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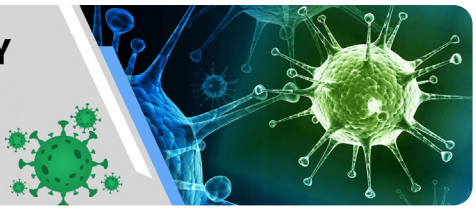

Assessment of Peripheral Perfusion in Severe Acute Respiratory Syndrome Corona virus 2 (Sars-Cov-2) Infection: An Exploratory Analysis with Near-Infrared Spectroscopy

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Abstract

Purpose: To investigate clinical and laboratory tissue perfusion in addition to near-infrared spectroscopy (NIRS) static and dynamic-derived parameters in critically ill COVID-19 patients.

Methods: A cross-sectional single-center exploratory study was performed. Twenty adult patients with confirmed COVID-19 admitted to the intensive care unit (ICU) within 24 hours were prospectively included in this study. A control group without COVID-19 was composed by forty patients included in recently published study. Accessed NIRS-derived parameters included basal tissue oxygen saturation (StO_2), descending slope (%/min), ascending slope (%/min), maximum value of StO2 (StO2max), recovery time (s) and the area under the curve of reactive hyperemia.

Results: The median (IQR) age of included patients was 58 (46-69) years. Patients with COVID-19 presented higher SAPS 3 score [50 (46-53) vs. 45 (30-53), p=0.04] compared with control patients. Patients with SARS-CoV-2 infection showed higher StO2 min [60 (49-79) vs. 54 (48-58) %; p=0.04] and lower descending slope [5.7 (3.4-8.8) vs. 8.1 (6.4-9.7) %/min; p<0.01] compared with ICU patients without COVID-19. Basal StO2 [80 (74-90) vs. 82 (76-86) %; p=0.89], StO2 max [(91 (83-95) vs. 90 (84-94) %; p=0.86], ascending slope [2.0 (1.1-2.9) vs. 2.2 (1.5-3.3) %/min; p=0.43], recovery time [14.5 (12.0-22.0) vs. 21.5 (14.3-28.3) s; p=0.13] and hyperemia area [10.3 (5.8-13.0) vs. 8.6 (4.0-14.3); p=0.55] did not differ between, respectively, COVID-19 and control groups.

Conclusion: Severe COVID-19 patients exhibited a lower rate of oxygen extraction by peripheral tissues than non-COVID-19 critically ill patients, which may represent an adaptive mechanism to hypoxemia. This hypothesis needs to be further investigated.

Keywords: Coronavirus, SARS-CoV-2, Intensive care unit, Hemodynamics, Microcirculation, Near-infrared spectroscopy.

Background

Coronavirus disease 2019 (COVID-19) was first identified in December 2019 in Wuhan, the capital of China's Hubei province [1]. Since then, COVID-19 has spread globally, resulting in the ongoing pandemic, with more than 600 million cases and more than 6 million deaths worldwide [2,

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3]. Studies from the early phase of the pandemic suggested that up to 20% of patients developed severe illness requiring hospitalization, and approximately 5 to 8 percent required admission to an intensive care unit (ICU) [1, 3, 4]. Critically ill patients with COVID-19 disease presented short-term mortality rates that ranged from 35% to 50% before instituting immunization [1, 3].

Endothelial damage is one of the most prominent mechanisms in the pathogenesis of severe COVID-19, resulting from a direct cytopathic damage of the virus on endothelial cells that express Angiotensin Converting Enzyme 2 (ACE2) [5, 6]. Patients with COVID-19 have increased plasma fibrinogen levels, decreased free protein S plasma levels and fibrinolysis, resulting in a hypercoagulability state [7]. The magnitude of the coagulation abnormalities seems to be correlated with the severity of the organ dysfunction [7].

The maintenance of a functional microcirculation is an essential condition for adequate tissue perfusion and cell oxygenation [8, 9]. Tissue oxygen saturation (StO2) measurement using near-infrared spectroscopy (NIRS) has been proposed as a hemodynamic monitoring tool to evaluate microcirculation and assess the balance between oxygen delivery and consumption at the tissue level [10, 11].

Although the correlation between microhemodynamic derangements and poor outcomes in different clinical conditions are well stablished [12-14], few studies have addressed the microvascular reactivity in COVID-19 patients with NIRS technology [15]. Therefore, the aim of the present study was to assess clinical and laboratory tissue perfusion parameters, in addition to NIRS static and dynamic-derived variables in patients with COVID-19 infection.

Materials and Methods

Study design and setting

This cross-sectional single-center exploratory study was conducted in an open medical-surgical ICU of a quaternary care hospital in São Paulo, Brazil. This study was approved by the institutional review board, and written informed consent was obtained from each study participant or their next of kin (CAAE: 33467020.0.0000.0071).

Participants

Twenty adult (≥18 years old) patients with laboratory-confirmed SARS-CoV-2 infection based on positive Reverse-Transcriptase-Polymerase Chain-Reaction (RT-PCR) assay [16] within 24 hours from ICU admission were eligible for this study. Moribund, palliative care and pregnant patients were excluded. A control group was composed of forty patients included in recently published study of our group aiming to evaluate peripheral perfusion in ICU (non-COVID-19) patients [17].

Data collection

Collected variables included demographics, comorbidities, Simplified Acute Physiology Score (SAPS 3 score) [18] at ICU admission, Sequential Organ Failure Assessment (SOFA) score [19], use of fluids, vasopressors (norepinephrine, epinephrine or vasopressin), inotropes, mechanical ventilation (MV) and renal replacement therapy (RRT) at the time of study inclusion and in-hospital mortality.

Systemic hemodynamics, arterial blood gas analysis and lactate

Systemic hemodynamics [heart rate (HR) and mean arterial blood pressure (MAP)] and the administered dose of vasopressors and inotropes were recorded simultaneously with the evaluation of the peripheral perfusion parameters. Fluid balance were recorded from ICU admission until study inclusion. Arterial pH, partial pressure of arterial oxygen (PaO2), partial pressure of arterial carbon dioxide (PaCO2), base excess (BE), hemoglobin and lactate levels were recorded from the closer time of study inclusion.

Peripheral perfusion

Peripheral perfusion was accessed with capillary refill time (CRT) [20] and peripheral perfusion index (PPI) [21]. Capillary refill time was determined by applying pressure on the distal phalanx of the index finger for 15 seconds. A chronometer recorded the time until return to the normal color [20]. Peripheral perfusion index (PPI) was obtained from pulse oximeter (Masimo® SET Radical-7, Masimo Corporation, Irvine, CA, USA) [21]. The PPI reflects changes in peripheral circulation and a value <1.4 was used to define the presence of peripheral vasoconstriction (poor peripheral perfusion) [21].

NIRS monitoring and analysis

The thenar StO2 was measured using the InSpectra StO2 Tissue Oxygenation Monitor (model 650; Hutchinson Technology, Hutchinson, MN, USA) with a 15-mm probe over the thenar eminence [17]. Basal StO2 values were recorded after 3 minutes of NIRS signal stabilization (minimal StO2 variation) [17]. The vascular occlusion test (VOT) was performed through cuff inflation (conventional sphygmomanometer pneumatic cuff) to 30 mmHg above systolic blood pressure (SBP) for 3 minutes and, after that, the occluded cuff was rapidly deflated to 0 mmHg to evaluate the reperfusion phase for 5 minutes [17]. StO2 (%) and tissue hemoglobin index (THI, %) were measured at baseline [17]. The descending slope (%/minute) was calculated from the StO2 baseline until the minimum value of StO2 (StO2min), and the ascending slope (%/minute) was calculated from the StO2min until the maximum value of StO2 (StO2max) [17]. The area under the curve (AUC) of reactive hyperemia was calculated from the StO2max until basal StO2 [17]. All NIRS-

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derived parameters were analyzed by a research software (Hutchinson Technology Inc., Hutchinson, MN, USA) [17].

Statistical analysis

Categorical variables are presented as absolute and relative frequencies. Continuous variables are presented as median with interquartile range (IQR). Normality was assessed using Kolmogorov-Smirnov test. Comparisons between the two groups [COVID-19 and non-COVID-19 (control group)] were performed. Categorical variables were compared with Chi-square test or Fisher exact test when appropriate. Continuous variables were compared using independent t test or Mann-Whitney U test in case of non-normal distribution. A p value of less than 0.05 was considered statistically significant. The software R version 2022.2.2 (R Foundation) was used to perform the analyses and GraphPad Prism version 9.3.0 (GraphPad Software, California, USA) was used for graph plotting.

Table 1: Baseline characteristics of study patients.

Characteristics	COVID-19 (n=20)	Non COVID-19 (n=40)	p-value
Age, years	58 (48-69)	58 (46-68)	0.79[a]
Men, n (%)	17 (85.0)	23 (57.5)	0.04[b]
SAPS 3 score, points	50 (46-53)	45 (30-53)	0.04[a]
SOFA score, points	5 (1-6)	5 (3-8)	0.17[c]
Time between ICU admission and inclusion, h	14 (10-18)	12 (7-16)	0.37[a]
Underlying condition, n (%)			
Systemic hypertension	9 (45.0)	15(37.5)	0.59[b]
Diabetes mellitus	2 (10.0)	8 (20.0)	0.47[b]
Coronary insufficiency	0 (0.0)	7 (17.5)	0.08[b]
Congestive heart failure	0 (0.0)	4 (10.0)	0.29[b]
Organ transplantation	2 (10.0)	2 (5.0)	0.60[b]
Vasoactive drugs, n (%)	4 (20.0)	21 (52.5)	0.03[b]
Norepinephrine	4 (20.0)	21 (53.8)	0.02[b]
Dobutamine	0 (0.0)	10 (25.0)	0.02[b]
Epinephrine	0 (0.0)	2 (5.0)	0.55[b]
Fluid balance, ml	101 (-281-293)	907 (77-1890)	<0.01[a]
Mechanical ventilation, n (%)	6 (30.0)	11 (27.5)	1.00[b]
Renal replacement therapy, n (%)	2 (10.0)	1 (2.5)	0.26[b]

Results

Patients' Characteristics

The median (IQR) age of included patients was 58 (46-69) years. Covid-19 patients were mostly men [17 (85.0%) vs. 23 (57.5%), p=0.04] and presented higher SAPS 3 score [50 (46–53) vs. 45 (30–53), p=0.04] than control group (Table 1).

Values represent median (IQR) or n (%). P values were calculated with (a) Independent *t*-test, (b) Fisher exact test, (c) Mann–Whitney *U* test. SAPS 3: Simplified acute physiology score 3, score range from 0 to 217, with higher scores indicating more severe illness and higher risk of death; SOFA score: sequential organ failure assessment score ranges from 0 to 24, with higher scores indicating more severe organ dysfunction. COVID-19 patients received less vasoactive drugs [4 (20.0%) vs. 21 (52.5%), p=0.03] and had a lower fluid balance [101 (-281-293) ml vs. 907 (77-1890), p<0.01] compared to control patients (Table 1). Hospital mortality did not differ between the groups [4 (20.0%) vs. 5 (12.5%), respectively COVID-19 and non-COVID-19 groups, p=0.46].

Systemic hemodynamics, arterial blood gas analysis and lactate

Systemic hemodynamics, hemoglobin, arterial blood gas analysis and arterial lactate are presented in Table 2. Patients with SARS-Cov2 infection had lower HR [69 (61-80) vs. 85 (75-98) bpm, p<0.01] and higher MAP [88 (82-103) vs. 73 (67-82) mmHg, p<0.01] compared with the control group (Table 2). Lactate [15 (13-21) vs. 25 (16-35) (mg/dl), p<0.01] and PaO_2 [87 (76-96) vs. 119 (89-140) (mmHg), p<0.01] were lower and hemoglobin [13.2 (11.4-14.5) vs. 10.2 (8.9-11.5) (g/dl), p<0.01] and BE [-1.7 (-2.4-1.8) vs. -4.7 (-7.6- -3.1), p<0.01] were higher in patients with COVID-19 compared with the control group (Table 2).

Table 2: Baseline systemic hemodynamics and arterial blood gas analysis

Characteristics	COVID-19 (n=20)	Non COVID-19 (n=40)	p-value
Heart rate, bpm*	69 (61-80)	85 (75-98)	<0.01[a]
MAP, mmHg*	88 (82-103)	73 (67-82)	<0.01[b]
Hemoglobin, g/dl	13.2 (11.4-14.5)	10.2 (8.9-11.5)	<0.01[a]
Arterial lactate, mg/dL[#]	15 (13-21)	25 (16-35)	<0.01[b]
Arterial pH[#]	7.38 (7.30-7.46)	7.37 (7.33-7.39)	0.59[a]
PaO_2, mmHg[#]	87 (76-96)	119 (89-140)	<0.01[a]
$PaCO_2$, mmHg[#]	37 (30-48)	36 (29-38)	0.27[a]
Base excess, mEq/L[#]	-1.7 (-2.4-1.8)	-4.7 (-7.6- -3.1)	<0.01[a]

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Values represent median (IQR). MAP: mean arterial blood pressure, PaO_2: partial pressure of arterial oxygen, $PaCO_2$: partial pressure of arterial carbon dioxide. P values were calculated with the use of (a) Independent *t*-test and (b) Mann–Whitney *U* test. *Systemic hemodynamic variables were recorded at the time of study inclusion simultaneously with the evaluation of the peripheral perfusion parameters. #Arterial blood gas analyses and lactate were recorded from the closer time of inclusion in the study

Peripheral perfusion parameters

PPI was higher in COVID-19 patients compared with the control group [4.5 (1.2-6.0) vs. 2.0 (0.9-2.7), p=0.02] while CRT [1.8 (1.4-2.6) vs. 1.8 (1.2-2.3) seconds, p=1.00] did not differ between groups (Figure 1).

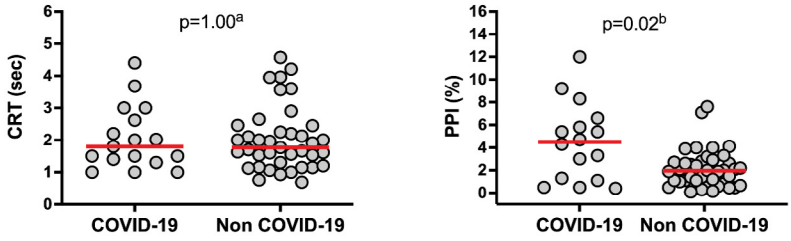

Figure 1: Peripheral perfusion parameters

Red horizontal bars represent median values. CRT: capillary refill time, PPI: peripheral perfusion index. P values were calculated with the use of (a) Mann–Whitney *U* test. and (b) Independent *t*-test

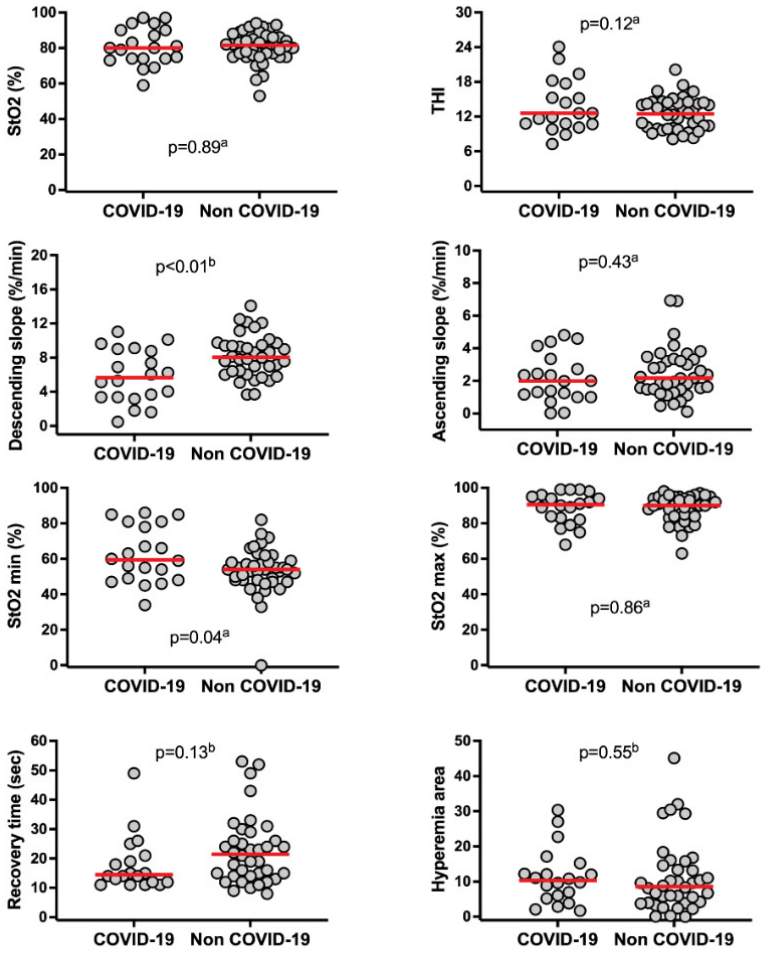

Figure 2: Near-infrared spectroscopy derived parameters

Red horizontal bars represent median values. THI: tissue hemoglobin index, StO_2: tissue oxygen saturation, StO_2 min: minimum StO_2 after arterial occlusion test, StO_2 max: maximum StO_2 after arterial occlusion test. P values were calculated using (a) independent *t* test and (b) Mann–Whitney *U* test.

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NIRS-Derived parameters

COVID-19 patients showed higher StO_2min [60 (49-79) vs. 54 (48-58) %, p=0.04] and lower descending slope [5.7 (3.4-8.8) vs. 8.1 (6.4-9.7) %/minute, p<0.01] compared with ICU patients without SARS-Cov2 infection (Figure 2).

Basal StO_2 [80 (74-90) vs. 82 (76-86) %, p=0.89], THI [12.6 (10.8-17.8) vs. 12.5 (10.0-14.4) %, p=0.12], ascending slope [2.0 (1.1-2.9) vs. 2.2 (1.5- 3.3) %/minute, p=0.43], StO_2max [(91 (83-95) vs. 90 (84-94) %, p=0.86], recovery time [14.5 (12.0-22.0) vs. 21.5 (14.3-28.3) s, p=0.13] and hyperemia area [10.3 (5.8-13.0) vs. 8.6 (4.0-14.3); p=0.55] did not differ between COVID-19 and the control group, respectively (Figure 2).

Red horizontal bars represent median values. THI: tissue hemoglobin index, StO_2: tissue oxygen saturation, StO_2 min: minimum StO_2 after arterial occlusion test, StO_2 max: maximum StO_2 after arterial occlusion test. P values were calculated using (a) independent *t* test and (b) Mann–Whitney *U* test.

Discussion

The main findings of this study are that, compared with non-COVID-19 patients, COVID-19 patients exhibited a higher StO2min and lower descending slope. Additionally, COVID-19 patients had high PPI values when compared with the control group. These findings might be related to fewer functional alterations in microcirculation in COVID-19 and may demonstrate that microvascular reactivity dysfunction is not the main mechanism of COVID-19 pathophysiology. The descending slope represents the drop in StO2 during an arterial occlusion test and is believed to reflect the local oxygen extraction rate at the NIRS-assessed area [22]. Therefore, the descending slope analysis provides an indirect estimate of the local balance between oxygen supply and oxygen demand [22, 23]. In our study the higher StO2min and the lower descending slope in COVID-19 patients compared with the control group may be explained by a lower local metabolic rate and a higher Hb concentration in the former. Mesquita and cols. recently demonstrated that, compared with health controls, COVID-19 associated acute respiratory distress syndrome (ARDS) patients exhibited a higher descending slope and a lower ascending slope [15]. Moreover, the authors demonstrated that the degree of microcirculatory abnormalities were correlated with the severity of ARDS [15].

The PPI has been used to noninvasively assess peripheral perfusion in critically ill patients [21]. Korkut and cols. demonstrated a positive correlation between the severity of SARS-Cov2 infection and the intensity of peripheral vasoconstriction assessed with PPI [24]. Furthermore, they demonstrated that PPI can be used to discriminate the more severe COVID-19 patients admitted to the emergency department [24]. Moreover, Akdur and cols. demonstrated that a PPI lower than 1.5 was independently associated with both 14 days and 90 days mortality [25]. A direct assessment of sublingual microcirculation of COVID-19 patients based on incident dark field (IDF) microscopy imaging has been reported [26-28]. Compared with health volunteers, mechanically ventilated COVID-19 patients exhibited an increased total vessel density (TVD), increased proportion of perfused vessels (PPV), increased perfused vessel density (functional capillary density), and increased capillary hematocrit [26], which represents an adaptative recruitment of microcirculation in response to hypoxia. In another study, compared with septic shock patients, COVID-19 admitted to the ICU had a higher microcirculatory flow index (MFI), a surrogate for microcirculatory perfusion, and a higher PPV than non-COVID-19 patients with septic shock [27].

Our study has limitations. First, this was a single center study. Therefore, our results may have been affected by selection bias. Secondly, we used as a control group a cohort of critically ill patients from another study. Since the first wave of COVID-19 in Brazil, all ICU beds in our hospital were designated to COVID-19 patients [29];therefore, we have not been able to constitute a temporal control group. Finally, peripheral tissue perfusion parameters are dynamically affected by disease severity and time span between disease onset and study inclusion. Thus, our findings must be interpreted with caution since only a punctual assessment of tissue perfusion parameters was performed.

Conclusion

In this prospective single center observational study, we found that critically ill COVID-19 patients exhibited a lower rate of oxygen extraction by peripheral tissues than non-COVID-19 patients, which may represent an adaptive mechanism to hypoxemia. This hypothesis needs to be further investigated.

Ethical Approval and Consent

This study was approved by the institutional review board, and written informed consent was obtained from each study participant or their next of kin (CAAE: 33467020.0.0000.0071).

Authors' Contributions

GMS, RRF and TDC conceived the study design. GMS, VBG, DLR, FSLV, CDM, AMV collected the data. RRF, RCFC and TDC analyzed the data. GMS, RRF, and TDC drafted the first manuscript draft. All authors critically revised the manuscript for important intellectual content. All authors approved the final manuscript and assumed responsibility for the integrity of the data and the accuracy of the data analysis.

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Availability of Data and Materials

The dataset used analyzed during the current study is available from the corresponding author on reasonable request.

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