ACUTE MYOCARDIAL INFARCTION IN THE CLINIKAL HOSPITAL IN STIP

Author: Prof. d-r Gordana Panova,Nenad Panov,L.Nikolovska, N.Velickova,V.Dzidrova, A. Stojanovski

 Introduction: Heart diseases in industrialized countries are the most common causes of death. Acute myocardial infarction (AIM) is a life threatening condition. Gold standard treatment is coronary intervention if angiographic laboratory is available in the first 6 hours of infarction. From quick and expert assessment of condition depends on patient outcome of the intervention. Angiographic and interventional lab team is available 24 hours for acceptance of patients in critical condition. Angiographic room at any time has the most sophisticated equipment and materials used in interventional cardiology or modified coronary catheters for single use and coronary wires, stents and balloons. Life-threatening complications in the room available fully equipped resuscitation cart with defibrillator, emergency drugs and equipment for intubation and respirator, aspirator, catheters and apparatus for intraaortalna kontrapulsacija (IABP), Autopulse ® - System automatska chest compression during resuscitation. After localization of the lesion, it is dilatira with balloon or stent. Objectives: To emphasize that the shortest time interval for detecting the site of occlusion and otvorenje the same, with balloon dilatation or stent, in order revascularization of ischemic region in the myocardium is a condition for avoiding further complications. Results: Of 289 patients admitted for coronary intervention in acute coronary anchor were 16 (5.4%) patients. After completion of the intervention pacintot remains of Unit of observation. Conclusion: The cutting edge equipment, standardized procedures and thoroughly trained staff provides timely and professional response to emergency situations.
**Key words**: acute myocardial, angiography, resuscitation, coronary syndrome

ACUTE MYOCARDIAL INFARCTION

**Introduction**: Acute myocardial infarction (MI) remains a leading cause of morbidity and mortality worldwide. Myocardial infarction occurs when myocardial ischemia, a diminished blood supply to the heart, exceeds a critical threshold and overwhelms myocardial cellular repair mechanisms designed to maintain normal operating function and homeostasis. Ischemia at this critical threshold level for an extended period results in irreversible myocardial cell damage or death.Critical myocardial ischemia can occur as a result of increased myocardial metabolic demand, decreased delivery of oxygen and nutrients to the myocardium via the coronary circulation, or both. An interruption in the supply of myocardial oxygen and nutrients occurs when a thrombus is superimposed on an ulcerated or unstable atherosclerotic plaque and results in coronary occlusion.[1](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#bib1) A high-grade (>75%) fixed coronary artery stenosis caused by atherosclerosis or a dynamic stenosis associated with coronary vasospasm can also limit the supply of oxygen and nutrients and precipitate an MI. Conditions associated with increased myocardial metabolic demand include extremes of physical exertion, severe hypertension (including forms of hypertrophic obstructive cardiomyopathy), and severe aortic valve stenosis. Other cardiac valvular pathologies and low cardiac output states associated with a decreased mean aortic pressure, which is the prime component of coronary perfusion pressure, can also precipitate MI.Myocardial infarction can be subcategorized on the basis of anatomic, morphologic, and diagnostic clinical information. From an anatomic or morphologic standpoint, the two types of MI are transmural and nontransmural. A transmural MI is characterized by ischemic necrosis of the full thickness of the affected muscle segment(s), extending from the endocardium through the myocardium to the epicardium. A nontransmural MI is defined as an area of ischemic necrosis that does not extend through the full thickness of myocardial wall segment(s). In a nontransmural MI, the area of ischemic necrosis is limited to the endocardium or to the endocardium and myocardium. It is the endocardial and subendocardial zones of the myocardial wall segment that are the least perfused regions of the heart and the most vulnerable to conditions of ischemia. An older subclassification of MI, based on clinical diagnostic criteria, is determined by the presence or absence of Q waves on an electrocardiogram (ECG). However, the presence or absence of Q waves does not distinguish a transmural from a nontransmural MI as determined by pathology.[2](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#bib2)A consensus statement was published to give a universal definition of the term myocardial infarction. The authors stated that MI should be used when there is evidence of myocardial necrosis in a clinical setting consistent with MI. Myocardial infarction was then classified by the clinical scenario into various subtypes. Type 1 is a spontaneous MI related to ischemia from a primary coronary event (e.g., plaque rupture, thrombotic occlusion). Type 2 is secondary to ischemia from a supply-and-demand mismatch. Type 3 is an MI resulting in sudden cardiac death. Type 4a is an MI associated with percutaneous coronary intervention, and 4b is associated with in-stent thrombosis. Type 5 is an MI associated with coronary artery bypass surgery.[3](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#bib3)A more common clinical diagnostic classification scheme is also based on electrocardiographic findings as a means of distinguishing between two types of MI, one that is marked by ST elevation (STEMI) and one that is not (NSTEMI). Management practice guidelines often distinguish between STEMI and non-STEMI, as do many of the studies on which recommendations are based. The distinction between STEMI and NSTEMI also does not distinguish a transmural from a nontransmural MI. The presence of Q waves or ST-segment elevation is associated with higher early mortality and morbidity; however, the absence of these two findings does not confer better long-term mortality and morbidity.[4](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#bib4)Prevalence and risk factors-Myocardial infarction is the leading cause of death in the United States and in most industrialized nations throughout the world. Approximately 450, 000 people in the United States die from coronary disease per year.[5](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#bib5) The survival rate for U.S. patients hospitalized with MI is approximately 95%. This represents a significant improvement in survival and is related to improvements in emergency medical response and treatment strategies.The incidence of MI increases with age; however, the actual incidence is dependent on predisposing risk factors for atherosclerosis. Approximately 50% of all MIs in the United States occur in people younger than 65 years. However, in the future, as demographics shift and the mean age of the population increases, a larger percentage of patients presenting with MI will be older than 65 years.Six primary risk factors have been identified with the development of atherosclerotic coronary artery disease and MI: hyperlipidemia, diabetes mellitus, hypertension, tobacco use, male gender, and family history of atherosclerotic arterial disease. The presence of any risk factor is associated with doubling the relative risk of developing atherosclerotic coronary artery disease.[1](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#bib1)Hyperlipidemia-Elevated levels of total cholesterol, LDL, or triglycerides are associated with an increased risk of coronary atherosclerosis and MI. Levels of HDL less than 40 mg/dL also portend an increased risk. A full summary of the National Heart, Lung, and Blood Institute's cholesterol guidelines is available online.[6](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#bib6)Diabetes Mellitus-Patients with diabetes have a substantially greater risk of atherosclerotic vascular disease in the heart as well as in other vascular beds. Diabetes increases the risk of MI because it increases the rate of atherosclerotic progression and adversely affects the lipid profile. This accelerated form of atherosclerosis occurs regardless of whether a patient has insulin-dependent or non–insulin-dependent diabetes.Hypertension-High blood pressure (BP) has consistently been associated with an increased risk of MI. This risk is associated with systolic and diastolic hypertension. The control of hypertension with appropriate medication has been shown to reduce the risk of MI significantly. A full summary of the National Heart, Lung, and Blood Institute's JNC 7 guidelines published in 2003 is available online.[7](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#bib7)Tobacco Use-Certain components of tobacco and tobacco combustion gases are known to damage blood vessel walls. The body's response to this type of injury elicits the formation of atherosclerosis and its progression, thereby increasing the risk of MI. A small study in a group of volunteers showed that smoking acutely increases platelet thrombus formation. This appears to target areas of high shear forces, such as stenotic vessels, independent of aspirin use.[8](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#bib8) The American Lung Association maintains a website with updates on the public health initiative to reduce tobacco use and is a resource for smoking-cessation strategies for patients and health care providers.Male Gender-The incidence of atherosclerotic vascular disease and MI is higher in men than women in all age groups. This gender difference in MI, however, narrows with increasing age.Family History-A family history of premature coronary disease increases an individual's risk of atherosclerosis and MI. The cause of familial coronary events is multifactorial and includes other elements, such as genetic components and acquired general health practices (e.g. smoking, high-fat diet).Pathophysiology and natural history-Most myocardial infarctions are caused by a disruption in the vascular endothelium associated with an unstable atherosclerotic plaque that stimulates the formation of an intracoronary thrombus, which results in coronary artery blood flow occlusion. If such an occlusion persists for more than 20 minutes, irreversible myocardial cell damage and cell death will occur.The development of atherosclerotic plaque occurs over a period of years to decades. The two primary characteristics of the clinically symptomatic atherosclerotic plaque are a fibromuscular cap and an underlying lipid-rich core. Plaque erosion can occur because of the actions of matrix metalloproteases and the release of other collagenases and proteases in the plaque, which result in thinning of the overlying fibromuscular cap. The action of proteases, in addition to hemodynamic forces applied to the arterial segment, can lead to a disruption of the endothelium and fissuring or rupture of the fibromuscular cap. The loss of structural stability of a plaque often occurs at the juncture of the fibromuscular cap and the vessel wall, a site otherwise known as the *shoulder region.* Disruption of the endothelial surface can cause the formation of thrombus via platelet-mediated activation of the coagulation cascade. If a thrombus is large enough to occlude coronary blood flow, an MI can result.The death of myocardial cells first occurs in the area of myocardium most distal to the arterial blood supply: the endocardium. As the duration of the occlusion increases, the area of myocardial cell death enlarges, extending from the endocardium to the myocardium and ultimately to the epicardium. The area of myocardial cell death then spreads laterally to areas of watershed or collateral perfusion. Generally, after a 6- to 8-hour period of coronary occlusion, most of the distal myocardium has died. The extent of myocardial cell death defines the magnitude of the MI. If blood flow can be restored to at-risk myocardium, more heart muscle can be saved from irreversible damage or death.The severity of an MI depends on three factors: the level of the occlusion in the coronary artery, the length of time of the occlusion, and the presence or absence of collateral circulation. Generally, the more proximal the coronary occlusion, the more extensive the amount of myocardium that will be at risk of necrosis. The larger the myocardial infarction, the greater the chance of death because of a mechanical complication or pump failure. The longer the period of vessel occlusion, the greater the chances of irreversible myocardial damage distal to the occlusion.STEMI is usually the result of complete coronary occlusion after plaque rupture. This arises most often from a plaque that previously caused less than 50% occlusion of the lumen. NSTEMI is usually associated with greater plaque burden without complete occlusion. This difference contributes to the increased early mortality seen in STEMI and the eventual equalization of mortality between STEMI and NSTEMI after 1 year.Signs and symptoms-Acute MI can have unique manifestations in individual patients. The degree of symptoms ranges from none at all to sudden cardiac death. An asymptomatic MI is not necessarily less severe than a symptomatic event, but patients who experience asymptomatic MIs are more likely to be diabetic. Despite the diversity of manifesting symptoms of MI, there are some characteristic symptoms.

* Chest pain described as a pressure sensation, fullness, or squeezing in the midportion of the thorax
* Radiation of chest pain into the jaw or teeth, shoulder, arm, and/or back
* Associated dyspnea or shortness of breath
* Associated epigastric discomfort with or without nausea and vomiting
* Associated diaphoresis or sweating
* Syncope or near syncope without other cause
* Impairment of cognitive function without other cause

An MI can occur at any time of the day, but most appear to be clustered around the early hours of the morning or are associated with demanding physical activity, or both. Approximately 50% of patients have some warning symptoms (angina pectoris or an anginal equivalent) before the infarct.

**Diagnosis**-Identifying a patient who is currently experiencing an MI can be straightforward, difficult, or somewhere in between. A straightforward diagnosis of MI can usually be made in patients who have a number of atherosclerotic risk factors along with the presence of symptoms consistent with a lack of blood flow to the heart. Patients who suspect that they are having an MI usually present to an emergency department. Once a patient's clinical picture raises a suspicion of MI, several confirmatory tests can be performed rapidly. These tests include electrocardiography, blood testing, and echocardiography.-Diagnostic ProceduresThe first diagnostic test is electrocardiography (ECG), which may demonstrate that a MI is in progress or has already occurred. Interpretation of an ECG is beyond the scope of this chapter; however, one feature of the ECG in a patient with an MI should be noted because it has a bearing on management. Practice guidelines on MI management consider patients whose ECG does or does not show ST-segment elevation separately. As noted earlier, the former is referred to as ST elevation MI ([Fig. 1](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#f0010)) and the latter as non-ST elevation MI ([Fig. 2](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#f0015)). In addition to ST-segment elevation, 81% of electrocardiograms during STEMI demonstrate reciprocal ST-segment depression as well.



[Figure 1: Click to Enlarge](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/images/amifig1_large.jpg)

Laboratory Tests

Living myocardial cells contain enzymes and proteins (e.g., creatine kinase, troponin I and T, myoglobin) associated with specialized cellular functions. When a myocardial cell dies, cellular membranes lose integrity, and intracellular enzymes and proteins slowly leak into the blood stream. These enzymes and proteins can be detected by a blood sample analysis. These values vary depending on the assay used in each laboratory. Given the acuity of a STEMI and the need for urgent intervention, the laboratory tests are usually not available at the time of diagnosis. Thus, good history taking and an ECG are used to initiate therapy in the appropriate situations. The real value of biomarkers such as troponin lies in the diagnosis and prognosis of NSTEMI ([Fig. 3](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/#f0020)).



[Figure 2: Click to Enlarge](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/images/amifig2_large.jpg)

**Summary**

* MI results from myocardial ischemia and cell death, most often because of an intra-arterial thrombus superimposed on an ulcerated or unstable atherosclerotic plaque.
* Despite advances in therapy, MI remains the leading cause of death in the United States.
* MI risk factors include hyperlipidemia, diabetes, hypertension, male gender, and tobacco use.
* Diagnosis is based on the clinical history, ECG, and blood test results, especially creatine phosphokinase (CK), CK-MB fraction, and troponin I and T levels.
* Outcome following an MI is determined by the infarct size and location, and by timely medical intervention.
* Aspirin, nitrates, and beta blockers are critically important early in the course of MI for all patients. For those with STEMI and for those with new left bundle branch block, coronary angiography with angioplasty and stenting should be undertaken within 90 minutes of arrival at facilities with expertise in these procedures. Fibrinolytic therapy should be used in situations in which early angiographic intervention is not possible.
* Postdischarge management requires ongoing pharmacotherapy and lifestyle modification.

References

1. Cotran RS, Kumar V, Robbins SL (eds): Robbins Pathologic Basis of Disease. 5th ed. Philadelphia: WB Saunders, 1994.
2. Rubin E, Farber JL (eds): Essential Pathology. 2nd ed. Philadelphia: JB Lippincott, 1995.
3. Thygesen K, Alpert JS, White HD. Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction: Universal definition of myocardial infarction. Eur Heart J. 2007, 28: 2525-2538.
4. Andreson J, Adams C, Antman E, et al: ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2007, 50: e1.
5. American Heart Association. Cardiovascular disease statistics. Available at <http://www.americanheart.org/presenter.jhtml?identifier=4478> (accessed March 2, 2009).
6. Adult Treatment Panel III. Detection, evaluation, and treatment of high blood cholesterol in adults. Available at <http://www.nhlbi.nih.gov/guidelines/cholesterol> (accessed March 2, 2009).
7. National Heart, Lung, and Blood Institute. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Available at <http://www.nhlbi.nih.gov/guidelines/hypertension> (accessed March 2, 2009).
8. Hung J, Lam JYT, Lacoste L, Letchacovski G. Cigarette smoking acutely increases platelet thrombus formation in patients with coronary artery disease taking aspirin. Circulation. 1995, 92: 2432-2436.
9. Antman E, Hand M, Armstrong P, et al: 2007 focused update of the ACC/AHA 2004 Guidelines for the Management of Patients with ST-Elevation Myocardial Infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2008, 51: 210-2147.
10. Second Chinese Cardiac Study Collaborative Group. Clopidogrel and Metoprolol in Myocardial Infarction Trial. Presented at the ACC annual conference on March 9, 2005, Orlando, Fla. Information available at <http://www.commit-ccs2.org> (accessed March 2, 2009).
11. Scirica BM, Sabatine MS, Morrow DA, et al: The Role of Clopidogrel in Early and Sustained Arterial Patency After Fibrinolysis for ST-Segment Elevation Myocardial Infarction The ECG CLARITY-TIMI 28 Study. J Am Coll Cardiol. 2006, 48: 37-42.
12. Cannon CP, Braunwald E, McCabe CH, et al: for the Pravastatin or Atorvastatin Evaluation and Infection Therapy-Thrombolysis in Myocardial Infarction 22 Investigators: Intensive versus moderate lipid lowering with statins after acute coronary syndromes. N Engl J Med. 2004, 350: 1495-1504.
13. Pitt B, Williams G, Remme W, et al: The EPHESUS Trial: Eplerenone in Patients with Heart Failure Due to Systolic Dysfunction Complicating Acute Myocardial Infarction. Cardio Drugs and Therapy. 2001, 15: 79-87.
14. Grines CL, Browne KF, Marco J, et al: A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction. The Primary Angioplasty in Myocardial Infarction Study Group. N Engl J Med. 1993, 328: 673-679.
15. Grines CL, Cox DA, Stone GW, et al: Coronary angioplasty with or without stent implantation for acute myocardial infarction. Stent Primary Angioplasty in Myocardial Infarction Study Group. N Engl J Med. 1999, 341: 1949-1956.
16. Moss AJ, Zareba W, Hall WJ, et al: for the Multicenter Automatic Defibrillator Implantation Trial II Investigators. Prophylactic Implantation of a Defibrillator in Patients with Myocardial Infarction and Reduced Ejection Fraction. N Engl J Med. 2002, 346: 877-883.
17. Epstein AE, DiMarco JP, Ellenbogen KA, et al: ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: Executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2008, 51: 2085-2105.
18. Wilhelmsson C, Vedin JA, Elmfeldt D, et al: Smoking and myocardial infarction. Lancet. 1975, 1: 415.
19. The MIAMI Trial Research Group. Metoprolol in Acute Myocardial Infarction (MIAMI): A randomized placebo-controlled international trial. Eur Heart J. 1985, 6: 199-226.
20. ISIS-1 Collaborative Group. Randomized trial of intravenous atenolol among 16027 cases of suspected acute myocardial infarction. Lancet. 1986, 2: 57-66.
21. Dargie HJ. Effect of carvedilol on outcome after myocardial infarction in patients with left ventricular dysfunction: the CAPRICORN randomised trial. Lancet. 2001, 357: 1385.
22. Pfeffer MA, Braunwald E, Moye LA, et al: Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction. Results of the Survival and Ventricular Enlargement trial. The SAVE Investigators. N Engl J Med. 1992, 327: 669-677.