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C-Reactive Protein as a Predictor of Fibrin Formation in Platelet Concentrates: A Prospective Observational Study on Donor Inflammation and Platelet Quality

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Abstract

Background: Fibrin formation in platelet concentrates (PCs) is a common yet underrecognized complication that compromises their clinical utility. This study investigates whether systemic inflammation in blood donors—quantified by C-reactive protein (CRP) levels—is associated with increased risk of fibrin deposition in PCs.

Methods: We conducted a prospective, controlled observational study including 60 standard PCs derived from whole blood. Platelet morphology, fibrin presence, and CRP levels were evaluated at 12 h and 72 h post-production. Phase-contrast microscopy was used to assess platelet activation.

Results: 71.7% (n=43) of PCs showed fibrin at 72 h. In this group, 65.1% of donors had CRP ≥10 µg/L, compared to 29.4% in controls (p = 0.0203). Platelets from fibrin-positive PCs demonstrated pronounced pseudopodia and activation profiles. Fibrin prevalence was higher in autumn-winter, coinciding with increased systemic inflammation due to seasonal infections.

Conclusion: Elevated donor CRP correlates with fibrin formation in PCs. Pre-donation CRP screening is a cost-effective and feasible measure to enhance the quality and usability of PCs.

Introduction

Platelet transfusion plays a crucial role in the supportive care of patients with thrombocytopenia, hemorrhage, or those undergoing myeloablative therapies. While the production of platelet concentrates (PCs) from whole blood is a routine practice, their functional integrity is occasionally compromised by the formation of fibrin strands or clots during storage. Such PCs must be discarded, leading to wastage of valuable resources, delays in therapy, and increased costs.

Although platelet activation is an expected physiological response in the context of hemostasis, inappropriate activation during storage—especially via inflammatory pathways—can lead to premature fibrin formation. C-reactive protein (CRP), a major acute-phase reactant synthesized by hepato-

cytes, is upregulated in systemic inflammation and plays a pleiotropic role in modulating thrombosis, platelet activation, and complement activation.

The association between donor inflammatory status and platelet product quality is still poorly explored. Several studies have shown that CRP, particularly in its monomeric form, can directly bind to platelet receptors and potentiate aggregation and clot formation. This study seeks to determine whether CRP levels in donor plasma correlate with fibrin deposition in PCs and whether this biomarker can be used as a predictive tool for assessing the quality of the end product.

Materials and Methods

Study Design and Setting

A prospective observational study was conducted over a 5-month period in the Department of Blood Processing, Regional Blood Transfusion Center. Sixty standard PCs were prepared from single units of whole donor blood using a standardized protocol.

Donor Selection and Inclusion Criteria

Healthy voluntary donors aged 18-60 years.

No recent infections, chronic inflammatory conditions, or medication use.

Hemoglobin ≥125 g/L, platelet count >150 × 10^9/L.

Production of Platelet Concentrates

Whole blood was collected and allowed to settle on cooling cloths to stabilize at 22°C. Dual centrifugation was performed:

First spin: 1500-2000 rpm for 15 minutes at 22°C.

This yields three layers: erythrocytes (bottom), platelet-leukocyte buffy coat (middle), and plasma (top).

The buffy coat and plasma were separated using a plasma extractor. The platelet-rich plasma (PRP) was further centrifuged:

Second spin: 3000 rpm for 15 minutes at 22°C.

Platelets sedimented; platelet-poor plasma (PPP) was removed.

Final PCs were resuspended in 60-80 mL plasma and stored at 22°C with constant gentle agitation in a thrombomixer.

Quality Assessment of PCs

Evaluated parameters at 12h and 72h:

Swirling phenomenon.

Visual presence of fibrin strands or clots.

Color and clarity.

Absence of red blood cells and hemolysis.

CRP Assay

CRP levels in donor plasma were measured using NADAL CRP rapid test (nal von minden GmbH, Germany), a semi-quantitative lateral flow immunoassay.

Ranges:

Normal: 0-6 μg/L.

Mild elevation: 10-40 μg/L.

High elevation: 40-80 μg/L.

Microscopic Assessment

Phase-contrast microscopy was used to evaluate platelet morphology, focusing on activation status (e.g., pseudopodia, granule extrusion). Digital images were obtained at ×1000 magnification.

Statistical Analysis

Fisher's exact test was used to compare proportions. A p-value <0.05 was considered statistically significant.

Results

Fibrin Presence in PCs

Out of 60 PCs:

43 (71.7%) showed fibrin at 72h.

17 (28.3%) were fibrin-free (controls).

CRP Levels

CRP Level	Fibrin-positive (n=43)	Control (n=17)
≤6 μg/L (normal)	34.9%	70.6%
10-40 μg/L (mild)	60.5%	29.4%
40-80 μg/L (high)	4.7%	0%

Overall CRP ≥10 μg/L:

Fibrin group: 65.1%.

Control group: 29.4%.

p = 0.0203 (statistically significant).

Microscopy Findings

Platelets in fibrin-positive PCs demonstrated:

Broad, irregular pseudopodia.

Granule degranulation.

Aggregation tendencies.

These features were absent or minimal in control samples.

Seasonal Variation

Increased frequency of fibrin-positive PCs was observed during autumn and winter months, coinciding with a rise in community-acquired respiratory infections.

Discussion

The study provides compelling evidence that subclinical inflammation in donors, reflected by elevated CRP, significantly correlates with fibrin formation in stored PCs. This phenomenon can be mechanistically linked to CRP's effects on platelet activation, particularly via its interaction with FcγRIIa and the complement system.

Our data confirm prior findings that CRP not only serves as a biomarker but also actively contributes to thrombogenesis by:

Inducing tissue factor expression in monocytes and platelets.

Enhancing platelet aggregation and prolonging activation.

Promoting fibrin polymerization.

The seasonal spike in fibrin-positive PCs may be a direct consequence of low-grade inflammation during viral infections, underscoring the need for dynamic donor screening strategies.

Conclusion

CRP levels in whole blood donors are a significant predictor of fibrin formation in PCs. Routine pre-donation screening using CRP assays, such as NADAL, is feasible, rapid, and cost-effective. Implementing this strategy could:

Reduce non-usable PCs by up to 70%.

Lower operational costs (reduced waste, consumables, labor).

Enhance clinical safety and transfusion efficiency.

Recommendations

Integrate CRP testing in donor pre-screening during peak infection seasons.

Establish CRP thresholds to defer donors with subclinical inflammation.

Combine with leukoreduction and pathogen inactivation for optimal PC quality.

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