Pathogenesis of myocardial infarction and the role of thrombosis.

Javed Hochman*

Department of Cardiology, University of Brasilia, Brasilia, Brazil

Introduction

Myocardial infarction (MI), commonly known as a heart attack, is a life-threatening condition that occurs due to the interruption of blood flow to the heart muscle. The interruption of blood flow leads to damage or death of the heart muscle tissue, which can have severe consequences. MI is a prevalent cause of death worldwide, and the pathogenesis of MI is complex, involving various factors such as atherosclerosis, thrombosis, and inflammation. Thrombosis, the formation of a blood clot within a blood vessel, is a crucial factor in the pathogenesis of MI. The formation of a thrombus occurs when there is a disruption in the balance between procoagulant and anticoagulant factors in the blood. The main process that triggers thrombosis is the rupture or erosion of atherosclerotic plaques in the coronary arteries [1].

Atherosclerosis is a chronic inflammatory disease that affects the inner lining of the arteries and leads to the accumulation of plaque. Plaque is composed of lipids, inflammatory cells, smooth muscle cells, and extracellular matrix. As the plaque accumulates, the artery narrows, reducing blood flow to the heart muscle. The plaque also becomes unstable and prone to rupture or erosion, exposing the underlying tissue to the blood. This exposure triggers the coagulation cascade and the formation of a thrombus. Thrombosis can also occur without plaque rupture, as in the case of endothelial injury or dysfunction. Endothelial dysfunction is a condition in which the inner lining of the blood vessels loses its ability to regulate vascular tone and function. Endothelial dysfunction can occur due to various factors such as hypertension, hypercholesterolemia, smoking, and diabetes mellitus. Endothelial dysfunction can lead to the activation of platelets and the formation of a thrombus [2].

The formation of a thrombus in the coronary arteries can have severe consequences, leading to MI. The thrombus can occlude the artery, blocking blood flow to the heart muscle. This blockage leads to ischemia, a condition in which the heart muscle does not receive enough oxygen and nutrients to function properly. Ischemia can cause chest pain, also known as angina pectoris, and can progress to MI if left untreated. MI occurs when the ischemic heart muscle tissue undergoes irreversible damage or death. The extent of the damage depends on the duration and severity of the ischemia. The heart muscle tissue can become necrotic, leading to the release of cardiac enzymes such as troponin and creatine kinase (CK) into the bloodstream. The elevation of these enzymes is a hallmark of MI and can help diagnose the condition [3].

The role of thrombosis in the pathogenesis of MI has led to the development of various treatments aimed at preventing or reducing the formation of thrombi. One of the most common treatments is antiplatelet therapy, which inhibits the activation of platelets and the formation of thrombi. Aspirin is a widely used antiplatelet agent that irreversibly inhibits cyclooxygenase-1 (COX-1), an enzyme that plays a crucial role in platelet activation. Other antiplatelet agents such as clopidogrel, prasugrel, and ticagrelor inhibit the adenosine diphosphate (ADP) pathway, which is another critical pathway in platelet activation [4].

Anticoagulant therapy is also used in the management of MI. Anticoagulants inhibit the coagulation cascade, preventing the formation of fibrin, the main component of thrombi. Heparin is a widely used anticoagulant that inhibits thrombin and factor Xa, two critical enzymes in the coagulation cascade. Direct oral anticoagulants (DOACs) such as dabigatran, rivaroxaban, and apixaban are also used in the management of MI and have shown promising results in clinical trials. Another treatment modality that targets thrombosis in the management of MI is reperfusion therapy. Reperfusion therapy aims to restore blood flow to the ischemic heart muscle tissue, reducing the extent of damage and improving outcomes. The two main forms of reperfusion therapy are primary percutaneous coronary intervention (PCI) and thrombolytic therapy [5].

Primary PCI involves the insertion of a catheter into the coronary artery, followed by the inflation of a balloon to open the artery and the placement of a stent to keep it open. Thrombolytic therapy involves the administration of drugs that dissolve the thrombus, restoring blood flow to the heart muscle tissue. Thrombolytic therapy is usually reserved for patients who cannot undergo primary PCI or present to the hospital outside the optimal time window for primary PCI.

Conclusion

Thrombosis plays a crucial role in the pathogenesis of MI. The disruption of the balance between procoagulant and anticoagulant factors in the blood leads to the formation of a thrombus in the coronary arteries, blocking blood flow to the heart muscle and leading to ischemia and ultimately MI. The development of treatments targeting thrombosis such as antiplatelet and anticoagulant therapy, as well as reperfusion therapy, has improved outcomes for patients with MI. Further research is needed to identify novel targets and treatments for thrombosis to further improve outcomes for patients with MI.

***Correspondence to:** Javed Hochman. Department of Cardiology, University of Brasilia, Brasilia, Brazil, E-mail: hochman.javed@unb.br

***Received:** 16-May-2023, Manuscript No. AAINIC-23-99677; **Editor assigned:** 19-May-2023, Pre QC No. AAINIC-23-99677(PQ); **Reviewed:** 02-Jun-2023, QC No. AAINIC-23-99677; **Revised:** 06-Jun-2023, Manuscript No. AAINIC-23-99677(R); **Published:** 13-Jun-2023, DOI:10.35841/aainic-6.3.147*

***Citation:** Hochman J. Pathogenesis of myocardial infarction and the role of thrombosis. J Invasive Noninvasive Cardiol. 2023;6(3):147*

References

1. Gorlin RI, Fuster VA, Ambrose JA. Anatomic-physiologic links between acute coronary syndromes. Circulation. 1986;74(1):6-9.
2. DeWood MA, Spores J, Notske R, et al. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. N Engl J Med. 1980;303(16):897-902.
3. Borissoff JI, Spronk HM, ten Cate H. The hemostatic system as a modulator of atherosclerosis. N Engl J Med. 2011;364(18):1746-60.
4. Javed U, Aftab W, Ambrose JA, et al. Frequency of elevated troponin I and diagnosis of acute myocardial infarction. Am J Cardiol. 2009;104(1):9-13.
5. Yeh RW, Sidney S, Chandra M, et al. Population trends in the incidence and outcomes of acute myocardial infarction. N Engl J Med. 2010;362(23):2155-65.

Citation: *Hochman J. Pathogenesis of myocardial infarction and the role of thrombosis. J Invasive Noninvasive Cardiol. 2023;6(3):147*