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ORIGINAL ARTICLE

Predictors of 30-day mortality and outcome in cases of myocardial infarction with cardiogenic shock treated by extracorporeal life support

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Abstract

OBJECTIVES: The twin aims of this study were to identify the independent predictors of 30-day mortality and to analyse the outcomes of patients with cardiogenic shock (CS) associated with acute myocardial infarction (AMI) and necessitating extracorporeal life support (ECLS).

METHODS: The investigation was a single-centre, retrospective study of 77 patients who required ECLS for AMI with CS. A logistic regression analysis was performed to identify the independent variables associated with 30-day mortality.

RESULTS: Between February 2006 and November 2009, 745 patients in our establishment received ECLS. In the single-centre cohort, we retrospectively reviewed 77 patients who had required ECLS support for AMI with CS. The delay between AMI and CS ECLS was 15 ± 4 h. PCI was performed in 58 patients (75.3%) and isolated emergency CABG in 12 (15.6%). The remaining 7 patients (9.1%) did not undergo revascularization. ECLS duration averaged 9.8 ± 7.1 days. Nineteen patients were successfully weaned from ECLS (24%). Fifty-eight patients did not undergo or did not tolerate the weaning trial (76%). Forty patients died during ECLS support, 13 were implanted with a mono-ventricular ($n = 9$) or biventricular assist device ($n = 4$) and 5 were bridged to heart transplantation. Complications consisted primarily in pneumonia (51.3%) and acute renal failure requiring haemofiltration (46.1%). Pulmonary oedema occurred in 24 patients (31.6%) and major bleeding in 16 (21.33%). 30-day and in-hospital survival rates were, respectively, 38.9 and 33.8%. Multivariable analysis identified preimplantation lactate serum level, preimplantation creatinine serum level and previous cardio-pulmonary resuscitation as independent predictors of 30-day mortality.

CONCLUSIONS: Prompt ECLS support is an effective strategy and provides a reasonable chance of survival in patients with AMI associated with profound CS. As shown in our results pertaining to predictive risk factors for 30-day mortality, reducing the duration of end-organ ischaemia is the keystone to management of this patient population. A major remaining challenge will consist in preventing pulmonary oedema following peripheral ECLS.

Keywords: Acute myocardial infarction • Cardiogenic shock • Extracorporeal life support

INTRODUCTION

The incidence of cardiogenic shock (CS) in patients with acute myocardial infarction (AMI) ranges from 7 to 10% [1]. Studies have shown that in patients presenting AMI with CS, medical therapy alone yields disappointing results, with in-hospital mortality rates exceeding 80% [2]. Despite aggressive treatment modalities such as coronary artery bypass graft (CABG), percutaneous coronary interventions (PCI) and use of an intra-aortic balloon pump (IABP), CS mortality has remained unacceptably high [1, 3]. In this context, extracorporeal life support (ECLS) should be considered as a means of rescuing patients with refractory CS [4, 5]. It has already been successfully used as a bridge to myocardial recovery, cardiac transplantation or implantation of a ventricular assist device (VAD) in cases of AMI with CS [6]. Although IABP is one of the most commonly utilized mechanical devices for CS [7–9], it provides only limited cardiac support and has been shown to be ineffective in the clinical setting of profound CS [8]. ECLS, on the other hand, allows for immediate and adequate systemic circulation and oxygenation. As a procedure, it is also much simpler, less expensive and more rapidly completed than left VAD [6, 10]. Little information is available on the efficacy of venoarterial ECLS in the management of AMI complicated by CS unresponsive to conventional treatment.

The aim of this single-centre study was to analyse the outcome of patients with CS related to AMI necessitating ECLS and to identify the independent predictors of 30-day mortality.

METHODS

Characteristics of study patients

Between February 2006 and November 2009, 745 patients in our establishment received ECLS.

In the single-centre cohort, we retrospectively reviewed 77 patients who had required ECLS support for AMI with CS. Indications for ECLS support were applicable to all patients having experienced profound CS related to AMI refractory to conventional therapy, including a maximal dose of vasopressor agent and/or IABP support and aortic time–velocity integral (VTI) <10 cm.

Procedure and management of ECLS

The ECLS system consists mainly of a heparin-bound centrifugal pump and a hollow-fibre microporous membrane oxygenator (Quadrox Bioline, Jostra-Maquet, Orléans, France). The entire system and all the instruments allowing for vascular access are installed on a mobile cart so as to facilitate prompt transportation within hospital facilities, including the catheterization room, emergency room, intensive care unit and operating room. In some cases, the Mobile Unit of Cardiac Assistance (MUCA) has been able to initiate and manage circulatory support in care facilities that do not host local circulatory support teams. The ECLS system is quickly set up through the femoral venoarterial route by accurate surgical cutdown and percutaneous puncture. The tip of the arterial cannula (15 or 17 Fr) is set at the aorto-iliac junction, while the tip of the venous cannula (27 or 29 Fr) is set at the junction between the inferior vena cava and the right atrium. The locations of the catheters are confirmed radiographically in the catheterization room or by estimating elsewhere in the establishment, the distance between the puncture site and right atrium. A catheter is put into place for antegrade distal limb perfusion. Heparinization is continued in order to maintain activated clotting time between 150 and 180 s in the absence of a haemorrhage. For severe haemorrhagic complications, heparinization was temporarily held for 12 h before dose adjustment. Extracorporeal blood flow was adjusted to maintain adequate systemic blood flow and oxygen supply as monitored by mean arterial pressure, urine output and plasma lactate concentrations. The doses of inotropic agents used before ECLS were progressively tapered following ECLS setup in order to reduce the left ventricular post-charge. Arterial pressure tracing was strictly monitored for reappearing pulsatile systemic blood flow, indicating residual left ventricular myocardial contractility facilitating left ventricular drainage. Dobutamine was infused to facilitate left ventricular decompression, minimizing the risks of pulmonary oedema and left ventricle blood stasis. In cases of total absence of cardiac contractility, a central cannulation was chosen and left-ventricular venting (through the pulmonary artery, the right superior pulmonary vein or the apex of the left ventricle) was implemented.

The circuit, pump head and oxygenator included, is replaced in the event of marked plasma leak, haemoglobinuria and/or blood clot formation in the circuit system. Serum cardiac enzyme levels are routinely measured and transthoracic or oesophagus echocardiography is performed daily to assess the recovery of heart function.

ECLS weaning

Successful weaning was defined as separation from ECLS without mortality over 48 h. An ECLS weaning trial was undertaken when the patient was considered haemodynamically stable, i.e. baseline mean blood pressure (MBP) >60 mmHg while receiving no or low-dose vasoactive agents and a pulsatile arterial waveform maintained for at least 24 h, and when pulmonary blood oxygenation was not compromised. The ECLS flow was decreased to 66% for 10–15 min, then to 33% and/or to a minimum of 1–1.5 l/min for another 10–15 min. If MBP dropped significantly and was constantly <60 mmHg during the trial, ECLS flow was returned to 100% of the initial flow, and the trial was discontinued. Doppler echocardiography was repeated each time ECLS flow was modified by the intensive care unit staff echocardiographist. When a patient had partially or fully recovered from the initial cardiac dysfunction, had tolerated the full weaning trial and shown left ventricular ejection fraction >20–25% and aortic VTI >10 cm with minimal support, ECLS removal was considered. If the patient remained stable after 15 min of complete-circuit clamping in the operating room, the machine was surgically removed, and the mediastinum or femoral access surgically repaired. When ECLS weaning was deemed impossible, bridging to VAD or to transplantation was considered as an alternative option.

Data collection

The following data were collected: age, gender, coronary risk factors, previous cardiac surgery, type of ECLS, peripheral femoral vs central intrathoracic; previous cardiopulmonary resuscitation (CPR), initiation under chest compressions, number of diseased vessels, Simplified Acute Physiology Score (SAPS) II; Sepsis-Related Organ Failure Assessment (SOFA) score; concomitant use of an IABP; in-hospital adverse events, weaning trial, bridge to bridge or heart transplantation and mortality (30-day and hospital mortality). Lactate, creatinine and SGOT serum levels at 0, 1 and 3 days after ECLS initiation were also recorded. Haemodynamic status was assessed daily by measuring systolic (SBP), diastolic (DBP) and mean (MBP) arterial blood pressure and heart rate. Data obtained from medical records, clinical case histories and laboratory investigations were retrospectively reviewed.

Statistical analysis

Descriptive statistics used mean ± standard deviation for quantitative variables and numbers and percentages for qualitative ones. The predictive score for 30-day mortality was built using three steps. In the first step, univariate statistical analysis was performed in order to select variables linked to 30-day mortality. This step used χ^2 tests for qualitative variables and two-sample Wilcoxon tests for quantitative ones. Variables with a P-value <0.05 in the first step were then entered into a stepwise logistic

regression in the second step. Variables with $P < 0.05$ by the Wald test were retained in the final logistic model. In the third step, the logistic model was evaluated: calibration was tested using the Hosmer and Lemeshow method, and discriminant power was evaluated by the area under the receiver operating characteristic (ROC) curve. The mean standard error for predicted probability of death was computed by nonparametric Boostrap estimation. Ten thousand bootstrap samples were randomly generated, model parameters of the logistic model were estimated for each, and probabilities of death were estimated for each sample and each subject. The standard errors for probability of death were then computed for each subject, and the weighted mean of the standard errors is the final estimate of the variability of the final result of the logistic formula. All the tests were two sided, with a *P*-value <0.05 considered as significant. Computations were performed using SAS V9, including the Bootstrap analysis, except for the ROC curve which was built using the XLSTAT software.

Ethics

The study was carried out according to the principles outlined in the Helsinki declaration of 1975 and in agreement with French laws on biomedical research. Because of the retrospective profile of the analysis, this study was not submitted to the approval of our ethical committee board (Comité de Protection des Personnes se Prêtant à la Recherche Biomédicale, CCPPRB Pitié-Salpêtrière, Paris, France). Informed consent for demographic, physiological and hospital outcome data analyses was not obtained, because this observational study did not modify existing diagnostic or therapeutic strategies.

RESULTS

Characteristics of study patients

Table 1 shows the baseline characteristics of 77 patients with a mean age of 56.1 years. The population was male-predominant, with current smoking as the most frequent cardiovascular risk factor. In 12 cases, ECLS was implanted by our MUCA. In the other cases, ECLS was implanted in the catheterization room ($n = 13$), emergency room ($n = 5$), intensive care unit ($n = 27$) or operating room ($n = 20$). All patients needed preoperative mechanical ventilation. Delay of CS-necessitating ECLS after AMI was 15 ± 4 h. Prior to ECLS implantation, 31 patients (40.3%) had undergone cardiopulmonary resuscitation over the previous 24 h, more than half of the patients were on IABP (55.8%) and, in 14 (18.2%) cases, ECLS was implanted under cardiac massage. Left ventricular ejection fraction was 17 ± 7% and MBP was 52.4 ± 14.10 mmHg. Twenty-four (24%) patients were treated with a single drug, and 40 (76%) with a combination of two drugs. Intravenous drugs included dobutamine in 53 patients (mean dose: 4.1 ± 5.6 γ/kg/min), and adrenaline in 45 (mean dose: 4.7 ± 6.9 mg/h). Laboratory findings showed that the preimplantation lactate serum level was elevated, as were creatinine and glutamate-oxaloacetate transaminase serum levels. However, preimplantation prothrombin time was pronouncedly low. Mortality predicted by the SAPS 2 score was 69.40%. As for myocardial infarction, the mean peak level of troponin-I was very high (286.08 µg/l). Emergency PCI was performed in 58 patients (75.3%) and isolated emergency CABG in 12 (15.6%). The remaining 7 patients (9.1%) did not undergo revascularization. A mechanical complication of AMI was detected in 5 patients (6.49%), whether by ventricular septum rupture ($n = 2$), left ventricular free wall rupture ($n = 2$) or papillary muscle rupture ($n = 1$). Three of these patients underwent surgical repair (mitral valve replacement, left ventricle free wall or ventricular septal rupture repair), and the other 2 died before arrival in the operating room (Fig. 1).

Table 1: Baseline characteristics of the 77 studied patients

Number of patients who received ECLS support (*n*)	All patients (*n* = 77)
Demographics and pre-existing comorbidity	
Age (years) (mean ± SD)	56.1 ± 0.7
Male gender [% (*n*)]	75.3 (58)
Body mass index (mean ± SD)	25.8 ± 4.8
Diabetes mellitus, [% (*n*)]	28.9 (22)
Hypertension [% (*n*)]	26.3 (20)
Current smoking [% (*n*)]	46.1 (35)
Hypercholesterolaemia [% (*n*)]	34.2 (26)
Previous cardiac surgery [% (*n*)]	5.3 (4)
ECLS implantation haemodynamics data	
Left ventricular ejection fraction (%) (mean ± SD)	17 ± 7
Mean blood pressure (mmHg) (mean ± SD)	52.4 ± 14.1
Intra-aortic balloon pump support [% (*n*)]	55.8 (43)
Previous cardiopulmonary resuscitation [% (*n*)]	40.3 (31)
ECLS implantation under cardiac massage [% (*n*)]	18.2 (14)
Inotropes at ECLS implantation	
Dobutamine (γ/kg/min) (mean ± SD)	4.1 ± 5.6
Adrenaline (mg/h) (mean ± SD)	4.7 ± 6.9
ECLS implantation laboratory	
Hb (g/dl) (mean ± SD)	10.2 ± 1.8
Platelet (10^3/µl) (mean ± SD)	215.5 ± 98.7
Creatinine (µmol/l) (mean ± SD)	158.1 ± 81.6
SGOT (UI/l) (mean ± SD)	1339.3 ± 2233.5
Prothrombin time (%) (mean ± SD)	50,1 ± 21.1
Lactate (mmol/l) (mean ± SD)	8.4 ± 4.9
Coronary disease	
Peak level of Troponin-I (µg/l) (mean ± SD)	286.1 ± 706.3
Uni-vessel stenosis [% (*n*)]	28.5 (18)
Double-vessel stenosis [% (*n*)]	38.1 (24)
Triple-vessel stenosis [% (*n*)]	33.3 (21)
Scores	
SOFA (mean ± SD)	11.5 ± 4.7
SAPS 2% (mean ± SD)	69.4 ± 22.1

ECLS: extracorporeal life support; Hb: haemoglobin; SOGT: serum glutamic oxaloacetic transaminase; SAPS: simplified acute physiology score; SOFA: sepsis-related organ failure assessment; INTERMACS: interagency registry for mechanically assisted circulatory support.

Early post-ECLS data

Fifty-nine patients were assisted with peripheral ECLS, while 18 immediately underwent central ECLS. Assistance duration averaged 9.8 ± 7.1 days. Complications consisted mostly in pneumonia and acute renal failure requiring haemofiltration (respectively, 51.3 and 46.1%). Pulmonary oedema occurred in 24 patients (31.6%), and 22 of them were supported by a peripheral venoarterial ECLS, requiring, in 8 cases, a centralization within a median of 3 (range 1–14) days postimplantation. As for IABP, the

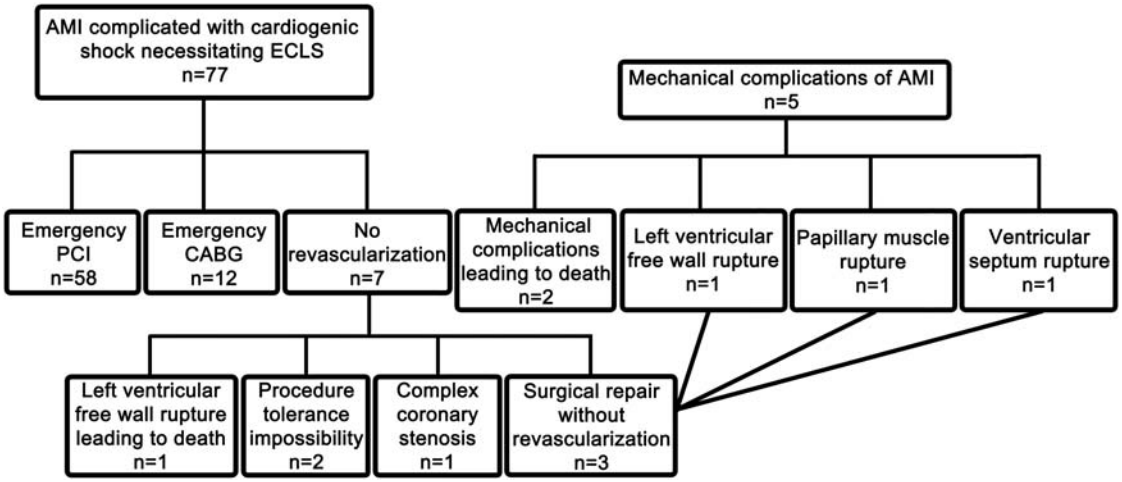

Figure 1: Revascularization and mechanical complications in patients with acute myocardial infarction (AMI) complicated with cardiogenic shock following of extracorporeal life support (ECLS). ECLS: extracorporeal life support; AMI: acute myocardial infarction; PCI: percutaneous coronary intervention; CABG: coronary artery bypass grafting.

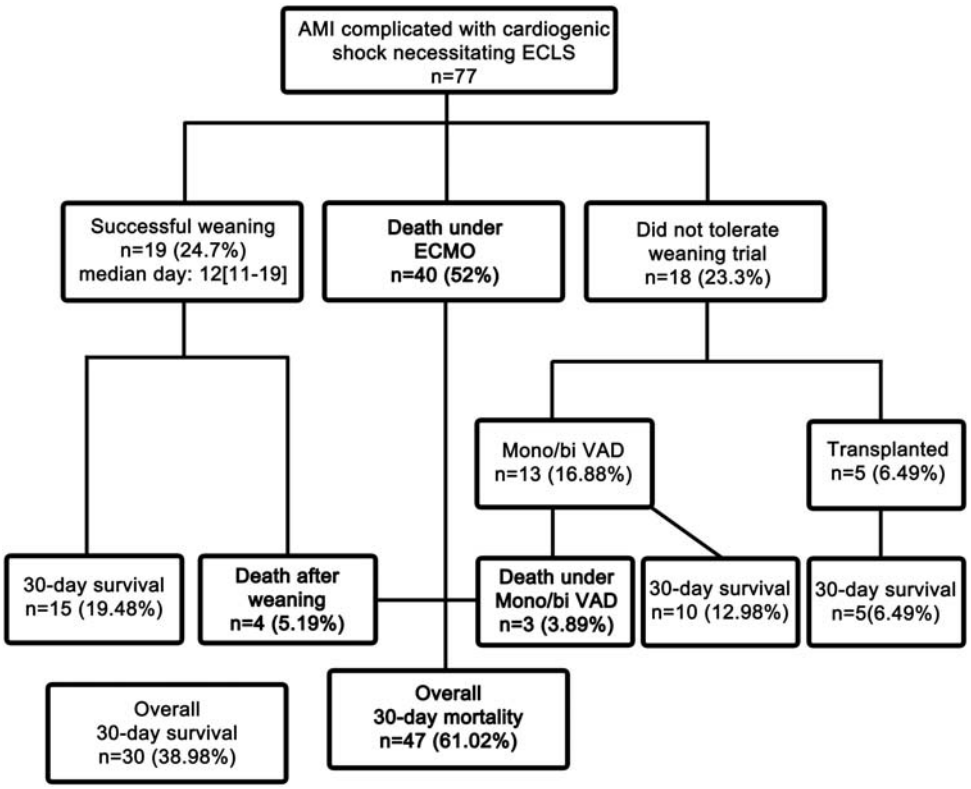

Figure 2: Thirty-day outcomes of patients with acute myocardial infarction (AMI) complicated by cardiogenic shock following extracorporeal life support (ECLS).

pulmonary oedema rate was 45.4% in patients assisted by ECLS and IABP vs 30.8% in the group with ECLS alone ($P = 0.14$). No patient underwent a transcutaneous septostomy or received a microaxial flow pump (Impella™ Recover LP 2.5 or 5.0) to decompress the left ventricle. Major bleeding occurred in 16 patients (21.3%), of whom 14 benefited from central ECLS. Following implantation, site-related adverse events were relatively infrequent, with, respectively, 7 and 6 cases of lower limb ischaemia (9.2%) and wound infection (8%). We also observed 2 cases of stroke (2.6%).

ECLS weaning, ventricular assistance device implantation and bridge to transplantation

Figure 2 shows the 30-day outcome of the 77 patients included in this series. Nineteen patients were successfully weaned (24.7%). Forty patients died during ECLS support without a weaning trial (52%). Eighteen patients did not tolerate the weaning trial (23.3%). They were therefore implanted with a mono- ($n = 9$) or bi-VAD ($n = 4$) and 5 were bridged to heart transplantation. As for the non-revascularized patients, none of

Table 2: Causes of all in-hospital deaths

ECLS weaning failure, *n* (%)	40 (51.95%)
Multiorgan failure	26
Failure of cardiac recovery	6
Massive bleeding	3
Septic shock	3
Aortic dissection	1
Left ventricle thrombosis	1
After ECLS weaning, *n* (%)	4 (5.19%)
Multiorgan failure	2
Neurological death	2
After bridge to mono/bi-VAD, *n* (%)	6 (7.80%)
Multiorgan failure	4
Septic shock	1
Massive bleeding	1
After heart transplantation, *n* (%)	1 (1.29%)
Septic shock	1
Total	51 (66.23%)

ECLS: extracorporeal life support; VAD: ventricular assist device.

Table 3: Univariate analysis of risk factors of 30-day death

Variables	*P*-value
Preimplantation lactate serum level	<0.0001
Preimplantation creatinine serum level	0.0046
Previous cardiopulmonary resuscitation	0.0007
1-day lactate serum level	0.0006
3-day lactate serum level	<0.0001
1-day creatinine serum level	0.0031
3-day creatinine serum level	0.0189
1-day SGOT level	0.0018
3-day SGOT level	<0.0001
Increasing SGOT between 0 day and 1 day	0.0373
Successful weaning	<0.0001
Surgical wound infection	0.0192
Acute renal failure necessitating haemofiltration	0.0391
SOFA score	0.0240

SOGT: serum glutamic oxaloacetic transaminase; SOFA: sepsis-related organ failure assessment.

them could be weaned ($n = 7$), 4 died under ECLS, 2 were bridged to VAD and 1 was bridged to transplantation.

Survival and predictive factors of 30-day mortality

Thirty-day and in-hospital survival rates were, respectively, 38.98 and 33.77%. Causes of in-hospital death are shown in Table 2. Table 3 shows univariate analysis of risk factors statistically associated with 30-day mortality. Only variables with *P*-value <0.05 were introduced in the logistic regression model. The results of the multivariable analysis are presented in Table 4. Three independent predictors of 30-day mortality emerged in this series: preimplantation lactate serum level, preimplantation creatinine serum level and previous CPR. The *P*-value for the Hosmer and Lemeshow test was 0.44, and the area under the ROC curve was

Table 4: Multivariate analysis of 30-day death risk factors

Variable	OR	95% CI	*P*-value
Previous cardiopulmonary resuscitation	4.729	1.258–17.770	0.022
Pre-ECLS lactate level	1.312	1.113–1.547	0.002
Pre-ECLS creatinine level	1.010	1.002–1.018	0.013

ECLS: extracorporeal life support.

0.867 (95% CI [0.792–0.943]). The discriminating ability of the logistic model (Fig. 3) allowed us to calculate an 'ECLS score' of 30-day mortality in cases of myocardial infarction with CS requiring ECLS support. The equation is given by the following formulas:

Calculating the risk score:

Risk score = −3.6321 + 0.2717 × preimplantation lactate serum level (mmol/l) + 0.00987 × preimplantation creatinine serum level (μmol/l) + 1.5536 × previous CPR (0 or 1)

Probability of 30-day mortality = 1/(1 + exp (− score)), where 'exp' denotes the exponential function (∼2.71828).

The computed probability of 30-day mortality has a Bootstrap mean standard error of 0.077.

DISCUSSION

The study showed a satisfactory rate of in-hospital survival (33.7%) in an extremely critical population of patients. AMI associated with CS usually carries a high mortality rate when the patient's status cannot be stabilized. Several therapeutic options appear useful in the rescue of critical patients: direct PCI, CABG, prompt ECLS with or without revascularization [6] and application of a mono- or biventricular assist device. Additionally, our data showed that preimplantation lactate and creatinine serum levels and previous CPR were the only three independent predictors of 30-day mortality.

Shock-induced organ damage was considered the primary cause of high mortality after AMI with CS. ECLS quickly restores haemodynamic function and decreases lactate, creatinine and glutamate-oxaloacetate transaminase serum levels. Successful initiation of ECLS not only eliminates the need to administer high doses of vasopressor and inotropic agents that might decrease myocardial oxygen consumption, but also improves end-organ microvascular perfusion [4]. As is the case in our study, the pre-ECLS serum lactate level has been shown to be an independent predictor of mortality [11]. With regard to its postimplantation level, Formica *et al*. [12] have reported a significant difference between survivors and non-survivors, whereas Ko *et al*. [13] and Bakhtiary *et al*. [14] have not. As for our outcomes in relation to preimplantation creatinine serum level and previous cardiopulmonary resuscitation showing that end-organ damage before implantation is a strong predictor factor of 30-day mortality, they also appear to show that when rapidly used to maintain normal systemic perfusion, ECLS may improve patient outcome. Renal failure may represent not only a common and important marker of high-risk status in these patients but also a predisposing factor for potentially lethal

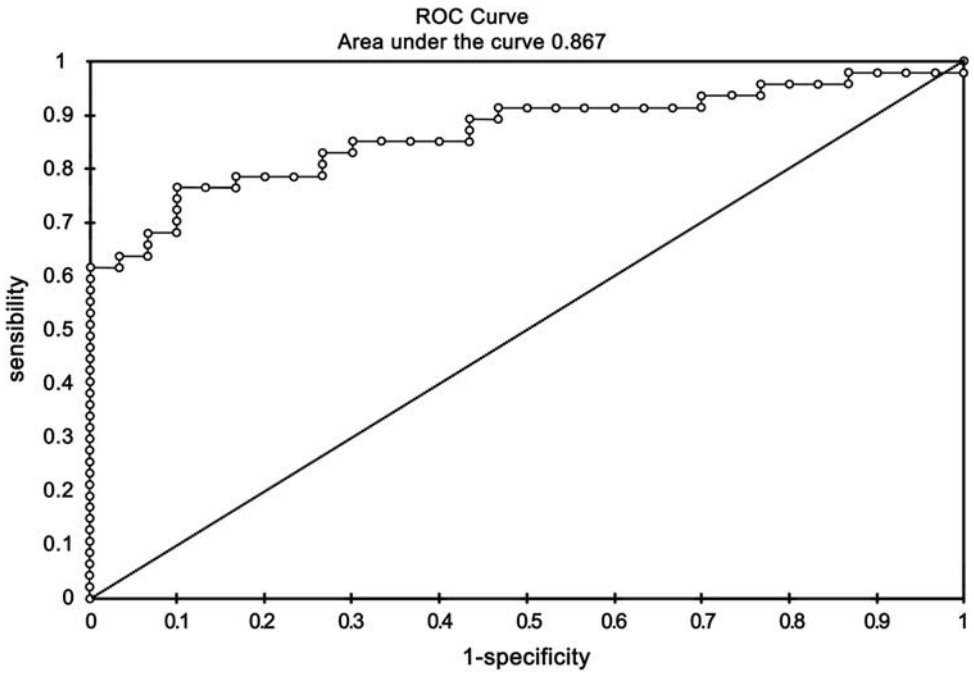

Figure 3: Receiver operating characteristic (ROC) curve for our logistic regression model developed from the analysis of 77 patients who required ECLS support for AMI with CS.

postimplantation complications. Timely management of these patients by our MUCA helps to reduce ischaemia duration in the end-organs and constitutes an additional factor contributing to improved patient outcome. However, as described by Kirsch *et al*. [15], some patients undergoing extracorporeal circulatory support may be subject to a mixed antagonistic (proinflammatory and anti-inflammatory) response syndrome. This response can result in immuno-paralysis, which might explain some of the failures encountered despite adequate haemodynamic support. On this subject, we did not evaluate the role of activation of the inflammatory cascade as a prognostic value for mortality or infection of ECLS recipients during their initial intensive care unit stay after implantation. Ongoing systemic inflammatory response syndrome (SIRS) in these patients might contribute to the development of multiorgan failure (MOF) [16]. Increased plasma levels of IL-6 and IL-8 during support have been shown to be prognostic of death in patients undergoing MCS as a bridge to transplantation [17]. It would also be interesting to determine whether postoperative monocytic human leucocyte antigen-DR (mHLA-DR) expression is of prognostic value for mortality or infection of ELCS recipients after implantation, as was shown by Kirsch *et al*. [15] in VAD recipients. In conclusion, when we note that CS patients requiring vasopressor support are particularly likely to have an adverse outcome despite adequate haemodynamic support, it would appear that a mechanistic approach aimed simply at re-establishing perfusion is insufficient. Future research will have to evaluate more precisely the role of SIRS in the development of MOF in these patients, and develop specific therapeutic strategies.

Unlike Sakomoto *et al*. [18], we did not find successful coronary revascularization to be a reliable predictor of 30-day survival. This result may be due to the deep and irreversible myocardial infarction expressed by the high mean level of troponin from our patients. Given their compromised status, many of the patients we studied required ECLS or a ventricular assist device as a bridge to heart transplantation. Weaning from ECLS using the protocol previously developed in our establishment [19] was nonetheless possible in 24% of the patients included in our group. In other studies, this rate was pronouncedly higher [11], probably due to a lower mean level of troponin, which allowed for easier myocardial recovery. Even though successful weaning is certainly statistically associated with in-hospital survival, we did not take into consideration its occurrence in our multivariate analysis, the reason being that it constitutes evolutionary data, and is consequently not available as a variable to be factored into a preimplantation predictive score of 30-day mortality.

ECMO is much less costly than the other systems, does not require operating room resources and avoids a sternotomy or ventriculotomy incision. Furthermore, current data do not suggest that ECMO is less efficacious for providing emergency circulatory support than alternative systems [6–10]. As for use of a left VAD, a BiVAD or a total artificial heart (TAH) in emergencies affecting this category of highly compromised patients, it might resolve the problem of pulmonary oedema and unsatisfactory left ventricle unloading, but given the elevated rate of mortality in these cases, the cost of such devices would, in our opinion, render any potential benefits moderate. It is nonetheless hardly obvious that ECLS is the ideal technique for these high-risk patients, and its use for cardiac failure in adult patients has its limitations. One concern is that left ventricular decompression may be inadequate and thus result in pulmonary hypertension and oedema, even though these complications may be prevented by non-invasive means that are discussed below. And yet, given the fact that many teams successfully use VAD as a measure of first resort when assisting this category of patients [20, 21], the question would appear to be largely discussed. Our policy consists in avoiding the use of VAD or TAH in overly unstable patients; in practice, we reserve this type of device for patients who have been stabilized with ECLS and who have not recovered cardiac function compatible with weaning. To

conclude, an initial period of resuscitation with ECMO is an effective strategy to salvage patients presenting with extreme haemodynamic instability and multiorgan injury. Use of VAD or TAH resources is improved by avoiding VAD or TAH implant in a very high-risk cohort of patients who do not survive ECLS [22].

As we have observed, the main limitation of peripheral ECLS consists in its inability to provide an adequate decompression of a heart with severely depressed left ventricular function [23]. When pump flow is increased in order to decrease cardiac preload, higher left ventricular afterload ensues and causes exacerbated dilatation, which finally leads to pulmonary oedema. Indeed, pulmonary oedema occurred in our patients at a high rate and occasionally required an invasive procedure to ensure ECLS centralization. The conclusion to be drawn is that a major remaining challenge consists in preventing pulmonary oedema following peripheral ECLS. As concerns the occurrence of pulmonary oedema and the need for centralization, we did not find any statistical difference between patients implanted with both peripheral ECLS and IABP vs ECLS alone. These results could be explained by the fact that IABP was not maintained throughout the support period and also because some patients already had a pulmonary oedema before implantation. It was consequently problematical in the framework of our study to assess the actual benefits of IABP on left ventricular loading. On this subject, an axial flow device has recently been developed, the Impella™ Recover LP 5.0 microaxial flow pump, which delivers a continuous flow of up to 5 l/min, and is presently available only as a surgical insertion [24]. Moreover, in pulmonary oedema management subsequent to ECLS, it would appear that in numerous cases, the association of peripheral ECLS with a means of unloading the left ventricle (transcutaneous septostomy, IABP or Impella™ Recover LP 5.0) should be considered as a possible option throughout support time. During our study, we did not use transcutaneous septostomy to decompress the left ventricle. However, a number of teams with trained operators have already used it quite successfully to unload the left ventricle [25]. In cases of total absence of cardiac contractility, a central cannulation was chosen and left ventricular venting (through the pulmonary artery, the right superior pulmonary vein or the apex of the left ventricle) was implemented. It has the drawback of carrying a high risk of bleeding. In order to prevent pulmonary oedema, we implement a medical treatment (cf. Methods section) now associated with IABP, but we recognize the need to have solid data and therefore to perform comparative studies designed to assess the efficacy of the different methods aimed at unloading the left ventricle.

There exists no specific score pertaining to patients with ECLS. While a presented score will not offer much help in decision-making in patients in deep CS and in urgent need of haemodynamic support, it might serve to provide patients and their families with personalized prognosis prediction and to establish benchmarks for outcome estimation and comparison. However, before it can be routinely used, the ECLS score needs to be validated in a prospective study and with a larger cohort.

Study limitations

A small number of patients included in a retrospective study entails numerous limitations. Moreover, our cohort was not uniform in terms of either preimplantation conditions or assistance and care management. A prospective and randomized trial would be necessary to evaluate the benefits of ECLS when associated with a means of unloading the left ventricle to prevent pulmonary oedema under peripheral ECLS. The 'ECLS score' suggested in this study should be evaluated more widely in order to more specifically define the risk profile for all patients with profound CS-necessitating ECLS. Finally, even though it is undeniably a factor influencing mortality, activation of the inflammatory cascade as a prognostic value for the latter or for infection of ECLS recipients during their initial intensive care unit stay following implantation has not been evaluated.

CONCLUSION

Prompt ECLS support is an effective management tool and provides reasonable chances for survival in highly compromised patients with AMI associated with profound CS. Its use as a bridge to recovery, transplantation and mono- or biventricular assist device could effectively decrease mortality.

As shown in our results pertaining to predictive risk factors for 30-day mortality, reducing the duration of end-organ ischaemia is the keystone to management of this patient population. A major remaining challenge consists in preventing pulmonary oedema following peripheral ECLS.

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