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Cardiac Dysfunction as an Early Outcome Predictor in Severe Sepsis

Asmaa Elsaeed Ebrahim*[1], Mohamed El-Said Ahmed[2], Samir Mohammed Attia[3], Afaf Abdel-Hafez Abdel-Mageed[4]

Departments of [1]Emergency Medicine and [2]Membership of Critical Care Medicine, Emergency Hospital, Mansoura University, Egypt

Departments of [3]Vascular Surgery and [4]Internal Medicine and Critical Care Medicine, Faculty of Medicine, Mansoura University, Egypt

***Corresponding author:** Asmaa Elsaeed Ebrahim, **Mobile:** (+20) 01033969279, **E-mail:** asmaaelsaeed510@gmail.com

ABSTRACT

Background: Sepsis has been considered as a major cause of morbimortality worldwide. The mortality rate of severe sepsis is 25%, representing about 40% of hospital mortality. Sepsis-induced cardiac dysfunction (SICD) is encountered in the intensive care unit (ICU), and its prevalence in septic cases ranges from 10 to 70%. Such discrepancies among researches are mainly owing to the absence of well-established diagnostic criteria.

Objective: To investigate if cardiac dysfunction as evidenced by positive troponin (cTnI) and left ventricular systolic and diastolic functions can predict bad outcome in non-cardiac patients with severe sepsis and/or septic shock.

Patients and Methods: This was a prospective observational cohort study carried out in Emergency Department (ED), Mansoura University on 100 patients, adults, non-cardiac, non-surgical patients, who were presented with severe sepsis over a period of 1 year from July 2022 to July 2023.

Results: 55% of the included patients were positive troponin. Troponin was a statistically significant independent predictor of mortality, bad outcome and need for vasopressor among studied cases. Possible predictors of mortality showed that lower albumin, lower ejection fraction and positive troponin were statistically significant predictors of mortality among studied cases.

Conclusion: High troponin levels may be detected in non-cardiac patients with sepsis and this group according to new guidelines are considered stage-B pre heart failure patients. Troponin is a statistically significant independent predictor of mortality, bad outcome and need for vasopressor among studied cases.

Keywords: Positive troponin, Sepsis-induced cardiac dysfunction.

INTRODUCTION

Sepsis has been considered as a primary cause of morbimortality all over the world. The mortality rate of severe sepsis is 25%, representing about 40% of hospital mortality. SICD is a gradually identified type of transient cardiac dysfunction in the septic cases. In spite of implications for patient outcomes, the critical healthcare communities haven't given considerable focus on such disease [(1)].

In addition, SICD is encountered in the ICU, and its incidence among septic cases ranges from 10 to 70% [(2)]. These discrepancies among studies are mostly owing to the absence of well-established diagnostic criteria. In addition, the epidemiological alteration highlights the complicated factors in sepsis: source and degree, onset of resuscitation, and therapeutic modalities. Low systolic, diastolic blood pressure and lower ejection fraction were predictors of SICD. Additionally, it was demonstrated that; SICD was accompanied by a higher possibility of hospital mortality. Hypoalbuminemia, higher creatinine levels and positive bacterial culture could be considered as independent predictors of SICD [(3)].

Of note, SICD often develops in critically ill cases; on the other hand, the clinical manifestations and prognostic impact of SICD on sepsis outcomes are still a matter of debate [(4)]. So, our study aims to investigate if cardiac dysfunction, as evidenced by positive troponin (cTnI) and left ventricular systolic (LVSF) and diastolic (LVDF) functions, can predict bad outcome in non-cardiac cases with severe sepsis and/or septic shock.

SUBJECTS AND METHODS

Subjects

Study design:

This was a prospective observational clinical study (cohort) carried out in ED on 100 patients, who were presented with severe sepsis to Mansoura University Emergency Hospital (MUEH) and admitted at Mansoura University Hospital (MUH). This study was conducted over the period of 1 year (from July 2022 to July 2023).

Study population:

Inclusion criteria included: Patients with age more than 18 years old; non-cardiac patients with severe sepsis, which is defined as "life-threatening organ dysfunction due to a dysregulated host response to infection".

Exclusion criteria included:

Age <18 years old; patients who didn't match the sepsis criteria; surgical patients; patients with pulmonary embolism; patients with history of cardiac diseases or previous cardiac surgeries such as myocardial infarction, angina pectoris, coronary angioplasty, coronary artery bypass graft (CABG), positive stress test for ischemia; admission ECG of ACS or typical changes of IHD; echo Doppler revealing cardiac disease as cardiomyopathies, EF < 0.50, significant valvular regurgitant or stenotic lesions or pericardial disease; and clinical evidence of congestive heart Failure.

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Methods

Study procedures: All participants underwent the following procedures:

History taking: All patients had a thorough history taking including: personal history, the "chief complaint" with special focusing on symptoms suggestive of infection, history of the present illness: details about the complaints including onset, course and duration and medications used to relieve this complaint before attending to ER, and past medical history.

Examinations:

[1] General examination: (pulse, blood pressure, temperature, respiratory rates). According to SOFA score and qSOFA score, the general examination of the patient was assessed when: Pulse (tachycardia >90 beats/minute), blood pressure (systolic blood pressure (SBP) ≤100 mmHg and mean arterial blood pressure (MAP) <60 mmHg), MAP was estimated according to the equation of: MAP = DBP+1/3 (SBP-DBP) or MAP = DBP + 1/3(PP), temperature (fever >38C or hypothermia <36C), RR ≥22 breaths/min [(5)].

[2] Abdominal examination.

[3] Chest examination.

[4] Cardiac examination.

[5] Neurological examination.

Laboratory assessment: [1] Measurement of serum troponin cTnI level: Blood samples were collected within 1 hour of hospital arrival. Serum cTnI levels were measured using the electrochemiluminescence immunoassay "ECLIA", which is intended for invitro use on Elecsys and Cobas e 411 immunoassay analyzers. Measuring range values: 0.1 -25 µg/L. Values below the limit of blank were reported as < 0.1 µg/L or negative. Values beyond the measuring range were recorded as > 25 µg/L should be for 10-fold diluted samples), negative value < 0.1 µg/L, positive value > 0.1 µg/L [(6)]. [2] CBC [3] Liver function tests; [4] Kidney function tests; [5] INR.

Radiological assessment:

Chest X-ray and NCCT chest for evidence of respiratory tract infection or congestion. ECG. Echo Doppler (systolic and diastolic functions): By 2D echo (LOGIC P6 PRO). Systolic functions, assessed by calculation of ejection fraction (EF) and fraction shortening (FS), were assessed by longitudinal parasternal four chamber view (by M mode) and apical view in echo. Normal ejection fraction (≥0.55 for men and 0.60 for women)**.** Ejection fraction was calculated from estimation of LVEDV (left ventricular end diastolic volume) and LVESV (left ventricular end systolic volume) from the formula: EF = (LVED volume – LV end-systolic volume)/LVED volume**.** Fraction shortening was calculated from estimation from LVEDD (left ventricular end diastolic dimension) and LVESD (left ventricular end systolic dimension) from the formula: FS= (LVEDD - LVESD / LVEDD) x 100**.** Diastolic function was assessed by bimensional (BD) mode together with pulsed wave Doppler (PWD) to measure the E/A ratio. Diastolic function was classified as next: normal, impairment of LV relaxation (mild DD, grade I), pseudo-normal (moderate DD, grade II), or restrictive pattern (severe DD, grade III) [(7)].

Ethical approval:

Our study was approved by the Ethics Committee, Mansoura University and written informed consents were obtained from patients' guardians. The Helsinki Declaration was followed throughout the study's conduct.

Statistical Analysis

The SPSS v25 computer software was utilized for data input. Quantitative data were presented as Mean±Standard deviation, range, and median and were compared by independent t-test. Qualitative data were presented as frequency and percentage and were compared by chi^2 test or Monte Carlo test. P-value was considered significant when its value was less than 0.05.

RESULTS

The current study showed that the age of the studied cases ranged from 28 to 90 years with mean 62.05 years. Also, we found that 65% of the studied cases were males and 60% were urban residence. Also, we found that 55% of studied cases were positive troponin (Table 1).

Table (1): Clinical, demographic characteristics and laboratory findings of the included patients:

Variables	N=100
Age/years Mean±SD (min-max)	 62.04±13.56 (28.0-90.0)
Sex Male Female	 65 (65%) 35 (35%)
Residence Urban Rural	 60 (60%) 40 (40%)
Comorbidities DM Hypertension CLD CKD	 50 (50%) 55 (55%) 20 (20%) 10 (10%)
GCS	13 (8.0-15.0)
Systolic blood pressure (mm/Hg)	115.0±27.99
Diastolic blood pressure (mm/Hg)	74.4±16.46
Mean arterial blood pressure (mm/Hg)	88.5±20.09
Pulse (beat/min)	93.8±23.13
Temperature (^{o}C)	37.54±0.66
Respiratory rate (breath/min)	17.90±4.37
WBCS/mm^{3}	22.74±5.65
Hemoglobin (g/dl)	10.84±2.58
Platelets count/mm^{3}	207.5(40-565)
Neutrophils /microlitre	17.56 (4.42-39.53)
Lymphocytes /microlitre	1.02(0.36-22.33)
Neutrophils/Lymphocytes ratio	20.45(0.2-51.3)
INR	1.33±0.24
ALT (IU/L)	33.5(14-716)
AST (IU/L)	52(16-318)
Bilirubin (umol/L)	0.595(0.32-26.8)
Albumin (g/l)	3.3(1.56-5.5)
Creatinine (mg/dl)	1.77(0.8-10)
Sodium (meq/L)	132.5(102-150)
Potassium (meq/L)	4.07±0.98
Troponin Negative Positive	 45 (45%) 55 (55%)

This study revealed statistically significant higher age in +ve troponin group compared with -ve troponin group. The current study revealed statistically significant higher number of patients with pneumonia as a cause of admission among positive troponin group compared with negative troponin group. Patients with positive troponin showed statistically significant higher pulse rate, temperature, lymphocytes, creatinine, AST, ALT and sodium. They also showed statistically significant lower hemoglobin level, neutrophils and albumin. They also showed statistically significant higher incidence of ECG findings (sinus tachycardia, normal sinus rhythm and atrial fibrillation) (Table 2).

Table (2): Comparison between patient's characteristics and clinical data among patients with negative and positive troponin:

	Troponin		Test of significance
	Negative N=45	**Positive N=55**	
Age/years (mean±SD)	57.07±15.36	66.11±10.37	t=3.49, p=0.001*
Pulse (beat/min)	82.33±20.54	103.18±16.48	t=6.096 p=0.001*
Temperature (^oC)	37.19±0.53	37.83±0.62	t=5.41 p=0.001*
WBCS/mm^3	23.51±5.84	22.09±4.07	t=1.06 p=0.293
Hemoglobin (g/dl)	11.59±2.44	10.22±2.55	t=2.73 p=0.007*
Neutrophils/microlitre	20.67±3.78	15.02±3.32	t=3.69 p=0.001*
Lymphocytes/microlitre	1.29±0.31	5.45±1.15	t=23.556 p=0.001*
Neutrophils/lymphocytes ratio	20.95±5.14	20.72±3.72	t=0.074 p=0.941
Albumin (g/l)	3.65±0.86	3.15±0.78	t=3.045 p=0.003*
Creatinine (mg/dl)	2.32±0.25	3.40±0.80	t=8.708, p=0.027*
Sodium (mmol/l)	129.11±11.98	133.25±6.23	t=2.23, p=0.028*
ECG Normal sinus rhythm AF (atrial fibrillation) Bradycardia Sinus tachycardia	 20(44.4) 5(11.1) 5(11.1) 15(33.3)	 20(36.4) 10(18.2) 0 25(45.5)	 χ^{2MC}=8.24 P=0.04*
Diastolic function Normal Dysfunction	 23(51.1) 22(48.9)	 30(54.5) 25(45.5)	 χ^2=0.117 P=0.732
Ejection fraction	0.603±0.06	0.581±0.07	t=1.77, p=0.08
LVEDD (cm)	5.18±0.51	5.02±0.44	t=1.68, p=0.096
LVESD (cm)	3.79±0.43	3.74±0.34	t=0.680, p=0.498
FS %	28.44±5.65	30.18±5.86	t=-1.497, p=0.138

t: Student t test, MC: Monte Carlo test, χ^2: Chi-Square test, *: Statistically significant

Possible predictors of positive troponin were higher pulse rate, creatinine, sodium levels and lower albumin (Table 3).

Table (3): Predictors of high troponin among studied cases:

	β	**P value**	**AOR (95% CI)**
Age/years	1.108	0.061	3.03(0.950-9.65)
Hypertension	-0.470	0.281	0.625(0.266-1.47)
Pulse (beat/min)	0.625	0.041*	1.869(1.03-3.41)
Temperature (^oC)	7.459	0.055	undefined
WBCS /mm^3	-4.705	0.071	0.009(0.001-1.50)
Hemoglobin (g/dl)	-3.187	0.324	0.041(0.002-23.29)
Neutrophils /microlitre	3.662	0.096	38.9340.524-350)
Lymphocytes /microlitre	8.127	0.067	33.85(0.562-56.8)
Albumin (g/l)	-0.495	0.021*	0.610 (0.401-0.927)
Creatinine(mg/dl)	0.218	0.027*	1.243(1.03-1.51)
Sodium (meq/l)	0.056	0.036*	1.058(1.00-1.12)
overall % predicted =90%			

Troponin was a statistically significant independent predictor of mortality, bad outcome and need for vasopressor among studied cases (Table 4).

Table (4): Predictive values of troponin:

	Troponin		Test of significance
	Negative (N=45)	Positive (N=55)	
Mortality (53%)	10(28.6)	25(71.4)	χ^2=5.87, P=0.02*
Vasopressors need (56%)	16	40	χ^2=13.88, P<0.001*
Bad outcome (71%)	30	49	χ^2=7.50, P=0.006*

Data are presented as frequency and percentage, χ^2: Chi-Square test, *: Statistically significant

When patients were classified according to survival, one of the most promising results were statistically significant positive troponin and ejection fraction among non-survived cases compared with survived cases. As well as, statistically significant higher pulse rate, neutrophils/lymphocytes ratio and LVESD among non-survived cases compared with survived cases. As well as, statistically significant lower hemoglobin level among non-survived cases compared with survived cases.

Table (5): Comparison between sociodemographic and clinical data between survived and non-survived cases:

	Survival N=65 (%)	Non survival N= 35 (%)	Test of significance
Age/years mean±SD (min-max)	61.03±14.55	63.91±11.49	t=1.01 p=0.313
GCS	8.2±0.97	13.31±2.69	t=6.76 p<0.001*
Systolic blood pressure (mmHg)	114.62±26.34	115.71±28.21	t=0.186 p=0.853
Diastolic blood pressure (mmHg)	72.92±15.78	77.14±17.54	t=1.23 p=0.220
Mean arterial blood pressure (mmHg)	87.69±17.54	90±21.53	t=0.587 p=0.546
WBCS /mm^3	22.71±5.3	22.78±2.87	t=0.051 p=0.960
Hemoglobin (g/dL)	11.36±2.20	9.86±2.97	t=2.88, p=0.005*
Neutrophils /microlitre	17.35±4.09	17.95±4.12	t=0.355, p=0.723
Lymphocytes /microlitre	3.044±0.64	3.84±0.66	t=0.295, p=0.769
Albumin (g/l)	3.62±0.82	2.91±0.71	t=3.43, p=0.001*
ECG Normal sinus rhythm AF (atrial fibrillation) Bradycardia Sinus tachycardia	 30(46.2) 5(7.7) 5(7.7) 25(38.5)	 10(28.6) 10(28.6) 0 15(42.9)	 χ^2=11.17 P=0.01*
Diastolic function Normal DDG1 DDG2	 33(50.8) 32(49.2)	 20(57.1) 15(42.9)	 χ^2=0.371 P=0.542
Ejection fraction	0.61±0.05	0.555±0.06	t=4.42 p<0.001*
LVEDD (cm)	5.08±0.5	5.11±0.44	t=0.372 p=0.710
LVESD (cm)	3.70±0.41	3.87±0.31	t=2.17 p=0.03*
FS%	28.4±5.38	31.25±6.18	t=2.04 p=0.018*

t: Student t test, χ^2: Chi-Square test, *: Statistically significant

Possible predictors of mortality showed that lower albumin, lower ejection fraction and positive troponin were statistically significant predictors of mortality among studied cases. Also, there was statistically significant positive troponin among patients who need for vasopressors compared with others who didn't need. Also, there was statistically significant positive troponin among patients with bad outcome compared with good outcome patients (Table 6).

Table (6): Predictors of mortality among studied cases:

Predictors of death	β	P value	AOR (95% CI)
GCS	0.301	0.426	1.351(0.643-2.84)
Pulse (beat/min)	0.052	0.206	1.053(0.972-1.14)
Hemoglobin (g/dl)	0.624	0.247	1.87(0.649-5.37)
Lymphocytes /microlitre	0.186	0.111	1.204(0.958-1.51)
Neutrophils/lymphocytes ratio	0.105	0.280	1.111(0.918-1.35)
INR	5.959	0.011*	387.193(UNDEFINED)
ALT (u/dl)	-0.001	0.955	0.999(0.949-1.05)
AST (u/dl)	0.081	0.094	1.085(0.986-1.19)
Bilirubin (umol/l)	0.065	0.076	1.067(0.993-1.15)
Albumin (g/l)	-0.948	0.001*	0.388(0.217-0.691)
Potassium (meq/l)	1.216	0.001*	3.38 (1.70-6.69)
SpO_2 %	0.719	0.004*	2.053(1.25-3.36)
PaO_2	-0.128	0.047*	0.880(0.776-0.998)
FiO_2 %	1.311	0.011*	3.71(1.36-10.17)
Troponin Positive	1.07	0.017*	2.92(1.21-7.04)
Ejection fraction	-19.796	0.002*	Undefined
LVESD (cm)	-0.575	0.578	0.562
% Predicted =70%			

DISCUSSION

An investigation of post-mortem necropsies in septic cases revealed that; more than 50% the cases had cardiac dysfunction. In addition, SICD was associated with a high mortality rate. The clinical results for cases with SICD may be improved if they are evaluated early and managed in time. Of note, SICD evaluation is primarily reliant on both echo and biomarkers. Echo needs operation skills, and errors in operators are difficult to avoid [8].

The main aim of this study was to study if cardiac dysfunction in non-cardiac patients as evidenced by positive troponin (cTnI) and LVSF and LFDF can predict bad outcome in septic patients. Our study was conducted at ED at Faculty of Medicine, Mansoura University. This study was conducted on 100 patients with severe sepsis and /or septic shock.

Our study revealed that 55% of the included non-cardiac patients with sepsis were positive troponin. Likewise, **Jendoubi *et al.*** [9] recorded that the prevalence of elevated cTnI vales in their study was 47%. They added that in a heterogenous population of cases with sepsis, severe sepsis, and septic shock, the mean incidence of increased cTnI was 70%. Also, **Mangi *et al.*** [10] recorded that the prevalence of positive cTnI in their study was 56.8%.

The current study revealed statistically higher age in positive troponin group as compared with negative troponin group. This in agreement with **Umeh *et al.*** [11] that revealed significant higher age in patients with positive troponin in comparison with patients with negative troponin. However, **Mehta *et al.*** [12] and **Lorson *et al.*** [13] showed no statistically significant differences in age between patients with positive and negative troponin.

Troponin is integral to heart function, generally, it's assumed that the greater a subject's cTnI, the greater their risk of death. **Kaura *et al.*** [14] analyzed the data of a quarter of a million cases to assess the significance of minor increase in troponin and the effect of increased troponin in all age groups. They classified their groups according to age and compared their troponin values with outcomes within a period of three years. They reported that in young cases (18-29 years), elevated cTnI values, even if they were minor, indicated a ten-fold increased risk of death. That reported risk diminished with age, ultimately reaching a 1.5-fold increased risk in cases aged 90 or more. Interestingly, raised troponin, however, was a predictor of mortality even in extremely old cases; following three years of follow-up, over half of the individuals over eighty with elevated cTnI levels died. The current study revealed statistically significant higher number of patients with pneumonia as a cause of admission among positive troponin group compared with negative troponin group. This was in agreement with **Efros *et al.*** [15] that reported similar results. The relation between community acquired pneumonia and cardiac diseases depends on a lot of factors. Pneumonia might be accompanied by a systemic inflammatory response inducing extensive hypo-perfusion and multi-organ failure (MOF). The potent systemic inflammatory reaction with a rapid boost of proinflammatory cytokines, C-reactive proteins (CRP), interleukins (IL), and TNF, causes the so-called "systemic inflammatory syndrome" that progresses in extensive hypoperfusion and MOF, comprising sepsis. The ventilation-perfusion mismatch and intrapulmonary shunt result in hypoxemia, a transient increasing in serum

endothelin-I, a vasoconstrictor, and an increase in the coagulation cascade.

The current study revealed statistically significant lower hemoglobin level, neutrophils and albumin among positive troponin group compared with negative troponin group. This was in agreement with **Efros *et al.*** [15] who reported statistically significant lower hemoglobin among high troponin group compared with normal troponin group. There was a significant lower albumin among positive troponin cases in comparison with negative troponin ones. **Kang *et al.*** [16] reported similar results.

The current study revealed statistically significant higher distribution of arrhythmia such as sinus tachycardia and atrial fibrillation among positive troponin group compared with negative troponin group. Likewise, **Poterucha *et al.*** [17] recorded that higher troponin levels was associated with ECG abnormalities such as sinus tachycardia, and atrial fibrillation (AF) in patients with severe sepsis. Also, **Ergün *et al.*** [18] reported new onset AF in septic patients with positive troponin compared with others with negative troponin [18]. Moreover, **Garcia *et al.*** [19] reported higher incidence of cardiovascular complications such as atrial fibrillation and heart failure in non-cardiac septic patients with positive troponin.

The current study revealed statistically significant lower neutrophils and higher lymphocytes among positive troponin group compared with negative troponin group. This in contrast with **Korkmaz *et al.*** [20] who reported higher neutrophils and lower lymphocytes among patients with positive troponin. There was statistically significant higher creatinine among positive troponin group compared with negative troponin group. Similarly, **Majure *et al.*** [21] revealed statistically significant higher serum creatinine levels among high troponin group compared with normal troponin group. However, **Wu *et al.*** [22] reported no significant difference as regard creatinine between normal and high troponin groups. This controversy may be explained by the difference of age between the two studies, difference in sample size and difference in duration of the study. There was statistically significant higher sodium among positive troponin group compared with negative troponin group. **Wu *et al.*** [22] and **Ammann *et al.*** [23] reported that no significant difference regarding sodium among high troponin group compared with normal troponin group. This is suggested to be due to near normalization of blood gases by proper treatment.

The current study revealed no statistically significant differences between patients with positive troponin and negative troponin as regards EF, FS, LVEDD, LVESD. In contrast, previous study, which included 250000 patients during 7 years of study, reported statistically significant lower EF, lower FS, higher LVEDD, higher LVESD, lower E/A ratio among high troponin as compared with normal troponin in patients with severe sepsis [14]. This controversy between the studies may be due to limited number of cases in the current study, limited duration of study and lack of follow up of the cases.

In the present study, multivariate analysis for predictors of positive troponin showed that higher pulse rate, higher creatinine and lower albumin were statistically significant predictors of high troponin among studied cases. This was in agreement with **Vallabhajosyula *et al.*** [24] who reported increased creatinine as a predictor of high troponin. Also, this study was in agreement with **Kang *et al.*** [16] who reported lower albumin as a predictor of high troponin.

Troponin was a statistically significant independent predictor of mortality, bad outcome and need for vasopressor among studied cases. Also**, Hanna *et al.*** [25] and **Lee *et al.*** [26] reported higher need of vasopressors among patients with raised troponin. Also, **Kim *et al.*** [1] reported that sepsis induced cardiac dysfunction (SICD) group showed higher troponin I as a predictor of poor clinical outcomes. Also, **Hanna *et al.*** [25] and **Lee *et al.*** [26] reported higher troponin was a predictor of bad outcome including higher mortality, higher need of vasopressors, higher number of mechanically ventilated cases and higher length of hospital stay (days).

When patients were classified according to survival, one of the most promising results were statistically significant positive troponin and lower ejection fraction among non-survived cases compared with survived cases. As well as, **Lundberg and Weitzberg** [27] demonstrated that there were statistically significant higher pulse rate, Neutrophils/ lymphocytes ratio and LVESD among non-survived cases compared with survived cases. As well as statistically significant lower hemoglobin level was found among non-survived cases compared with survived cases.

In the current study, multivariate analysis for predictors of mortality showed that lower albumin, lower ejection fraction and positive troponin were statistically significant predictors of mortality among studied cases. **Weng *et al.*** [28] have found that lower EF was predictive of mortality in septic cases. On the other hand, **Rolando *et al.*** [29] didn't find differences in the EF value between the survivors and non-survivors. **Garcia *et al.*** [19] concluded that among patients without pre-existing cardiovascular diseases, troponin elevation during sepsis identified patients at a higher possibility for post-sepsis cardiovascular adverse events [19]. Also, **Jendoubi *et al.*** [9] concluded that increased troponin concentrations at 72 hours was accompanied by 28-day mortality in patients with septic shock. **Yin *et al.*** [30] reported that low albumin was a predictor of mortality in cases with severe sepsis.

CONCLUSION

We concluded that high troponin levels may be detected in non-cardiac patients with sepsis and this

group according to new guidelines is considered stage-B pre heart failure patients. Also, we concluded that troponin was an independent predictor of mortality, bad outcome and need for vasopressor among studied cases.

RECOMMENDATION

Future studies should be conducted using well-designed randomized controlled trials or large, comparative observational studies.

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