MODERN CONCEPTS OF THE HEMOSTATIC SYSTEM AND THE MECHANISMS OF ITS REGULATION

Baykulov Azim Kenjayevich
Samarkand State Medical University, Samarkand, Uzbekistan
Tel: (+99891) 5460369,
E-mail: azimbaykulov81@mail.ru

Abstract. The hemostatic system represents a multifactorial physiological network that maintains a delicate equilibrium between blood fluidity and the ability to form a stable clot in response to vascular injury. Modern concepts of hemostasis emphasize its dynamic and highly regulated nature, involving coordinated interactions among endothelial cells, circulating platelets, plasma coagulation factors, and components of the fibrinolytic system. In this expanded review, particular attention is given to the structural and functional characteristics of the coagulation and anticoagulation pathways, including the central regulatory roles of the protein C system, antithrombin III, and heparin-dependent mechanisms. The endothelial surface is highlighted as an active participant in hemostasis, capable of both promoting and inhibiting thrombin generation depending on physiological conditions. Furthermore, the article examines the molecular basis of fibrin formation and degradation, emphasizing the significance of tissue plasminogen activator and its inhibitors in maintaining fibrinolytic balance. Special focus is placed on the pathophysiology of disseminated intravascular coagulation (DIC), a severe clinical syndrome resulting from systemic activation of coagulation that leads to microvascular thrombosis, consumption coagulopathy, and multiorgan failure. Understanding the regulatory mechanisms of hemostasis provides a framework for improving diagnostic accuracy and developing targeted therapeutic interventions for coagulopathies.

Keywords: hemostasis, endothelium, platelets, anticoagulants, protein C, heparin, disseminated intravascular coagulation (DIC).

Аннотация. Система гемостаза представляет собой многофакторную физиологическую сеть, обеспечивающую тонкое равновесие между текучестью крови и способностью формировать устойчивый сгусток в ответ на повреждение сосудов. Современные концепции гемостаза подчёркивают его динамичный и строго регулируемый характер, включающий согласованные взаимодействия между эндотелиальными клетками, циркулирующими тромбоцитами, плазменными факторами свёртывания и компонентами фибринолитической системы. В данном расширенном обзоре особое внимание уделяется структурно-функциональным характеристикам путей коагуляции и антикоагуляции, включая ключевую регуляторную роль системы протеина С, антитромбина III и гепарин-зависимых механизмов.

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Эндотелиальная поверхность рассматривается как активный участник гемостаза, способный как стимулировать, так и подавлять генерацию тромбина в зависимости от физиологических условий. Кроме того, в статье анализируются молекулярные основы образования и деградации фибрина, с акцентом на значении тканевого активатора плазминогена и его ингибиторов в поддержании фибринолитического равновесия. Особое внимание уделяется патофизиологии диссеминированного внутрисосудистого свёртывания (ДВС-синдрома) — тяжёлого клинического синдрома, возникающего вследствие системной активации коагуляции, приводящей к микрососудистому тромбозу, коагулопатии потребления и полиорганной недостаточности. Понимание механизмов регуляции гемостаза создаёт основу для повышения точности диагностики и разработки целенаправленных терапевтических вмешательств при коагулопатиях.

Ключевые слова: гемостаз, эндотелий, тромбоциты, антикоагулянты, протеин С, гепарин, диссеминированное внутрисосудистое свёртывание (ДВС).

Annotatsiya. Gemostatik tizim qonning suyuqligini saqlash va tomir shikastlanganda barqaror ivindini hosil qilish qobiliyati o‘rtasidagi nozik muvozanatni ta’minlovchi ko‘p omilli fiziologik tizim hisoblanadi. Gemostazning zamonaviy konsepsiyalari uning dinamik va qat’iy boshqariladigan jarayon ekanligini ta’kidlab, endotelial hujayralar, aylanib yuruvchi trombotsitlar, plazmadagi ivish omillari hamda fibrinolitik tizim komponentlari o‘rtasidagi muvofiqlashtirilgan o‘zaro ta’sirlarni o‘z ichiga oladi. Ushbu kengaytirilgan sharhda koagulyatsiya va antikoagulyatsiya yo‘llarining tuzilma-funksional xususiyatlariga, xususan protein C tizimi, antitrombin III va geparinga bog‘liq mexanizmlarning markaziy tartibga soluvchi roliga alohida e’tibor qaratiladi. Endotelial yuzaning gemostazda faol ishtirok etuvchi tuzilma sifatida, fiziologik sharoitga qarab trombin hosil bo‘lishini rag‘batlantirishi yoki bostirishi mumkinligi yoritiladi. Bundan tashqari, maqolada fibrin hosil bo‘lishi va parchalanishining molekulyar asoslari tahlil qilinib, fibrinolitik muvozanatni saqlashda to‘qima plazminogen faollashtiruvchisi va uning ingibitorlarining ahamiyati ta’kidlanadi. Disseminatsiyalangan tomir ichidagi ivish (DVS-sindrom) patofiziologiyasiga alohida e’tibor qaratilib, u koagulyatsiyaning tizimli faollashuvi natijasida yuzaga keladigan, mikrotomir trombozi, iste’mol koagulopatiyasi va ko‘p a’zolar yetishmovchiligi bilan kechuvchi og‘ir klinik holat sifatida tavsiflanadi. Gemostazni boshqarish mexanizmlarini tushunish koagulopatiyalarni aniq tashxislash va maqsadli davolash usullarini ishlab chiqish uchun muhim ilmiy asos yaratadi.

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Kalit so‘zlar: gemostaz, endoteliy, trombotsitlar, antikoagulyantlar, protein C, geparin, disseminatsiyalangan tomir ichidagi ivish (DVS).

Introduction

Hemostasis is a complex, multilevel biological system responsible for preserving the fluidity of blood under physiological conditions while ensuring a rapid and localized clotting response following vascular injury. Its proper functioning is essential for effective microcirculation, tissue oxygenation, and overall hemodynamic stability. Modern views on hemostasis emphasize that it is not a static mechanism but rather a dynamic regulatory network involving continuous interactions between cellular and plasma components. These interactions maintain a finely tuned balance between procoagulant, anticoagulant, and fibrinolytic forces.

The classical tripartite model of hemostasis includes the vascular–platelet component, the plasma coagulation cascade, and the fibrinolytic system. The vascular endothelium plays a pivotal role as a modulator, switching between anticoagulant and procoagulant phenotypes in response to biochemical stimuli, inflammation, or mechanical damage. Platelets serve as key effector cells that initiate primary hemostasis by adhering to exposed subendothelial structures, aggregating, and providing a phospholipid surface for coagulation enzyme complexes. Simultaneously, plasma factors participate in a tightly regulated enzymatic cascade that culminates in thrombin formation and the conversion of fibrinogen into a stable fibrin clot.

Disturbances in the intricate equilibrium among these mechanisms can manifest as either hypercoagulability, associated with thrombosis and vascular occlusion, or hypocoagulability, characterized by pathological bleeding. Recent advances in molecular biology and vascular physiology have contributed to a deeper understanding of the mechanisms underlying these disruptions, including genetic thrombophilia, endothelial dysfunction, inflammation-induced coagulopathy, and dysregulation of natural anticoagulant pathways. Because alterations in hemostasis play a central role in many critical conditions—such as sepsis, trauma, cardiovascular disease, and disseminated intravascular coagulation (DIC)—the study of its regulatory mechanisms remains an important focus of modern clinical and experimental research.

Materials and Methods

This work is based on a comparative analysis of more than 20 scientific sources, including peer-reviewed articles, clinical guidelines, and monographs in hematology. Content analysis and systematization methods were applied to identify key regulatory factors and molecular mechanisms involved in maintaining hemostatic equilibrium.

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Results and Discussion

Endothelial Regulation of Hemostasis

The vascular endothelium plays a central role in maintaining hemostatic balance through synthesis of vasodilators such as prostacyclin and nitric oxide, degradation of platelet activators, and activation of endogenous anticoagulants such as antithrombin III and heparin cofactor II.

The Protein C Pathway

The protein C system modulates thrombin generation by inactivating factors Va and VIIIa. Deficiencies or genetic mutations within this pathway, such as factor V Leiden, significantly increase the risk of thrombosis.

Fibrinolysis

The fibrinolytic system dissolves fibrin clots via activation of plasminogen by t-PA. Inhibitors such as PAI-1 strictly regulate this process, and their elevation is associated with hypofibrinolysis and thrombotic risk.

Mechanisms of Blood Coagulation

Coagulation proceeds through intrinsic and extrinsic pathways converging on thrombin formation and fibrin polymerization. Platelets provide the necessary structural matrix for clot stabilization.

Disseminated Intravascular Coagulation (DIC)

DIC is characterized by uncontrolled activation of coagulation, resulting in microthrombi formation and consumption of clotting factors, ultimately leading to hemorrhage and organ dysfunction.

Recent advances in hemostasis research have demonstrated the critical role of endothelial–platelet interactions in early thrombus formation. Novel data suggests that endothelial-derived extracellular vesicles contribute to coagulation activation under inflammatory conditions.

Moreover, the concept of immunothrombosis emphasizes the role of innate immune cells—particularly neutrophils—in amplifying thrombin generation through the release of neutrophil extracellular traps (NETs). These structures provide a scaffold that binds coagulation factors and promotes fibrin formation.

In addition, modern molecular techniques have revealed significant genetic determinants of thrombophilia, including mutations affecting anticoagulant pathways and fibrinolytic activity. Understanding these factors is essential for developing personalized anticoagulant therapies.

Table 1.

Key Components of the Hemostatic System

Hemostatic Component	Primary Function	Regulatory Factors
Endothelium	Maintains anti-thrombotic surface	NO, PGI_2, AT III activation

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Platelets	Initial plug formation	ADP, thromboxane A_2
Protein C system	Inactivation of factors Va and VIIIa	Thrombomodulin, Protein S
Fibrinolysis	Clot dissolution	t-PA, PAI-1

Conclusion

The hemostatic system represents a complex and tightly regulated network. Understanding its molecular mechanisms, particularly those involving endothelial regulation, the protein C pathway, and fibrinolysis, is essential for improving the diagnosis and treatment of coagulopathies.

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