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CLINICAL RESEARCH

Improved outcome of cardiogenic shock at the acute stage of myocardial infarction: a report from the USIK 1995, USIC 2000, and FAST-MI French Nationwide Registries

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Aim The historical evolution of incidence and outcome of cardiogenic shock (CS) in acute myocardial infarction (AMI) patients is debated. This study compared outcomes in AMI patients from 1995 to 2005, according to the presence of CS.

Method and results Three nationwide French registries were conducted 5 years apart, using a similar methodology in consecutive patients admitted over a 1-month period. All 7531 AMI patients presenting ≤48 h of symptom onset were included. The evolution of mortality was compared in the 486 patients with CS vs. those without CS. The incidence of CS tended to decrease over time (6.9% in 1995; 5.7% in 2005, $P = 0.07$). Thirty-day mortality was considerably higher in CS patients (60.9 vs. 5.2%). Over the 10-year period, mortality decreased for both patients with (70–51%, $P = 0.003$) and without CS (9–4%, $P < 0.001$). In CS patients, the use of percutaneous coronary intervention (PCI) increased from 20 to 50% ($P < 0.001$). Time period was an independent predictor of early mortality in CS patients (OR for death, 2005 vs. 1995 = 0.45; 95% CI: 0.27–0.75, $P = 0.005$), along with age, diabetes, and smoking status. When added to the multivariate model, PCI was associated with decreased mortality (OR = 0.38; 95% CI: 0.24–0.58, $P < 0.001$). In propensity-score-matched cohorts, CS patients with PCI had a significantly higher survival.

Conclusions Cardiogenic shock remains a clinical concern, although early mortality has decreased. Improved survival is concomitant with a broader use of PCI and recommended medications at the acute stage. Beyond the acute stage, however, 1-year survival has remained unchanged.

Keywords Cardiogenic Shock • Myocardial infarction • Percutaneous coronary intervention • Epidemiology

Introduction

Cardiogenic shock (CS) is the leading cause of hospital mortality associated with acute myocardial infarction (AMI). Its prevalence varies from 5 to 15%.[1–7] Despite the many recent advances in the prevention and management of AMI, however, the reported historic trends in the prevalence of CS in AMI patients are discrepant.[8–13] Although reperfusion treatment in ST-segment elevation

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myocardial infarction (STEMI) improves prognosis, mortality remains very high, close to 50% in most recent studies.[14–17]

USIK 1995, USIC 2000, and FAST MI are three prospective observational studies conducted in 1995, 2000, and 2005 in a large number of the French centres taking care of AMI patients. Each survey assessed the characteristics, management, and outcome of patients admitted to hospital with AMI over a 1-month period.[18–21]

The aim of the present study is to analyse factors related to the occurrence of CS, as well as trends in the prevalence, management, and short- and long-term outcomes over the past 10 years.

Methods

Study

All patients of the three nationwide French registries USIK (1995), USIC 2000, and FAST-MI (2005) were included ($n = 7531$).

Methods of these registries have been described in detail elsewhere.[18–21] Briefly, the primary objectives were to evaluate MI management in 'real-life' practice and to assess short- and long-term outcomes of patients admitted to intensive care units (ICUs) for MI. Patients were recruited consecutively from ICU departments over a period of 1 month (November 1995 and 2000 and October 2005). Participation in the study was offered to all French institutions, university teaching hospitals, general and regional hospitals, and private clinics with ICUs authorized to receive acute coronary syndrome (ACS) emergencies. In each centre, a physician was in charge of the registry and provided a full list of all patients admitted to the unit. The number of participating centres was 312 in 1995, 369 in 2000, 223 in 2005, and, respectively, 2152, 2320, and 3059 patients were included. The percentage of participating centres compared with all centres taking care of AMI patients in France was 74% in 1995, 83% in 2000, and 60% in 2005.

Inclusion criteria were (i) men or women, >18 years old; (ii) patients admitted within 48 h after symptom onset in an ICU for an AMI[22,23] characterized by increased troponin or creatine kinase-MB associated with at least one of the following elements: symptoms compatible with myocardial ischaemia, appearance of pathological Q-waves, or ST-T changes compatible with myocardial ischaemia (ST-segment elevation or depression, T-wave inversion); and (iii) consent to take part in the study. Patients who died very soon after admission and for whom cardiac markers were not measured were included if they had compatible signs or symptoms associated with typical ST-segment changes.

Exclusion criteria were (i) refusal to participate, (ii) MI admitted >48 h after symptom onset, (iii) iatrogenic MIs, defined as MIs occurring within 48 h of a therapeutic procedure (bypass surgery, coronary angioplasty, or any other medical or surgical intervention), (iv) ACS diagnosis invalidated in favour of another diagnosis, and (v) patients with unstable angina and no increase in cardiac biomarkers.

The study was conducted in compliance with good clinical practice, French law, and the French data protection law. The data file of the three studies were declared to and authorized by the French data protection committee (Commission Nationale Informatique et Liberté).

Participating in the protocol did not change the therapeutic approach of the cardiologist in any way.

Definitions

Cardiogenic shock was defined as systolic blood pressure <90 mmHg in the absence of hypovolaemia and associated with cyanosis, cold extremities, changes in mental status, persistent oliguria, or congestive heart failure.[22,23] The definition of CS remained the same during all periods studied, and was such that patients with classic signs and symptoms of this clinical syndrome would be included.

Data collection

Baseline characteristics, namely demographics (age, gender), risk factors (hypertension, body mass index >30 kg/m^2, diabetes, current smoking, hyperlipidaemia), and medical history (previous AMI, previous percutaneous coronary intervention (PCI), previous coronary artery bypass grafting, previous stroke, previous heart failure, prior peripheral arterial disease, and previous chronic renal failure), were collected prospectively and stored electronically as previously described.

Clinical complications at the time of admission (second-and third-degree atrio-ventricular block, atrial fibrillation, ventricular fibrillation, stroke, anterior location of AMI and ejection fraction) were also collected.

Also, we collected the use of cardiac procedures and the presence and type of reperfusion therapy in STEMI (with PCI or thrombolysis). In 2000 and 2005, the timing of fibrinolytic treatment (pre-hospital or in-hospital), and the use of inotropes, intra-aortic balloon pump, and other haemodynamic support were collected. Medications (antiplatelet agents, diuretics, beta-blockers, ACE-inhibitors, and lipid-lowering agents) used in the first 48 h and at hospital discharge in early CS survivors were recorded.

Outcome

Mortality was assessed at 30 days and 1 year for each cohort (1995, 2000, and 2005), both in patients with and without CS. The one-year follow-up was obtained directly by the physician in charge of the study in each centre for the 1995 and 2000 surveys. For the 2005 survey, the 1-year follow-up was centralized at the French Society of Cardiology and dedicated research technicians contacted both the physicians and the patients themselves, after checking the patients' vital status in municipal registers. The rate of patients lost to the follow-up at 1-year was 2.7% in 1995, 8.3% in 2000, and 0.3% in 2005.

Statistical analysis

Statistical analysis was performed using IBM SPSS 20.0 (IBM SPSS, Inc., Chicago, IL, USA). For quantitative variables, mean and standard deviations were calculated. Discrete variables are presented as percentages. Comparisons were performed with the χ^2 or Fisher's exact test for discrete variables and by the unpaired *t*-test, the Wilcoxon sign-rank test, or the analysis of variance for continuous variables. When comparing continuous variables between the three periods, we used the analysis of variance followed by Student's *t*-tests comparing 1995 and 2005, when appropriate.

Odds ratios or hazard ratios are presented with their 95% confidence interval (CI).

One-year mortality was calculated using the Kaplan–Meier method and comparisons were made using log-rank tests.

Multivariate analyses of predictors of short-term outcome (30 days) were performed using stepwise multiple logistic regression analysis. Correlates of 1-year mortality were determined using a multivariate stepwise Cox backward model. Variables included in the final multivariate models were selected *ad hoc*, upon their physiological relevance and potential to be associated with outcomes; thus, we included variables likely to influence outcome negatively (age ≥75 years, history of heart failure, history of diabetes, history of prior

AMI, history of stroke, history of peripheral artery disease) or positively (history of hypertension, current smoking, fibrinolytic treatment) as well as sex, type of MI (STEMI vs. NSTEMI) and time period (1995, 2000, and 2005).

In addition, to assess the potential role of PCI in CS patients, we repeated the multivariate model including the use of PCI (model 2). We also calculated a propensity score for getting PCI in these patients, using logistic regression analysis (*c*-statistic = 0.79) and two cohorts (121 patients each) were constituted, matched on the propensity score, and their outcome was compared.

For confirmation purposes, we also used a model excluding patients dying on Day 1 (because these patients might have died before PCI could actually be performed), and a model including variables with a *P*-value < 0.15 in univariate analyses. Variance inflation factors were calculated for testing potential colinearity of the variables, and goodness of fit was assessed by Hosmer–Lemeshow tests. For all analyses, a *P* value < 0.05 was considered significant.

Results

Correlates of cardiogenic shock in the whole population

The total study sample consisted of 7531 patients with AMI, of whom 6.5% ($n = 486$) developed CS. The mean age was 66 ± 14 years and 29.4% ($n = 2025$) were women. Detailed results are shown in *Table 1*.

Patients who developed CS were significantly older and were more likely to be women, or to have a history of diabetes mellitus, heart failure, myocardial infarction, stroke, peripheral arterial disease, or renal failure. They were also more likely to have a STEMI and clinical concurrent complications at admission (second- and third-degree block, atrial fibrillation, ventricular fibrillation, stroke, anterior MI, and ejection fraction <40%) compared with patients who did not develop CS. Conversely, patients with CS were significantly less often smokers and had less often known hyperlipidaemia.

Patients who developed CS during hospitalization for AMI were significantly less likely to be treated with aspirin and lipid-lowering agents and more likely to get diuretics during hospitalization. Fewer patients with CS were treated with fibrinolytic therapy or PCI. Intra-aortic balloon pump (IABP) was used more often (*Table 2*).

Trends in the prevalence, characteristics, and management of cardiogenic shock patients

The prevalence of CS tended to decrease from 1995 to 2005 (6.9% in 1995; 7% in 2000, 5.7% in 2005, $P = 0.07$).

Over the 10-year period, the characteristics of AMI patients with CS remained unchanged, although the proportion of STEMI was lower in 2005, reflecting the higher proportion of patients with NSTEMI after the widespread use of troponin measurements (*Table 3*). The use of aspirin, lipid-lowering agents, and beta-blockers increased markedly over time in patients with or without CS. The incidence of atrial fibrillation decreased in patients without CS, whereas it remained constant in the CS patients. The use of fibrinolytic therapy declined considerably in patients with CS, whereas the use of PCI increased markedly. The use of inotropes and haemodynamic support in CS patients did not differ between 2000 and 2005. Inotropes were used in 54% of the patients with CS in 2000 and 49% in 2005. Intra-aortic balloon pump was used in 20 and 15%, respectively. Finally, other types of ventricular-assist devices were used in 2.5 and 1%, respectively.

The use of recommended medications at hospital discharge increased notably over the study period (e.g. in CS survivors, lipid-lowering use increased from 9% in 1995 to 82% in 2005) (*Table 4*).

Trends in outcomes

Thirty-day mortality in the whole population

Overall, 60.9% of patients with AMI who developed CS died during the first month compared with 5.2% of patients who did not develop CS ($P < 0.001$).

Over the 10-year period, mortality decreased for both patients with (70–51%, $P = 0.003$) and without CS (9–4%, $P < 0.001$) (*Figure 1*). Thirty-day mortality in CS patients decreased both in patients ≥75 years of age (from 83.5 to 68%, $P = 0.03$) and in younger patients (from 54 to 32%, $P = 0.006$). Using multivariate analysis in the whole population, both the time period and the presence of CS were independent predictors of 30-day mortality.

Thirty-day mortality in cardiogenic shock patients

Patients who survived an episode of CS were significantly younger, were less likely to have a history of diabetes mellitus, and were more likely to smoke than patients who died of CS (*Table 5*).

In CS patients, time period was an independent predictor of mortality (odds ratio for 30-day death, 2005 vs. 1995 = 0.45; 95% CI: 0.27–0.75; $P = 0.002$).When the use of PCI was added to the multivariate model, however, PCI was associated with decreased mortality (odds ratio = 0.38; 95% CI: 0.24–0.58, $P < 0.001$) and time period was no longer significant. Repeating model 2 after excluding patients dying on Day 1 yielded similar results (odds ratio: 0.43, 95% CI: 0.27–0.69).

When using the multivariate models including all variables with a *P*-value < 0.15 on univariate analyses instead of *ad hoc* variables, the time period remained significantly associated with 30-day mortality even when PCI was added to the model (Supplementary material online, *Table S1*).

In the propensity-score-matched cohorts (Supplementary material online, *Table S2*), 30-day mortality was 48% in patients with PCI, vs. 66% in those without ($P = 0.004$).

One-year survival in cardiogenic shock patients

One-year survival was 37% in 2005, compared with 24.5% in 2000 and 25% in 1995. Cox multivariate analysis identified the historical period as an independent predictor of survival, both when PCI was (hazard ratio for 2005 compared with 1995: 0.67, 95% CI: 0.50–0.92; for 2000 compared with 1995: 0.81, 95% CI: 0.61–1.08) or was not [hazard ratio for 2005 compared with 1995: 0.58, 95% CI: (0.43–0.77), for 2000 compared with 1995: 0.72, 95% CI: 0.37–1.06], included into the multivariate model. Using the multivariate models including all variables with a *P*-value < 0.15 on univariate analyses instead of *ad hoc* variables yielded similar results (Supplementary material online, *Table S1*).

Table 1 Characteristics of patients according to the presence of cardiogenic shock

Characteristics	Whole cohort (n = 7531)	Shock present (n = 486)	Shock absent (n = 7045)	P-value
Age (years) mean ± SD	66 ± 14	74 ± 12	66 ± 14	<0.001
Age >75 years, n (%)	2429 (32)	268 (55)	2161 (31)	<0.001
Female, n (%)	2215 (29)	190 (39)	2025 (29)	<0.001
STEMI, n (%)	4979 (67)	344 (71)	4635 (66)	0.02
Pre-existing conditions, n (%)				
Body mass index >30 kg/m^2	1214 (18)	52 (14)	1162 (18)	0.051
Diabetes mellitus	1575 (21)	132 (27)	1443 (21)	0.001
Hypertension	3806 (51)	263 (55)	3543 (50)	0.09
Treated hyperlipidaemia	3147 (42)	160 (34)	2987 (43)	<0.001
Smoking current	2324 (31)	89 (19)	2235 (32)	<0.001
Prior myocardial infarction	1330 (18)	112 (23)	1218 (17)	0.002
Prior PCI[a]	616 (12)	34 (10)	582 (12)	0.48
Prior heart failure	520 (7)	80 (17)	440 (6)	<0.001
Prior stroke	483 (6)	52 (11)	431 (6)	<0.001
Prior peripheral arterial disease	718 (10)	77 (16)	641 (9)	<0.001
Prior coronary artery bypass	264 (5)[a]	18 (5)	246 (5)	0.70
Renal failure	271 (5)[a]	39 (12)	232 (5)	<0.001
Clinical complications, n (%)				
Recurrent MI[a]	113 (2)	18 (5)	95 (2)	<0.001
Second-and third-degree heart block	302 (4)	81 (17)	221 (3)	<0.001
Atrial fibrillation	629 (8)	92 (19)	537 (8)	<0.001
Ventricular fibrillation	232 (3)	69 (14)	163 (2)	<0.001
Ejection fraction <40%[b]	1217 (21)	175 (63)	1042 (19)	<0.001
Anterior necrosis	2130 (28)	190 (39)	1940 (28)	<0.001
Stroke[a]	51 (1)	12 (4)	39 (1)	<0.001

SD, standard deviation; MI, myocardial infarction; PCI, percutaneous coronary intervention.
[a]Data not available in 1995.
[b]Data not available in 23% of the population.

Table 2 In-hospital management according to the presence of cardiogenic shock

	Whole cohort (n = 7531)	Shock present (n = 486)	Shock absent (n = 7045)	P-value
Medications, n (%)				
Aspirin	7078 (94)	397 (82)	6681 (95)	<0.001
Lipid-lowering agents	3541 (47)	128 (26)	3413 (48)	<0.001
Diuretics	2383 (32)	332 (68)	2051 (29)	<0.001
Procedures, n (%)				
PCI	3675 (49)	182 (37)	3493 (50)	<0.001
Reperfusion therapy in STEMI patients[a]				
Intravenous fibrinolysis		117 (24)	(32)	0.005
Primary PCI		117 (24)	(23)	
Pre-hospital thrombolysis	1550 (21)	75 (15)	1475 (21)	0.006
In-hospital thrombolysis	171 (2)	16 (3)	156 (2)	0.004
Intra-aortic balloon pump	94 (2)[b]	56 (17)	38 (1)	<0.001

PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.
[a]By thrombolysis or PCI.
[b]Not available in 1995.

Table 3 Trends characteristics and management of acute myocardial infarction patients with cardiogenic shock

Characteristics	1995		2000		2005		*P*-value*	
	No CS (*n* = 2004)	CS (*n* = 148)	No CS (*n* = 2157)	CS (*n* = 163)	No CS (*n* = 2884)	CS (*n* = 175)	No CS	CS
Age, mean, years	66 ± 14	74 ± 12	65 ± 14	75 ± 12	67 ± 14	74 ± 13	<0.001	0.88
Female, *n* (%)	545 (28)	65 (44)	570 (26)	58 (36)	901 (31)	67 (38)	0.003	0.26
STEMI, *n* (%)	1422 (72)	114 (79)	1714 (80)	130 (80)	1499 (52)	100 (57)	<0.001	<0.001
Medical history and risk factors, *n* (%)								
Body mass index ≥30 kg/m^2, *n* (%)	265 (14)	15 (12)	346 (18)	16 (14)	1157 (18)	21 (17)	0.001	0.28
Diabetes mellitus, *n* (%)	324 (17)	32 (22)	434 (20)	53 (33)	677 (24)	47 (27)	<0.001	0.43
Hypertension, *n* (%)	881 (45)	76 (53)	994 (46)	82 (50)	1650 (57)	104 (59)	<0.001	0.23
Hypercholesterolaemia, *n* (%)	703 (37)	36 (25.5)	895 (42)	49 (31)	1373 (48)	75 (43)	<0.001	0.001
Current smoking, *n* (%)	619 (32)	24 (17)	729 (34)	25 (16)	882 (31)	40 (23)	0.25	0.17
Prior myocardial infarction, *n* (%)	339 (17)	42 (28)	375 (17)	36 (22)	491 (17)	34 (19)	0.83	0.05
Prior heart failure, *n* (%)	159 (8)	30 (21)	119 (5.5)	30 (18)	153 (5)	20 (12)	<0.001	0.02
Management, *n* (%)								
Antiplatelet agents	1829 (93)	103 (71)	2065 (96)	139 (85)	2756 (96)	153 (87)	<0.001	<0.001
Lipid-lowering agents	202 (10)	6 (4)	1020 (47)	25 (15)	2186 (76)	97 (55)	<0.001	<0.001
Diuretics	641 (33)	107 (72)	514 (24)	99 (61)	876 (30)	126 (72)	0.33	0.95
Beta-blockers	1343 (67)	30 (20)	1608 (74.5)	46 (28)	2049 (71)	68 (39)	0.009	<0.001
ACE-inhibitors	946 (47)	46 (31)	868 (40)	40 (24.5)	1342 (46.5)	67 (38)	0.96	0.134
Procedures, *n* %								
Reperfusion therapy in STEMI		45/114 (39.5)		58/130 (45)		63/100 (63)		<0.001
Fibrinolysis in STEMI patients		31/114 (27)		25/130 (19)		28/100 (28)		0.95
Pre-hospital fibrinolysis		NA		5/130 (4)		13/100 (13)		0.01
PCI during the hospital stay	342 (17)	30 (20)	1276 (59)	65 (40)	1870 (65)	87 (50)	<0.001	<0.001
Intra-aortic balloon pump		NA	25 (1)	32 (20)	13 (0.5)	24 (14)	0.23	0.58
Concomitant complications								
Atrial fibrillation	228 (12)	27 (19)	162 (8)	32 (20)	142 (5)	33 (19)	<0.001	0.97
Ventricular fibrillation	61 (3)	21 (14.5)	52 (2)	29 (18)	48 (2)	19 (11)	0.001	0.31
AV block	107 (5)	34 (23)	77 (4)	30 (18.5)	36 (1)	15 (9)	<0.001	<0.001
Reinfarction	50 (2)	7 (4)	45 (2)	11 (6)	95 (2)	18 (5)	0.05	0.42
Stroke	17 (0.8)	5 (3)	22 (0.8)	7 (4)	39 (0.8)	12 (4)	0.91	0.65

ACE, angiotensin-converting enzyme; AV, atrio-ventricular; NA, not available; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.
**P*-values are *P* for trends by period using χ^2 tests for discrete variables. For continuous variables, *P*-values are for two-way analysis of variance.

In the propensity-score-matched cohorts, 1-year survival was 22% in the cohort, which did not get PCI, vs. 40.5% in the cohort with PCI ($P = 0.001$) (*Figure 2*).

In patients surviving to 1 month, however, late survival at 1 year did not improve over time (1995: 82%; 2000: 67%; and 2005: 76.5%).

Discussion

The results of these three nationwide registries implemented 5 years apart provide insights into changing trends in the management, and hospital outcomes of patients with CS complicating AMI. Although the prevalence of CS tended to decrease, the main baseline characteristics of the patients with shock remained essentially similar. In contrast, the use of reperfusion therapy in STEMI patients with shock increased from <40–63% in the latter period; likewise, the use of PCI at any time during the hospital stay more than doubled (from 20 to 50%). In addition, medical therapy also evolved with more patients receiving early treatment with aspirin, beta-blockers, and lipid-lowering agents in the most recent period. Early mortality decreased in relation with a higher use of PCI at the acute stage. In contrast, mortality beyond the first month remained constant. Overall, 1-year survival increased by 48% over this 10-year period.

Incidence of cardiogenic shock

The overall rate of CS after AMI observed in the present study is 6.5% and is in agreement with the rates of CS described in previously published studies, ranging from 5 to 15%.[1–13] This relatively wide range reflects the various definitions of AMI and CS used, the use of representative as opposed to more highly selected patient samples, the time periods under study, and the use of therapeutic options that may reduce the risk of CS.

The previous studies that have examined changing trends in the incidence of CS after AMI have yielded conflicting results.[8–13] In the study conducted by Fang *et al.*,[8] using the National Hospital Discharge Survey data, declines in the frequency of CS were observed between 1979 and 2003. Goldberg *et al.*[9] reported a decrease in the incidence of CS between the late 1990s (7.5%) and (4.1%) in the residents of the Worcester metropolitan area hospitalized for AMI. Similar results were reported in the GRACE multinational cohort study conducted between 1990 and 2006.[10]

Table 4 Discharge medications in patients with cardiogenic shock who survived the acute phase

Medications, *n* (%)	1995 (*n* = 45)	2000 (*n* = 60)	2005 (*n* = 85)	*P* for trend
Antiplatelet agents	36 (80)	52 (87)	71 (83.5)	0.71
Beta-blockers	12 (27)	26 (43)	51 (60)	<0.001
ACE-inhibitors	25 (56)	35 (58)	53 (62)	0.44
Lipid-lowering agents	4 (9)	21 (35)	55 (65)	<0.001

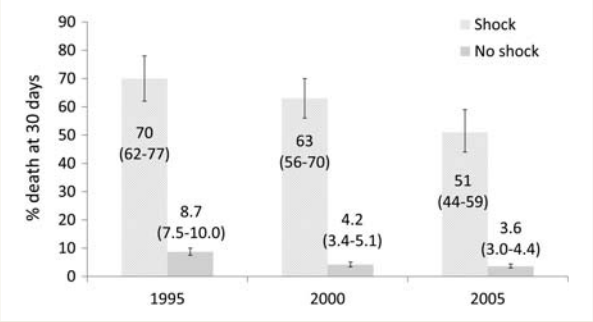

Figure 1 Trends in thirty-day mortality in patients with acute myocardial infarction according to the presence of cardiogenic shock.

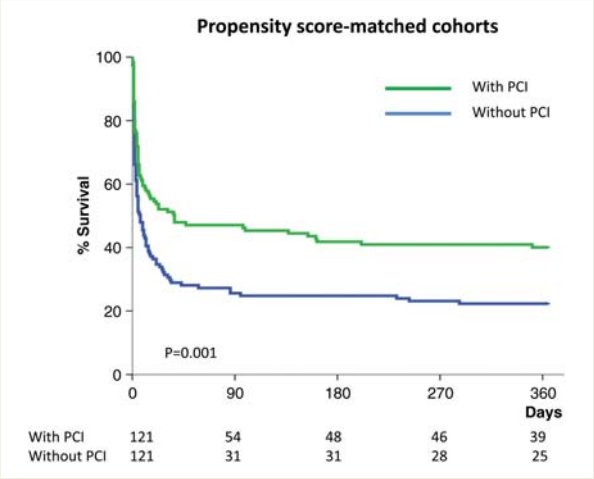

Figure 2 One-year survival in patients with cardiogenic shock matched on a propensity score for getting percutaneous coronary intervention during the hospital stay.

Table 5 Independent predictors of 30-day mortality for myocardial infarction complicated by cardiogenic shock

Variable	Model 1, odd ratio (95% CI)	*P*-value	Model 2[a], odds ratio (95% CI)	*P*-value
Time period (reference 1995)				
2000	0.63 (0.37–1.06)	0.08	—	
2005	0.45 (0.27–0.75)	0.002		
Age ≥75 years	2.93 (1.88–4.56)	<0.001	2.46 (1.56–3.88)	<0.001
Diabetes	1.96 (1.21–3.19)	0.007	1.88 (1.15–3.06)	0.01
Current smoking	0.55 (0.32–0.96)	0.035	0.54 (0.31–0.95)	0.03
History of hypertension	0.62 (0.40–0.96)	0.03	0.60 (0.38–0.93)	0.02
Type of MI (reference: anterior STEMI)				
Non-anterior STEMI	—		0.72 (0.44–1.20)	0.21
NSTEMI			0.46 (0.27–0.78)	0.004
PCI	—		0.38 (0.24–0.58)	<0.001
Hosmer–Lemeshow *P*-value	0.17		0.97	

AMI, acute myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.
[a]Model 2 includes the use of PCI during the initial hospital stay.

Likewise, Abdel-Qadir *et al.*[11] described a significant decline in the incidence of CS in the Ontario Myocardial Infarction Database between 1992 and 2008.

In contrast, findings from the National Registry of Myocardial Infarction showed either stable or slightly increasing incidence rates of CS over the 10-year period beginning in 1995 in patients hospitalized with ST-segment elevation MI.[12] The results of our study suggest that patients hospitalized with AMI in 2005 were only slightly less likely to develop CS than patients hospitalized 10 years before (6.9% in 1995; 5.7% in 2005).

In a Swiss national registry of 23 060 patients with ACS s admitted from 1997 to 2006, Jeger *et al.*[13] showed different trends in the incidence of CS, depending on its timing: a stable incidence of CS on admission and a decreasing incidence of CS occurring after hospital admission. In our study, the timing of CS was not recorded in 1995. From 2000 to 2005, the percentage of shock on admission decreased from 2.6 to 1.7%, as did the percentage of shock developing after admission (4.4 to 4.1%).

Finally, the incidence of CS tended to decrease both in patients $\geq$75 years of age (from 11.6 to 9.0%) and in younger patients (from 4.7 to 4.0%).

Management

In current guidelines, AMI complicated by CS is listed as a class IA indication for PCI and a class IA indication for coronary artery bypass graft surgery if the patient has suitable coronary anatomy.[22,23] There is evidence that aggressive intervention may result in improved survival rates among patients in whom CS has developed.[24,25]

The use of PCI among our patients with shock was comparatively low: in 2005, PCI was performed in 50% of the patients. Although this rate is lower than what might be expected on a theoretical basis, it is actually higher than the rates reported in most observational cohorts.[5,12,26] Coronary angiography was done in a greater percentage of the patients (63%). Of note, 55% of our patients were 75 years of age or older; older age is associated with a lower use of invasive procedures in all registries and, following the results of the Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) trial, the usefulness of PCI in elderly patients CS has been debated.[5,27] Since 2005, the overall use of PCI in AMI has continued to increase in France and was 77% in FAST-MI 2010.[28]

Contrary to the use of PCI, there was no increase in the use of intra-aortic balloon pump or ventricular-assist devices, which may reflect the relatively poor level of evidence supporting the use of these devices, although the European Society of Cardiology STEMI guidelines consider that IABP should be used in patients with CS (IC recommendation).[23] Likewise, the use of inotropes remained stable (~50%). These figures are in line with those found for CS patients in the SWISS registry,[13] and therefore seem to adequately reflect the real-world practice in Western Europe at that time. The use of diuretics also remained stable.

In contrast, there was an increase in the early prescription of statins and antiplatelet agents, which paralleled the prescription pattern in patients without CS. Finally, the prescription of beta-blockers and ACE-inhibitors was concordant with that in the SWISS registry[13] and also increased over time.

Outcome

Although CS remains a most severe condition, survival improved notably during this 10-year period. This improvement (+48%) was associated with a 150% increase in the use of PCI. Numerous studies suggest that PCI improves short-term survival in patients with CS, with survival contingent on the successful establishment of coronary reperfusion.[5,8–17,25] In all studies which assessed changing trends in the incidence rates and the outcome of CS after AMI, an increase was found in the use of PCI with a significant decline in short-term mortality associated with CS.[8–13] Also supporting the role of PCI was the fact that mortality decreased to a greater extent in patients with CS on admission (2000: 60%; 2005: 40%) in whom PCI use increased from 43 to 59%, compared with those in whom CS developed later during the hospital course (2000: 65%; 2005: 56%), in whom the increase in PCI use was less (38–46%).

It must be kept in mind, however, that the improvement in early mortality may also have been related to the use of other recommended measures and to the global management of the patients, as suggested by the fact that 1-year mortality significantly decreased according to the time period, independently of the use of PCI.

In contrast with the marked decline in early mortality, mortality from 1 month to 1 year remained high (>20%) and did not improve over time, in spite of the higher use of recommended medications at hospital discharge over the study period (e.g. in CS survivors, beta-blockers at discharge increased from 27% in 1995 to 60% in 2005). Further studies in this regard will be needed, in particular as regards the long-term (beyond 1 year) outcome of early survivors of CS.

Strengths and limitations

All three registries were performed using a similar methodology at institutions representing a vast majority of those taking care of patients with AMI in France during the study period.

In this registry, as in all registries, there was no independent review of source documents to confirm the diagnosis of CS. However, the reported rate of CS was in agreement with the incidence rates of CS described in previously published studies, supporting the validity of the diagnosis.

The clinical definition of CS did not include any time frame. In the 2000 and 2005 registries, however, we separated patients with CS on admission and those in whom CS developed later. For the purpose of the historic comparison, we therefore considered as CS patients those developing CS at any time during the hospital stay. Regarding correlations with long-term outcomes, we had no information on implantable cardioverter defibrillator (ICD) implantation in the 1995 and 2000 registries. In 2005, very few patients had an ICD implanted during their hospital stay (one in the CS group, 0.6%; and six in the patients without CS, 0.2%), and we have no information to date on subsequent implantation of ICDs.

Finally, as in all observational studies, we are only able to describe associations and not causality, between baseline characteristics, management strategies, and outcomes.

Conclusions

In these three nationwide surveys conducted over a period of 10 years, CS remains a clinical concern, particularly in elderly patients. There is, however, a favourable trend regarding early survival, translating into an improved long-term outcome. Improved survival is concomitant with a broader use of PCI and recommended medications at the acute stage. Although our results cannot prove any causal relationship, they suggest that a more aggressive management (both in terms of early revascularization and medications) is particularly warranted in these patients, including those over the age of 75 years.

Supplementary material

Supplementary material is available at *European Heart Journal* online.

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CARDIOVASCULAR FLASHLIGHT

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Acute myocardial infarction and swinging heart: not always a cardiac rupture—haemopericardium due to infiltrative lung cancer

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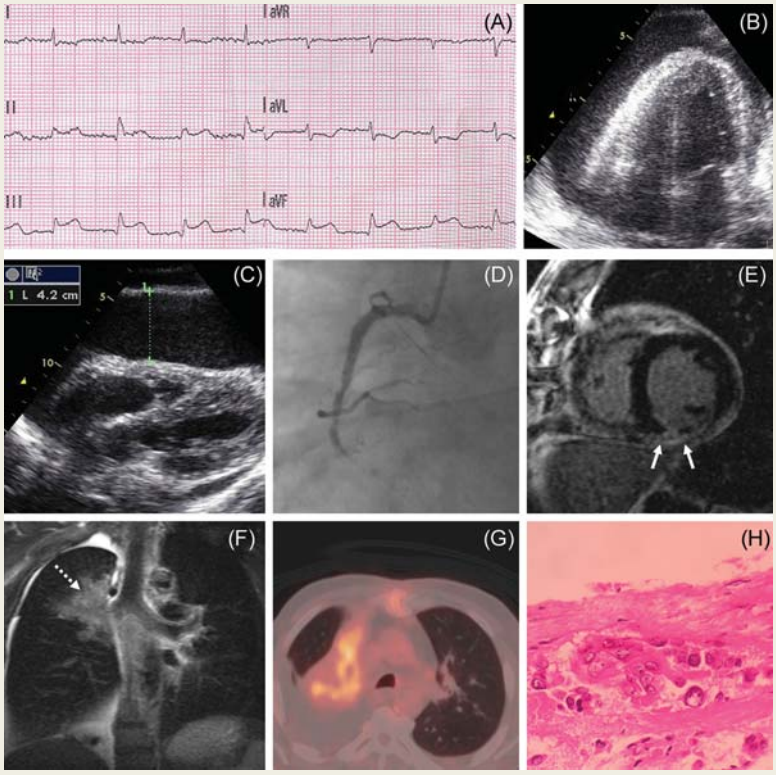
A 56-year-old diabetic patient was admitted with oppressive chest pain, hypotension, and ST-segment elevation on inferior leads (*Panel A*). The transthoracic echocardiogram carried out in the emergency room revealed a severe pericardial effusion with a swinging heart (*Panels B* and *C*, Supplementary material online, *Video S1*). After being diagnosed with acute myocardial infarction, the patient underwent urgent coronariography, which confirmed thrombotic occlusion of the right coronary artery (*Panel D*, Supplementary material online, *Video S2*). During the procedure, the patient developed progressive cardiogenic shock and was referred for emergency cardiovascular surgery with a presumptive diagnosis of cardiac rupture. A large haemopericardium was drained through an open sternotomy, but there was no evidence of myocardial rupture. To verify the integrity of the myocardial wall, a contrast-enhanced cardiac magnetic resonance was carried out, which confirmed an inferior necrosis area (*Panel E*, arrows indicate the region of late gadolinium enhancement) and revealed a right lobar mass infiltrating the pericardium (*Panel F*, dotted arrow). Subsequently, the thoracic positron emission tomography-computerized tomography confirmed the infiltrative and hypermetabolic profile of the tumour (*Panel G*). Finally, a bronchoscopic biopsy diagnosed an adenocarcinoma infiltrating the right upper bronchus (*Panel H*, haematoxylin-eosin stain).



Supplementary material is available at *European Heart Journal* online.