OPEN ACCESS Freely available online

Anatomy and Physiology : Current Research

Perspective

The Interplay of Low Protein and Iron Metabolism in Atherosclerosis: Pathogenesis and Therapeutic Implications

Alice Willow*

Department of Pharmaceutical Sciences, The University of Tennessee Health Science Center, Memphis, USA

DESCRIPTION

Atherosclerosis is a chronic inflammatory disease characterized by the progressive buildup of plaques within arterial walls, leading to narrowing and hardening of the arteries. It is a major cause of cardiovascular diseases, including heart attacks and strokes, which are leading causes of mortality worldwide. While traditional risk factors such as high cholesterol, hypertension, and smoking are well-established, emerging research suggests that altered protein and iron metabolism may also play a significant role in the development and progression of atherosclerosis. This article provides an overview of the relationship between low protein and iron metabolism and the pathogenesis of atherosclerosis.

Protein metabolism and atherosclerosis

Cholecystitis Proteins are essential macromolecules involved in various physiological processes, including lipid metabolism, inflammation, and coagulation. Dysregulation of protein metabolism, specifically low protein intake or impaired protein synthesis, has been associated with an increased risk of atherosclerosis. Studies have shown that insufficient dietary protein intake or conditions that lead to protein malnutrition can contribute to endothelial dysfunction, impaired lipid metabolism, and increased oxidative stress, all of which promote the development of atherosclerotic plaques.

Low protein intake has been linked to alterations in lipoprotein metabolism, specifically decreased levels of High-Density Lipoprotein (HDL) cholesterol, commonly referred to as "good" cholesterol. HDL cholesterol plays a crucial role in reverse cholesterol transport, which involves removing excess cholesterol from peripheral tissues and transporting it back to the liver for excretion. Reduced HDL cholesterol levels impair this process, leading to the accumulation of cholesterol within arterial walls and the initiation of atherosclerosis.

Furthermore, low protein intake has been associated with increased levels of pro-inflammatory cytokines, such as Interleukin-6 (IL-6) and Tumor Necrosis Factor-Alpha (TNF-α). Chronic inflammation contributes to endothelial dysfunction, characterized by impaired nitric oxide production and increased adhesion molecule expression, facilitating the adhesion and migration of monocytes into the arterial wall. Once in the intima, monocytes differentiate into macrophages, which take up oxidized Low-Density Lipoprotein (LDL) particles, forming foam cells, a hallmark of early atherosclerotic lesions.

Iron metabolism and atherosclerosis

The Iron is an essential micronutrient involved in several physiological processes, including oxygen transport, energy production, and DNA synthesis. However, excessive iron accumulation or impaired iron metabolism can lead to oxidative stress, endothelial dysfunction, and inflammation, all of which contribute to atherosclerosis.

Iron overload, as observed in conditions such as hereditary hemochromatosis or excessive dietary iron intake, leads to the generation of Reactive Oxygen Species (ROS) through the Fenton reaction. ROS can promote lipid peroxidation, damage DNA, and trigger inflammation within the arterial wall. Additionally, excess iron can directly stimulate the expression of adhesion molecules and promote the adhesion of monocytes to endothelial cells, facilitating their entry into the arterial intima.

Conversely, iron deficiency has also been associated with an increased risk of atherosclerosis. Iron deficiency anemia leads to reduced oxygen-carrying capacity, forcing the heart to work harder and potentially inducing cardiac hypertrophy and remodeling. Iron deficiency can also impair mitochondrial function and reduce antioxidant defenses, promoting oxidative stress and endothelial dysfunction.

Interplay between protein and iron metabolism

The Protein and iron metabolism are interconnected, and disruptions in one can influence the other. For example, iron is required for the synthesis of several proteins involved in lipid metabolism, including lipoprotein lipase, which hydrolyzes triglycerides from circulating lipoproteins. Iron deficiency can impair the activity of these proteins, leading to dyslipidemia.

Correspondence to: Alice Willow, Department of Pharmaceutical Sciences, The University of Tennessee Health Science Center, Memphis, USA, E-mail: willalice@Oxford.edu

Received: 28-Apr-2023, Manuscript No. APCR-23- 24690; **Editor assigned:** 02-May-2023, Pre QC No. APCR-23- 24690(PQ); **Reviewed:** 16-May-2023, QC No. APCR-23- 24690; **Revised:** 23-May-2023, Manuscript No. APCR-23- 24690(R); **Published:** 30-May-2023, DOI:10.35248/2161-0940.23.13.428

Citation: Willow A (2023) The Interplay of Low Protein and Iron Metabolism in Atherosclerosis: Pathogenesis and Therapeutic Implications. Anat Physiol. 13:428.

Moreover, protein malnutrition can affect iron metabolism. Proteins such as transferrin and ferritin are responsible for transporting and storing iron in the body. Insufficient protein intake can lead to decreased production of these iron-binding proteins, resulting in impaired iron transport and storage. Consequently, iron availability for essential processes, including the synthesis of enzymes involved in antioxidant defense mechanisms, may be compromised. This can further exacerbate oxidative stress and inflammation, contributing to the development of atherosclerosis.

Interestingly, emerging evidence suggests that specific proteins involved in both protein and iron metabolism may directly influence atherosclerosis development. For instance, Apolipoprotein B (apoB), a protein present in LDL cholesterol particles, has been implicated in the progression of atherosclerosis. Iron-induced oxidative modifications of apoB can enhance its pro-atherogenic properties, promoting LDL cholesterol retention within the arterial wall and subsequent foam cell formation. These findings highlight the intricate relationship between protein, iron, and lipid metabolism in atherosclerosis pathogenesis.

Therapeutic implications

Understanding the role of protein and iron metabolism in atherosclerosis opens avenues for potential therapeutic interventions. Modulating protein intake and optimizing iron status may have beneficial effects in preventing or managing atherosclerosis. Ensuring an adequate dietary protein intake, particularly sources rich in essential amino acids, can help maintain endothelial function, lipid metabolism, and immune response, reducing the risk of atherosclerotic plaque formation.

Regarding iron metabolism, iron chelation therapy has shown potential in reducing iron overload-associated oxidative stress and inflammation. In individuals with iron deficiency, iron supplementation can correct the deficiency and potentially alleviate the associated cardiovascular risks. However, careful monitoring and individualized treatment approaches are necessary, as both iron deficiency and excess can have adverse effects.

Additionally, targeting specific proteins involved in protein and iron metabolism pathways could be explored. For instance, therapies aimed at modulating lipoprotein metabolism, such as increasing HDL cholesterol levels or promoting reverse cholesterol transport, may be beneficial in preventing or treating atherosclerosis. Similarly, strategies targeting iron-regulating proteins, like hepcidin, could help maintain iron homeostasis and reduce iron-induced oxidative stress and inflammation.

CONCLUSION

The relationship between low protein and iron metabolism and the development of atherosclerosis is an emerging area of research. Dysregulation in protein and iron metabolism can contribute to endothelial dysfunction, inflammation, oxidative stress, and lipid abnormalities, all of which play critical roles in atherosclerotic plaque formation and progression. Understanding the interplay between protein and iron metabolism pathways provides valuable insights into the pathogenesis of atherosclerosis and offers potential therapeutic avenues to mitigate the disease burden. Further research is needed to elucidate the underlying mechanisms fully and translate these findings into clinical practice, ultimately improving the prevention and management of atherosclerosis and its associated cardiovascular complications.