Complications of Myocardial Infarction

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Case Presentation

A 61 year old woman with a history of hypertension, tobacco abuse, and Wolf-Parkinson-White syndrome with prior ablation 15 years ago, presented to the emergency department via ambulance with 4 days of indigestion and generalized chest discomfort, with more severe pain lasting several hours. EMS noted her to be tachycardic with HR 102 bpm, and hypotensive with BP 83/61 mmHg. The following 12-lead ECG was obtained (Fig. 13.1).

The initial physical exam was notable for an anxious and diaphoretic patient with elevated jugular venous pressure, crackles at the lung bases, and a left ventricular S3 with a soft systolic murmur adjacent to the lower left sternal boarder. Initial blood tests showed WBC 12.5, hemoglobin 15.5 mg/dL, serum creatinine 1.2 mg/dl, and cardiac troponin I 6.06 ug/L. She was brought emergently to the cardiac catheterization laboratory where she was found to have 100 % occlusion of a dominant, mid-left circumflex artery. She underwent successful PCI with placement of two overlapping drug-eluting stents, and restoration of TIMI 3 flow to the infarct related artery (Table 13.1). At the conclusion of the case, the patient became agitated and pulse oximetry showed 88 % saturation despite administering 100 % oxygen by non-rebreather facemask. Blood-pressure was 78/58 mmHg at this time, and the patient required endotracheal intubation and the initiation of vasopressor medications to stabilize her blood pressure.

Question What is the differential diagnosis for the patient's hemodynamic and respiratory decompensation, and what should be done next to confirm the diagnosis?

Answer The differential diagnosis must include mechanical complications of AMI including ventricular septal rupture (VSR) (Video 13.1), papillary muscle rupture leading to acute mitral regurgitation (MR) (Video 13.2), or free-wall rupture leading to pseudoaneurysm or cardiac tamponade (Video 13.3). Other etiologies to be considered include RV infarction, acute blood loss, iatrogenic hypotension secondary to medication, and cath lab complications such as aortic dissection or coronary perforation. **The most**

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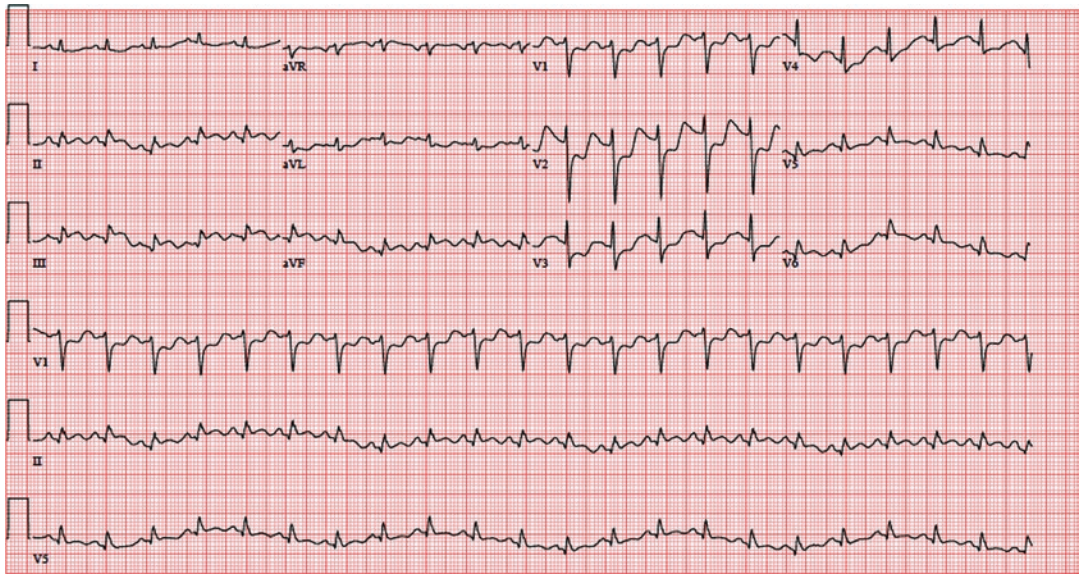


Fig. 13.1 12-lead ECG

Table 13.1 TIMI flow definitions

TIMI Grade 0 (no perfusion)	There is no antegrade flow beyond the point of occlusion
TIMI Grade 1 (penetration without perfusion)	The contrast material passes beyond the area of obstruction but "hangs up" and fails to opacify the entire coronary bed distal to the obstruction for the duration of the cineangiographic filming sequence
TIMI Grade 2 (partial perfusion)	The contrast material passes across the obstruction and opacifies the coronary bed distal to the obstruction. However, the rate of entry of contrast material into the vessel distal to the obstruction or its rate of clearance from the distal bed (or both) are perceptibly slower than its entry into or clearance from comparable areas not perfused by the previously occluded vessel – e.g., the opposite coronary artery or the coronary bed proximal to the obstruction
TIMI Grade 3 (complete perfusion)	Antegrade flow into the bed distal to the obstruction occurs as promptly as antegrade flow into the bed proximal to the obstruction, and clearance of contrast material from the involved bed as rapid as clearance from an uninvolved bed in the same vessel or the opposite artery

Data from Chesebro et al. [16]

rapid way to confirm the diagnosis is with immediate, trans-thoracic echocardiography.

The patient has demonstrated hemodynamic instability despite patency of the infarct related artery in the setting of a delayed presentation of acute myocardial infarction (AMI). Based on her clinical and hemodynamic findings she is now Killip Class IV (Table 13.2). Understanding the etiology of her hemodynamic instability is now crucial to guide further management. While this may represent LV failure in the setting of a large MI, it is unusual that an isolated infarction in the distribution described will account for these findings. Mechanical complications of MI, as listed above, must be considered and rapidly identified or excluded. In this specific case, the bedside echo demonstrated a severe, eccentric jet of mitral regurgitation. On a subsequent trans-esophageal echocardiogram (TEE), the diagnosis was confirmed to be due to rupture and flail of the papillary muscle with prolapse into the left atrium during systole. An image from the trans-esophageal echocardiogram obtained in the standard 4-chamber view is provided in Fig. 13.2. The patient was taken emergently to the operating room where a bioprosthetic mitral valve replacement was performed. The patient was successfully

discharged to a sub-acute nursing facility on hospital day 15, and made a full recovery.

Standard Approach to Management

Epidemiology

Cardiogenic shock in the peri-infarct setting is defined as inadequate tissue perfusion and sustained hypotension despite adequate intravascular volume. Cardiogenic shock complicates between 5 and 8 % of cases of AMI in contemporary databases and trials. Mortality for AMI in general is <5 % in contemporary series, but can exceed 50 % in patients with shock [1]. The most common cause of shock after AMI is left ventricular (LV) failure which represents 80 % of cases, but mechanical complications of AMI must be considered including isolated right ventricular (RV) infarct, ventricular septal rupture (VSR), free-wall rupture with ensuing cardiac tamponade, and papillary muscle rupture leading to acute mitral regurgitation (MR). Data from the SHOCK trial and the subsequent SHOCK registry documented the most common etiologies of cardiogenic shock among 1,422 patients after AMI (Fig. 13.3) [2].

Table 13.2 Killip Class definitions

	Definition	30-day mortality (in 1967) (%)
Killip Class I	No clinical signs of heart failure	<6
Killip Class II	Signs of heart failure including rhales or crackles in the lower lung fields, an S3, or elevated jugular venous pressure	<17
Killip Class III	Frank, acute pulmonary edema.	38
Killip Class IV	Cardiogenic shock or hypotension (measured as systolic blood pressure < 90 mmHg) and evidence of peripheral vasoconstriction (oliguria, cyanosis, or sweating)	81

Data from Killip and Kimball [17]

Diagnosis

The first step in managing patients with cardiogenic shock after AMI is to identify the cause. Specifically, one must attempt to differentiate between mechanical complications that require immediate operative treatment as compared to LV failure alone. A rapid but thorough physical examination is important in any patient with AMI and shock. Cardiac auscultation may reveal the

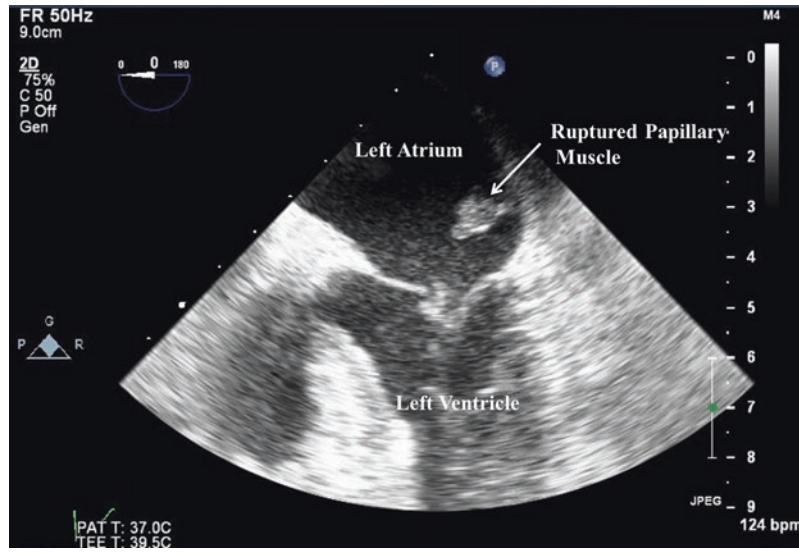


Fig. 13.2 Transesophageal echocardiogram

characteristic, harsh, holosystolic murmur of a VSR or a softer, apical murmur consistent with acute mitral regurgitation. Cardiac tamponade may be suspected based on jugular venous distention (JVD) and a pulsus paradoxus, or Beck's triad of hypotension, distended neck veins, and muffled heart sounds. Patients with RV infarction may display a pulsus paradoxus along with the Kussmaul sign (increase in JVD with inspiration) which is usually absent in patients with cardiac tamponade. Physical exam may be challenging due to the degree of hypotension, the need for mechanical ventilation, and the physical environment. The murmur of acute MR may be soft and difficult to appreciate due to rapid elevation in left atrial pressures. Similarly a large VSR in the setting of hypotension may also be under appreciated.

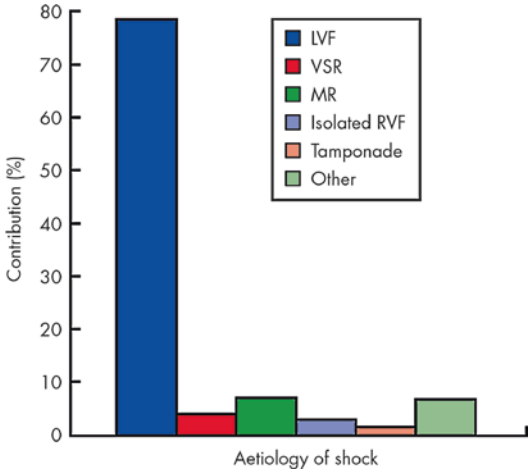
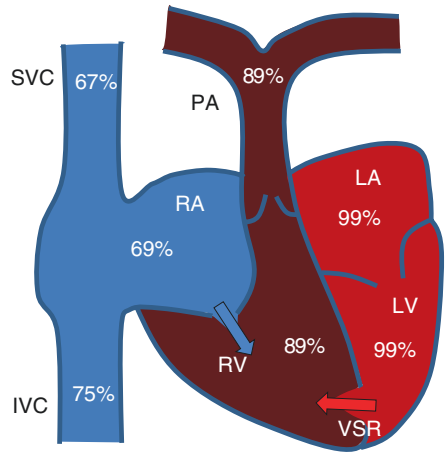


Fig. 13.3 Etiology of suspected cardiogenic shock in the combined SHOCK trial registry and trial (total n = 1422, only first 232 trial patients are included). "Other" includes shock caused by prior severe valvular disease, dilated cardiomyopathy, excess beta-blockade/calcium channel blockade, hemorrhage, and procedural complications. Aortic dissection, pulmonary embolism, and dynamic subaortic outflow obstruction should also be considered. *LVF* left ventricular failure, *MR* mitral regurgitation, *RVF* right ventricular failure, *VSR* ventricular septal rupture (Reproduced with permission from Menon, Heart, 2002 [2] with permission from BMJ Publishing Group Ltd)

Practically speaking, most mechanical complications of AMI can be definitively diagnosed by prompt, bedside, trans-thoracic echocardiography (TTE). In patients who are brought immediately to the cardiac catheterization lab and/or in whom echocardiography is not immediately available or is inconclusive, the diagnosis can be made by LV angiography and/or right heart catheterization. Using a balloon-tipped catheter, VSR can usually be confirmed by demonstrating a "step-up" in the oxygen saturation of blood sampled from the right ventricle or pulmonary artery as compared to the right atrium, reflecting shunting of oxygenated blood from the LV to the RV through the ruptured septum, and subsequent mixing with relatively deoxygenated blood returning to the right side of the heart (Fig. 13.4). Comparatively, patients with severe mitral regurgitation will likely have tall v-waves in the pulmonary capillary wedge tracing without a significant step-up in oxygen saturations. Importantly, not all mitral regurgitation in the setting of AMI is due to papillary muscle rupture, and it is important to distinguish MR due to LV



$$\text{Qp/Qs} = \frac{\%\ \text{Sat Artery} - \%\ \text{Sat RA}}{\%\ \text{Sat Artery} - \%\ \text{Sat PA}}$$

$$\text{Qp/Qs} = \frac{99\% - 69\%}{99\% - 89\%}$$

$$\text{Qp/Qs} = \frac{30\%}{10\%}$$

$$\text{Qp/Qs} = 3$$

Fig. 13.4 Oximetry run and shunt fraction calculation in a patient with an apical VSR showing a "step up" in the RV where there is mixing of venous blood with oxygenated blood from the left ventricle that is crossing the apical septal defect. Note: RA sat can be estimated by (3× SVC + IVC)/4. Note: A peripheral arterial sat is usually used as a surrogate for LV saturation

dilation or posterior mitral leaflet restriction in the setting of an akinetic inferior wall (Video 13.4), neither of which would typically be managed with emergent surgery. Thus, there should be a low threshold to proceed to trans-esophageal echocardiography (TEE) when the mechanism for the MR is ambiguous on surface echocardiogram.

Management of LV Failure

When LV failure is felt to be responsible for cardiogenic shock, the next step in management is to ensure complete revascularization, as was clearly demonstrated in the SHOCK trial [1]. Patients with ongoing hemodynamic embarrassment despite revascularization may require urgent mechanical support, most commonly in the form of an intra-aortic balloon pump (IABP). Inotropic and vasopressor agents are sometimes used in emergent situations to stabilize hemodynamics, but have not been shown to improve hospital survival. Other mechanical support devices that have been used include the TandemHeart, Impella, and veno-arterial extra-corporeal membrane oxygenation (ECMO). In this emergent setting, mechanical support is usually utilized as a bridge to decision. In those who stabilize and recover, support is subsequently withdrawn. In others who continue to be unstable, destination support with an LVAD or consideration of the LVAD as a bridge to transplantation may be considered. Finally in the group of patients where care may be deemed futile, support is not performed or may be subsequently withdrawn.

Management of Mechanical Complications

Mechanical complications of AMI have become less common in the era of early reperfusion, but mortality for these patients remains high (Fig. 13.5) [3]. Medical management of papillary

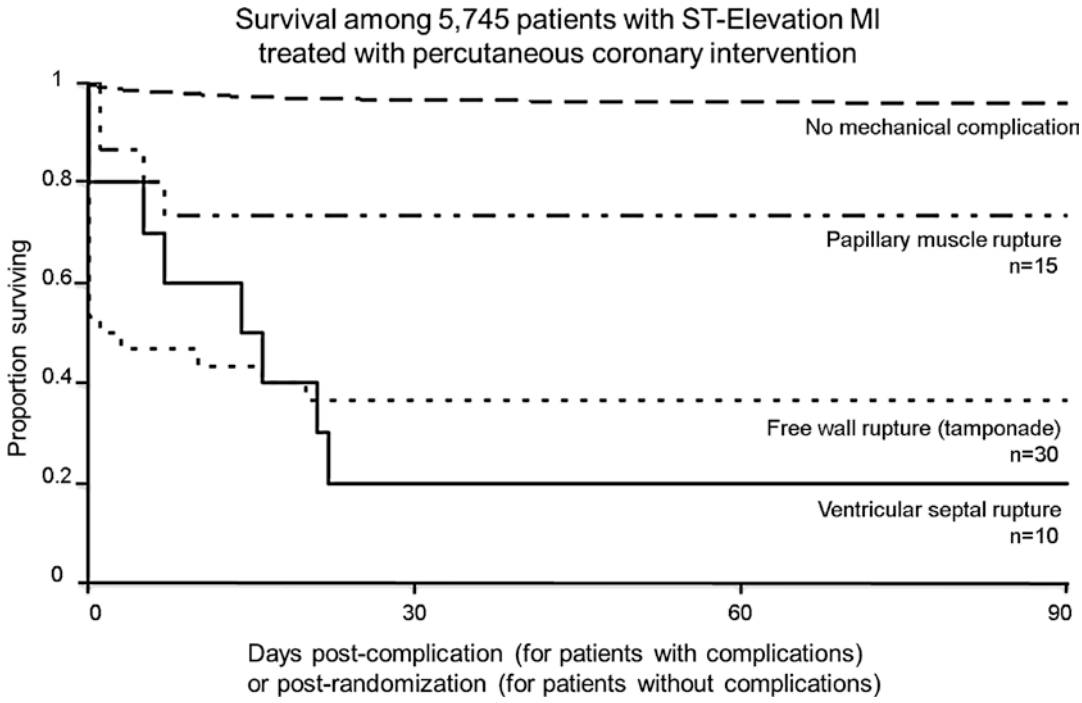


Fig. 13.5 Survival among 5,745 patients undergoing primary percutaneous coronary intervention (PCI) after ST-elevation myocardial infarction in the APEX-AMI trial. A total of 52 patients (0.91 %) had a mechanical complication of which 15 were papillary muscle rupture, 30 were free wall rupture with tamponade, and 10 were ventricular septal rupture (Adapted from French [3] with permission from Elsevier Limited)

muscle rupture and VSR is limited, but involves afterload reduction with intravenous sodium nitroprusside. IABP placement is considered routine care in hemodynamically unstable patients. The only definitive management strategy is surgical repair, although a variety of mechanical support devices have been used to temporarily stabilize hemodynamically unstable patients who were not initially felt to be candidates for surgery. When the diagnosis of a mechanical complication can be made prior to primary percutaneous coronary intervention (PCI), immediate collaboration between the interventional cardiologist and cardiac surgeon is needed to determine the optimal approach for prompt restoration of coronary flow, while also considering the competing need to limit the administration of dual-antiplatelet therapy in patients undergoing emergent surgery. In some cases, balloon angioplasty without stent placement, followed by combined coronary artery bypass graft placement with immediate mechanical repair, may be the preferred option.

Evidence Contour

Timing of Surgery in Ventricular Septal Rupture

The optimal timing for surgery in unstable patients with ventricular septal rupture is controversial given the exceedingly high mortality of patients who are operated on in the immediate, post-infarct period [4]. Complexity of the operation, relative surgical inexperience, acute right ventricular dysfunction from infarction, ischemia or volume overload, patient comorbidities, or exposure to potent antiplatelet treatment prior to surgery may all contribute towards this finding. The overall mortality of patients in the Society of Thoracic Surgeons National Database for patients with VSR was 42.9%, but was significantly higher (>60%) in patients who underwent surgery in the first 24 h, as compared to 18.4% in patients in whom the repair was delayed until after 7 days [5]. Unfortunately, unstable patients are very unlikely to survive long enough for surgery to be done electively, so this data is mostly reflective of survival bias. Many have hypothesized however that the particularly poor outcomes with immediate surgery may be in part due to the weak, friable tissue of an acute infarct which holds sutures poorly. Thus, some experienced centers have attempted a strategy of full mechanical support with a planned, delayed surgery in select patients, although the success of this strategy is currently limited to case reports [6, 7]. In general, patients who are considered acceptable candidates for immediate repair based on factors including comorbidities, willingness of the patient to pursue an aggressive strategy, and comfort of the surgeon/medical center in managing the patient, should undergo surgical repair without delay.

Percutaneous Closure of VSR

Given the exciting evolution of structural cardiac interventions over the past decade, many have considered options for percutaneous closure of acute VSR, either as a definitive strategy, or as a bridge to surgery after initial stabilization. This strategy has been attempted for both primary closure and in the treatment of residual shunts after surgical repair. Unfortunately, when attempted in the immediate, post-infarct period, or in patients with cardiogenic shock, mortality remains high, similar to surgical series [8]. The procedure can be done safely however, and with technical success in the overwhelming majority of cases. Thus, procedures done in the acute setting should likely be limited to patients who are not initially surgical candidates, and undertaken with the goal of reducing shunt fraction and improving hemodynamic stability as a bridge to a more definitive surgical repair. For patients in the sub-acute to chronic period whose comorbidities continue to prevent definitive repair, percutaneous closure is an especially attractive option, and initial clinical series have shown excellent success and low 30-day mortality in these populations [9–13].

Intra-aortic Balloon Pump

IABP use in patients with cardiogenic shock after AMI has come under scrutiny given the results of the IABP-SHOCK II trial which failed to show a difference in 30-day mortality among patients that were randomized to IABP placement vs. open label controls [14]. Although well performed, IABP placement in this study was performed only after revascularization and it included large subsets of patients with NSTEMI ACS and post cardiac arrest. Criticisms of this study have also included a high cross-over rate with 10% of patients undergoing IABP placement despite randomization to the control group, and a trend toward higher utilization of LV assist devices in the control group. Most clinicians feel that IAPB utilization continues to be a useful adjunct in the hemodynamically unstable patient with AMI. Based on the IABP –SHOCK II trial however, its utilization has been downgraded to a Class IIa LOE B recommendation from the 2013 ACCF/AHA guidelines on management of ST-elevation MI [15]. Importantly, the IABP-Shock II trial was not powered to look at the utility of IABP placement in the setting of VSR, where it is considered routine care.

References

1. Hochman JS, Sleeper LA, White HD, Dzavik V, Wong SC, Menon V, et al. One-year survival following early revascularization for cardiogenic shock. JAMA. 2001; 285(2):190–2.
2. Menon V, Hochman JS. Management of cardiogenic shock complicating acute myocardial infarction. Heart. 2002;88(5):531–7.
3. French JK, Hellkamp AS, Armstrong PW, Cohen E, Kleiman NS, O'Connor CM, et al. Mechanical complications after percutaneous coronary intervention in ST-elevation myocardial infarction (from APEX-AMI). Am J Cardiol. 2010;105(1):59–63.
4. Jones BM, Kapadia SR, Smedira NG, Robich M, Tuzcu EM, Menon V, et al. Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. Eur Heart J. 2014;35(31):2060–8.
5. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database. Ann Thorac Surg. 2012;94(2):436–43; discussion 43–4.
6. Tsai MT, Wu HY, Chan SH, Luo CY. Extracorporeal membrane oxygenation as a bridge to definite surgery in recurrent postinfarction ventricular septal defect. ASAIO J. 2012;58(1):88–9.
7. Neragi-Miandoab S, Michler RE, Goldstein D, D'Alessandro D. Extracorporeal membrane oxygenation as a temporizing approach in a patient with shock, myocardial infarct, and a large ventricle septal defect; successful repair after six days. J Card Surg. 2013;28(2):193–5.
8. Thiele H, Kaulfersch C, Daehnert I, Schoenauer M, Eitel I, Borger M, et al. Immediate primary transcatheter closure of postinfarction ventricular septal defects. Eur Heart J. 2009;30(1):81–8.
9. Assenza GE, McElhinney DB, Valente AM, Pearson DD, Volpe M, Martucci G, et al. Transcatheter closure of post-myocardial infarction ventricular septal rupture. Circ Cardiovasc Interv. 2013;6(1):59–67.
10. Holzer R, Balzer D, Amin Z, Ruiz CE, Feinstein J, Bass J, et al. Transcatheter closure of postinfarction ventricular septal defects using the new Amplatzer muscular VSD occluder: Results of a U.S. Registry. Catheter Cardiovasc Interv. 2004;61(2):196–201.
11. Maltais S, Ibrahim R, Basmadjian AJ, Carrier M, Bouchard D, Cartier R, et al. Postinfarction ventricular septal defects: towards a new treatment algorithm? Ann Thorac Surg. 2009;87(3):687–92.
12. Bialkowski J, Szkutnik M, Zembala M. Ventricular septal defect closure – importance of cardiac surgery and transcatheter intervention. Kardiol Pol. 2007; 65(8):1022–4.
13. Demkow M, Ruzyllo W, Kepka C, Chmielak Z, Konka M, Dzielinska Z, et al. Primary transcatheter closure of postinfarction ventricular septal defects with the Amplatzer septal occluder- immediate results and up-to 5 years follow-up. EuroIntervention. 2005;1(1):43–7.
14. Thiele H, Zeymer U, Neumann FJ, Ferenc M, Olbrich HG, Hausleiter J, et al. Intraaortic balloon support for myocardial infarction with cardiogenic shock. N Engl J Med. 2012;367(14):1287–96.
15. O'Gara PT, Kushner FG, Ascheim DD, Casey Jr DE, Chung MK, de Lemos JA, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2013;61(4):e78–140.
16. Chesebro JH, et al. Thrombolysis in Myocardial Infarction (TIMI) Trial, Phase I: a comparison between intravenous tissue plasminogen activator and intravenous streptokinase. Clinical findings through hospital discharge. Circulation. 1987;76(1):142–54.
17. Killip T, Kimball JT. Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. Am J Cardiol. 1967;20(4): 457–64.