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ICU mortality of post-myocardial infarction ventricular septal defect complicated by cardiogenic shock: a retrospective multicentric cohort

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Abstract

Purpose Post myocardial infarction ventricular septal defect (PMI-VSD) complicated by refractory cardiogenic shock is associated with an extremely high mortality rate. We sought to evaluate the factors associated with in-ICU mortality in patients with PMI-VSD-related cardiogenic shock.

Methods Patients with PMI-VSD complicated by cardiogenic shock, admitted in 10 French tertiary centers between 2008 and 2022, were retrospectively included. The primary outcome was in-ICU mortality. The timing of surgery was classified as early (≤ 7 days) or late (> 7 days). Multivariable analysis was performed to identify the variables associated with in-ICU mortality.

Results A total of 138 patients were included (mean age 70 (± 10) years, female sex 54%). Of these, 116 patients (84%) received MCS, including 43 patients (31%) with VA-ECMO. VSD surgical closure was performed in 93 patients (67%, 60 early, 33 late). Only 2 patients had percutaneous closure without surgical repair. A total of 84 patients (61%) died. The type of surgical management strategy was significantly associated with in-ICU mortality (no surgery, 100%; early surgery, 45%; late surgery, 27%; $p_{trend} < 0.001$). In all patients, the variables independently associated with in-ICU mortality were: old age (adjusted OR = 1.1, 95%CI [1.02–1.12.], $p = 0.004$), SOFA score (adjusted OR = 1.2, 95%CI [1.07.-1.37], $p = 0.003$), and VA-ECMO (adjusted OR = 2.9, 95%CI [1.2–7.7], $p = 0.02$). In patients with VSD surgical closure, a longer delay between ICU admission and VSD surgical closure was independently associated with decreased in-ICU mortality (adjusted OR = 0.9, 95%CI [0.79–0.96], $p = 0.003$).

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Take Home Message Critically ill patients with PMI-VSD may benefit from delayed VSD surgical closure. Defining the optimal management of these unstable patients is yet to be defined and warrants further research.

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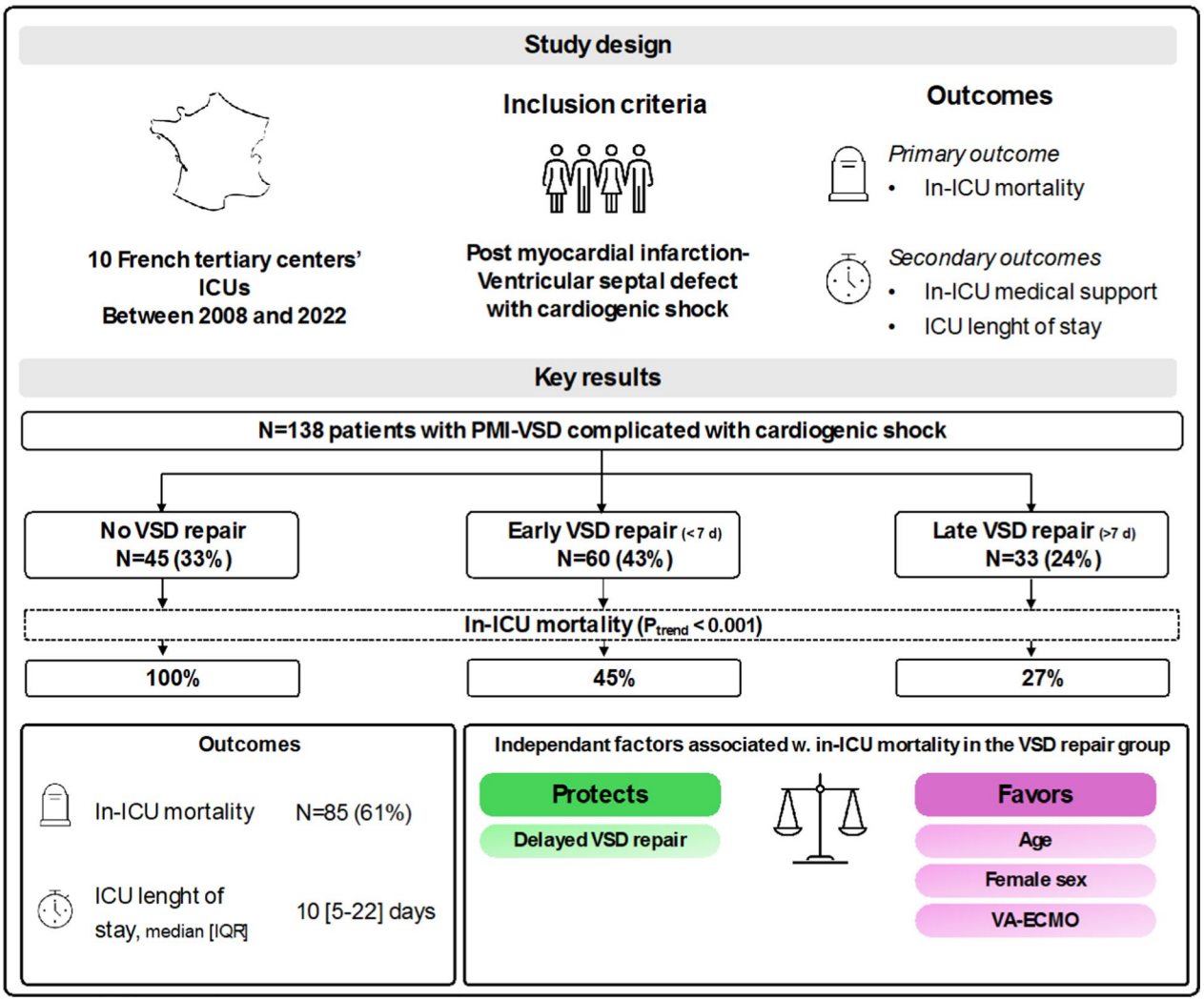
Conclusion Delayed VSD closure is associated with improved outcomes in PMI-VSD complicated by cardiogenic shock.

Trial registration #CE SRLF 19-34, #CNIL MR004 2224973, retrospectively registered 04 July 2019

Keywords Ventricular septal defect, Extra corporeal membrane oxygenation, Mechanical circulatory support, Cardiogenic shock, Mechanical complication, Myocardial infarction, Surgical timing

Graphical Abstract



Abbreviations: d day; ICU intensive care unit; SOFA sequential organ failure assessment; VA-ECMO veno-arterial extracorporeal membrane oxygenation; VSD ventricular septal defect; w with.VSD repair is equivalent to VSD surgical closure and do not include the patients (n=2) who underwent percutaneous closure only.

Background

Post-myocardial infarction ventricular septal defect (PMI-VSD) is a rare but very serious condition. Although it is associated with an extremely high mortality rate (nearly 40% and up to 60% in patients with

cardiogenic shock), optimal management is poorly defined and usually entails surgical intervention to correct the septal defect [1–3]. However, early closure of PMI-VSD is associated with a high rate of failure due to (i) tissue inflammation, and necrosis, (ii) technical challenges associated with the surgical procedure in this context and (iii) hemodynamic instability. Therefore, a delayed VSD repair strategy is often recommended [4]. However, the most severe patients with refractory cardiogenic shock may not benefit from this delayed management strategy. The use of mechanical circulatory support (MCS), mostly veno-arterial extracorporeal membrane oxygenation (VA-ECMO), has been proposed to stabilize patients before surgery. However, the contribution of VA-ECMO as a bridge to surgery and its impact in this setting is not well defined [5].

With the development of coronary reperfusion strategies, PMI-VSD is becoming rarer, with a current incidence of 0.2% compared to 1–2% in the pre-thrombolytic era[3, 6]. VSD is the main mechanical complication of MI (e.g., compared to 0.05% for mitral regurgitation due to ischemic papillary muscle rupture and 0.01% for free wall rupture) [7, 8]. In this context, only a few, small, retrospective cohorts and case reports appear in the literature. Furthermore, most of these studies have included heterogeneous patient cohorts in terms of hemodynamic status [9, 10].

Here we have sought to investigate the factors associated with the timing of surgical closure and its impact on early mortality in patients with a PMI-VSD-related cardiogenic shock in a large, contemporary French cohort.

Methods

Study population and design

We retrospectively included patients with PMI-VSD complicated by cardiogenic shock admitted to the intensive care unit (ICU) in 10 French tertiary centers certified for cardiac surgery between June 2008 and March 2022. Cardiogenic shock was diagnosed by local investigators based on the common clinical definition; the association of a systemic arterial hypotension or low cardiac output requiring vasopressor/inotrope support, with left ventricular overload and signs of impaired organ perfusion [11]. Patients younger than 18 years old were excluded. The study was approved by our Institutional Review Board (Ethics committee of the French Intensive Care Society (SRLF) CE SRLF 19–34, retrospectively registered 04 July 2019) and was conducted in accordance with the French Data Protection Authority (CNIL MR004 2224973) and the Declaration of Helsinki. Written consent was not required due to the retrospective nature of the study. This report follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines [12] (**Table S1**).

Data collection and study endpoints

Medical records were thoroughly reviewed to assess patient outcomes. Standardized forms were used to collect the following information: baseline general characteristics; cardiovascular risk factors and history of coronary artery disease (CAD); clinical parameters and laboratory findings on ICU admission; MI characteristics; echocardiographic parameters; organ failure and support; ICU treatments including use of inotropes, vasopressors and MCS [intra-aortic balloon pump (IABP), VA-ECMO, intravascular micro-axial flow pump (AFP) or paracorporeal left ventricular assist device (pLVAD)]; VSD surgical closure and/or percutaneous closure; complications and outcomes. The diagnosis of VSD was based on transthoracic echocardiography. Variables were defined prior to data collection. The primary outcome was in-ICU mortality. The secondary outcomes were in-ICU medical support need (including cardiovascular, respiratory and renal support therapies) and ICU length of stay.

Statistical analysis

Normally distributed continuous variables were expressed as mean ± standard deviation, and non-normally distributed continuous variables were expressed as median with interquartile range. Categorical variables were expressed as number and percentages. Comparisons were made using $\chi 2$ or Fisher's exact test for categorical variables and Student's t-test or Mann–Whitney-Wilcoxon test for continuous variables, as appropriate. First, a descriptive analysis of the cohort was performed. Second, patient characteristics and outcomes were compared with respect to VA-ECMO implantation and the timing of VSD surgical closure. Patients were divided into three groups according to the following surgical management strategies: (i) no surgical closure, (ii) early surgical closure (≤ 7 days after ICU admission), and (iii) late surgical closure (>7 days after ICU admission) as previously published [4]. Percutaneous closure without surgery was excluded from these subgroup stratifications due to a small sample size ($n=2$) and to allow for interpretation of results within the context of surgery. The inclusion date used was the date of admission to the ICU. Third, multivariable analysis was performed to identify variables independently associated with mortality. Variables were included in the multivariable analysis if they were clinically relevant. Multiple imputation was used for variables with missing data (less than 30% missing), i.e. SOFA score and the delay between admission and surgery. Th adjusted odds ratios (aOR) presented in the

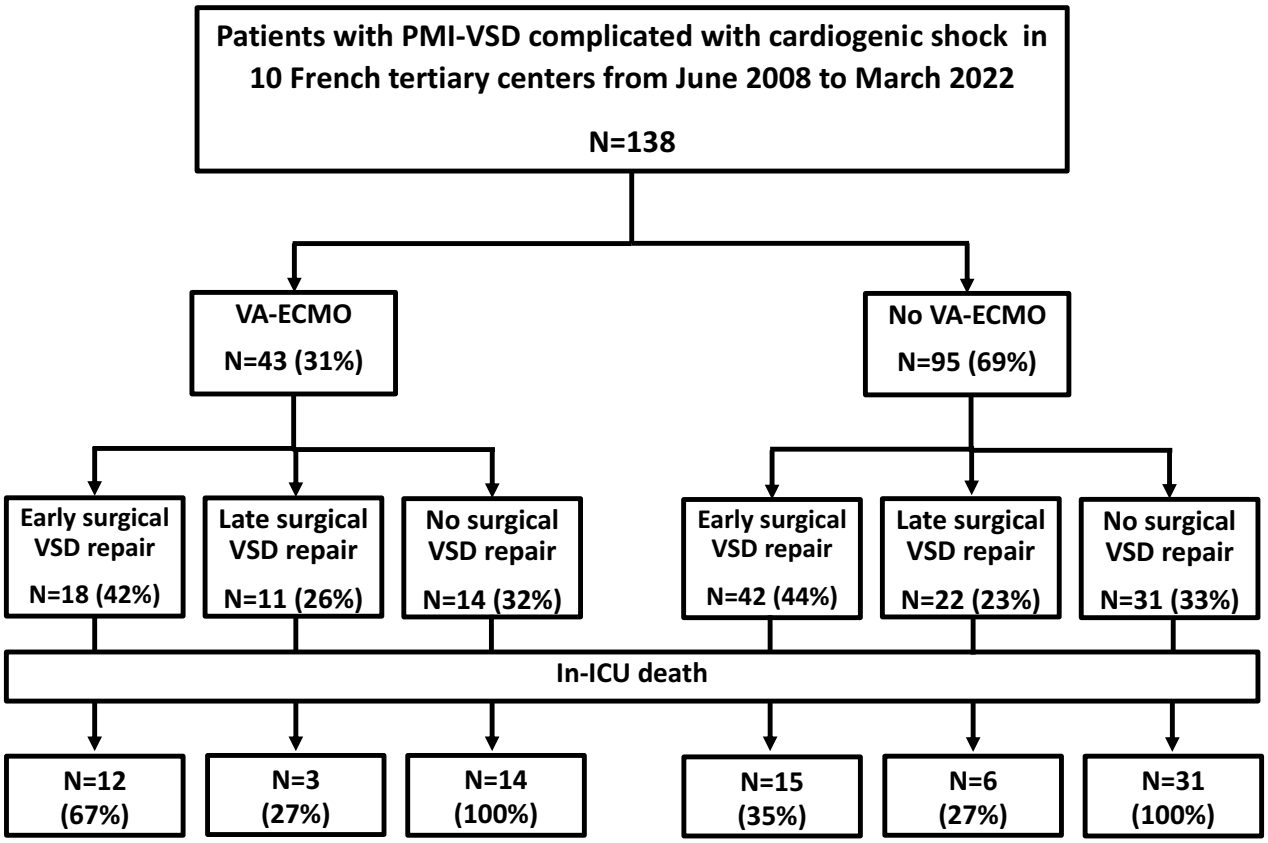


Fig. 1 Study Flow Chart. Abbreviations: VA-ECMO veno-arterial extracorporeal membrane oxygenation; ICU intensive care unit; PMI post-myocardial infarction; VSD ventricular septal defect

manuscript correspond to analyses with multiple imputation. Results were considered statistically significant with a p-value < 0.05 and all analyses were two-tailed. Statistical analyses were performed using RStudio software, version 4.0.5 (https://www.R-project.org/).

Results

Study population

A total of 138 patients were enrolled (Fig. 1). The mean age at admission was 70 ± 10 years. A majority of cases had no prior history of CAD (n = 124, 90%). Coronary angiography was performed in most patients (n = 128, 93%) and showed mono-vessel lesions in the majority (n = 71, 55%). A culprit lesion was identified in most patients (n = 128, 93%), typically involving the left anterior descending artery (n = 74 54%), with complete vessel occlusion in most patients (n = 98, 71%). The median delay between chest pain and ICU admission was 2 days [0–4]. All patients were in cardiogenic shock with a mean ICU admission SOFA score of 7 ± 3. Mean LVEF was 42% (± 13). Characteristics of the study population are shown in Table 1.

VSD closure

A total of 95 patients (69%) underwent VSD closure; having either surgical closure only (n = 81), surgical and percutaneous closure (n = 12) or percutaneous closure only (n = 2). Concomitant coronary artery bypass grafting (CABG) was performed in 20 patients (14%). All patients who did not benefit from surgical VSD closure died (n = 43). Comparison of clinical characteristics of patients with and without VSD surgical closure is shown in Table 1**.** Early (< 7 days) surgical closure was performed in 60 patients (44%) and late surgical closure in 33 patients (24%). A total of 27 patients (45%) died in the early surgery group versus 9 patients (27%) in the late surgery group (p = 0.09). Overall, death rates were significantly associated with surgical management strategies (p-trend < 0.001) (Fig. 2**, A**).

Mechanical circulatory support

A total of 116 patients (84%) received MCS: 43 VA-ECMO (31%), 99 IABP (72%), 6 of other types (4%) (AFP (n = 5) or pLVAD (n = 1)). Of the patients on VA-ECMO, 30 (70%) received concomitant IABP support and 1 concomitant AFP (Tables S2 and S3). Variables associated

Table 1 Characteristics and outcomes of patients with VSD according to closure status

	All patients (n = 138)	Surgical closure (n = 93)	No surgical closure (n = 45)	*P*-value
Baseline characteristics				
Age (years)	70 (± 10)	68 (10)	74 (9)	**0.002**
Female gender	63 (46%)	43 (46%)	20 (44%)	0.843
Chronic kidney disease	4 (3%)	3 (3%)	1 (2%)	1
Known coronary artery disease	14 (10%)	7 (7%)	7 (15%)	0.143
COPD	11 (8%)	7 (7%)	4 (9%)	0.835
Baseline organ failures				
SOFA score at ICU admission (n = 101)	7 (3)	6 (3)	8 (4)	**0.004**
Lactatemia at ICU admission (mmol/L) (n = 74)	4.9 [3.4; 6.4]	3.9 [2.5; 5.3]	6.5 [3.8; 9.1]	0.083
Creatinine at ICU admission (µmol/l) (n = 96)	131 [83; 179]	108 [72; 144]	164 [97; 231]	**< 0.001**
Norepinephrine at ICU admission	106 (82%)	77 (83%)	29 (64%)	**0.017**
Dobutamine at ICU admission	105 (81%)	73 (78%)	32 (71%)	0.340
Epinephrine at ICU admission	28 (28%)	21 (32%)	7 (25%)	0.336
Myocardial infarction				
Delay between first symptoms and admission (d)	2 [1; 5]	2 [1; 5]	2 [1; 4]	0.563
Myocardial infarction site:				
- Anterior	61 (44%)	42 (45%)	19 (42%)	0.744
- Inferior	77 (56%)	51 (55%)	26 (58%)	
Culprit lesion:				
- Left anterior descending artery	74 (54%)	51 (55%)	23 (51%)	0.068
- Left circumflex artery	2 (1.5%)	0	2 (4%)	
- Right coronary artery	52 (38%)	38 (41%)	14 (31%)	
Number of affected coronary vessels:				
- 1	71 (51%)	56 (60%)	15 (33%)	**0.014**
- 2	26 (19%)	17 (18%)	9 (20%)	
- 3	31 (22%)	16 (17%)	15 (33%)	
PCI of culprit lesion	64 (46%)	39 (42%)	25 (55%)	0.101
Echocardiographic parameters				
LVEF (%)	42 (13)	43 (14)	42 (13)	0.638
Right ventricle dysfunction	68 (49%)	47 (50%)	21 (47%)	0.761
VSD diameter (mm, n = 125)	18 [10; 26]	20 [12; 28]	19 [9; 29]	0.626
VSD location:				
- Anterior/apical	74 (54%)	52 (56%)	22 (50%)	0.825
- Posterior/inferior	63 (46%)	41 (44%)	22 (50%)	
Pericardial effusion	17 (12%)	10 (11%)	7 (15%)	0.647
Mechanical circulatory support				
MCS use	116 (84%)	82 (88%)	34 (75%)	0.058
IABP	99 (72%)	71 (76%)	28 (62%)	0.084
Veno-arterial ECMO	43 (31%)	29 (31%)	14 (31%)	1
Others	7 (5%)	4 (4%)	3 (7%)	0.682
Percutaneous VSD surgical closure	14 (10%)	12 (13%)	2 (4%)	0.145
Outcomes				
MCS duration (d)	5 [3; 9]	7 [4; 10]	3 [2; 6]	**0.010**
Norepinephrine duration (d, n = 57)	3 [1; 6]	3 [2; 8]	3 [2; 6]	0.094
Dobutamine duration (d, n = 154)	5 [2; 10]	7 [3; 12]	3 [1; 5]	**0.007**
Ventilation duration (d, n = 98)	5 [1; 13]	6 [1; 15]	3 [1; 10]	0.066
RRT use during ICU stay	43 (31%)	29 (31%)	14 (31%)	0.993
ICU length of stay (d)	10 [5; 22]	15 [7; 30]	6 [3; 10]	**< 0.001**
In-ICU mortality	84 (61%)	39 (42%)	45 (100%)	**< 0.001**

Table 1 (continued)

Continuous variables are expressed as mean ± standard deviation or median [interquartile range (IQR)]; categorical variables are expressed as n (%). Abbreviations: VSD ventricular septal defect; CABG coronary artery bypass graft; COPD chronic obstructive pulmonary disease; d days; ECMO extra corporeal membrane oxygenation; IABP intra-aortic balloon pump counter pulsation; ICU intensive care unit; LVEF left ventricular ejection fraction; MCS mechanical circulatory support; PCI percutaneous coronary intervention; RRT renal replacement therapy; SOFA sequential organ failure assessment. Bolded results are statistically significant at the *P*-value < 0.05 level. When not precised, percentages were calculated over the whole cohort (i.e. 138 patients). Chronic kidney disease denoted patients with an estimated glomerular filtration rate below 60 mL/min/1.73m^2

with ECMO implantation were: younger age (66 vs. 72 years, $p=0.003$), a culprit lesion involving the right coronary artery (58% vs. 28%, $p<0.001$) and posterior VSD location (51% vs. 36%, $p=0.02$). Patients on ECMO had a longer duration of mechanical ventilation (10 vs. 2 days, $p<0.001$) and higher rates of renal replacement therapy (53% vs. 21%, $p<0.001$). Mortality did not differ between the VA-ECMO and nonVA-ECMO groups (55% vs. 33%, $p=0.08$). A comparison of clinical characteristics between these groups is shown in Table S3. Comparison of ECMO patients with and without another type of MCS is reported in Table S4.

ECMO as a bridge to VSD surgical closure

A subset of 29 patients on ECMO underwent VSD surgical closure (18 early (62%), 11 late (38%)). Of these, 15 (55%) patients died (Supplementary Table S5). In-ICU mortality rate was significantly associated with surgical timing (12 deaths (67%) in the early VSD group vs. 3 deaths (27%) in the late group, $p=0.04$) (Fig. 2, B).

AFP and pLVAD

Five patients had an AFP (2/5 were on concurrent VA-ECMO, did not benefit from surgery and died; 3/5 were not on VA-ECMO: 2/3 underwent surgery, of these two patients, one was discharged from ICU alive) and one had pLVAD (underwent surgery without VA-ECMO and was discharged alive from ICU).

In-ICU mortality

A total of 84 (61%) patients died, including all those who underwent surgical VSD closure. On univariate analysis, variables associated with death in the ICU were: old age; female gender; high SOFA score and serum creatinine level at ICU admission; pericardial effusion; PCI of the culprit lesion; absence of VSD surgical closure; no concomitant CABG and use of renal replacement therapy. Comparison of the clinical characteristics with respect to survival is shown in Table 2.

On multivariable analysis old age (aOR = 1.1, 95%CI [1.02–1.12.], $p=0.004$), high SOFA score (aOR = 1.2, 95%CI [1.07.-1.37], $p=0.003$), and the use of VA-ECMO (aOR = 2.9, 95%CI [1.2–7.7], $p=0.02$) were independently associated with poor outcomes. In patients with VSD surgical closure, a longer delay between ICU admission and VSD surgical closure was independently associated with decreased in-ICU mortality in patients with VSD surgical closure (aOR = 0.9, 95%CI [0.79–0.96], $p=0.003$) (Table 3**).**

Discussion

Herein, we report the clinical characteristics, and outcomes associated with PMI-VSD complicated by cardiogenic shock in a large, contemporary French cohort. The main findings of this study are as follows: i) in-hospital mortality rate was 61%; ii) all patients who did not undergo VSD surgical closure died; iii) old age, high SOFA score, VA-ECMO treatment and early surgery were independently associated with death in the ICU; iv) a longer duration from ICU admission to VSD surgical closure was associated with lower mortality among patients undergoing surgery (Graphical Abstract).

The baseline characteristics of the patients considered in our series were roughly equivalent to those of critically ill patients with PMI-VSD reported in the literature in terms of age and sex [1]. In addition, the all-cause mortality and operative mortality rates were approximately 60% and 40%, respectively, which are close to those reported in patients with cardiogenic shock [2].

Overall, the variables associated with mortality in our series were consistent with the literature. Old age and female sex have been reported to be associated with worse outcomes in previous studies [13–16]. High serum creatinine and lactate levels have also been associated with worse outcomes in previous studies [13–15]. As previously reported in the literature, posterior location of the VSD and right coronary involvement were associated with an increase in ECMO treatment and higher mortality rates [3, 17, 18].

The means and timing of revascularization play a key role in the management of these patients. Considering that VSD is a post-MI complication, treatment of the underlying CAD is advocated to provide ischemic border perfusion. However, in our study, PCI of the culprit lesion was associated with higher mortality in univariate analysis, consistent with previous studies [1, 15]. Our reasoning for this increased risk is related to the late reperfusion of necrotic areas and the elevated hemorrhagic risk due to the antiplatelet treatments required after percutaneous procedures, which may accelerate

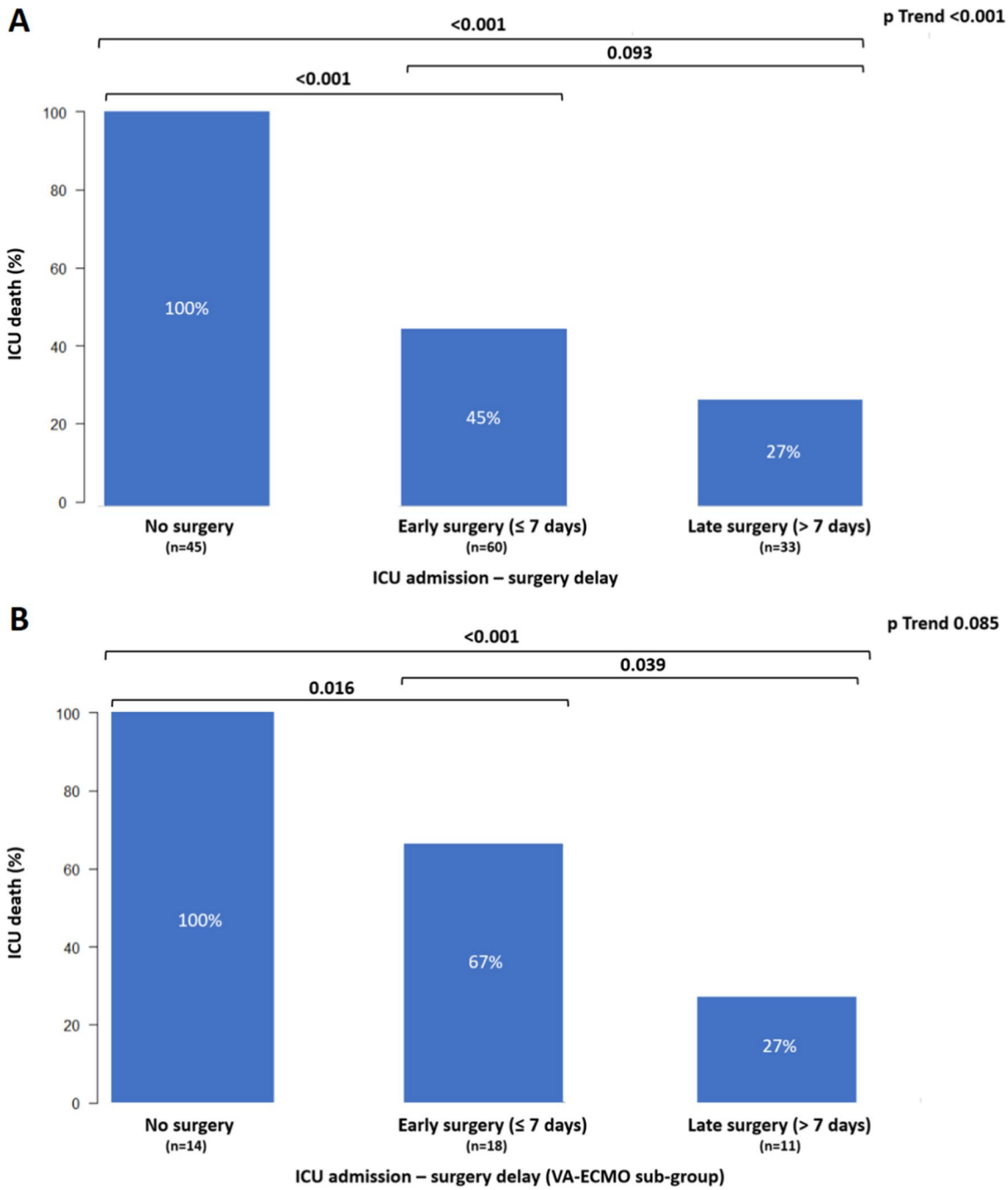


Fig. 2 In-ICU death according to surgery strategy and delay. **A**. In all patients. **B**. In VA-ECMO implanted patients. Abbreviations: VA-ECMO, veno-arterial extracorporeal membrane oxygenation; ICU, intensive care unit

myocardial rupture. Notably, a shorter delay between MI and VSD has been observed in the thrombolytic era [6]. Additionally, PCI may be associated with adverse effects from iodine injection and delayed medical support [19]. Although the findings of the French FAST-MI registry support the benefits of coronary revascularization for latecomer (> 12 h) STEMI patients, the outcomes associated with longer delays remain unknown, as patients revascularized after 48 h were not included [20]. In our study, the median delay between chest pain onset and admission was 2 days as previously reported [5]. In VSD patients, the maximum time after which PCI may be detrimental, if any, is unknown. Besides PCI, the role of concomitant CABG in this setting is still debated because

Table 2 Characteristics of patients with postinfarction ventricular septal defect by ICU survival

	All patients (*n* = 138)	ICU survivors (*n* = 54)	ICU non-survivors (*n* = 84)	*P*-value
Baseline characteristics				
Age (years)	70 (10)	66 (11)	73 (9)	**< 0.001**
Female gender	63 (46%)	18 (33%)	45 (53%)	**0.020**
Baseline organ failures				
SOFA at ICU admission (n = 101)	7.1 (3)	5.5 (3)	8.1 (4)	**< 0.001**
Lactatemia at ICU admission (mmol/L) (n = 74)	4.9 [3.4; 6.4]	3 [2; 5]	4 [3; 6]	0.076
sCr at ICU admission (µmol/L) (n = 96)	131 [83;179]	108 [79;152]	145 [98; 215]	**0.005**
Norepinephrine at ICU admission	106 (82%)	42 (86%)	64 (79%)	0.5
Dobutamine at ICU admission	105 (81%)	40 (78%)	65 (82%)	0.75
Epinephrine at ICU admission	28 (28%)	11 (28%)	17 (27%)	1
Myocardial infarction				
Delay between symptom and admission (d)	2 [0; 4]	3 [1; 4]	2 [1; 4]	0.313
Myocardial infarction site:				
- Anterior	61 (44%)	31 (57%)	46 (55%)	0.760
- Inferior	77 (56%)	23 (42%)	38 (45%)	
Culprit lesion:				
- Left anterior descending artery	74 (54%)	31 (57%)	43 (51%)	0.813
- Left circumflex artery	2 (1.5%)	0	2 (2%)	
- Right coronary artery	52 (38%)	21 (39%)	31 (37%)	
Occlusion of culprit lesion	98 (71%)	41 (76%)	57 (68%)	0.756
Number of affected coronary vessels:				
- 1	71 (51%)	31 (57%)	40 (48%)	0.378
- 2	26 (19%)	12 (22%)	14 (17%)	
- 3	31 (22%)	9 (17%)	22 (26%)	
PCI of culprit lesion	64 (46%)	19 (35%)	45 (54%)	**0.032**
Thrombolysis	4 (3%)	1 (2%)	3 (4%)	1
EF (%)	42 (13)	42 (12)	43 (14)	
Right ventricle dysfunction	68 (49%)	26 (48%)	42 (50%)	0.752
VSD diameter (mm, n = 125)	18 [10; 26]	17 [12; 25]	19 [10; 30]	0.863
VSD location:				
- Anterior/apical	74 (54%)	32 (59%)	42 (50%)	0.563
- Posterior/inferior	63 (46%)	22 (41%)	41 (50%)	
Pericardial effusion	17 (12%)	2 (2%)	15 (18%)	**0.015**
Mechanical circulatory support				
IABP	99 (72%)	41 (76%)	58 (69%)	0.381
Veno-arterial ECMO	43 (31%)	13 (24%)	30 (36%)	0.150
Veno-arterial ECMO with LV unloading	30 (22%)	11 (20%)	19 (23%)	1
Others	7 (5%)	3 (6%)	4 (5%)	1
MCS duration (d)	5 [2; 8]	5 [4; 12]	5 [3; 8]	0.154
VSD surgical closure				
VSD surgical closure	93 (67%)	54 (100%)	39 (46%)	**< 0.001**
Delay between symptom and VSD surgical closure (d)	7 [2.5; 11.5]	8 [5; 18]	7 [4; 10]	0.117
Delay between admission and VSD surgical closure (d)	4 [0; 8]	5 [1; 11]	3 [1; 7]	0.224
Delay between MCS and VSD surgical closure (d)	3 [0; 6]	3 [0; 9]	3 [1; 6]	0.925
Percutaneous VSD surgical closure	14 (10%)	5 (9%)	9 (11%)	0.782
Cardiopulmonary bypass time (min, n = 60)	127 (44)	124 (46)	132 (38)	0.562
Concomitant CABG	20 (14%)	14 (26%)	6 (7%)	**0.003**
Surgical revision	19 (14%)	9 (17%)	10 (12%)	0.445

Table 2 (continued)

	All patients (*n* = 138)	ICU survivors (*n* = 54)	ICU non-survivors (*n* = 84)	*P*-value
Outcomes				
Norepinephrine duration (d, n = 57)	3 [1; 6]	3 [2; 5]	3 [1; 6]	0.941
Dobutamine duration (d, n = 54)	5 [1.7; 8.3]	8 [5; 15]	4 [2; 7]	**0.004**
Mechanical ventilation duration (d, n = 98)	5 [1; 9]	6 [1; 17]	4 [1; 10]	0.260
RRT use during ICU stay	43 (31%)	9 (17%)	34 (40%)	**0.003**
ICU length of stay (d)	10 [2; 18]	20 [10; 35]	7 [4; 13]	**< 0.001**

Continuous variables are expressed as mean ± standard deviation or median [interquartile range (IQR)]; categorical variables are expressed as n (%). CABG: coronary artery bypass graft; COPD: chronic obstructive pulmonary disease; ECMO: extra corporeal membrane oxygenation; IABP: intra-aortic balloon pump; ICU: intensive care unit; LVEF: left ventricular ejection fraction; VSD: ventricular septal defect; MCS: mechanical circulatory support; PCI: percutaneous coronary intervention; RRT: renal replacement therapy; sCr, serum creatinine; SOFA: sequential organ failure assessment. Bolded results are statistically significant at the *P*-value < 0.05 level. When not precised, percentages were calculated over the whole cohort (i.e. 138 patients)

Table 3 Factors associated with ICU death according to surgical management and extracorporeal support

	Without imputation				With Imputation			
	Univariate	*p*-value	Multivariable	*p*-value	Univariate	*p*-value	Multivariable	*p*-value
All patients (n = 138)								
Age	1.06 [1.02; 1.10]	**0.001**	1.07 [1.02; 1.14]	**0.004**	1.06 [1.02; 1.10]	**0.001**	1.07 [1.02; 1.12]	**0.004**
Sexe (female)	1.81 [0.92; 3.64]	0.090	1.08 [0.40; 2.92]	0.877	1.81 [0.92; 3.64]	0.090	1.61 [0.72; 3.63]	0.243
PCI of culprit lesion	1.96 [0.98; 3.99]	0.058	1.31 [0.48; 3.51]	0.597	1.78 [0.90; 3.55]	0.100	1.08 [0.48; 2.39]	0.850
SOFA at ICU admission	1.29 [1.11; 1.47]	**< 0.001**	1.26 [1.09; 1.48]	**0.003**	1.20 [1.08; 1.35]	**0.001**	1.20 [1.07; 1.37]	**0.003**
VA-ECMO	1.86 [0.89; 4.05]	0.106	2.94 [0.90; 10.9]	0.087	1.86 [0.89; 4.05]	0.106	2.94 [1.19; 7.70]	**0.022**
Pericardial effusion	4.15 [1.12; 4.15]	**0.032**	2.21 [0.49; 12.6]	0.327	4.02 [1.23; 18.1]	**0.035**	3.56 [0.97; 17.3]	0.075
With surgical VSD surgical closure (n = 93)								
Age	1.05 [1.01; 1.11]	**0.023**	1.09 [1.02; 1.20]	**0.020**	1.05 [1.01; 1.11]	**0.023**	1.08 [1.02; 1.16]	**0.018**
Sexe (female)	2.69 [1.15; 6.48]	**0.024**	2.78 [0.73; 11.7]	0.142	2.69 [1.15; 6.48]	**0.024**	3.65 [1.28; 11.4]	**0.019**
PCI of culprit lesion	2.08 [0.88; 4.99]	0.096	1.73 [0.43; 6.74]	0.429	1.92 [0.82; 4.52]	0.132	1.86 [0.63; 5.59]	0.259
SOFA at ICU admission	1.19 [1.01; 1.43]	**0.044**	1.17 [0.95; 1.46]	0.145	1.20 [1.05; 1.39]	**0.008**	1.14 [0.98; 1.36]	0.100
VA-ECMO	2.19 [0.90; 5.44]	0.086	3.97 [0.71; 28.2]	0.132	2.19 [0.90; 5.44]	0.086	6.23 [1.78; 26.2]	**0.007**
Pericardial effusion	4.5 [1.15; 22.14]	**0.039**	1.92 [0.27; 15.1]	0.511	4.34 [1.12; 21.3]	**0.043**	3.82 [0.74; 25]	0.123
ICU admission to VSD surgical closure delay (day)	0.94 [0.86; 1.01]	0.090	0.88 [0.75; 0.99]	**0.047**	0.94 [0.86; 1.00]	0.090	0.88 [0.79; 0.96]	**0.010**

Continuous variables are expressed as mean ± standard deviation or median [interquartile range (IQR)]; categorical variables are expressed as n (%). When not precised otherwise, percentages were calculated over the whole cohort (i.e. 138 patients). CABG: coronary artery bypass graft; COPD: chronic obstructive pulmonary disease; ECMO: extra corporeal membrane oxygenation; IABP: intra-aortic balloon pump; ICU: intensive care unit; LVEF: left ventricular ejection fraction; VSD: ventricular septal defect; MCS: mechanical circulatory support; PCI: percutaneous coronary intervention; RRT: renal replacement therapy; SOFA: sequential organ failure assessment. Bolded results are statistically significant at the *P*-value < 0.05 level

the advantages of revascularization are often offset by higher surgical risks and longer cardiopulmonary bypass and aortic cross-clamping durations. However, CABG may be particularly important in multivessel CAD, which has also been associated with higher mortality [1, 3, 21]. In our study, unlike PCI, CABG was associated with improved outcomes in univariable analysis. Nevertheless, patients who undergo CABG are typically highly selected and show good prognostic factors.

Surgical closure is the cornerstone treatment for VSD. Although delayed surgical closure is generally preferred due to tissue inflammation, critically ill patients are at high risk for early death [9]. Our results suggest that in critically ill patients, delaying surgery has beneficial effects on outcomes [16]. ECMO as a bridge-to-surgery may stabilize patients and allow for tissue scarring prior to surgery. Rob et al. published an important study reporting an overall 30-day mortality of 71% in 14 patients with cardiogenic shock and noted a beneficial impact on outcomes in patients receiving VA-ECMO [22]. Recently, increasing data has been published on using ECMO as a bridge to surgical closure in unstable patients [1, 14, 15, 23–30]. However, only two series have specifically included critically ill patients in cardiogenic shock [2, 31]. In this subgroup of severe patients, the optimal delay for surgery was unknown [32]. However, previous studies have consistently reported results supporting a delayed strategy irrespective of the circulatory status'[14,15]. Arnaoutakis et al. reported a 17% mortality in patients undergoing surgery after 7 days compared to 54% in those undergoing surgery before 7 days [14]. As suggested in recent literature reviews [36], early surgery in PMI-VSD appears to be associated with worse outcomes, and the use of MCS as a bridge to delayed surgery is likely a viable alternative.

In most series, patients were included irrespective of their hemodynamic status. In our series, which only included patients with cardiogenic shock, those treated with ECMO were more severely ill than those who were not, as indicated by their creatinine and lactate levels and their SOFA scores. In-ICU mortality rates of patients with ECMO were not significantly different from those without ECMO on univariate analysis, but ECMO was associated with higher rates of death in multivariable analysis. Overall, our results do not demonstrate a clear benefit of ECMO. However, this potential benefit may be difficult to assess considering the confusion bias resulting from the close links between ECMO treatment and patient severity. Further, MCS may allow delayed surgery, which was associated with improved outcomes [37].

There is no consensus on the ideal MCS strategy for patients with PMI-VSD complicated by cardiogenic shock [37]. Randomized controlled trials and meta-analyses have reported that IABP alone does not significantly improve outcomes for patients with all-cause cardiogenic shock [38]. An IABP reduces the afterload of the failing heart and improves forward systemic cardiac output and coronary flow but does not improve end-organ perfusion [38]. Theoretically, it also reduces the shunt across the VSD and thereby decreases right heart overload. Although we were not able to demonstrate a survival benefit with its use, IABP support may have provided adequate stabilization for subsequent surgical closure [37].

In cases of refractory cardiogenic shock, VA-ECMO is the most widely used MCS device for restoring blood flow. However, VA-ECMO increases left-to-right shunting through the VSD and the afterload of the left ventricle. Due to these hemodynamic considerations, some authors have advocated for the use of percutaneous left ventricular assist devices, such as an AFP (Impella—Abiomed, Danvers, MA), as a bridge to surgery or transplant. These devices could be associated with improved tissue scarring compared to VA-ECMO as recently suggested [39]. Additionally, postoperative left ventricular unloading with MCS likely protects the surgical patch and surrounding sutured tissue, leading to better outcomes [40].

The combination of VA-ECMO and left ventricular unloading with an AFP (referred to as ECMELLA or ECPELLA) could provide an attractive strategy to optimize hemodynamics during the perioperative period [41]. The ECPELLA combination theoretically offers the optimal combination of high cardiac output and significant left ventricular unloading. However, it comes with higher costs and increased rates of complications such as intravascular hemolysis, acute kidney injury, bleeding, stroke, and the risk of VSD enlargement [42]. This approach should be evaluated in future studies.

Deciphering the mechanisms underlying cardiogenic shock is crucial, and two situations should be distinguished: one scenario where the low output is associated with a massive shunt and mild to moderate LV dysfunction (usually posterior MI), and another scenario where the low output is caused by an extensive MI with large areas of myocardial damage and severe LV dysfunction (usually anterior MI). However, both in our series and in the literature, neither LVEF nor VSD diameter were associated with worse outcomes. This should be considered alongside the known facts that (i) the shunt can overestimate the LVEF, and (ii) VSD may be underestimated on transthoracic echocardiography at admission. Overall, although clinically challenging, distinguishing these two phenotypes may help identify the subgroup of patients who may recover after VSD surgical closure.

This work has limitations. First, it was a retrospective study with multiple potential sources of bias. Additionally,

some patient data were missing, particularly regarding the dose of vasopressors, which prevented the calculation of the vaso-inotropic score. Second, long-term follow-up was not available, limiting the extent of our conclusions. Third, regarding surgical timing, relatively few patients (n = 9) underwent salvage surgery (within 24 h post-ICU admission). It is possible that most centers were using a delayed VSD surgical closure approach. Only survivors to initial management could benefit from delayed surgery. Therefore, this may have contributed to an underestimation of mortality in patients with delayed surgical management. Patients who could not be stabilized and so underwent early surgery or patients who died early prior surgery were included in the early or no surgery groups, likely leading to a selection bias towards including healthier individuals within the delayed surgery group. This bias may help in explaining the lower mortality observed in these patients [43]. The association observed herein cannot be interpreted as causation. Importantly, MCS and VA-ECMO implantation rates were similar between patients who did and who did not undergo surgery. Additionally, very frail and unfit patients are usually not admitted in the ICU. These elements advocate for a fairly limited number of unfit patients within the "no surgery" group. Fourth, matching patients with and without ECMO using a propensity score was not possible due to a small sample size and significant differences between the groups. Fifth, data pertaining to percutaneous closure and AFP/pLVAD were too limited to draw robust conclusions from the current work. Sixth, although valuable, invasive hemodynamic data was missing due to current practices in France. Last, the time span of this study covers a long period of time during which medical knowledge increased, guidelines have changed and therefore clinical practice too. This point suggests the existence of a possible time-related bias in our study. Nevertheless, this work has several strengths: it was a multicenter study; the cohort of patients was homogeneous, as all patients had cardiogenic shock; and, most notably, it is the first large multicenter cohort study reporting on the experience of PMI-VSD-related cardiogenic shock.

Conclusions

In this contemporary series of patients with PMI-VSD complicated by cardiogenic shock, almost two thirds of the population died. Importantly, all patients who did not undergo VSD surgical closure died. Old age, SOFA score, and VA-ECMO were independently associated with in-ICU death, whereas a longer duration from ICU admission to VSD surgical closure was associated with lower mortality in patients undergoing surgery. Overall, critically ill patients with PMI-VSD may benefit from delayed VSD surgical closure. Optimal management of these unstable patients is yet to be defined but delayed surgery warrants further research.

Abbreviations

aOR	Adjusted odds-ratio
AFP	Intravascular micro-axial flow pump
CABG	Coronary artery bypass graft
CAD	Coronary artery disease
IABP	Intra-aortic ballon pump
ICU	Intensive care unit
MCS	Mechanical circulatory support
LV	Left ventricle
LVEF	Left ventricular ejection fraction
MCS	Mechanical circulatory support
MI	Myocardial infarction
OR	Odds-ratio
PCI	Percutaneous coronary intervention
pLVAD	Paracorporeal left ventricular assist device
SOFA	Sequential organ failure assessment
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
VA-ECMO	Veno-arterial extracorporeal membrane oxygenation
VSD	Ventricular septal defect

Supplementary Information

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Additional file 1.

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Author contributions

L-D.A, F.B, A.G and A.M-D designed the study. L-D.A, F.B, C.D, O.AA, C.B, S.P, T.MB, N.M, P.S, H.N, A.K, T.K, M.H, A.S collected the data.L-D.A, F.B and A.G conducted the statistical analysis.L-D.A, F.B, C.D, A.G, and A.M-D analyzed and interpreted the data.L-D.A, F.B, A.G, C.D and A.M-D wrote the manuscript. All authors critically reviewed and approved the final version of the manuscript.All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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Availability of data and materials

All data generated or analysed during this study are included in this published article [and its supplementary information files]. No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The study was approved by our Institutional Review Board (Ethics committee of the French Intensive Care Society (SRLF) CE SRLF 19–34) and was conducted in accordance with the French Data Protection Authority (CNIL

MR004 2224973) and with the Declaration of Helsinki. Written consent was not required due to the retrospective nature of the study.

Consent for publication
Written consent was not required due to the retrospective nature of the study.

Competing interests
The authors declare no competing interests.

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