Van Med J 31 (3): 232-238, 2024 DOI: <u>10.5505/vmj.2024. 92486</u>

# **Electrocardiographic Findings in Sepsis**

Hatice Aslan Sirakaya<sup>1</sup>, Kaniye Aydin<sup>2</sup>, Ali Cetinkaya<sup>1</sup>

<sup>1</sup>Health Science University, Kayseri City Hospital, Department of Internal Medicine, Kayseri, Türkiye <sup>2</sup>Cukurova University, Faculty of Medicine, Department of Internal Medicine, Division of Medical Intensive Care Unit, Adana, Türkiye

#### Abstract

Introduction: Sepsis continues to be among the important causes of death in the world. This study aimed to determine the electrocardiography (ECG) findings observed in patients with sepsis and to reveal their relationship with survival.

**Materials and Methods:** This single-center, observational and descriptive study included 45 patients aged 18 and over who were followed up in the Intensive Care Unit (ICU) of Kayseri City Hospital in the last year and were diagnosed with sepsis. Demographic characteristics, laboratory results, clinical course and ECG findings of the patients were documented. Mortality status of the patients was recorded. Patients were divided into two groups (survivors and non-survivors) according to 28-day mortality results. Demographic characteristics, laboratory data and ECG findings were compared between these groups.

**Results:** Among the patients included in the study, 60% were female and 40% were male. Comparing patients in terms of survival, the median APACHE II score, median SOFA score, and the presence of atrial fibrillation (AF) were significantly higher in the non-surviving group (p=0.001; p=0.034; p=0.034; p=0.047). Multivariate analysis revealed that factors influencing survival were the APACHE II score and the presence of AF (p=0.001 and p=0.048).

Conclusion: Cardiac effects can be seen in patients with sepsis. AF may be a sign indicating mortality in patients with sepsis.

Key words: Atrial fibrillation; ECG; QT prolonged; sepsis.

## Intoduction

Sepsis is a highly fatal condition that can lead to organ dysfunction resulting from an abnormal immune response to inflammation and infection (1). In sepsis, organ dysfunctions are evaluated using the Sequential Organ Failure Assessment (SOFA) scoring system, with organ dysfunction defined as a two or more-point increase in the SOFA score. Therefore, sepsis should be considered in patients presenting with novel and unexplained organ dysfunctions. Septic shock is a subgroup of sepsis that has increased mortality rates due to various mechanisms and is seen as the progression of sepsis. The APACHE scoring system is a logistic regression equation generated by a computer, using a large number of clinical variables, to predict hospital mortality and, in some cases, the length of hospital stay. The necessary variables include factors such as age, diagnosis, prior treatment location, and numerous acute physiological and chronic health variables.

The pathophysiology of sepsis is a highly complex process that involves nearly all organs and systems. Vasodilation and increased capillary permeability are common outcomes of inflammatory processes in sepsis. Physiological changes such as reduced systemic vascular resistance, diminished cardiac output, myocardial dysfunction, and decreased cardiac contractility are particularly observed in septic shock. The main reason for the development of myocardial dysfunction in sepsis is the release of inflammatory cytokines induced by endotoxins of pathogenic microorganisms and causing endothelial damage. Myocardial depression, vasodilatation, increased capillary permeability and oxide release following dysregulated nitric inflammatory cytokine release result in myocardial ischemia. In addition, myocardial dysfunction occurs following deprivation of supportive mechanisms in autonomic nervous system (2). Atrial fibrillation is the most common arrhythmia encountered, affecting more than 30 million people worldwide (3). Atrial fibrillation leads to an increase in cerebrovascular events, heart failure, dementia, and mortality (4). In intensive care patients, AF can lead to a ventricular response and hemodynamic instability, which may increase mortality and morbidity. A meta-analysis showed that new-onset AF leads to a 1.69-fold increase in mortality (5). The literature contains numerous studies investigating the impact of cardiac failure, cardiac arrhythmias, and cardiac abnormalities on mortality in patients with sepsis (6-8). However, which comprehensive study in the no



<sup>\*</sup>Corresponding Author: Hatice Aslan Sirakaya, Health Science University, Kayseri City Hospital, Department of Internal Medicine, Kayseri, Turkey, E-mail:hasirakaya@gmail.com **Orcid:** Hatice Aslan Sirakaya <u>0000-0001-6933-6459</u>, Kaniye Aydin <u>0000-0001-5538-3692</u>, Ali Cetinkaya 0000-0001-8485-0982

electrocardiographic findings of sepsis patients are evaluated and interpreted in detail has been found in the literature. This study aimed to determine the ECG findings observed in sepsis patients and to reveal their relationship with survival.

## Materials and Methods

**Study Design and Setting:** Forty-five patients over the age of 18 who were followed up with a diagnosis of sepsis in the intensive care unit (ICU) of Kayseri City Hospital in the last year were included in this retrospective, single-center, observational and descriptive study. The study adhered to the principles of the Declaration of Helsinki and the Patients' Bill of Rights.

Study Population and Data Collection: The sample included patients who had been diagnosed with sepsis in the medical ICU between August 1, 2019, and December 31, 2019. The patients were diagnosed with sepsis or septic shock according to the definition of Sepsis (1). All patients were treated according to the Surviving Sepsis Campaign (SSC) guidelines. Patients over 18 years of age, diagnosed with sepsis, without malignancy, and treated by admission to the intensive care clinic were included in the study. Exclusion criteria include being under 18 years of age, not having an ECG at the time of hospitalization, not being able to access clinical data, exitus within the first 24 hours of hospitalization, having a malignancy, and being followed up with non-sepsy diagnoses. The patients were divided into two groups: surviving (Group 1) and non-surviving (Group 2). There were 22 patients in Group 1 and 23 patients in Group 2. We prepared a form to record the information of the patients included in the study. Patient data from patient files, clinical information forms and hospital information management system were retrospectively scanned and recorded in the prepared form. The patients' comorbidities, demographic characteristics, Glasgow coma scale (GCS), body mass index, Acute Physiology and Chronic Health Evaluation II (APACHE-II) scores, SOFA scores, laboratory values, treatments, intensive care unit stay times and 28-day mortality rate were analyzed. The 12lead ECGs routinely taken for each patient in our intensive care unit were also examined and pathological findings were recorded.

**Ethical approval:** The study was approved by the Kayseri City Hospital Ethics Committee (approval #2020/209).

Statistical analysis: Descriptive statistics were presented as frequencies and percentages for categorical variables. The Kolmogorov-Smirnov test was used for normality assumption for continuous variables. Descriptive statistics for continuous variables with normal distribution were reported as mean  $\pm$  SD, and variables with skewed distribution were presented as median (25<sup>th</sup>-75<sup>th</sup> percentile). According to the 28-day mortality, we divided patients into two groups (survivors and non-survivors) and compared the demographic, clinical characteristics and ECG findings. Chi-square test or the Fisher's exact test was performed to determine the relationship between categorical variables. Continuous variables were compared by t test (normally distributed variable) or MannWhitney U test (not normally distributed variable).

**Table 1:** Demographic and clinical characteristics

 of the patients

| of the patients       |                  |
|-----------------------|------------------|
| Sex (male/female)     | 18/27(40/60)     |
| Age (years)           | 75.5(26-96)      |
| BMI                   | $24.04 \pm 3.37$ |
| GCS                   | 12.5 (3-15)      |
| APACHE II score       | 34(27-40)        |
| SOFA score            | 8(7-11)          |
| Site of infection     |                  |
| Lung                  | 19(42.2)         |
| Abdomen               | 10(22.2)         |
| Urogenital            | 8(17.8)          |
| Bloodstream           | 7(15.6)          |
| Soft tissue           | 1(2.2)           |
| Comorbidities         |                  |
| Diabetes mellitus     | 18(19.4)         |
| Hypertension          | 18(19.4)         |
| Coronary artery       | 4(4.3)           |
| disease               |                  |
| Heart failure         | 4(4.3)           |
| Chronic kidney        | 30(32.3)         |
| damage                |                  |
| Chronic liver disease | 1(1.1)           |
| Chronic obstructive   | 4(4.3)           |
| pulmonary disease     |                  |
| Neurological disease  |                  |
| Rheumatological       | 13(14)           |
| disease               |                  |
| Septic shock          | 1(1.1)           |
| Renal replacement     | 28(62.2)         |
| therapy               |                  |
| Acute renal failure   | 29(64.4)         |
| Insulin use           | 39(86.7)         |
| Sex (male/female)     | 15(33.3          |

Values are presented as number (%) of patients. **BMI:** Body mass index, **GCS:** Glasgow coma scale APACHE II score: Acute Physiology and Chronic Health Evaluation II Score, SOFA score: sequential organ failure assessment score.,

In addition, Logistic regression analysis was performed to determine the relationships between exitus rate and the explanatory variables. Statistical significance level was considered as 5%. All calculations were performed in the Statistical Package for the Social Sciences version 20.0 (IBM Corp., Armonk, NY, USA).

### Results

In this study, which included 45 patients, 60% of the patients were female and 40% were male. Table 1 includes the demographic and clinical characteristics of the patients.

Table 2: Laboratory findings and Clinical course

| Variable                               | Values                    |
|--|---------------------------|
| White blood cell (10 <sup>3</sup> /µL) | 12.3(2.1-33) <sup>a</sup> |
| Hematocrit (%)                         | $35.5\pm8.4^{a}$          |
| Platelet (10 <sup>3</sup> /µĹ)         | 185.13±95.59 <sup>b</sup> |
| Glucose (mg/dL)                        | $150(60-451)^{a}$         |
| Blood Urea Nitrogen                    | 55.8±25.99 <sup>b</sup>   |
| (mg/dL)                                |                           |
| Serum creatinine (mg/dL)               | $3.17(0.75-9.7)^{a}$      |
| Serum sodium (mmol/L)                  | 134.5(125-                |
|  | 158)ª                     |
| Serum potassium                        | 4.54±1.38 <sup>b</sup>    |
| (mmol/L)                               |                           |
| Serum calcium (mg/dL)                  | $8.16 \pm 0.92^{b}$       |
| Serum phosphorus                       | $4.45(2.19-8.5)^{a}$      |
| (mg/dL)                                |                           |
| Serum magnesium                        | $1.9(1.24-3.6)^{a}$       |
| (mg/dL)                                |                           |
| Albumin (g/L)                          | 28.99±0.66 <sup>b</sup>   |
| Uric acid (mg/dL)                      | 6.24±2.92ь                |
| Lactate dehydrogenase                  | 323.5(126-                |
| (U/L)                                  | 1037)ª                    |
| C-reactive protein,                    | 82.8(3.8-377)ª            |
| (mg/dL)                                |                           |
| Procalcitonin, (ng/mL                  | 17.99(0.17-               |
|  | 100)                      |
| NT-proBNP, (pg/mL)                     | 17375.5(35-               |
|  | 35000)ª                   |
| pH                                     | 7.29±0.13 <sup>b</sup>    |
| HCO <sup>3</sup> , (mmol/L)            | 19.36±5.12 <sup>b</sup>   |
| Lactate (mmol/L)                       | 2.6(0.7-14)               |
| Length of ICU stay (days)              | 8(1-45)°                  |
| Mode of discharge from                 |                           |
| ICU                                    |                           |
| Transfer to internal                   | 19(42.2) <sup>c</sup>     |
| medicine clinic                        |                           |
| Discharge from hospital                | $3(6.7)^{c}$              |
| Exitus                                 | 23(51.1)°                 |
| 28 day mortality                       | 23(51.1) <sup>c</sup>     |

a: Median (25<sup>th</sup>-75<sup>th</sup> percentile) <sup>b</sup>: mean ±standard deviation c: number (%) of patients **NT-proBNP**: N terminal probrain natriuretic peptide, **HCO**<sup>3</sup>: Bicarbonate.

Table 2 summarizes the laboratory findings and clinical course of the patients. Some of the findings in the ECG of the patients are as follows; ST segment elevation in 13.3%, tachycardia in 55.6%, atrial fibrillation in 17.8%, pathological Q wave in 26.7%, left bundle branch block in 4.4%. (Table 3) The 28-day mortality of the patients was

found to be 51.1%. The patients were divided into groups: survivors and non-survivors, two according to their 28-day mortality. Demographic characteristics, laboratory findings and ECG findings were compared between the groups. (Table 4). Among non-survivors, median APACHE II score and SOFA score were statistically significant higher than among survivors (p=0.001 and p=0.034). The presence of atrial fibrillation was more among non-survivors than among survivors (p=0.047). In univariate analysis, parameters affecting the 28-days of mortality (APACHE II score, SOFA score, and presence of atrial fibrillation) were evaluated by binary logistic regression analysis. According to the multivariate analysis, the factors affecting mortality were the APACHE II score and presence of atrial fibrillation (Table 5).

## Discussion

In this study, when patients were compared regarding survival, it was found that APACHE II score, SOFA score and presence of AF were significantly higher in non-survivor group. Based on multivariate analysis, APACHE II score and AF were identified as factors affection on mortality. Many mechanisms explaining the cardiac involvement that develops in sepsis have been reported in the literature (2),(8). However, there is a scarcity in studies on ECG findings in patients with sepsis, most of which are not up-todate.In a study by Mangano et al., it was shown that tissue perfusion was dependent to vascular resistance rather than cardiac output in patients with sepsis. Thus, it was also shown that the reduction in heart rate by esmolol, a cardioselective beta-blocker, decreased myocardial performance and risk for myocardial ischemia without impairment in organ perfusion (9). According to research, it has been reported that left ventricular dysfunction due to sepsis may occur as Takotsubo Syndrome pattern (10),(11). Takotsubo Syndrome, also known as broken heart syndrome, causes systolic dysfunction. Clinical, laboratory and ECG findings are similar to those in acute coronary syndrome, all which are irreversible. In our patient group, there left branch bundle block in 2 patients (4.4%), ST segment elevation in 6 patients (13.3%) and pathological Q wave in 12 patients (26.7%). However, acute coronary syndrome was not detected in any patients by clinical and laboratory findings. When compared regarding survival, there was no significant difference regarding pathological Q wave, ST elevation and left branch bundle block.

## Table 3: ECG findings

| Tachycardia                         |                |
|-------------------------------------|----------------|
| Presence                            | 25(55.6)       |
| Absence                             | 20(44.4)       |
| Bradycardia                         |                |
| Presence                            | 2(4.4)         |
| Absence                             | 43(95.6)       |
| Normal axis                         |                |
|                                     | 28(62.2)       |
| Left axis deviation                 | 14(31.1)       |
| Right axis deviation                | 3(6.7)         |
| Left atrial abnormality             |                |
| Presence                            | 9(20)          |
| Absence                             | 36(80)         |
| Right atrial abnormality            |                |
| Presence                            | 2(4.4)         |
| Absence                             | 43(95.6)       |
| PR segment deviation                | 15(55.0)       |
| -                                   | 2(1, 1)        |
| Presence                            | 2(4.4)         |
| Absence                             | 43(95.6)       |
| First-degree atrioventricular block |                |
| Presence                            | 9(20)          |
| Absence                             | 36(80)         |
| PR interval, (ms)                   | 154.28±38.35   |
| QRS duration                        | 86.33±20.04    |
| Atrial fibrillation                 | 00000000       |
| Presence                            | 8(17.8)        |
| Absence                             |                |
|                                     | 37(82.2)       |
| Left bundle branch block            | - / /          |
| Presence                            | 2(4.4)         |
| Absence                             | 43(95.6)       |
| Right bundle branch block           |                |
| Presence                            | 2(4.4)         |
| Absence                             | 43(95.6)       |
| Pathologic Q wave                   |                |
| Presence                            | 12(26.7)       |
| Absence                             | 33(73.3)       |
|                                     | 35(75.5)       |
| Left ventricular hypertrophy        |                |
| Presence                            | 11(24.4)       |
| Absence                             | 34(75.6)       |
| Right ventricular hypertrophy       |                |
| Presence                            | 1(2.2)         |
| Absence                             | 44(97.8)       |
| ST segment elevation                |                |
| Presence                            | 6(13.3)        |
| Absence                             | 39(86.7)       |
| Early repolarization                | 35(00.7)       |
| • •                                 | 45(100)        |
| Absence                             | 45(100)        |
| Primary T wave inversion            |                |
| Presence                            | 21(46.7)       |
| Absence                             | 24(53.3)       |
| Pathological U wave                 |                |
| Presence                            | 1(2.2)         |
| Absence                             | 44(97.8)       |
| Corrected QT interval, (ms)         | 459.56±44.15   |
| Corrected QT interval, (ins)        | 137.30 - 77.13 |
|                                     | 2(/57 0)       |
| Presence                            | 26(57.8)       |
| Absence                             | 19(42.2)       |
| Corrected QT interval shortening    |                |
| Presence                            | 1(2.2)         |
| Absence                             | 44(97.8)       |
|                                     |                |

Values are presented as number (%) of patients or mean  $\pm$ standard deviation.

Van Med J Volume:31, Issue:3, July/2024

|  | Survivor (n=22)     | Nonsurvivor (n=23)  | p-value |  |
|--|---------------------|---------------------|---------|--|
| Age (years)                              | 72.5(63.75-82,5)    | 68(57-84)           | 0.585   |  |
| APACHE II score                          | 29.5(21.75-33.25)   | 38(35-45)           | 0.001   |  |
| SOFA score                               | 7.5(6-10)           | 10(7-12)            | 0.034   |  |
| C-reactive protein (mg/dL)               | 63.55(2.8-330)      | 86.1(3.8-377)       | 0.411   |  |
| Procalcitonin (ng/mL)                    | 11.95(0.14-100)     | 9.15(0.24-100)      | 0.624   |  |
| NT-proBNP (pg/mL)                        | 15763(165-35000)    | 18466(35-35000)     | 0.531   |  |
| Lactate (mmol/L)                         | 2.05(0.6-8.9)       | 3.1(0.9-18)         | 0.104   |  |
| Tachycardia                              |                     |                     |         |  |
| Presence                                 | 9(40.9)             | 16(69.6)            |         |  |
| Absence                                  | 13(59.1)            | 7 (30.4)            | 0.750   |  |
| Left axis deviation                      |                     |                     |         |  |
| Presence                                 | 6(27.3)             | 8(34.8)             |         |  |
| Absence                                  | 16(72.7)            | 15(65.2)            | 0.749   |  |
| Left atrial abnormality                  |                     |                     |         |  |
| Presence                                 | 5(22.7)             | 4(17.4)             |         |  |
| Absence                                  | 17(77.3)            | 19(82.6)            | 0.722   |  |
| First-degree atrioventricular            | 17(11.3)            | 19(02.0)            | 0.122   |  |
| block                                    |                     |                     |         |  |
| Presence                                 | 5(22.7)             | 4(17.4)             |         |  |
| Absence                                  | 17(77.3)            | 19(82.6)            | 0.722   |  |
| Atrial fibrillation                      | 17(77.3)            | 17(02.0)            | 0.722   |  |
| Presence                                 | 1(4.5)              | 7(30.4)             |         |  |
| Absence                                  | 21(95.5)            | 16(69.6)            | 0.047   |  |
| QRS prolongation                         | 21(75.5)            | 10(09:0)            | 0.047   |  |
| Presence                                 | 7(31.8)             | 5(21.7)             |         |  |
| Absence                                  | 15(68.2)            | 18(78.3)            | 0.445   |  |
| Low voltage                              | 15(00.2)            | 10(70.5)            | 0.445   |  |
| Presence                                 | 1(4.5)              | 4(17.4)             |         |  |
| Absence                                  | 21(95.5)            | 19(82.6)            | 0.346   |  |
| Pathologic Q wave                        | 21(95.5)            | 17(02.0)            | 0.540   |  |
| Presence                                 | 5(22.7)             | 7(30.4)             |         |  |
| Absence                                  | 17(77.3)            | 16(69.6)            | 0.738   |  |
|  | 17(77.3)            | 10(09.0)            | 0.738   |  |
| ST segment elevation<br>Presence         | 2(0, 1)             | 4(17.4)             |         |  |
|  | 2(9.1)              |                     | 0.665   |  |
| Absence                                  | 20(90.9)            | 19(82.6)            | 0.005   |  |
| Left ventricular hypertrophy<br>Presence | 6(27.2)             | 5(21 7)             |         |  |
|  | 6(27.3)<br>16(72.7) | 5(21.7)<br>18(78.3) | 0.738   |  |
| Absence                                  | 16(72.7)            | 18(78.3)            | 0.738   |  |
| Poor R progression                       |                     |                     |         |  |
| Presence                                 | 10(45.4)            | 16(69.6)            | 0.401   |  |
| Absence                                  | 12(54.5)            | 7(30.4)             | 0.136   |  |
| Primary T wave inversion                 |                     |                     |         |  |
| Presence                                 | 11(50)              | 10(43.5)            | ~       |  |
| Absence                                  | 11(50)              | 13(56.5)            | 0.768   |  |
| Corrected QT prolongation                |                     |                     |         |  |
| Presence                                 | 16(77.3)            | 10(43.5)            |         |  |
| Absence                                  | 6(22.7)             | 13(56.5)            | 0.071   |  |

Table 4: Demographic characteristics, laboratory and ECG findings according to 28 day mortality

Values are presented as number (%) of patients or median (25<sup>th</sup>-75<sup>th</sup> percentile).**APACHE II score:** Acute Physiology and Chronic Health Evaluation II Score, **SOFA score:** sequential organ failure assessment score, **NT-proBNP:** N terminal probrain natriuretic peptide.

| Table 5: Summa | rized result | 's of Logistic | regression | analysis |
|----------------|--------------|----------------|------------|----------|
|                |              |                |            |          |

| Findings Beta       | Beta p-value | Odds ratio | 95% CI |       |        |
|---------------------|--------------|------------|--------|-------|--------|
|                     | p value      |            | Lower  | Upper |        |
| Atrial fibrillation | 2.218        | 0.048      | 9.187  | 1.024 | 82.414 |
| APACHE II score     | -0.160       | 0.001      | 0.852  | 0.772 | 0.940  |

APACHE II score: Acute Physiology and Chronic Health Evaluation II Score, CI: Confidence interval.

Van Med J Volume:31, Issue:3, July/2024

In a study of patients with septic shock by Parker et al., 50% of patients had mild to severe reduction in ejection fraction ( $\leq 40\%$ ; as graded by radionuclide cineangiography and hemodynamic measurements). It was observed that ventricular functions returned to normal in these patients 10 days after the initial period of septic shock (12). In a study involving 35 culture-positive patients, Ellrodt et al. illustrated segmental ventricular wall movement abnormalities in 22 individuals. It was reported that these pathological findings resolved upon recovery from sepsis (13). In our study, no comparison was made before and after sepsis. In a case report by Varriale et al., it was observed that QTc time was prolonged up to 600 msec in a patient with sepsis and decreased to 400 msec after recovery of sepsis. Torsades de Pointes was developed in the patient and a temporary pacemaker was implanted (14). In another study, QT prolongation was found as an independent risk factor for sepsis in hospitalized patients (15). In a study on 17 patients with septic shock, Rich et al. observed that there was decrease in QRS amplitude in 14 patients and prolonged QRS in eight patients on ECGs obtained during and after septic shock (7). In our study, no significant difference was observed in terms of QTc duration. However, a significant decrease in QRS amplitude was noted among the patients. Additionally, while QTc prolongation was observed in some patients, no significant difference was detected between the groups. Although a decrease in QRS width was observed, it did not significantly correlate with survival outcomes. Despite these cardiac effects not influencing mortality, pathological changes were evident in the ECG, consistent with findings from previous studies. In the study by Rich et al., vasopressor agents were required in all patients with septic shock admitted to intensive care unit and 50% of patients died (7). In our study, vasopressor agents were required in 62.2% of patients. It was seen that almost half of our patients died. The APACHE II score is used in many intensive care units to determine the severity of illness and predict mortality. In our study, it also played an effective role in predicting mortality in patients with sepsis (16),(17). A study reported that AF was observed in 25% of patients hospitalized due to sepsis (18). In another cohort study, the effects of acute AF development in patients with sepsis were examined, and AF was detected in 23%, and it was observed that patients with AF were older, had more comorbid diseases, and had a higher APACHE IV score. Again, in this study, new-onset AF was found to be associated with increased mortality, prolonged

hospital stay, and higher mortality risk (19). Similarly, in our study, patients with AF were seen to have a significant impact on mortality. In a meta-analysis conducted by Corica and colleagues, it was also found that newly developed atrial fibrillation (AF) leads to an increase in mortality, which is consistent with our study (5).

**Study limitations:** The limitations of our study include its single-center retrospective design and relatively small sample size. Additionally, the lack of comparison of ECG findings after Sepsis treatment is also among the limitations. The study is not supported by transthoracic and other cardiac imaging methods, and the effects of the antibiotics and drugs used were not evaluated, which are other limitations of the study.

## Conclusion

As a result, cardiac involvement can be observed frequently in patients with sepsis. The presence of AF may be a finding that predicts mortality in sepsis patients.

**Ethics committee approval:** Kayseri City Hospital Education and Research Hospital Ethics Committee, date: 15.10.2020, number: 209).

**Conflict of interest:** No potential conflict of interest relevant to this article was reported.

**Financial support:** This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution: Concept-Design: HAS, KA, AC; Literature review: HAS, KA; Data collection: HAS, KA, AC; Writing-review-revision: HAS, KA.

## References

- Evans L, Rhodes A, Alhazzani W, Antonelli M, Coopersmith CM, French C, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. Intensive Care Med 2021;47(11):1181–1247.
- 2. Tartavoulle T, Fowler L. Cardiogenic Shock in the Septic Patient: Early Identification and Evidence-Based Management. Crit Care Nurs Clin North Am. 2018;30(3):379– 387.
- Chugh SS, Havmoeller R, Narayanan K, Singh D, Rienstra M, Benjamin EJ, et al. Worldwide epidemiology of atrial fibrillation: a Global Burden of Disease 2010 Study. Circulation. 2014;129(8):837– 847.
- 4. January CT, Wann LS, Calkins H, Chen LY, Cigarroa JE, Cleveland JCJ, et al. 2019

AHA/ACC/HRS Focused Update of the 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart J Am Coll Cardiol 2019;74(1):104–132.

- Corica B, Romiti GF, Basili S, Proietti M. Prevalence of New-Onset Atrial Fibrillation and Associated Outcomes in Patients with Sepsis: A Systematic Review and Meta-Analysis. J Pers Med 2022;12(4):547-559.
- Mehta S, Granton J, Lapinsky S, Newton G, Bandayrel K, Little A, et al. Agreement in ECG interpretation in patients with septic shock. Crit Care Med. 2011;39:2080–2086.
- Rich MM, McGarvey ML, Teener JW, Frame LH. ECG changes during septic shock. Cardiology. 2002;97(4):187–196.
- Vallabhajosyula S, Jentzer JC, Geske JB, Kumar M, Sakhuja A, Singhal A, et al. New-Onset Heart Failure and Mortality in Hospital Survivors of Sepsis-Related Left Ventricular Dysfunction. Shock. 2018;49(2):144–149.
- Mangano DT, Layug EL, Wallace A, Tateo I. Effect of atenolol on mortality and cardiovascular morbidity after noncardiac surgery. Multicenter Study of Perioperative Ischemia Research Group. N Engl J Med 1996;335(23):1713–1720.
- 10. Y-Hassan S, Settergren M, Henareh L. Sepsis-induced myocardial depression and takotsubo syndrome. Acute Card Care 2014;16(3):102–109.
- 11. Santoro F, Di Biase M, Brunetti ND. Urinary sepsis associated with Takotsubo cardiomyopathy. Int J Urol. 2014 ;21(4):432-433.
- 12. Parker MM, McCarthy KE, Ognibene FP, Parrillo JE. Right ventricular dysfunction and dilatation, similar to left ventricular changes, characterize the cardiac depression of septic shock in humans. Chest 1990;97(1):126–131.

- 13. Ellrodt AG, Riedinger MS, Kimchi A, Berman DS, Maddahi J, Swan HJ, et al. Left ventricular performance in septic shock: reversible segmental and global abnormalities. Am Heart J 1985;110(2):402– 409.
- 14. Varriale P, Ramaprasad S. Septic cardiomyopathy as a cause of long QT syndrome. J Electrocardiol 1995 ;28(4):327– 329.
- 15. Tisdale JE, Jaynes HA, Kingery JR, Mourad NA, Trujillo TN, Overholser BR, et al. Development and validation of a risk score to predict QT interval prolongation in hospitalized patients. Circ Cardiovasc Qual Outcomes. 2013 ;6(4):479–487.
- 16. Rowan KM, Kerr JH, Major E, McPherson K, Short A, Vessey MP. Intensive Care Society's Acute Physiology and Chronic Health Evaluation (APACHE II) study in Britain and Ireland: a prospective, multicenter, cohort study comparing two methods for predicting outcome for adult intensive care patients. Crit Care Med 1994;22(9):1392–1401.
- 17. Ho KM, Dobb GJ, Knuiman M, Finn J, Lee KY, Webb SAR. A comparison of admission and worst 24-hour Acute Physiology and Chronic Health Evaluation II scores in predicting hospital mortality: a retrospective cohort study. Crit Care 2006;10(1):466-473.
- Walkey AJ, Greiner MA, Heckbert SR, Jensen PN, Piccini JP, Sinner MF, et al. Atrial fibrillation among Medicare beneficiaries hospitalized with sepsis: incidence and risk factors. Am Heart J 2013;165(6):949–955.
- 19. Klein Klouwenberg PMC, Frencken JF, Kuipers S, Ong DSY, Peelen LM, van Vught LA, et al. Incidence, Predictors, and Outcomes of New-Onset Atrial Fibrillation in Critically Ill Patients with Sepsis. A Cohort Study. Am J Respir Crit Care Med 2017;195(2):205–211.