Difference Between Atheroma and Atherosclerosis

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Key Difference – Atheroma vs Atherosclerosis

Atherosclerosis is a pathological condition of the arteries characterized by the buildup of fat deposits inside the arterial wall. These fat deposits that are formed as a result of atherosclerosis are called atheromas. This is the key difference between atheroma and atherosclerosis. Atherosclerosis is by far the commonest cause of cardiac, cerebral and peripheral vascular diseases and consequently, it has mortality and morbidity rates that surpass most of the other illnesses.

What is Atherosclerosis?

Atherosclerosis is a pathological condition of the arteries that is characterized by the buildup of fat deposits inside the arterial wall. There are different factors and comorbidities that contribute to the development of atherosclerosis. These contributory factors can be basically divided into two categories as modifiable factors and nonmodifiable factors.

Modifiable Factors

- Hyperlipidemia
- Hypertension
- Diabetes
- Inflammation
- Cigarette smoking

Nonmodifiable Factors

- Genetic defects
- Family history
- Increasing age
- Male gender

Pathogenesis of Atherosclerosis
“Response to injury” is the most widely accepted hypothesis that explains the pathogenesis of this condition by integrating the aforementioned risk factors with the pathological events taking place in the arterial wall. This hypothesis suggests a seven step mechanism for the development of an atheroma.

1. **Endothelial injury and dysfunction** which increase the vascular permeability, **leukocyte adhesion** and the likelihood of **thrombosis**.
2. **Accumulation of lipids** inside the vessel wall – LDL and its oxidized forms are the types of fat that accumulate abundantly.
3. **Monocyte adhesion** to the **endothelium** – these monocytes then migrate into the intima and transform into foam cells or macrophages.
4. **Platelet adhesion**
5. Platelets, **macrophages** and various other types of cells that are accumulated at the site of injury start to release different chemical mediators that initiate the recruitment of **smooth muscle** cells either from the media or from the circulating precursors.
6. The recruited smooth muscle cells proliferate while synthesizing extracellular matrix substances and attracting **T cells** towards the damaged vessel.
7. Lipid accumulates both extracellularly and intracellularly (inside macrophages and smooth muscle cells) forming an atheroma.

**Morphology**

The two hallmark morphological features of atherosclerosis are the presence of fatty streaks and atheromas.

Fatty streaks contain foamy macrophages filled with lipids. At the beginning, they appear as tiny yellow spots and later they coalesce, forming streaks that are usually around 1cm in length. Since they are not sufficiently elevated from the surface, blood flow through the vessel is not interrupted. Although the fatty streaks can advance into atheromas, most of them spontaneously disappear. The aortas of healthy infants and adolescents can also have these fatty streaks.

(Morphology of atheromas is discussed under the heading “atheroma”)


Atherosclerosis mainly affects large arteries such as the aorta and medium size arteries like coronary arteries. Although it is possible for this pathological process to happen anywhere in the body, a person becomes symptomatic only when atherosclerosis damages the arteries supplying blood to heart, brain and the lower extremities. Therefore the major complications of atherosclerosis are,

- Myocardial infarction
- Cerebral infarction
- Gangrene of the lower limbs
- Aortic aneurysms
What is an Atheroma?

The fat deposits formed inside the arterial wall as a result of the atherosclerosis are called atheromas. These are intimal lesions composed of a lipid core covered by a fibrous cap.

Morphology of Atheroma

Atherosclerotic plaques have a typical yellowish white color but the presence of a superimposed thrombus can give a reddish brown color to the plaque. They protrude into the lumen of the arteries impeding the blood flow through the vessels. Plaques are formed in different sizes but they can coalesce into large masses capable of completely occluding the vascular lumen.

An atheroma has three main components:

- Smooth muscles, macrophages, T cells
- Extracellular matrix having collagen, elastic fibers and proteoglycan
Intracellular and extracellular lipid

As mentioned above, an atheroma has a fibrous cap made of smooth muscle cells and dense collagen fibers. Beneath this cap lies the fat that has accumulated at the damaged site along with other cells and debris. New blood capillaries start to appear around the periphery of the lesion, and this phenomenon is called Neovascularization. Unlike the typical atheromatous plaques, fibrous atheromas have a very little amount of fat, and they are principally made of fibrous connective tissues and smooth muscle cells. With time, atheromas gradually enlarge and get calcified. This calcification stiffens the arterial wall, making it less compliant and increasing the risk of coronary arterial diseases.

Clinically Significant Pathological Changes of Atheromas

- Rupture, ulceration or erosion of the fibrous cap exposes the underlying thrombogenic substances resulting in thrombosis.
- Hemorrhage into a plaque
- Atheroembolism
- Formation of aneurysms

What is the difference between Atherosclerosis and Atheroma?

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### Relationship

| Atherosclerosis is a pathological process. | Atheromas are the products of atherosclerosis. |

### Summary – Atherosclerosis vs Atheroma

Atheromas are the fat deposits formed inside the arterial wall whereas atherosclerosis is a pathological condition of the arteries that is characterized by the buildup of fat deposits inside the arterial wall. This is the basic difference
between atheromas and atherosclerosis. As discussed here, a balanced diet, exercise, and the self-restraint to stay away from cigarettes decrease the risk of atherosclerosis drastically. If you have these risk factors, it is important to get rid of them as soon as possible to live a long and healthy life.

References:


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