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Short Communication

Acute Kidney Injury does not Alter Energy Metabolism of Septic Patients in Intensive Care Unit

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- Energy expenditure
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Abstract

Background: The determination of resting energy expenditure (REE) in critically ill patients is essential to prevent complications such as hypo and hyper alimentation.

Objectives: This study aims to describe the REE in septic patients with and without acute kidney injury (AKI) and compare the REE estimated by the Harris-Benedict equation (HB) with the REE measured by indirect calorimetry (IC).

Methods: Prospective and observational study was performed. Septic patients older than 18 years, undergoing mechanical ventilation, with or without AKI defined by KDIGO criteria, and admitted to the Intensive Care Unit of University Hospital from Brazil were included. The REE was estimated by HB equation and measured by the IC within 72 hours after the diagnosis of sepsis and seven days after the initial measure.

Results: Sixty-eight patients were evaluated, age was 62.49 ± 16.6 years, 64.7% were male, 63.2% had AKI, and SOFA was 9.81 ± 2.35. The measured REE was 1857.53 ± 685.32 kcal, while the estimated REE was 1514.87 ± 356.72 kcal, with adequacy of 123.49 ± 43%. Septic patients without AKI (n = 25) and with AKI (n = 43) had measured REE statistically higher than the estimated one (1855.0 kcal (1631.75-2052.75) vs. 1551.0 (1349.0 -1719.25), p=0.007 and 1868.0 kcal (1219.5-2364.75) vs. 1388.0 kcal (1254.0-1665.5), p=0.026, respectively). There was no significant difference between the two groups (with and without AKI) in measured and estimated REE (p = 0.63 and 0.64, respectively). There was no significant difference in evolutional REE (1845.95 ± 658.27 kcal vs. 1809.54 ± 755.08 kcal, p = 0.86).

Conclusion: The REE measured by IC was significantly higher than that estimated by the equation HB in both septic with and without AKI. There was no significant difference between the septic patients with and without AKI in REE, suggesting that AKI does not influence the energy metabolism of septic patients.

INTRODUCTION

Sepsis, defined as systemic inflammatory response syndrome associated with infection, is an important cause of morbidity and mortality in patients admitted to intensive care units (ICU) [1,2].

It is the most frequent cause of acute kidney injury (AKI) in critically ill patients, occurring in approximately 19% of patients with sepsis, 23% of patients with severe sepsis and in 51% of patients with septic shock and positive culturesand septic patients developing severe renal failure suffer, despite advanced vital organ support, a high risk of dying [3,4].

It is well know that sepsis and AKI can affect the energy metabolism and treatments based on a better understanding of these alterations may help to prevent weight loss and muscle wasting [5]. Accurate determination of energy needs is obviously important in critically ill patients because both over and underfeeding may be associated with complications and undesired consequences [6].

Few authors studied the energy metabolism in patients with renal failure, with conflicting results [7-14]. Studies have suggested that chronic kidney disease (CKD) is associated with hypometabolic state due to abnormalities in cell metabolism

[10,11]. In contrast, a hypermetabolic state was frequently observed in AKI patients and associated with its cause and severity [15]. The hypermetabolism may be present in AKI patients since AKI is a part of a more complex illness such as sepsis and not necessarily the direct consequence of renal failure per se [15-21]. Thus, it is unknown whether possible changes in energy metabolism observed in septic AKI patients are directly related to AKI itself.

Given the lack of studies on energy metabolism in AKI patients, we decided to measure and compare the resting energy expenditure (REE) in septic patients with and without AKI using indirect calorimetry (IC). This study also aims to compare the REE estimated by the Harris-Benedict equation (HB) with that measured by IC.

PATIENTS AND METHODS

A prospective and observational study was conducted from November 2013 to May 2015 in patients admitted to ICUs from Botucatu Medical School that is a teaching hospital in the city of Botucatu, São Paulo State, southeastern Brazil. It is reference for population for an area comprising 500,000 inhabitants. We included patients 18 years of age or older who had sepsis according to "Survival Sepsis Campaign 2012 [22], andmechanically ventilated using of inspired oxygen (FiO_2) < 0.60. Exclusion criteria were patients with CKD stage 4 and 5 (creatinine clearance lower than 30 mL/min/1.73 m^2, estimated by the modification of diet in renal disease (MDRD) equation) [23].

Septic patients were divided into two groups according to presence or not of AKI associated with sepsis. AKI was defined using KDIGO 2012 criteria [24].

Variables previously reported to be associated with AKI, sepsis or energy metabolism were collected prospectively on each patient by review of the medical record: sex, age, the presence of comorbidities (diabetes, CKD, and hypertension), primary diagnosis, the a etiology of sepsis, prognostic score specific for AKI (ATN-ISS) [25], Sequential Organ Failure Assessment SOFA [26] use of vasoactive drug and neuromuscular blockers, creatinine and urea levels, C reactive protein (CRP) and leukocytes.

The REE was estimated by HB equation [27] and measured by the IC within 72 hours after the diagnosis of sepsis and seven days after the initial measure. IC was performed using QUARK RMR (Cosmed, Rome, Italy). The calorimeter was calibrated before each use. The protocol required that patients be inactive and undisturbed for 30 minutes prior to testing and for 30-minute duration of the data collection. It is recommended that patients achieve steady state during testing. Steady state was defined as a variability of <10% in the measurements of oxygen consumption and carbon dioxide production, and< 5% in the respiratory quotient from minute to minute. The REE was also estimated using HB formula and injury factor for sepsis as suggested by Long et al [28].

Patient height was measured when possible, or it was considered the value documented in the medical record at the time. Weight was measured using calibrated hospital scales in most patients or estimated using Chumlea formula [29].

The Ethics Committee of the Botucatu School of Medicine – UNESP approved this study (approved protocol number 322,535) with a waiver of informed consent given its observational nature.

Statistical analysis

The sample size calculated was 61 patients considering standard deviation 200 kcal, estimated maximum error de 50 kcal in critically ill patients and p value =0.05.

Data analysis was performed using SAS for Windows (version 9.2: SAS Institute, Cary, NC, USA, 2012). Results were expressed as mean and standard deviation or median and interquartile range. The chi-square test was used to compare categorical variables. We used ANOVA to compare parametric variables of clinical, laboratory and nutritional data. For non-parametric variables, the Mann-Whitney test was used, $p<0.05$. Variables with significant univariate associations ($p<0.10$) were candidates for multivariable analysis, which was performed using stepwise variable selection. Repeated measures analysis using the mixed procedure was used for the evolutional REE.

RESULTS

Sixty-eight septic patients admitted to ICU were evaluated. Age was 62.49 ± 16.6 years, 64.7% were male, 64.71% were Caucasian, SOFA was 9.81 ± 2.35, shock septic was the classification of sepsis more frequent (64.71%), lung was the main site of infection (70.6%) and comorbidities were present in 82.85% of patients. The measured REE was 1857 (1308-2261.5) kcal, while the estimated REE was 1449 (1255.5-1677.5) kcal. AKI were present in the most of patients (63.2%) and mortality was high (77.94%). A comparison of baseline characteristics between those who did and did not develop AKI is shown in (Table 1).

AKI group had higher SOFA (11.0±1.73 vs. 7.76±1.79, $p<0.0001$), CRP ($p=0.01$), comorbidities ($p=0.01$) and mortality ($p=0.046$). The groups were similar in gender, age, site of infection. In multivariable regression analysis, comorbidities (OR: 0.07; CI95%: 1.0-1.8) and SOFA (OR: 0.32; CI95%: 0.1-0.5) were identified as predictors of AKI (Table 2).

The (Table 3) shows the comparison between estimated and measured REE in both groups of patients. Septic patients without AKI (n =25) and with AKI (n = 43) had measured REE statistically higher than the estimated one (1855.0 kcal (1636.75-2052.75) vs. 1551.0 (1349.0 -1719.25), $p = 0.007$ and 1868.0 kcal (1219.5-2364.75) vs. 1388.0 kcal (1254.0-1665.5), $p = 0.026$, respectively). The equation HB without using injury factor underestimated the REE in 16.4% in septic patients without AKI and in 25.7% in septic patients with AKI.

When injury factor was used, the measured REE was statistically lower than the estimated one in both groups. Measured and estimated REE were 2467.2(1322-2213.8) vs.1855 (1636.75 – 2052.75), $p<0.001$ in non-AKI group and 2370.63(1456-2451) vs. 1868.0 (1219.5 – 2364.75), $p<0.001$ in AKI group. Thus, the equation HB using injury factor was not precise and overestimated the REE in 33% in septic patients without AKI and in 26.9% in septic patients with AKI.

There was no significant difference between the two groups (with and without AKI) in measured and estimated REE (p =

Table 1: Patients demographics and clinical characteristics (n=68) according to presence of AKI.

Variables	Septic patients (n=68)	AKI septic patients (N=43)	Non-AKI septic patients (N=25)	P
Age (years)	62.49 ± 16.60	65.28 ± 14.50	57.68 ± 19.05	0.068
Male sex (%)	44 (64.71)	27 (62.79)	17 (68.0)	0.66
Race (%) Caucasian	60 (88.24)	38 (88.37)	22 (88.0)	0.91
Sepsis classification (%) shock septic Severe sepsis Sepsis	 44 (64.71) 14 (20.59) 10 (14.70)	 26 (60.47) 11 (25.58) 6 (13.95)	 18 (72.0) 3 (12.0) 4 (16.0)	0.40
Site ofinfection (%)				
Pulmonary	48 (70.59)	26 (60.47)	22 (88.0)	0.085
Abdominal	14 (20.59)	11 (25.58)	3 (12.0)	0.07
Others	6 (8.82)	6 (13.95)	-	0.13
SOFA score*	9.81 ± 2.35	11.0 ± 1.73	7.76 ± 1.79	<0.001
Presence Comorbidities (%) Hipertension Diabetes Mellitus Dislypidemia Obesity	56 (82.85) 42 (61.76) 18 (26.47) 13 (19.11) 8 (11.76)	39 (90.7) 31 (72.09) 15 (34.88) 10 (23.25) 6 (13.95)	17 (68.0) 11 (44.0) 3 (12.0) 3 (12.0) 2 (8.0)	 0.01 0.02 0.09 0.15
Ventilationmode (%) Controled Espontaneous	 54 (79.41) 14 (20.59)	 36 (83.72) 7 (16.28)	 18 (72.0) 7 (28.0)	 0.24
FiO_2	35.97 ± 9.49	35.84 ± 9.59	36.20 ± 9.50	0.88
Use ofvasoactivedrugs (%)	50 (73.53)	34 (79.07)	16 (64.0)	0.17
Use ofsedatives (%)	34 (50.0)	21 (48.84)	13 (52.0)	0.50
Use ofantibiotics (%)	65 (95.59)	41 (95.35)	24 (96.0)	0.69
Presenceoffever (%)	46 (67.64)	29 (67.44)	17 (68.0)	0.59
Urea (mg/dl)	118.82 ± 65.46	150.28 ± 58.32	64.72 ± 34.50	<0.001
Creatinine (mg/dl)	2.35 ± 1.68	3.22 ± 1.51	0.84 ± 0.32	<0.001
CRP (mg/dl)**	29.27 ± 15.72	32.94 ± 14.63	22.98 ± 15.82	0.01
Leukocytes (mm^3)	16392.81± 8761.79	17226.63± 9240.82	14992 ± 7872.42	0.31
Outcomes (%) Death	 53 (77.94)	 38 (88.37)	 15 (60.0)	 0.04

*Sequential Organ Failure Assessment Score; **Acute Kidney Injury; *** C Reactive Protein reactive

Table 2: Multivariable analysis for AKI risk (n=68).

Factors	OR	CI 95%	P
Site of infection	0.4	0.09 – 1.7	0.22
Presence of comorbidities	0.07	1.0 – 1.8	0.03
CRP*	0.94	0.88 – 1.0	0.06
SOFA score **	0.32	0.1 – 0.5	0.0002

Note: OR: Odds Ratio; CI95%: confidence interval of 95%; p: statistical significance
* C Reactive Protein reactive; **Sequential Organ Failure Assessment Score

0.638 and 0.64, respectively). There was no significant difference in evolutional REE (day 1 vs. day 7) in general septic population (1845.95 ± 658.27 kcal vs. 1809.54 ± 755.08 kcal, p = 0.86) and after patients have been divided into AKI (1873.5±718.43 vs.1610.5 ± 629.98, p=0.70) and non-AKI groups (1795.83 ± 557.73 vs. 1915 ± 756.21, p=0.76).

DISCUSSION

This study described and compared the REE estimated by the HB equation and measured by IC in septic patients who developed and did not develop AKI during ICU stay. Its results indicate that HB equation does not agree well with energy expenditure measured by IC in critically ill patients and that AKI

Table 3: Anthropometric characteristics and energetic metabolism of septic patients admitted to intensive care unit according to presence of AKI.

Variables	Septic patients (n=68)	AKI septic patients (n=43)	Non-AKI septic patients (n=25)	P
Weight (Kg)	76.74 ± 25.40	77.23 ± 26.35	75.88 ± 24.17	0.83
Height (cm)	157.82 ± 35.23	160.87 ± 27.05	152.58 ± 46,25	0.35
BMI (Kg/m²)*	27.83 ± 8.59	28.04 ± 7.97	27.48 ± 9.55	0.72
Measured REE (kcal)**	1857(1308-2261.5)[a]	1868 (1219.5-2364 75)[b]	1855 (1636.75-2052.75) [c]	0.62
Estimated REE (kcal)	1449 (1255.5-1677.5)	1388 (1254.0-1665.5)	1551 (1349.0 -1719.25)	0.63
Estimated REE using FI (kcal)	2283.2(1308-2261.5)	2370.63(1456-2451)	2467.2(1322-2213.8)	0.59

*Body Mass Index; ** Resting Energy Expenditure
[a]Different from estimated REE using HB formula without and with injury factor, p<0.001
[b]Different from estimated REE using HB formula without and with injury factor, p=0.007 and < 0.001 respectively
[c]Different from estimated REE using HB formula without and with injury factor, p=0.026 and < 0.001 respectively

Table 4: Evolutional resting energy expendure (day 1 vs day 7) in septic patients according to presence of AKI.

	After seven days		
	measured REE D1 (kcal)	measured REE D7 (kcal)	P
Septic patients (n=22)	1845.95 ± 658.27	1809.54 ± 755.08	0.86
AKI* septic patients (n=16)	1873.5 ± 718.43	1610.5 ± 629.98	0.70
Non-AKI septic patients (n=6)	1795.83 ± 557.73	1915 ± 756.21	0.76

*Acute Kidney Injury, *REE: Rest Energy Expenditure

itself apparently has no direct effect on energy metabolism of septic patients.

The measured REE was higher than the estimated one in general septic population and in groups that developed and did not developed AKI. The equation HB without using injury factor was not precise and underestimated the REE in 16.4% (-304 kcal) in septic patients without AKI and in 25.7% (-480 kcal) in septic group with AKI.

Due to theinaccuracyof this equation,the correction factor was applied. The calculated HB equation was multiplied by injury factor (1.6). However, when injury factor was used, the measured REE was statistically lower than the estimated one in all groups. Thus, the equation HB using injury factor was not precise and overestimated the REE in 33% (+612.2 kcal) in septic patients who did not develop AKI and in 26.9% (+502.6 kcal) in septic patients who developed AKI.

Similar results were observed in previous studies [30-33]. Coletto et al [30] reported in septic patients that HB equation underestimated the REE in 7.6% and when the injury factor as used, the REE was overestimated in more than 50%. In a systematic review, Frankenfield e cols [31] reported the results of an evidence analysis of the accuracy of metabolic rate calculation methods. HB equation had mean differences between measured resting metabolic rate and predicted values ranged from 250 to 900 kcal/ day (meaning that some individual differences can be much higher). Other reviewstudy have suggested not to use the HB equation, with or without correction factors, in critically AKI patients because it underestimated and/or overestimated REE and was inaccurate and unreliable for ICU patient [32].

It may be argued that inaccurate predictions are expected because HB equation was developed long ago and based on data from healthy volunteers. Others equations**as**the Ireton-Jones, Penn state and Faisy were developed from REE measurements of hospitalized and critically ill patients, and dynamic variables as body temperature andminute ventilation that reflect the metabolicstate of the patient were added. Although they areintendedto criticalpatients, several studies showed theseformulashad poor agreement with measured REE by IC [33,34].

Boulattaet al[33] evaluated the energy expenditure equations in a total of 365 patients. They found there were poor accuracy between REE measured by IC and REE predicted by the HB, Mifflin, Penn State and the Ireton-Jones equations. In all cases, the predictive equations underestimated measured REE. In another study, Krooset al [34] evaluated The REE in 927 patients, including 401 obese patients. They also found there were poor agreement between REE measured by IC and REE predicted by the HB, American College of Chest Physicians, Mifflin, and the Ireton-Jones equations. In all cases, except using Ireton-Jones, the predictive equations underestimated measured REE.

Our study agrees with review studies that also suggest that none of these equations has sufficient accuracy and agreement with measured REE in critically ill patients and should not replace the use of IC [33]. Using universal prediction equations to critical ill AKI patients, errors of prediction can occur and lead to overfeeding or underfeeding if they are used to guide the feeding regimen of these patient [35].

Using this data set, we also have demonstrated that there was no significant difference between the groups of septic patients

who developed and did not developed AKI. This suggests that AKI does not affect the energy metabolism of septic patients. Similar results were observed by Schneeweiss e cols [35]. It was the only study that evaluated the REE also in AKI patients. In that study, energy metabolism was measured by IC in 86 patients with AKI and chronic kidney disease (CKD) and in 24 control subjects. In AKI patients with sepsis, the REE was increased *($p < 0.05$)*. In other groups with renal failure (AKI without sepsis, CKD with conservative treatment or hemodialysis, and severe untreated azotemia) the REE was not different from those of control subjects. The authors concluded that renal failure has no influence on energy expenditure as long as septicemia is absent.

Others studies agree with our results that suggest the hypermetabolism may be present in AKI patients since AKI is a part of a more complex illness such as sepsis and not necessarily the direct consequence of renal failure per se. suggesting that AKI does not influence the energy metabolism of septic patients [15-21]

There was no significant difference in evolutional REE (day 1 vs. day 7) in general septic population and after patients have been divided into AKI and non-AKI groups. Different results were observed by Vermeij et al [36] who investigated if only a daily measure of REE could be extrapolated for the whole length of stay in the ICU. The authors noted that there were variations higher than 31% for the same patient, although the daily average is close to the average seven-day study.

Some limitations should be recognized. First, we did not examine others predictive equations currently used in practice such as Mifflin, Penn State and the Ireton-Jones equations. However, the HB equation that we evaluated contain clinical information readily available to practitioners, making it clinically useful equation. Second, we did not have information about treatments that might influence energy expenditure and carbon dioxide production, including type of nutrition and energy intake, catecholamine, neuromuscular blocking agents, and opioids. Finally, we studied a select population of patients, our findings may not be generalizable to all AKI, or critically ill patients.

Despite limitations, this is the largest study to reports that predictive HB equation does not accurately estimate REE in critically ill septic patients and that possible changes in energy metabolism observed in septic AKI patients are not directly related to AKI itself. Our findings suggest that the REE measured by IC was significantly higher than that estimated by the equation HB in both septic with and without AKI and that the equation HB using injury factor also was not precise and overestimated the REE. The lack of difference in REE between the septic patients with and without AKI, suggesting that AKI does not influence the energy metabolism of septic patients and that possible changes in energy metabolism observed in septic AKI patients are not directly related to AKI itself.

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