Journal of Clinical & Experimental Cardiology

Ninkovic et al, J Clin Exp Cardiolog 2013, 4:7
DOI: 10.4172/2155-9880.1000253

Research Article Open Access

Predictors of In-Hospital Mortality in Patients with Acute Inferior Infarction of the Left Ventricle Accompanied by Right Ventricular Infarction when Treated with Percutaneous Coronary Intervention

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Abstract

Introduction: Right ventricular infarction (RVI) significantly increases the risk of in-hospital mortality in patients with acute inferior infarction of the left ventricle (AIILV). The present study evaluated pre-procedural and intra-procedural predictors of in-hospital mortality in patients with AIILV, accompanied by right RVI, when treated with primary percutaneous coronary intervention (PCI).

Methods: 237 patients with acute inferior infarction of the left ventricle accompanied with RVI and treated with PCI were admitted to the Coronary Care Unit at Clinical Centre of Serbia, from 2007 until 2009. In-hospital mortality was 4.2%. **Results:** Using a multivariate logistic regression model, two independent risk-factors for in-hospital mortality were identified: cardiogenic shock caused by right ventricular insufficiency (54.5% vs. 4.5% without cardiogenic shock, p=0.002) and post-procedural TIMI flow <3 (50.0% with TIMI flow <3 vs. 5.6% with TIMI flow 3, p=0.026). In the group of patients without cardiogenic shock, independent risk-factors of in-hospital mortality were Killip class >1 (p = 0.019) and previous PCI (p=0.021).

Conclusions and clinical implications: Cardiogenic shock and post-procedural TIMI flow <3 in the infarcted artery are independent predictors of in-hospital mortality. In patients without cardiogenic shock, the risk for in-hospital mortality correlates with the degree of left ventricular insufficiency and previous PCI.

Keywords: Mortality; Predictors; Infarction; Cardiogenic shock; Killip class; TIMI flow

Abbreviation

AIILV: Acute Inferior Infarction of the Left Ventricle; CASH: Cardiogenic Shock; PCI: Percutaneous Coronary Intervention; RV: Right Ventricular; RVI: Right Ventricular Infarction

Introduction

It is estimated that 30% of all acute inferior myocardial infarctions of the left ventricle (AIILV) is associated with right ventricular infarction (RVI) [1,2]. RVI is frequently caused by proximal occlusion of the dominant right coronary artery [3,4]. Hemodynamic consequences of RVI are asymptomatic right ventricular (RV) dysfunction, RV insufficiency with distended jugular veins together with maintained blood pressure. In most severe cases RVI is followed by a systemic hypotension and cardiogenic shock (CASH). RVI is commonly used term to indicate acute RV dysfunction caused by free wall motion abnormalities and/or dilation of right ventricle in the presence of ischaemic but viable myocardium [2-5]. The occurrence of the RVI during AIILV is associated with high in-hospital mortality and morbidity [6,7]. The treatment of these patients involves primarily early reperfusion. Reperfusion, either by thrombolysis or percutaneous coronary intervention (PCI) enhances the recovery of RV function and improves clinical outcome and survival [8-10].

Successful thrombolysis improves survival in patients with RVI, while the absence of reperfusion is associated with persistent dysfunction of the right ventricle and increased mortality [8-11]. Unsuccessful recanalization of the infarcted artery during fibrinolytic therapy occurs primarily in patients with proximal occlusion of the right coronary artery. Poor coronary delivery of fibrinolytic agents and restenosis may occur due to hypotension following such intervention. In contrast, the PCI has a higher success rate for recanalization of the occluded artery and has rapid beneficial effects on the global RV performance and clinical outcome [10,12].

There is a large number of models and scoring systems to assess the patient's risk of mortality in patients suffering acute myocardial infarction of the left ventricle [13-15]. Although RVI significantly increases the risk of in-hospital mortality in patients with AIILV, limited information is available about predictors of in-hospital mortality in these patients. Therefore the aim of the present study was to determine pre-procedural and intra-procedural predictors of in-hospital mortality in patients with AIILV, accompanied by RVI, who were treated with percutaneous coronary intervention.

Methods

Data were collected retrospectively from patients treated at the Coronary Care Unit at the Clinical Center of Serbia in Belgrade. During the three year period, from beginning of 2007 until the end of 2009, a total of 237 patients with acute inferior infarction of the left ventricle accompanied with infarction of the right ventricle were treated with primary PCI. Ten patients died during hospitalization and

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Received May 03, 2013; **Accepted** June 26, 2013; **Published** June 28, 2013

Citation: Ninkovic VM, Perunicic JZ, Ninkovic SM, Miloradovic VM, Vojislav G, et al. (2013) Predictors of In-Hospital Mortality in Patients with Acute Inferior Infarction of the Left Ventricle Accompanied by Right Ventricular Infarction when Treated with Percutaneous Coronary Intervention. J Clin Exp Cardiolog 4: 253. doi:10.4172/2155-9880.1000253

227 survived. For determination of predictors of in-hospital mortality, medical records of survivors and non-survivors were analysed.

AIILV accompanied by RVI was diagnosed when the following criteria were fulfilled: persistent discomfort in the chest that lasted ≥ 20 min with ST segment elevation ≥1 mm in at least two inferior leads and ST segment elevation ≥ 1 mm in the right precordial lead V4 (V4R), and confirmed by significantly increased concentration in cardiac enzymes. The ST-segment elevation in the V4R was used for diagnosis of the RV dysfunction due to its high sensitivity and specificity, and correlation with early mortality [16-18].

Following diagnosis of AIILV accompanied by RVI all patients were treated with double antiplatelet therapy (i.e. aspirin and clopidogrel) and were sent to the catheterization labaratory for PCI. In order to reduce time to PCI, we did not perform echocardiography prior to intervention. Similarly, the Swan-Gantz catheterization was only performed in few patients who were haemodinamically unstable. Due to a significant increase in time to PCI associated with Swan-Gantz catheterization, these haemodynamically unstable patients were excluded from analysis.

Cardiogenic shock was defined as systolic blood pressure less than 90 mmHg for more than 30 minutes, or a requirement for inotropic drugs to maintain systolic blood pressure ≥ 90 mmHg, after excluding other causes including hypovolemia and hemorrhage. Cardiogenic shock with clear lung fields (Killip class 1), and distended jugular veins was considered as a consequence of RV insufficiency.

Pre-procedural flow in the infarcted artery and procedural success was evaluated by a cardiologist who performed a PCI according to the TIMI flow grading system [16].

Statistical analysis

All statistical analyses were performed using the Statistical Package for Social Sciences, version 15.0 (SPSS Inc, Chicago, IL, USA). Data are presented as means and standard deviation for continuous variables and as frequencies and percentages for categorical variables. Categorical values were compared by χ^2 analysis. Association between ischemia time and in-hospital mortality was assessed with Mann-Whitney U Test. Relationships between each variable and in-hospital mortality rates were re-assessed by calculating the odds ratio.

Multiple logistic regression analysis was performed to identify independent variables associated with in-hospital mortality. Stepwise selection of the variable and estimation of significant probabilities were computed by means of the maximal likelihood ratio test. The variables were entered in the multivariate model when they were significantly related to mortality. Further logistic regression analysis was performed taking into account factors influencing mortality in patients without CASH. All statistical tests were two sided, with α=0.05.

Results

Patients

Mean (SD) age of the patients was 59.5 (10.8) years and 67% of patients were men. Cardiogenic shock was present in 11% and three vessel disease in 25%. A history of previous myocardial infarction was present in 10% and previous PCI in 5%. Mean ischemia time was 268.5 (160.9) minutes and mean door to balloon time was 88.5 (37.0) minutes (Table 1). Overall in-hospital mortality was 4.2% (10 out of 237).

Angiographic success and in-hospital mortality

TIMI grade 3 flow was achieved in 90% of all patients. In-hospital mortality was 5.6% in patients with post-procedural TIMI flow 3 compared with 50% in patients with post-procedural TIMI flow <3 (p <0.0001). In-hospital mortality was significantly lower among patients who started treatment within 90 minutes after admission (p=0.018), who were under 60 years old (p = 0.019), without history of angina (p=0.046), previous myocardial infarction (p=0.026) or PCI (p =0.022), without cardiogenic shock (p <0.0001), three vessel disease (p=0.007), with Killip class1 (p <0.0001), heart rate <100 bpm (p=0.007) and no distension of jugular veins (p=0.013). Using regression analysis, the following predictors of in-hospital mortality were identified: age (i.e. ≥60 years), previous myocardial infarction, previous PCI, heart rate ≥100 bpm, Killip class >1, distension of jugular veins, cardiogenic shock, longer ischemia time, door to balloon time >90 min, post-procedural TIMI flow <3, and three vessel disease (Table 2).

Independent predictors of in-hospital mortality were CASH (54.5% vs. 4.5% without CASH, p=0.002) and technical success (50% with TIMI flow <3 vs. 5.6% with TIMI flow 3, p=0.026) (Table 3).

Variables	Mean ± SD
Age (years)	59.5 ± 10.8
Weight (kg)	81.3 ± 5.1
Body mass index (kg/m^2)	27.5 ± 4.0
Heart rate (bpm)	75.4 ± 20.4
SBP (mmHg)*	138.2 ± 27.6
Ishemia time (min)	268.5 ± 160.9
Door to baloon time (min)	88.5 ± 37.0
	%
Male	67
Angina pectoris	17
Hypertension	57
Hyperlipidemia	41
Family history of CAD	30
Previous MI	10
Previous PCI	5
Previous CVA	8
Smoking	57
Diabetes	11
Killip class >1	8
Distension of jugular veins (%)	14
Cardiogenic shock (%)	11
ST changes in leads V1-V6 (%)	42
PVF (%)	12
Complete AV block (%)	13
Atrial fibrillation	17
Preprocedural TIMI 0-1	99
Postprocedural TIMI 3	90
Three vessel disease	25
Number of stents-one	58
Transferred	28

CAD indicates coronary artery disease; CVA, cerebrovascular accident; MI, myocardial infarction; PVF, primary ventricular fibrillation; AV, atrioventricular; SBP, systolic blood pressure.

*Patients with cardiogenic shock were excluded.

Table 1: Demographic, Clinical, and Angiographic Characteristics of Patient Population

Parameter	In-hospital Mortality (%)	p Value	OR (95% CI)	p Value
Age ≥ 60 years	17.8	0.019	5.73 (1.15-28.53)	0.033
Age < 60 years	3.6			
Male sex	9.0	0.620	1.40 (0.37-5.36)	0.621
Female sex	12.1			
Weight ≤ 70kg	3.8	0.544	1.95 (0.22-17.56)	0.550
Weight >70kg	7.2			
Body mass index ≤ 25 kg/m²	3.7	0.500	2.10 (0.23-18.83)	0.509
Body mass index >25 kg/m²	7.5			
Angina pectoris	23.5	0.046	3.85 (0.95-15.53)	0.058
No angina pectoris	7.4			
Hypertension	10.5	0.870	1.12 (0.30-4.24)	0.870
No hypertension	9.5			
Hyperlipidaemia	12.2	0.561	1.47 (0.40-5.46)	0.563
No hyperlipidaemia	8.6			
Family history of CAD	3.3	0.141	0.23 (0.03-1.90)	0.173
No family history of CAD	13.0			
Previous MI	30.0	0.026	5.08 (1.07-24.11)	0.041
No previous MI	7.8			
Previous PCI	40.0	0.022	7.25 (1.05-49.96)	0.044
No previous PCI	8.4			
Previous CVA	22.2	0.200	3.54 (0.89-14.09)	0.072
No previous CVA	8.8			
Smoking	7.0	0.236	0.45 (0.12-1.72)	0.244
No smoking	14.3			
Diabetes	8.3	0.203	2.54 (0.58-11.09)	0.216
No diabetes	18.8			
SBP <100 mmHg	18.2	0.338	2.25 (0.41-12.26)	0.349
SBP ≥100 mmHg	9.0			
Heart rate ≥ 100 bpm	37.5	0.007	7.20 (1.42-36.59)	0.017
Heart rate <100 bpm†	7.7			
Killip class >1	50.0	<0.0001	14.33 (2.85-72.01)	0.001
Killip class 1	6.5			
Distension of jugular veins	28.6	0.013	5.33 (1.28-22.19)	0.021
No distension of jugular veins	7.0			
Cardiogenic shock	54.5	<0.0001	25.5 (5.39-120.59)	<0.0001
No cardiogenic shock	4.5			
ST changes in leads V1-V6	9.5	0.893	0.91 (0.24-3.46)	0.893
No ST changes in leads V1-V6	10.3			
PVF	25.0	0.065	3.86 (0.85-17.60)	0.081
No PVF	8.0			
Complete AV block	15.4	0.488	1.79 (0.34-9.57)	0.493
No complete AV block	9.2			
Atrial fibrillation	5.9	0.613	0.58 (0.07-4.95)	0.617
No atrial fibrillation	9.8			
Ischemia time, mean (min)	(259.10/361.22)*	(0.102)	1.006 (1.001-1.011)	0.023
Ischemia time >360 min	20.0	0.115	3.21 (0.71-14.57)	0.131
Ischemia time ≤360 min	7.2			
Door to balloon time >90 min	17.5	0.018	9.12 (1.07-77.82)	0.043
Door to balloon time ≤90 min	2.3			
Preprocedural TIMI 0-1	10.5	0.444	0,70 (0,14-3,52)	0,665
Preprocedural TIMI >1	0.0			
Postprocedural TIMI 3	5.6	<0.0001	17.00 (3.67-78.74)	<0.0001
Postprocedural TIMI <3	50.0			
3- vessel disease	24.0	0.007	5.6 0(1.43-21.9)	0.013
1 or 2 - vessel disease	5.3			
Number of stents-one	12.1	0.418	1.78(0.43-7.35)	0.423
Number of stents-two or more	7.1			
Transferred	10.7	0.724	1.3 (0.30-5.60)	0.725
No transferred	8.5			

(*-survived/dead; † - excludes patients with atrioventricular block)

Table 2: Predictors of in-hospital mortality: univariate analysis

Independent predictors of in-hospital mortality in group of patients without CASH were previous PCI (p = 0.021) and Killip class >1 (p=0.019) (Table 4).

Discussion

This is the first study to assess predictors of in-hospital mortality in patients suffering acute myocardial infarction accompanied with right ventricular infarction and threated with percutaneous coronary intervention. The major findings suggest that the strongest predictors of in-hospital mortality are cardiogenic shock and TIMI flow<3 in the infarcted artery. In the group of patients without cardiogenic shock the major predictors of in-hospital mortality are previous PCI and Killip Class >1.

There are a large number of models and scoring systems to assess the patient's risk of mortality in patients suffering acute myocardial infarction of the left ventricle [13-15]. Although RVI significantly increases the risk of in-hospital mortality in patients with AIILV, prognostic risk factors for such outcome are still poorly established. Gumina and co-workers evaluated the application of TIMI risk score in patients with RVI and found that the initial TIMI risk score had the capability to predict in-hospital morbidity and mortality and long-term mortality [19]. However, the inclusion criteria for this study have been questioned in the relation to high in-hospital mortality rate of 22% [20]. It has been suggested that in-hospital and long-term mortality correlated with the degree of RV insufficiency during RVI but not with the admission TIMI risk score [21].

When PCI was established for treatment of patients with left ventricular infarction with ST elevation, angiographic parameters were found to be independent predictors of in-hospital and long-term mortality. The data from a large German registry of PCI (ALKK), with approximately 5,000 patients from 80 hospitals, showed that the independent predictors of in-hospital mortality in patients with left ventricular infarction with ST elevation, who underwent the PCI, were CASH, age, post-procedural TIMI flow less than 3, three-vessel disease, anterior infarction, the year of inclusion and, the number of PCI procedures in each center (other observed parameters were left bundle branch block, ischemia time and door to balloon time) [22]. In another study with more than 1,700 patients who suffered left ventricular infarction and underwent PCI, six independent predictors of 30-day mortality were identified: Killip class, age, anterior infarction, ischemia time, post-procedural TIMI flow and three-vessel disease, but not door to balloon time [23]. In our study independent predictors of in-hospital mortality in patients with AIILV accompanied by RVI who underwent primary PCI were CASH and post-procedural TIMI flow <3. It should be emphasized that in our study the CASH was primarily due to RV insufficiency (out of five surviving patients with CASH, three were without left ventricular insufficiency (Killip-1), one had mild insufficiency (Killip-2) and one had moderate to severe left ventricular insufficiency (Killip-3). Among the non-survivors, out of six patients with CASH, four patients were classified as Killip-1, one was classified as Killip-2 and one was classified as Killip-3.

Ischemia time and door to balloon time

In our study we distinguished the two time-domain variables: 1) ischemia time i.e. the time from the beginning of chest discomfort to first balloon inflation, and 2) door to balloon time i.e. the time from admission to hospital to first balloon inflation. These times are important because their influence on in-hospital mortality has been debated. The

Parameter	Adjusted OR	95% CI	p Value
Cardiogenic shock	18.26	3.00-111.17	0.002
Postprocedural TIMI <3	8.89	1.30-60.74	0.026

Table 3: Predictors of in-hospital mortality: Multivariate logistic regression analysis

Parameter	Adjusted OR	95% CI	p Value
Previous PCI	39.00	1.74-871.72	0.021
Killip class>1	36.90	1.69-849.34	0.019

Table 4: Predictors of in-hospital mortality: Multivariate logistic regression analysis (group of patients without cardiogenic shock)

two large showed that there was no significant increase in in-hospital mortality as time to PCI increases, if this time was within the 12 hours after symptom onset [22,24]. This was further supported by Shöming and associates who demonstrated the amount of rescued myocardium (i.e. Salvage index) depends on the time from symptom onset to the beginning of the fibrinolytic therapy [25]. In contrast, the time until the beginning of PCI is not a predictor of mortality if performed within 12 hours after the onset of symptoms [25]. In contrast, more recent study suggests that shorter time from the onset of symptoms to PCI was associated with smaller infarct size, less microvascular obstruction and larger amounts of rescued myocardium [26]. It is clear that the duration of ischemia time is inversely related to the size of myocardial infarciton. Indeed, it seems that duration of ischemia time can be critical in high risk patients with large ischemia as supported with correlation between ishemia time and in-hospital mortality in studies with high-risk patients [23,27,28]. In addition to strong relationship between door to balloon time and in-hospital mortality, evidence further suggests that there is increased risk for in-hospital mortality when this time is longer than 2 hours [24]. Interestingly, these results were not confirmed during large multi-centre trials [22,29]. This discrepancy is likely to be explained by different time interval between door to balloon which was 83 vs. 55 minutes [22,26,29]. It has further been suggested that an additional non-linear reduction in in-hospital mortality was associated with progressive reduction of door to balloon time below 90 minutes [30]. Current recommendation suggests that this time should be shorter than 90 minutes [31]. In our study, although the ischemia time and door to balloon time were risk factors for in-hospital mortality, their impact at multi-variate analysis was diminished by the strong influence of CASH, procedural failure on in-hospital mortality, left ventricular insufficiency, and previous PCI in patients without CASH.

The present study is not without limitations. The sample size is relatively small with only 10 mortality events, and therefore the study may have not sufficient power. Nevertheless we strongly believe that our findings are clinically relevant and will drive development of future large studies in this area.

Conclusion

In patients with acute infarction of the inferior wall of the left ventricle, which is considered to be low risk, when accompanied by right ventricular infarction, and treated with percutaneous coronary intervention within 12 hours after symptom onset, the independent predictors of in-hospital mortality are cardiogenic shock caused by right ventricular insufficiency and procedural failure of percutaneous coronary intervention confirmed by post-procedural TIMI flow <3 in the infarcted artery. In the group of patients without cardiogenic shock, the risk for in-hospital mortality is associated with the degree of left ventricular insufficiency during infarction and previous percutaneous coronary intervention. Future large clinical studies are required to enhance our understanding of mortality predictors in right ventricular infraction.

Competing Interests

The authors declare no conflict of interests.

Acknowledgements

We are grateful to Dr Guy A MacGowan from Freeman Hospital, Newcastle uypon Tyne, UK for his critical review of the manuscript.

References

1. Kinch JW, Ryan TJ (1994) Right ventricular infarction. N Engl J Med 330: 1211-1217.
2. Goldstein JA (1998) Right heart ischemia: pathophysiology, natural history, and clinical management. Prog Cardiovasc Dis 40: 325-341.
3. Isner JM, Roberts WC (1978) Right ventricular infarction complicating left ventricular infarction secondary to coronary heart disease. Frequency, location, associated findings and significance from analysis of 236 necropsy patients with acute or healed myocardial infarction. Am J Cardiol 42: 885-894.
4. Andersen HR, Falk E, Nielsen D (1987) Right ventricular infarction: frequency, size and topography in coronary heart disease: a prospective study comprising 107 consecutive autopsies from a coronary care unit. J Am Coll Cardiol 10: 1223-1232.
5. Lim St, Marcovitz P, O´ Neill WA, Goldstein JA (2003) Right ventricular performance is preserved during stress in patients with chronic proximal right coronary occlussion. Am J Cardiol 92: 1203-1206.
6. Zehender M, Kasper W, Kauder E, Schönthaler M, Geibel A, et al. (1993) Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. N Engl J Med 328: 981-988.
7. Hamon M, Agostini D, Le Page O, Riddell JW, Hamon M (2008) Prognostic impact of right ventricular involvement in patients with acute myocardial infarction: meta-analysis. Crit Care Med 36: 2023-2033.
8. Zehender M, Kasper W, Kauder E, Geibel A, Schönthaler M, et al. (1994) Eligibility for and benefit of thrombolytic therapy in inferior myocardial infarction: focus on the prognostic importance of right ventricular infarction. J Am Coll Cardiol 24: 362-369.
9. Kinn JW, Ajluni SC, Samyn JG, Bates ER, Grines CL, et al. (1995) Rapid hemodynamic improvement after reperfusion during right ventricular infarction. J Am Coll Cardiol 26: 1230-1234.
10. Bowers TR, O'Neill WW, Grines C, Pica MC, Safian RD, et al. (1998) Effect of reperfusion on biventricular function and survival after right ventricular infarction. N Engl J Med 338: 933-940.
11. Giannitsis E, Potratz J, Wiegand U, Stierle U, Djonlagic H, et al. (1997) Impact of early accelerated dose tissue plasminogen activator on in-hospital patency of the infarcted vessel in patients with acute right ventricular infarction. Heart 77: 512-516.
12. Goldstein JA (2002) Pathophysiology and management of right heart ischemia. J Am Coll Cardiol 40: 841-853.
13. Lee KL, Woodlief LH, Topol EJ, Weaver WD, Betriu A, et al. (1995) Predictors of 30-day mortality in the era of reperfusion for acute myocardial infarction. Results from an international trial of 41,021 patients. GUSTO-I Investigators. Circulation 91: 1659-1668.
14. Maseri A, Rebuzzi AG, Cianflone D (1997) Need for a composite risk stratification of patients with unstable coronary syndromes tailored to clinical practice. Circulation 96: 4141-4142.
15. Morrow DA, Antman EM, Charlesworth A, Cairns R, Murphy SA, et al. (2000) TIMI risk score for ST-elevation myocardial infarction: A convenient, bedside, clinical score for risk assessment at presentation: An intravenous nPA for treatment of infarcting myocardium early II trial substudy. Circulation 102: 2031-2037.
16. [No authors listed] (1985) The Thrombolysis in Myocardial Infarction (TIMI) trial. Phase I findings. TIMI Study Group. N Engl J Med 312: 932-936.
17. Candell-Riera J, Figueras J, Valle V, Alvarez A, Gutierrez L, et al. (1981) Right ventricular infarction: relationships between ST segment elevation in V4R and hemodynamic, scintigraphic, and echocardiographic findings in patients with acute inferior myocardial infarction. Am Heart J 101: 281–287.
18. Braat SH, Brugada P, de Zwaan C, Coenegracht JM, Wellens HJ (1983) Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction. Br Heart J 49: 368-372.
19. Gumina RJ, Wright RS, Kopecky SL, Miller WL, Williams BA, et al. (2002) Strong predictive value of TIMI risk score analysis for in-hospital and long-term survival of patients with right ventricular infarction. Eur Heart J 23: 1678-1683.
20. Wong CK, White HD (2002) Risk stratification of patients with right ventricular infarction: is there a need for a specific risk score? Eur Heart J 23: 1642-1645.

21. Lupi-Herrera E, Chuquiure-Valenzuela E, González-Pacheco H, Juárez-Herrera U, Martínez-Sánchez C, et al. (2008) A proposed functional clinical classification predicts in-hospital and long-term survival in the setting of acute right ventricular infarction. Arch Cardiol Mex 78: 369-378.

22. Zahn R, Vogt A, Zeymer U, Gitt AK, Seidl K, et al. (2005) In-hospital time to treatment of patients with acute ST elevation myocardial infarction treated with primary angioplasty: determinants and outcome. Results from the registry of percutaneous coronary interventions in acute myocardial infarction of the Arbeitsgemeinschaft Leitender Kardiologischer Krankenhausarzte. Heart 91: 1041-1046.

23. De Luca G, Suryapranata H, van 't Hof AW, de Boer MJ, Hoorntje JC, et al. (2004) Prognostic assessment of patients with acute myocardial infarction treated with primary angioplasty: implications for early discharge. Circulation 109: 2737-2743.

24. Cannon CP, Gibson CM, Lambrew CT, Shoultz DA, Levy D, et al. (2000) Relationship of symptom-onset-to-balloon time and door-to-balloon time with mortality in patients undergoing angioplasty for acute myocardial infarction. JAMA 283: 2941-2947.

25. Schömig A, Ndrepepa G, Mehilli J, Schwaiger M, Schühlen H, et al. (2003) Therapy-dependent influence of time-to-treatment interval of myocardial salvage in patients with acute myocardial infarction treated with coronary artery stenting or thrombolysis. Circulation 108: 1084-1088.

26. Francone M, Bucciarelli-Ducci C, Carbone I, Canali E, Scardala R, et al. (2009) Impact of primary coronary angioplasty delay on myocardial salvage, infarct size, and microvascular damage in patients with ST-segment elevation myocardial infarction. Insight from cardiovascular magnetic resonance. J Am Coll Cardiol 54: 2145-2153.

27. Brodie BR, Stuckey TD, Muncy DB, Hansen CJ, Wall TC, et al. (2003) Importance of time-to-reperfusion in patients with acute myocardial infarction with and without cardiogenic shock treated with primary percutaneous coronary intervention. Am Heart J 145: 708-715.

28. Zeymer U, Vogt A, Zahn R, Weber MA, Tebbe U, et al. (2004) Predictors of in-hospital mortality in 1333 patients with acute myocardial infarction complicated by cardiogenic shock treated with primary percutaneous coronary intervention (PCI); Results of the primary PCI registry of the Arbeitsgemeinschaft Leitende Kardiologische Krankenhausärzte (ALKK). Eur Heart J 25: 322-328.

29. De Luca G, Suryapranata H, Zijlstra F, van 't Hof AW, Hoorntje JC, et al. (2003) Symptom-onset-to-balloon time and mortality in patients with acute myocardial infarction treated by primary angioplasty. J Am Coll Cardiol 42: 991-997.

30. Rathore SS, Curtis JP, Chen J, Wang Y, Nallamothu BK, et al. (2009) Association of door-to-balloon time and mortality in patients admitted to hospital with ST elevation myocardial infarction: national cohort study. BMJ 338: b1807.

31. Steq PG, James SK, Atar D, Badano LP, BlÖmstrom-Lundqvist C, Borqer MA, et al. (2012) ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J 33: 2569-2619.