary artery disease, cerebrovascular disease, and peripheral artery disease. Globally, cardiovascular diseases remain the leading cause of morbidity and mortality, and dyslipidemia is recognized as a central contributor to this health burden (Murray & Lopez, 2017; Silverman et al., 2016).

Epidemiological studies indicate that the prevalence of dyslipidemia among adults ranges from 30% to 50%, depending on geographic region, age, sex, and lifestyle factors (Zhang et al., 2020). The condition is increasing in low- and middle-income countries due to urbanization, dietary transitions toward high-calorie, high-fat foods, and reduced physical activity (Narayan et al., 2019). Dyslipidemia can occur in isolation or in combination with other metabolic disorders, such as obesity, diabetes mellitus, and hypertension, which together amplify cardiovascular risk (Reaven, 1988; Ginsberg, 2000).

The pathophysiology of dyslipidemia involves complex interactions between genetic, metabolic, and environmental factors. Elevated LDL-C contributes to endothelial dysfunction, inflammation, and plaque formation in arterial walls, while low HDL-C impairs reverse cholesterol transport, reducing the clearance of excess cholesterol from peripheral tissues (Ross, 1999; Libby, 2013; Barter & Rye, 2006). Triglyceride-rich lipoproteins further exacerbate atherogenesis by promoting small dense LDL particles and endothelial injury (Nordestgaard, 2016).

Despite its high prevalence and serious health consequences, dyslipidemia is often underdiagnosed and undertreated. Guidelines recommend routine screening for adults, especially those with risk factors such as diabetes, obesity, smoking, and a family history of cardiovascular disease (Grundy et al., 2018; Sniderman et al., 2012).



PATHOPHYSIOLOGY OF ADULT DYSLIPIDEMIA:

Adult dyslipidemia involves abnormal lipid levels, primarily elevated low-density lipoprotein (LDL) cholesterol, triglycerides, or reduced high-density lipoprotein (HDL) cholesterol, driven by disruptions in lipid metabolism, transport, and clearance. These imbalances stem from genetic factors in primary forms or acquired influences like insulin resistance and oxidative stress in secondary forms, promoting atherosclerosis. **Key Mechanisms:**

Lipids such as cholesterol and triglycerides are transported via lipoproteins (LDL, HDL, VLDL), but impaired synthesis, secretion, or clearance leads to accumulation. In insulin resistance or metabolic syndrome, excess free fatty acids from adipose tissue increase hepatic VLDL production and reduce lipoprotein lipase activity, raising triglycerides and small dense LDL while lowering HDL. Genetic mutations, as in familial hypercholesterolemia, impair LDL receptor function, causing persistent high LDL levels.

Role in Atherosclerosis:

Oxidized LDL (oxLDL) particles trigger endothelial dysfunction by promoting reactive oxygen species (ROS) production, monocyte adhesion, and infiltration into arterial walls. Macrophages engulf oxLDL via scavenger receptors, forming foam cells that initiate fatty streaks and atherosclerotic plaques, eventually leading to cardiovascular events. Chronic inflammation and plaque instability exacerbate risks in dyslipidemic adults. According to Richardson and smith(1993) to make the model more effective and efficient the selection criteria for the shares in the period are: Shares with no missing values in the period, Shares with adjusted R² < 0 or F significant (p-value) >0.05of the first pass regression of the excess returns on the market risk premium are excluded. And Shares are grouped by alphabetic order into group of 30 individual securities (Roll and Ross, 1980).

RISK FACTOR OF ADULT DYSLIPIDEMIA:

Modifiable Risk Factors

- 1. Obesity and abdominal obesity increase free fatty acid release, elevating triglycerides and LDL.
- 2. Sedentary lifestyle and lack of exercise reduce HDL cholesterol levels.

Non-Modifiable Risk Factors

- 1. Older age correlates with declining lipid clearance mechanisms.
- 2. Family history of dyslipidemia or premature cardiovascular disease indicates genetic predisposition.

Associated Medical Conditions

- 1. Type 2 diabetes and insulin resistance impair lipoprotein lipase activity.
- 2. Hypertension and metabolic syndrome amplify cardiovascular risk alongside dyslipidemia.

CLINICAL SIGNIFICANCE OF ADULT DYSLIPIDEMIA:

Adult dyslipidemia significantly elevates the risk of atherosclerotic cardiovascular disease (ASCVD), including coronary heart disease, myocardial infarction, stroke, and peripheral artery disease, due to lipid-driven plaque formation.

Cardiovascular Risks:

Elevated LDL cholesterol promotes atherosclerosis by oxidizing and infiltrating arterial walls, forming plaques that can rupture and cause acute events like unstable angina or death. Low HDL cholesterol impairs reverse cholesterol transport and anti-inflammatory effects, independently increasing coronary risk by 2-3% per 1 mg/dL decrease. High triglycerides contribute to small dense LDL particles and remnant lipoproteins, raising ASCVD risk by up to 72% in elevated ranges.

Clinical Impact:

Dyslipidemia accelerates plaque burden and inflammation (e.g., via hsCRP and IL-6), leading to higher morbidity and mortality in cardiac patients. In high-risk groups like those with diabetes or metabolic syndrome, combined lipid abnormalities amplify event rates, with studies showing 17-39% higher risks for major CV events. Early intervention reduces coronary events by 34-37% through lipid lowering.

DIAGNOSIS OF ADULT DYSLIPIDEMIA:

Diagnosis of adult dyslipidemia begins with a comprehensive clinical history assessing family history of premature cardiovascular disease, lifestyle factors, and secondary causes like diabetes or hypothyroidism, followed by a fasting lipid panel (9-12 hours) measuring total cholesterol (TC), LDL-C, HDL-C, non-HDL-C, triglycerides (TG), and optionally apolipoprotein B (apoB) and lipoprotein(a) [Lp(a)] once in a lifetime. Non-fasting profiles suffice for initial screening in low-risk adults, but fasting is essential if TG >400 mg/dL (prevents inaccurate Friedewald LDL-C calculation) or for confirmation. Repeat testing confirms persistence, with targets varying by risk: LDL-C <100 mg/dL optimal generally, <70 mg/dL for high-risk (e.g., ASCVD), HDL-C >40 mg/dL (men)/>50 mg/dL (women), TG <150 mg/dL

The diagnosis of adult dyslipidemia primarily relies on a fasting lipid profile test that measures total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides. LDL cholesterol is often calculated using the Friedewald formula unless triglycerides exceed 400 mg/dL, in which case direct measurement is necessary. The diagnosis is confirmed when lipid levels deviate from established normal ranges, such as total cholesterol above 200 mg/dL, LDL cholesterol above

100 mg/dL, HDL cholesterol below 40 mg/dL (men) or 50 mg/dL (women), and triglycerides above 150 mg/dL.

GOALS OF TREATMENT OF ADULT DYSKIPIDEMIA:

Primary Goals:

risk.

The main goals of treating adult dyslipidemia focus on reducing low-density lipoprotein cholesterol (LDL- C) levels to minimize atherosclerotic cardiovascular disease (ASCVD) risk, with targets varying by patient risk category. High-risk patients, such as those with established ASCVD or diabetes, aim for LDL-C <55-70 mg/dL or a ≥50% reduction from baseline, while very high-risk individuals target <55 mg/dL with further intensification if >40 mg/dL persists. Secondary objectives include non-HDL-C <85-100 mg/dL, apolipoprotein B (ApoB) <80-100 mg/dL, and triglycerides <150 mg/dL to address residual

Risk-Stratified Targets:

Very high risk (ASCVD, familial hypercholesterolemia with events): LDL-C <55 mg/dL; reassesss if >40 mg/dL on maximal therapy.

High risk (diabetes >20 years, multiple risk factors, chronic kidney disease):

Moderate risk (10-year ASCVD risk 7.5-19.9%): LDL-C <100 mg/dL; intensify if >70-100 mg/dL. Low risk: LDL-C <130 mg/dL, with lifestyle focus unless other enhancers present.

Additional Lipid Goals:

HDL-C goals emphasize raising levels above 40 mg/dL (men) or 50 mg/dL (women) through lifestyle, though pharmacologic targets are less emphasized. For elevated triglycerides (150-499 mg/dL), prioritize statin therapy first, then add agents like icosapent ethyl if ≥150 mg/dL persists in high-risk cases.

Overall Objectives:

Ultimate aims extend beyond lipids to reduce major ASCVD events (e.g., myocardial infarction, stroke) by 20-25% per 39 mg/dL LDL-C drop, alongside addressing comorbidities and improving quality of life. Guidelines prioritize thresholds for intensification over rigid targets for practical management

MANAGMENT OF ADULT DYSLIPIDEMIA:

Management of adult dyslipidemia involves a combination of lifestyle changes and pharmacologic interventions aimed at reducing cardiovascular risk by improving lipid profiles.

Lifestyle Modification

- 1. Dietary changes: Adopt a heart-healthy diet low in saturated fats (<7% of total calories), trans fats, and cholesterol; increase intake of fiber, fruits, vegetables, and omega-3 fatty acids.
- 2. Physical activity: Engage in moderate-intensity aerobic exercise (e.g., brisk walking) for at least 150 minutes per week.
- 3. Weight management: Achieve and maintain a healthy body weight.

Pharmacological Treatment

Pharmacologic therapy is recommended based on cardiovascular risk stratification and lipid levels:

- 1. Statins (HMG-CoA reductase inhibitors) are the first-line treatment, effectively lowering LDL cholesterol by 20-50% and reducing cardiovascular events. Therapy should be initiated in patients with atherosclerotic cardiovascular disease (ASCVD), LDL-C≥190 mg/dL, or diabetes with LDL-C >70 mg/dL, or intermediate-to-high 10-year cardiovascular risk.
- 2. Ezetimibe, a cholesterol absorption inhibitor, is added when statin monotherapy does not achieve LDL-C goals or in statin-intolerant patients.
- 3. PCSK9 inhibitors (e.g., evolocumab, alirocumab) are recommended for patients with ASCVD or familial hypercholesterolemia who have persistently elevated LDL-C despite maximally tolerated statin and ezetimibe therapy.

CONCLUSION:

Adult dyslipidemia is a prevalent metabolic disorder characterized by abnormal lipid levels, significantly increasing the risk of cardiovascular diseases such as coronary artery disease and stroke. Its management requires a comprehensive approach combining lifestyle modifications—including a heart-healthy diet, regular physical activity, weight management, smoking cessation, and moderation of alcohol intake—with pharmacological treatments tailored to individual risk profiles. Statins remain the cornerstone of drug therapy, effectively reducing LDL cholesterol and cardiovascular events, while other agents like ezetimibe, PCSK9 inhibitors, fibrates, and omega-3 fatty acids provide options for patients with persistent or complex lipid abnormalities.

The primary goal is to achieve lipid targets that correspond to the patient's cardiovascular risk, aiming to lower LDL cholesterol as much as safely possible to reduce atherosclerosis progression and prevent major adverse cardiovascular events. Secondary goals include improving HDL cholesterol and managing elevated triglycerides, along with controlling comorbid conditions that exacerbate dyslipidemia. Regular monitoring and risk assessment are critical to individualize therapy and ensure adherence.

Overall, adult dyslipidemia requires early identification and persistent management to effectively reduce morbidity and mortality, emphasizing prevention through lifestyle and precision medicine approaches guided by evolving clinical guidelines.

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