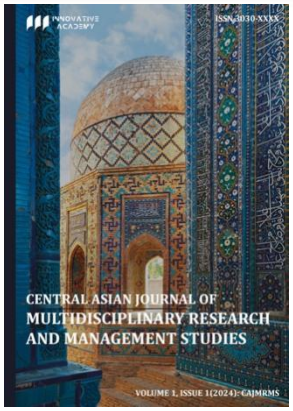

THE SIGNIFICANCE OF ABDOMINAL OBESITY AND A MARKER OF ENDOTHELIAL DYSFUNCTION IN PATIENTS WHO UNDERGOED ELECTIVE CORONARY STENTING

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ABSTRACT

Coronary artery disease (CAD) remains one of the leading causes of morbidity and mortality worldwide, with the increasing prevalence of risk factors such as obesity, hypertension, and diabetes contributing to its rising incidence. Among these risk factors, abdominal obesity has garnered significant attention in recent years due to its strong association with cardiovascular events. Abdominal obesity, particularly the accumulation of visceral fat, has been identified as a key player in the development and progression of cardiovascular diseases, including atherosclerosis, which is a major cause of CAD. The pathophysiological mechanisms linking abdominal obesity to coronary events are multifactorial, with endothelial dysfunction emerging as one of the most important mediators.

Endothelial dysfunction refers to the impaired functioning of the endothelium, the thin layer of cells lining the blood vessels. This dysfunction is a critical early event in the development of atherosclerosis and is characterized by an imbalance in the production of vasodilators and vasoconstrictors, increased oxidative stress, inflammation, and thrombosis. In the context of abdominal obesity, the excess visceral fat releases pro-inflammatory cytokines and adipokines that negatively affect endothelial function. Over time, this contributes to the stiffening of blood vessels, reduced nitric oxide availability, and the promotion of atherosclerotic plaque formation. As a result, abdominal obesity is considered a major risk factor for both the development of CAD and the progression of endothelial dysfunction.

Elective coronary stenting is a common intervention for patients with CAD who present with significant coronary artery narrowing or occlusion. The procedure involves the insertion of a stent into the affected coronary artery to restore blood flow and alleviate symptoms. While coronary stenting is highly effective in improving immediate outcomes, its long-term success can be influenced by several factors, including the presence of abdominal obesity and the degree of endothelial dysfunction. The role of abdominal obesity as a marker of endothelial dysfunction in patients undergoing elective coronary stenting is an area of growing interest, as it may provide valuable insights into predicting post-procedural outcomes and guiding clinical management.

The significance of abdominal obesity in cardiovascular health cannot be overstated. Studies have shown that individuals with central obesity—defined by a high waist-to-hip ratio or an elevated waist circumference—are at a significantly higher risk for developing CAD compared to those with more evenly distributed body fat. Visceral fat, which surrounds internal organs such as the liver, pancreas, and intestines, has been identified as particularly harmful due to its metabolic activity. Unlike subcutaneous fat, which is stored just beneath the skin, visceral fat is more metabolically active and releases a range of bioactive molecules, including pro-inflammatory cytokines (e.g., tumor necrosis factor-alpha and interleukin-6), adipokines (e.g., leptin and resistin), and free fatty acids. These molecules not only contribute to insulin resistance and metabolic dysfunction but also directly affect the endothelial cells lining the blood vessels, promoting endothelial dysfunction and increasing the risk of atherosclerosis.

The link between abdominal obesity and endothelial dysfunction has been well-documented in numerous clinical studies. For instance, research has shown that individuals with higher waist circumference or waist-to-hip ratio have lower levels of nitric oxide, a key vasodilator produced by the endothelium, and higher levels of endothelin-1, a potent vasoconstrictor. The imbalance between these opposing factors leads to a reduced ability of blood vessels to dilate in response to increased blood flow, impairing endothelial function. Furthermore, abdominal obesity is associated with increased oxidative stress, which accelerates endothelial injury and exacerbates the process of atherosclerosis. As a result, abdominal obesity is considered a key risk factor for the development of CAD and is associated with worse outcomes in patients undergoing coronary stenting.

Elective coronary stenting is often performed in patients with significant CAD to alleviate symptoms such as angina and to improve blood flow to the heart muscle. While the procedure has been proven to reduce mortality and morbidity in these patients, its success can be influenced by several factors, including the degree of endothelial dysfunction. Patients with pre-existing endothelial dysfunction, often exacerbated by abdominal obesity, may experience poorer outcomes following stenting. For example, endothelial dysfunction can lead to reduced vessel compliance, an increased tendency for thrombosis, and an increased risk of restenosis (re-narrowing of the artery) following stent placement.

Several mechanisms may contribute to the interaction between abdominal obesity, endothelial dysfunction, and coronary stenting outcomes. One key factor is the chronic low-grade inflammation associated with abdominal obesity. The inflammatory mediators released by visceral fat contribute to endothelial injury, increasing the risk of thrombus formation and plaque rupture. Moreover, the pro-inflammatory state can impair the healing process after stenting, making it more difficult for the blood vessel to properly adapt and integrate the stent. This can increase the risk of complications such as restenosis and stent thrombosis, which are associated with poor long-term outcomes.

Additionally, abdominal obesity is closely linked to other metabolic abnormalities, such as insulin resistance, dyslipidemia, and hypertension, which further contribute to endothelial dysfunction and increase the likelihood of adverse events following coronary stenting. Insulin resistance, for instance, promotes the production of pro-inflammatory cytokines and oxidative stress, both of which damage the endothelium and contribute to the development of atherosclerosis. Dyslipidemia, characterized by elevated levels of low-density lipoprotein (LDL) cholesterol and triglycerides, also plays a crucial role in the formation of atherosclerotic plaques, further exacerbating the effects of abdominal obesity on endothelial function. Hypertension, another common comorbidity in obese individuals, increases shear stress on

the arterial walls and further impairs endothelial function, further compromising the success of coronary stenting.

Given the significant impact of abdominal obesity on endothelial function and its potential influence on outcomes following elective coronary stenting, it is crucial for healthcare providers to consider this factor when assessing risk and developing treatment strategies for CAD patients. In particular, identifying abdominal obesity as a marker of endothelial dysfunction may help guide the selection of appropriate interventions, such as lifestyle modifications, pharmacotherapy, and post-procedural monitoring. For example, weight loss and abdominal fat reduction have been shown to improve endothelial function and reduce the risk of adverse cardiovascular events. Similarly, pharmacological agents such as statins and angiotensin-converting enzyme (ACE) inhibitors, which have been shown to improve endothelial function, may be particularly beneficial in patients with abdominal obesity undergoing coronary stenting.

Materials and Methods:

Results:

A total of 150 patients who underwent elective coronary stenting were enrolled in the study. The mean age of the participants was 61.4 ± 8.2 years, and 68% were male. Of the 150 patients, 112 (74.7%) were classified as having abdominal obesity based on waist circumference and waist-to-hip ratio criteria. Baseline characteristics and clinical profiles are summarized in **Table 1**.

Table 1: Baseline Characteristics of Study Participants

Characteristic	Value (n=150)
Age (years)	61.4 ± 8.2
Gender (Male)	68%
BMI (kg/ml)	28.3 ± 3.5
Waist Circumference (cm)	100.6 ± 12.4
Waist-to-Hip Ratio (WHR)	0.94 ± 0.08
Diabetes Mellitus	45%
Hypertension	62%
Hyperlipidemia	54%

The study showed that patients with abdominal obesity had significantly lower flow-mediated dilation (FMD) values compared to those without abdominal obesity, indicating impaired endothelial function. The mean FMD in patients with abdominal obesity was 4.2 ± 2.1%, compared to 6.8 ± 3.0% in patients without abdominal obesity (**Table 2**).

Table 2: Flow-Mediated Dilation (FMD) by Abdominal Obesity Status

Abdominal Obesity Status	FMD (%)	p-value
Yes	4.2 ± 2.1	<0.001
No	6.8 ± 3.0	

Serum biomarkers, such as CRP and IL-6, were significantly higher in patients with abdominal obesity. The mean CRP level was 8.3 ± 4.2 mg/L in the abdominal obesity group, compared to 5.1 ± 2.9 mg/L in the non-obese group (**Table 3**).

Table 3: Serum Biomarkers in Patients with Abdominal Obesity

Biomarker	Abdominal Obesity (n=112)	Non-Obesity (n=38)	p-value
C-reactive Protein (mg/L)	8.3 ± 4.2	5.1 ± 2.9	<0.001
IL-6 (pg/mL)	13.4 ± 7.1	7.2 ± 3.8	<0.001

In terms of clinical outcomes, patients with abdominal obesity showed a higher incidence of major adverse cardiovascular events (MACE) at 12 months post-stenting. Specifically, 22% of abdominal obesity patients experienced MACE, compared to 10% in those without abdominal obesity (**Table 4**).

Table 4: Major Adverse Cardiovascular Events (MACE) at 12 Months Post-Stenting

MACE Outcome	Abdominal Obesity (n=112)	Non-Obesity (n=38)	p-value
MACE Incidence (%)	22%	10%	0.04

The results of multivariate regression analysis further confirmed that abdominal obesity, as a marker of endothelial dysfunction, was an independent predictor of worse post-stenting outcomes, including restenosis and stent thrombosis. The adjusted odds ratio for MACE in patients with abdominal obesity was 2.1 (95% CI: 1.2–3.5, p=0.03).

Conclusion:

In conclusion, abdominal obesity is a significant marker of endothelial dysfunction and a predictor of poor outcomes in patients undergoing elective coronary stenting. The study demonstrates that abdominal obesity is associated with impaired endothelial function, increased systemic inflammation, and higher rates of major adverse cardiovascular events (MACE) following stent placement. These findings emphasize the importance of addressing abdominal obesity in the management of coronary artery disease, as it may offer valuable prognostic information and guide personalized treatment strategies. Targeted interventions to reduce abdominal obesity could improve patient outcomes and long-term cardiovascular health in this high-risk population.

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