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Targeting Atherosclerotic Plaques with Nanoparticles: Emerging Strategies to Prevent Heart Attacks

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A B S T R A C T

Atherosclerosis is a highly frequent heart disease that has killed and made many people sick all over the world. There are a lot of treatment alternatives accessible in clinical practice, but it's still challenging to totally stop the atherosclerotic process. A lot of old-fashioned anti-arteriosclerosis drugs don't dissolve well, don't act well on certain targets, and have clear negative effects. Nanomedicine-based drug delivery technologies have recently become possible strategies to treat atherosclerosis since they can convey different drugs to specific cells, microstructures, and molecules in lesion sites. These new features make medications work better and more bioavailable while also lowering their side effects. Nanomedicine not only keeps plaque from forming, but it also decreases lesions and eventually heals atherosclerosis, according to a lot of research. People think that nanomedicine is an important technique to minimize the risks that come with atherosclerosis even further. This review presents a complete overview of novel nanomedicine-based ways to get anti-atherosclerosis drugs to certain places. It tells you how these methods operate and how well they function to stop atherosclerosis. There are also some huge problems and big promises for the future when it comes to researching and using anti-arteriosclerosis nanomedicine.

Keywords: Nano medicine; Drug Carrier; Drug delivery; Atherosclerosis treatment; Targeted strategy

1. INTRODUCTION

For a long time, cardiovascular diseases (CVDs) have been the leading cause of death in the globe. The World Health Organization (WHO) says that some 18 million people die from heart and blood vessel diseases every year. Four out of five of these deaths are caused by strokes or heart attacks. CVDs are diseases that affect the heart and blood vessels. Coronary heart disease, which includes angina pectoris and myocardial infarction, is one example. Atherosclerosis is the most common cause of coronary heart disease. Cerebrovascular disease, rheumatic heart disease, heart failure, aortic aneurysm, myocarditis, hypertension, and pulmonary artery hypertension and thrombosis are more instances. Atherosclerosis is one of the most common causes of a sudden ischemic heart attack, which can be fatal. It also raises the risk of ischemic stroke. Atherosclerosis is a long-term, widespread inflammatory disease. Atherosclerosis is when atherosclerotic plaques, which are made up of cholesterol, other lipids, cells, and platelets, develop up. These plaques collect in the intima, which is the inner membrane of arteries. This happens most often in the aorta, coronary arteries, and cerebral arteries. The plaques that are getting bigger obstruct blood flow through the vessel and make it more likely that a clot may form, especially if they rupture. In certain cases, the arterial lumen can suddenly collapse, stopping blood flow to an organ like the heart or brain, which can be fatal.

Making atherosclerotic plaque takes a number of phases (see Figure 1). Damage to the vascular endothelium and long-term inflammation are likely to be the main causes. The damaged endothelium enables LDL lipoproteins through and releases cytokines like ICAM-1 and VCAM-1, which attract leukocytes, especially monocytes. The lipids that have accumulated up are oxidized, and monocytes that have gone into the intima convert into macrophages and then into foam cells, which store the oxidized lipids. These deposits also cling to platelets. After that, macrophages and other activated cells release proinflammatory chemicals. This makes smooth muscle cells travel to the intima. Their structure changes, which makes them less flexible, and they create things like collagen and proteoglycans that are outside of cells. These atherosclerotic lesions often turn to stone, and difficulties with the endothelium make it easier for platelets to stick together, which causes atherosclerotic ulcers and thrombi. Macrophages create a lot of enzymes and proinflammatory chemicals that can inhibit collagen production, destroy muscle cells, and break up plaque. This can lead to thrombosis and obstruction of the vessel lumen.

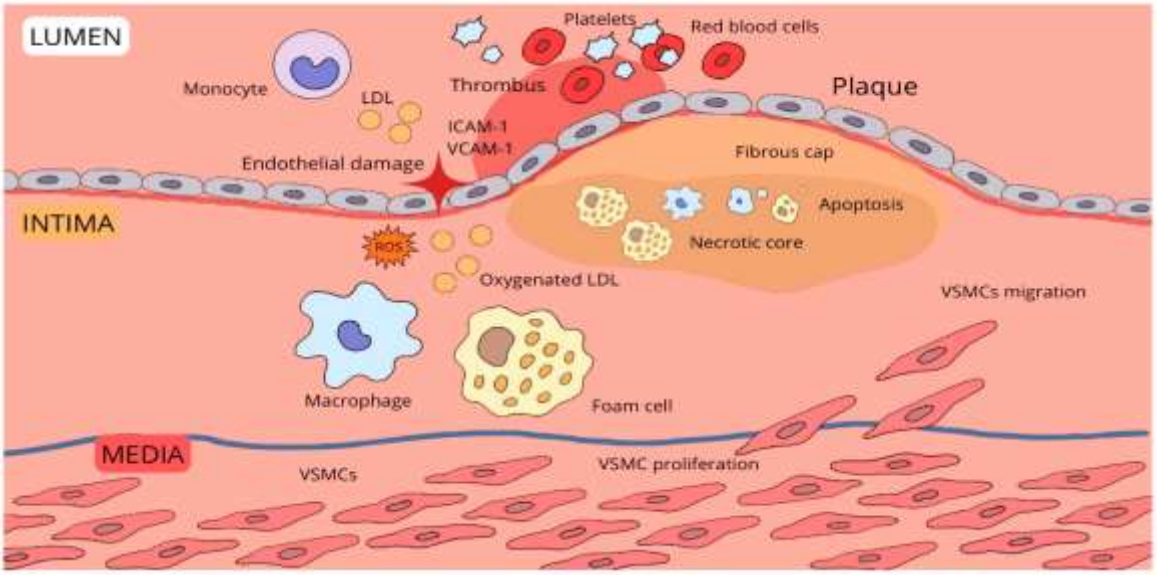

Figure 1. Scheme of atherosclerotic plaque formation.

Doctors can utilize a lot of various anti-atherosclerotic medications right now. In clinical trials, some of these methods have been demonstrated to work quite well for treating patients. Statins are drugs that decrease lipids and have proven very useful for a long time in lowering the number of deaths and illnesses caused by atherosclerosis. Having high levels of lipids in your blood, high blood pressure, diabetes, being overweight, smoking, being a man, living a sedentary lifestyle with little physical activity, and eating unhealthy foods are all things that can make you more likely to get atherosclerosis. As you get older, your risk of acquiring atherosclerosis also goes up. Ultrasound, magnetic resonance imaging, and computed tomography are some of the imaging methods that can be used to see atherosclerotic plaques in blood vessels. The best way to look at coronary vessels is via invasive coronary angiography (ICA). It shows the coronary arteries using X-rays by injecting a contrast chemical into them. Computed tomography coronary angiography (CCTA) is another way to discover those who are at low or medium risk. There are novel hypolipidemic drugs on the market now, like ATP-citrate lyase (ACL) inhibitors and proprotein convertase subtilisin/kexin 9 (PCSK9) inhibitors. These medications are helping the business grow. Antihypertensive and hypoglycemic drugs are also commonly used to treat typical risk factors for atherosclerosis.

Nanomedicine is the use of nanotechnology to manufacture medicines. Some people think that it is a useful way to treat diseases that don't respond to current treatments, like atherosclerosis. First, nanomedicine is good for delivering a lot of different anti-atherosclerosis drugs, like proteins, peptides, nucleic acids, and chemical compounds. Nanomedicine may also be easily modified to vary its physical and chemical properties so that it can be employed in different pathological situations in atherosclerosis. Nanomedicine not only makes loaded medicines more stable and enhances their pharmacokinetic qualities, but it also makes passive targeting easier and inflammatory endothelium permeability better. This means that more drugs can build up in plaques. Nanomedicine might also be able to target a number of cells and molecules that are involved in the pathophysiological process of atherosclerosis. These include cytokines, receptors on cell surfaces, and proteins that send signals. More importantly, the atherosclerotic milieu has a lot of unique properties, such as a high level of ROS, a low pH, shear stress, and specific enzymes. These make it an excellent area for drug delivery systems that respond to stimuli. Nanomedicine has been very popular in the past few years for treating and reversing atherosclerosis because of the great outcomes it has gotten in studies.

The first thing that happens to an arterial vessel that causes atherosclerotic plaque to form is damage to the endothelial cells, which are the cells that border the vessel from the lumen. The damaged endothelium causes the adhesion molecules ICAM-1 and VCAM-1 work harder, which makes it easier for leukocytes like monocytes to attach to the wall of the blood vessel. The deepest layer of the vessel wall is called the intima, and LDL particles enter inside it. Reactive oxygen species (ROS) cause LDL to oxidize (oxLDL), which is a key step in commencing the inflammatory response. After that, monocytes go to the intima and become macrophages. When macrophages ingest oxLDL, they transform into foam cells, which are cells that are loaded of fat deposits, as seen in the diagram. When foam cells die, they produce a necrotic core that speeds up the death of other cells. These items make the inflammation worse and the plaque that causes atherosclerosis less stable. Vascular smooth muscle cells (VSMCs) from the media layer begin to develop and move into the intima. These cells help form the fibrous cap that protects the plaque. When the endothelium is damaged, platelets become active and form thrombus, which can block or slow down blood flow.

2.MACROPHAGE- TARGETED NANOPARTICLES

Macrophages are particularly crucial in the development of atherosclerosis because they induce lesions to start and proliferate through long-term inflammation and issues with lipid metabolism. Proinflammatory (M1) macrophages create too many cytokines and reactive oxygen species (ROS) once they are recruited and differentiated in the arterial wall. Foam cells form when cholesterol builds up in their bodies. This process turns on pathways that send inflammatory signals, notably those that include Toll-like receptors (TLRs), the NLRP3 inflammasome, and NF-κB. This generates an inflammatory

loop that continues getting bigger. Modern nanoplatforms can contain advanced payloads like siRNA, miRNA, or proteins to selectively intervene in macrophage signalling pathways, therefore lowering their proliferation and enabling inflammation resolution . For instance, a study that used Fe3O4@M2 NPs (nanoparticles covered with M2-type macrophage membranes) found that fluorescence and MRI can be used to see atherosclerotic lesions quite well. The M2 membrane coating made it easier for active macrophages to take up the platform, which showed that it is highly safe and useful for diagnostic application. .

3.ENDOTHELIAL CELL -TARGETED NANOPARTICLES

One of the key elements that makes atherosclerotic plaque form is damage to the vascular endothelium. When the endothelium is hurt, it makes adhesion molecules like VCAM-1 and ICAM-1 more active. This makes it easier for monocytes to get in and become foam cells full with lipids. This rise in surface molecules is a clear potential for nanotherapeutics that target specific cells.

For example, MM/RAPNPs are biomimetic nanoparticles made of a rapamycin-PLGA core coated in a macrophage membrane. They were able to successfully target activated endothelial cells, build up in lesions, and halt the progression of disease in a mouse model. LFP/PCDPD is another nanoparticle that can do a lot of things. It was created to target VCAM-1 and CD44. It could take images of lesions, get rid of lipids, and release the anti-inflammatory drug prednisolone when local ROS levels were high. This made the treatment incredibly targeted. TM-GW micro micelles also went after VCAM-1. They gave a PPARδ receptor agonist to aortic smooth muscle cells that were under oxidative stress to better control apoptosis and migration.

4. VASCULAR SMOOTH MUSCLE CELLS (VSMC'S)-TARGETED NANOPARTICLES

Vascular smooth muscle cells (VSMCs) are very important and have a lot of diverse duties to do during the life of an atherosclerotic plaque. They help with inflammation, changing the extracellular matrix, and eventually, keeping the plaque stable or making it unstable. VSMCs can change their shape and function in many ways, such as by turning into cells that look and act like macrophages or osteochondrogenic cells. In an advanced plaque, these cells can compose up to 70% of all the cells.

Changing the phenotype and development of VSMCs has been an important therapy aim. One way to do this is to provide the VSMC miRNA, such miR-145, to modulate how it contracts. Researchers observed that micelle-type nanoparticles that carried miR-145 and were directed at the CCR2 receptor on sick VSMCs were able to restore their protective phenotype and halt lesions from occurring in mice. Another interesting method uses functionalized nanoparticles, such as GW1516@NP-OPN, to target proteins that are overexpressed, such as osteopontin (OPN). These particles let off a PPARδ agonist that halted VSMC recruitment and apoptosis, which made the lesion area get a lot smaller. There are different nanosystems being produced to deliver miRNA, but carriers made from living things are becoming more common. To do this, extracellular vesicles (EVs) designed with the MCP-1 peptide to carry miR-145 worked exactly as well as synthetic carriers, but they only carried 25,000 times less miRNA, which made the danger of side effects much lower. .

5.LOWERING LDL LEVELS

Atherosclerosis is caused by long-term inflammation and the buildup of lipoproteins, predominantly LDL, in the walls of blood vessels. Statins, ezetimibe, and PCSK9 inhibitors are all common ways to lower LDL cholesterol levels, but they don't lower the risk of heart issues. Nanoparticle-based therapy systems have become more popular in recent years because they let drugs be delivered exactly where they need to go and improve the pharmacokinetic profile of existing active substances.

At the same time, researchers are developing nanoparticle-based vaccines that target PCSK9. PCSK9 is a protein that breaks down the LDL receptor (LDLR), which raises the amount of LDL in the blood. One way to do this is to mix the catalytic domain of PCSK9 with ferritin nanoparticles that can build themselves. This makes the immune system react and make antibodies against PCSK9, which lowers lipids and stops atherosclerosis in multiple mice models. Liposomal vaccinations (L-IFPTA+) had similar effects, causing a long-lasting immune response and lowering LDL levels in animals that were eating a diet that induced atherosclerosis.

Nanotechnology is also being used to modify drugs that are already available. Statins do work, but not very well because they don't dissolve well in water, don't stay in the body very long, and break down quickly. These difficulties can be avoided by using new therapeutic procedures that target the lipid component very precisely and just in certain areas. Nanoparticles can let the drug stay in the body longer, which helps it build up and spread to the tissues it needs to reach. This gives the most advantage to the treatment with the least harm to the rest of the body. Researchers are working on nanosystems such polymer nanoparticles, lipid-based nanoparticles, chitosan nanoparticles, nanoliposomes, nanoemulsions, nanotransferomal carriers, self-emulsifying systems, and cerium oxide nanoparticles to do this. One example is statins that are encased in PLGA nanoparticles that break down over time. These nanoparticles help the medicine stay stable, release it more slowly, and perform better at lower doses. .

6.ANTI- INFLAMMATORY AND ANTI- OXIDATIVE ACTING

Atherosclerosis (AS) is a long-term inflammatory disease of the artery intima that happens when lipids and inflammatory cells like macrophages, mast cells, and T lymphocytes build up. These cells produce cytokines, which in turn form reactive oxygen species (ROS). ROS assist smooth muscle cells move and make plaque that builds up in arteries. Too much ROS damages DNA in the mitochondria and nucleus and activates the MAPK, NF-κB, and JAK/STAT pathways, which kills cells. Mitochondria are the principal source of ROS, but other organelles, like peroxisomes and the endoplasmic reticulum, can also make them. You can make them with or without enzymes. The NF-kappaB (NF-κB) pathway can be turned on by ROS. This can cause the release of inflammatory substances such vascular cell adhesion molecule-1 (VCAM-1), interleukin-6 (IL-6), interleukin-1β (IL-1β), and other inflammatory substances. It can also make vascular smooth muscle cells (VSMCs) grow, stick to things, and move around. Oxidative stress gets worse because of things in the environment and choices people make. Monocyte membrane-coated nanoparticles (MoNP) are a promising method because they feature proteins that help them stick to the inflammatory endothelium and deliver the therapeutic payload, which in this case is verteporfin. MoNPs were very effective at stopping the formation of atherosclerotic lesions and lowering inflammatory infiltrates in mice via blocking the YAP/TAZ pathway. They did this without causing any big problems. The LLC NPs system, which has low-molecular-weight heparin (LMWH), lipoic acid (LA), and curcumin (Cur), is another notable example of a blend of anti-inflammatory and antioxidant actions. When these nanoparticles encounter ROS, they can identify P-selectin on endothelial cells and release Cur. Curcumin, a powerful antioxidant, made the treatment function better. It halted atherosclerotic lesions from growing in mice by a lot. .

7. CHALLENGES AND FUTURE DIRECTIONS IN NANOPARTICLE -BASED ATHEROSCELROSIS THERAPY

Nanotechnology could be very helpful in treating atherosclerosis, but there are still several huge issues that need to be worked out before it can be employed in real life. One of the biggest worries is nanotoxicity. This includes negative immune responses, oxidative stress, and effects that aren't what they were supposed to be. Researchers have discovered that some nanoparticles may actually make atherosclerosis worse. This is surprising because many nanomaterials, such as lipid- or polymer-based nanoparticles, are supposed to be harmless for living organisms. For example, amorphous silica nanoparticles have been shown to make macrophages get into plaques more easily, stress the endoplasmic reticulum, and boost levels of LDL and triglycerides in the blood. All of these things make the disease proceed faster. Also, employing ferumoxytol nanoparticles throughout the body, even if they are FDA-approved as iron supplements, may cause iron to build up more and make proinflammatory M1-like macrophages in lesions more polarized.

The biodistribution and clearance of nanoparticles are still very critical issues. Uncontrolled buildup in organs that aren't the aim, such the liver or spleen, could make therapies less effective and create safety concerns. Another issue with using nanotherapeutics in medicine is that they are difficult to create and scale up. For industrial-scale manufacture, Good Manufacturing Practice (GMP) requires tight quality control to make sure that each batch has the same size, surface charge, drug loading, and stability. Also, the rules that come with intricate nanostructures make it extra harder to get to the clinical stage. There are also basic biological restrictions because preclinical models and human pathophysiology are not the same. Atherosclerosis develops over the period of weeks in animal models, especially mice. This is not the same as human plaques, which take decades to form and are more varied and complicated. These alterations could be why outcomes from preclinical studies don't always match up with those from humans. People are actively looking at a number of new ways to get past these issues. Targeted nanoparticle systems that are made to attack lesional macrophages or plaque-specific receptors have been demonstrated to be less hazardous to the body as a whole and more effective at treating diseases. You can control when and where drugs are given with nanoparticles that respond to changes in pH, redox environment, or enzyme activity in plaques and release therapeutics in response to these changes. Dual or multi-drug nanocarriers that target different detrimental pathways in atherosclerosis, like cholesterol accumulation and inflammation, may operate better together and give superior outcomes. Also, making new materials, such DNA nanostructures, gives drug carriers that can be tailored, programmed, and precisely functionalized new options. There hasn't been much research on their application in atherosclerosis yet, but their modularity and tunable properties make them quite attractive for future research.

8.CONCLUSION

Nanoparticle-based therapeutics are going to transform how we treat atherosclerosis, going from lowering the risk of the illness as a whole to treating atherosclerotic plaques in a very targeted way. This review has brought to light a number of critical methods that help this transition happen. Nanoparticles that target certain biological actors, such as proinflammatory macrophages, activated endothelial cells, and VSMCs that have changed their phenotype, have been found to be able to affect critical pathogenic pathways with a high degree of specificity. One of the most notable advancements in this field is the use of biomimetic nanoparticles that are hidden from host cells like macrophages or platelets by membranes. This technique not only leverages natural biological targeting mechanisms, but it also helps the immune system stay hidden, which makes the treatment work better by increasing circulation time. Functional nanoparticles that lower LDL, neutralize ROS, or deliver anti-inflammatory medications directly to the lesion microenvironment are also strong, multi-pronged strategies to inhibit plaque formation and improve stability. These are good steps in the right direction, but there are still huge difficulties and things we don't know that need to be fixed before they can be used in real life. Nanotoxicity and long-term biocompatibility are still big issues since some materials can make inflammation worse or build up in organs that aren't the aim, including the liver and spleen, which makes safety a concern. It is very crucial to understand the difference between preclinical models and real human sickness. For instance, the plaques that form in mouse models are usually too simple and grow too quickly to demonstrate how complex and varied human atherosclerosis is, which takes decades to develop. This can often make it hard to connect positive preclinical outcomes with clinical trial results. Finally, there are still major challenges with making things that are GMP-compliant and can be scaled up, as well as figuring out how to follow the rigorous rules for multi-component nanotherapeutics. To avoid

these concerns, future research needs to be carefully planned. We need to quickly make and test better preclinical models, including large animal models or organ-on-a-chip systems, that better show how plaque impacts the body in people. Nanoparticles for the next generation should be able to do more than one thing and react to different things. They should be able to send different medications to different places or only release their payload when particular biochemical signals are present in the plaque. This will make it work best and have the fewest adverse effects. We also need to do a lot of research on the long-term consequences of nanoparticles and how they might be dangerous to living organisms over time. In the end, the effectiveness of nanomedicine in treating atherosclerosis in the clinic will likely depend on a tailored approach, where the treatment regimens are based on the specific cellular and molecular makeup of a patient's plaque. Nanoparticle-based platforms offer a lot of potential to make atherosclerosis a curable and maybe even reversible condition by directly addressing these issues.

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