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Case report

Treatment of cardiogenic shock in PCI center without the possibility of mechanical circulatory support - case report and review of the contemporary data

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Abstract

Introduction: Cardiogenic shock (CS) is one of the most severe conditions met in the cardiac intensive care unit, with a short-term mortality rate of 30–40%. It represents a syndrome of hypoperfusion and organ dysfunction due to a primarily cardiac cause, most commonly as a consequence of acute myocardial infarction (AMI).

Aim: To present a case of an advanced age female patient treated for CS following acute myocardial infarction in a PCI center without the availability of mechanical circulatory support.

Case report: An 85-year-old female patient was admitted to the Invasive cardiology department of Health Center Zaječar, presenting with an anterior wall STEMI, 9 hours after the onset of chest pain in Forrester class III CS. Primary PCI of the LAD was performed, after which she was treated with inotropic support of norepinephrine in a maximal dose of 0.35 mcg/kg/min, along with cautious infusion of crystalloid fluids and other heart failure therapy. Upon the administered therapy, the patient hemodynamically stabilized and inotropic stimulation ceased despite ongoing severe left ventricular systolic dysfunction with an EF of 15–20% and severe mitral regurgitation. The further clinical course was complicated by aspiration pneumonia, diarrheal syndrome, and dehydration with hypernatremia, eventually resulting in a fatal outcome after 21 days of treatment.

Conclusion: Emergency primary PCI procedure in the nearest PCI center, along with the administration of inotropic agents and other supportive measures in the cardiac intensive care unit, can lead to hemodynamic stabilization even in critically ill patients of advanced age with CS.

Key-words acute myocardial infarction, STEMI, cardiogenic shock, inotropic agents, norepinephrine

Introduction

Cardiogenic shock (CS) represents a syndrome of hypoperfusion and organ dysfunction due to a primarily cardiac cause, with a decreased cardiac output[1]. The clinical definition implies a systolic pressure below 90 mmHg lasting longer than 30 minutes, or maintained above these values by the administration of inotropes/vasopressors; and signs of tissue hypoperfusion: oliguria (diuresis less than 30 ml/h), altered mental status, skin pallor and coldness. Laboratory markers of tissue hypoperfusion are elevated levels of serum lactate above 2 mmol/L; alanine transaminaase (ALT) above 200 or more than 3 times the upper reference limit; serum creatinine more than 2 times the upper reference limit; arterial blood pH less than 7.2, without other causes of acidosis. CS is one of the most common causes of admission to the cardiac intensive care unit (coronary care unit), with a short-term mortality rate of 30–40% and a one-year mortality rate of around or over 50%[2].

Here we present a case of an elderly female patient with CS caused by acute myocardial infarction (AMI), treated with primary percutaneous coronary intervention (pPCI) and inotropic agents in a secondary-level healthcare facility, without the possibility of mechanical circulatory support.

Case report

An 85-year-old female patient was admitted to the Department of invasive cardiology of the Zaječar Health Center nine hours after the onset of continuous chest pain. Electrocardiography revealed ST-segment elevation in precordial leads (Figure 1). Upon admission, the patient was hemodynamically unstable with BP 70/40 mmHg, HR 110/min, pale, cold skin, and without signs of pulmonary congestion, corresponding to CS (Killip IV, Forrester III class). Emergency coronary angiography was performed, revealing lesions in the ostial segment of the Cx and a longer atherosclerotically narrowed segment of OM2, as well as an ostially occluded LAD (Figures 2 and 3). Standard coronary guidewires (BMW, Asahi SIon Blue) could not cross the lesion, so a Pilot 50 coronary wire was used, followed by predilatations with Sapphire 1x10 and Sprinter Legend 2x20 balloons. Two lesions were identified—one in the ostial part of LAD and the

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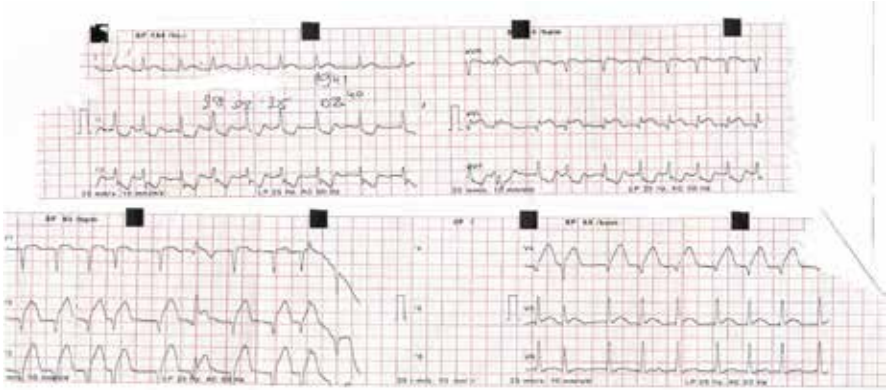
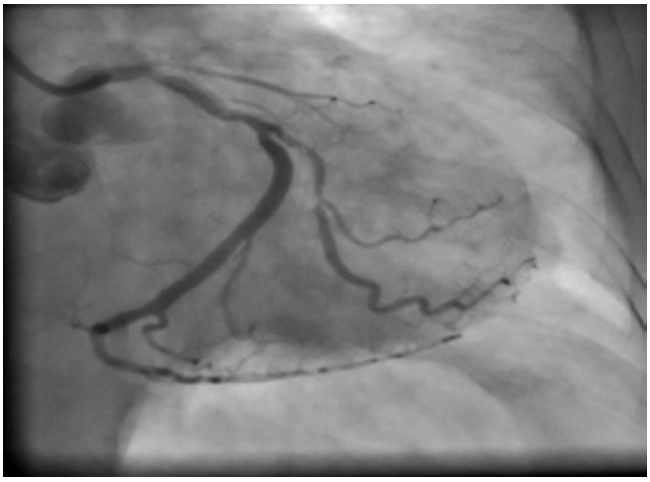


Figure 1.



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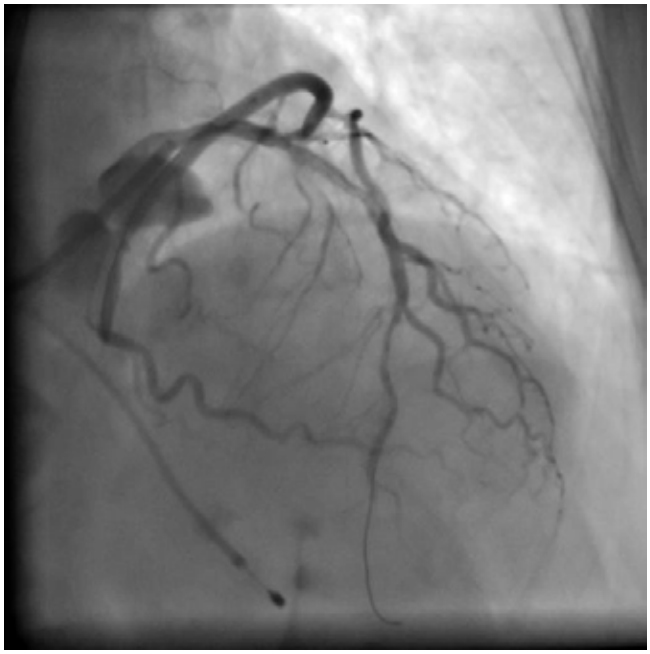

Figure 4.

other in the medial segment of LAD. A Synsiro PRO 2.75x26 stent was implanted in the medial stenosis, after which a *no-reflow* phenomenon occurred. Through a microcatheter, 500 mcg of Verapamil was applied into the distal segment of the artery, resulting in a TIMI 2–3 flow through the LAD with a residual LM–LAD stenosis of 50–60% (Figure 4). A temporary pacemaker electrode was placed, with a pacing rate of 100/min.

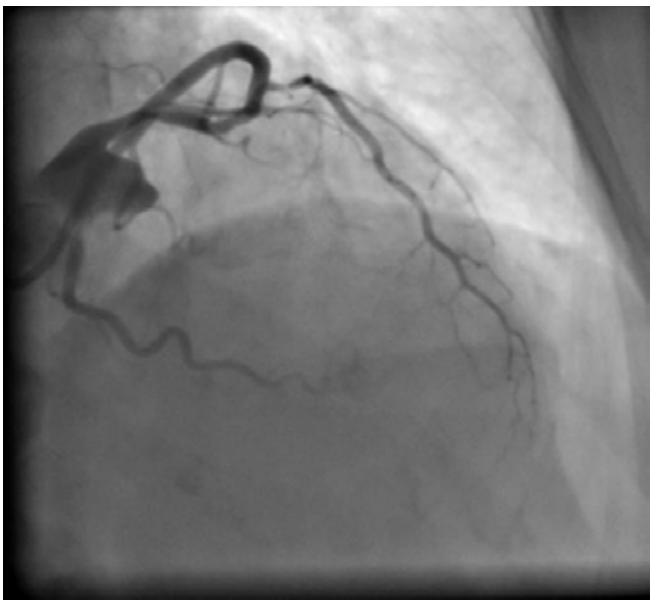

Figure 3.

During the procedure, the patient remained hemodynamically unstable, on oxygen therapy, with a brief respiratory arrest followed by spontaneous breathing. After the procedure, she was transferred to the Coronary Care Unit, and inotropic support with norepinephrine was initiated through the right femoral vein at an initial dose of 0.2 mcg/kg/min, titrated according to therapeutic response over 48 hours up to a maximum of 0.35 mcg/kg/min. From the second hospital day, as there were no signs of pulmonary congestion, a "fluid challenge" with 1 L of crystalloids per day was administered, without the development of dyspnea or pulmonary congestion. Hemodynamic improvement was achieved, and after 48 hours inotropes and oxygen therapy were discontinued, and the temporary pacemaker electrode was removed, with a daily diuresis of 1000 ml.

The subsequent hospital course was complicated by right-sided pneumonia, treated empirically with cefepime and levofloxacin, followed by diarrheal syndrome with hypertonic dehydration and severe hypernatremia (up to 164 mmol/L). Throughout the hospital course, the patient remained hemodynamically stable with "dry" lungs. Echocardiography performed on the fifth hospital day indicated severely impaired global left ventricular function with an estimated left ventricular ejection fraction (LVEF) of 15–20% and akinesia of the anterior sep-

tum, apical and medial segment of the inferior septum, as well as the entire anterior and lateral wall. The right heart chambers were dilated, with TR 3–4+ and estimated pulmonary artery systolic pressure of about 50 mmHg. After 21 days of treatment, despite all therapeutic measures applied and the patient's hemodynamic stability, the patient died.

Discussion

CS may arise from multiple etiologies, among which AMI has traditionally been the most frequent cause. Less common etiologies include various forms of *de novo* cardiac dysfunction such as right ventricular failure, Takotsubo syndrome, peripartum cardiomyopathy, fulminant myocarditis, valvular heart disease, or decompensation of chronic heart failure[3]. Although historically about 80% of cardiogenic shock cases were attributed to acute myocardial infarction[4], more recent registry data suggest that the epidemiology has shifted in recent decades, with decompensated heart failure now representing an increasingly prevalent cause[5–7].Given that the case report involves AMI-related CS, the following discussion focuses on this clinical subtype.

Before the advent of pharmacologic and mechanical reperfusion strategies, CS frequently complicated AMI. In the landmark study by Killip *et al*[8], published in 1967, 250 patients had a definite diagnosis of myocardial infarction in a single hospital in a two-year period, with CS occurring in 19% of cases. In the early 2000s, the incidence of CS was reported in approximately 8% of STEMI and 5% of NSTEMI patients[9].

Despite major advances in myocardial revascularization and the development of short-term mechanical circulatory support over the past two decades, randomized clinical trials have largely failed to identify additional therapeutic interventions that significantly reduce mortality from AMI-related cardiogenic shock beyond revascularization itself[10]. Notably, the SHOCK registry found no statistically significant difference in 30-day mortality between patients who underwent urgent myocardial revascularization (PCI or surgical) and those initially managed with medical stabilization including systemic fibrinolysis. However, at 6- and 12-month follow-up, patients who underwent early mechanical revascularization demonstrated substantially lower mortality (50.3% and 53.3% vs. 63.1% and 66.4%, respectively)[11]. The registry also described the temporal profile of shock onset. On average, CS developed 6.2 hours after the onset of chest pain: 1.7 hours when the culprit lesion was in the left main coronary artery, 3.5 hours in the right coronary artery, 3.9 hours in the circumflex artery, 11 hours in the LAD, and 10.9 hours in a saphenous vein graft. Early shock (within 24 hours of symptom onset) was associated with chest pain as the presenting symptom, ST-segment elevation in two or more ECG leads, smoking, multivessel disease, and left main artery disease. Late shock (more than 24 hours after symptom onset) was more often linked to recurrent ischemia, development of Q waves in two or more leads, and anterior infarction. Mortality was higher in early compared with late shock (62.6% vs. 53.6%)[12].

Yang *et al*[13] proposed the EARLY SHOCK scoring system, identifying eight prehospital variables independently predicting high risk of developing CS: heart rate, systolic blood pressure at presentation, diabetes, dialysis status, age, prehospital cardiac arrest, and infarct localization. The strongest predictors were systolic blood pressure at presentation below 90 mmHg (OR 17.76) and prehospital cardiac arrest (OR 18.32), while anterior infarction carried a moderately increased risk compared with non-anterior localization (OR 1.58).

Our patient presented nine hours after symptom onset with an ostial LAD occlusion, and the prolonged duration of ischemia involving a large myocardial territory resulting in the development of CS upon presentation. The time interval between symptom onset and first medical contact (FMC) remains prolonged in various parts of the world. Data from the GRACE registry indicate that this interval averages around two hours in the United States and Canada[14]. In a cohort study from Ontario, Canada, as many as 66% of patients had a symptom-onset-to-FMC time exceeding 120 minutes[15]. Unfortunately, this trend has persisted over the past two decades despite public awareness campaigns and other interventions. Delayed presentation is more common among women and elderly patients[16–18]. Local data from Eastern Serbia show a similar pattern: in 2019, the average time to FMC was 222 minutes, and in 2020, due to the COVID-19 pandemic and fear of seeking medical care, increased to 302 minutes[19].

Our patient's clinical presentation corresponded to Forrester class III cardiogenic shock ("dry and cold")[20]. She showed no clinical signs of pulmonary congestion and was therefore able to undergo primary PCI in the supine position with supplemental oxygen therapy, without the need for mechanical ventilation. In their seminal study, Forrester *et al* performed right heart catheterization in patients with AMI-related-CSI and, using hemodynamic parameters (cardiac index and pulmonary capillary wedge pressure), defined four clinical subsets. The first two classes were characterized by the absence of hypoperfusion (cardiac index > 2.2 L/min/m²), whereas classes III and IV demonstrated a reduced cardiac index, with normal (class III) or elevated (class IV) pulmonary wedge pressure. Mortality increased progressively with higher class—from 2.2% in class I to 55.5% in class IV. Class III, into which our patient was stratified, carries an estimated mortality rate of 22.4%. As right heart catheterization was not feasible in our facility, classification was based on clinical parameters. Notably, the accuracy of clinical assessment in predicting hemodynamic abnormalities was found to be satisfactory—approximately 83%—in a subsequent analysis by Forrester *et al*.[21].

An important question arises regarding the appropriateness of fluid challenge in patients with Forrester class III CS. Most clinicians in everyday practice tend to avoid fluid administration due to the fear of inducing pulmonary edema, even when the pulmonary capillary wedge pressure (PCWP) is not elevated. In the present case, after initial stabilization, the patient received 1LL of crystalloid solutions daily without the development of pulmonary congestion. Early studies involving right heart catheterization demonstrated that intravascular volume

expansion may improve cardiac index. It has been shown that a 50% increase in PCWP can lead to a 19% increase in cardiac index, without affecting systolic arterial pressure. Thus, an improvement in cardiac index through crystalloid infusion can be achieved while maintaining PCWP below the pulmonary congestion treshold[22].

Following hospital admission and the diagnosis of anterior wall STEMI in our patient, coronary angiography and primary PCI were performed. Due to hemodynamic instability, only the left coronary system was cannulated. In addition to the culprit lesion involving the left main and LAD, a significant angiographic lesion was observed in the OM2 branch. Only the LAD lesions were treated, in accordance with the findings of the CULPRIT-SHOCK trial, which demonstrated that in patients with multivessel coronary disease, culprit lesion only treatment was associated with lower 30-day mortality (OR 0.84) and a reduced need for renal replacement therapy (OR 0.71) compared to complete revascularization of all angiographically significant lesions[23].

Accurate and comprehensive monitoring of patients with CS is of paramount importance, given the inherently unpredictable clinical course and high mortality associated with this condition. According to expert consensus recommendations, all patients should have an arterial line placed for continuous invasive blood pressure monitoring. A low diastolic pressure measured via this route, in the absence of bradycardia, suggests a loss of arterial tone and warrants initiation or escalation of inotropic therapy. Serial plasma lactate measurement should be performed, provided that adrenaline is not used as an inotropic agent, as lactate serves as a reliable marker of tissue (hypo)perfusion during treatment. In addition, periodic assessment of organ function markers, particularly hepatic and renal parameters, is advised. Regular determination of central venous oxygen saturation (ScvO2) is recommended, while measurement of mixed venous oxygen saturation (SvO2)—obtained from the pulmonary artery and requiring right heart catheterization—should be reserved for cases of refractory shock. Right heart catheterization itself carries only a weak recommendation and is primarily indicated in patients with refractory shock and right ventricular dysfunction. Conversely, measurement of central venous pressure (CVP) is discouraged, because it has limited accuracy and does not reliably reflect cardiac preload. Echocardiography should be performed to identify the underlying cause of shock, assess hemodynamic status, and detect mechanical complications such as cardiac tamponade[24].

Early revascularization remains the cornerstone of therapy in CS; however, pharmacologic modulation of vascular tone and myocardial performance through vasoactive agents represents an equally critical determinant of hemodynamic stabilization. Vasoactive agents are typically classified according to their predominant pharmacodynamic profile into three principal categories: inopressors (which increase cardiac index while inducing vasoconstriction), vasopressors (which elevate arterial blood pressure primarily through vasoconstriction and increased systemic vascular resistance, without direct inotropic stimulation), and inodilators (which augment cardiac output while promoting vasodilation).

The administration of these agents is exceedingly common among critically ill patients: approximately one quarter of all patients in intensive care units receive at least one inotropic agent, while in CS this proportion exceeds 90%. A large-scale study encompassing over 10,000 patients treated in a tertiary cardiac intensive care unit demonstrated a clear trend toward increased norepinephrine use, accompanied by a gradual decline in dopamine administration. In that cohort, inotropic therapy was employed in 24.7% of all admissions.

A widely used parameter for quantifying the cumulative inotropic and vasoactive burden is the Vasoactive–Inotropic Score (VIS**)**, which is defined as the sum of the maximal doses of all vasoactive agents administered, adjusted for their equipotent potencies. For example, 0.2 μg/kg/min of norepinephrine is considered equivalent to 20 μg/kg/min of dopamine. Thus, a patient receiving both agents concurrently at these doses would have a VIS of *0.2 × 100 (norepinephrine) + 20 × 1 (dopamine) = 40*, representing a high level of pharmacologic circulatory support.

In our patient, norepinephrine was administered as the sole vasoactive agent, reaching a maximal infusion rate of 0.35 μg/kg/min, corresponding to a VIS of 35. Elevated VIS values have been consistently shown to represent an independent adverse prognostic indicator, as the need for higher inotropic support reflects greater underlying disease severity.Among patients receiving vasoactive therapy in the aforementioned cohort—after adjustment for disease severity and VIS—norepinephrine use was associated with significantly lower mortality (OR 0.66)[25]. The SOAP II trial, which randomized 1,679 patients with shock of various etiologies to norepinephrine or dopamine, found no difference in overall mortality; however, dopamine administration was associated with a higher incidence of arrhythmic events (24.1% vs. 12.1%). In the subgroup of 280 patients with cardiogenic shock, dopamine use correlated with a significantly higher mortality rate[26]. The OPTIMA CC study compared epinephrine and norepinephrine in patients with cardiogenic shock following acute myocardial infarction. No difference was observed in the doses required to achieve target mean arterial pressure, hemodynamic parameters were comparable between groups. Nevertheless, epinephrine administration resulted in significantly higher lactate levels and a more pronounced metabolic acidosis. Moreover, epinephrine produced a greater increase in heart rate and in the rate–pressure product, a surrogate of myocardial oxygen consumption. The study was terminated prematurely due to the frequent occurrence of refractory shock among epinephrine-treated patients (OR 8.24)[27].

The most plausible pathophysiological explanation for the superior clinical performance of norepinephrine compared to other vasoactive agents lies in its effect on myocardial oxygen consumption. Oxygen consumption is a critically important determinant of myocardial metabolism, particularly under conditions of arterial occlusion and severely compromised coronary perfusion, as

observed in cardiogenic shock. It has been well established that heart rate exerts the greatest influence on myocardial oxygen demand. Estimates suggest that variations in heart rate account for approximately 50–70% of total myocardial oxygen consumption[28,29]. By contrast, increases in myocardial contractility during stress or elevated workload contribute an additional 15–25%[28]. Unlike most other inotropic agents, norepinephrine exerts minimal chronotropic effects, and thus only modestly elevates myocardial oxygen consumption in already ischemic regions. This pharmacodynamic profile promotes a more favorable balance between myocardial oxygen supply and demand in patients with cardiogenic shock.

Based on this physiological rationale, American guidelines recommend norepinephrine as the first-line vasoactive agent in the management of CS accompanied by hypotension (mean arterial pressure <65 mmHg). If hypotension persists despite norepinephrine administration, vasopressin may be considered as an adjunct. In cases where blood pressure stabilizes but low stroke volume persists, the addition of an inodilator (such as milrinone or dobutamine) is advisable. Conversely, in patients who are not initially hypotensive but present with a low stroke volume, an inodilator may be initiated first, with norepinephrine added subsequently if hypotension develops during the course of treatment[30].

At every stage of management—particularly in cases of refractory CS—mechanical circulatory support may be employed. The intra-aortic balloon pump (IABP) remains the most frequently used device owing to its relative simplicity, while extracorporeal membrane oxygenation (ECMO) and microaxial flow pumps (Impella) are utilized less commonly. However, meta-analyses of mechanical support in CS following AMI have failed to demonstrate a significant reduction in 30-day mortality with any modality[31]. The DANGER-SHOCK trial represents the first randomized multicenter study to demonstrate a clear survival benefit of mechanical circulatory support in AMI related CS. Among 355 patients, treatment with the Impella CP microaxial flow pump was associated with improved 6-month survival compared to standard medical therapy (OR 0.74). This benefit, however, was accompanied by a higher incidence of adverse events, including increased requirements for renal replacement therapy (OR 1.98), moderate-to-severe bleeding (OR 2.06), limb ischemia (OR 5.15), and sepsis (OR 2.76). The number needed to harm (NNH) for Impella CP use was 6. In our case, none of the above-mentioned modalities of mechanical circulatory support were available.

Conclusion

Early presentation of the patient to a PCI-capable center and prompt culprit lesion revascularisation combined with the judicious use of inotropic agents and comprehensive supportive care in an intensive care unit setting and under continuous hemodynamic monitoring can significantly improve outcomes in patients with AMI complicated with CS.

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Sažetak

Perkutana koronarna intervencija kod pacijenta sa kardiogenim šokom u centru bez mogućnosti mehaničke cirkulatorne potpore: Prikaz slučaja i pregled postojeće literature

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***Uvod**: Kardiogeni šok je jedno od najtežih stanja u kardiološkoj jedinici intenzivne nege sa kratkoročnim mortalitetom 30-40%. Predstavlja sindrom hipoperfuzije i disfunkcije organa zbog primarno srčanog uzroka, najčešće kao posledica akutnog infarkta miokarda.*

***Cilj rada**: Prikaz bolesnice odmakle životne dobi lečene pod slikom kardiogenog šoka nakon akutnog infarkta miokarda u PCI centru bez mogućnosti primene mehaničke cirkulatorne potpore.*

***Prikaz slučaja**: Bolesnica starosti 85 godina primljena je na Odeljenje invazivne kardiologije ZC Zaječar sa slikom STEMI anteriorne lokaliazacije 9h nakon početka bola u grudima i razvijenim kardiogenim šokom klase III po Foresteru. Urađena je primarna PCI LAD nakon čega je lečena intortopnom potporom noradrenalinom u maksimlanoj dozi od 0.34 mcg/kg/min uz opreznu infuziju kristaloidnih rastvora i ostalu terapiju srčane insuficijencije. Na primenjenu terapiju dolazi do hemodinamske stabilizacije i isključenja inotropne stimulacije i pored teške sistolne disfunkcije leve komore sa EF 15-20% i teške mitralne regurgitacije. Dalji tok bolesti komplikovan je aspiracionom pneumonijom, dijarealnim sindromom i d*

*ehidratacijom sa hipernatrijemijom i naposletku smrtnog ishoda nakon 21 dana lečenja. **Zaključak**: Hitna primarna PCI procedura u najbližem PCI centru uz davanje inotropnih lekova i druge suportivne mere u kardiološkoj jedinici intenzivnog lečenja može dovesti do hemodinamske stabilizacije i veoma teških bolenika odmakle životne dobi sa kardiogenim šokom.*

***Ključne reči**: akutni infarkt miokarda, STEMI, kardiogeni šok, inotropni lekovi, noradrenalin*