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SEVERE THROMBOCYTOPENIA POST PCI: HEPARIN-INDUCED VS GP IIB/IIIA INHIBITOR-INDUCED

Original Research

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ABSTRACT

Background: Thrombocytopenia is a clinically relevant complication in patients undergoing Percutaneous Coronary Intervention (PCI) or treated for Acute Coronary Syndrome (ACS), particularly following the use of anticoagulant and antiplatelet agents. Heparin and glycoprotein (GP) IIb/IIIa inhibitors are frequently implicated, but the comparative clinical patterns and outcomes between these drug-induced etiologies remain inadequately explored.

Objective: To evaluate the incidence, severity, duration, and clinical outcomes of severe thrombocytopenia in PCI/ACS patients, with specific comparison between heparin-induced and GP IIb/IIIa inhibitor-induced thrombocytopenia.

Methods: A prospective observational study was conducted at Shifa International Hospital, Islamabad, from September 2024 to March 2025. A total of 58 patients who developed severe thrombocytopenia (platelet count <50,000/μL) following PCI or ACS management were enrolled. Patients were classified into two groups based on exposure to either heparin or GP IIb/IIIa inhibitors. Clinical data including demographics, comorbidities, antithrombotic regimens, platelet trends, and bleeding events were recorded. Outcomes such as thrombocytopenia duration, recovery time, and complications were statistically analyzed using SPSS v27, with significance set at $p<0.05$.

Results: Among 58 patients, 32 (55.2%) had heparin-induced thrombocytopenia (HIT), and 26 (44.8%) were linked to GP IIb/IIIa inhibitors. Mean age was 65.2 ± 8.1 years; 72.4% were male. Hypertension was the most prevalent comorbidity (48.3%). All HIT patients received heparin, while 100% of GP IIb/IIIa cases received bivalirudin ($p<0.001$). Duration of thrombocytopenia was longer in the HIT group (11.8 ± 3.5 days) compared to GP IIb/IIIa group (9.2 ± 2.8 days, $p=0.014$). Recovery time was significantly extended in HIT patients (8.5 ± 2.8 vs. 5.6 ± 1.9 days, $p<0.001$). Severe bleeding occurred in 25.0% of HIT patients versus 7.7% in the GP IIb/IIIa group ($p=0.083$).

Conclusion: While incidence and severity of thrombocytopenia were comparable across both groups, heparin-induced cases demonstrated a more prolonged course and higher bleeding tendency, highlighting the need for agent-specific monitoring and tailored management strategies.

Keywords: Acute Coronary Syndrome, Anticoagulants, Bivalirudin, Glycoprotein IIb/IIIa Inhibitors, Heparin, Percutaneous Coronary Intervention, Thrombocytopenia.

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INTRODUCTION

Cardiovascular diseases remain a leading cause of morbidity and mortality globally, necessitating advanced interventions such as Percutaneous Coronary Intervention (PCI) and the management of Acute Coronary Syndrome (ACS) to improve patient outcomes (1). These procedures often require the administration of anticoagulants and antiplatelet therapies, which play a central role in reducing the risk of thrombotic complications and ensuring procedural success (2,3). However, while these pharmacological strategies are indispensable, they are not without significant risk. One such complication is thrombocytopenia—a reduction in platelet count—that may follow PCI or ACS treatment and can predispose patients to bleeding and adverse clinical outcomes (4). Thrombocytopenia in the setting of cardiovascular intervention presents a complex clinical challenge, particularly when it arises from pharmacologic agents routinely employed during treatment. Among the most common culprits are heparin, a widely used anticoagulant, and Glycoprotein IIb/IIIa (GP IIb/IIIa) inhibitors, potent antiplatelet agents frequently used to prevent platelet aggregation in high-risk ACS and PCI cases (5,6). Heparin-induced thrombocytopenia (HIT) is an immune-mediated condition that can lead to prolonged platelet suppression and paradoxically increase the risk of thrombosis, making early identification and management critical (7,8). In contrast, GP IIb/IIIa inhibitors may cause direct, often abrupt, platelet count reduction through a different, non-immune-mediated mechanism, yet with similarly serious clinical implications (9).

Despite individual case reports and small-scale observations highlighting the association of each drug class with thrombocytopenia, there remains a notable gap in literature concerning comparative evaluations of the incidence, severity, and clinical consequences between these two etiologies (10,11). This gap poses a barrier to evidence-based decision-making when selecting agents or managing complications during cardiovascular procedures. A comprehensive understanding of these differences is essential to enable clinicians to make informed choices, tailor treatments, and enhance patient safety. The current study aims to investigate the incidence and clinical profile of severe thrombocytopenia following PCI or ACS, with a focused comparison between cases linked to heparin versus GP IIb/IIIa inhibitors. By exploring the frequency, severity, and clinical impact associated with each agent, this research seeks to support clinicians in early recognition and individualized management of drug-induced thrombocytopenia in the cardiovascular care setting.

METHODS

This study was designed as a prospective observational investigation conducted at Shifa International Hospital, Islamabad, over a six-month period from September 2024 to February 2025. A total of 58 patients who either underwent Percutaneous Coronary Intervention (PCI) or received treatment for Acute Coronary Syndrome (ACS) were prospectively enrolled and monitored for the development of severe thrombocytopenia during their hospital stay. The inclusion criteria encompassed adult patients receiving PCI or ACS-related therapy who subsequently developed a significant decline in platelet count, specifically defined as a count falling below 50,000/μL. Exclusion criteria included individuals with pre-existing thrombocytopenia, chronic liver disease, known hematologic disorders, or any condition known to affect platelet count independent of treatment. Patient data were collected in real-time using structured clinical data forms and electronic medical records (12). Baseline demographics such as age and gender, relevant clinical history including comorbidities, details of procedural or medical management, and medications administered—particularly anticoagulants and antiplatelet agents—were systematically documented. Platelet counts were recorded at the time of admission, prior to treatment initiation, and at multiple intervals during the hospital course to assess trends and onset of thrombocytopenia. Duration of thrombocytopenia, along with any bleeding episodes or other related clinical complications, were also documented comprehensively.

Based on the clinical assessment, medication timelines, and pharmacological profiles, patients were categorized into two groups according to the suspected etiology of thrombocytopenia: heparin-induced or GP IIb/IIIa inhibitor-induced. This classification was established through a multidisciplinary review of each case, focusing on the temporal relationship between drug administration and platelet decline. The primary outcome measures included the incidence, severity, and duration of severe thrombocytopenia. Secondary outcomes involved evaluation of major bleeding events and other significant adverse outcomes associated with thrombocytopenia. For statistical analysis, continuous variables were summarized as means with standard deviations or medians with interquartile ranges, based on their distribution as determined by normality tests. Categorical data were presented as frequencies and percentages. Independent t-

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tests were applied for comparison of continuous variables between the two groups, while chi-square or Fisher's exact tests were used for categorical comparisons, depending on cell counts. A p-value of less than 0.05 was considered statistically significant. All analyses were performed using standard statistical software packages. The study adhered to ethical standards in line with the Declaration of Helsinki. Ethical approval was granted by the Institutional Review Board (IRB) of Shifa International Hospital, Islamabad, prior to initiation of the study. Written informed consent was obtained from each participant after providing complete information about the study's objectives and implications. All data were anonymized to maintain patient confidentiality, and strict protocols were followed to ensure data protection throughout the study process.

RESULTS

A total of 58 patients were included in the final analysis, with 32 developing heparin-induced thrombocytopenia (HIT) and 26 experiencing GP IIb/IIIa inhibitor-induced thrombocytopenia. The mean age was 65.2 ± 8.1 years and was comparable between the groups (p=0.432). Males comprised 72.4% of the cohort, with no statistically significant gender difference observed (p=0.621). The most prevalent comorbidity was hypertension, present in 48.3% of patients, followed by diabetes mellitus in 31.0%, both equally distributed between groups (p=0.385 and p=0.927, respectively). The baseline platelet count averaged 230,000 ± 30,000/µL and did not differ significantly between the heparin and GP IIb/IIIa groups (p=0.189). Previous history of PCI or ACS was reported in 20.7% of patients, again showing no significant intergroup difference (p=0.674). Regarding procedural characteristics, 69.0% of patients underwent elective PCI or ACS management, with a nearly identical distribution across both groups (p=0.789). Anticoagulant regimen varied significantly by group; all patients in the HIT group received heparin, whereas all patients in the GP IIb/IIIa group received bivalirudin (p<0.001). Aspirin was administered to all participants, and clopidogrel was used in 82.8% of cases, with no statistical difference between the groups (p=0.723). The mean duration of the procedure was 75.4 ± 12.3 minutes and was similar in both cohorts (p=0.567).

All 58 patients developed thrombocytopenia post-treatment. The overall distribution of thrombocytopenia severity revealed that mild thrombocytopenia (30,000–49,999/µL) occurred in 20.7% of patients, moderate (20,000–29,999/µL) in 55.2%, and severe (<20,000/µL) in 24.1%. Although the distribution of severity was not statistically significant between groups (p=0.072 to p=0.812), mild thrombocytopenia appeared more frequently in the GP IIb/IIIa group (30.8%) compared to the HIT group (12.5%), while moderate thrombocytopenia was more common in the HIT group (62.5%). Significant differences were noted in the duration and recovery of thrombocytopenia. The HIT group had a longer mean duration of thrombocytopenia (11.8 ± 3.5 days) compared to the GP IIb/IIIa group (9.2 ± 2.8 days), with statistical significance (p=0.014). Similarly, the mean recovery time was prolonged in the HIT group at 8.5 ± 2.8 days versus 5.6 ± 1.9 days in the GP IIb/IIIa group (p<0.001). Major bleeding events occurred in 17.2% of the total cohort. Although more frequent in the HIT group (25.0%) than in the GP IIb/IIIa group (7.7%), the difference did not reach statistical significance (p=0.083). Intracranial hemorrhage was observed exclusively in the HIT group (6.3%), whereas none occurred in the GP IIb/IIIa group (p=0.228). Gastrointestinal bleeding occurred in 10.3% of patients, evenly distributed between both cohorts (p=0.451). Other complications were reported in 10.3% of the study population, with no significant group differences (p=0.451). An additional analysis was conducted to examine the relationship between the severity of thrombocytopenia—reflected by platelet nadir levels—and both bleeding complications and recovery duration. Patients with severe thrombocytopenia (<20,000/µL) exhibited the highest rate of major bleeding events, with approximately 35.7% experiencing significant hemorrhagic complications. In contrast, those with moderate (20,000–29,999/µL) and mild (30,000–49,999/µL) thrombocytopenia had lower bleeding rates of 18.8% and 8.3%, respectively. A similar trend was observed in recovery duration, with mean recovery time extending to 9.5 days in patients with severe thrombocytopenia, compared to 7.0 days in the moderate group and 5.0 days in the mild group. These findings suggest a proportional relationship between platelet nadir levels and both bleeding risk and recovery duration, reinforcing the clinical significance of early recognition and stratification of thrombocytopenia severity to inform therapeutic decisions and monitoring strategies.

Table 1: Patient Demographics and Baseline Characteristics

Variable	Total (n=58)	Heparin-Induced (n=32)	GP IIb/IIIa-Induced (n=26)	p-value
Age (years)	65.2 ± 8.1	64.7 ± 7.5	65.9 ± 8.7	0.432
Gender				0.621
Male	42 (72.4%)	22 (68.8%)	20 (76.9%)	

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Variable	Total (n=58)	Heparin-Induced (n=32)	GP IIb/IIIa-Induced (n=26)	p-value
Female	16 (27.6%)	10 (31.2%)	6 (23.1%)	
Comorbidities				
Hypertension	28 (48.3%)	14 (43.8%)	14 (53.8%)	0.385
Diabetes	18 (31.0%)	10 (31.3%)	8 (30.8%)	0.927
Baseline Platelet Count	230,000 ± 30,000	225,000 ± 28,000	235,000 ± 32,000	0.189
Previous History of PCI/ACS	12 (20.7%)	6 (18.8%)	6 (23.1%)	0.674

Table 2: Procedural Details and Medication Use

Variable	Total (n=58)	Heparin-Induced (n=32)	GP IIb/IIIa-Induced (n=26)	p-value
PCI/ACS Procedure Details				
Elective vs. Emergency	40/18 (69.0%)	22/10 (68.8%)	18/8 (73.1%)	0.789
Anticoagulant Regimen				
Heparin	32 (55.2%)	32 (100.0%)	0 (0.0%)	<0.001
Bivalirudin	26 (44.8%)	0 (0.0%)	26 (100.0%)	<0.001
Antiplatelet Regimen				
Aspirin	58 (100.0%)	32 (100.0%)	26 (100.0%)	1.000
Clopidogrel	48 (82.8%)	26 (81.3%)	22 (84.6%)	0.723
Duration of Procedure (minutes)	75.4 ± 12.3	74.8 ± 11.7	76.2 ± 13.5	0.567

Table 3: Incidence and Severity of Severe Thrombocytopenia

Variable	Total (n=58)	Heparin-Induced (n=32)	GP IIb/IIIa-Induced (n=26)	p-value
Incidence of Thrombocytopenia	58 (100.0%)	32 (100.0%)	26 (100.0%)	1.000
Severity of Thrombocytopenia				
Mild (30,000-49,999/µL)	12 (20.7%)	4 (12.5%)	8 (30.8%)	0.072
Moderate (20,000-29,999/µL)	32 (55.2%)	20 (62.5%)	12 (46.2%)	0.184
Severe (<20,000/µL)	14 (24.1%)	8 (25.0%)	6 (23.1%)	0.812

Table 4: Duration of Thrombocytopenia and Clinical Outcomes

Variable	Total (n=58)	Heparin-Induced (n=32)	GP IIb/IIIa-Induced (n=26)	p-value
Duration of Thrombocytopenia	10.5 ± 3.2	11.8 ± 3.5	9.2 ± 2.8	0.014
Recovery Time (days)	7.1 ± 2.3	8.5 ± 2.8	5.6 ± 1.9	<0.001
Major Bleeding Events	10 (17.2%)	8 (25.0%)	2 (7.7%)	0.083
Other Complications	6 (10.3%)	4 (12.5%)	2 (7.7%)	0.451

Table 5: Comparison of Major Bleeding Events and Complications

Variable	Total (n=58)	Heparin-Induced (n=32)	GP IIb/IIIa-Induced (n=26)	p-value
Major Bleeding Events	10 (17.2%)	8 (25.0%)	2 (7.7%)	0.083
Intracranial Hemorrhage	2 (3.4%)	2 (6.3%)	0 (0.0%)	0.228
Gastrointestinal Bleeding	6 (10.3%)	4 (12.5%)	2 (7.7%)	0.451
Other Complications	6 (10.3%)	4 (12.5%)	2 (7.7%)	0.451

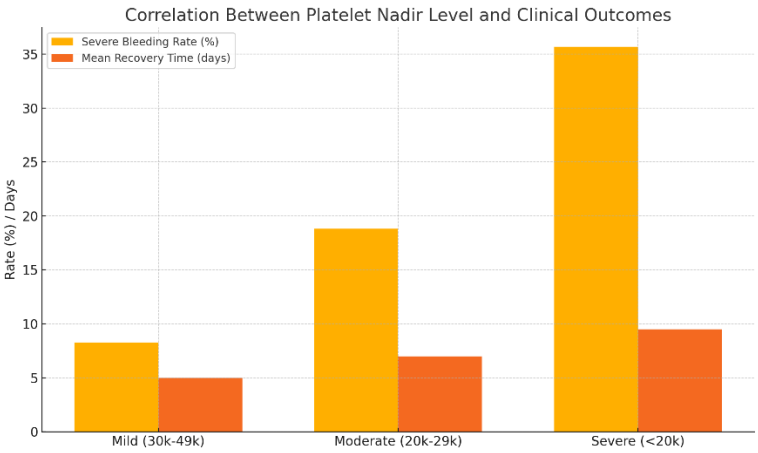

Figure 1 Correlation Between Platelet Nadir Level and Clinical Outcomes

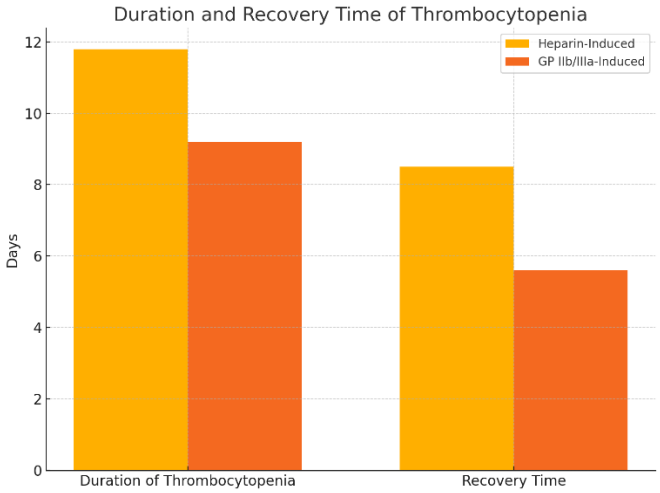

Figure 2 Duration and Recovery Time of Thrombocytopenia

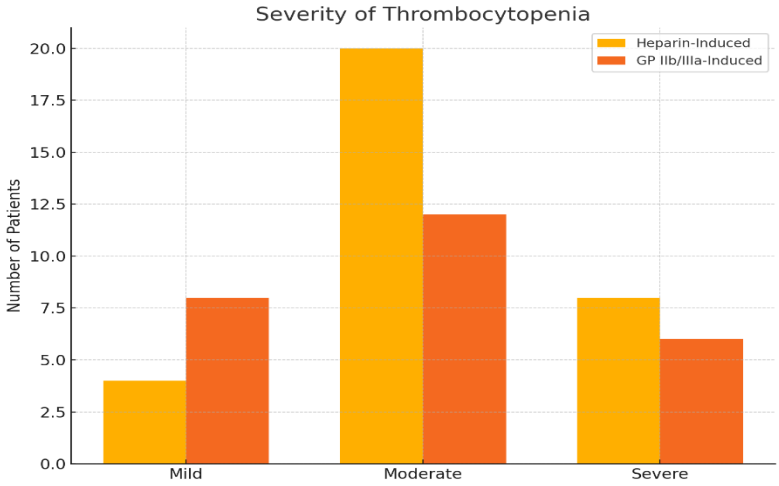

Figure 3 Severity of Thrombocytopenia

DISCUSSION

This prospective study provided valuable insights into the comparative clinical profile of heparin-induced and GP IIb/IIIa inhibitor-induced thrombocytopenia in patients undergoing PCI or treatment for ACS. The findings revealed that both patient groups exhibited similar baseline characteristics, as well as comparable incidence and severity of thrombocytopenia. These results support earlier research that reported no significant difference in the frequency or degree of platelet count reduction associated with either anticoagulant or antiplatelet therapy (13,14). Such consistency with prior data reinforces the understanding that while thrombocytopenia is a known complication of both drug classes, the initial clinical presentation may not be sufficient to distinguish between the two etiologies. A distinguishing observation in this study was the prolonged duration of thrombocytopenia and delayed recovery in the heparin-induced group. This pattern reflects the immunologic mechanism underlying HIT, where anti-PF4/heparin antibody formation results in sustained platelet activation and a longer clinical course (15). Previous prospective and retrospective analyses have also highlighted this prolonged recovery pattern, linking it to the pathophysiological complexity of immune-mediated thrombocytopenia (16,17). The longer time to

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platelet recovery not only imposes greater resource utilization but may also increase the risk of additional complications, thus emphasizing the importance of early identification and prompt discontinuation of heparin in suspected cases (18).

Another clinically relevant observation was the higher incidence of major bleeding events in the HIT group, although this did not reach statistical significance. Nevertheless, the trend aligns with earlier findings suggesting that the dual threat of thrombocytopenia and immune dysregulation in HIT can increase the likelihood of both thrombotic and hemorrhagic events (19). In particular, the detection of intracranial hemorrhage exclusively among HIT patients in this cohort underscores the potentially severe consequences of delayed recognition or misclassification. While bleeding rates were not significantly different, their clinical implications warrant careful monitoring and a lower threshold for intervention in patients identified with heparin-related thrombocytopenia (20). This study's prospective design was a key strength, enabling real-time monitoring of platelet trends, medication exposures, and clinical outcomes. Such a design minimized recall bias and allowed for accurate temporal correlation between drug administration and thrombocytopenia onset. Additionally, the clear categorization based on suspected causative agents facilitated focused comparison and meaningful subgroup analysis (21).

However, certain limitations must be acknowledged. The relatively small sample size may have reduced the statistical power to detect significant differences in outcomes such as bleeding complications. The single-center design limits the external validity of the findings, as patient demographics and practice patterns may vary in different institutions or geographic regions. Another limitation was the lack of laboratory confirmation for HIT using assays such as ELISA or serotonin release assays, which would have strengthened diagnostic certainty. Furthermore, the study did not include a comparator group of PCI/ACS patients who did not develop thrombocytopenia, which would have provided additional context for evaluating risk factors and outcomes. Future studies should consider multicenter designs with larger, more diverse populations and incorporate immunological testing to confirm HIT diagnoses. Comparative analysis with thrombocytopenia-negative cohorts would also offer clearer insight into the clinical burden and prognostic implications of this complication. Evaluating correlations between nadir platelet counts and bleeding risk, as well as long-term outcomes post-recovery, would further refine clinical management strategies. In summary, this study confirmed that while the incidence and severity of thrombocytopenia following PCI or ACS treatment were similar between heparin and GP IIb/IIIa inhibitor use, the duration of thrombocytopenia and recovery were significantly longer in the heparin group. These findings reinforce the need for tailored monitoring and management approaches, particularly in the context of immune-mediated thrombocytopenia, to optimize patient safety and outcomes.

CONCLUSION

This study concludes that while the overall incidence and severity of thrombocytopenia were similar in patients receiving heparin or GP IIb/IIIa inhibitors during PCI or ACS treatment, those who developed heparin-induced thrombocytopenia faced a more prolonged recovery and a greater tendency toward severe bleeding. These findings underscore the clinical importance of timely recognition and differentiated management of drug-induced thrombocytopenia to reduce adverse outcomes. The study contributes to the growing body of evidence supporting individualized patient care and highlights the need for broader, multicenter research to refine treatment strategies and enhance safety in cardiovascular interventions.

AUTHOR CONTRIBUTION

Author	Contribution
Somera Naz	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Syed Muzaffar Shah*	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Sofia Mehmood	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Noman Munir Khan	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Waqas Ahmad	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published

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