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Commentary

A Brief Note on Hyperlipidemia

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DESCRIPTION

Hyperlipidemia is defined as unusually high blood levels of lipids (fats, cholesterol, or triglycerides) or lipoproteins. Hyperlipidemia is a phrase that refers to a laboratory finding as well as an umbrella term that encompasses a variety of acquired or genetic illnesses that result in that finding. A subset of dyslipidemia, hyperlipidemia is a superset of hypercholesterolemia. Hyperlipidemia is usually a chronic condition that necessitates long-term therapy to keep blood lipid levels under control.

A protein capsule transports lipids (water-insoluble compounds). The density of a capsule (or lipoprotein) is determined by its size. The particle's fate and metabolic impact are determined by the density of lipoproteins and the type of apolipoproteins it contains. There are two forms of hyperlipidemia: primary and secondary. Primary hyperlipidemia is caused by genetic factors (for example, a mutation in a receptor protein), whereas secondary hyperlipidemia is caused by extrinsic factors such as diabetes. Because of their impact on atherosclerosis, lipid and lipoprotein abnormalities are frequent in the general population and are considered modifiable risk factors for cardiovascular disease. Furthermore, some types may put you at risk for acute pancreatitis [1].

Hyperlipidemias are classed as familial (also known as primary) when caused by specific genetic defects, or acquired (also known as secondary) when caused by another underlying illness that causes changes in plasma lipid and lipoprotein metabolism. Hyperlipidemia can also be idiopathic, meaning it has no known aetiology.

Eruptive xanthomata and stomach colic are common symptoms of type I hyperlipoproteinemia in children. Retinal vein blockage, acute pancreatitis, steatosis, organomegaly, and lipemia retinalis are all complications. Hyperlipoproteinemia type II is further classified into types IIa and IIb, depending mainly on whether elevation in the triglyceride level occurs in addition to LDL cholesterol.

This may be sporadic (due to dietary factors), polygenic, or truly familial as a result of a mutation either in the LDL receptor gene on chromosome 19 (0.2% of the population) or the ApoB gene (0.2%). The familial form is characterized by tendon xanthoma, xanthelasma, and premature cardiovascular disease. The incidence of this disease is about one in 500 for heterozygotes, and one in 1,000,000 for homozygotes. HLPIIa is a rare genetic disorder characterised by elevated LDL cholesterol levels in the blood due to a lack of LDL particle uptake (no Apo B receptors). However, among the numerous hyperlipoproteinemias, this illness is the second most prevalent, with one in every 500 people having a heterozygotic predisposition and one in every million having a homozygotic tendency [2]. These individuals may present with a unique set of physical characteristics such as xanthelasmas (yellow deposits of fat underneath the skin often presenting in the nasal portion of the eye), tendon and tuberous xanthomas, arcus juvenilis (the graying of the eye often characterized in older individuals), arterial bruits, claudication, and of course atherosclerosis. Laboratory findings for these individuals are significant for total serum cholesterol levels two to three times greater than normal, as well as increased LDL cholesterol, but their triglycerides and VLDL values fall in the normal ranges.

Taking extreme measures to control people with HLPIIa may be necessary, especially if their HDL cholesterol levels are less than 30 mg/dL and their LDL cholesterol levels are greater than 160 mg/dL. For these people, a healthy diet should limit total fat to less than 30% of total calories, with a 1:1:1 ratio of monounsaturated:polyunsaturated:saturated fat. Cholesterol intake should be reduced to less than 300 mg per day, with animal products avoided, and fibre intake increased to more than 20 g per day, with 6 g of soluble fibre per day. Exercise should be encouraged because it can help to raise HDL levels. In the worst-case scenario, uncontrolled and untreated individuals may die before they reach the age of 20, but if a prudent diet is combined with proper medical intervention, the individual may see an increased incidence of xanthomas with each decade, as well as Achilles tendinitis and accelerated atherosclerosis [3].

Hypercholesterolemia (8–12 mmol/L), hypertriglyceridemia (5–20 mmol/L), a normal ApoB levels, and two types of skin symptoms are all connected with it (palmar xanthomata or orange discoloration of skin creases, and tuberoeruptive

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xanthomata on the elbows and knees). It is characterised by the beginning of cardiovascular and peripheral vascular illness at an early age. The ApoE receptor, which is ordinarily necessary for the elimination of chylomicron remnants and IDL from the circulation, malfunctions, resulting in remnant hyperlipidemia. Because of the receptor deficiency, blood levels of chylomicron remnants and IDL are greater than usual. An autosomal recessive mutation or polymorphism causes the receptor deficiency.

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