The American Journal of Medical Sciences and Pharmaceutical Research ISSN 2689-1026 Volume 08 - 2026

Physiology of Blood Stasis

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Received: 22 April 2026 | Received Revised Version: 08 May 2026 | Accepted: 16 May 2026 | Published: 08 June 2026

Volume 08 Issue 06 2026 | Crossref DOI: 10.37547/tajmspr/Volume08Issue06-12

Abstract

Blood stasis is a complex pathophysiological process characterized by a significant slowdown or complete cessation of blood flow in the microcirculatory vessels. In recent years, researchers have focused on the mechanisms of interaction between blood cells, endothelial cells, and vascular regulatory factors.

Keywords: Vascular disorders, capillaries, erythrocytes, microcirculation, ischemia.

Cite This Article: Kamalova Zilola Mirsabitovna. (2026). Physiology of Blood Stasis. The American Journal of Medical Sciences and Pharmaceutical Research, 8(06), 77–80. https://doi.org/10.37547/tajmspr/Volume08Issue06-12

1. Introduction

The human circulatory system ensures the continuous supply of oxygen and nutrients to tissues, as well as the removal of metabolic waste products. Maintaining adequate blood flow is essential for cell survival. Any disruption to blood flow inevitably affects the functional state of organs and tissues. Among the various forms of vascular disorders, blood stasis—a condition in which blood flow within the microvasculature slows significantly or ceases entirely—holds a significant place. This phenomenon can occur in both localized pathological processes and severe systemic diseases.

Studying the physiology of blood stasis is crucial for understanding the mechanisms underlying ischemia, thrombosis, inflammation, and organ failure. As early as the 19th century, the German pathologist Rudolf Virchow highlighted the role of impaired blood flow in the development of vascular complications. Modern research has significantly expanded our understanding of the nature of this process, establishing that changes in blood rheology, the functional state of the endothelium, and the activity of cellular elements play a key role.

In clinical practice, stasis is most frequently observed in cases of chronic venous insufficiency, diabetes mellitus, septic conditions, severe infections, heart failure, and certain forms of shock. Stasis impairs oxygen delivery to tissues, increases blood viscosity, and enhances erythrocyte and platelet aggregation. Simultaneously, mechanisms of inflammation and vascular wall injury are activated. All of this creates a vicious cycle of pathological changes.

According to data from the Ministry of Health of the Republic of Uzbekistan and national statistical surveys conducted in recent years, diseases of the circulatory system remain a leading cause of mortality. A significant proportion of complications associated with these conditions stems specifically from microcirculatory disorders. In regions with a high prevalence of diabetes mellitus and arterial hypertension, the incidence of microangiopathies continues to rise. Such trends are observed not only in Uzbekistan but also in many Central Asian countries.

The physiology of stasis is of interest not only to

pathophysiologists but also to cardiologists, surgeons, endocrinologists, and intensive care specialists. Understanding the mechanisms of its development enables more effective prevention of vascular complications and the development of new treatment methods for patients with microcirculatory disorders.

This study takes the form of an analytical review of contemporary scientific literature concerning the physiology and pathophysiology of blood stasis. The analysis utilized publications by domestic and international authors indexed in international scientific databases between 2010 and 2025. Particular attention was paid to studies examining the mechanisms of microcirculatory impairment, alterations in blood rheological properties, and the role of endothelial dysfunction in the development of stasis. Methods of comparative analysis, systematization, and critical evaluation of scientific data were employed throughout the study.

The data were compared with the results of modern clinical observations regarding cardiovascular diseases, endocrine pathologies, and inflammatory processes. Additionally, statistical data reflecting the prevalence of diseases associated with impaired microcirculation were analyzed.

To evaluate the physiological mechanisms of stasis, data were examined regarding the condition of the vessel wall, the dynamics of blood cells, and hemodynamic changes in various segments of the microcirculatory bed

The state of the endothelium plays a significant role. Under normal conditions, endothelial cells synthesize substances that inhibit platelet aggregation and ensure adequate vasodilation. Inflammation, hypoxia, or exposure to toxic factors disrupts these functions; nitric oxide production decreases, and the expression of adhesion molecules increases, promoting the attachment of leukocytes and platelets to the vessel wall.

Changes in the rheological properties of blood are a key factor in the development of stasis. Elevated fibrinogen levels, dehydration, and an increased count of formed elements lead to higher blood viscosity. Erythrocytes begin to form so-called "rouleaux" (stacks resembling coins), which impede their movement through the capillaries. Such changes are frequently observed in patients with diabetes mellitus, chronic inflammatory diseases, and metabolic disorders.

Studies show that prolonged stasis leads to severe tissue hypoxia. Oxygen deprivation causes cells to switch to an anaerobic pathway for energy production. This results in the accumulation of lactic acid, alterations in the acid-base balance of tissues, and impaired cellular membrane function. If blood flow is not restored in a timely manner, irreversible structural damage occurs.

Data regarding the relationship between stasis and thrombus formation are of particular interest. Slowed blood flow creates favorable conditions for the activation of blood coagulation factors. Against a background of endothelial dysfunction and increased platelet aggregation, the risk of microthrombus formation rises significantly. This is confirmed by studies involving patients with severe infections, sepsis, and cardiovascular disease. An analysis of the literature leads to the conclusion that blood stasis cannot be viewed solely as a local vascular phenomenon.

It is a complex biological process involving mechanisms of hemodynamics, cellular metabolism, inflammation, and hemostasis. For this reason, the study of its physiological underpinnings remains a relevant area of modern medical science.

The physiology of blood stasis remains one of the most complex yet fundamental issues in modern pathophysiology. Despite significant progress in the study of microcirculation, many aspects of the development of stasis continue to be the subject of scientific debate.

According to several studies, even moderate endothelial damage can trigger pronounced changes in microcirculation. Hypoxia leads to the activation of pro-inflammatory cytokines and increased vascular wall permeability, creating conditions conducive to blood cell aggregation. It is noteworthy that the rate at which stasis develops varies significantly depending on the type of vessel and the nature of the damaging factor. This process occurs much more rapidly in capillaries than in arterioles, a phenomenon attributed to the hemodynamic characteristics of the microcirculatory bed.

Accumulated data allow stasis to be viewed as a universal response of the microcirculatory system to injury of various origins.

The role of erythrocytes in the development of stasis warrants particular attention. For a long time, the primary function of these cells was considered to be limited to the transport of oxygen and carbon dioxide; however, modern research demonstrates their active involvement in the regulation of microcirculation. Alterations in the physicochemical properties of blood plasma lead to increased erythrocyte aggregation.

So-called "rouleaux" aggregates (resembling stacks of coins) are formed. These structures significantly impede blood flow through small-diameter vessels. Such changes are particularly common in cases of diabetes mellitus. Studies conducted in various Central Asian countries, including Uzbekistan, indicate an increased incidence of microangiopathy among patients with long-standing diabetes. Reduced erythrocyte deformability acts as an additional factor contributing to impaired microcirculation.

The less effectively cells deform while passing through capillaries, the higher the risk of local stasis. In some cases, blood flow virtually ceases in specific sections of the microvasculature. The resulting hypoxia further exacerbates tissue damage. A pathological cycle develops in which one abnormality triggers another; for this reason, correcting the rheological properties of blood is considered a key approach to preventing vascular complications.

The link between blood stasis and inflammatory reactions is of significant interest. Many researchers view these processes as mutually reinforcing phenomena. Large quantities of biologically active substances are released at the site of inflammation; interleukins, tumor necrosis factor, histamine, and prostaglandins are of particular importance among them. These compounds alter vascular tone and increase capillary permeability. Simultaneously, leukocyte adhesion to the endothelium rises, and leukocytes begin to migrate through the vessel wall into the tissues. At a certain stage, this process contributes to the slowing of blood flow.

In some cases, microscopic areas of complete cessation of blood flow develop. Such changes are frequently observed in severe infectious diseases and septic conditions. They were particularly evident during the COVID-19 pandemic, when microcirculatory disturbances were identified as a key mechanism underlying damage to the lungs and other organs. Numerous observations have shown that the severity of stasis is directly linked to the intensity of the inflammatory response. This fact underscores the close interrelationship between the vascular and immune mechanisms of the pathology.

The role of platelets warrants separate consideration. Under normal conditions, these cells maintain the integrity of the vascular wall. Upon endothelial injury, their activity increases sharply. Platelets begin to release substances that promote aggregation and thrombus formation. When blood flow slows, the likelihood of their interaction rises significantly. Consequently, conditions favorable for the formation of microthrombi are created. These processes are particularly pronounced in elderly patients, as the functional reserves of the vascular system naturally decline with age.

Atherosclerosis, arterial hypertension, and lipid metabolism disorders exert an additional influence. This is precisely why the risk of vascular complications is significantly higher in the elderly. Some researchers consider microthrombosis to be a logical progression of long-standing stasis, while others maintain that these processes are independent. Nevertheless, the majority of specialists acknowledge a close link between them. Clinical observations support this view. Tissue hypoxia plays a significant role in the pathogenesis of stasis; insufficient oxygen supply leads to serious metabolic disturbances.

Cells begin to utilize anaerobic energy-generating mechanisms. This is accompanied by the accumulation of lactic acid and a decrease in interstitial pH. Simultaneously, the function of cell membrane ion pumps is impaired. Cellular edema gradually develops. In severe cases, irreversible damage to intracellular structures occurs. Nervous tissue and the myocardium are particularly sensitive to hypoxia. Even brief disruptions in microcirculation can cause serious functional disorders.

This explains the high risk associated with vascular disorders in the central nervous system. Clinical practice includes well-documented cases where even minor microcirculatory disturbances led to severe neurological consequences. Such observations underscore the need for the early diagnosis of blood stasis.

The significance of blood stasis in cardiovascular diseases cannot be overlooked. Cardiovascular pathology currently remains the leading cause of death in many countries worldwide. According to recent national statistics from Uzbekistan, diseases of the circulatory system rank first among the causes of death in the adult population. A significant proportion of complications is associated with microcirculatory disturbances. Chronic heart failure results in reduced efficiency of the heart's pumping function, leading to a slowing of peripheral blood flow.

Particularly pronounced changes develop in the venous system. Venous congestion creates a favorable environment for the development of stasis. The persistence of such abnormalities contributes to the development of trophic disorders and the impairment of internal organ function. These findings suggest that microcirculatory disturbances serve as an important prognostic factor in the course of

cardiovascular diseases.

Blood stasis is a complex, multifactorial process involving virtually all components of the microcirculatory system. Its development involves hemodynamic disturbances, alterations in blood rheological properties, endothelial damage, inflammatory reactions, and activation of the hemostatic system. An analysis of current literature indicates that stasis is not merely a localized vascular disorder; it serves as a significant pathogenetic mechanism in cardiovascular, endocrine, infectious, and inflammatory diseases. Impaired microcirculation leads to tissue hypoxia, metabolic shifts, and the development of structural organ damage.

Of particular importance are the processes of erythrocyte and platelet aggregation, which contribute to the further deterioration of blood flow. The findings underscore the need for the early detection of microcirculatory disturbances and the timely management of risk factors. Future research should focus on improving methods for diagnosing and preventing blood stasis, thereby enhancing the effectiveness of treatment for patients with vascular pathology.

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