

# The Effect of Serum Lactate Level and Shock Index on Morbidity and Mortality in Aortic Dissection and Aneurysm

© Hakan Çelik, © Abuzer Coşkun, © Burak Demirci, © Betül Çam, © Burak Akın

University of Health Sciences Turkey, Bağcılar Training and Research Hospital, Clinic of Emergency Medicine, İstanbul, Turkey

## Abstract

**Objective:** Acute aortic dissections and aneurysms are vascular emergencies and have high mortality rates. Since early diagnosis is valuable in terms of mortality and prognosis, the effects of shock index (SI) and lactate levels on mortality and prognosis in patients with aortic dissection or aneurysm were investigated.

**Materials and Methods:** The study was conducted by retrospectively examining 71 patients older than 18 years, who were diagnosed with aortic dissection or aneurysm, who applied to the emergency department between January 01, 2015, and December 31, 2020. The patients were divided into two groups in terms of dissection and aneurysm. Three groups were defined for dissections and two groups for aneurysms. Age, gender, lactate level, SI, and mortality of the patients were evaluated.

**Results:** The mean age of the 71 patients included in the study was  $62.08 \pm 13.80$  years, 15 (21.1%) were female, and the age range was 32-94 years. Lactate levels of aortic dissection and abdominal aneurysm cases were  $3.22 \pm 2.09$  mmol/L and  $1.90 \pm 1.51$  mmol/L, respectively ( $p=0.004$ ). The mean shock indices of patients with aortic dissection and aneurysms were  $0.71 \pm 0.18$  and  $0.63 \pm 0.15$ , respectively ( $p=0.018$ ). While 19 (54.2%) of 35 cases with aortic dissection survived, 16 (45.8%) died. While 30 (83.3%) of the aortic aneurysm cases survived, 6 (25%) died ( $p=0.008$ ).

**Conclusion:** SI and serum lactate levels may be prognostic values in terms of morbidity and mortality in aortic dissection and/or aortic aneurysm.

**Keywords:** Emergency department, aortic dissection, aortic aneurysm, SI, serum lactate level

## Introduction

Acute aortic dissections (AAD) are a dramatic life-threatening disease. It is defined as the separation of the media and intima layers of the aorta along the long axis of the vessel. It is one of the leading diseases in terms of mortality and morbidity. Aortic dissection, one of the most urgent cardiovascular diseases, requires rapid diagnosis and treatment. Appropriate diagnosis and treatment methods should be used to prevent this sudden-onset pathology from becoming fatal. AAD are estimated to occur with a frequency of 5-10/1,000,000 in the United States [1]. Unfortunately, there is no large-scale incidental study in our country. Mortality in the first two weeks is approximately 80-90% in patients with AAD due to failure to diagnose or delay in treatment. It has been reported that mortality increases

by approximately 1-3% per hour in the first two days [2]. The incidence of the disease is higher in men than in women. Many predisposing factors and etiological causes can be shown as precursors for damage to the aorta and dissection. These etiological and predisposing factors are currently accepted under seven main headings. These; hypertension, medial degenerative disease, congenital anomalies, atherosclerosis, inflammatory diseases, trauma, and pregnancy [3]. According to DeBakey's et al. [2] definition of dissection, it is defined as acute in the first 14 days, subacute between 15 days and 60 days, and chronic if it lasts longer than 2 months.

Clinical suspicion is the most important step for the diagnosis of aortic dissection, which is one of the most life-threatening diseases. In reaching the diagnosis, anamnesis, physical



Address for Correspondence: Hakan Çelik MD, University of Health Sciences Turkey, Bağcılar Training and Research Hospital, Clinic of Emergency Medicine, İstanbul, Turkey

Phone: +90 212 440 40 00 E-mail: dr.celik91@gmail.com ORCID-ID: orcid.org/0000-0001-9500-8440

Received: 21.03.2022 Accepted: 28.03.2022

© Copyright 2022 by the Turkish Emergency Medicine Foundation, Global Emergency and Critical Care published by Galenos Publishing House.

examination and telecardiography are important steps. However, the specificity of telecardiography in the diagnosis of aortic dissection is low [4]. Clinically, typical symptoms of aortic dissection are sudden onset, tearing, severe, chest pain that starts from the back and neck and spreads to the jaw [5]. However, patients with aortic dissection do not always present with these classic symptoms. It has been reported that some patients did not describe any pain, and some patients with pain did not present with classical complaints. Although diagnosis is delayed in these patients who do not present with classic symptoms and have atypical complaints, there is an increase in mortality rates [6].

Patients diagnosed with AAD and aneurysm have high morbidity and mortality rates. Therefore, it was determined the effect of shock index (SI) and serum lactate levels on mortality in patients with aortic dissection and aneurysms who applied to the emergency department.

## Materials and Methods

### Population and Laboratory Design

In this retrospective study, the data of 71 patients (mean age  $62.08 \pm 13.79$  years, mean range 32-94 years) diagnosed with aortic dissection and aneurysm in the emergency department between January 01, 2015, and December 31, 2020, were included. The patients were selected from those whose clinical findings, vital signs, serum lactate levels, and diagnostic data were recorded in the hospital data recording system. Patients under the age of 18 and patients with insufficient data were excluded from the study.

Age, gender, SI, serum lactate levels, hematological parameters, and urea and creatinine values were recorded from the patient files. Hemograms were measured using a Beckman Coulter Automated CBC Analyzer (Beckman Coulter, Inc., Fullerton, CA, USA). Biochemistry blood was analyzed with a Cobas 6000 (C6000-Core, Cobas c-501 series, Hitachi, Roche, USA). Venous blood gas samples were taken for lactate level. Patients' lactate levels were obtained from venous blood gas analyses using the Cobas® b221 blood gas system (Roche, Basel, Switzerland).

The SI was calculated based on the vital signs of the patient at the time of admission to the emergency room. The formula "SI: Heart rate (HR) (HR/minute)/systolic blood pressure (SBP) (mmHg)" was used.

Dissection types were grouped according to the DeBakey (types 1, 2, and 3) classification. According to the DeBakey classification, type 1 dissection is seen in the entire aorta, type 2 dissection is seen only in the ascending aorta, and type 3 dissection is seen in the descending aorta [7]. The aneurysm patients were determined as ascending and descending. Groups were determined according to mortality or not.

The study was conducted in accordance with the Helsinki Declaration of Human Research after the approval of the Health Sciences University, Kanuni Sultan Suleyman Training and Research Hospital Ethics Committee (subject number: KAEK/2022.02.41, decision no: 41, date: 23.02.2022). After all, patients were informed, their consent was obtained for inclusion in the study.

### Statistical Analysis

The data obtained from the study were analyzed with the SPSS 20 (SPSS Inc., Chicago, IL, USA) package program. The Kolmogorov-Smirnov test was used while investigating the normal distributions of the variables. Descriptive statistics and continuous variables were presented as mean  $\pm$  standard deviation or median (minimum-maximum) and nominal variables were presented as many cases and percentage (%). While examining the differences between the groups, Mann-Whitney U and Kruskal-Wallis H tests were used because the variables did not come from a normal distribution. Chi-square analysis was performed when examining the relationships between groups of nominal variables. Spearman's rho analysis was used for correlation with variables. Receiver operating characteristic (ROC) curve was used for sensitivity and specificity analysis of dissection, aneurysm, and mortality. When interpreting the results, values below the significance level of 0.05 were considered statistically significant.

## Results

The mean age of all patients was  $62.08 \pm 13.79$  years, and the mean range was 32-94 years. The mean age of patients with aortic dissection or aortic aneurysm was  $56.74 \pm 14.01$  years and  $67.27 \pm 11.56$  years, respectively ( $p=0.001$ ). The mean serum lactate levels of the cases were  $3.22 \pm 2.09$  mmol/L and  $1.90 \pm 1.51$  mmol/L, respectively ( $p=0.004$ ). The mean shock indices of the cases were  $0.71 \pm 0.18$  and  $0.63 \pm 0.15$ , respectively ( $p=0.018$ ). The mean hematocrit values of the patients were found to be  $35.58 \pm 6.51\%$  and  $32.46 \pm 6.07\%$ , respectively ( $p=0.046$ ). Seventy one patients were included in the study. Fifty six of these cases (78.8%) were male. The relationship between gender and group was not significant ( $p=0.132$ , Table 1).

In the subtypes of aortic dissection in the study, the mean ages for type 1, type 2, and type 3 dissections were  $59.14 \pm 13.97$  years,  $63.42 \pm 10.75$  years, and  $42.85 \pm 6.96$  years, ( $p=0.001$ ), serum lactate levels were  $3.49 \pm 2.05$  mmol/L,  $2.65 \pm 1.80$  mmol/L, and  $2.95 \pm 2.61$  mmol/L, ( $p=0.025$ ) and SIs of the cases were  $0.75 \pm 0.19$ ,  $0.64 \pm 0.19$ , and  $0.66 \pm 0.13$ , respectively ( $p=0.032$ ). Of 21 patients with type 1 dissection, 9 (42.8%) survived, while 12 (57.8%) died. While 5 (71.4%) of 7 cases with type 2 dissection were alive, 2 (28.6%) died. While 5 (71.4%) of 7 patients with type 3 dissection were alive, 2 (28.6%) died ( $p=0.020$ , Table 2).

**Table 1. Evaluation of age, gender, shock index and laboratory variables with aneurysm and dissection groups**

	All patient mean ± SD	Dissection mean ± SD	Aneurysm mean ± SD	p value*	
Age (year)	62.08±13.79	56.74±14.01	67.27±11.56	<b>0.001</b>	
Lactate (mmol/L)	2.55±1.93	3.22±2.09	1.90±1.51	<b>0.004</b>	
Shock index	0.67±0.17	0.71±0.18	0.63±0.15	<b>0.018</b>	
Urea (mg/dL)	50.98±34.90	48.27±24.52	53.61±42.84	0.845	
Creatinine (mg/dL)	1.34±1.07	1.24±0.63	1.43±1.37	0.516	
Sodium (mmol/L)	140.25±6.60	140.44±7.12	140.07±6.11	0.945	
Potassium (mmol/L)	4.16±0.65	4.09±0.61	4.23±0.69	0.486	
WBC (×10 <sup>3</sup> /mcl)	12.95±6.20	14.25±7.37	11.69±4.56	0.110	
Hemoglobin (g/dL)	11.16±2.24	11.66±2.27	10.67±2.12	0.086	
Hematocrit (%)	34.00±6.44	35.58±6.51	32.46±6.07	<b>0.046</b>	
MCV (fL)	87.29±5.70	87.44±5.83	87.15±5.64	0.730	
MCHC (g/dL)	32.23±1.68	32.06±1.46	32.39±1.87	0.309	
PLT (×10 <sup>3</sup> /mcl)	207.59±108.82	220.64±130.9	194.91±81.82	0.585	
Gender	n (%)	n (%)	n (%)	p value**	
	Male	56 (78.8)	25 (71.4)		31 (86.1)
	Female	15 (21.2)	10 (28.6)		5 (13.9)
	Total	71 (100)	35 (100)		36 (100)

\*Mann-Whitney U test, \*\*Chi-square analysis, WBC: White blood cell, MCV: Mean cell volume, MCHC: Mean corpuscular hemoglobin concentration, PLT: Platelet, SD: Standard deviation

**Table 2. Relationship of variables with aortic dissection types**

	Type 1 dissection mean ± SD	Type 2 dissection mean ± SD	Type 3 dissection mean ±SD	p value*	
Age (year)	59.14±13.97	63.42±10.75	42.85±6.96	0.001	
Lactate (mmol/L)	3.49±2.05	2.65±1.80	2.95±2.61	0.025	
Shock index	0.75±0.19	0.64±0.19	0.66±0.13	0.032	
Urea (mg/dL)	52.83±25.12	47.61±26.79	35.25±17.78	0.364	
Creatinine (mg/dL)	1.37±0.74	1.01±0.36	1.10±0.43	0.568	
Sodium (mmol/L)	139.70±6.72	141.85±9.75	141.28±6.07	0.807	
Potassium (mmol/L)	4.14±0.60	4.24±0.50	3.79±0.72	0.501	
WBC (×10 <sup>3</sup> /mcl)	13.61±5.74	10.68±3.83	19.72±11.48	0.073	
Hemoglobin (g/dL)	11.55±2.02	12.02±2.55	11.62±3.01	0.367	
Hematocrit (%)	35.36±5.48	36.91±7.08	34.91±9.34	0.245	
MCV (fL)	87.19±6.34	88.40±4.94	87.22±5.73	0.956	
MCHC (g/dL)	31.90±1.52	31.54±1.28	33.07±1.10	0.151	
PLT (×10 <sup>3</sup> /mcl)	206.14±125.74	232.00±123.53	252.78±164.64	0.772	
Mortality	n (%)	n (%)	n (%)	p value	
	No	9 (42.8)	5 (71.4)		5 (71.4)
	Yes	12 (57.8)	2 (28.6)		2 (28.6)
	Total	21 (100)	7 (100)		7 (100)

\*Kruskal-Wallis H test, \*\*Chi-square analysis, WBC: White blood cell, MCV: Mean cell volume, MCHC: Mean corpuscular hemoglobin concentration, PLT: Platelet

The mean age in ascending and descending aortic aneurysms was 50.33±20.84 years and 68.81±9.47 years, respectively (p=0.001). Serum lactate levels were 1.33±0.32 mmol/L and 1.95±1.57 mmol/L (p=0.013). Mean corpuscular volume (MCV)

values were 78.06±3.10 fL and 87.97±5.07 fL (p=0.034). The mean corpuscular hemoglobin concentration (MCHC) values were 29.90±0.62 g/dL and 32.62±1.77 g/dL (p=0.012). While 3 (100%) of 3 patients with ascending aneurysms survived, 6

(18.2) of 33 patients with descending aneurysm died (p=0.024, Table 3).

While 19 (54.2%) of 35 cases with aortic dissection in the study survived, 16 (45.8%) resulted in mortality. Thirty (83.3%) patients with aortic aneurysm survived, while 6 (16.6%) died (p=0.008, Table 4).

In the correlation analysis, there was a strong negative correlation between age and dissection, and a strong positive correlation between aneurysm. Additionally, both lactate and

SI had a strong positive correlation with mortality (Table 5).

According to the ROC curve analysis of the patients, the optimal cut-off values of lactate and SI [area under the curve (AUC): 95% confidence interval (CI): 95%] to determine the positivity of dissection, aneurysm and mortality;

**1. Dissection; Lactate:** Sensitivity 77.1% and specificity 63.9%; (AUC; 0.700, 95% CI; 0.573-0.826, p=0.004). **SI:** Sensitivity 74.3% and specificity 61.3%; (AUC; 0.663, 95% CI; 0.534-0.793, p=0.018 (Figure 1).

**Table 3. Relationship of variables with aortic dissection types**

	Ascending mean ± SD		Descending mean ± SD	p value*
Age (year)	50.33±20.84		68.81±9.47	<b>0.001</b>
Lactate (mmol/L)	1.33±0.32		1.95±1.57	<b>0.013</b>
Shock index	0.58±0.03		0.63±0.15	0.056
Urea (mg/dL)	37.66±25.71		55.06±44.05	0.574
Creatinine (mg/dL)	0.76±0.32		1.49±1.41	0.312
Sodium (mmol/L)	133.66±8.50		140.65±5.66	0.386
Potassium (mmol/L)	4.22±0.78		4.23±0.70	0.769
WBC (×10 <sup>3</sup> /mcl)	12.60±3.63		11.61±4.67	0.248
Hemoglobin (g/dL)	10.56±2.97		10.68±2.08	0.225
Hematocrit (%)	33.43±9.19		32.37±5.91	0.136
MCV (fL)	78.06±3.10		87.97±5.07	<b>0.034</b>
MCHC (g/dL)	29.90±0.62		32.62±1.77	<b>0.012</b>
PLT (×10 <sup>3</sup> /mcl)	195.33±76.69		194.87±83.39	0.841
	n (%)		n (%)	p value**
Mortality	No	3 (100)	27 (81.8)	<b>0.024</b>
	Yes	0 (0)	6 (18.2)	

\*Mann-Whitney U test, \*\*Chi-square analysis, WBC: White blood cell, MCV: Mean cell volume, MCHC: Mean corpuscular hemoglobin concentration, PLT: Platelet

**Table 4. Mortality comparison in aortic dissection and aneurysm**

		Dissection n (%)	Aneurysm n (%)	All patients n (%)	p value*
Mortality	No	19 (54.2)	30 (83.3)	49 (69.1)	0.132
	Yes	16 (43.8)	6 (16.6)	24 (30.9)	
Total		35 (100)	36 (100)	71 (100)	-

\*Chi-square analysis

**Table 5. Correlation analysis between variables**

	Dissection		Aneurysm		Mortality	
	r	p*	r	p*	r	p*
Gender	0.119	0.328	-0.196	0.101	0.194	0.105
Age	<b>-0.412</b>	<b>0.001</b>	<b>0.455</b>	<b>0.001</b>	0.047	0.694
Lactate	<b>0.279</b>	<b>0.019</b>	<b>-0.329</b>	<b>0.005</b>	<b>0.741</b>	<b>0.001</b>
Shock index	0.195	0.105	<b>-0.268</b>	<b>0.024</b>	<b>0.768</b>	<b>0.001</b>

\*Spearman's rho analysis

**2. Aneurysm; Lactate:** Sensitivity 52.8% and specificity 48.6%; (AUC; 0.300, 95% CI; 0.174-0.427, p=0.004). SI: Sensitivity 47.2% and specificity 45.8%; (AUC; 0.337, 95% CI; 0.207-0.466, p=0.018 (Figure 2).

**3. Mortality; Lactate:** Sensitivity 98% and specificity 95.2%; (AUC; 0.968, 95% CI; 0.915-0.1020, p=0.001). SI: Sensitivity

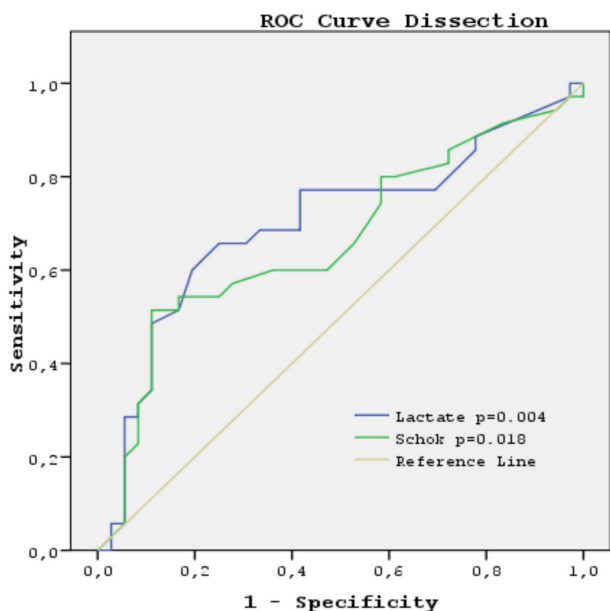
96.7% and specificity 92.2%; (AUC; 0.980, 95% CI; 0.955-0.1005, p=0.001 (Figure 3).

**Discussion**

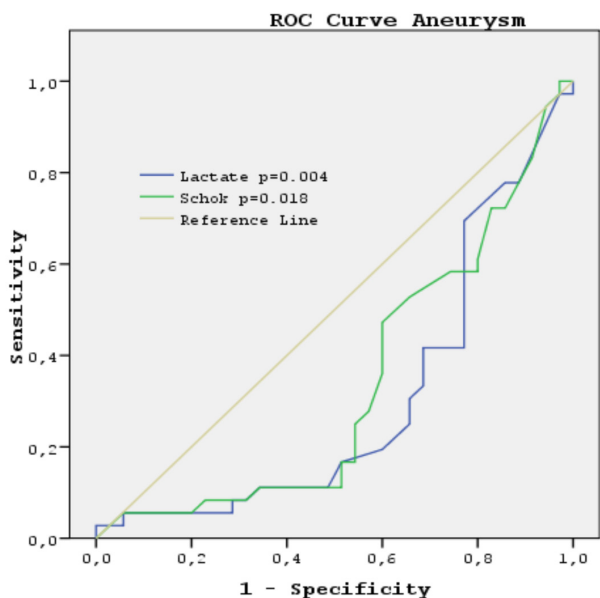
In aortic aneurysm and dissection, which is an important etiology for admission to emergency services, while seconds are so precious for the survival of the patient, providing faster and more practical approaches in diagnosis and treatment will bring the chance for rapid intervention and provide a significant reduction in morbidity and mortality.

Nutrition and oxygenation of the outer half of the aorta with the media layer are provided by vasa vasorum. Developing hypoxemia causes degeneration in the elastic structures in the tunica media, weakening the wall and paving the way for developing an aneurysm [8,9]. Aortic dissection results from a tear in the aortic intima because of pulsatile blood flow in the medial layer. Progressive separation of the aortic wall layer results in the formation of a false lumen, and re-entry into the true lumen via another intimal tear may also occur. This leads to rapid blood loss and death [10,11]. At the molecular level, aortic dissection is the result of remodeling of the aortic wall structure because of inflammation and extracellular matrix disruption. Activated macrophages infiltrate the tunica environment and release matrix metalloproteinases and proinflammatory cytokines [12].

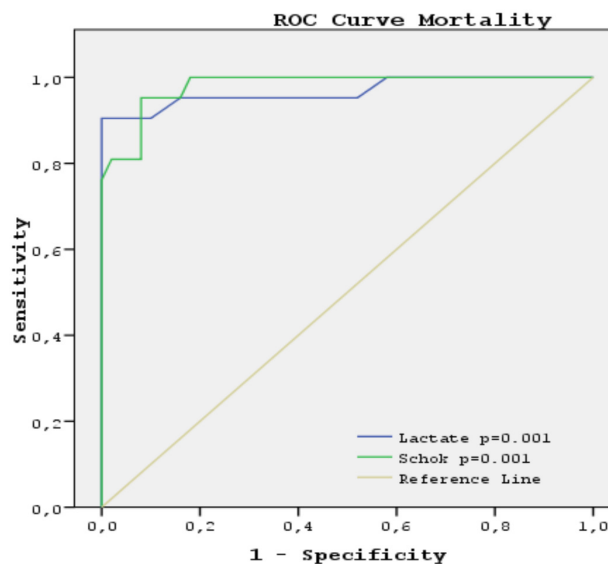
Age is considered an independent factor in revealing vascular damage [13]. It is predicted that cardiovascular diseases will continue to be the leading cause of death in the greater than 65 age group. Aortic dissection is most common in this population



**Figure 1.** ROC curve analysis according to lactate V and shock index acute aortic dissection positivity  
ROC: Receiver operating characteristic



**Figure 2.** ROC curve analysis according to lactate V and shock index aortic aneurysm positivity  
ROC: Receiver operating characteristic



**Figure 3.** ROC curve analysis according to lactate V and shock index mortality positivity  
ROC: Receiver operating characteristic



aged 65-75 years, with an incidence of 35 cases per 100,000 people per year [14]. The most important risk factor for its development is hypertension. Additionally, aortic dissection is seen 2-3 times more frequently in men than in women of the same age. The incidence of this disease under the age of 40 years is relatively rare, except family history, congenital heart diseases such as bicuspid aortic valve or aortic coarctation, Marfan syndrome, and pregnancy. In the literature, it is stated that the male/female ratio in relation to gender is 2-5/1. Again, in studies, it is seen more frequently in the age of 40-70 [2]. In the studies of Sbarouni et al. [15] it was reported that the mean age of patients with aortic dissection was  $63\pm 14$  years, and the female/male ratio was 89/31. In our study, 56 (78.8%) of the patients were male and 15 (21.2%) were female. The mean age and gender distribution of our cases were similar to that of other studies.

In a study, hemogram values in dissection patients were evaluated whether they have any prognostic value in predicting hospitalizations and long-term mortality [16]. The MCV value makes a significant difference in the mortality rate in aortic dissection [17]. In the study by Hirst et al. [17] mortality was found to be higher in patients with distal type, malperfusion, or ruptured aortic dissection patients aged 70 and over. In our study, MCV and MCHC values in the patient group with ascending aortic aneurysm were found to be significantly higher in the patient group with descending aneurysm. This suggests that MCV and MCHC elevations may be indicators of both mortality and clinical severity.

Lactate level is a fast and easily accessible, safe parameter that increases especially in perfusion disorders. Lactate measurements in arterial blood gas can be used as a predictor of tissue hypoxia and metabolic acidosis after aortic dissection diagnosis. Serial lactate measurements may be useful in predicting mortality [18]. Lactate formation in body metabolism results from anaerobic glycolysis by causing bleeding, inadequate ventilation, hypovolemia, hypoxemia, and end-organ hypoperfusion following a traumatic injury [19,20]. Intense adrenergic discharge increases lactate formation in patients with acute bleeding [21]. In their study, Kruse et al. [22] suggested that the results obtained from blood lactate monitoring and especially serial lactate sampling in the risk assessment of patients admitted to the hospital acutely are valuable in predicting in-hospital mortality. This study recommended that all patients with lactate greater than 2.5 mmol/L at presentation require close clinical follow-up, and that serial lactate samples should be taken in patients with lower lactate levels [22]. The acute stage of vascular damage in the aorta and the extent of damage are related to lactate levels. In previous studies, it was observed that pulse fullness, heart rate, SBP, SI,  $O_2$  saturation, and end-tidal  $CO_2$  levels, which indicate end-organ perfusion, were correlated with plasma

lactate levels [23-25]. The pain and/or stress experienced by patients with aortic dissection contribute to prehospital lactate levels. In a study conducted with 122 patients with aortic aneurysms, it was concluded that serum lactate levels were helpful in predicting mortality [26]. In another study conducted with 228 patients, it was concluded that one of the most important parameters determining the survival of patients in the perioperative period is serum lactate level [27]. In a study involving the cardiovascular system, Kawase et al. [28] investigated the effect of hospitalized blood lactate levels on early mortality in patients hospitalized with acute heart failure and found that SBP and HR were associated with early mortality. In our study, the lactate level was found to be  $1.90\pm 1.51$  mmol/L and  $3.22\pm 2.09$  mmol/L, respectively, in patients with aortic aneurysm or aortic dissection, and it was found to be useful in predicting mortality, and this elevation could also be interpreted as the effect of shock after dissection on the lactate level.

SI, which can be calculated from the patient's vital follow-up, is closely related to mortality. This index is an important parameter that can be calculated quickly and easily, without requiring additional laboratory results in patients, and helps in identifying critically ill patients in the emergency department. In the study by Hoff et al. [29], it was shown that acute aortic injury and mortality were associated with high SI. It is a critical clinical parameter that reflects changes in hemodynamics. According to literature reports, the SI is widely used in risk analysis for many diseases such as trauma, pulmonary embolism, severe pneumonia, and ectopic pregnancy [30]. In a study conducted with 313 patients with aortic aneurysms, it was shown that SI was directly related to mortality [31]. Studies have reported that an SI of 0.9 is an indicator of the risk of shock and even death for critically ill patients [32,33]. Mortality rates were found to be higher in patients with a SI of  $0.97\pm 0.54$  than those with a SI of  $0.52\pm 0.12$ . It has been suggested that considering the SI is an important clinical indicator in the follow-up of patients with aortic dissection [34]. In our study, the shock indices of patients with type 1, type 2, and type 3 dissection were found to be  $0.75\pm 0.19$ ,  $0.64\pm 0.19$ , and  $0.66\pm 0.13$ , respectively. It is thought that the SI may be a prognostic parameter in predicting mortality in patients with aortic aneurysm or dissection.

DeBakey type 1 was found most frequently in studies, followed by types 3 and type 2, respectively. In the study by Açıklan et al. [35], type 1 dissection was observed in 10 of 22 patients, whereas type 1 dissection was detected in 13 of 14 patients with AAD in the study by Buket et al. [36]. In our study, 21 (60%) of 35 dissection patients were type 1 dissection and mortality was also significantly higher. Dissection types were at similar rates with other studies, and mortality was also significantly higher in type 1. Due to the increase in mortality, we believe

that all parameters we evaluated can give an idea about the types of aortic dissection, as well as be markers for mortality.

Despite advances in invasive diagnostic methods, AAD has high mortality. Accurate and rapid diagnosis can reduce the mortality rate below 50%. Although the mortality rate of the disease is 1% per hour for the first 24 h, particularly in dissections involving the ascending aorta, this rate reaches 75% at the end of the second week. In this respect, early diagnosis in these cases is an important factor that positively affects the prognosis [4]. In the study by Yeşilaras et al. [37], 89.4% of the patients were hospitalized, 2.1% died in the emergency room, and 8.5% were referred to another health institution. In the study by Sarıtaş et al. [38] no patients were hospitalized, 91.7% were referred to another health institution, and 8.3% died in the emergency room. In our study, 16 (43.8%) patients with dissection and 6 (16.6%) patients with aneurysm resulted in mortality. We attribute the fact that we have no patients transferred to another center and that our mortality rate is below 50%, because we are a tertiary healthcare institution.

With all these data, both lactate level and SI showed significant results in the relationship between aneurysm dissection and dissection types. Lactate and SI was high in type 1 dissection, and both parameters were found to be significantly higher in the aneurysm dissection relationship. We believe that their usability for diagnosis and classification will be clarified with additional studies.

### Study Limitations

The limitations of the study include the single-center and retrospective nature of the study, difficulties in accessing the records, the lack of certainty that the registry represents all patients even though the participating researchers tried including all patients in their institutions, and the inability to fully evaluate the changes in the cause of death over time.

### Conclusion

Although aortic dissection and aneurysm are not very common diseases in emergency services, they are among the real emergencies that require rapid diagnosis and treatment in terms of mortality. Emergency physicians should be careful about these diseases, which are uncommon but have very high mortality and should be careful about medical first treatment and consultation requests. Although the mean age of patients with aortic aneurysm is higher, the mortality after dissection is much higher. We think that lactate level and SI are important parameters in predicting mortality in aortic aneurysms and dissections, and prospective and multicenter studies are needed to ensure their practical use.

### Ethics

**Ethics Committee Approval:** The study was conducted in accordance with the Helsinki Declaration of Human Research after the approval of the Health Sciences University, Kanuni Sultan Suleyman Training and Research Hospital Ethics Committee (subject number: KAEK/2022.02.41, decision no: 41, date: 23.02.2022).

**Informed Consent:** All patients were informed, their consent was obtained for inclusion in the study.

**Peer-review:** Externally peer-reviewed.

### Authorship Contributions

Surgical and Medical Practices: H.Ç., Concept: H.Ç., A.C., B.D., Design: H.Ç., A.C., B.D., Data Collection or Processing: H.Ç., B.Ç., B.A., Analysis or Interpretation: A.C., B.D., B.Ç., B.A., Literature Search: H.Ç., A.C., B.D., B.Ç., Writing: H.Ç., B.D., B.Ç., B.A.

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** The authors declared that this study received no financial support.

### References

1. Sorensen HR, Olsen H. Ruptured and dissecting aneurysms of the aorta. Incidence and prospects of surgery. *Acta Chir Scand.* 1964;128:644-50.
2. DeBakey ME, McCollum CH, Crawford ES, Morris GC Jr, Howell J, Noon GP, et al. Dissection and dissecting aneurysms of the aorta: twenty-year follow-up of five hundred twenty-seven patients treated surgically. *Surgery.* 1982;92:1118-34.
3. Roberts WC. Aortic dissection: anatomy, consequences, and causes. *Am Heart J.* 1981;101:195-214.
4. Güven FMK, Korkmaz İ, Doğan Z, Döleş KA, Eren ŞH. Acute aortic dissection: unusual presentations. *Turk J Emerg Med.* 2009;9:79-83.
5. De Bakey ME, Cooley DA, Creech O Jr. Surgical considerations of dissecting aneurysm of the aorta. *Ann Surg.* 1955;142:586-612.
6. Park SW, Hutchison S, Mehta RH, Isselbacher EM, Cooper JV, Fang J, et al. Association of painless acute aortic dissection with increased mortality. *Mayo Clin Proc.* 2004;79:1252-7.
7. DeBakey ME, Henly WS, Cooley DA, Morris GC Jr, Crawford ES, Beall AC Jr. Surgical management of dissecting aneurysms of the aorta. *J Thorac Cardiovasc Surg.* 1965;49:130-49.
8. Holmes DR, Liao S, Parks WC, Thompson RW. Medial neovascularization in abdominal aortic aneurysms: a histopathologic marker of aneurysmal degeneration with pathophysiologic implications. *J Vasc Surg.* 1995;21:761-71.
9. Tintinalli J, Stapczynski J, Ma OJ, Cline D, Cydulka R, Meckler G. *Tintinalli's emergency medicine: a comprehensive study guide, seventh edition (book and DVD):* Mcgraw-hill; 2010.
10. Vilacosta I, Aragoncillo P, Cañadas V, San Román JA, Ferreirós J, Rodríguez E. Acute aortic syndrome: a new look at an old conundrum. *Postgrad Med J.* 2010;86:52-61.

11. White A, Broder J, Mando-Vandrick J, Wendell J, Crowe J. Acute aortic emergencies--part 2: aortic dissections. *Adv Emerg Nurs J*. 2013;35:28-52.
12. Hahn AW, Jonas U, Bühler FR, Resink TJ. Activation of human peripheral monocytes by angiotensin II. *FEBS Lett*. 1994;347:178-80.
13. North BJ, Sinclair DA. The intersection between aging and cardiovascular disease. *Circ Res*. 2012;110:1097-108.
14. Nienaber CA, Clough RE, Sakalihasan N, Suzuki T, Gibbs R, Mussa F, et al. Aortic dissection. *Nat Rev Dis Primers*. 2016;2:16053.
15. Sbarouni E, Georgiadou P, Kosmas E, Analitis A, Voudris V. Platelet to lymphocyte ratio in acute aortic dissection. *J Clin Lab Anal*. 2018;32:e22447.
16. Wen D, Du X, Dong JZ, Zhou XL, Ma CS. Value of D-dimer and C reactive protein in predicting inhospital death in acute aortic dissection. *Heart*. 2013;99:1192-7.
17. Hirst AE Jr, Johns VJ Jr, Kime SW Jr. Dissecting aneurysm of the aorta: a review of 505 cases. *Medicine (Baltimore)*. 1958;37:217-79.
18. Dübendorfer C, Billeter AT, Seifert B, Keel M, Turina M. Serial lactate and admission SOFA scores in trauma: an analysis of predictive value in 724 patients with and without traumatic brain injury. *Eur J Trauma Emerg Surg*. 2013;39:25-34.
19. Kushimoto S, Akaishi S, Sato T, Nomura R, Fujita M, Kudo D, et al. Lactate, a useful marker for disease mortality and severity but an unreliable marker of tissue hypoxia/hypoperfusion in critically ill patients. *Acute Med Surg*. 2016;3:293-7.
20. Lewis CT, Naumann DN, Crombie N, Midwinter MJ. Prehospital point-of-care lactate following trauma: A systematic review. *J Trauma Acute Care Surg*. 2016;81:748-55.
21. Levy B, Desebbe O, Montemont C, Gibot S. Increased aerobic glycolysis through beta2 stimulation is a common mechanism involved in lactate formation during shock states. *Shock*. 2008;30:417-21.
22. Kruse O, Grunnet N, Barfod C. Blood lactate as a predictor for in-hospital mortality in patients admitted acutely to hospital: a systematic review. *Scand J Trauma Resusc Emerg Med*. 2011;19:74.
23. Ter Avest E, Griggs J, Wijesuriya J, Russell MQ, Lyon RM. Determinants of prehospital lactate in trauma patients: a retrospective cohort study. *BMC Emerg Med*. 2020;20:18.
24. Mullen M, Cerri G, Murray R, Talbot A, Sanseverino A, McCahill P, et al. Use of point-of-care lactate in the prehospital aeromedical environment. *Prehosp Disaster Med*. 2014;29:200-3.
25. Brown JB, Lerner EB, Sperry JL, Billiar TR, Peitzman AB, Guyette FX. Prehospital lactate improves accuracy of prehospital criteria for designating trauma activation level. *J Trauma Acute Care Surg*. 2016;81:445-52.
26. Gemelli M, Di Tommaso E, Chivasso P, Sinha S, Ahmed EM, Rajakaruna C, et al. Blood lactate predicts mortality after surgical repair of type A acute aortic dissection. *J Card Surg*. 2022;37:1206-11.
27. Lieberg J, Pruks LL, Kals M, Paapstel K, Aavik A, Kals J. Mortality after elective and ruptured abdominal aortic aneurysm surgical repair: 12-year single-center experience of estonia. *Scand J Surg*. 2018;107:152-7.
28. Kawase T, Toyofuku M, Higashihara T, Okubo Y, Takahashi L, Kagawa Y, et al. Validation of lactate level as a predictor of early mortality in acute decompensated heart failure patients who entered intensive care unit. *J Cardiol*. 2015;65:164-70.
29. Hoff E, Eagle T, Pyeritz RE, Ehrlich M, Voehringer M, Bossone E, et al. Pulse pressure and type A acute aortic dissection in-hospital outcomes (from the international registry of acute aortic dissection). *Am J Cardiol*. 2014;113:1255-9.
30. Rappaport LD, Deakyn S, Carcillo JA, McFann K, Sills MR. Age- and sex-specific normal values for shock index in National Health and Nutrition Examination Survey 1999-2008 for ages 8 years and older. *Am J Emerg Med*. 2013;31:838-42.
31. Dirks NPM, Mestrom M, van der Lugt M, van Osch F, Peters NALR, Elshof JM, et al. Utility of shock index for suspected rupture of abdominal aortic aneurysms. *Prehosp Emerg Care*. 2021;25:496-503.
32. Vandromme MJ, Griffin RL, Kerby JD, McGwin G Jr, Rue LW 3rd, Weinberg JA. Identifying risk for massive transfusion in the relatively normotensive patient: utility of the prehospital shock index. *J Trauma*. 2011;70:384-8.
33. Cannon CM, Braxton CC, Kling-Smith M, Mahnken JD, Carlton E, Moncure M. Utility of the shock index in predicting mortality in traumatically injured patients. *J Trauma*. 2009;67:1426-30.
34. Guo ZJ, Lin Q, Zi XR, Xu Q, Liu HT, Lu JY, et al. Correlation of computed tomography angiography parameters and shock index to assess the transportation risk in aortic dissection patients. *Radiol Med*. 2015;120:386-92.
35. Açıklın A, Satar S, Akpınar O, Kuvandık G, Sarı A, Kanadaşı M, et al. Aortic dissection: two years of clinical experience in patients presenting to the emergency department of a university hospital. *Turk J Emerg Med*. 2005;5:32-5.
36. Buket S, Apaydın A, Hamulu A, Özbaran M, Alayunt A, Yüksel M, et al. Surgical treatment in acute aortic dissections. *GKD Cer Derg*. 1995;3:147-52.
37. Yeşilaras M, Sönmez N, Karcıoğlu Ö, Topaçoğlu H, Aksakallı S, Bayram B. Clinical characteristics of patients diagnosed with aortic dissection in the emergency department: a case series. *Turk J Emerg Med*. 2006;6:1-6.
38. Sarıtaş A, Güneş H, Kandış H, Çıkman M, Çandar M, Korkut S, et al. A retrospective analysis of patients admitted to our clinic with aortic dissection. *JAEM* 2011;10:152-5.