Falsely low values of oxygen saturation measured by pulse oximetry in patients with coronavirus disease 2019

Sir,

Digital pulse oximetry is a rapid noninvasive test and is used to estimate arterial oxygen saturation. However, falsely low readings are common due to a range of causes including motion artifact, hypotension, nail polish, darker skin pigmentation, and venous pulsations. We recently encountered a number of patients with coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) with falsely low oxygen saturation detected by digital pulse oximetry.

Following the first confirmed COVID-19 case on March 11 in Turkey, our hospital started to serve as a coronavirus pandemic hospital on April 1, 2020. Three hundred and forty-five patients were hospitalized to Istanbul University of Health Sciences Gaziosmanpasa Research and Training Hospital between April 1, 2020, and April 14, 2020. Among these, we identified 17 patients who had a discordance of oxygen saturation measured by digital pulse oximetry and arterial blood gas analysis. This study was approved by our institutional ethics committee (Approvement protocol number: 58/06.05.2020).

The mean (standard deviation [SD]) age was 65.7 ± 17.0 years, and 10 of 17 patients (58.8%) were men. Nine of these 17 patients had fever. Hypotension and tachycardia were not observed in all the patients. Laboratory parameters showed elevated aspartate transaminase (median: 46 U/L, interquartile range [IQR]: 34–58), ferritin (median: 447 ng/mL, IQR: 237–1119), D-dimer (median: 1062 ng/mL, IQR: 774–1387), C-reactive protein (mean ± SD: 141.4 ± 103.1 mg/L), fibrinogen levels (mean ± SD: 382.6 ± 72.0 mg/dL), and low lymphocyte count (mean ± SD: 790 ± 409 cells/μL). The findings of oxygen saturation levels measured by pulse oximetry and arterial blood gas analysis are shown in Table 1. Repeated measurements of oxygen saturation by different pulse oximetry devices were still falsely low in all the patients. Hypertension and chronic ischemic heart disease were the most common comorbidities. In addition, one of them was diagnosed with acute pulmonary thromboembolism. Eleven patients were treated by therapeutic dose enoxaparin, whereas 6 were treated by prophylactic dose enoxaparin. Among the 17 patients, 8 (47%) remained hospitalized at the final study follow-up date, 7 (41.1%) were discharged alive, and the remaining 2 (11.9%) were transferred to the intensive care unit.

The relationship between inflammation due to viral infection and hypercoagulation has already been known.[1] Similarly, extensive intravascular microthrombosis was observed in autopsy series including four deceased cases with COVID-19.[2] Endothelial cell involvement has been suggested a possible reason for impaired microcirculatory function in different vascular beds.[3,4] Ciceri *et al.* have recently proposed a new hypothesis called "microvascular COVID-19 lung vessels obstructive thromboinflammatory syndrome (MicroCLOTS)."[5] This hypothesis was based on the following findings. First, SARS-CoV-2 enters into endothelial cell through the receptor angiotensin-converting enzyme 2. Second, replication of the virus causes release of pro-inflammatory cytokines and activation of macrophages and the complement cascade in endothelial cells. Activation of complement cascade triggers further immune response, tissue injury, and microvascular thrombosis. Third, the progression of endothelial damage with microvascular thrombosis can spread locally in the lung and potentially extends to the microvascular bed of several organs. In our case series,

Table 1: Findings of oxygen saturation measured by pulse oximeter and arterial blood gas

	FiO_2 levels applied	Oxygen saturation (pulse oximeter) (%)	Oxygen saturation (arterial blood gas) (%)
Patient 1	0.21	84	95
Patient 2	0.44	82	97
Patient 3	0.50	79	93
Patient 4	0.44	80	95
Patient 5	0.21	84	95
Patient 6	0.52	70	99
Patient 7	0.21	87	97
Patient 8	0.37	85	94
Patient 9	0.37	84	93
Patient 10	0.40	83	95
Patient 11	0.34	87	95
Patient 12	0.52	80	93
Patient 13	0.37	80	97
Patient 14	0.21	85	92
Patient 15	0.21	89	95
Patient 16	0.34	80	94
Patient 17	0.34	85	91

all of the patients were hemodynamically stable and had no clinical sign of hypoperfusion. In addition, none of the patients had anemia, hypothermia, nail polish, pigmented skin, dyshemoglobinemia, and hyperbilirubinemia that may cause misconceptions in reporting oxygen saturation. Although inappropriate placement of pulse oximeter probe may be a limitation of our observation, repeated measurements by different pulse oximetry devices were consistent. We suggest that discordance between pulse oximeter and arterial blood gas analysis may also indicate widespread microvascular thrombosis, not limited to the lung, and may further support MicroCLOTS hypothesis. Moreover, oxygen saturation is an essential monitoring tool in the management of COVID-19. Some patients may experience silent hypoxemia,[6] and there is an ongoing discussion whether pulse oximetry screening at home could provide an early warning system for COVID-19 pneumonia. However, arterial blood gas analysis seems to be more appropriate to assess arterial oxygen saturation.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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Submitted: 18-May-2020 **Accepted:** 19-May-2020
Published: 30-Oct-2020

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Access this article online

Quick Response Code:

Website: www.lungindia.com

DOI: 10.4103/lungindia.lungindia_392_20

How to cite this article: Satici C, Demirkol MA, Alkan M, Esatoglu SN. Falsely low values of oxygen saturation measured by pulse oximetry in patients with coronavirus disease 2019. Lung India 2020;37:553-4.