

**VASCULAR-PLATELET HEMOSTASIS IN CHRONIC VIRAL LIVER DISEASES:
CURRENT CONCEPTS AND CLINICAL IMPLICATIONS**

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The review of the literature provides information on changes in vascular-platelet hemostasis, platelet count, disorders of adhesion and aggregation properties, hemorrhagic complications in chronic liver disease. The results of the study of vascular-platelet hemostasis in patients with chronic liver diffuse disease were analyzed.

Keywords: hepatotropic viruses, hepatitis, liver cirrhosis, platelets, adhesions, aggregation.

Introduction

The liver plays a pivotal role in maintaining hemostatic balance through the synthesis of most coagulation factors, anticoagulant proteins, and fibrinolytic components. Consequently, chronic liver diseases are frequently accompanied by significant alterations in hemostatic function [1,2]. These abnormalities may manifest as both hemorrhagic and thrombotic complications, reflecting the complex interplay between coagulation pathways, endothelial integrity, and platelet function [3]. Chronic viral hepatitis and liver cirrhosis remain major global health concerns due to their high prevalence and association with substantial morbidity and mortality. Progressive hepatic fibrosis and cirrhosis are accompanied by profound changes in vascular-platelet hemostasis, including thrombocytopenia, impaired platelet aggregation, endothelial dysfunction, and disturbances in microcirculation [4,5].

Among the various components of hemostasis, platelet-related mechanisms play a critical role in disease progression. Platelets are involved not only in primary hemostasis but also in inflammation, tissue repair, angiogenesis, and liver regeneration [18,23].

Thrombocytopenia is one of the most common hematological abnormalities observed in patients with chronic liver disease [8,20]. A platelet count below $150 \times 10^9/L$ may occur in both chronic hepatitis and liver cirrhosis.

Several mechanisms contribute to platelet reduction in chronic liver disorders. Hypersplenism resulting from portal hypertension is considered the primary cause of thrombocytopenia in cirrhotic patients [8,19]. Additional factors include reduced thrombopoietin production by damaged hepatocytes, bone marrow suppression, folate deficiency, autoimmune platelet destruction, and disseminated intravascular coagulation [6,19].

Clinical studies have consistently demonstrated a significant decline in platelet count among patients with chronic hepatitis C and liver cirrhosis [20]. Beyond quantitative abnormalities, platelet function is also impaired. Alterations in platelet adhesion, activation, and aggregation have been reported in various forms of chronic liver disease [7,17].

Endothelial dysfunction represents a central component in the pathogenesis of chronic liver disease [1,9]. Persistent viral infection, immune-mediated injury, oxidative stress, and inflammatory cytokines contribute to endothelial damage [2,9].

One of the most important markers of endothelial dysfunction is von Willebrand factor (vWF). Elevated plasma concentrations of vWF have been consistently reported in patients with advanced liver disease [14,15]. Increased vWF activity partially compensates for

thrombocytopenia by enhancing platelet adhesion. However, despite elevated vWF levels, platelet-vWF interactions may remain impaired, contributing to an increased risk of bleeding [12].

Nitric oxide and prostacyclin released by endothelial cells regulate platelet activation and vascular tone, thereby influencing primary hemostasis [11].

Recent studies have demonstrated that platelets possess functions extending beyond hemostasis. They serve as reservoirs of numerous growth factors, cytokines, and bioactive mediators that influence tissue repair and regeneration [18].

Platelets contribute to liver regeneration through direct interactions with hepatocytes, sinusoidal endothelial cells, and Kupffer cells [23]. Experimental evidence suggests that platelet-derived mediators may reduce hepatic fibrosis by modulating stellate cell activity [23].

Although platelet transfusion has been reported to improve liver function in selected cirrhotic patients, routine transfusion remains controversial due to the risk of transfusion-related complications and infectious transmission [10,18].

Hemorrhagic manifestations include epistaxis, gingival bleeding, petechiae, gastrointestinal bleeding, and variceal hemorrhage [4]. The risk of bleeding is influenced by thrombocytopenia, platelet dysfunction, reduced coagulation factor synthesis, and portal hypertension [3,8].

Conversely, thrombotic events such as portal vein thrombosis, mesenteric vein thrombosis, and pulmonary embolism may occur in advanced liver disease [16]. Increasing evidence suggests that hypercoagulability contributes to hepatic fibrogenesis and disease progression [19,22].

The coexistence of bleeding and thrombotic tendencies presents significant diagnostic and therapeutic challenges in clinical practice [11].

Despite substantial advances in understanding hemostatic abnormalities associated with chronic liver disease, several challenges remain unresolved. Conventional coagulation tests often fail to accurately reflect the true hemostatic balance in cirrhotic patients [4,22].

Future research should focus on identifying reliable predictors of bleeding and thrombosis, improving risk stratification, and developing individualized therapeutic strategies [9,13].

Conclusion

Chronic viral liver diseases are associated with complex disturbances of vascular–platelet hemostasis that significantly influence disease progression and clinical outcomes. Thrombocytopenia, platelet dysfunction, endothelial injury, and alterations in coagulation pathways represent key components of the hemostatic imbalance observed in patients with chronic hepatitis and liver cirrhosis. These abnormalities contribute not only to hemorrhagic manifestations but also to an increased risk of thrombotic complications.

Current evidence indicates that platelet-related disorders in chronic liver disease are multifactorial and result from hypersplenism, impaired thrombopoietin production, immune-mediated mechanisms, and endothelial dysfunction. In addition to their role in primary hemostasis, platelets participate in inflammatory processes, tissue repair, and liver regeneration, highlighting their broader biological significance in hepatic pathology.

Endothelial dysfunction and elevated von Willebrand factor activity further modify platelet function and contribute to the fragile equilibrium between bleeding and thrombosis. Therefore, comprehensive assessment of vascular–platelet hemostasis is essential for evaluating disease severity, predicting complications, and optimizing patient management.

In conclusion, vascular–platelet hemostatic abnormalities should be regarded as an integral component of chronic viral liver diseases. A deeper understanding of the underlying mechanisms may improve diagnostic approaches, prognostic assessment, and the development of targeted therapeutic strategies aimed at reducing both hemorrhagic and thrombotic risks in affected patients.

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