INTERNATIONAL MULTIDISCIPLINARY JOURNAL FOR RESEARCH & DEVELOPMENT
SJIF 2019: 5.222 2020: 5.552 2021: 5.637 2022:5.479 2023:6.563
eISSN 2394-6334 https://www.ijmrd.in/index.php/imjrd Volume 10, issue 10 (2023)

THE MAIN FACTORS THAT CAUSE ATHEROSCLEROSIS

Tojiddinov Xusniddin Saloxiddinovich

Andijan State Medical Institute

Abstract: Atherosclerosis is a complicated sickness characterised by way of the accumulation of plaque in the arterial walls. This buildup restricts blood flow, main to a number of cardiovascular complications. Understanding the fundamental elements that make a contribution to the improvement of atherosclerosis is quintessential in devising advantageous prevention and cure strategies. Several chance factors, ranging from way of life preferences to genetic predispositions, have been recognized and appreciably studied. This article objectives to delve into the key elements that play a massive function in the pathogenesis of atherosclerosis.

Keywords: Mankkeberg, lipids, plaque, elements, fibrotic thickness, resistance.

Introduction: Atherosclerosis is the most frequent structure of arteriosclerosis. This is a frequent time period for countless illnesses that motive thickening and loss of elasticity in the arterial walls. This sickness is additionally the most serious and clinically full-size structure of arteriosclerosis. Because it motives coronary heart ischemia and cerebrovascular diseases. Non-atheromatous types of arteriosclerosis encompass Mankkeberg arteriosclerosis and arteriolosclerosis.

Atherosclerosis disorder statistics

This disorder is a main purpose of morbidity and mortality in the United States and most developed countries. Age-related mortality due to atherosclerosis has reduced in current years. But in 2016, cardiovascular diseases, generally atherosclerosis of coronary arteries and cerebral vessels, triggered 18 million deaths worldwide.

This is 30% of all deaths. In 2016, 800,000 human beings died from cardiovascular sickness in the United States. This corresponds to nearly each 0.33 death. The occurrence of atherosclerosis is growing in creating countries.

Because human beings stay longer in developed countries, and as a result, such ailments increase. Atherosclerosis is turning into the major purpose of dying worldwide.

Atherosclerotic plaque is a attribute characteristic of atherosclerosis; it develops from fatty tissue and has three vital components:

Lipids

Inflammation and clean muscle cells

Connective tissue matrix that might also incorporate a variety of degrees of calcium deposits and blood clots

Atherosclerotic plaque formation

All degrees of the disease—from plaque formation and increase to complications—are seen as an inflammatory response to injury by using particular cytokines. Endothelial injury is regarded to play a main role.

Non-laminar or turbulent blood waft (for example, where arterial bushes branch) leads to endothelial dysfunction. Also, it stops the endothelial manufacturing of nitric oxide, which is a sturdy vasodilator and anti-inflammatory factor. This blood waft motives endothelial cells to produce adhesion molecules that entice and bind inflammatory cells.

Risk elements for sickness (eg, dyslipidemia, diabetes, smoking, arterial hypertension), oxidative stressors (eg, superoxide radicals), angiotensin II, and systemic contamination or irritation attenuate nitric oxide release, and adhesion molecules, inflammatory cytokines, hemotaxis stimulates the formation of proteins and vasoconstrictor substances; extra unique mechanisms are unknown. As a result, monocytes and T cells are constant in the endothelium. These cells migrate to the subendothelial area and the neighborhood vascular inflammatory response begins.

Monocytes in the subendothelium end up macrophages. Blood lipids, specifically low-density lipoprotein (LDL) ldl cholesterol and very low-density lipoprotein (VLDL) cholesterol, bind to endothelial cells and are oxidized in the subendothelial space. Uptake of oxidized lipids and conversion of macrophages into lipid-filled foam cells is a regular atherosclerotic impact (so-called fatty streaks). Degradation of erythrocyte membranes prompted by using vasa vasorum rupture and intraplaque hemorrhage may also be an necessary extra supply of intraplaque lipids.

Macrophages secrete proinflammatory cytokines. This motives the migration of easy muscle cells from the environment. As a result, it attracts macrophages and stimulates their growth. Various elements stimulate the proliferation of easy muscle cells and amplify the formation of a dense extracellular matrix.

As a result, a thickened subendothelial fibrous plaque is formed, consisting of easy muscle cells, the internal wall of which is surrounded through connective tissue and intracellular and extracellular lipids. A technique comparable to bone formation leads to calcification inside the plaque.

Stable plaques regress. It then grows slowly over countless a long time till it leads to stenosis or occlusion.

Unstable plaques are susceptible to direct erosion or rupture. They motive acute thrombosis, occlusion, and infarction lengthy earlier than hemodynamically vast stenosis. Many scientific occasions are triggered by using unstable plaques that do now not exhibit sizeable adjustments in the angiogram; Thus, stabilization of atherosclerotic plaques may additionally be a way to limit morbidity and mortality.

The elasticity of the fibrotic thickness and its resistance to harm rely on the stability between the procedures of collagen deposition and destruction. Plaque rupture is brought on through the launch of metalloproteases, cathepsins, and collagenases by using activated macrophages inside the plaque.

These enzymes decrease the thickening of the fibers, specially round the edges, inflicting the pill to skinny and sooner or later rupture. Plaque T cells help destroy down cytokines. The latter slows the synthesis and deposition of collagen in easy muscle cells, which normally will increase plaque formation.

Risk of atherosclerosis plaques

When the plaque ruptures, its contents come into contact with the circulating blood, which reasons thrombosis; macrophages additionally stimulate the system of thrombus formation.

Because they comprise a tissue issue that helps the formation of thrombus in vivo. Then one of 5 results can occur:

The ensuing thrombus can merge into a plaque. Later, structural adjustments show up and the plaque grows rapidly.

A thrombus can shortly block vascular permeability, main to acute ischemia.

Development of thrombotic embolism.

Plaque fills with blood and enlarges and shortly blocks the artery.

Plaque steadiness relies upon on many factors, such as its composition (proportion of lipids, inflammatory cells, clean muscle cells, connective tissue, and thrombus), wall anxiety (splint stretch), size, core, blood flow, and plaque location. depends. Internal bleeding can play an vital function in plaque stability.

In general, unstable plaques of coronary arteries include a giant quantity of macrophages, a massive lipid core, and a skinny fibrous fibrous capsule; they slender the permeability of blood vessels with the aid of much less than 50% and are susceptible to surprising rupture. Unstable plaques in the carotid arteries have the identical composition and generally reason issues due to extreme stenosis and occlusion or platelet aggregation, and embolism is greater frequent than rupture. Low-risk atherosclerotic plaques have a thicker pill and comprise much less lipids; they frequently slender vascular permeability via 50% and lead to secure angina pectoris.

Atherosclerosis factors

Certain elements regularly manifest concurrently with metabolic syndrome. This syndrome consists of belly obesity, atherogenic dyslipidemia, arterial hypertension, insulin resistance, and propensity for thrombosis in sedentary sufferers and regularly occurring inflammatory reactions. Insulin resistance is no longer synonymous with metabolic syndrome, however might also play a key position in its etiology.

Dyslipidemia is a thing of atherosclerosis

Dyslipidemia is excessive complete cholesterol, low LDL cholesterol, or low low-density lipoprotein cholesterol. Arterial hypertension and diabetes make a contribution to the improvement of atherosclerosis. At the identical time, it will increase the dysfunction of the endothelium and will increase the irritation in the vascular endothelium.

With dyslipidemia, the subendothelial content material and LDL oxidation stage increase. Oxidized lipids stimulate the synthesis of adhesion molecules and inflammatory cytokines. They can be antigenic, ensuing in T-mediated immunity and irritation of the arterial wall.

Although HDL was once until now notion to shield in opposition to atherosclerosis via reverse ldl cholesterol transport and transport of antioxidant enzymes that can degrade and neutralize oxidized lipids, latest proof from randomized trials and genetics suggests that HDL performs little position in atherogenesis. The position of hypertriglyceridemia in atherosclerosis is complex, though it has a small unbiased effect.

Arterial hypertension is a aspect of atherosclerosis

It can motive irritation of blood vessels via mechanisms associated to angiotensin II. The latter stimulates endothelial cells, vascular easy muscle cells, and macrophages to produce pro-

atherogenic mediators, together with cytokines, superoxide anions, prothrombotic factors, boom factors, and oxidized lectin-like LDL receptors.

Diabetes is a issue of atherosclerosis

Diabetes motives the formation of glycolysis merchandise that amplify the synthesis of anti-inflammatory cytokines in endothelial cells. Oxidative stress and oxygen radicals shaped in diabetes without delay injury the endothelium and make contributions to atherogenesis.

Cigarette smoke is a thing of atherosclerosis

Cigarette smoke carries nicotine and different chemical substances that are poisonous to the vascular endothelium. Smoking, consisting of passive smoking, will increase platelet reactivity. It additionally will increase plasma fibrinogen and hematocrit (which will increase blood viscosity). Smoking will increase LDL and lowers HDL; ensuing in narrowing of the arteries, which is in particular unsafe for arteries that are already narrowed by way of atherosclerosis. HDL tiers upward jab with the aid of about 6-8 mg/dL inside 1 month of quitting smoking.

Lipoprotein is a thing of atherosclerosis

(a) [Lp(a)] is proatherogenic and is an impartial threat thing for the improvement of cardiovascular diseases, which include myocardial infarction, vascular and aortic valve stenosis. It has a shape comparable to that of LDL. Also, B-100 has a hydrophilic apolipoprotein (a) covalently linked to a hydrophobic apolipoprotein. Lp(a) tiers are genetically decided and continue to be pretty steady in the course of life. A degree of Lp(a) above 50 mg/dL is pathological.

Apolipoprotein is a thing of atherosclerosis

(B) (apoB) is a particle with two isoforms: apoB-100, synthesized in the liver, and apoB-46, synthesized in the intestine. ApoB-100 has the capability to bind to LDL receptors and is accountable for ldl cholesterol transfer. In addition, it is accountable for transporting oxidized phospholipids and has anti-inflammatory properties. The presence of apoB particles in the arterial wall is an preliminary element in the improvement of atherosclerotic lesions.

LDL is atherogenic - the issue of atherosclerosis

A very giant quantity of LDL is attribute of atherogenic diabetes. The mechanism may also enlarge susceptibility to oxidative and non-susceptible endothelial damage.

Atherosclerosis regularly develops besides signs and symptoms for decades. Symptoms show up when blood go with the flow is obstructed. Transient ischemic signs and symptoms (eg, secure exertional angina, transient ischemic attacks, periodic claudication) may additionally advance when continual plaque increase reduces arterial permeability by means of > 70%. Vasoconstriction can exacerbate harm to the vessel partitions (even if it has no longer beforehand limited blood flow) and lead to extreme or entire stenosis. Symptoms of unstable angina, myocardial infarction, ischemic stroke, or leg ache can be brought on through thrombosis or embolism when unstable plaques rupture and all at once block a massive artery. Atherosclerosis can additionally reason surprising dying besides a preceding steady or unstable angina pectoris.

Conclusion

Atherosclerosis is a multifaceted disease influenced by various factors. Elevated cholesterol levels, hypertension, smoking, diabetes, obesity, sedentary lifestyle, genetic predispositions, and other lesser-known factors all contribute to the initiation and progression of atherosclerotic plaque. Recognizing and addressing these risk factors are essential in preventing and managing this prevalent and potentially life-threatening condition. By adopting a holistic approach that targets lifestyle modifications and medical interventions, the burden of atherosclerosis can be significantly reduced.

References:

1. Murtalibov Sh. A. i ssoavtr. ―Psihiatriya ma‘lomotnoma‖ 1993.
2. Xodjaeva N.I.Shoyusupova A.I., «Psihiatriya», Tashkent, 2013.
3. Coeytaux A., Jallon P., Morabia A. Incidence of status epilepticus in Frenchspeaking Switzerland: (EPISTAR). Neurology 2000;55:693–97.
4. Allergology and Immunology. National leadership. GEOTAR Media 2013 (Russian magazine)