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Review Article

The No-reflow Phenomenon in Acute Coronary Syndromes: A Comprehensive Review

Akut Koroner Sendromlarda No-reflow Fenomeni: Kapsamlı Bir İnceleme

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Abstract

Invasive interventions have long been widely used in acute coronary syndromes. Effective reperfusion of the ischemic myocardium should be the primary strategy in patients with acute coronary syndrome. Successful reperfusion is strongly associated with improved clinical outcomes. Conversely, inadequate reperfusion is associated with increased mortality and cardiovascular events in the subsequent period. In some patients, mechanical opening of the coronary arteries is insufficient. Restoring epicardial blood flow does not always ensure microvascular circulation and tissue perfusion. In this microcirculation disorder, called the "no-reflow" phenomenon, there is no mechanical occlusion in the coronary artery, but there is significant myocardial perfusion impairment. Given the recent advances in diagnostic tools and pharmacological treatments, this review aims to provide a comprehensive update on the no-reflow phenomenon to help guide clinical management.

Keywords: Acute coronary syndrome, No-reflow

ÖZ

Akut koroner sendromlarda (AKS) invaziv girişimler uzun süredir yaygın olarak uygulanmaktadır. Akut koroner sendromlu hastalarda iskemik miyokardın etkili bir şekilde reperfüze edilmesi ana stratejiyi oluşturmalıdır. Başarılı reperfüzyon, klinik sonuçların iyileşmesiyle güçlü biçimde ilişkilidir. Buna karşın, yetersiz reperfüzyon, takip eden dönemde mortalite ve kardiyovasküler olaylarda artışla ilişkilidir. Bazı hastalarda koroner arterlerin mekanik olarak açılması yeterli olmamaktadır. Epikardiyal kan akımının sağlanması her zaman mikrovasküler dolaşım ve doku perfüzyonunu sağlamamaktadır. "No-reflow" fenomeni adı verilen bu mikrosirkülasyon bozukluğunda koroner arterde mekanik bir tıkanma yokken miyokardda önemli perfüzyon bozukluğu mevcuttur. Tanısal araçlardaki ve farmakolojik tedavilerdeki son gelişmeler göz önüne alındığında, bu derleme, klinik yönetime/uygulamaya rehberlik etmek amacıyla no-reflow fenomeni üzerine kapsamlı bir güncelleme sunmayı amaçlamaktadır.

Anahtar Kelimeler: Akut koroner sendrom, No-reflow

Highlights

- Successful mechanical opening of the coronary artery does not always ensure tissue perfusion**.**
- The no-reflow phenomenon is associated with higher mortality and adverse clinical outcomes.
- Prevention strategies such as direct stenting are often more effective than treating established no-reflow.

Introduction

Percutaneous coronary interventions (PCIs) continue to be the primary treatment modality for acute coronary syndromes, aiming to achieve effective reperfusion. he primary goal is to achieve revascularization and reperfusion of the coronary arteries. After percutaneous coronary intervention, the abrupt cessation of coronary blood flow (TIMI 0–1) in the absence of angiographic evidence of dissection, thrombus, spasm, or significant residual stenosis is defined as "no-reflow." No-reflow can occur in both acute coronary syndromes and stable coronary artery disease and is caused by structural and functional changes in the coronary microcirculation. No-reflow is also associated with an increased incidence of rehospitalization, negative ventricular remodelling, malignant arrhythmias, and heart failure. It is an independent predictor of myocardial infarction and heart-related mortality (1). Risk factors include age over 65 years, hypertension, smoking, dyslipidaemia, diabetes, renal insufficiency, atrial fibrillation, high thrombus burden, delayed primary interventions, and the use of high-pressure or oversized stents (2). Many methods have been developed to assess reperfusion effectiveness, and these have undergone some changes in recent years, reflecting both evolving treatment strategies and technological advances.

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With percutaneous coronary intervention becoming the standard of care, angiographic evaluation has become the primary standard for determining reperfusion success. Cardiac magnetic resonance imaging, on the other hand, has demonstrated higher sensitivity and better prognostic classification than angiography for detecting no-reflow and reperfusion injury. Despite this, coronary angiography remains the most common and accessible method for assessing no-reflow in practice.

Material and Methods

This review was conducted by searching the PubMed, Scopus, and Google Scholar databases for studies published between 1974 and 2024. The search focused on keywords such as 'no-reflow phenomenon', 'microvascular obstruction', 'acute coronary syndromes', 'percutaneous coronary intervention', and 'myocardial reperfusion'. Priority was given to clinical trials, meta-analyses, and the most recent clinical practice guidelines. Only studies published in the English language were included to ensure that the information presented is up-to-date and clinically accurate.

Ethical approve

Ethical committee approval is not required for this study.

Pathophysiology

The “no-reflow” phenomenon is characterized by complex functional and structural alterations within the coronary microcirculation. While it was initially demonstrated in the myocardium by Kloner et al. (3), contemporary research highlights that the process is multifactorial rather than a single event (2). During percutaneous intervention, the disruption of an atherosclerotic plaque may lead to distal embolization. This increase in vascular resistance, combined with the release of proinflammatory and vasoconstrictive mediators, contributes to further microinfarctions (4). The primary mechanisms include distal atherothrombotic embolization, ischemic damage, reperfusion injury, and individual susceptibility to microvascular injury (5). Prolonged ischemia results in myocyte and endothelial cell death, which impairs nitric oxide production and increases vascular endothelial growth factor (VEGF) levels, leading to interstitial edema (5). Furthermore, the sudden restoration of blood flow triggers reperfusion injury through neutrophil influx and the production of free oxygen radicals (6). These processes collectively result in the characteristic endothelial edema and intraluminal obstruction observed in no-reflow areas **(Table 1).**

Table 1. Pathophysiological Components of No-Reflow

Mechanism	Key Factors	Microvascular Impact
Distal Embolization	Plaque debris, microthrombi	Increased resistance and microinfarctions
Ischemic Damage	ATP depletion, NO impairment	Endothelial swelling and interstitial edema
Reperfusion Injury	Neutrophils, free oxygen radicals	Post-ischemic inflammation and capillary plugging
Structural Changes	Fibrin clumps, endothelial protrusions	Physical obstruction of the microluminal space

Prognosis and frequency

No-reflow leads to reduced five-year survival in patients with acute coronary syndromes (with a mortality rate of 18.2% in those with no-reflow compared to 9.5% in patients with normal coronary flow). The increase in mortality associated with no-reflow was observed to be independent of other variables such as infarct size, multivessel disease, or Killip class (7). Larger infarct size was also noted in the no-reflow group (15% of the left ventricle in the no-reflow group compared with 8% in normal-flow patients). Furthermore, no-reflow has been associated with an increased incidence of systolic heart failure and malignant arrhythmias (ventricular tachycardia, ventricular fibrillation, and high-degree atrioventricular [AV] block).

The no-reflow rate varies depending on the patient subgroups included in the studies. The incidence of no-reflow during percutaneous coronary interventions has been reported to range from 2% to 5% (8-9). No-reflow is more frequently observed in coronary lesions with high thrombus burden and in degenerated saphenous vein grafts. The rate was higher (1.2-9.0%) when coronary rotational atherectomy was used and was generally reversible (10-11).

Diagnosis

Several different classifications are used to assess coronary blood flow:

TIMI flow classification is a standard angiographic measure used to assess the degree of reperfusion in coronary arteries. It plays a critical role in determining the success of reperfusion after percutaneous coronary intervention, particularly in acute coronary syndromes. According to this classification, TIMI 0 flow represents complete

obstruction, with no antegrade blood flow distal to the lesion.
1. TIMI 1 flow represents a situation where blood flow partially bypasses the occlusive lesion but cannot adequately fill the distal coronary bed, and perfusion is severely inadequate.
TIMI 2 flow represents a situation where the distal coronary bed is filled, but the flow rate is significantly slower than normal.
TIMI 3 flow indicates that complete reperfusion, equivalent to a normal flow rate, has been achieved.
TIMI 0–1 flow is considered no-reflow.
2. Coronary Myocardial Perfusion Classification (Myocardial Blush Grade, MPG) complements the TIMI flow classification and directly reflects the degree of myocardial capillary perfusion.
MPG 0 indicates no myocardial blush in the distribution area of the treated artery and significant microvascular obstruction.
MPG 1 indicates minimal, but very weak, insufficient staining.
MPG 2 indicates myocardial staining is present but less intense than usual.
MPG 3 indicates complete microvascular reperfusion with normal intensity and rapid myocardial staining.
MPG 0–1 is associated with no reflow.
3. TIMI Myocardial Perfusion Grade (TMP) is an angiographic assessment method developed to measure true perfusion at the myocardial level, unlike the TIMI flow classification, which assesses epicardial vascular patency. This classification is based on the rate of contrast medium filling and disappearance in the myocardial tissue.
TMP 0 indicates that contrast is completely absent from the myocardial tissue and indicates the presence of severe microvascular obstruction.
TMP 1 indicates that contrast reaches the myocardium but distributes very slowly and is limited, resulting in a significant perfusion defect.
TMP 2 indicates that contrast distributes to some extent in the myocardium but clears more slowly than normal and represents partial suboptimal reperfusion.
TMP 3 indicates that contrast rapidly fills and disappears normally in the myocardial tissue, indicating complete and effective microvascular reperfusion.
TMP 0–1 is associated with no-reflow.
In addition to angiographic assessment, functional measurements such as Coronary Flow Reserve (CFR) and the Index of Microcirculatory Resistance (IMR) provide crucial diagnostic data; specifically, an IMR value >40 is highly predictive of significant microcirculatory dysfunction (12). Other modalities, including myocardial contrast echocardiography and positron emission tomography, may also be utilized. However, gadolinium-enhanced cardiovascular magnetic resonance imaging (CMR) remains the gold standard, as it allows for the precise visualization of microvascular obstruction (MVO) and intramyocardial haemorrhage (12). Table 2 provides a comparative overview of the standard angiographic perfusion grades used in clinical practice. **(Table 2).**

Table 2. Comparison of Angiographic Diagnostic Classifications (TIMI, MBG, and TMP)

Parameter	Definition	Grading (0-3)	Clinical Significance
TIMI Flow Grade	Rate of epicardial coronary blood flow.	0: No perfusion; 1: Penetration without albumin; 2: Partial perfusion (sluggish); 3: Complete perfusion.	Primary tool for epicardial patency; may overlook microvascular obstruction (MVO).
Myocardial Blush Grade (MBG)	Intensity of myocardial contrast opacification (density-based).	0: No blush; 1: Minimal blush; 2: Moderate blush; 3: Normal myocardial blush.	Visual assessment of myocardial tissue-level perfusion and microvascular integrity.
TIMI Myocardial Perfusion (TMP)	Rate of contrast clearance from the myocardium (time-based).	0: No entry; 1: Entry but no clearance; 2: Delayed clearance; 3: Normal entry and clearance.	Superior to MBG in predicting long-term survival and recovery of left ventricular function.

Prevention

In patients with acute coronary syndromes or those at high risk for no-reflow, direct stenting may be considered—provided the plaque is soft and non-calcified—whereas high-pressure post-dilatation should generally be avoided. Furthermore, when using rotational atherectomy, shorter burr runs (<20 seconds) and lower speeds (140,000 to 150,000 rpm) are advisable, while decelerations of more than 5,000 rpm should be avoided. For SVG PCI, the use of distal embolic protection devices and pretreatment with intracoronary vasodilators should be considered.

In 2024, Luke P. Dawson et al. developed and externally validated the NORPACS risk score to predict the development of angiographic no-reflow in patients undergoing PCI for acute coronary syndrome. They created a simple and practical scoring system consisting of six variables strongly associated with no-reflow. The results indicate that the score can be used in clinical practice to identify high-risk patients at an early stage, thereby allowing the implementation of preventive strategies. (13). The specific weight of each parameter and the clinical risk threshold are detailed in **(Table 3).**

Table 3. The NORPACS Scoring System and Risk Stratification

Variable	Definition / Criteria	Points
Cardiogenic Shock	Presence of cardiogenic shock at presentation	4
Symptom Duration	STEMI symptom duration **>195 minutes**	3
Lesion Location	Specific coronary segment involved	3
Stent Specifications	Anticipated stent length **≥20 mm**	2
Vessel Anatomy	Vessel diameter **<2.5 mm**	2
Baseline Flow	Pre-procedural TIMI flow grade **<3**	2
Total Score Assessment	**Score ≥8 indicates a no-reflow risk exceeding 20%**	**Max: 16**

Beside the NORPACS score, recent studies have used artificial intelligence to evaluate various hematological markers. These studies suggest that parameters such as the mean platelet volume-to-lymphocyte ratio (MPVLR) may provide additional help in predicting the risk of no-reflow phenomenon (14).

Treatment

If no-reflow occurs following percutaneous coronary intervention, alternative causes—including coronary dissection, systemic hypotension, distal thrombus migration, and vasospasm—must first be excluded through angiographic imaging. Once vascular patency is confirmed, vasodilators should be administered directly into the distal vessel, preferably via a microcatheter. Throughout this process, maintaining a therapeutic activated clotting time (ACT) is essential, and hemodynamic support devices (e.g., Impella, intra-aortic balloon pump) should be utilized if clinically indicated. In cases involving a high thrombus burden, thrombectomy should be performed. The pharmacological agents utilized, including their specific administration routes and dosages, are summarized in **(Table 4)**. (2, 15, 16).

For intracoronary administration of drugs, a microcatheter or over-the-wire balloon should be used in the distal coronary artery. This minimizes systemic side effects and ensures drug access to microcirculation. A double-lumen microcatheter or thrombectomy catheter enables distal drug delivery without compromising wire position. If no-reflow persists and the patient is hemodynamically stable, drugs can be administered as multiple boluses. If flow improvement is not achieved with the first bolus, different vasodilators should be used. Calcium channel blockers (CCBs) (verapamil, diltiazem, nicardipine) cause smooth muscle relaxation and coronary vasodilation through channel binding in vascular smooth muscle, cardiac myocytes, and nodal cells. (CCBs) have been shown to have limited benefits in no-reflow therapy in a limited number of studies (17,18). Adenosine causes vasodilation in the coronary microcirculation and relaxes smooth muscle by binding to A2 receptors, and its effect lasts quite briefly. It also has anti-inflammatory properties and inhibits platelet aggregation, supporting ischemic preconditioning by limiting reperfusion injury. Side effects include bradycardia, hypotension, dyspnoea, bronchospasm, and flushing (19). The REOPEN-AMI (Comparison of Intracoronary Nitroprusside and Adenosine in Acute Myocardial Infarction) trial demonstrated a significant improvement in coronary circulation and peak troponin compared with placebo or sodium nitroprusside, leading to a reduction in major cardiovascular events (20). In addition to traditional antiplatelet therapy, ticagrelor loading plays a key role in preventing microvascular injury. Beyond potent P2Y12 inhibition, ticagrelor improves microcirculatory flow by increasing local adenosine levels. This mechanism provides an additional protective effect against the no-reflow phenomenon during primary percutaneous interventions (2, 12). Sodium nitroprusside is a potent vasodilator that inhibits platelet aggregation in the coronary circulation. It is metabolized to its active form, nitric oxide, and its duration of action is longer than that of vasodilators. (21).

Table 4. Pharmacological Agents and Clinical Considerations in No-Reflow Management

Drug/Group	Route	Dose	Side Effects	Contraindications
Adenosine	IC/IV	• 50–200 μg bolus (IC) • 70 μg/kg/min (IV)	Bradycardia, transient AV block, hypotension	High-grade AV block, severe asthma
Verapamil	IC	• 100–250 μg bolus	Hypotension, heart block, negative inotropy	Severe heart failure, advanced AV block
Diltiazem	IC	• 400 μg bolus	Hypotension, bradycardia	Heart failure with low EF
Nicardipine	IC	• 50–200 μg bolus	Hypotension, tachycardia	Severe aortic stenosis
Nitroprusside	IC	• 50–200 μg bolus	Profound hypotension, headache	Severe hypotension, hypovolemia
Epinephrine	IC	• 50–200 μg bolus	VT/VF risk, tachycardia, hypertension	Severe tachyarrhythmias
GP IIb/IIIa inhibitors	IV			
Abciximab	IV	• Bolus: 0.25 μg /kg • Infusion: 0.125 μg /kg/min	Bleeding, thrombocytopenia	Active bleeding, recent surgery
Eptifibatide	IV	• Bolus: 180 μg /kg • Supplement: 25 μg /kg IV bolus • Infusion: 2 μg /kg/min	Bleeding, hypotension	Severe renal failure (if not adjusted)
Tirofiban	IV	• Bolus: 25 μg /kg • Infusion: 0.15 μg /kg/min	Bleeding	Hypersensitivity, active bleeding

Epinephrine has been effectively used in the treatment of no-reflow syndromes resistant to other treatments or in cases where these cannot be used. The recently published COAR study compared intracoronary epinephrine and adenosine in the treatment of no-reflow in normotensive patients with acute coronary syndromes and found epinephrine to be more effective than adenosine in correcting no-reflow, with a higher rate of TIMI III coronary flow (90.1% vs. 78%, p=0.019) (22). However, despite its efficacy, the use of intracoronary epinephrine carries a significant risk of inducing malignant arrhythmias, specifically ventricular tachycardia and ventricular fibrillation. Therefore, continuous electrocardiographic and hemodynamic monitoring is essential during its administration. (12, 22,23) Glycoprotein IIB/IIIA inhibitors are potent antiplatelet agents that block platelet aggregation. While some studies suggest limited routine benefit, according to the 2023 ESC guidelines, GP IIb/IIIa inhibitors should be considered as a 'bail-out' therapy if there is evidence of no-reflow or thrombotic complications (recommendation class IIa, level of evidence C) (12). Recent evidence indicates that their intracoronary administration may be particularly useful in managing microvascular obstruction in high-risk patients (12, 24). The role of intracoronary fibrinolytic agents in the treatment of no-reflow is still debated. Although some initial positive data documented benefits for myocardial reperfusion, subsequent studies have not confirmed these findings (25).

Study limitations

There are some limitations to this review. First, since it is a narrative review rather than a systematic meta-analysis, we did not perform a statistical analysis of the data. Second, we only included studies published in English, which means we might have missed relevant findings in other languages. Finally, because the review covers a long period (1974–2024), the definitions of no-reflow have changed over time, making it difficult to compare older studies with modern ones directly.

Conclusion

The no-reflow phenomenon is a complex condition that increases mortality and morbidity after percutaneous coronary intervention in patients with acute coronary syndrome. Necessary precautions should be taken when performing interventions on lesions at high risk of no-reflow, and patients' treatment should be managed using a combination of pharmacological and non-pharmacological methods.

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