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P R O F E S S I O N A L A R T I C L E S

Current Therapy of the Right Ventricle Myocardial Infarction

Vjekoslav Orozović, Branko Gligić, Momčilo Krgović, Saša Rafajlovski, Milić Marković, Radenko Spasić, Nenad Ratković, Radoslav Romanović

Military Medical Academy, Clinic for Emergency Medicine**, Belgrade**

Background. *Acute myocardial infarction of the right venricle (AMI-RV) is a separate subgroup within the scope of inferoposterior infarction of the left ventricle. It still represents the population of patients at high risk due to numerous, often hardly predictable complications and high mortality rate.* ***Methods****. In fifteen-year period (1987–2001) 3 765 patients with the acute myocardial infarction (AMI) of different localizations of both sexes – 2 283 males and 1 482 females of the average age 61.4 ± 4.6 years were treated in our institution. Anterior myocardial infarction was diagnosed in 2 146 (56.9%) patients, inferior in 1 619 (43.1%) patients, out of whom right ventricular infarction (RVI) was confirmed in 384 (23.7%). Thrombolytic therapy was administered in 163 (42.4%) patients with RVI, and in 53 (41.7%) of these patients balloon dilatation was performed with coronary stent implantation in 24 (45.2%).* ***Results.*** *Favorable clinical effect of the combined thrombolytic therapy and percutaneous transluminal coronary angioplasty (PTCA) was achieved in 51 (96.1%), and in only 2 (3.9%) of patients the expected effect wasn't achieved. Myocardial revascularization was accomplished in 6 (3.6%) and 1 patient died. In 3 (3.4%) patients primary balloon dilatation with the implantation of intracoronary stent was performed within 6 hours from the onset of anginal pain. In the other group of 221 (57.5%) patients with RVI who did not receive thrombolytic therapy, or it had no effect, 26 (11.7%) patients died, which indicated the validity and the efficacy of this treatment (p<0,01). In the whole group of patients with myocardial infarction of the right ventricle 31 (8.1%) died; in the group that received thrombolytic therapy and PTCA 5 (3.1%) died, while in the group treated in a conservative way 26 (11.7%) died.* ***Conclusion.*** *Combined therapy was successful in the treatment of patients with RVI and should be administered whenever possible, since it was the best prevention of life-threatening complications and the decrease in the mortality of those patients.*

K e y w o r d s : **myocardial infarction; ventricular dysfunction, right; fibrinolytic agents; balloon dilatation; angioplasty, transluminal, percutaneous coronary; stents; combined modality therapy; treatment outcome.**

Introduction

Current treatment of AIM comprises volume compensation due to the reduced minute volume, combined thrombolytic, antiaggregation and anticoagulant therapy, together with primary or delayed (elective) percutaneous transluminal coronary angioplasty (PTCA) and intracoronary stent implantation, if necessary (1–6).

According to many previously published studies the incidence of AMI-RV ranged from 13 to 56%, depending on population, clinical, ECG, hemodynamic or patho-anatomic criteria. Mortality rate ranged from 20 to 50% (7–12). The results of the GUSTO IIb study showed that PTCA alone led to 45% of new restenoses during the four month follow-up period and therefore the effect of such therapy was practically annullated, although in the first act

it resulted with the complete potency of the infarcted artery (13–15). Owing to this, new studies – CADILLAC before all, as well as the others, suggested the administration of direct inhibitors of the platelet glycoprotein IIb/IIIa (abcximab) together with intracoronary low-molecular heparin. This modified therapy proved itself much more effective not only in infarction size and mortality rate reduction, but also in the lowered incidence of the complications such as myocardial reinfarction, restenosis, cerebrovascular accidents, during the hospital treatment, but also during one-year follow-up period after patients were discharged from the hospital (16–17). The application of any of these individual treatment methods or of their combination was aimed for primary preservetion of the myocardium, as well as for the management of reocclusion and restenosis (18–19). Widely accepted thrombolytic therapy together with more and more frequent PTCA generally used in the treatment of myocardial infarction and particularly in patients with AMI-RV, started a new era of interventional cardiology, as well as of the active approach in the treatment of these patients (20–24).

The aim of this study was to present our results with combined fibrinolytic therapy achived in the treatment of patients with AMI-RV, using accelerated tissue-type plasminogen activator protocol and primary or delayed PTCA together with intracoronary stent implantation, if necessary. Obtained results within one month intrahospital period were correlated with the clinical, ECG, echocardiographic and scintigraphic (SPECT) findings when necessary, radionuclide ventriculography (RV) and coronarography, or autopsy findings in patients who died.

Methods

Within the fifteen-year period (1987-2001), 3 765 patients of both sexes, 2 283 (60.6%) males and 1 482 (39.4%) females aged 60.1± 4.6 years in average underwent treatment of the acute myocardial infarction (AMI) of various localizations at the Clinic of Emergency Internal Medicine, Military Medical Academy (MMA). Infarction of anterior localization occurred in 2 146 (56.9%) and inferoposterior one in 1 619 (43.1%) of patients, which was confirmed in 384 (23.7%) cases of AMI-RV. Thrombolytic therapy was administered in the first 6–12 hours from the onset of anginous pain, and primary or delayed PTCA 6 hours up to 21 days after the infarction being confirmed clinically. They were performed by ECG, enzyme tests, echocardiography or radionucleide imaging of the myocardium, if necessary, and by autopsy in fatal cases. Selective coronarography was performed in all patients on thrombolytic therapy, aiming to evalute the obtained effect and to come to decision concerning further treatment. All results were analysed using χ^2 test in statistical analysis. During the last three years the modified treatment protocol was used introducing platelet glycoprotein IIb/IIIa inhibitors (abcximab) together with low-molecular heparin – enoxaparin in the dosis of 20 mg intracoronary.

Results

There were 384 patients in the group with AMI-RV, 229 (59.6%) males and 155 (40.4%) females and the average age of the whole group was 60.2 ± 5.8 years. Fibrinolytic therapy was administered to 163 (42.4%) patients and satisfactory reperfusion according to clinical, ECG and enzime criteria was achieved in 111 (68.1%) patients, but without the expected effect in 52 (31.9%) of patients. In the thrombolytic therapy group in 53 (41.7%) patients delayed or elective PTCA was performed in the later period and complete dilatation only by balloon was achieved in 27 (50.9%) patients. Partial effect of dilatation with remained lower degree stenosis was successfully managed by intracoronary stent implantation in 24 (45.2%) patients. Myocardial surgical revascularisation was done in 2 (3.9%) patients.

Primary percutaneous transluminal coronary angioplasty (PPTCA) was performed in 3 (3.9%) patients (2 females and 1 male) within the first 6 hours from the onset of anginous pain due to threatening cardiogenic shock and marked conduction disorders (AV block I^0–III^0). Complete effect of thrombolytic therapy combined with PTCA with coronary stenting was accomplished in 51 (96.1%) of patients with no fatal outcome and no serious clinical complications in the later course. Myocardial revascularization was found in 6 (3.6%) patients, while in the whole group receiving thrombolytic therapy together with primary or delayed PTCA and intracoronary stent 5 (3.1%) patients died – 3 in cardiogenic schock, 1 after a bypass surgery and 1 due to myocardial rupture. Coronary angiography showed pathologic changes in one blood vessel in 28 (22%), two in 36 (28.4%), and three in 47 (37%) patients, while 16 (11.5%) of them had tortuous changes of coronary arteries without significant stenosis.

In the group of 221 patients with AMI-RV who did not receive thrombolytic therapy or it was ineffective, 26 (11.7%) of them died. In the group receiving combined fibrinolytic therapy and PTCA 5 (3.1%) patients died (χ^2 test-9.56; $p<0.0l$), and in the whole group of 384 patients with the established AMI-RV 31 (8.1%) patient died, which undoubtedly confirmed the efficiency and justification of fibrinolytic therapy administration combined with PTCA in accordance with the modified protocol.

Discussion

Owing to the size of necrosis affecting the lower wall of both ventricles, distal half of interventricular septum, free wall of the right ventricle, papillary muscles together with the valvular apparatus, as well as cardiac conduction system, the infarction of the left and the right ventricle was in fact combined in patients with AMI-RV. It made their prognosis uncertain and mortality rate was still very high (25–27). The underlying physiopathologic substrate was stenosis or occlusion of proximally one third of the right

coronary artery (RCA), less frequently of the circumflex artery which was always predominant so that both sinus and AV node perfusions could have been compromised. This was clinically manifested as sinus bradycardia, hypotension, high degree AV block (II^0–III^0), or as the rapid development of cardiogenic shock (28–30). Current therapy recommends thrombolytic agents together with PTCA as an obligation aiming to open infarcted arteries as soon as possible and to definitely manage occlusion or stenosis (31–32).

More than 15 years had passed before the first communications on thrombolytic therapy administration in AMI-RV resulted in satisfactory reperfusion and increased the ejection fraction together with the less frequent AV blocks in patients with AMI-RV, comparing those in whom the reperfusion effect was not achieved with thrombolytic therapy (33–34). Such observation was also confirmed by the results in later TIMI 2 study in which thrombolytic therapy administered to patients with inferior infarction contributed to the reduced incidence of AMI-RV in patients with diaphragmal infarction. In those patients satisfactory reperfusion of the dominant RCA was achieved in comparison with those in whom this effect failed. This was an introduction into the new treatment strategy for patients with inferoposterior infarction in whom initial clinical signs or ECG findings suggested possible development of AMI-RV (35–37). The first study on successful primary angioplasty in patients with AMI-RV in whom satisfactory hemodynamic improvement of ejection fraction and rapid clinical recovery were recorded (38) was published at the end of eighties. Our experience and the obtained results in the treatment of these patients within the fifteen-year period were in accordance with many previous similarly designed studies and our attitude was that in all these patients, except for unavoidable fibrinolytic therapy, primary or most often delayed PTCA should be performed whenever necessary and possible, aiming to definitely manage residual stenosis. The effect of this combined therapy was best noticeable in the fact that in our subgroup of 27 patients with AMI-RV treated in this way there were no fatal outcomes during the first month of hospital treatment and that following complications were of no clinical significance. Meta-analysis of 10 world extensive randomised studies showed that primary PTCA reduced mortality rate and non-fatal myocardial infarction for 7.4% in comparison with intravenous administration of fibrinolytic therapy alone with the maintaining maximum TIMI 3 blood flow through the infarcted artery in about 90% of patients (39). Comparing clinical, ECG and coronarographic findings it was noticed that long and repeated anginous pain lasting longer than 40 minutes as the well as development of cardiogenic shock within the first 24 hours were the indicators of the unfavourable outcome, while the elevation of ST segment in V4 R–V5R >2 mV or bradycardia < 50/min were ECG signs suggesting the development of AV block III^0 or the rapid development of cardiogenic shock. Special precaution ought to have been taken in all these patients because these were the early signs suggesting possible lethal outcome. These results were confirmed in other authors' communications and they were the signs of poor prognosis and possible serious complications (40–41). Sufficient konowledge of early clinical and ECG indicators together with echocardiographic follow-up of patients enabled timely undertaken measures for elective balloon dilatation, and our previous experience and the obtained results completely confirmed it. The administration of the modified protocol combined with abciximab and enoxaparin multiply reduced the number of possible complications and the mortality rate (42). However, in about 30% of patients with maintained adequate angiographic blood flow (TIMI-3), there were clinical signs or marked, often irreversible cardiogenic shock, because they did not have the same blood flow on microcirculation level due to possible massive microembolism from the dilated subepicardial artery, or spontaneously developed microvascular thrombosis associated with the spasm and edema of the surrounding infarcted tissue (43, 44). Although angiography revealed completely potent and completely dilated artery, they died in an irreversible cardiogenic shock due to the so called „no-reflow“ phenomenon (45, 46). Thus, in spite of the great progress in the treatment of these patients, there are still many unanswered questions because we do not know why these patients still belong to the group at high risk with the uncertain outcome.

Conclusion

Our results showed that thrombolytic therapy combined with PTCA in a modified treatment protocol reduced the incidence of complications and mortality rate in patients with AMI-RV. This method of treatment undoubtedly reduced the size of necrosis and soon preserved the remained portion of the myocardium in the blood supply area of the infarcted artery which was shown to be the best prevention of the life threatening complications and the best way to reduce the moratality rate in these patients. Interventional cardiology also reduced the number of patients requiring additional myocardial revascularization risks related to this intervention and significantly lowered treatment costs. Thrombolytic therapy combined with PTCA most often resulted in rapid and complete potency of the infarcted artery, fast clinical recovery and favourable clinical course even after patients were discharged from the hospital. Out of 384 with AMI-RV thrombolytic therapy was administered patients to 163 (42.4%) of them, while the delayed PTCA together with intracoronary stent implantation was performed in 53 (29.6%) of patients. Complete clinical effect of this combined treatment was achived in 51 (96.1%) patients without the fatal outcome and serious clinical complications in the further course. PPTCA and intracoronary stent implantation were performed in 3 patients and their later clinical course and recovery were also without complications.

Unfortunately, in spite of the most up-to-date methods of treatment, mortality rate in these patients was still 4–5 times higher comparing to the ones with common infero-posterior left ventricle infarction in which this aggressive approach was neither necessary, nor recommendable.

The development of interventional cardiology thus introduced a new strategy in the treatment of these patients and our previous results, as well as the experience, completely confirmed this method as a very reliable one.

REFERENCES

1. *Sanders AO.* Coronary thrombosis with complete heart-block and relative ventricular tachycardia: a case report. Am Heart J 1930; 31(G): 820–3.
2. *Cohn JN, Guiha NH, Broder MI, Limas CJ.* Right ventricular infartion: clinical and haemodynamic features. Am J Cardiol 1974; 33(2): 209–14.
3. *Dell' Italia LJ.* The right ventricule: anatomy, physiology and clinical importance. Curr Probl Cardiol 1991; 16(10): 653–720.
4. *Kinch JW, Ryan TJ.* Right ventricular infarction. Engl J Med 1994; 330:(17); 1211–7.
5. *Isner JM, Roberts WC.* Right ventricular infarction complicating left ventricular infarction secondary to coronary heart disease: frequency, location, associated findings and significanse from analysis of 236 necropsy patients with acute or healed myocardial infarction. Am J Cardiol 1978; 42(6): 885–94.
6. *Etiene Y, Boshat J.* Fonction ventriculaire droite à la phase de convalescence d' un primoinfarctus posterieur. Arc Mal Coeur 1985; 78(3): 396–403.
7. *Zehender M, Kasper W, Kaunder E, Schonthaler M, Geibel A, Olschewski M,* et al. Right ventricular infarctions as independent predictor of prognosis after acute inferior myocardial infarction. N Engl J Med 1993; 328(14): 981–8.
8. *Serrano Junior CV, Ramires JA, Cesar LA, Zweier JL, Rati M, Deluz P,* et al. Prognostic significance of right ventricular dysfunction in patients with acute inferor myocardial infarction and right ventricular involvement. Clin Cadiol 1995; 18(4): 199–205.
9. *Reddy GV, Scharmroth L.* The electrocardiology of right ventricular myocardial infarction. Chest 1986; 90(5): 756–60.
10. *Erhardt LR.* Clinical and pathological observations in different types of acute myocardial infarction. A study of 84 patients deceased after treatment in a coronary care unit. Acta Med Scand 1974; 560(Suppl): 1–78.
11. *Cohn JN.* Right ventricular infarction revisted. Am J Cardiol 1979; 43(3): 666–8.
12. *Lloyd EA, Gerch BJ.* Haemodynamic spectrum of "dominant" right ventricular infarction in 19 patients. Am J Cardiol 1981; 48(6): 1016–22.
13. *O'Rourke RA.* Treatment of right ventricular infarction: thrombolytic therapy, coronary angioplasty or neither? J Am Coll Cardiol 1998; 32(4): 882–4.
14. *Zeymer U, Neuhaus KL, Wegsheider K, Tebbe LI, Molhoek P, Schroder R.* Effect of thrombolytic therapy in acute inferior myocardial infarction with or without right ventricular involvement. HIT-4 Trial Group Hirudin for Improvement of Thrombolysis. 1998; 32(4): : 876–81.
15. *THE GUSTO IIb INVESTIGATORS:* A clinical trial comparing primary coronary angioplasty with tissue plasminogen activator for acute myocardial infarction.The Global Use of Strategies to Open Occluded Coronary Arteries in Acute Coronary Syndromes (GUSTO IIb) Angioplasty Substudy Investigators. N Engl J Med 1997; 336(23): 1621–8.
16. *Chan AW, Moliterno DJ.* Defining the role of abciximab for acute coronary syndromes: lessons from CADILLAC, ADMIRAL, GUSTO IV, GUSTO V and TARGET. Curr Opin Cardiol 2001; 16(6): 375–83.
17. *Montalescot G, Barragan P, Wittenberg O, Ecollan P, Elhadad S, Villain P,* et al. Platelet glycoprotein IIb/IIIa inhibition with coronary stenting for acute myocardial infarction. N Engl J Med 2000; 344(25): : 1895–903.
18. *Bowers TR, O'Neill WW, Grines C, Pica MC, Safian RD, Goldstein JA.* Effect of reperfusion on biventricular function and survival after right ventricular infarction. N Engl J Med 1998; 338(14): 933–40.
19. *Goldstein JA.* Right heart ischemia: pathophysiology, natural history, and clinical management. Prog Cardiovasc Dis 1998; 40(4): 325–41.
20. *Giannitsis E, Potratz J, Wiegand U, Stierle U, Djonlagic H, Sheikzadeh A.* Impact of early accelerated dose tissue plasminogen activator on in-hospital patency of the infarcted vessel in patients with acute right ventricular infarction. Heart 1997; 77(6): 512–6.
21. *Jorgensen B, Simonsen S, Forgang K, Endresen K, Thaulow E.* Efect of percutaneous transluminal coronary angioplasty on exercise in patients with and without previous myocardial infarction. Am J Cardiol 1998; 82(9): 1030–3.
22. *Berger PB, Ryan TJ.* Inferior myocardial infarction. High risk subgroups. Circulation 1990; 81(2): 401–11.
23. *Orozović V, Matunović A, Marković S.* Clinical forms of combined infero-posterior myocardial infarction of the left and right ventricle. Kardiologija 1994; 15(1): : 59–65.

24. *Ryan TJ, Anderson JL, Antman EM, Braniff BA, Brooks NH, Califf RM,* et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: Executive summary and recommendations: a report of the American College of Cardiology/American Heart Association task force on practice guidelines. J Am Coll Cardiol 1996; 94(9): 2341–50.

25. *Isner JM.* Right ventricular myocardial infarction. JAMA 1988; 259(5): 712–8.

26. *Love JC, Haffajee CI, Gore JM, Alpert JS.* Reversibility of hypotension and shock by atrial or atrioventricular sequential pacing in patients with right ventricular infarction. Am Heart J 1984; 108(1): 5–13.

27. *Gueret P, Lacroix P, Leymarie JL, Bensaid J.* Infarction of the right ventricle: from physiopatological mechanisms to therapeutic strategy. Ann Med Interne (Paris) 1987; 138(4): 301–7.

28. *Kurose M, Okamoto K, Sato T, Ogata K, Yasumoto M, Terasaki H, Morioka T.* Extracorporeal life support for patients undergoing prolonged external cardiac massage. Resuscitation 1993; 25(1): 34–40.

29. *Dell' Italia LJ.* Reperfusion for right ventricular infarction. N Engl J Med 1998; 338(14): 978–80.

30. *Movahed A.* Treatment of right ventricular infarction. Am Fam Physician 1999; 60(6): 1640–9.

31. *Schuler G, Hofmann M, Schwarz F, Mehmel H, Menthey J, Tillmanns H,* et al. Effect of successful thrombolytic therapy on right ventricular function in acute inferor wall myocardial infarction. Am J cardiol 1984; 54(8): 951–7.

32. *Braat SH, Ramentol M, Halders S, Wellens HJ.* Reperfusion with streptokinase of an occluded right coronary artery: effects on early and late right and left ventricular ejection fraction. Am Heart J 1987; 113(2Pt1): : 257–60.

33. *Orozović V, Krgović M, Rafajlovski S, Gligić B, Damjanović M, Marković M,* et al. Trombolitička terapija i perkutana transluminalna koronarna angioplastika u bolesnika sa infarktom desne komore. Kardiologija 2001; 22(1–2): 33–41.

34. *Berger PB, Ruocco NA Jr, Ryan TJ, Jacobs AK, Zaret BL, Wackers FJ,* et al. Frequency and significance of right ventricular dysfunction during inferior wall left ventricular myocardial infarction treated with thrombolytic therapy (results from the thrombolysis in myocardial infarction [TIMI] II trial). Am J Cardiol 1993; 71(13): 1148–52.

35. *THE TIMI STUDY GROUP.* Comparison of invasive and conservative strategies after treatment with intravenous tissue plasminogen activator in acute myocardial infarction. Results of the thrombolysis in myocardial infarction [TIMI] phase II trial. N Engl J Med 1989; 320(10): 618–27.

36. *Robalino BD, Whitlow PL, Underwood DA, Salcedo EE.* Electrocardiographic manifestations of right ventricular infarction. Am Heart J 1989; 118(1): 138–44.

37. *Orozović V, Krgović M, Rafajlovski S, Gligić B, Marković M, Spasić S,* et al. Thrombolytic therapy and percutaneous transluminal coronary angioplasty in patients with right ventricular myocardial infarction. In: *Lewis BS, Halon DA, Flugelman MY, Toubol P*, editors. Coronary Artery Disease-Prevention to Intervention. Bologna, Italy: Monduzzi Editore; 2000. p. 499–506.

38. *Moreyra AE, Suh C, Porway MN, Kostis JB*. Rapid hemodynamic improvement in right ventricular infarction after coronary angioplasty. Chest 1988; 94(1): : 197–9.

39. *Topol EJ.* Reperfusion therapy for acute myocardial infarction with fibrinolytic therapy or combination reduced fibrinolytic therapy and platelet glycoprotein IIb/IIIa inhibition: the GUSTO V randomised trial. Lancet 2001; 357(9272): 1905–14.

40. *Braat SH, Gorgels AP, Bar FW, Wellens HJ.* Value of the ST - T segmet in lead V4R in inferior acute myocardial infarction to predict the site of coronar arterial occlusion. Am J Cardiol 1988; 62(1): 140–2.

41. *Weaver WD, Simes RJ, Betriu A, Grines CL, Zijlstra F, Garcia E,* et al. Comparison of primary coronary angioplasty and intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review. JAMA 1997; 278(23): 2093–8.

42. *Brener SJ, Barr LA, Burchenal JE, Katz S, George BS, Jones AA,* et al. Randomized, placebo-controlled trial of platelet glycoprotein IIb/IIIa blockade with primary angioplasty for acute myocardial infarction. ReoPro and Primary PTCA Organization and Randomized Trial (RAPPORT) Investigators. Circulation 1998; 98(8): 734–41.

43. *Pirwitz MJ, Hillis LD.* Emergency coronary artery bypass surgery for acute myocardial infarction. Coron Artery Dis 1994; 5(5): 385–91.

44. *Corbalan R, Larrain G, Nazzal C, Castro PF, Acevedo M, Dominguez JM,* et al. Association of noninvasive markers of coronary artery reperfusion to assess microvascular obstruction in patients with acute myocardial infarction treated with primary angioplasty. Am J Cardiol 2001; 88(4): 324–6.

45. *Ito H, Maruyama A, Iwakura K,Takiuchi S, Masuyama T, Hori M,* et al. Clinical implications of the 'no reflow' phenomenon. A predictor of complications and left ventricular remodeling in reperfused anterior wall myocardial infarction. Circulation 1996; 93(2): 223–8.

46. *Eeckhout E, Kern MJ.* The coronary no-reflow phenomenon: a review of mechanism and therapies. Eur Heart J 2001; 22(9): 729–39.

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A p s t r a k t

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SAVREMENA TERAPIJA INFARKTA MIOKARDA DESNE KOMORE

Uvod. Akutni infarkt miokarda desne komore (AIM-DK), posebna podgrupa u okviru inferoposteriornog infarkta leve komore, predstavlja veoma rizičnu bolest zbog brojnih, često teško predvidljivih komplikacija i visokog mortaliteta. **Metode.** U periodu od 15 godina (1987–2001) ukupno je od akutnog infarkta miokarda (AIM) različitih lokalizacija, lečeno 3 765 bolesnika oba pola, 2 283 muškaraca i 1 482 žena, prosečne starosti 61,4 ± 4,6 g. Infarkt prednjeg zida imalo je 2 146 (56,9%) bolesnika, inferiorni infarkt je imalo 1 619 (43,1%) bolesnika, od kojih je kod 384 (23,7%) bio dokazan infarkt desne komore. Trombolitičku terapiju dobilo je 163 (42,4%) sa AIM-DK, a kod 53 (41,7%) ovih bolesnika urađena je balon dilatacija, uz implantaciju intrakoronarnog stenta kod 24 (45,2%). **Rezultati.** Potpuni klinički efekat primenom kombinovane trombolitičke terapije i perkutane translumenske koronarne angioplastike (PTKA) postignut je kod 51 (96,1%), a očekivani efekat izostao je samo kod 2 (3,9%) bolesnika. Revaskularizacija miokarda urađena je kod 6 (3,6%) i jedan bolesnik je umro. Kod 3 (3,4%) bolesnika koji su primljeni na bolničko lečenje u prvih 6 sati od početka anginoznih bolova urađena je i direktna balon dilatacija sa implantacijom intrakoronarnog stenta i postignut potpuni klinički efekat. Nije bilo ni jednog smrtnog slučaja u grupi koja je lečena kombinovanom fibrinolitičkom terapijom i PTKA, kao ni u grupi u kojoj je primenjena primarna PTKA. U drugoj grupi od 221 (57,5%) bolesnika sa AIM-DK koji nisu dobili trombolitičku terapiju, ili je ona ostala bez efekta, umrlo je 26 (11,7%) što najbolje govori o uspešnosti i efikasnosti ovog načina lečenja ($p<0,01$). U celoj grupi bolesnika sa AIM-DK umrlo je 31 (8,1%) – u grupi koja je primila trombolitičku terapiju i PTKA umrlo je 5 (3,1%), dok je u grupi koja je lečena konzervativnom terapijom umrlo 26 (11,7%). **Zaključak.** Kombinovana terapija je veoma uspešna u lečenju bolesnika sa AIM-DK treba je primeniti u svim slučajevima gde je to moguće, jer je to najbolja prevencija životno opasnih komplikacija uz smanjenje smrtnosti ovih bolesnika.

K lj u č n e r e č i : **infarkt miokarda; srce, disfunkcija desne komore; fibrinolitici; dilatacija balonom; angioplastika, translumenska perkutana, koronarna; stentovi; lečenje, kombinovano, lečenje, ishod.**