International Clinical and Medical Case Reports Journal
Case Report (ISSN: 2832-5788)

# Inhibitors of Atherosclerotic Plaque Crystallization Centers

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**Citation:** Maciej Pawlikowski. Inhibitors of Atherosclerotic Plaque Crystallization Centers. Int Clinc Med Case Rep Jour. 2026;5(11):1-7.

**Received Date:** 04 November 2026; **Accepted Date:** 07 November 2026; **Published Date:** 10 November 2026

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## Synopsis

This paper proposes a method for blocking the sites of atherosclerotic plaque development in arteries. It was created based on research findings from the years 1987–2026 [1–26]. The approach is theoretical and grounded in established chemical, physical, mineralogical, and medical phenomena. Considering the significance of the subject, the practical application of this method should be subject to clinical validation.

## Atherosclerotic Plaque Crystallization Centers

Crystallization centers are sites where the biological structures of tissues—in this case, parts of the arteries and the heart—are damaged. Such damage leads to breaking of interatomic bonds within these biological structures. The consequence of this process is the presence of free, electrically charged bonds (an electric field) at these locations. Ions circulating in the oxygenated blood attach themselves to these sites, leading to the formation of atherosclerotic plaque.

Among the various types of such centers of atherosclerotic plaque formation, we can distinguish:

**Primary**

- Genetic defects of biological structures

**Secondary**

- Results of physical damage
- Effects of lysis caused by toxins produced by microorganisms infecting the body (bacteria, viruses, fungi, etc.)
- Results of the impact of various types of harmful substances entering the body from the outside.

Regardless of their origin, centers of atherosclerotic plaque formation all look similar in scanning observations (Photo 1).

The sites where such centers develop may be random. Most likely, they are areas where the atomic structure of the tissue became weakened, which is why atherosclerotic plaque forms in various locations in the arteries and on heart valves. This type of mineralization may also occur in tissues outside the circulatory system. In any case, there may be a single cause of its development, or several coexisting factors.

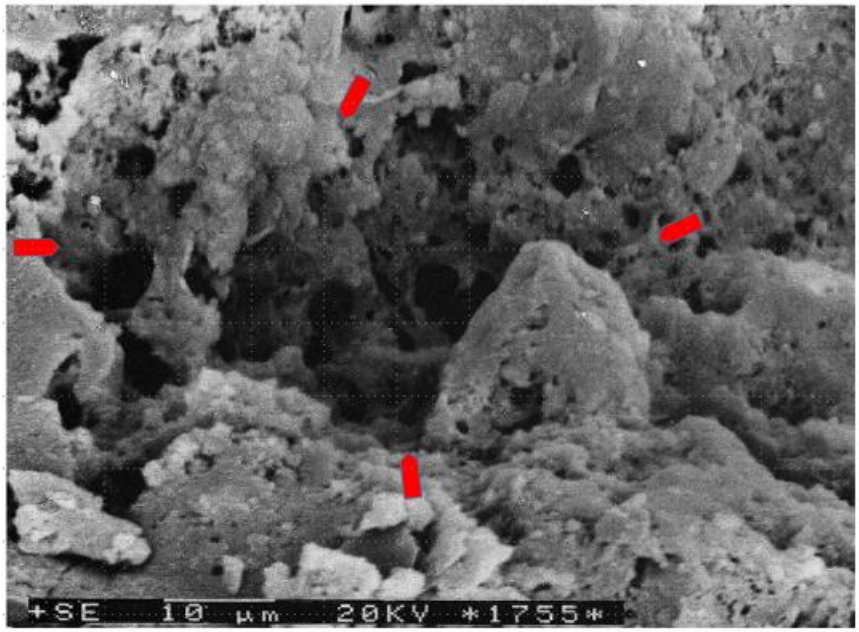



**Photo 1:** Damaged surface of an aortic valve leaflet (arrows)—a potential atherosclerotic plaque formation center. SEM.

## Healing Of Damaged Sites in Arterial Walls (Centers of Atherosclerotic Plaque Formation)

Examinations of the surface of arterial walls allowed us to observe the process of healing in their damaged sites (Photo 2). This phenomenon was caused by proliferation of fibrous protein structures. A better understanding of this process could help stimulate the healing of arterial damage and, consequently, prevent the formation of atherosclerotic plaque.

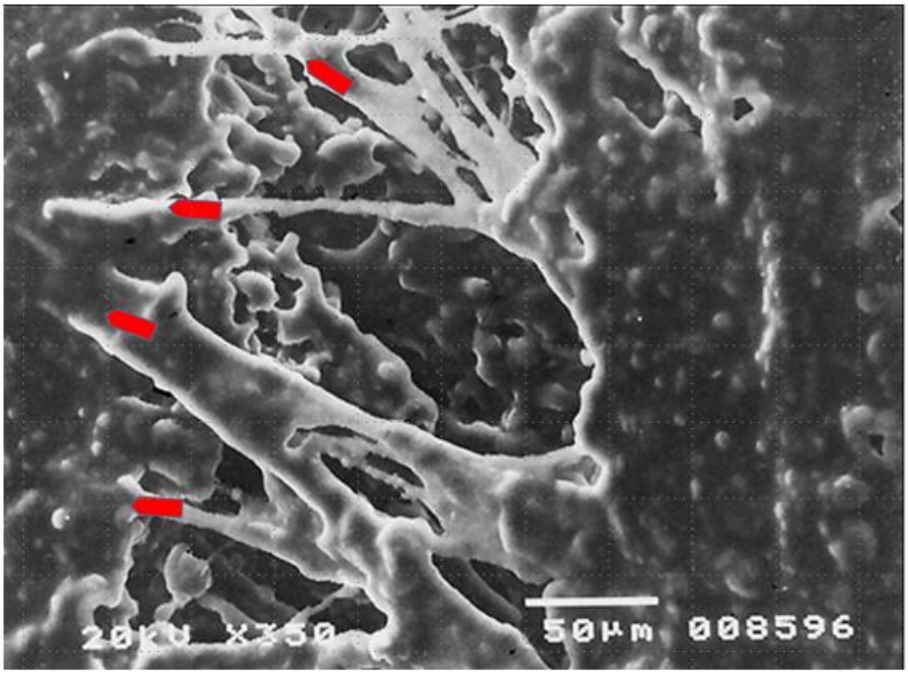



**Photo 2:** Healing of the damaged areas of the femoral artery wall. Arrows indicate the direction of proliferation of the system responsible for healing the damage. SEM.

## The Process of Atherosclerotic Plaque Development

This process, leading to the narrowing of the inner diameter of an artery, is widely recognized as harmful. However, looking at this phenomenon from a different perspective, atherosclerotic plaque formation may be seen as a process of healing ("sealing") the damaged sites in arterial walls (Figure 1).

Observations indicate that atherosclerotic plaque is usually built of cholesterol that is accompanied by weakly crystalline hydrated calcium phosphates. Their atomic structure is poorly organized (Photo 3).

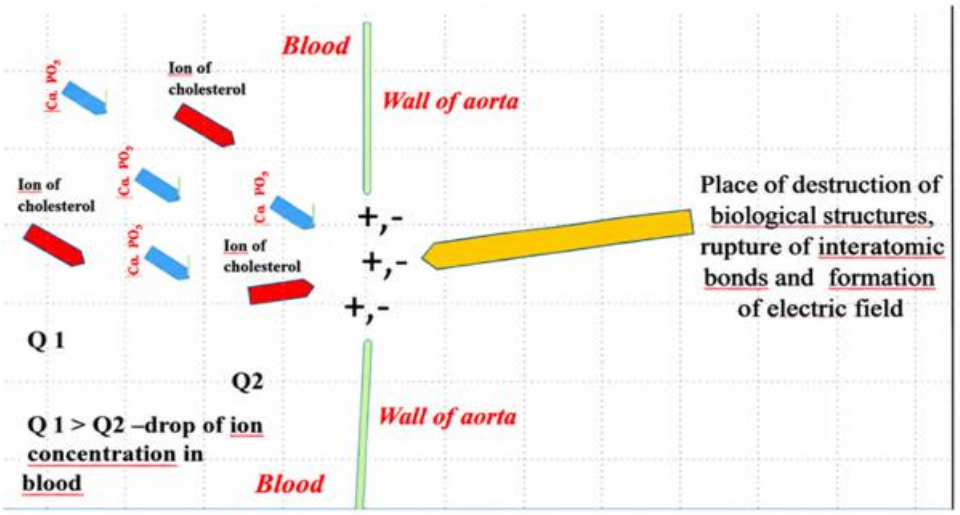



**Figure 1:** Diagram showing the formation of atherosclerotic plaque at an arterial wall damage location. The free, electrically charged interatomic bonds that emerge at the damaged site form a center where the development of an atherosclerotic plaque begins. Ions circulating in the arterial blood become attached to these free, electrically charged bonds.

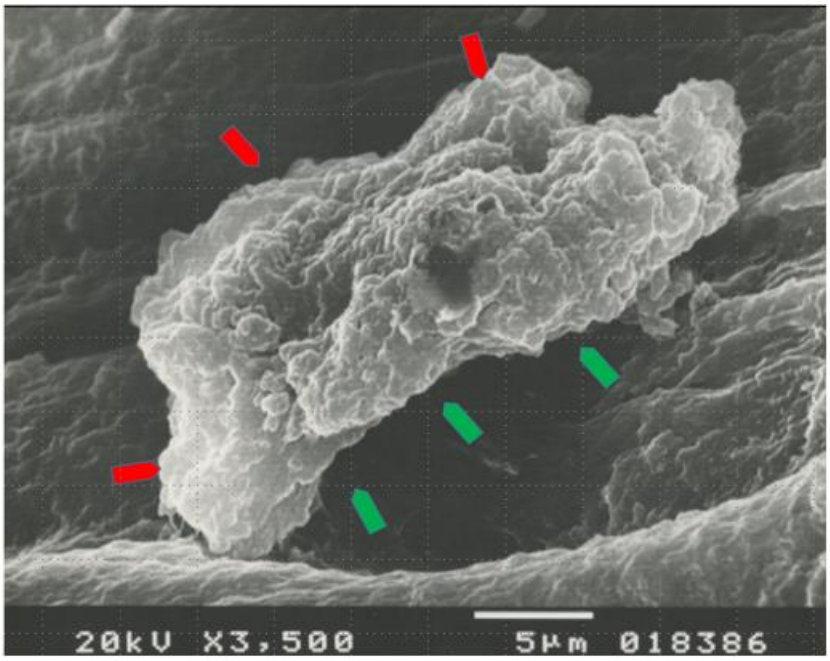



**Photo 3:** Cholesterol - phosphate atherosclerotic plaque in an early stage or development (red arrows) on a damaged surface of endothelium (green arrows). SEM.

## Main Components of Atherosclerotic Plaque

When discussing the formation of atherosclerotic plaque and how to prevent it, it is essential to consider the composition of the plaque, its crystallinity, and the components that build it (Figure 2). These are calcium phosphates (so called calcifications) and cholesterol. The main components of calcifications are Ca2+, PO43-, CO32-, OH-, F-2. In cholesterol, the components are carbon, hydrogen, and oxygen. Both organic and inorganic substances are made of ions with electric charges. After binding to the structure of the developing atherosclerotic plaque, these ions, both organic and inorganic, are built into it in such a way that not all their electric charges are bound with the structure of the crystallization center. Some of their bonds "stick out" of the developing plaque and become points of attachment for more ions in the blood. Therefore, the surface of atherosclerotic plaque becomes a center for binding ions, causing further growth of the plaque.

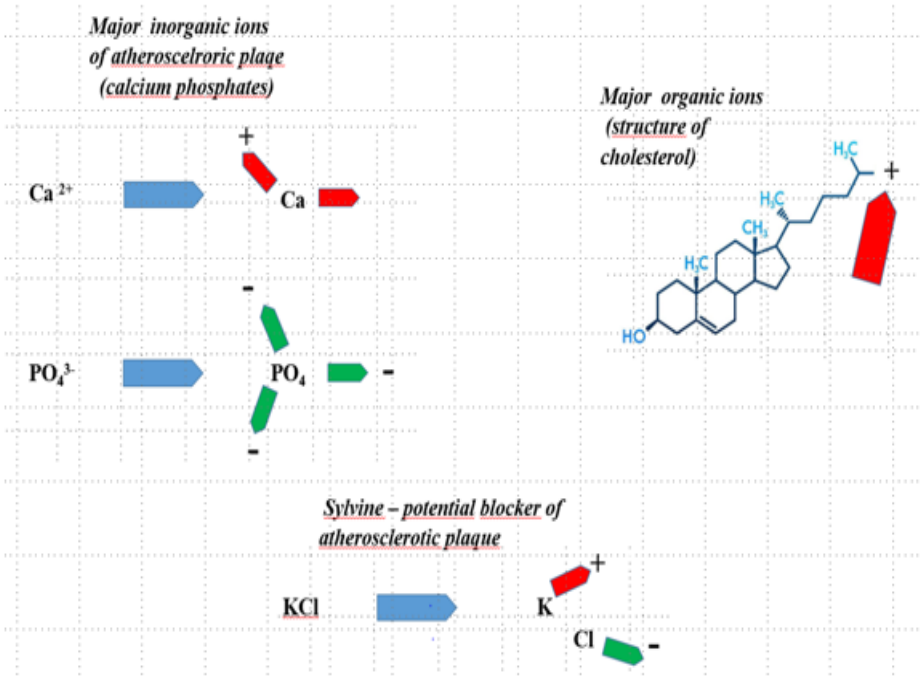



**Figure 2:** Primary ions that build atherosclerotic plaque, and sylvine-a potential blocker of atherosclerotic plaque crystallization centers.

## Blocking the Centers of Atherosclerotic Plaque Formation (Biomineralization)

The diagram in Figure 3 presents the process of blocking the center of atherosclerotic plaque development (a site where the arterial wall is damaged).

If the atherosclerotic plaque is already developed, the K and Cl ions block its further growth by attaching to the available bonds on the surface of the plaque [Figure 4]. In this way, the surface becomes "electrically neutral" and does not attract any more ions that make up the plaque.

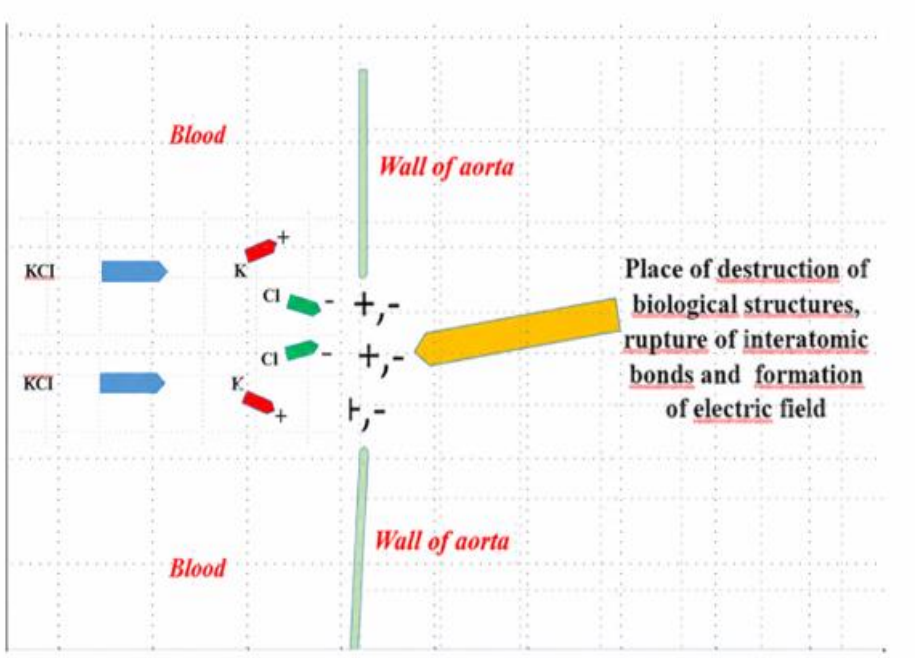



**Figure 3:** Diagram showing the process of blocking the site of arterial wall damage—the place where atherosclerotic plaque forms. Monovalent ions of potassium and chloride, drawn from the blood to the plaque's crystallization center, carry only a single electrical charge. Upon attaching to the biological structures at the site of the damage, they block the available interatomic bonds, thereby preventing the formation of atherosclerotic plaque.

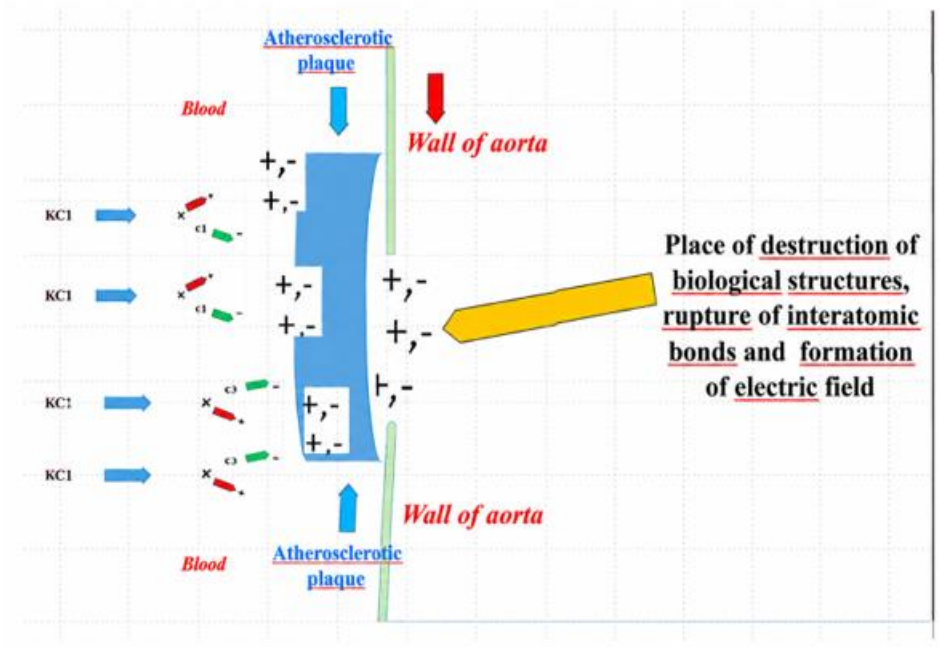



**Figure 4:** Diagram of blocking the available bonds (electric charges) on the surface of existing atherosclerotic plaque by the K+ i Cl – ions present in blood.

## Summary

Formation of atherosclerotic plaque requires the presence of damaged areas within the arteries, as well as elevated levels of cholesterol, ionized calcium, and phosphorus in the blood. Elevated cholesterol is a consequence of impaired liver function, as cholesterol derived from external sources is not as dangerous as that produced by the body itself. Meanwhile, blood calcium levels can be affected by poor thyroid (and parathyroid) function—specifically through fluctuations in hormones such as parathormone, calcitonin, and others.

Damage to the arterial walls, which can become a starting point of crystallization for atherosclerotic plaque, can be caused by various factors. It is essential to block such centers of crystallization, i.e. sites of damage in the arteries and structures of the heart, because they contain free interatomic bonds and an electric field, capable of capturing electrically charged ions from the bloodstream.

KCl is an effective agent for blocking the atherosclerotic plaque crystallization centers, as it readily dissociates into K+ i Cl- ions. These ions easily attach to free bonds at the site of damage, saturating either a positive or a negative bond, thereby eliminating any remaining free bonds and neutralizing the local electric field.

Thus, by attaching themselves one after another, monovalent ions gradually neutralize the electrical charge at the site of damage—a potential location for the development of atherosclerotic plaque—thereby stripping it of its ability to bind ions of cholesterol, calcium phosphates, or other such substances. In this way, the center of plaque formation becomes inactive; even if blood cholesterol levels are elevated, the development of atherosclerotic plaque cannot occur.

The situation is different when the arteries are already “clogged.” Experiments have shown that cholesterol in atherosclerotic plaque dissolves in alcohol, while the so-called calcification gradually dissolves in water [11].

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