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Original article

Prediction of fluid status and survival by electrical cardiometry in septic patients with acute circulatory failure

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ABSTRACT

Prediction of fluid status and survival by electrical cardiometry in septic patients with acute circulatory failure.

Background: Septic hemodynamic instability imposes challenges to critical care physician in deciding fluid management to optimize preload dependency state.

Methods: Thirty patients with severe sepsis and hypotension (Mean arterial pressure i.e. MAP < 65 mmHg) and evidence of tissue hypotension i.e. lactate level ≥4 mmol/L were enrolled in our study. Fluid resuscitation (30 ml/kg) was administered. Fluid response was defined as MAP ≥ 65 mmHg with lactate level <4 mmol/L cardiac output (CO), measured by electrical cardiometry, in guiding fluid therapy.

Results: The study included 13 males (43.3%) with age 47.8 ± 19.7. Paired comparison showed significant change in MAP readings (P value < 0.001). ROC curve showed cutoff 12.5% for delta CO to predict fluid responsiveness with Area under Curve (AUC) 0.927, sensitivity 90.0%, and specificity 70.0%. ROC also showed delta CO cutoff 12.5% to predict survival with AUC 0.756, sensitivity 66.7% and specificity 66.7%.

Conclusion: Delta change in cardiac output, measured by electric cardiometry could be used to predict fluid response and survival in acute circulatory failure in septic critically ill patients.

1. Background

Patients in severe sepsis are at risk of acute hemodynamic instability with resultant serious sequelae upon both morbidity and mortality measures. Sepsis results into an unopposed heterogonous vasodilatation associated with cardiac contractile impairment. Fluid therapy poses an important step in managing this acute circulatory failure and reversing morbidities.

However, this created the need to predict fluid responders to optimize fluid resuscitation and organ support therapies. Static measures were studied extensively, but the poor predictive value of static measures and clinical examination has led to investigation of the dynamic measures of fluid responsiveness. In contrast to static measures, dynamic indices rely on the changing physiology of heart lung interactions to determine whether a patient will benefit from increased preload. Increase in cardiac output emerged as an acceptable surrogate for positive fluid response [1].

This has led to investigating goal-directed fluid optimization which showed superiority over standard protocols in surgical settings. A recent meta-analysis of randomized controlled studies also showed superiority of early goal directed protocol over standard protocol in terms of survival benefits [2]. Currently, Surviving Sepsis Campaign (SSC) offers clarification on the implications of the new definition statements and guidance for hospitals [3].

Electrical velocimetry has been validated to monitor cardiac output non-invasively [4–7].

2. Aim of study

To examine an increase in cardiac output, measured by electrical cardiometry as a predictor of survival and fluid response in management of critically ill septic patients with hemodynamic instability.

3. Methods

This is a prospective observational study, which was conducted on thirty patients with the diagnosis of severe sepsis, admitted to the Critical Care department of Cairo University from June 2015 to April 2016. Sepsis was defined using the standard Surviving Sepsis Campaign criteria [2]. Our protocol was approved by the Ethical Committee Review Board of the Faculty of Medicine, Cairo University. All patients consented to participation.

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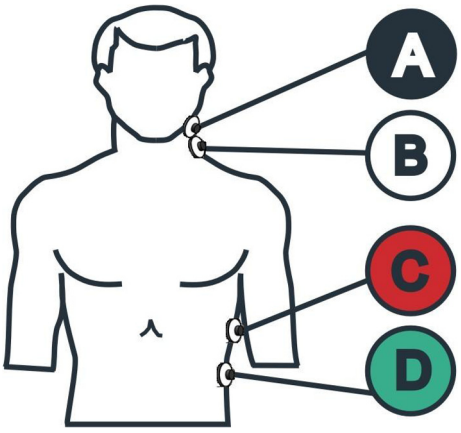

Fig. 1. The Electrical cardiometry sensors.

Patients who experienced hypotension (defined as MAP < 65 mmHg) or lactate ≥4 mmol/L were enrolled in our study. *Exclusion criteria:* Age < 18 years, history of heart disease (e.g. valvular, myopathy, ischemic), history of hepatic or renal diseases, evidence of pulmonary embolism or dysrhythmias. Acute Physiological And Chronic Health Evaluation (APACHE II) scoring system was calculated and lactate withdrawn on admission and 3 h later to assess tissue hypoperfusion, as recommended by current SSC guidelines [2].

Fluid resuscitation (30 ml/kg) was given, patients who exhibited persistent hypotension were maintained on vasopressors. Fluid response was defined as improvement in MAP after fluid resuscitation i.e. MAP ≥ 65 mmHg and lactate <4 mmol/L.

For volume expansion, 30 ml/kg Normal Saline 0.9% was infused over 2 h. Measurements were taken before and after fluid administration: cardiac output (CO) and pulse pressure by ICON ® CARDIOTRONIC, OSYPKA MEDICAL. Patients were studied in a supine position, measured before and after fluid administration. For this purpose, four sensors were applied- first: approximately 5 cm above left base of the neck, second on the left base of neck, third on the lower left thorax at level of xiphoid and the fourth one on the lower left thorax approximately 5 cm below the 3rd electrode at the level of anterior axillary line Fig. 1. The Electrical cardiometry monitor (Electrical Cardiometry monitor, ICON Cardiotronics, Inc., La Jolla, CA 92307; Osypka Medical GmbH, Berlin, and Germany) was connected to the sensor cable and the patient data were fed. The ICON monitor incorporates an algorithm which transforms the ohmic equivalent of mean aortic blood flow acceleration into an equivalent of mean aortic blood flow velocity [8–9].

4. Statistical methods

Numerical variables were described as Mean ± SD. Categorical variables were described as percentages. Comparisons were done using Student 't' test for numerical variables, paired 't' test for paired comparisons and Chi square test for categorical variables. ROC curves were plotted to predict survival and positive fluid response. P value was considered significant if ≤0.05. Statistics were calculated using SPSS 21 package [10].

5. Results

Thirty patients were enrolled in the current study. The study included 13 males (43.3%) with age 47.8 ± 19.7. Average length of ICU stay (LOS) was 10.7 ± 6.2 days. Sepsis with an identified pathogen (proved by microbiological culture) was documented in twenty-five patients (83.3%). APACHE II was 16.7 ± 5.6. Lactate was 3.5 ± 1.9 mmol/L. Mortality was 50.0%.

Paired comparisons between MAP and pulse pressure before and after fluid challenge showed significant differences in MAP readings after fluid challenge (P value < 0.001) while pulse pressure readings did not differ significantly, (P value 0.11) as shown in Table 1. Also paired comparisons for CO showed significant differences in both fluid responders and non-responders, (P < 0.001 for all).

In the current study, electrical impedance was applied to measure cardiac output. Fluid response was determined in 10 patients (33.3%). Fluid non-responders had higher length of ICU stay (12.9 ± 6.5 vs. 6.5 ± 2.3, P 0.001), worse APACHE II score (19.2 ± 4.1 vs. 11.7 ± 4.9, P < 0.001) and higher lactate levels on

Table 1
Paired comparison between initial and follow-up hemodynamic readings.

Hemodynamic parameters	Before fluid challenge	After fluid challenge	P value
MAP	52.9 ± 7.9	63.8 ± 9.0	**<0.001**
Pulse pressure	34.0 ± 11.3	36.0 ± 10.7	0.110

Table 2
Comparison between fluid responders and non-responders for preload assessment.

	Responders	Non-responders	P value
Baseline CO	2.4 ± 0.4	2.6 ± 0.4	0.347
Follow-up CO	2.9 ± 0.4	2.8 ± 0.4	0.882
Delta change CO	17.6 ± 3.4%	11.6 ± 3.0%	**<0.001**

Table 3
Comparison between survivors and non-survivors for preload and volume status assessment.

	Survivors	Non-survivors	P value
Baseline CO	2.4 ± 0.4	2.6 ± 0.4	0.197
Follow-up CO	2.8 ± 0.5	2.9 ± 0.4	0.556
Delta change CO	15.6 ± 4.3%	11.6 ± 3.2%	**0.008**

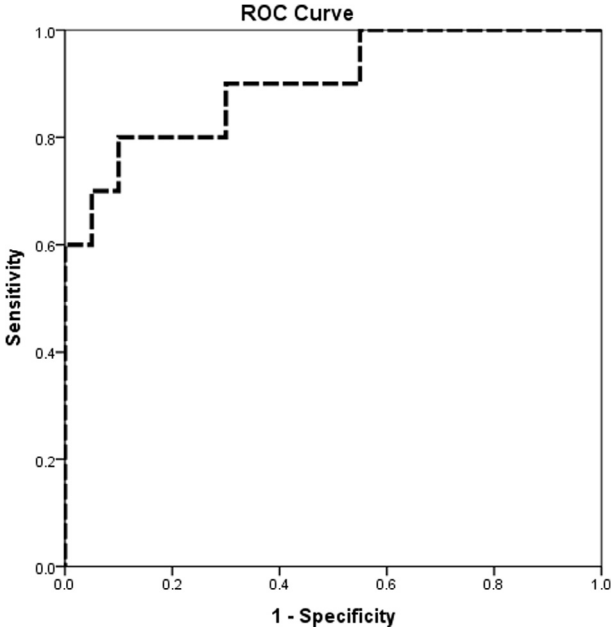

Fig. 2. ROC curve for delta CO to predict positive fluid response.

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