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ENDOTHELIAL DYSFUNCTION IN KAWASAKI DISEASE: FROM ACUTE INFLAMMATION TO LONG-TERM CARDIOVASCULAR SEQUELAE

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ABSTRACT

Kawasaki disease primarily affects children under 5 years of age. Lesions primarily involve generalized inflammation of small and medium-sized vessels. Structural and functional changes in the coronary arteries make this disease the most common coronary artery disease in children. The disease involves destruction of the vascular endothelium, leading to remodeling and the formation of aneurysms, among other conditions. We are learning more and more about the factors causing this damage. Currently, inflammatory mediators, immune cells, and oxidative stress are involved in the pathogenesis of endothelial destruction. Pyroptosis, leading to the death of vascular endothelial cells, also appears to be important. Numerous genetic changes promoting these changes in non-coding miRNA sequences have also been described. There is a noticeable connection between changes occurring in the coronary vessels and subsequent cardiovascular incidents, especially when diseases such as hypercholesterolemia, diabetes and atherosclerosis occur. Because the disease was first described in 1967, data on its long-term effects are still being collected.

KEYWORDS

Kawasaki Disease, Vasculitis, Endothelial Dysfunction, Cardiovascular Sequelae in Kawasaki Disease

CITATION

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Introduction

Kawasaki disease is an acute systemic vasculitis, was first described by Dr. Tomisaku Kawasaki in 1967. Generally, the highest prevalence is observed in Asia. Moreover, the highest number of patients is reported in Japan, South Korea, and China. It most commonly occurs in children under 5 years of age, many authors consider it the most common coronary artery disease in childhood. [1,8] This disease mainly affects small and medium-sized vessels, within which aneurysms may develop. [1,2,3,6,7,9] Endothelium lining the inner surface of vessels is in direct contact with circulating inflammatory cells, through it, molecules capable of damaging the outer layers. Endothelial cells can also secrete pro-inflammatory cytokines. There are several genes involved in the activation of inflammation, namely NOD1 and NLPR1. These genes participate in the development of Kawasaki disease and regulate autophagy. Endothelial dysfunction is systemic in nature. [1,2,3,9] The wellbeing of blood vessels also depends on nitric oxide (NO) produced by the single-layer endothelium, which limits inflammation, cell proliferation, and thrombosis, thereby enabling laminar blood flow and providing protective effects on the deepest layer of the vessel wall. [2] In children with Kawasaki disease, independent risk factors associated with predicting the development of coronary artery lesions were identified, including prolonged fever periods and an increased neutrophil-to-lymphocyte ratio. The molecular cause of the aforementioned damage is the oxidation of low-density lipoproteins and the activation of signaling via receptors. Experimental studies on human umbilical cord mesenchymal stem cells show that their regulation of CD54 and CD105 expression levels in vascular endothelial cells in Kawasaki disease can reduce the severity of inflammatory processes and decrease endothelial damage, which may offer hope for their use in treatment. [1] It is important to understand how the described changes affect the development of coronary artery damage and other vessels, which will make it possible to prevent serious disruptions of homeostasis and select the appropriate therapy, which is what we will focus on in this article.

Methodology

The aim of this paper is to review the latest medical reports on the impact of endothelial dysfunction on the development of Kawasaki disease and its complications. To provide a thorough understanding and presentation of this topic, review articles, research papers, and meta-analyses were reviewed using a standard PubMed search using the phrase "Kawasaki disease, endothelial dysfunction". Sixty-four search results were found for fully accessible free articles from 2018-2025, from which those describing the role of the vascular endothelium in the pathophysiology of this condition were selected.

Results

The functional and structural changes in the vessels observed in Kawasaki disease lead to changes in the coronary arteries, such as coronary artery aneurysm or dilatation. [1,2,3,4,9] These changes are the most common serious complications that can cause thrombosis, which in older patients can lead to myocardial infarction or coronary artery disease. [1,2,9] When examining autopsy samples from patients with Kawasaki disease, pathophysiological processes leading to vascular damage, such as necrotizing arteritis, subacute or chronic vasculitis, and inflammation of the lumen of fibroblasts, were discovered using light microscopy and transmission electron microscopy. [2] Necrotizing arteritis is a self-limiting, neutrophilic endothelial cell process that gradually destroys the homeostasis of the intima, media, and extrinsic parts of the coronary artery. [2] Wall destruction leads to the development of cystic aneurysms, which increases the risk of rupture and leads to thrombosis. [1,2,4,8] This is a common cause of premature death in patients with this disease. [2] The involvement of inflammatory factors, which accumulate in the endothelial cells of coronary arteries, is crucial for understanding the pathophysiology of these disorders. [2] These factors then act on certain receptors and regulate factors that initiate vascular inflammation. [2] A significant association has also been observed in the Chinese pediatric population between the rs2069952, rs9574, and rs1415774 gene endothelial protein C receptor and a higher risk of Kawasaki disease. [2]

The inflammatory phase od coronary vasculitis lasts the first 6 weeks. Histopathological changes first appear in the adventitia and intima, after which inflammatory cells penetrate the media, at which point the entire coronary artery wall becomes inflammatory. [4] Due to significant damage to the arterial components, the artery begins to dilate approximately 10-12 days after the onset of Kawasaki disease. [4] The inflammatory cell infiltration lasts about two weeks, then gradually subsides, forming scar that heal after approximately 40 days of illness. [4] A whole-body examination for Kawasaki disease, which assessed the presence of systemic vasculitis, revealed that vascular destruction, such as dilatation or aneurysm, may be present in large arteries, particularly the subclavian, axillary, brachial, and iliac arteries. [4] It was noted that the vasculitis in Kawasaki disease is synchronous throughout the body. [4]

Cardiovascular Events

Endothelial dysfunction and destruction resulting from the inflammatory response leads to vascular wall dysfunction, which is important in the development of atherosclerosis. [4] Elevated and altered levels of low-density lipoprotein cholesterol also play a significant role in the development of atherosclerotic lesions. [4] The concomitant presence of Kawasaki disease promotes the development of atherosclerosis, which increases the risk of cardiovascular events. [4]

Pathophysiologically, endothelial dysfunction in Kawasaki disease is the result of chronic inflammatory activation and vascular damage, which may contribute to the subsequent progression of cardiomyopathy, hypertension, and other cardiovascular complications, regardless of the presence of coronary artery lesions. [14] The pathogenesis of endothelial dysfunction in Kawasaki disease (KD) is associated with the activation of endothelial cells by biologically active factors present in patient serum, such as cytokines and VEGF. [15] Activation of the Ca^{2+}/NFAT pathway leads to the translocation of NFATc1 and NFATc3 to the cell nucleus, which increases the expression of adhesion molecules, including nVCAM-1, E-selectin, and inflammatory mediators such as MCP-1 and tissue factor. [15,16] This results in increased leukocyte adhesion, an enhanced inflammatory response, and vascular wall damage. [15] These disorders lead to endothelial proliferation and excessive angiogenesis, which alters vascular homeostasis. Genetic predispositions also exist, such as mutations in the Ca^{2+}/NFAT pathway genes, increasing susceptibility to its excessive activation. [15,16]

It is not controversial that the stiffness of vessels in which atherosclerotic plaques appear is significantly increased. [4] The previously mentioned occurrence of systemic vasculitis in patients with Kawasaki disease is believed to place these patients at increased risk of developing systemic atherosclerosis involving multiple vessels. [4]

The production of proinflammatory mediators, including IL-1, IL-6, tumor necrosis factor (TNF) and vascular endothelial growth factor (VEGF), causes inflammation and tissue destruction and is the basis of coronary artery endothelial damage associated with Kawasaki disease. [5,8,16] In the acute phase, endothelial reorganization may occur even in the absence of known coronary vessel abnormalities. [1,2,5,8,16]

Oxidative Stress

Oxidative stress is associated with a wide variety of pathophysiological changes in cardiovascular disease, such as endothelial dysfunction, vascular inflammation, and atherosclerosis. [5] In areas where active inflammation occurs, inflammatory cells, endothelial cells, and vascular smooth muscle cells release reactive oxygen species, chemical mediators, and enzymes, which collectively contribute to oxidative stress. [5] Molecules affected by this phenomenon, such as oxidized low-density lipoprotein, are also involved in regulating nuclear-kappa B factor (NF-kB) activation, thrombosis, inflammation, angiogenesis, endothelial function, and immune tolerance. [5] Ox-phospholipids are implicated in the development of coronary artery pathology associated with Kawasaki disease, where increased expression of an adhesion molecule called endothelial E-selectin plays a significant role. [5] In a clinical study, children with Kawasaki disease (KD) had elevated levels of reactive oxygen metabolite derivatives, reflecting oxidative stress and endothelial dysfunction, confirmed by decreased percent flow-mediated dilation. [13] Levels of reactive oxygen metabolite derivatives were higher in both patients with and without vascular complications, and longer duration of fever independently correlated with lower percent flow-mediated dilation, suggesting that chronic inflammation and free radical overproduction increase the risk of early cardiovascular events. [13]

Structural and functional changes of the endolium

It has been discovered that the vascular endothelium of patients with Kawasaki disease is characterized by high expression of neurocapillary protein 1 and VEGF receptor 2, which interact with angiopoietin-like protein 4 and VEGF, respectively, leading to increased endothelial permeability. [5,16] Vascular endothelial cell dysfunction is associated with non-coding miRNA sequences that participate in post-transcriptional protein expression. These include miR-233, miR-197-3p, miR-483, miR-483, miR-27b, SOCS2-AS1, miR-324-5p, miR-320a, miR-320a, miR-145-5p, PINC, miR-125a-5p, miR-186, and miR-93. [1,7] These sequences actively participate in the regulation of cell proliferation, apoptosis, inflammation, autoimmunity, and maintenance of function, which may result in the death of cells that make up the innermost layer of vessels. [1,5] Changes also concern adhesion and infiltration, where platelet endothelial cell adhesion molecule-1 (PECAM-1) plays a key role, which is important because disturbed expression of the PECAM-1 gene may cause atherosclerosis and other vascular diseases. [5] The glycocalyx covering the endothelium may also undergo remodeling, which is another factor facilitating the formation of atherosclerotic plaques. [5]

The mechanism of pyroptosis as a form of vascular endothelial cell destruction

As described earlier, acute inflammation in Kawasaki disease is accompanied by obvious endothelial cell dysfunction and increased levels of cytokines, including interleukin 1ß, which may indicate the involvement of pyroptosis in the pathophysiology of this disease. [6,7] Pyroptosis is a type of proinflammatory cell death that combines the features of necrosis and apoptosis. [1,5,6,7] This phenomenon may be mediated by inflammasome-dependent activation of caspase-113, which is responsible for the cleavage and/or maturation of Gasdermin D (GSDMD), IL-1ß and IL-18. [6] Canonical caspase-1-mediated pyroptosis can be activated by seven types of inflammasomes, including NLRP1, NLRP3, NLRP6, NLRP9, NLRC4, AIM2, and Pyrin13. [6] High-mobility group box 1 (HMGB1) is a highly conserved nuclear protein that acts as an extracellular signal when released by immune or necrotic cells. [6] Released HMGB1 is capable of mediating proinflammatory responses in endothelial cells and inducing cell apoptosis, autophagy, or pyroptosis. [6] Its important role in cardiovascular diseases such as atherosclerosis, diabetic cardiomyopathy and myocardial ischemia/reperfusion injury has been noted. [6] The current area of research is the damage to the endothelium of coronary vessels observed in Kawasaki disease and the role of pyroptosis in this. [6,7]

The role of long non-coding RNAs in the pathological process of vascular changes in Kawasaki disease

Long non-coding RNAs (lncRNAs) are crucial in regulating the pathological process of Kawasaki disease by mediating endothelial cell function and/or the immune response. [7] The lncRNA PINC participates in the tumor necrosis factor (TNF)-α pathway and initiates apoptosis of vascular endothelial cells. [7] High-throughput sequencing technology has led to the identification of more circular RNAs (circRNAs) associated with Kawasaki disease. [7] Studies have shown that serum levels of circRNAs in patients with this disease were lower before treatment than in controls but higher after treatment. [7] Little is known about the mechanism of action of circRNAs in Kawasaki disease, but it is known that they can induce endothelial–mesenchymal transition (EndoMT). [7,16] EndoMT is responsible for vascular structure damage, remodeling and fibrosis. [7,16] Recently, there is increasing evidence implicating EndoMT in cardiovascular diseases, including atherosclerosis, pulmonary hypertension, valve disease, and fibroelastosis. [7] Therefore, these sequences should be further studied in the hope of better understanding this disease.

USP/NFATC1/TLR4/NF-kB axis

In Kawasaki disease, NFATC1 expression is elevated in the serum of patients, leading to endothelial dysfunction through increased apoptosis, oxidative stress, and proinflammatory cytokine secretion. [12,17] The deubiquitinase USP5 stabilizes NFATC1, and its interaction with TLR4 activates the NF-κB pathway, enhancing the inflammatory response of endothelial cells. [12] Inhibition of USP5 or NFATC1 reduces these abnormalities, indicating that the USP5/NFATC1/TLR4/NF-κB axis is a key mechanism of endothelial dysfunction and a potential factor in increasing cardiovascular risk in patients with Kawasaki disease. [12]

The role of endothelial dysfunction and destruction in cardiovascular diseases.

Oxidative stress, pathological increase in vascular endothelial permeability, endothelial cell death, increased adhesion and infiltration of vascular endothelium, and disturbance of the structure and balance of endothelial glycocalyx are components of vascular endothelial damage associated with Kawasaki disease, the chronic nature of which is associated with an increased risk of age-related vascular diseases. [5,9]

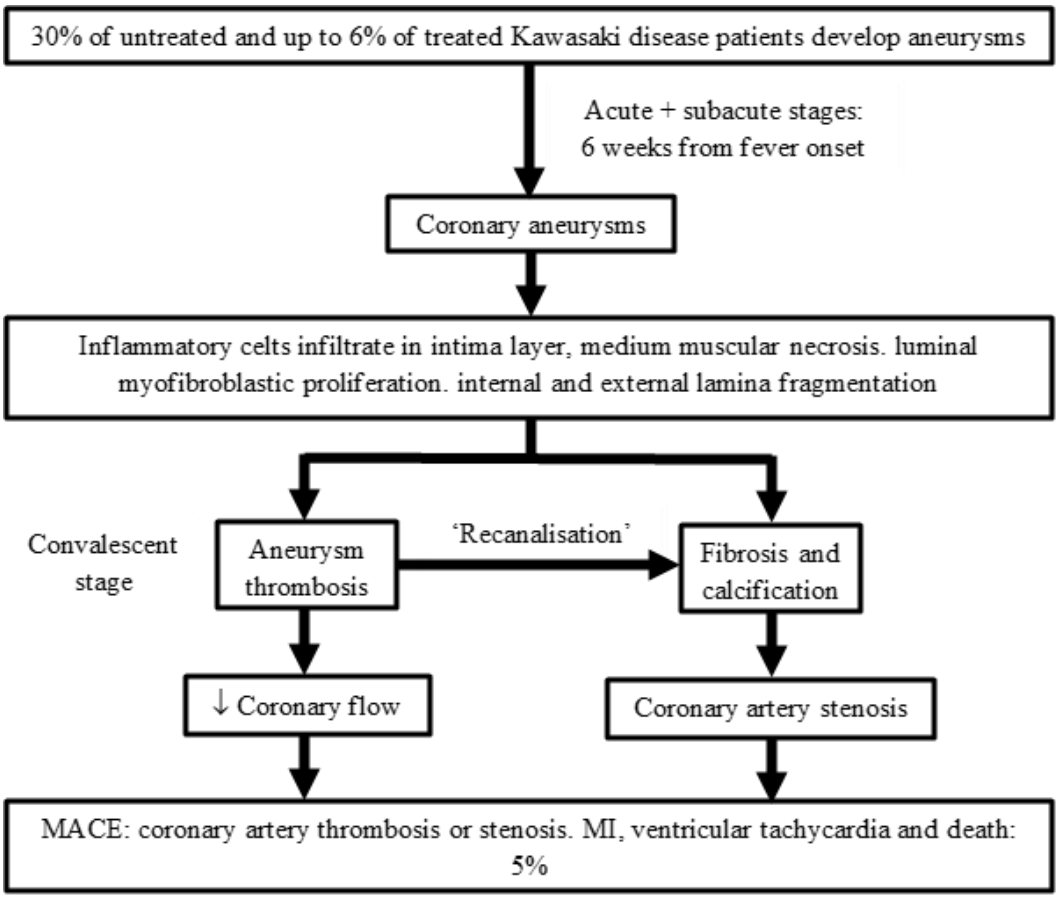

Fig. 1. *Coronary artery in Kawasaki disease*

Coronary artery in Kawasaki disease – schematic description

In Kawasaki disease, dynamic and multi-stage alterations occur in the coronary arterial wall, untimately leading to aneurysm formation, trombosismm stenosis, and vascular remodeling. The presented schematic illustrates these changes from the acute to the convalescent stage, encompassing typical inflammatory and reparative events. During the acute and subacute stages, lasting up to six weeks from fever onset, intense inflammation develops in the vessel wall. The intima shows inflammatory cell infiltration composed of lymphocytes, macrophages, and neutrophils, accompanied by necrosis of the medial smooth muscle and fragmentation od internal and external elastic laminae. These events weaken the arterial wall, predisposing it to dilation. Endothelial permeability increases, and pro-inflammatory cytokines such as IL-7, TNF-α, and IL-1β drive endothelial injury. Endothelial dysfunction results in loss of barrier integrity, leukocyte adhesion, and platelet activation, promoting trombotic changes.

Structural degradation of the vascular wall during the acute phase leads to the development of coronary artery aneurysms. These may be single or multiple, most frequently located in the proximal segments of the right or left coronary arteries. In untreated patients, coronary aneurysms occur in approximately 30%, while timely immunoglobulin therapy reduces this incidence to 5–6%. Disturbed flow within aneurysmal segments fosters thrombosis formation, and occlusive thrombi can cause myocardial ischemia, infarction, or sudden death.

In the subacute and healing phases, reparative processes dominate, including intimal myofibroblast proliferation, collagen deposition, and fibrotic and calcific transformation of the vessel wall. These lead to

coronary artery stenosis, often occurring at sites of prior aneurysms or unaffected segments. Fibrotic and calcified areas cause loss of arterial elasticity, reducing vasomotor responsiveness. Partial recanalization of previous thrombotic occlusions may occur, yet structural remodeling persists.

The convalescent stage is characterized by the resolution of inflammation, though fibrotic remodeling remains evident. Persistent endothelial dysfunction and early atherosclerotic features have been documented by vascular imaging studies assessing coronary flow. Although the risk of major complications such as recurrent thrombosis or sudden cardiac death is reduced, it is not completely eliminated.

In summary, the schematic reflects the full spectrum of pathological changes within coronary arteries during Kawasaki disease: from acute necrotizing vasculitis to aneurysm and thrombosis, followed by fibrosis, calcification, stenosis, and partial recanalization. Each stage represents the interplay between vascular injury and repair, with endothelial dysfunction and immune dysregulation as central mechanisms.

Endothelial Function Assessment

Percent flow-mediated dilation (%FMD) can be used as a test of vascular function and also serves as a noninvasive indicator of endothelial function. [17] This method utilizes ultrasound imaging to measure the arterial response during reactive hyperemia. [17] This study demonstrated that patients with Kawasaki disease (KD) and coronary artery aneurysms had significantly reduced endothelial function, as assessed by the systolic (FMDs) and diastolic (FMDd) flow-dependent dilation (FDD) index, compared to healthy children. [11,17] These changes occurred despite no significant differences in BMI, blood pressure, or traditional cardiovascular risk markers such as cholesterol, glucose, or ADMA levels. [11] The results suggest that patients with CAA undergo a chronic vascular remodeling process, which may predispose them to subsequent cardiovascular events. [11]

Discussion

Kawasaki disease is a condition whose pathophysiology can fascinate physicians of all specialties, from rheumatologists, cardiologists, and radiologists to pathologists and geneticists. Thanks to the growing number of scientific publications, knowledge on the topic we study is expanding. A better understanding of the pathological phenomena requires further research, which will provide diagnostic tools, establish new treatment standards, and improve patient outcomes. In our article, we focus on changes in the vascular endothelium, particularly those in the coronary arteries. We demonstrate the mechanisms of structural and functional damage to the inner vessel wall and demonstrate how comorbidities contribute to the development of these conditions. The search for markers of endothelial damage in Kawasaki disease continues.

Conclusions

Kawasaki disease, which manifests as vasculitis within the coronary arteries, may increase the risk of cardiovascular events in association with chronic diseases such as diabetes, hypertension, and hypercholesterolemia. Many proinflammatory cytokines circulating during the active inflammatory phase damage the vascular endothelium, altering its permeability and destroying the structure of the outer arterial walls. Studies indicate that %FMD may soon be used for early assessment of cardiovascular risk in patients with Kawasaki disease.

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