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# Changes in Platelet Count and Function in Chronic Pancreatitis

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**Abstract** Chronic pancreatitis is one of the most common diseases in the population today. Alleviating the clinical course of this disease, increasing the effectiveness of treatment, and reducing the incidence of complications are among the primary healthcare goals. Analyzing the relationship between the hemostatic system and chronic pancreatitis is key to achieving this goal. Therefore, in this article, we will focus on studying changes in the number and function of platelets, which are part of the hemostatic system, in chronic pancreatitis.

**Keywords** Chronic pancreatitis, Thrombocytopenia, DIC, Thrombocytosis, Megakaryocyte

## 1. Introduction

Chronic pancreatitis (CP) is a disease characterized by fibrosis of the exocrine and endocrine pancreatic tissue as a result of prolonged inflammation. This process enhances the systemic inflammatory response and leads to significant alterations in the hemostatic system, particularly resulting in increased platelet count and functional activity [1,3].

Studies have demonstrated that two types of platelet abnormalities may occur in chronic pancreatitis: thrombocytopenia (decreased platelet count) and reactive thrombocytosis (increased platelet count) [2,4]. Thrombocytopenia is more commonly observed in severe or complicated cases of chronic pancreatitis and may be associated with coagulopathy, sepsis, or deep vein thrombosis [1,3].

Splenic vein thrombosis is also a frequent finding in chronic pancreatitis, in which platelet count in the peripheral blood decreases due to sequestration in the spleen [4]. Therefore, some patients with CP may present with a combination of thrombocytopenia, splenomegaly, and portal hypertension [4].

Moreover, reactive thrombocytosis may develop as a response to chronic inflammation. This condition is associated with increased megakaryocyte activity in the bone marrow under the influence of inflammatory mediators, particularly interleukin-6 (IL-6) [2]. Reactive thrombocytosis is often regarded as a marker of active inflammation and disease severity [2,3].

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Platelet activation-related changes play an important role in the pathogenesis of CP. Activated platelets interact with endothelial cells, promoting microthrombosis, vascular alterations, and ischemic tissue injury [3,5]. Therefore, evaluating platelet count and activity has diagnostic value for monitoring the coagulation system in CP.

In recent years, platelet indices such as mean platelet volume (MPV) and the MPV-to-platelet count ratio (MPR) have been investigated as potential biomarkers for assessing the degree of inflammation and thrombosis risk in chronic pancreatitis [5,6]. Increased MPV indicates elevated platelet activation and active participation in inflammatory and coagulative processes [6]. Consequently, both quantitative and functional platelet alterations in chronic pancreatitis represent important clinical indicators for evaluating inflammation severity, risk of complications, and disease prognosis [1,2,3,4,5,6].

**Changes in Platelet Function in Chronic Pancreatitis**

Platelet activation and aggregation: Inflammation and endothelial dysfunction in chronic pancreatitis promote platelet aggregation through prior activation. Activated platelets bind to each other via glycoprotein IIb/IIIa receptors, forming platelet aggregates that increase the risk of microthrombosis and tissue ischemia.

Secretion and mediators: Activated platelets release mediators from their alpha-granules, such as P-selectin (CD62P) and platelet factor 4 (PF4). These molecules interact with endothelial cells and leukocytes, enhancing inflammation and amplifying inflammatory signaling in the bloodstream.

P-selectin and platelet-leukocyte aggregates: In chronic pancreatitis, elevated P-selectin expression and increased platelet-leukocyte aggregate formation may occur, intensifying the cross-talk between immune and coagulation pathways and elevating the risk of thrombotic complications.

Platelet microparticles (PMPs): Activated platelets release microparticles with procoagulant properties that further promote inflammation and thrombosis. Elevated PMP levels are associated with a higher risk of vascular complications.

Morphological and laboratory parameters: Indices such as mean platelet volume (MPV) and the MPV-to-platelet ratio (MPR) reflect platelet functional status; elevated MPV indicates the presence of larger, more active platelets and is associated with a procoagulant state.

Splenic venous system and platelets: Thrombosis of the splenic or portal veins in chronic pancreatitis affects both platelet count and function. Sequestration or thrombosis may lead to platelet hyperreactivity or, conversely, peripheral depletion.

Platelet dysfunction both hyperactivation and impaired function can result in microvascular thrombosis, splenic vein thrombosis, and hemostatic disturbances in CP, which increase complication rates and worsen disease prognosis.

## 2. Conclusions

In conclusion, monitoring platelet function (MPV, P-selectin, platelet-leukocyte aggregates, PMP) may have diagnostic and prognostic significance in CP management. However, decisions regarding antiplatelet or anticoagulant therapy should be individualized, based on a comprehensive risk-benefit assessment, and require further clinical investigation.

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