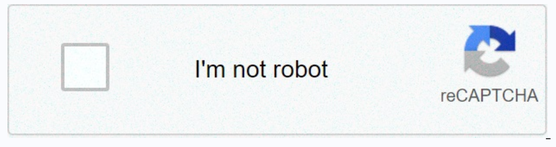

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Atheroma formation video

Accumulation of degenerative material in the inner layer of the artery walls AtheromaOther namestheromata (multiple number), atheromas (multiple number)Atherosclerotic plaque is a carotenous endarterectomy specimen. It shows the division of the joint between the inner and outer carotid arteries. SpecialtyCardiology ComplicationsTrombbosis, embolism of atheroma, or atheromatous plaque (plaque), is an abnormal accumulation of material in the inner layer of the wall of an artery. [1] The substance consists mainly of macrophage cells,[2][3] or debris containing lipids, calcium and varying amounts of fibrous connective tissue. Accumulated material forms a swelling in the wall of the artery, which can penetrate into the lumen of the artery, narrowing and limiting blood flow. Atheroma is the pathological basis of the disease entity atherosclerosis, a subtype of atherosclerosis. [summons required] Signs and symptoms in most people, the first symptoms stem from atheroma progression of the heart arteries, most often leading to heart attack and subsequent weakness. Cardiac arteries are difficult to track because (a) they are small (from about 5 mm to microscopic), (b) hidden deep in the chest, and (c) never stop. In addition, all mass clinical strategies focus on the minimum costs and (b) the overall safety of the procedure. Therefore, existing diagnostic strategies for detecting atheroma and monitoring the response to treatment are extremely limited. The most commonly used methods, the patient's symptoms and heart stress tests, do not detect the symptoms of the problem until the atheromatous disease is very advanced, because the arteries are enlarged, do not narrow in response to an increase in atheroma. [4] This plaque ruptures, producing debris and blood clots that hinder blood flow later, sometimes locally (as seen in the angiogram), which reduces/halts blood flow. Still, these events occur suddenly, and neither stress tests, stress tests or angiograms are revealed in advance. Mechanism Healthy epicardial coronary artery consists of three layers, tunica intima, media and adventitia. [5] Changes in atheroma and artery walls usually result in smaller aneurysms (enlargements) large enough to compensate for the extra wall thickness, and the diameter of the lumen has not changed. However, in the end, typically, as a result of rupture of vulnerable plaques and blood clots in the lumen above the plaque, narrowing (narrowing) of the vessel develops in some areas. Less often, the artery is enlarged so much that the gross aneurysm enlarges the artery results. All three results are often observed in different places within the same individual. [summons required] Throes and closure Over time, atheromata tend to progress in size and thickness, and induces the surrounding muscular central region (the media) of the artery that stretches out, called transformation, typically just enough to compensate for their size to the The opening of the artery (lumen) remains unchanged until typically more than 50% of the artery wall cross-sectional area consists of atheromatous tissue. [4] The narrowed arterial artery, which is blocked by anteromy. If the enlargement of the muscular wall eventually can not keep up with the enlargement of the volume of the atheroma, or if a blood clot forms and organizes the plaque, then the lumen of the artery narrows over the tissue separating the atheroma from the bloodstream due to repeated fractures, blood clots & fibrosis. This narrowing is becoming more common after decades of life, becoming more common after people in their 30s and 40s. The endothelium (the cell is single-layered inside the vessel) and which tissue, called a fibrous cap, separates the atheroma of the blood from the lumen. If rupture of the endothelium and fibrous cap (see vulnerable plaque) occurs, within a fraction of a second, the shower of plaque debris and (b) the platelet and blood cling response (both to the debris and the fracture site) occur within a fraction of a second. As a result of the rupture (a) a shower of debris closing smaller later vessels (debris greater than 5 micrometers is too large to pass through the capillaries)) (b) the accumulation of platelets and clots above the rupture (injury/repair response), resulting in rupture, sometimes closure, of the lumen. Subsequent tissue damage is due to a) closure of the downstream microvascular and/or b) of the lumen at rupture, resulting in loss of blood flow to subsequent capillary microvascular. This is the main mechanism of heart attack, stroke or other related cardiovascular disease problems. While blood clots at the fracture site tend to decrease in volume over time, some blood clots can become organized fibrotic tissue, causing narrowing of the artery lumen; The hydrological ingest is sometimes seen in angiography tests, if severe enough. Since angiographic methods can reveal only larger lumens, typically more than 200 micrometers, angiography usually does not reveal what happened after a cardiovascular event. Artery enlargementIf the muscle wall enlargement is exaggerated over time, then the gross expansion of the artery results, usually several decades of life. It's a less common result. Atheroma inside the aneurysm enlargement (vessel bulge) can tear and shower debris from the atheroma and blood clot down. If arterial enlargement continues 2-3 times the usual diameter, the walls often become weak enough that only the stress of the pulse, the loss of wall integrity can occur, leading to sudden bleeding (bleeding), main symptoms and weakness; often a quick death. The main shirt for the formation of an aneurysm is the pressure atrophy of structural support of muscle layers. The main structural proteins are collagen and elastin. This causes thinning and wall balloons allow gross enlargement, as shown in the Necessary] Histology The accumulation (swelling) is always in the tunic intima, between the endothelium lining and the smooth muscle middle layer of the artery wall. While in the early stages of the gross appearance, traditionally referred to as fatty streaks by pathologists, they are not available in fat cells, but rather in the accumulation of white blood cells, especially macrophates, that have been taken up by oxidized low-density lipoprotein (LDL). After they accumulate large amounts of cytoplasmic membranes (associated with high cholesterol content) these are called foam cells. When foam cells die, their contents are released, which attracts more macrophag and creates extracellular lipid cores near the center of each atherosclerotic plaque near the inner surface. Conversely, the outer, older parts of the plaque become more calcical, less metabolically active and physically rigid over time. [summons required] Atheromata does not develop in the veins because they are not subjected to the same hemodynamic pressure as arteries, [7] unless surgically moved to act as arteries as in bypass surgery. Diagnosis Illustration comparison of normal blood vessels and partially blocked vessel due to atherosclerotic plaque. Notice the expansion & absence of many luminaire constation. [4] As arterial walls dilate in places with atheroma,[4] atheroma is found before death and autopsies have long been problematic. Most methods focused on openings in the arteries; it is very important, but there is a complete lack of atheroma inside the artery walls. Historically, arterial wall fixation, staining and thin section were the gold standard for detecting and describing atheroma, death and autopsy. With special stains and tests, micromemes are detected[8], typically in the smooth muscle cells of the arterial medium, near fatty streaks within a year or two. Interventional and non-interventional methods for detecting atherosclerosis, in particular vulnerable plaque (non-occlusive or soft plaque), are now widely used in research and clinical practice. Carotis Intima-media thickness The Study (CIMT can be measured by b-mode ultrasound testing) measurement is recommended by the American Heart Association as the most useful method for identifying atherosclerosis, and now it can be very well for the detection of gold standard. [summons required] IVUS is the current most sensitive method for detecting and measuring more advanced atheroma in individuals living within, although it is not usually used until decades after atheroma begins to form due to cost and body invasiveness. [summons required] Ct using the most up-to-speed higher resolution spiral, or higher-speed EBT, machines were the most effective method of detecting calcification present in plaque. However, the atheroma must be developed enough to provide relatively large areas of calcification in them to create large enough regions ~ 130 Hounsfield units that are the CT scanner software from other surrounding tissues. Typically, such regions begin occurring in heart arteries about 2-3 decades after atheroma begins to develop. The presence of smaller, patchy plaques is actually more dangerous in developing acute heart attacks. [9] Arterial ultrasound, especially in the carotid arteries, by measuring the thickness of the artery wall, provides an opportunity to partially monitor the progression of the disease. Since 2006, the thickness, commonly referred to as IMT's intimal-medial thickness, has not been measured clinically though it has been used by some researchers since the mid-1990s to track changes in arterial walls. Traditionally, clinical carotenography ultrasound is only an estimate of the degree of blood lumen restriction, narrowing, as a result of very advanced disease. The National Institute of Health had a five-year $5 million study, headed by medical researcher Kenneth Ouriel, to study intravascular ultrasound techniques regarding atherosclerotic plaque. [summons required] More progressive clinicians began using IMT measurement to quantify and track disease progression or stability within individual patients. [summons required] Angiography, since the 1960s, has been a traditional way of evaluating atheroma. However, angiography is just movement or stills of paint mixed with blood with arterial lumen and never show atherom; the walls of the arteries, including the atherom with the arterial wall, remain invisible. The limited exception to this rule is that with very advanced atheromy, extensive calcification within the wall, most older people can see the halo-like radio density ring, especially if the arterial lumens have been made visible at the end. For cine-floro, cardiologists and radiologists usually look for these calcification shadows to detect arteries before injecting any contrast agent during an angiogram. [summons required] Classification of lesions Type I: Ulcerative, hemorrhaic, thrombosis lesion[5][10] VII, more specifically: Only 4 out of ten points are referred to, and only one of them is a primary, peer-reviewed reference. Without medical refs, it's unclear how reliable any proposed approach applies to the treatment of the disease, even if it's a good general advice. Please review the contents of this section and provide the appropriate links if possible. Materials without a source or from poor sources can be attacked and removed. Search for sources: Atheroma News · newspapers · books · scientist · JSTOR (October 2019) A number of approaches have been supported[who?] as a method of reducing or reversing atheroma[11] necessary] eating a diet of raw fruits, vegetables, nuts, beans, berries, and grains; [11] Foods containing omega-3 fatty acids such as fish, fish-derived supplements, as well as flaxseed oil, borage oil, and other non-animal based oils; abdominal fat reduction; aerobic exercise; [11] inhibits cholesterol synthesis (known as statins); [11] low normal blood sugar (gllycosylated hemoglobin, also known as HbA1c); consumption of micronuts (vitamins, potassium and magnesium); maintaining normal or healthy blood pressure levels; Aspirin supplemental cyclodextrin also dissolves cholesterol, removing it from plaques[12] A history of research in developed countries, improved public health, infection control and increasing lifespan, ateroma processes have become an increasingly important problem and burden for society. Atheromata remains the primary basis for disability and death, despite a gradual improvement since the early 1960s (adjusted for the age of patients). Thus, the increase in efforts to better understand, address and prevent the problem is constantly evolving. [summons required] According to U.S. data, in 2004, about 65% of men and 47% of women, the first symptoms of cardiovascular disease are heart attacks (heart attacks) or sudden death (death within an hour of symptom formation). [summons required] A significant part of the artery flow-disrupting events occur in places with less than 50% lumenal throomens. Heart stress testing, traditionally the most commonly performed noninvasive method of testing blood flow limits, usually only detects lumen constation of ~75% or more, although some doctors advocate nuclear stress methods, which sometimes detect only 50%. [summons required] The sudden nature of complications of pre-existing atheroma, vulnerable plaque (non-occlusive or soft plaque) has led to the development of intensive care units and complex medical and surgical interventions since the 1950s. Angiography and later heart stress testing were launched to either visualize or indirectly detect thomens. Then came bypass surgery, with plumb transplanted veins, sometimes arteries, around the stenoses and more recently angioplasty, now including a stent, most recently drug-coated stents that stretch the stenoses more open. [summons required] Yet despite these medical advances, success in reducing symptoms of angina and decreased blood flow, atheroma rupture events remain the main problem and still sometimes lead to sudden disability and death despite even the fastest, massive and qualified medical and surgical intervention available anywhere today. According to some clinical trials, bypass surgery and angioplasty procedures have had the best minimal effect, if any, on improving overall survival. The death rate for bypass operations is typically between 1 and 4%, angioplasty is between 1 and 1.5%. [summons required] Furthermore, these Often it occurs only if the individual is symptomatic, often already partially disabled, as a result of the disease. It is also clear that both angioplasty and bypass interventions do not prevent future heart attacks. [summons required] Older methods of understanding atheroma, which dates to before World War II, relied on autopsy data. Autopsy data have long shown initiation of fatty streaks in later childhood with slow asymptomatic progression for decades. [4] One way to get atheroma is through very invasive and costly IVUS ultrasound technology; this gives us the exact volume of the inner intima, as well as the central media layers of about 25 mm (1 in) of artery length. Unfortunately, it does not give information about the structural strength of the artery. Angiography does not imagine atherom; it only makes the blood flow to the blood vessels visible. Alternative methods have been used that are not or less physically invasive and less expensive per individual study, such as computed tomography (CT; higher speed driven by electron beam tomography) and magnetic resonance imaging (MRI). The most promising since the early 1990s has been EBT, detecting calcification in atherom before most individuals start having clinically recognized symptoms and debility. Statin therapy (to lower cholesterol) does not slow down the speed of calcification determined by CT. MRI coronary artery wall imaging, although currently only in research studies, has demonstrated the ability to detect vascular wall thickening in asymptomatic high-risk individuals. [13] As a non-invasive, ionising radiation-free technique, MRI-based techniques may continue to be used to monitor disease progression and regression. Most visualization techniques are used in research, they are not widely available to most patients, have significant technical limitations, are not widely accepted and are generally not covered by health insurers. [summons required] In human clinical trials, it has become increasingly clear that a more effective focus on treatment is slowing down, stopping and even partially reversing the atheroma growth process. [14] There are a number of prospective epidemiological studies, including the Atherosclerosis Risk in Communities (ARIC) study and the Cardiovascular Health Study (CHS), which supported the direct correlation between carotis intima-media thickness (CIMT) and cardiovascular disease history. The ARIC study was conducted on 15,792 people between the age of 5 and 65 in four different regions of the United States between 1987 and 1989. In this study, the baseline CIMT was measured and the measurements were repeated at intervals of 4 to 7 years by carotide mode B ultrasound. The increase in CIMT correlated with the increased risk of CAD. CHS was initiated in 1988 and the risk of CIMT and myocardial infarction and stroke 4476 patients aged 65 years or younger. At the end of approximately six years of follow-up, CIMT measurements correlated with cardiovascular events. [summons required] Paroi arteryelle et Risque Cardiovasculaire asia africa/middle east and latin america (PARC-AALA) is another important large-scale study in which 79 centers from countries in Asia, Africa, the Middle East and Latin America participated, and the distribution of cimt, according to various ethnic groups and associations of Framingham cardiovascular score was studied. Multilinear regression analysis showed that the Framingham cardiovascular score was associated with CIMT and carotid plaque regardless of geographical differences. [summons required] Cahn et al. prospectively tracked 152 patients with coronary artery disease for 6-11 months with carotenic artery ultrasonography and noted 22 vascular events (myocardial infarction, transient ischemic seizure, stroke, and coronary angioplasty) during that period. It was concluded that cervical atherosclerosis measured this non-interventional method of prognostic significance in coronary artery patients. [summons required] In the Rotterdam study, Bots and his mts followed 7,983 patients at the age of >55 for an average of 4.6 years, and 194 events of myocardial infarction were reported during this period. CIMT was significantly higher in the heart attack group than in the other group. Demircan et al. found that CIMT in patients with acute coronary syndrome increased significantly compared to patients with stable angina pectoris. [summons required] In another study, it was reported that the maximum CIMT of 0.956 mm had a sensitivity of 85.7% and a specificity of 85.1% for the prediction of angiographic CAD. The study team consisted of patients admitted to the cardiac outpatient clinic with symptoms of stable angina pectoris. The study showed that CIMT was higher in patients with significant CAD than in patients with non-critical coronary artery lesions. Regression analysis showed that a thickening of the average intima-media complex of more than 1.0 predicted significant CAD patients. In CIMT, the number of coronary artery affected gradually increased significantly. According to the literature, cimt was found to be significantly higher in the presence of CAD. Furthermore, CIMT was increased as the number of affected vessels increased and the highest CIMT values were observed in patients with the main coronary artery affected left. However, human clinical trials have been slow to make clinical & medical evidence, in part because of the asymptomatic nature of atheromata that they are particularly difficult to study. Promising results have been found in carotid intima-media thickness scanning (CIMT measurable B-mode ultrasound), B vitamins that reduce protein corrosive, homocysteine, and which reduce the amount and thickness of carotid artery plaque, and stroke, even in section section Furthermore, understanding what drives the development of atheroma complex is several factors involved, only a few, such as lipoproteins, which is even more important lipoprotein subclass analysis, blood sugar and hypertension are best known and researched. More recently, some of the complex immune system patterns that promote or inhibit the inherent inflammatory macrophage trigger processes involved in atheroma progression are slowly better clarified in animal models of atherosclerosis. [summons required] See also: Angiogram ApoA-1 Milano Atherosclerosis Atherothrombosis Coronary Circulation Imiate ET Hemorheologic-Hemodynamic Theory of Atherosclerosis Lipoprotein LDL, HDL, IDL and VLDL References ^ Lusis, Aldons J. (September 2000). Atherosclerosis. Nature. 407 (6801): 233-241. doi:10.1038/35025203 PMC 2826222. PMID 11001066. ^ Hotamisligil, Gökhan S (April 2010). Endoplasmic retikulum stress and atherosclerosis. Natural medicine. 16 (4): 396-399. doi:10.1038/nm0410-396. PMC 2897068. PMID 20376052. ^ Oh, Jisu; Riek, Amy E.; Weng, Sherry; Petty, Marvin; Kim, David; Colonna, Marco; Cell, Marina; Bernal-Mizrachi, Carlos (April 6, 2012). Endoplasmic Reticulum stress controls M2 macrophage differentiation and foam cell formation. 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