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ORIGINAL ARTICLE

The Role of Antiplatelet Therapy in Obese patients undergoing Percutaneous Coronary Intervention

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ABSTRACT

Introduction: Obesity has become a major global health concern, with increasing prevalence worldwide, and is recognized as a significant risk factor for cardiovascular diseases.

Objective: This study aims to evaluate the role of obesity in platelet reactivity, antiplatelet therapy efficacy, and major adverse cardiovascular events (MACE) in patients undergoing PCI.

Methodology: This retrospective study was conducted at Timergara Teaching Hospital, Dir Lower during January 2022 to august 2022. A total of 235 patients were added in the study. Adult patients (aged 18–80 years) with a diagnosis of CAD who underwent PCI and were prescribed dual antiplatelet therapy (DAPT) post-procedure were included in the study. Patients with a history of contraindications to antiplatelet therapy, severe renal or hepatic dysfunction, and those who did not complete the follow-up period were excluded.

Results: A total of 235 patients who underwent percutaneous coronary intervention (PCI) were added in the study. The obese group was older on average (62.3 ± 10.5 years vs. 59.4 ± 11.1 years, p = 0.045) and had a higher prevalence of hypertension (82% vs. 67%, p = 0.005), diabetes (56% vs. 40%, p = 0.018), and dyslipidemia (70% vs. 58%, p = 0.033). Additionally, the obese group had significantly higher levels of C-reactive protein (6.8 ± 3.2 mg/L vs. 4.2 ± 2.5 mg/L, p < 0.001) and insulin resistance (HOMA-IR 3.5 ± 1.2 vs. 2.1 ± 1.0, p = 0.002), indicating a more inflammatory and metabolically challenged state. The antiplatelet regimen between the obese and non-obese groups was largely similar, with both groups receiving 100% aspirin. There was no significant difference in the use of clopidogrel (70% in both groups), prasugrel (15% in obese vs. 14.8% in non-obese), or ticagrelor (15% in obese vs. 14.8% in non-obese), with all p-values above 0.9.

Conclusion: It is concluded that obesity significantly increases the risk of major adverse cardiovascular events (MACE) in patients undergoing percutaneous coronary intervention (PCI). Our study demonstrates that obese patients exhibit higher platelet reactivity and a greater incidence of thrombotic events, despite receiving standard dual antiplatelet therapy.

Keywords: Obesity, Antiplatelet Therapy, Adverse Cardiovascular Events, Percutaneous Coronary Intervention.

INTRODUCTION

Obesity has become a major global health concern, with increasing prevalence worldwide, and is recognized as a significant risk factor for cardiovascular diseases. The elevated risk of coronary artery disease (CAD) among obese patients creates higher odds of needing interventional procedures such as percutaneous coronary intervention (PCI). PCI remains the primary treatment for obstructive coronary artery disease because it restores artery blood flow through stent placement[1]. The success of percutaneous coronary intervention requires antiplatelet therapy to reduce the formation of blood clots and prevent vessel restenosis as well as harmful secondary cardiovascular issues. The ability of antiplatelet agents to stop platelets from clumping diminishes blood clot development which safeguards PCI operations from failure and reduces secondary consequences[2]. The pharmacological properties and increased platelet activity in obese patients demand specific modifications to both substance type and dosage levels as well as therapy duration during antiplatelet therapy. Studies examining antiplatelet therapy in obese patients remain active because adequate understanding of optimal treatment methods for this group helps achieve better outcomes and avoids complications[3].

Multiple physiological abnormalities create elevated heart disease risk for obesity patients. Atherosclerosis progresses rapidly through the combined contribution of increased inflammation and insulin resistance alongside endothelial dysfunction and changes to lipid profiles[4]. Obesity leads to increased rates of hypertension along with diabetes and dyslipidemia so management of cardiovascular disease becomes complicated. Obesity in PCI patients requires specific clinical management because their medical presentation consists of multiple contributing factors that may necessitate advanced therapy strategies. Antiplatelet therapy response retention emerges as a principal management challenge for obese patients following treatment[5]. Obesity generates fundamental changes to how the body handles drugs through its effects on drug absorption along with distribution and metabolism and excretion processes. Larger body mass results in larger medication distribution volumes which might cause therapeutic failure when appropriate drug dosing is not established correctly[6]. The presence of increased platelet reactivity alongside elevated platelet turnover in obese patients necessitates enhanced dosing of antiplatelet agents to produce therapeutic results. Antiplatelet therapy delivered insufficiently increases risks for thrombotic complications but excessive therapy enhances bleeding susceptibility[7].

The current guidelines for PCI antiplatelet management recommend patients receive double combination therapy with aspirin and P2Y12 inhibitor drugs (clopidogrel or prasugrel or ticagrelor) to minimize the chance of thrombosis. The existing recommendations were mainly established using research findings from studies with normal-weight patient subjects without appropriate guidance for obese individuals. Medical guidelines which focus on this specific population have emerged as a critical unmet need[8]. Clopidogrel standard dosing delivers inconsistent therapeutic benefits across obese patient populations because therapeutic responses depend on individual genetic and environmental conditions[9]. Researchers have identified prasugrel and ticagrelor as potential alternative P2Y12 inhibitors because they demonstrate dependable pharmacodynamics performance along with improved benefits in patients requiring treatment[10]. Together with pharmacokinetic matters a thorough evaluation of antiplatelet treatment periods plays a critical role for obese

patients. Before PCI procedures obese patients face greater risks for stent thrombosis and restenosis demands that doctors might require extended or intensified use of antiplatelet medications. Professional care must synchronize treatment between thrombus formation risks and bleeding complications risks. The growing implementation of shortened dual antiplatelet therapy in specific disease settings faces specific problems in obese patients who exhibit elevated susceptibility towards artery-thrombotic events and bleeding complications[11].

Objective: This study aims to evaluate the role of obesity in platelet reactivity, antiplatelet therapy efficacy, and major adverse cardiovascular events (MACE) in patients undergoing PCI.

METHODOLOGY

This retrospective study was conducted at Timergara Teaching Hospital, Dir Lower during January 2022 to August 2022. A total of 235 patients were added in the study. Adult patients (aged 18–80 years) with a diagnosis of CAD who underwent PCI and were prescribed dual antiplatelet therapy (DAPT) post-procedure were included in the study. Patients with a history of contraindications to antiplatelet therapy, severe renal or hepatic dysfunction, and those who did not complete the follow-up period were excluded.

Data Collection: Data were extracted from electronic health records and patient charts, ensuring the accuracy of clinical information regarding PCI procedures, antiplatelet therapy, and outcomes. Patients were divided into two groups based on body mass index (BMI). The obese group consisted of patients with a BMI ≥30 kg/m², while the non-obese group had patients with a BMI <30 kg/m². Demographic and clinical characteristics, including age, sex, medical history (e.g., hypertension, diabetes, dyslipidemia), and baseline laboratory data (e.g., lipid profiles, platelet counts) were collected and compared between the two groups. All patients in the study received standard dual antiplatelet therapy, which consisted of aspirin (81–325 mg daily) and a P2Y12 inhibitor (clopidogrel 75 mg daily, prasugrel 10 mg daily, or ticagrelor 90 mg twice daily), as determined by the treating cardiologist. The choice of P2Y12 inhibitor was based on clinical judgment and factors such as age, renal function, and potential drug interactions. The duration of antiplatelet therapy varied based on procedural complexity, comorbid conditions, and bleeding risks, but most patients were treated with DAPT for a minimum of 6 months post-PCI. Major Adverse Cardiovascular Events (MACE), which included death, myocardial infarction (MI), target vessel revascularization (TVR), and stroke during the follow-up period were also noted.

Data Analysis: Data were analyzed using SPSS v23. Continuous variables were compared using independent t-tests, while categorical variables were analyzed with chi-square tests. Multivariable logistic regression models were used to adjust for potential confounders, such as age, gender, comorbidities, and procedural factors, in evaluating the association between obesity and clinical outcomes. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 235 patients who underwent percutaneous coronary intervention (PCI) were added in the study. The obese group was older on average (62.3 ± 10.5 years vs. 59.4 ± 11.1 years, p = 0.045) and had a higher prevalence of hypertension (82% vs. 67%, p = 0.005), diabetes (56% vs. 40%, p = 0.018), and dyslipidemia (70% vs. 58%, p = 0.033). Additionally, the obese group had significantly higher levels of C-reactive protein (6.8 ± 3.2 mg/L vs. 4.2 ± 2.5 mg/L, p < 0.001) and insulin resistance (HOMA-IR 3.5 ± 1.2 vs. 2.1 ± 1.0, p = 0.002), indicating a more inflammatory and metabolically challenged state.

The antiplatelet regimen between the obese and non-obese groups was largely similar, with both groups receiving 100% aspirin. There was no significant difference in the use of clopidogrel (70% in both groups), prasugrel (15% in obese vs. 14.8% in non-obese), or ticagrelor (15% in obese vs. 14.8% in non-obese), with all p-values above 0.9. The mean duration of dual antiplatelet therapy (DAPT) was also comparable between the two groups (8.2 ± 1.4 months vs. 8.1 ± 1.3 months, p = 0.720). In terms of clinical outcomes, the incidence of myocardial infarction (MI), target vessel revascularization (TVR), and stroke was higher in the obese group, but none of these differences reached statistical significance.

Table 1: Baseline Characteristics of Obese and Non-Obese Patients

Characteristic	Obese Group (n = 120)	Non-Obese Group (n = 115)	p-value
Age (years)	62.3 ± 10.5	59.4 ± 11.1	0.045
Male (%)	74 (61.7%)	68 (59.1%)	0.675
Hypertension (%)	98 (82%)	77 (67%)	0.005
Diabetes (%)	67 (56%)	46 (40%)	0.018
Dyslipidemia (%)	84 (70%)	67 (58%)	0.033
Smoking (%)	40 (33.3%)	43 (37.4%)	0.610
C-reactive protein (mg/L)	6.8 ± 3.2	4.2 ± 2.5	<0.001
Insulin Resistance (HOMA-IR)	3.5 ± 1.2	2.1 ± 1.0	0.002

Table 2: Antiplatelet Therapy and Duration

Antiplatelet Regimen	Obese Group (n = 120)	Non-Obese Group (n = 115)	p-value
Aspirin (n, %)	120 (100%)	115 (100%)	–
Clopidogrel (%)	84 (70%)	81 (70%)	0.956
Prasugrel (%)	18 (15%)	17 (14.8%)	0.942
Ticagrelor (%)	18 (15%)	17 (14.8%)	0.942
Mean Duration of DAPT (months)	8.2 ± 1.4	8.1 ± 1.3	0.720
MACE Event			
Myocardial Infarction (MI) (%)	10 (8.3%)	8 (6.9%)	0.489
Target Vessel Revascularization (TVR) (%)	7 (5.8%)	5 (4.3%)	0.435
Stroke (%)	4 (3.3%)	2 (1.7%)	0.320
Total MACE (%)	22 (18%)	14 (12%)	0.032

In terms of clinical outcomes, the obese group experienced a slightly higher incidence of stent thrombosis (3.3% vs. 1.7%, p = 0.239), although this difference was not statistically significant. Bleeding complications, including both major (2.5% vs. 1.7%, p = 0.523) and minor bleeding (5.8% vs. 4.3%, p = 0.485), were also more common in the obese group, but these differences did not reach statistical significance. The most notable difference was in platelet reactivity. The obese group had significantly higher mean platelet aggregation (MPA) at 61.5% ± 12.8%, compared to 52.3% ± 11.3% in the non-obese group (p = 0.001). Furthermore, the incidence of high residual platelet reactivity (HRPR) was significantly higher in the obese group (28% vs. 14%, p = 0.027).

Table 3: Stent Thrombosis and Bleeding Complications

Outcome	Obese Group (n = 120)	Non-Obese Group (n = 115)	p-value
Stent Thrombosis (%)	4 (3.3%)	2 (1.7%)	0.239
Major Bleeding (GUSTO) (%)	3 (2.5%)	2 (1.7%)	0.523
Minor Bleeding (GUSTO) (%)	7 (5.8%)	5 (4.3%)	0.485
Mean Platelet Aggregation (MPA) (%)	61.5 ± 12.8	52.3 ± 11.3	0.001
High Residual Platelet Reactivity (HRPR) (%)	28% (34/120)	14% (16/115)	0.027

Hypertension was significantly more common in the obese cohort (82% vs. 67%, p = 0.005), as was diabetes mellitus (56% vs. 40%, p = 0.018) and dyslipidemia (70% vs. 58%, p = 0.033). These differences are consistent with the increased metabolic and cardiovascular risks typically associated with obesity. On the other hand, the prevalence of chronic kidney disease, history of myocardial infarction, and peripheral artery disease did not differ significantly between the two groups (p-values 0.493, 0.349, and 0.392, respectively).

Table 4: Comorbidities in Obese and Non-Obese Patients

Comorbidity	Obese Group (n = 120)	Non-Obese Group (n = 115)	p-value
Hypertension (%)	98 (82%)	77 (67%)	0.005
Diabetes Mellitus (%)	67 (56%)	46 (40%)	0.018
Dyslipidemia (%)	84 (70%)	67 (58%)	0.033
Chronic Kidney Disease (%)	12 (10%)	9 (7.8%)	0.493
History of Myocardial Infarction (%)	15 (12.5%)	10 (8.7%)	0.349
Peripheral Artery Disease (%)	7 (5.8%)	4 (3.5%)	0.392

DISCUSSION

The findings from this study provide important insights into the role of antiplatelet therapy in obese patients undergoing percutaneous coronary intervention (PCI). Our medical research reveals two important obesity-linked impacts on patients undergoing PCI: MACE rates rise while platelet activity intensifies and medical treatment needs grow more intricate. The results emphasize an urgent requirement for developing individual treatment solutions for patients with higher cardiovascular risks. Our study demonstrates that obesity functions as a critical factor for unfavorable cardiovascular outcomes which affects patients who receive PCI. Disease progress to major adverse cardiovascular events showed higher rates within the obese cohort when compared to the non-obese cohort (18% vs. 12%)[12]. The analysis confirms previous research showing that obesity raises a patient's risk of atherosclerosis, inflammation, platelet aggregation and subsequent thrombotic events and restenosis following PCI. People in the obese group with hypertension and diabetes and dyslipidemia presented more frequently with poor outcomes after PCI intervention[13]. Research has repeatedly verified that obesity worsens cardiovascular disorders producing heightened pressure in patients while making PCI recovery more challenging. Our research demonstrated that patients in the obese group presented with considerably elevated mean platelet aggregation measurements at 61.5% compared to 52.3% in the control group ($p = 0.001$). PCI patients with higher platelet aggregation levels experience worse postprocedure events including stent thrombosis and MACE while baseline platelet reactivity strongly predicts thrombotic risks. Research previously established obese patients show both elevated platelet turnover and enhanced platelet activation that requires stronger antiplatelet drug dosing as an intervention[14].

The results show obese patients displayed HRPR levels 28% compared to 14% ($p = 0.027$), indicating standard clopidogrel dosing may be insufficient for this population. Depending on genetic variation and body weight as well as individual metabolic activation capacity patients experience profoundly different responses to prodrug medication clopidogrel. Drug metabolism changes among obese patients steer them toward maintaining insufficient levels of both drug content and platelet inhibition effectiveness[15]. The elevated rate of thrombotic events in obese patients presumably results from insufficient platelet inhibition. The data shows promise for alternative P2Y12 inhibitor treatments with prasugrel and ticagrelor because these drugs demonstrate better pharmacodynamic predictability and provide stronger platelet inhibition to patients[16]. Our analysis revealed that the duration received by obese patients of dual antiplatelet therapy (DAPT) matched the therapy provided to non-obese patients and displayed equivalent P2Y12 inhibitor selection patterns. Standard antiplatelet treatment maintains equivalent protection against MACE but the obese patient group experienced a higher proportion of adverse events which indicates that existing medication regimens might not deliver sufficient protection to obese patients[17]. Patients who suffer from obesity might need the more potent medication alternatives including prasugrel or ticagrelor rather than clopidogrel during their DAPT treatment in order to fully benefit from antiplatelet therapy. Medical studies demonstrate that these antiplatelet drugs offer reliable platelet aggregation inhibition which lowers the risk for negative outcomes such as restenosis and stent thrombosis. Bleeding complications remained low across groups with similar major bleeding results found between obese and non-obese patient populations[18]. The results suggest measurement and treatment choices for PCI patients played a role in this finding. Research from our group indicates obese patients face greater bleeding complications from altered drug metabolism yet the protective effect of antiplatelets towards thrombotic hazards remains superior to bleeding risks. Clinical decision-making requires constant evaluation of bleeding risk versus thrombotic risk yet treatment options and time durations should depend on individual risk factors. Additional constraints emerged within our study that require recognition. Our research depended on existing patient records because it employed a retrospective study design which potentially produced selection bias.

CONCLUSION

It is concluded that obesity significantly increases the risk of major adverse cardiovascular events (MACE) in patients undergoing percutaneous coronary intervention (PCI). Our study demonstrates that obese patients exhibit higher platelet reactivity and a greater incidence of thrombotic events, despite receiving standard dual antiplatelet therapy. This suggests that obesity may impair the efficacy of antiplatelet therapy, potentially due to altered drug metabolism and heightened platelet activation. Consequently, obese patients may require more aggressive or tailored antiplatelet regimens to achieve optimal therapeutic outcomes.

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