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Platelet hemostasis disorders in burn disease: a review

Abstract. ***Background.*** *Platelets are highly sensitive to changes in the internal environment and play a key role in hemostasis, inflammation, and immune response. Burn injury is associated with significant alterations in platelet count and function, contributing to coagulopathy and adverse clinical outcomes. The purpose was to summarize current evidence on platelet hemostasis disorders in burn disease, with a focus on pathophysiology, biomarkers, and treatment approaches.* ***Materials and methods.*** *A literature search was conducted using CrossRef, Scopus, Google Scholar, and PubMed databases for the period from 2017 to 2026. The search strategy combined structured descriptive approaches with manual screening of relevant publications. Observational studies and reviews addressing platelet dysfunction in burn patients were included.* ***Results.*** *Thrombocytopenia in burn patients is primarily associated with increased platelet consumption and destruction due to inflammatory mediators, endothelial damage, and activation of coagulation pathways. Early decreases in fibrinogen may impair platelet aggregation. Several biomarkers demonstrate diagnostic and prognostic potential, including platelet count and its dynamics, mean platelet volume, platelet distribution width, immature platelet fraction, inflammatory markers (C-reactive protein, procalcitonin, interleukin-6), and coagulation parameters (D-dimer, fibrinogen, antithrombin III). Additional indicators include markers of endothelial dysfunction and organ failure. Severe thrombocytopenia (< 50 × 10^9/L) requires urgent clinical management. However, platelet transfusion in sepsis remains controversial due to potential risks such as hypercoagulability and thrombotic complications.* ***Conclusions.*** *Platelet dysfunction plays a central role in the pathogenesis of burn-related coagulopathy and sepsis. Further research is needed to improve diagnostic strategies and develop targeted therapies aimed at optimizing platelet function and reducing inflammation in burn patients.*

Keywords: *burn injury; thrombocytopenia; platelet dysfunction; coagulopathy; sepsis; biomarkers*

Introduction

Severe burn injury significantly affects the hemostatic system, leading to alterations in both platelet count and function. Platelets play a critical role not only in coagulation but also in inflammation and immune regulation. In burn patients, these processes are often dysregulated, contributing to the development of coagulopathy and multiple organ dysfunction [1–3].

Early studies have demonstrated that thrombocytopenia frequently occurs in patients with burn injury and may be associated with sepsis, systemic inflammation, drug exposure, and coagulation disorders. Platelets are highly sensitive to environmental changes and rapidly respond to alterations in blood composition caused by tissue injury or infection [2].

Endothelial damage following burn injury triggers inflammatory and coagulation cascades, resulting in platelet activation, aggregation, and increased consumption. These processes contribute to both bleeding risk and hypercoagulability, complicating clinical management [2].

Given the complex role of platelets in burn disease, understanding the mechanisms of platelet dysfunction is essential for improving diagnostic and therapeutic strategies.

Materials and methods

A literature search was conducted in CrossRef, Scopus, Google Scholar, and PubMed databases for studies published between 2017 and 2026. The search strategy included keywords related to burn injury, thrombocytopenia, platelet function, and coagulopathy.

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Studies were included if they: 1) were published in English, Ukrainian, or Chinese; 2) addressed platelet hemostasis disorders in burn patients; 3) used observational (cohort or cross-sectional) or review designs.

Manual screening of reference lists was performed to identify additional relevant publications.

A total of 33 studies were included in the analysis, the majority of which were published within the last 5–10 years.

Analysis and discussion

The severity of thrombocytopenia in burn patients is closely associated with the extent of the injury. Patients with burns involving less than 10 % of the total body surface area rarely develop thrombocytopenia [1, 4]. In contrast, more extensive burns are significantly associated with a higher incidence and severity of platelet depletion. Clinical observations indicate that patients with thrombocytopenia tend to have a greater mean burn area compared to those without it [5].

In burn patients, a decrease in platelet count typically occurs within the first 48 hours after injury. This early decline is primarily related to platelet consumption caused by thermal damage, as well as hemodilution resulting from aggressive fluid resuscitation [6]. Platelet consumption is considered a characteristic feature of burn disease [1].

Platelets in burn patients exhibit increased reactivity and are more prone to activation, which contributes to thrombotic complications and adverse cardiovascular events. In addition, platelet activation promotes the release of inflammatory mediators, thereby amplifying the inflammatory response [2]. Under conditions of tissue damage, platelets adhere to exposed subendothelial structures, including collagen, von Willebrand factor, laminin, fibronectin, and thrombospondins, initiating multiple signaling cascades that enhance platelet adhesion and aggregation [7].

Exposure of subendothelial collagen activates circulating platelets and promotes recruitment of additional platelets to the site of injury. Various factors, including neutrophil-derived proteases, histones, and tissue thromboplastin, can directly activate platelets. Activated platelets contribute to hemostasis by forming aggregates at the site of injury, while also releasing chemokines and growth factors that modulate leukocyte activity and support wound healing [4, 8]. Despite their important role in tissue repair, platelet counts in burn patients are often reduced compared to healthy individuals [9].

Thrombocytopenia in burn disease is primarily associated with increased platelet consumption and destruction due to inflammatory mediators. Although bone marrow suppression may contribute to reduced platelet production, current evidence suggests that increased consumption rather than impaired production is the dominant mechanism [5].

Endothelial activation plays a central role in platelet dysfunction. Activated endothelial cells express von Willebrand factor and integrins, which facilitate platelet adhesion and activation. Increased levels of thrombopoietin may further enhance platelet sensitivity to activating stimuli [4, 10, 11]. Additionally, an early decrease in fibrinogen levels following burn injury may impair platelet aggregation and contribute to coagulopathy [11–13].

Neutrophil extracellular traps (NETs) represent another important mechanism linking inflammation and coagulation. Platelets become activated upon interaction with NETs, which promote coagulation and endothelial damage. NET-mediated intravascular coagulation involves complex interactions between histones, platelets, and inorganic polyphosphates [14, 15]. NETs also serve as a structural scaffold for platelet aggregation and exhibit pronounced procoagulant activity in sepsis [16].

Platelet indices such as platelet distribution width (PDW) and platelet-large cell ratio (P-LCR) reflect platelet size heterogeneity and activation status. Elevated PDW and increased P-LCR (> 35 %) are associated with inflammation, thrombosis, and platelet activation. In the early phase of burn injury, increased platelet activation and aggregation, combined with hemodilution, lead to a progressive decrease in platelet count. Monitoring the dynamics of PDW and related indices may therefore provide clinically relevant information [17].

Systemic platelet activation precedes platelet consumption and plays a key role in the development of a hypercoagulable state in burn patients [18]. Flow cytometric studies have demonstrated increased expression of P-selectin on platelets in response to lipopolysaccharides, indicating the presence of primed platelets in circulation [7, 11].

Endothelial injury and inflammation further promote platelet interaction with immune cells through Toll-like receptors (TLR), thereby sustaining the inflammatory response. Experimental studies have shown increased levels of platelet-derived microvesicles in circulation following burn injury. These microvesicles exhibit procoagulant properties and may exacerbate hypercoagulability [7, 18].

Altered platelet responsiveness to agonists has also been described in burn patients. Increased platelet response to arachidonic acid has been associated with thrombotic complications, while impaired responses to ristocetin and thrombin receptor-activating peptides have been observed [1, 18]. Low concentrations of prostaglandin E2 may enhance platelet aggregation by modulating intracellular signaling pathways [18].

The glycoprotein IIb/IIIa receptor (GPIIb/IIIa) plays a central role in platelet aggregation by mediating fibrinogen binding. Increased activation of GPIIb/IIIa and P-selectin expression in burn patients reflects altered platelet signaling. These changes are associated with markers of coagulation and endothelial dysfunction, including D-dimer and soluble P-selectin levels [19].

Additional molecular mechanisms include activation of purinergic P2X1 receptors, which mediate rapid calcium influx and contribute to platelet aggregation [8]. The content of platelet-derived growth factors correlates with platelet count, reflecting their role in tissue repair and regeneration [20].

Platelets also play an important role in immune defense. Conditions such as heparin-induced thrombocytopenia illustrate the complex interaction between platelet activation and immune mechanisms. Antibodies directed against platelet factor 4-heparin complexes can lead to platelet activation even in the absence of prior heparin exposure [7, 21].

Clinically, thrombocytopenia in burn patients can be divided into early and late forms. Early thrombocytopenia occurs within the first week after injury and is associated with stress response and platelet consumption, whereas late thrombocytopenia is typically related to sepsis [2]. The type

of burn injury may also influence platelet dynamics, with flame burns more frequently associated with thrombocytopenia compared to scald injuries [5].

Even in cases where platelet counts remain within normal limits, platelet function may be impaired [22]. Early reductions in platelet function and coagulation factors are typically observed within the first 48 hours and may persist for several days despite intensive therapy [3, 5]. This contributes to increased transfusion requirements in the later stages of treatment [6].

During the first 72 hours after burn injury, abnormalities in coagulation parameters and thrombocytopenia are inversely related to total body surface area, with more extensive burns associated with more pronounced changes [6, 23]. Platelet counts usually recover within the first week and may subsequently increase above normal levels, with reactive thrombocytosis peaking around day 21 [1, 4, 21, 24, 25]. Normalization may take several months [3].

In severe cases, late thrombocytopenia may develop in association with septic complications and consumption coagulopathy. This condition is characterized by depletion of coagulation factors and activation of coagulation pathways, often mediated by contact activation mechanisms [6]. Regardless of the underlying cause, burn patients frequently exhibit a hypercoagulable state [2].

Sepsis remains the most common and clinically significant cause of thrombocytopenia in burn patients [5, 26].

Thrombocytopenia occurs in approximately half of patients with burn sepsis and serves as an important prognostic marker. Platelets contribute to host defense by interacting with pathogens and modulating immune responses, highlighting their role beyond hemostasis [7].

The pathogenesis of thrombocytopenia in sepsis is multifactorial and includes decreased production, increased destruction due to disseminated intravascular coagulation, and sequestration in the spleen and other organs. Additional factors associated with thrombocytopenia include higher organ failure scores, impaired oxygenation, and vasopressor use [7].

A key mechanism underlying organ dysfunction in sepsis is the disruption of the balance between procoagulant and anticoagulant pathways. Proinflammatory cytokines promote the release of procoagulant factors, leading to thrombosis, endothelial dysfunction, and impaired tissue perfusion, ultimately contributing to multiple organ failure [11].

Overall, platelet dysfunction in burn disease represents a complex and dynamic process involving interactions between inflammation, coagulation, and immune responses. Understanding these mechanisms is essential for improving clinical outcomes and developing targeted therapeutic strategies.

Markers. Currently, no single biomarker has been established as a definitive predictor of thrombocytopenia or clinical outcomes in burn patients. However, a number of laboratory parameters demonstrate diagnostic and prognostic value when used in combination.

Platelet level and its changes over time remain among the most accessible and clinically relevant indicators. Both the baseline platelet count and the rate of decline are important for early risk stratification. In addition, platelet indices such as mean platelet volume, PDW, and the immature (reticulated) platelet fraction provide insight into platelet production and activation status [7].

Markers of systemic inflammation, including C-reactive protein, procalcitonin, and interleukin (IL) 6, are widely used to assess the severity of inflammatory response and the risk of sepsis. Coagulation parameters such as D-dimer, fibrinogen, and antithrombin III reflect activation of coagulation pathways and may indicate the development of coagulopathy. Indicators of liver function, like bilirubin and albumin levels, as well as markers of endothelial dysfunction (e.g., von Willebrand factor, angiopoietin-2, and nitric oxide), further contribute to the assessment of disease severity [7].

Immune-related indices such as the neutrophil-to-lymphocyte ratio have also been proposed as simple and cost-effective markers of systemic inflammation and immune dysregulation. In addition, composite clinical scores that assess organ dysfunction, acute physiology, and overall disease severity are valuable for predicting outcomes in burn patients [7].

Recent studies have focused on the identification of novel molecular biomarkers, including microRNAs and TLR signaling components, which may improve early diagnosis and prognostic accuracy. Furthermore, clinical factors such as age, comorbidities (particularly hepatic, renal, and hematological disorders), and the type of infectious pathogen (e.g., Gram-negative bacteria) significantly influence the risk of coagulopathy and thrombocytopenia [7].

In the context of burn sepsis, thrombocytopenia below 100,000/μL is generally not considered a reliable diagnostic marker during the first three days following injury, which limits its early predictive value [27].

Infectious processes also influence platelet activity through the stimulation of extracellular vesicle release. Circulating extracellular vesicles and their molecular content change significantly during sepsis and may correlate with the severity of organ dysfunction, suggesting their potential role as emerging biomarkers [7].

Treatment. Management of thrombocytopenia in burn patients, particularly in the setting of sepsis, requires an individualized approach that considers the underlying pathophysiological mechanisms, severity of illness, and patient-specific factors. The primary goals of therapy are to prevent bleeding complications, restore platelet count, and address the underlying cause.

Platelet transfusion remains the most commonly used intervention for severe thrombocytopenia. In general, transfusion is recommended when platelet count decreases below 10×10^9/L, even in the absence of active bleeding [28, 29]. In patients at increased risk of bleeding or those undergoing invasive procedures, higher thresholds (e.g., $< 20 \times 10^9$/L) may be considered, although these recommendations remain controversial [30]. In cases of severe thrombocytopenia ($< 50 \times 10^9$/L), urgent clinical management is required, including treatment of the underlying condition and supportive care [2, 7].

However, the use of platelet transfusion in sepsis is associated with several limitations. These include low post-transfusion platelet increment, increased platelet consumption or destruction, and adverse reactions such as fever, splenomegaly, and disseminated intravascular coagulation. Furthermore, platelet transfusion may contribute to a prothrombotic state and potentially worsen infectious complications. Therefore,

it is not considered a sufficient standalone therapy in patients with burn sepsis and thrombocytopenia [2, 7].

Current guidelines also vary regarding transfusion thresholds. For example, the Association for the Promotion of Hematology and Biotherapy recommends prophylactic platelet transfusion at counts ≤ 10×10^9/L to prevent spontaneous bleeding, < 20×10^9/L prior to invasive procedures, and < 50×10^9/L before surgical interventions. Nevertheless, there are no universally accepted protocols specifically for burn-related thrombocytopenia, and the overall efficacy and safety of platelet transfusion in this population remain uncertain [7].

Intravenous immunoglobulin (IVIG) represents an alternative therapeutic option, particularly in cases where immune-mediated platelet destruction is suspected. IVIG may provide a more rapid increase in platelet count compared to glucocorticoids. IgM-enriched IVIG has been associated with reduced mortality, shorter hospital stay, and improved severity scores (e.g., APACHE II) in patients with burn sepsis [7, 31]. However, its use is not recommended in patients with IgA deficiency.

Recombinant human thrombopoietin stimulates platelet production by acting on megakaryocytes and promoting their differentiation and maturation. Clinical data suggest that it effectively increases platelet count with minimal adverse effects and may reduce the need for platelet transfusion as well as 28-day mortality in patients with burn sepsis [7].

Recombinant human IL-11 has also demonstrated thrombopoietic and anti-inflammatory effects. Administration of IL-11 has been associated with increased platelet counts, reduced levels of inflammatory markers such as IL-6 and procalcitonin, and improved clinical outcomes. However, its role in the treatment of sepsis remains insufficiently defined and requires further investigation.

Targeted anti-inflammatory therapy may also influence platelet function. *Tocilizumab*, a monoclonal antibody against the IL-6 receptor, reduces inflammation and may contribute to normalization of platelet count by inhibiting IL-6-mediated pathways [7]. In addition, inhibition of platelet-activating factor, including the use of *recombinant platelet-activating factor acetylhydrolase*, has been proposed as a potential therapeutic strategy. Other agents such as *ginkgolides*, *nonsteroidal anti-inflammatory drugs*, *glucocorticoids*, and *ketotifen* may exert similar but less pronounced effects [32].

Emerging therapeutic approaches include inhibition of TLR4 signaling. Agents like *TAK-242* (*resatorvid*, *eritoran*) selectively block TLR4 activation and may reduce platelet activation, aggregation, and the formation of platelet-leukocyte complexes in response to endotoxins [7].

Thrombopoietin receptor agonists, including *avatrombopag*, *eltrombopag*, and *romiplostim*, are effective in the treatment of chronic thrombocytopenia and may represent a promising option in selected patients by enhancing platelet production.

Overall, treatment of thrombocytopenia in burn disease remains challenging and requires a comprehensive approach targeting both the underlying disease and the complex interactions between coagulation, inflammation, and immune responses.

Prognosis. Currently, thrombocytopenia appears to be the earliest and most reliable independent predictor of severe complications in patients with major burns [5, 21].

Thrombocytopenia in burn patients is a clinically significant marker associated with adverse outcomes, including increased risk of sepsis, multiple organ dysfunction, and mortality. Both the absolute platelet count and its temporal dynamics are important for prognostic assessment. A rapid decline in platelet levels or persistently low counts is generally associated with worse clinical outcomes [7].

Several studies have demonstrated that thrombocytopenia correlates with the severity of burn injury and the extent of systemic inflammatory response. In particular, a progressive decrease in platelet count during the early post-burn period may indicate the development of sepsis or disseminated intravascular coagulation, both of which are associated with poor prognosis [2, 7].

In addition to platelet count, combined assessment with other laboratory and clinical parameters improves prognostic accuracy. These include inflammatory markers, coagulation indices, and organ dysfunction scores. Persistent thrombocytopenia despite ongoing therapy may reflect uncontrolled infection, severe endothelial damage, or bone marrow suppression, all of which are associated with increased mortality risk [7, 33].

Importantly, recovery of platelet count over time is generally considered a favorable prognostic sign, indicating resolution of inflammation and stabilization of hemostatic processes. Conversely, failure of platelet recovery may suggest ongoing pathological processes requiring further diagnostic and therapeutic interventions [7].

Conclusions

Platelet dysfunction plays a central role in the pathogenesis of burn-related coagulopathy and is closely linked to inflammation, endothelial injury, and immune dysregulation. Thrombocytopenia is not only a laboratory abnormality but also an important clinical marker associated with disease severity and patient outcomes.

A comprehensive assessment of platelet-related parameters, including platelet count, functional indices, and associated biomarkers, is essential for early diagnosis, risk stratification, and monitoring of burn patients. However, no single biomarker provides sufficient sensitivity and specificity, highlighting the need for a multimodal diagnostic approach.

Current treatment strategies remain largely supportive and are often limited by insufficient evidence and lack of standardized protocols, particularly in the context of burn sepsis. While platelet transfusion, immunotherapy, and thrombopoietic agents show potential benefits, their use requires careful consideration of risks and individual patient characteristics.

Further research is needed to better understand the mechanisms of platelet dysfunction in burn disease and to develop targeted therapeutic strategies aimed at improving clinical outcomes. Future studies should focus on the identification of reliable biomarkers and the evaluation of novel treatments that modulate the complex interplay between coagulation, inflammation, and immune responses.

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Розлади тромбоцитарного гемостазу при опіковій хворобі: огляд

Резюме. ***Актуальність.*** Тромбоцити дуже чутливі до змін внутрішнього середовища і відіграють ключову роль у гемостазі, запаленні й імунній відповіді. Опікова травма пов'язана зі значними змінами кількості та функції тромбоцитів, призводячи до коагулопатії та несприятливих клінічних результатів. ***Мета:*** узагальнити сучасні дані про порушення тромбоцитарного гемостазу при опіковій хворобі з акцентом на патофізіології, біомаркерах і підходах до лікування. ***Матеріали та методи.*** Пошук літератури проводився з використанням баз даних CrossRef, Scopus, Google Scholar та PubMed за період з 2017 до 2026 р. Стратегія пошуку поєднувала структуровані описові підходи з ручним відбором відповідних публікацій. Були включені обсерваційні дослідження та огляди щодо дисфункції тромбоцитів у хворих з опіками. ***Результати.*** Тромбоцитопенія в опікових пацієнтів пов'язана в першу чергу з підвищеним споживанням і руйнуванням тромбоцитів через медіатори запалення, пошкодження ендотелію та активацію шляхів згортання крові. Раннє зниження фібриногену може порушити агрегацію тромбоцитів. Кілька біомаркерів демонструють діагностичний та прогностичний потенціал, включаючи кількість тромбоцитів і їх динаміку, середній об'єм тромбоцитів, ширину розподілу тромбоцитів, незрілу фракцію тромбоцитів, маркери запалення (С-реактивний білок, прокальцитонін, інтерлейкін-6) і параметри коагуляції (D-димер, фібриноген, антитромбін III). Додатковими показниками є маркери ендотеліальної дисфункції та органної недостатності. Тяжка тромбоцитопенія ($< 50 \times 10^9$/л) вимагає термінового клінічного лікування. Однак переливання тромбоцитів при сепсисі залишається суперечливим через потенційні ризики, як-от гіперкоагуляція і тромботичні ускладнення. ***Висновки.*** Дисфункція тромбоцитів відіграє центральну роль у патогенезі опікової коагулопатії та сепсису. Потрібні подальші дослідження для вдосконалення діагностичних стратегій і розробки цільової терапії, спрямованої на оптимізацію функції тромбоцитів та зменшення запалення в пацієнтів з опіками.

Ключові слова: опікова травма; тромбоцитопенія; тромбоцитарна дисфункція; коагулопатія; сепсис; біомаркери