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Hyperlactatemia as a Risk Stratification for Postinfarction Ventricular Septal Rupture

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ABSTRACT

Background: Surgical repair for postinfarction ventricular septal rupture (VSR) is a challenging operation, especially in the acute phase with unstable hemodynamics. We retrospectively examined the value of measuring lactate, which can be a quantitative indicator of end-organ ischemia caused by multiple factors including ineffective cardiac output, for risk stratification in patients undergoing surgical repair.

Methods: From April 2008 to December 2018, 33 consecutive patients were admitted to our hospital with a diagnosis of VSR and underwent VSR repair surgery within 24 hours of admission. We categorized patients into 3 groups based on the distribution of preoperative blood lactate level: <5 mmol/L (n = 16), 5 to 10 mmol/L (n = 11), and >10 mmol/L (n = 6).

Results: There was no intraoperative death, but the prognosis for patients with lactate >10 mmol/L was extremely poor, with a median postoperative survival time of only 23 days [25th percentile 6, 75th percentile 30]. Five of 6 patients (83%) died within 30 days after surgery. The risk-adjusted hazard ratio (95% confidence interval) for mortality per 1 SD (0.41-unit) increase in log-transformed preoperative lactate level (equivalent to a 4.10-fold increase) was 2.85 (1.57 to 5.19). Patients with lactate 5 to 10 mmol/L had 3.95-fold and those with lactate >10 mmol/L had 6.03-fold higher risk of mortality than those with lactate <5 mmol/L.

Conclusion: Elevation of preoperative serum lactate is significantly associated with mortality in patients who underwent VSR repair. The findings of this study highlight the value of measuring lactate level for risk stratification.

INTRODUCTION

Ventricular septal rupture (VSR) is a rare and often fatal complication of myocardial infarction [Crenshaw BS 2000; Birnbaum 2002; Moreyra 2010]. Surgical repair remains the treatment of choice, but it is a challenging operation carrying high early mortality, especially when attempted in the acute phase complicated with cardiogenic shock (CS) [Jones 2014; Daggett 1977; David 1995; Jeppsson 2005; Lundblad 2009; Arnaoutakis 2012]. According to the largest recent research using the database of the Society of Thoracic Surgeons, operative mortality was 54.1% when repairs were attempted within 7 days after myocardial infarction [Arnaoutakis 2012]. These patients tend to be in worse general condition, with unstable hemodynamics, preoperative intubation, or multiorgan dysfunction. In addition, repair surgery itself is technically difficult because of the fragile necrotic myocardium.

In such critically ill patients, a mechanical circulatory support device such as extracorporeal membrane oxygenation (ECMO) and Impella (Abiomed, Danvers, MA) might be a useful as a bridging therapy to stabilize hemodynamic conditions, improve end-organ malperfusion, and delay surgery [La Torre 2011; Rob 2017; Furui 2018; Matos 2020; Morimura 2020]. However, there are no clear criteria for selecting patients who should undergo emergency VSR repair and those who should undergo mechanically assisted bridging therapy. Risk stratification is crucial for promoting effective decision-making for VSR in the acute phase.

Here we focus on preoperative serum lactate concentration, which is used as a sensitive and quick indicator of end-organ malperfusion. Other parameters generated by arterial blood gas analysis such as pH and base excess are easily altered by prolonged therapy with potassium-wasting diuretics, steroids, or ingestion/overdose of sodium. These measurements are also easily affected by hyperventilation or chronic lung disease with CO2 retention (chronic obstructive pulmonary disease, respiratory depression from severe asthma, or pulmonary edema). In this study, we retrospectively analyzed our database to examine the association between preoperative serum lactate level and mortality in patients who underwent VSR repair.

METHODS

From April 2008 to December 2018, 33 consecutive patients were admitted to our hospital with a diagnosis of VSR and underwent VSR repair surgery within 24 hours of admission. No patient died or recovered without intervention. No patient received nonsurgical approaches such as trans-catheter closure, left ventricular assist device, or

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transplantation. The details of VSR repair have been reported previously [Asai 2012; Hosoba 2013; Asai 2016]. This study was approved by the institutional review board (no. R2018-189), and written informed consent was waived because of the retrospective design.

Outcome Measures and Definitions

Blood lactate concentrations were first measured at the time of induction of anesthesia, several times intraoperatively, at the time of admission to the intensive care unit after surgery, and then every 3 to 6 hours. Blood lactate level was analyzed by a Radiometer ABL800 Flex Analyzer (Radiometer Medical ApS, Bronshoj-Husum, Denmark). The primary outcome was 30-day (in-hospital) mortality. All data were obtained directly from electronic medical records at our hospital.

Data Analysis

According to the distribution of preoperative blood lactate level (Fig. 1), we categorized patients into 3 groups: low lactate, <5 mmol/L (n = 16); intermediate lactate, 5 to 10 mmol/L (n = 11); and high lactate, >10 mmol/L (n = 6). Linear relationships across the 3 groups were analyzed by use of the Mantel–Haenszel linear-by-linear association χ^2 test for binary variables and the Jonckheere–Terpstra test for continuous variables. After evaluation of normality by Kolmogorov–Smirnov tests, the natural log transform was used to normalize the distributions and improve the linearity. To further clarify the association between preoperative lactate and baseline characteristics, a multivariate linear regression was conducted. In an attempt to eliminate multicollinearity, variables that measured essentially the same thing were not entered into the model. Furthermore, multicollinearity between explanatory variables was reduced by centering the variables by subtracting the mean from individual values. Multicollinearity of the regression model was examined by calculating variance inflation factor for each variable. None of them exceeded 2 (greatest, 1.52), which indicates that multicollinearity was not a significant issue. Postoperative serum lactate levels were examined using linear mixed-effect models for repeated measures with random intercepts. The 95% confidence interval (CI) of the least-squares means was obtained to perform within-group and between-group pairwise comparisons. The estimated survival rates were calculated using the Kaplan–Meier method and compared using the log rank test. The association between mortality and preoperative serum lactate was estimated using the Cox proportional hazard model. Results are presented as adjusted hazard ratio (HR) with 95% CI derived from 1,000 bootstrap samples. Variables are reported as median [25th, 75th percentile] or number (percentage). Values of $P < 0.05$ were considered to indicate statistical significance. All statistical analyses were performed using the SPSS statistical package

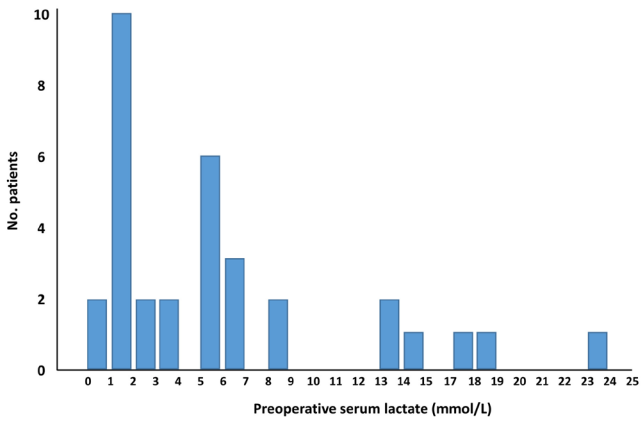

Figure 1. Distribution of preoperative serum lactate levels.

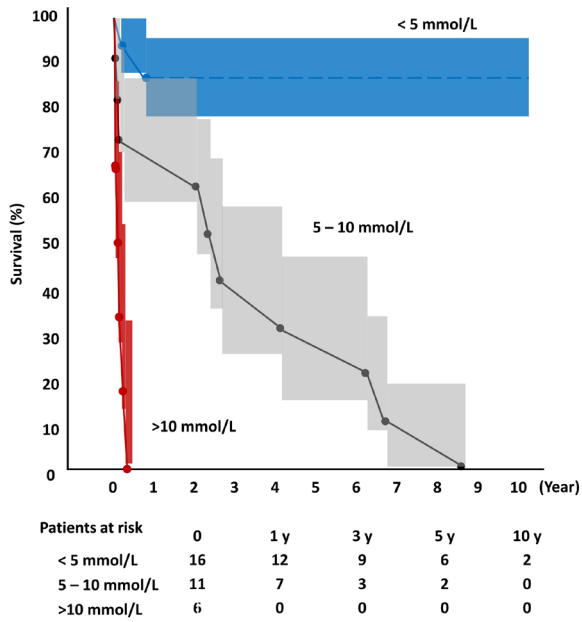

Figure 2. Kaplan–Meier survival curves stratified by preoperative serum lactate levels. Each symbol represents an event being traced. Shaded area shows 95% CI. Lower panel shows number at risk for each group. The P value of the log-rank test is 0.01.

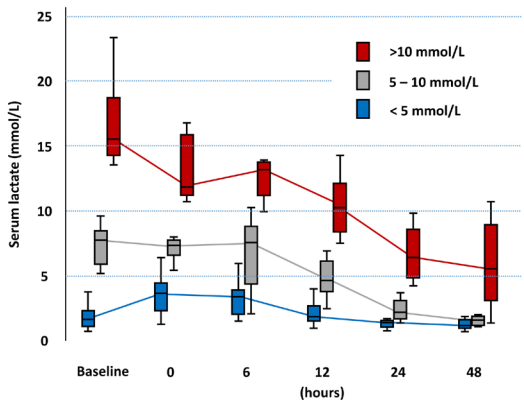

Figure 3. Postoperative changes of serum lactate levels. Boxes denote the lower and upper quartiles; whiskers denote the full range; and the horizontal bar denotes the median.

version 25.0 (SPSS, Chicago, IL) and R software (https://www.r-project.org/).

RESULTS

Baseline Characteristics

The overall mean age of this cohort was 79 years [74, 83]. The location of VSR was anterior in 21 patients and posterior in 12. Twenty-five patients (76%) received VSR repair within 1 week from the onset of myocardial infarction, and 18 patients within 24 hours. The distribution of preoperative lactate levels is shown in Table 1. Patients with higher preoperative lactate levels were older and more likely to have lower base excess, higher lactate dehydrogenase levels, and maximum creatine phosphokinase (CPK) levels. Five of 6 patients in the high-lactate group received intra-aortic balloon pump (IABP) support, and 2 received ECMO at admission; 2 were

Table 1. Baseline characteristics by preoperative lactate levels*

Characteristic	Preoperative Lactate (mmol/L)			*P* for Trend
	<5	5 to 10	>10	
Patients (n)	16	11	6	
Age (y)	74 [62, 82]	80 [77, 82]	84 [83, 88]	0.01
Male sex	9 (56)	4 (36)	2 (33)	0.27
Hypertension	9 (56)	5 (45)	4 (67)	0.84
Diabetes mellitus	6 (38)	4 (36)	3 (50)	0.66
Hyperlipidemia	6 (38)	2 (18)	2 (33)	0.63
Smoking	8 (50)	2 (18)	1 (17)	0.14
Estimated GFR (ml/min/1.73 m2)	52 [33. 70]	32 [28, 49]	26 [23, 37]	0.15
Atrial fibrillation	1 (6)	2 (18)	1 (17)	0.49
Intra-aortic balloon pump	11 (69)	8 (73)	5 (83)	0.52
Percutaneous cardiopulmonary support	0	0	2 (33)	0.07
Preoperative PCI for culprit lesion	8 (50)	5 (45)	1 (17)	0.21
Preoperative intubation	0	3 (27)	2 (33)	0.03
Anterior VSR	10 (63)	9 (82)	2 (33)	0.44
LV ejection fraction (%)	49 [43, 55]	48 [42, 56]	46 [40, 51]	0.15
Coronary artery disease				0.59
1-vessel disease	13 (81)	7 (64)	5 (83)	
2-vessel disease	2 (13)	2 (18)	0	
3-vessel disease	1 (6)	2 (18)	1 (17)	
VSR repair within 1 week after MI	10 (63)	9 (82)	6 (100)	0.16
Laboratory data				
Lactate during induction of anesthesia (mmol/L)	1.8 [1.1, 2.3]	7.7 [6.1, 8.2]	15.9 [14.0, 18.6]	0.01
Base excess	−1.1 [−3.9, 0]	−8.0 [−11.2, −6.5]	−12.6 [−15.0, −5.5]	0.01
Aspartate transaminase (IU/L)	110 [33, 337]	209 [80, 708]	1109 [174, 1290]	0.26
Alanine aminotransferase (IU/L)	47 [28, 111]	55 [28, 312]	544 [140, 794]	0.59
Lactate dehydrogenase (IU/L)	500 [350, 993]	587 [450, 1057]	1609 [855, 2816]	0.03
Alkaline phosphatase (IU/L)	227 [162, 308]	207 [170, 261]	322 [225, 380]	0.72
Total bilirubin (mg/dL)	0.67 [0.35, 0.84]	0.85 [0.59, 1.25]	1.26 [1.22, 1.33]	0.19
Brain natriuretic peptide (pg/mL)	523 [206, 1120]	816 [322, 1931]	742 [343, 1305]	0.39
Maximum creatine phosphokinase (IU/L)	701 [542, 2815]	1275 [886, 2810]	4234 [2008, 5656]	0.01

Data are median [25th, 75th percentile] or n (%).

*LV indicates left ventricle; MI, myocardial infarction; PCI, percutaneous coronary intervention.

intubated before transfer to our hospital. Multivariate linear regression analysis revealed that preoperative maximum CPK level was the only independent factor associated with preoperative lactate level (Supplementary Table S1).

Intra- and Postoperative Outcomes

There was no significant difference in concomitant procedures, operative time, cardiopulmonary bypass time, and cardioplegic arrest time between the 3 groups (Table 2). No patient needed a second pump run for residual VSR shunt. Regarding postoperative complications, except acute kidney disease requiring hemodialysis, no significant difference was observed in any variables. One patient underwent reoperation for recurrent VSR shunt 9 days after the first surgery. Intraoperatively, a new ventricular septal defect 1 cm in diameter was identified more basally from the patch placed in the first operation. No dehiscence was found in the suture line of that patch, and a new patch was placed.

Mortality

Estimated survival stratified by preoperative lactate is shown in Fig. 2. There was no intraoperative death, but the prognosis for the high-lactate group was extremely poor, with a median postoperative survival time of only 23 days [6, 30]. Five of 6 in the high lactate group (83%) died within 30 days after surgery. Of these, 3 died of multiorgan failure due to prolonged low cardiac output syndrome before surgery. The other 2 died of subdural hemorrhage and nonocclusive mesenteric ischemia. The 30-day mortality in the intermediate-lactate group (27%, 3/11) was significantly better than that in the high-lactate group (P = 0.01), but deaths were observed even after discharge, and the survival was significantly worse than that in the low-lactate group (P = 0.01). Causes of death within 30 days in the intermediate-lactate group were diffuse alveolar hemorrhage, massive gastrointestinal hemorrhage, and nonocclusive mesenteric ischemia. In the low-lactate group, except for 1 patient who died of pneumonia on

Table 2. Intraoperative Outcomes and Postoperative Complications*

Event	Preoperative Lactate (mmol/L)			*P* for Trend
	<5	5 to 10	>10	
Patients (n)	16	11	6	
Coronary bypass surgery	7 (44)	4 (36)	2 (33)	0.76
Number of coronary bypass grafts				
0	9 (56)	7 (64)	4 (67)	
1	4 (25)	1 (9)	1 (17)	
2	2 (13)	2 (18)	1 (17)	
3	1 (6)	1 (9)	0	
Aortic valve replacement	1 (6)	1 (9)	0	0.65
Mitral valve repair	1 (6)	0	0	0.22
Operation time (min)	208 [182, 277]	252 [210, 278]	281 [235, 313]	0.29
Cardiopulmonary bypass time (min)	109 [102, 141]	124 [102, 173]	149 [127, 166]	0.17
Cardioplegic arrest time (min)	80 [64, 115]	82 [58, 96]	96 [81, 103]	0.79
Postoperative complication				
Re-exploration for bleeding	0	1 (9)	1 (17)	0.13
Residual VSR shunt	1 (6)	0	0	0.22
Acute kidney disease requiring hemodialysis	1 (6)	3 (27)	3 (50)	0.02
New cerebral infarction	1 (6)	0	0	0.22
Permanent pacemaker implantation	0	1 (9)	0	0.69
Mediastinitis requiring surgical debridement	0	0	0	0.99
Pneumonia requiring antibiotic therapy	3 (19)	4 (36)	2 (33)	0.82
Mortality				
In-hospital mortality	1 (6)	3 (27)	6 (100)	0.01
30-d mortality	0	3 (27)	5 (83)	0.01

Data are median [25th, 75th percentile] or n (%).

*VSR indicates ventricular septal rupture.

postoperative day 72, all patients were discharged alive, and postdischarge survival was excellent except for 1 who died of aortic aneurysm rupture at 1 year.

The results of bivariate comparisons between patients with and without 30-day mortality are summarized in Table 3. Patients who died within 30 days were significantly older, had lower estimated glomerular filtration rate (GFR) at admission, and had higher preoperative serum lactate. The following variables with *P* value of <0.25 were included in the multivariate Cox model (Table 4): higher age, male sex, smoking, estimated GFR, preoperative ECMO, preoperative intubation, preoperative lactate, maximum CPK, and brain natriuretic peptide. HR (95% CI) for mortality per 1 SD (0.41-unit) increase in log-transformed preoperative lactate level (equivalent to a 4.10-fold increase) was 2.85 (1.57 to 5.19). Patients with lactate 5 to 10 mmol/L had a 3.95-fold

Table 3. Bivariate Analysis of 30-Day Mortality*

Characteristic	30-Day Mortality		*P*
	Yes	No	
Patients (n)	8	25	
Age (y)	84 [77, 89]	77 [66, 82]	0.03
Male sex	2 (25)	13 (52)	0.24
Hypertension	5 (63)	13 (52)	0.69
Diabetes mellitus	3 (38)	10 (40)	0.99
Hyperlipidemia	2 (25)	8 (32)	0.99
Smoking	1 (13)	10 (40)	0.22
Estimated GFR (ml/min/1.73 m2)	29 [25, 37]	48 [32, 67]	0.04
Atrial fibrillation	0	4 (16)	0.55
Intra-aortic balloon pump	6 (75)	18 (72)	0.87
Percutaneous cardiopulmonary support	2 (25)	0	0.07
Preoperative PCI for culprit lesion	3 (38)	11 (44)	0.99
Preoperative intubation	3 (38)	2 (8)	0.06
Anterior wall VSR	4 (50)	17 (68)	0.42
LV ejection fraction (%)	47 [41, 53]	48 [42, 55]	0.78
Coronary artery disease			0.48
1-vessel disease	7 (88)	18 (72)	
2-vessel disease	0	4 (16)	
3-vessel disease	1 (13)	3 (12)	
VSR repair within 1 week after MI	7 (88)	18 (72)	0.64
Lactate during induction of anesthesia, mmol/L	9.4 [2.3, 14.3]	1.9 [1.3, 4.8]	0.01
0 to 4.9	0	16 (64)	
5.0 to 9.9	3 (38)	8 (32)	
>10.0	5 (63)	1 (4)	
Aspartate transaminase (IU/L)	142 [29, 729]	159 [42, 362]	0.57
Alanine aminotransferase (IU/L)	86 [29, 159]	48 [27, 160]	0.88
Lactate dehydrogenase (IU/L)	770 [514, 1249]	540 [350, 1016]	0.29
Alkaline phosphatase (IU/L)	249 [166, 399]	215 [162, 286]	0.82
Total bilirubin (mg/dL)	1.28 [1.19, 1,81]	0.65 [0.37, 0.84]	0.11
Brain natriuretic peptide (pg/mL)	1506 [469, 1931]	463 [223, 1005]	0.18
Maximum creatine phosphokinase (IU/L)	3743 [1009, 5091]	1036 [561, 2810]	0.26

Data are median [25th, 75th percentile] or n (%).

*LV indicates left ventricle; MI, myocardial infarction; VSR, ventricular septal rupture.

Table 4. Multivariate Cox Hazard Model for All-Cause Mortality

Variable	Hazard Ratio	95% CI*	*P*
Lactate during induction of anesthesia			
<5 mmol/L	1	Reference	
5 to 10 mmol/L	3.95	1.25 to 12.51	0.03
>10 mmol/L	6.03	2.51 to 14.46	0.01
Age (per 10-y increment)	1.38	0.96 to 1.98	0.08
Male sex	0.56	0.25 to 1.27	0.12
Smoker	0.46	0.13 to 1.63	0.19
Estimated GFR (per 10-ml/min/1.73 m2 increment)	0.98	0.89 to 1.09	0.39
Percutaneous cardiopulmonary support	0.74	0.13 to 4.26	0.74
Preoperative intubation	1.65	0.51 to 5.31	0.40
Brain natriuretic peptide†	3.69	0.89 to 15.31	0.08
Maximum creatine phosphokinase†	1.33	0.15 to 11.53	0.79

Hosmer–Lemeshow goodness-of-fit test, P = 0.38; 2 statistics = 0.67.

*Estimated from 1,000 bootstrap samples.

†For each 1-unit increment of log-transformed biomarker levels.

Table 5. Change of Lactate Level after Surgery

Lactate (mmol/L)	Time after surgery (h)				
	0	6	12	24	48
>10	13.2 (12.4, 13.9)*†	12.6 (12.1, 13.1)*†	10.8 (10.1, 11.6)*†	7.7 (7.2, 8.3)*†	5.8 (5.1, 6.5)*†
5 to 10	7.6 (7.1, 8.2)†	7.9 (7.3, 8.4)†	4.3 (3.9, 4.7)†	2.9 (2.4, 3.3)†	1.5 (1.1, 2.0)
<5	4.2 (3.7, 4.7)	4.0 (3.5, 4.5)	2.7 (2.4, 3.1)	1.1 (0.8, 1.4)	1.2 (0.9, 1.4)

Data are expressed as least square means (95% CI). Statistical significance of between-group comparisons is expressed as *P < 0.01 versus lactate 5 to 10 mmol/L and †P < 0.01 versus lactate <5 mmol/L. Within-group changes (95% CI) were significant from 0 to 48 h in patients with lactate >10 mmol/L [−7.5 mmol/L (−9.1, −6.0), P < 0.01]; lactate 5 to 10 mmol/L [−6.0 (−7.1, −5.0), P < 0.01]; and lactate <5 mmol/L [−3.0 mmol/L (−3.8, −2.3), P < 0.01].

higher risk of 30-day mortality, and those with lactate >10 mmol/L had a 6.03-fold higher risk than those with lactate <5 mmol/L. No interaction term was significantly associated with mortality and preoperative lactate.

Postoperative Lactate Change

Fig. 3 summarizes postoperative serum lactate changes. Measured lactate levels immediately after surgery were significantly higher in patients with higher preoperative lactate levels (*P* = 0.01). Thereafter, lactate levels begun to decrease after 6 hours in all 3 groups. By 48 hours, the difference between the intermediate- and low-lactate groups disappeared, and lactate levels dropped to almost normal range. In the high-lactate group, lactate levels continued to decrease over time, but hyperlactatemia persisted, and the median lactate level still exceeded 5 mmol/L at 48 hours. Statistical output of mixed-effect model analysis for within- and between-group comparisons is demonstrated in Table 5.

DISCUSSION

In this study, we investigated the association between preoperative serum lactate level and mortality in patients who underwent VSR repair. The results showed that elevated preoperative lactate levels were independently associated with increased mortality.

Cardiogenic shock (CS) has been reported to be significantly associated with a higher risk of mortality in patients undergoing VSR repair [Crenshaw 2000; Arnaoutakis 2012; Menon

Supplemental Table S1. Patient Factors Associated with Preoperative Lactate Levels*

Factor	Unstandardized Coefficient		Standardized Coefficient		
	B	SE	β	t	*P*
Age	0.009	0.01	0.257	0.918	0.37
Male	0.465	0.312	0.584	1.492	0.15
Hypertension	0.041	0.189	0.052	0.219	0.83
Diabetes mellitus	–0.042	0.145	–0.052	–0.292	0.77
Smoking	–0.496	0.337	–0.608	–1.474	0.16
Estimated GFR	0.004	0.004	0.208	0.941	0.36
Anterior wall VSR	0.085	0.184	0.102	0.461	0.65
No. diseased coronary	0.172	0.142	0.311	1.219	0.24
Surgery within 1 wk after MI	–0.172	0.24	–0.194	–0.714	0.49
CPK (maximal value)	0.785	0.263	0.839	2.988	0.01
Brain natriuretic peptide	0.254	0.222	0.3	1.142	0.27

*CPK indicates creatine phosphokinase; MI, myocardial infarction; SE, standard error; VSR, ventricular septal rupture. χ^2 statistics = 0.71. Preoperative lactate, creatine phosphokinase, and brain natriuretic peptide are log transformed.

2000; Labrousse 2002; Barker 2003], but the definitions of CS vary depending on investigators. Furthermore, the majority of patients with VSR have already received IABP support with large doses of catecholamines before surgery, and it is not easy to diagnosis CS based on blood pressure, cardiac index, left ventricular filling pressure, or urine output. The generally accepted definition of CS is a clinical condition in which ineffective cardiac output, due to a primary cardiac dysfunction, causes inadequate tissue (end organ) perfusion. End-organ ischemia observed in patients with VSR is multifactorial, and its extent is affected by many factors such as the amount of left-to-right shunt, myocardial dysfunction due to ischemia, arrhythmia, anemia, diabetic vasculopathy, chronic kidney disease, and pulmonary disease. We believe that the advantage of measuring lactate is that the severity of end-organ malperfusion can be summarized by a single index. The main clinical takeaway of the present study is that the prognosis of patients who already had severe hyperlactatemia (>10 mmol/L) before surgery was extremely poor, despite the elimination of VSR shunt by emergency surgery. Of course, one should not rush to judgment about whether VSR repair should be delayed in every patient with severe hyperlactatemia. However, it may be necessary to consider watchful waiting to gain time for some improvement in the hemodynamics and improve end-organ ischemia using a mechanical circulator.

Using ECMO as a bridging therapy has been reported in recent years [Rob 2017, Furui 2018; Matos 2020; Morimura 2020]. Morimura et al [2020] reported that ECMO was used safely to delay surgery in 8 acute VSR patients with cardiogenic shock. They showed that the introduction of mechanical assistance markedly improved laboratory data, including serum lactate. When using mechanical assistance, the question is how long it should continue. It is clear that the mechanical assistance should be as short as possible to avoid hemorrhagic or neurological complications.

Limitations

The main limitation of our analysis is the inability to address hidden biases due to unobserved or unrecorded variables. As a result, our results may reflect the effects of unknown or unmeasured confounders. Our cohort represents a single-center and single-surgeon experience, limiting its generalizability. Our surgical series of 33 patients may not provide adequate power for robust statistical comparisons. The cutoff values used for preoperative lactate (5 and 10 mmol/L) are somewhat arbitrary, but risk stratification by preoperative lactate was statistically significant when these criteria were examined as continuous variables, suggesting that risk increases with increasing levels of preoperative lactate and is not dependent on the choice of specific cutoff values. Additionally, risk stratification remained statistically significant even if alternative thresholds of 2 and 8 mmol/L were used, further suggesting that the overall predictive value of these measures is not strongly dependent on the precise cutoff values selected.

Conclusions

Elevation of preoperative serum lactate was significantly associated with mortality in patients who underwent VSR repair. The findings of this study highlight the value of measuring lactate level for risk stratification.

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