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Publication date

09-06-2023

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Document Version

Accepted version

Citation for this work (American Psychological Association 7th edition)

Schwarz, B., Abdel-Wahab, M., Robinson, D. R., & Richardt, G. (2016). *Predictors of mortality in patients with cardiogenic shock treated with primary percutaneous coronary intervention and intra-aortic balloon counterpulsation* (Version 1). University of Sussex. <https://hdl.handle.net/10779/uos.23441693.v1>

Published in

Medizinische Klinik – Intensivmedizin und Notfallmedizin

Link to external publisher version

<https://doi.org/10.1007/s00063-015-0118-8>

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Predictors of mortality in patients with cardiogenic shock treated with primary percutaneous coronary intervention and intra-aortic balloon counterpulsation

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Running title: Mortality predictors in cardiogenic shock

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Abstract

Background

Cardiogenic shock remains the most serious complication of patients hospitalized with acute myocardial infarction (AMI). Early revascularization is the cornerstone of invasive therapy, while mechanical support with intra-aortic balloon pump (IABP) is debatable. From our institutional shock registry we sought to determine predictors of in-hospital mortality - including the aspect of IABP timing - and to develop a clinical risk score for shock patients with AMI.

Methods

From January 2005 till December 2010, 102 patients with cardiogenic shock due to AMI treated with primary percutaneous coronary intervention (PCI) and IABP were analyzed. Univariate and multivariate logistic regression analyses were used to identify independent predictors of in-hospital mortality. Logistic regression analysis and receiver-operating curves were used to generate a mortality risk score.

Results

The mean age of the cohort was 70.1 ± 11.0 years and 70% were males. One third of patients had a non-ST segment elevation myocardial infarction and 30% had to be resuscitated before coronary intervention. Mean left ventricular ejection fraction was 25%. After admission, 23% of patients developed an acute renal failure and 10% needed renal dialysis during hospital stay. In 52% of patients IABP therapy was initiated after primary PCI, while the remaining patients had an IABP-assisted primary PCI. All cause in-hospital mortality was 40.2%.

Using multivariate analysis, age (odds ratio [OR] 1.08, $p=0.006$), resuscitation before PCI (OR 3.46, $p=0.045$), vasopressor use (OR 7.88, $p=0.003$), acute renal failure (OR 11.18, $p=0.001$) and IABP-implantation after PCI (OR 4.36, $p=0.011$) were independently associated with in-hospital mortality. Based on these predictors, a mortality-risk score was calculated as follows: $1.5 \times \text{IABP-timing before PCI} + 0.1 \times \text{age} + \text{resuscitation before PCI} + 2 \times \text{vasopressor use} + 2.5 \times \text{acute renal}$

failure . Using a cut-off value of 10.4, this score had a specificity of 83% and a sensitivity of 82% for prediction of in-hospital death.

Conclusions

We identified age, vasopressor use, resuscitation before PCI, acute renal failure and IABP-implantation after PCI as independent predictors of in-hospital mortality in patients with cardiogenic shock due to AMI. The timing of IABP insertion was the only modifiable factor predicting in-hospital mortality in our cohort. Consequently, balloon pumping should be started before PCI to improve outcome of CS patients.

Keywords: cardiogenic shock; myocardial infarction; intra-aortic balloon; outcome

Introduction

Cardiogenic shock (CS) affects 5-8% of patients with acute myocardial infarction (AMI) (Thiele et al., 2010). Although modern revascularization strategies have achieved a significant mortality reduction (Hochmann et al., 1999), CS remains the most serious complication of patients hospitalized for AMI, and mortality is still approaching 50%.

Despite the obvious impact of CS on public health, uncertainties remain concerning pathophysiology and treatment. Most recently, mechanical support with intra-aortic balloon pump counterpulsation (IABP) failed to reduce mortality at 30 days and 12 months in a large multicenter randomized controlled trial (Thiele et al. 2012, Thiele et al. 2013). These results challenged the traditional concept of mechanical support in CS, provoked a debate about use and misuse of IABP, and ultimately left clinicians in uncertainty while treating individuals with CS (Perera et al. 2013). Moreover, currently available ICU outcome scores appear to be inappropriate to guide the management of CS patients (Kellner et al. 2013).

With this background, we sought to determine predictors of in-hospital mortality including the aspect of IABP-timing, and to develop a clinical risk score for shock patients with AMI from our institutional shock registry. All patients received primary percutaneous coronary intervention (PCI) and an IABP – either before or after PCI - as well as pharmacological and fluid management according to current guidelines. In this cohort, identification of mortality predictors may not only yield patient subgroups with higher or lower likelihood to survive CS, but can also define the impact of different treatment strategies on overall clinical outcome.

Methods

Study design and patient population

Patients with CS are consecutively included in our institutional shock registry since January 2005. The present analysis comprises patients with CS complicating AMI treated with primary PCI and IABP. The study complied with the Declaration of Helsinki and data collection was approved by

the local ethics committee. All patients provided written informed consent for the retrospective analysis of their anonymized data.

Cardiogenic shock was confirmed clinically by the presence of hypotension (systolic blood pressure of < 90 mmHg for > 30 minutes or the need for supportive measures to maintain the systolic blood pressure > 90 mmHg) and end organ hypoperfusion (cool extremities or a urine output < 30 ml/hour) after adequate correction of preload and major arrhythmias. To obtain a more homogenous population, only patients with AMI and CS due to left ventricular failure were included in the present analysis. Patients with mechanical complications such as ventricular rupture or acute severe mitral regurgitation, isolated right ventricular infarction, and shock resulting from excess β -blockade or calcium channel blockade or as a complication of cardiac catheterization, were excluded. Patients who did not have IABP support within 24 hours from the index PCI were also not considered.

Coronary intervention and IABP technique

Immediately after the diagnosis of AMI, a loading dose of intravenous aspirin (500 mg) and clopidogrel (600 mg) was given to all patients. Unfractionated heparin was given at 70 U/kg at initial presentation and additional heparin doses were given during PCI to maintain an activated clotting time of 250 to 300 seconds and between 200 and 250 seconds if a glycoprotein IIb/IIIa inhibitor was administered. Cardiac catheterization was performed through the femoral route using 6Fr systems in all patients. Contrast ventriculography was routinely performed in the right anterior oblique projection. Coronary angiography and PCI were performed in a conventional manner. IABP was inserted either before or after PCI; the exact timing was dependent on the operator's decision following clinical and/or logistic considerations.

In case of IABP before PCI, implantation was mostly performed through the contralateral femoral artery. Patients receiving IABP support after PCI had the pump inserted using the same femoral artery access which had been used for cardiac catheterization and PCI. The IABP was inserted through an 8Fr sheath and was guided into the descending aorta, approximately 2 cm from the left

subclavian artery. Aortic counterpulsation was electrocardiographically triggered in all patients, and the balloon was generally left for 48 hours at a rate of 1:1. The patient was then gradually weaned off the pump during a 12-hour period before removal. Aortic counterpulsation was stopped earlier in case of complications such as limb ischemia or access site bleeding.

Medical therapy

Standard coronary care management was provided. After the procedure, clopidogrel was continued for at least 6 months (according to local practice), and aspirin was prescribed indefinitely for all patients. If no contraindications were present, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, β -blockers, and statins were applied. Vasopressor drugs - mainly norepinephrine - were used in hemodynamically unstable patients.

Study endpoints

The primary end point for the present analysis was all cause in-hospital death. The secondary end points evaluated included cardiac death, recurrent nonfatal myocardial infarction, target vessel revascularization, and the occurrence of cerebrovascular ischemic or hemorrhagic events. In addition, the occurrence of renal failure (defined as serum creatinine > 2.5 mg/dl) and major bleeding leading to a decrease of hemoglobin level > 5 g/dl or that requiring blood transfusion were analyzed. Cardiac death was defined as any death due to an approximate cardiac cause, death of unknown cause, and all procedure-related death (Cutlip et al.,2007). The recurrence of myocardial infarction was defined as recurrent chest pain lasting for > 30 minutes after the index procedure, associated with new Q waves in > 2 leads or recurrent ST- segment elevation > 0.1 mV in > 2 contiguous leads, and/or re-elevation of creatine kinase-MB levels to at least twice the upper limit of normal and $> 50\%$ greater than the previous value. Target vessel revascularization was defined as repeat PCI or surgical bypass grafting of any segment of the target vessel.

Statistical analysis

Data evaluation was performed using a statistical soft-ware package (Minitab, version 13.1). Continuous variables are expressed as mean \pm SD or median and interquartile range and were

analyzed using the Student's t test or Mann-Whitney test, as appropriate. Discrete variables are presented as counts and percentages and were analyzed using the Pearson chi-square test or Fisher's exact test, as appropriate. All potential predictors for in-hospital mortality were studied using univariate logistic regression analysis. Promising variables in the univariate analysis ($p < 0.1$) were included in a multivariate logistic regression model with a backward selection approach. Adjusted odds ratios are presented with 95% confidence intervals. The logistic regression model was used to determine a preliminary prognostic score for in-hospital mortality. The ROC curve for this score (i.e. a plot of sensitivity against 1-specificity for each cut-off value) was plotted, the area under the curve (AUC) determined, and a 95% confidence interval for the AUC found using the bootstrap method. A p value < 0.05 was considered statistically significant.

Results

Baseline clinical and hemodynamic characteristics

From January 2005 till December 2010, 102 consecutive patients with AMI and CS treated with primary PCI and IABP were identified and included in the current analysis. The study cohort represents a typical contemporary CS population (table 1). Mean age of the study population was 70 years, most patients were men, and nearly one third of patients had a non-ST-segment-elevation myocardial infarction. Cardiovascular risk factors were present in a high proportion of patients, with 46.1% of patients having diabetes, 72.5% hypertension, 56.9% hyperlipidemia, and 32.4% active smokers. History of previous MI and previous CABG was 28.4% and 14.7%, respectively. Nearly two thirds of the population had chronic renal impairment, and about one third had to be resuscitated before coronary intervention. The mean lactate level of our cohort was clearly elevated (4.3 ± 4.1 U/l). The mean systolic and diastolic blood pressure levels were 102.1 ± 24.1 mmHg and 59.2 ± 17.5 mmHg, respectively, with a heart rate of 88.8 ± 22.4 beats/min. Notably, 41.2% of patients had to be treated with vasopressors before PCI. Inflammatory markers such as white blood cells and C-reactive protein were slightly elevated (13.9 ± 11.9 and 9.1 ± 8.4 , respectively).

Procedural details

Periprocedural characteristics (table 2) revealed a mean left ventricular ejection fraction of 24.7%. While 85.3% of patients presented with multivessel disease, approximately half of the population had their culprit lesion in the left anterior descending coronary artery (LAD). In 52% of patients IABP-support was initiated after coronary intervention, while the remaining patients had an IABP-assisted primary PCI. The mean procedural duration was 94.8 ± 44.2 minutes.

In-hospital outcome

Nearly 60% of patients had to be treated with vasopressors during hospital stay. Regarding renal function, 22.5% of patients developed acute renal failure and 9.8% needed renal dialysis during hospitalization. Peak serum levels of creatine-kinase (CK) and CK-MB were substantially elevated (3395 ± 4901 U/l and 323 ± 377 U/l, respectively).

The in-hospital outcome of the cohort is shown in table 3. The average hospital stay was 16.5 ± 14.6 days and patients had a mean duration of mechanical ventilation of 5 ± 8.5 days. All-cause mortality was 40.2%, and most patients died from cardiac complications. Acute myocardial re-infarction occurred in 2 patients (2%), and 4 patients (3.9%) had a cerebrovascular event. Major bleeding was a dominant complication and occurred in 25 patients (25.5%).

As listed in table 4, age, atrial fibrillation and chronic renal failure were the strongest mortality predictors among the demographic characteristics. Resuscitation before PCI also increased the mortality risk, while shock indicators such as blood pressure and serum lactate were less important. The need to use vasopressors and the occurrence of acute renal failure were both strongly associated with in-hospital death, and delayed IABP use after PCI was the only procedural variable with major impact on mortality.

Predictors of in-hospital mortality

Using multivariate analysis, five independent predictors of in-hospital mortality could be identified: age (odds ratio [OR] 1.08, 95% confidence interval [CI] 1.02-1.15), resuscitation before PCI (OR 3.46, 95%CI 1.03-11.62), vasopressor use (OR 7.88, 95%CI 2.01-30.88), acute renal

failure (OR 11.18, 95%CI 2.71-46.07) and IABP-implantation after PCI (OR 4.36, 95%CI 1.39-13.62) (figure 1).

Based on these predictors a mortality-risk score was calculated as follows:

$1.5 \times \text{IABP-Timing (before PCI = 0; after PCI = 1)} + 0.1 \times (\text{age}) + \text{resuscitation before PCI (no = 0; yes = 1)} + 2 \times \text{vasopressor use (no = 0; yes = 1)} + 2.5 \times \text{acute renal failure (no = 0; yes = 1)}$.

Using a cut-off value of 10.4, this score had a specificity of 83% and a sensitivity of 82% for prediction of in-hospital death (figure 2).

Discussion

This is a reasonably large cohort study in patients with AMI and CS. All patients were treated with primary PCI and mechanical support (IABP). Although patients received a contemporary and guideline conform treatment, in-hospital mortality was still high with a rate of 40% (Antman et al., 2004; van de Werf et al., 2003). The mortality rate and the patient characteristics are very similar to the recently published IABP-Shock II trial. Notably, the mean ejection fraction of our cohort was lower (25% vs. 35%), and less patients were resuscitated before PCI (30% vs. 45%) compared to IABP-SHOCK II. Both factors, the relatively high ejection fraction and the high rate of post-resuscitation patients, are regarded as limitations of the IABP-Shock II trial.

We identified age, vasopressor use, resuscitation before PCI, acute renal failure and IABP-implantation after PCI as independent predictors of in-hospital mortality. Age, renal failure, vasopressor use and CRP had been reported as adjusted predictors for 30-day mortality in recent studies (Katz et al., 2009; Cheng et al., 2009). Other factors reported in the latter studies such as blood pressure, lactate levels or mechanical ventilation did not prove to be independent predictors in our multivariate analysis. Based on these predictors, a mortality-risk score was calculated, and using a cut-off value of 10.4, this score had an excellent specificity (83%) and sensitivity (82%) for prediction of in-hospital death. Risk scores have gained increasing importance for decision making in critically ill patients. Currently, the APACHE II and the SAPS II scores are the most useful ICU assessment tools for the prognostic outcome of critically ill patients (Knaus et al., 1985;

LeGall et al., 1984), but for CS patients these scores are less useful, since a recent study reported sensitivity and specificity rates below 80% in a contemporary CS cohort (Kellner et al., 2013). Moreover, all mentioned ICU risk scores focus on physiological measurements and do not include specific therapeutic strategies. Even a reliable risk score for patients with advanced coronary artery disease undergoing PCI such as the SYNTAX score had no prognostic impact in our shock patients as previously reported (Abdel-Wahab et al, Abstract, Eurointervention). In contrast, our risk score is based on a few parameters, which can be easily assessed and applied in a short equation.

In our cohort, timing of IABP insertion was assessed and turned out to be the only modifiable factor predicting in-hospital mortality. IABP is an established technology and still the most widely used mechanical system for hemodynamic support in CS. However, the evidence for IABP use in CS has been challenged by recent studies. A meta-analysis of observational studies found a 6% mortality increase with IABP (Sjauw et al.), and the IABP-SHOCK II trial found a neutral effect of balloon pumping in 600 randomized CS patients.

At this point the question arises, how our findings fit into the landscape of current IABP literature. So far, three large randomized studies have investigated IABP in different indications (IABP SHOCK II, CRISP-AMI, BCIS-1). The IABP shock II trial randomized CS patients undergoing primary PCI to either IABP or optimal medical therapy. Mechanical support was started after PCI in 87% of patients in the IABP arm. The primary endpoint (30-day mortality) was not different between both treatment arms, and mortality was almost identical at 12 months (Thiele et al., 2013). Among patients with acute anterior STEMI without shock, initiation of IABP before primary PCI did not reduce infarct size compared to primary PCI alone in the CRISP AMI trial, but at six months, 1.9% of patients in the IABP group and 5.2% in the PCI alone group had died ($p<0.12$) (Patel et al., 2011) . In the BCIS-1 study, balloon-pump-assisted PCI was tested against PCI without planned IABP support in patients with severe ischemic cardiomyopathy (ejection fraction 26.6 % in both arms). Elective IABP use was associated with a significant 33% mortality reduction at long term follow-up (51 month) (Perera et al., 2013). In this context, it appears that the effectiveness of

IABP in high-risk PCI is only given if the counterpulsation is active while PCI is being performed. In an earlier analysis of our shock registry, we already reported that patients with IABP-supported PCI did much better than those who received the pump after PCI (Abdel-Wahab et al., 2010). In order to understand the benefit of hemodynamic support during PCI in CS, we have to realize the risks of a complex PCI in hemodynamically compromised patient. The injection of dye, several runs of short coronary occlusions, suboptimal ventilation, analgosedative drugs etc. are significant procedural hazards. In this setting, IABP prevents hypotension and improves diastolic coronary flow, and thereby allows proper plaque preparation and stent placement. Notably, most of the CS patients have complex anatomies and need multilesion/multivessel interventions with several stents. Theoretically, left ventricular unloading may still be helpful after reopening of an infarct artery and prevent infarct expansion and ventricular remodeling, but this concept did not translate into improved survival in IABP Shock II. Most of CS patients present too late to expect relevant myocardial salvage. It is rather the quality and completeness of coronary revascularization which improves survival in CS, and this is probably the mechanism by which IABP support can provide a benefit in CS patients.

Study limitations

This study has all the limitations of a retrospective observational study. Particularly, we cannot exclude that unmeasured confounders have driven the decision to implant the IABP prior or after PCI. Moreover, the relevance of our mortality risk score has to be affirmed in a larger cohort of shock patients.

Conclusions

In conclusion, this study presents an easily applicable risk score for contemporary CS patients. In addition, it indicates that IABP can be a useful therapy in CS patients if initiated early to support the coronary revascularization procedure.

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Table 1**Baseline-characteristics of study population**

Baseline-Characteristics	
Age [years]	70.1 ± 11.0
Male	696 %
Diabetes	46.1 %
Hypertension	72.5 %
Hyperlipidemia	56.9 %
Smoker	32.4 %
pAVK	8.8 %
Atrial Fibrillation	32.4 %
Previous MI	28.4 %
Previous CABG	14.7 %
Chronic renal failure	56.9 %
Vasopressor use pre PCI	41.2 %
Resuscitation pre PCI	30.4 %
Blood Pressure sys pre PCI	102.1 ± 24.1 mmHg
Blood Pressure dias. pre PCI	59.2 ± 17.5 mmHg
Heart rate pre PCI	88.8 ± 22.4 b/min
Serum lactate	4.3 ± 4.1 mmol/l
Whithe blood cells	13.9 ± 5.2
C-reactiv protein	9.6 ± 8.4

Data are presented as n (%), mean ± SD, or median (interquartile range)

Table 2**Periprocedural characteristics of study population**

Periprocedural Characteristics	
STEMI	67.6 %
Ejection fraction	24.7 %
Culprit lesion LAD	45.1 %
Multivessel disease	85.3 %
GP IIb/IIIa inhibitor	59.8 %
Procedural duration	94.8 ± 44.2 min
IABP-Insertion after PCI	52%
Nummers of stents	2.2 ± 1.4
Length of stent	36.2 ± 24.4 mm
Vasopressor use	59.8 %
Acute renal failure	22.5 %
Dialysis	9.8 %
Creatinin Kinase _[max] (CK)	3395 ± 4901 U/l
Creatinin Kinase MB _[max] (CKMB)	323 ± 377 U/l

Data are presented as n (%), mean ± SD, or median (interquartile range)

Table 3**Outcome of study population**

Outcome	
Hospital Stay	16.5 ± 14.6 days
Mechanical Ventilation	5 ± 8.5 days
Inhospital Death	40.2 %
Cardiac Death	75.6 %
Bleeding	25.5 %
Emergency CABG	4.9 %
Cerebrovascular Events	3.9 %
Myocardial Infarction	2.0 %

Data are presented as n (%), mean ± SD, or median (interquartile range)

Table 4**Univariate predictors of in-hospital mortality**

Variable	Odds ratio	95% CI		p-value
		Lower	Upper	
IABP-Implantation after PCI	3.73	1.60	8.69	0.002
Age	1.06	1.02	1.11	0.004
Smoking	0.43	0.18	1.07	0.069
Atrial Fibrillation	3.52	1.48	8.36	0.004
Multivessel disease	5.28	1.12	24.82	0.035
Prev. CABG	3.61	1.13	11.52	0.030
Resuscitation pre-PCI	2.89	1.21	6.89	0.017
Lactate (Ln)	1.84	0.97	3.49	0.061
CK_[max] (Ln)	1.40	1.02	1.92	0.040
CK-MB_[max] (Ln)	1.88	1.27	2.78	0.002
Blood pressure dias. pre-PCI	0.97	0.94	0.99	0.009
Vasopressor pre-PCI	2.05	0.91	4.64	0.084
Vasopressor use	7.85	2.88	21.43	<0.001
Chronic renal failure	3.90	1.63	9.35	0.002
Acute renal failure	8.77	2.91	26.42	<0.001
Dialysis	7.03	1.41	35.08	0.017
Mechanical Ventilation (Ln)	1.50	1.02	2.20	0.038

Fig. 1

Forest plot of independent predictors for in-hospital mortality; OR : odds ratio; CI : confidence interval

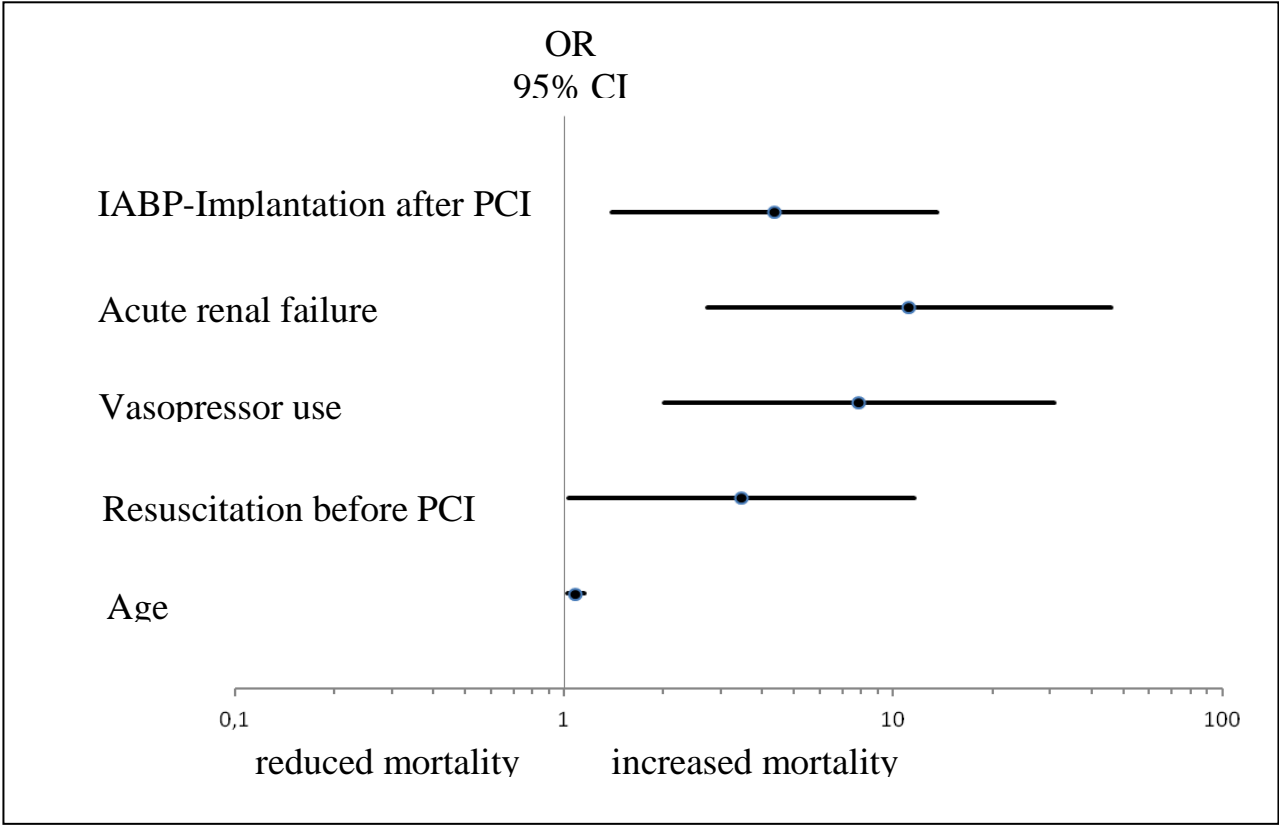


Fig. 2
Histogram of mortality score

