

ORIGINAL RESEARCH



Vital signs and work of breathing assessment in the emergency department as predictor for acute respiratory failure in COVID-19 pneumonia

Mia Elhidsi^{1,*}, Menaldi Rasmin¹, Riana Agustin¹, Prasenhadi Pradono¹

¹Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, Universitas Indonesia, Persahabatan National Respiratory Referral Hospital, 13220 Jakarta, Indonesia

***Correspondence**

miapulmo.ui@gmail.com

(Mia Elhidsi)

Abstract

Coronavirus disease 2019 (COVID-19) with acute respiratory failure (ARF) has a high mortality rate. This study aimed to investigate the vital signs and work of breathing parameters in COVID-19 pneumonia patients to predict ARF. We predicted the risk of acute respiratory distress syndrome (ARDS) in COVID-19 patients within 72 hours of admission to the emergency department (ED) and determined cut-off values. We performed an observational prospective cohort study at the tertiary referral Persahabatan Hospital in Jakarta, Indonesia, from July to December 2020. The vital signs were as follows: of respiratory rate (RR), heart rate (HR), pulse oxygen saturation (SpO₂), mean arterial pressure (MAP), and axillary body temperature. The work of breathing, which was indicated by nasal flaring and the contraction of the sternocleidomastoid and abdominal muscles, was assessed one hour after a triage examination. The ARF was monitored within 72 hours. The cut-off values of vital signs were determined using the Youden index. In total, 71 (13.65%) of the 520 patients had ARF within 72 hours of admission. The mean values of RR, HR, MAP and SpO₂ in the ARF group were 26 breaths/minute, and 102 pulses/minute, at 100 and 92%, respectively. All ARF patients had nasal flaring, 86.4% had a contraction of the sternocleidomastoid, and 67.6% had a contraction of the abdominal muscle. The cut-off values for predicting ARF were as follows: RR >23 breaths/minute (sensitivity 83.1%; specificity 86%), SpO₂ <93% (sensitivity 80.5%; specificity 75.2%), HR = 92 pulses/minute (sensitivity 71.8%; specificity 75.2%), and MAP = 93.5 (specificity 64.8%; sensitivity 60.4%). Our results indicate that vital signs and work of breathing within the first hour in the emergency department can predict ARF in COVID-19 pneumonia patients within 72 hours.

Keywords

Acute respiratory failure; COVID-19; Pneumonia; Vital signs; Work of breathing

1. Introduction

Acute respiratory failure (ARF) is defined as a progressive and acute condition of hypoxemia. In general, ARF can be caused by various cardiovascular disorders or systemic diseases [1]. Although not yet fully understood, the pathomechanism of (ARF) due to COVID-19 infection differs from that of ARF caused by other factors and involving mechanisms associated with viral effects and host cell-derived substances. The activation of proinflammatory cytokines, such as interleukin 6 (IL-6) and tumour necrosis factor-alpha (TNF- α), increased activity of neutrophils, natural killer cells, reactive oxygen species (ROS), and various proteases are also involved and contribute to the lung damage, both directly and indirectly [2, 3]. In addition, ARF in COVID-19 pneumonia patients has been under-recognised until the patient experiences infection, inflammation, worsening hypoxemia, ventilatory failure and

organ failure [4–6].

Previous studies have indicated that ARF due to COVID-19 has a higher mortality rate than ARF caused by other factors [7]. A global literature survey reported that ARF related to acute respiratory distress syndrome (ARDS) developed in 33% of patients presenting with COVID-19 pneumonia, of whom, 16% died [8]. Other studies reported that ARF in COVID-19 patients had an incidence rate reaching 20%, with 10 to 20% of the patients requiring mechanical ventilation [9, 10].

Missed or delayed recognition is both common and serious problem [11]. Hence, the early recognition of ARF in the emergency department (ED) can be accomplished by measuring the patient's vital signs and work of breathing. The accurate measurement of vital signs and work of breathing can indicate the patient's outcomes, such as by predicting the risk of patient deterioration [12, 13]. Hence, the early identification of patients whose vital signs indicate an unfavourable progno-

sis could facilitate their prompt referral to a more appropriate hospital.

Vital signs and work of breathing measurements are important parameters in determining patients at risk of deterioration [12, 13]. Vital signs, including respiratory rate (RR), heart rate (HR), oxygen saturation, blood pressure, body temperature and work of breathing, such as nasal flaring, and the contraction of sternocleidomastoids and abdominal muscles, are easy to detect, but valuable in assessing patients' clinical responses and risk of respiratory muscle fatigue, which can lead to hypoventilation and ARF [14]. Therefore, this procedure is highly beneficial, especially when it is performed correctly. They also have the potential to serve as a cost-effective prognostic indicator for patients. A study conducted by Ikran AS in India found that measuring the vital signs of COVID-19 patients in the ED could be used to predict the patient outcomes in a resource-limited hospital setting. In addition, knowledge of the cut-off value of vital signs was required to assess the patients' clinical conditions and determine the risk of ARF [15, 16].

Because of the variations in characteristics, incidences and mortality rates between ARF in COVID-19 and ARF resulting from other diseases, experts have considered developing new specific diagnostic criteria for ARF specifically related to COVID-19 infections [17]. This study aimed to investigate the vital signs and work of breathing to predict ARF in pneumonia COVID-19 patients within 72 hours. The cut-off values for the vital signs were also determined.

2. Materials and methods

2.1 Study design and population

In this cohort observational study, primary data were collected on patients with pneumonia COVID-19 who were admitted to ED in a tertiary national referral hospital, Persahabatan Hospital, from July to December 2020. The inclusion criteria were COVID-19 pneumonia, 18 years old or older, and no diagnosis of ARF at triage. The exclusion criteria were trauma cases, acute cardiac failure, acute coronary syndrome, malignancy, cerebrovascular diseases and pregnancy. After the patients gave their consent to participate in the study, vital signs examination and work of breathing were measured within one hour of admission.

The sample size was determined using a categorical descriptive with a 95% confidence interval (CI), based on a 33% incidence of ARF in COVID-19, 5% precision, and a risk of loss to follow-up of 10% [18]. The minimal required sample size was 374.

2.2 Definition and measurements

COVID-19 pneumonia was determined by clinical, radiological findings and real-time Polymerase Chain Reaction (PCR) examinations of Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [19]. The ARF criteria were as follows: using >6 liters per minute (lpm) of oxygen to achieve SpO₂ 94% or arterial oxygen pressure (PaO₂) <60 mmHg or requiring invasive or non-invasive mechanical ventilation [20–22]. Cardiac disease was defined as a non-ischaemic

acute cardiac dysfunction [23]. Chronic kidney disease (CKD) was defined as abnormalities in kidney structure or function, present for >3 months, with health implications [24].

The patients were examined for vital signs in the supine position without supplemental oxygen. Blood pressure was measured non-invasively in mmHg by the brachial artery using RGB medical comfort sense electronic sphygmomanometer (type BPM001, Sinko Prima Alloy Company, Surabaya, Indonesia). Mean arterial pressure (MAP) was calculated as follows: $MAP = DP + 1/3 (SP - DP)$ or $MAP = DP + 1/3 (PP)$, where DP is the diastolic blood pressure, SP is the systolic blood pressure, and PP is the pulse pressure [25]. RR was measured in one minute and given in breaths/minute [26]. HR was measured by counting the number of pulse beats per minute of the pulse in the wrist [27]. The axillary temperature in degrees Celsius (°C) was measured using a thermometer i-care thermos checker DT-pen [28].

The work of breathing was assessed indirectly through the assessment of nasal flaring and contraction of the sternocleidomastoid and abdominal muscles. Nasal flaring was determined by the movement of the nostrils. Contraction of the sternocleidomastoid was assessed by palpations of its clavicular insertion using two fingers with the hand ipsilateral to the patient's side and detecting increased tension during inspiration. Contraction of the abdominal muscles was assessed by palpating of the abdomen using the hand ipsilateral to the patient's side and detecting increased tension during expiration [29]. These examinations were performed by a doctor or nurse on duty who had been trained prior to the study. The ARF was then monitored for 72 hours after admission.

2.3 Data analysis

Data were recorded primarily and analyzed using IBM SPSS (version 21, Armonk, NY, USA). Only data on patients who were monitored within 72 hours were included in the analysis. Numeric data were presented as median (minimal–maximal), and proportions were presented as quantity and percentage. Vital signs were compared between ARF and without ARF using an independent *t*-test or the Mann-Whitney test. The proportion of work of breathing was compared between patients with ARF and those without ARF using the chi-square test. A *p*-value < 0.05 was assumed to be statistically significant. The cut-off values of vital signs and their sensitivity and specificity were determined using the Youden index.

3. Results

Of 2621 patients admitted to the adult ED, 916 patients were diagnosed with ARF at admission, 534 patients were <18 years old, 613 patients were excluded, and 558 patients were enrolled in this study. Thirty-eight patients were discharged before 72 hours of observation, 520 patients gave consent and their data were analyzed (Fig. 1). In total, 71 (13.65%) of the 520 patients had respiratory failure within 72 hours of admission. The ARF patients were predominantly elderly, male and had a history of hypertension. The ARF group was admitted to the ED on the seventh day of onset (3–12th day) (Table 1).

In our study, the mean values of RR, HR and MAP in the

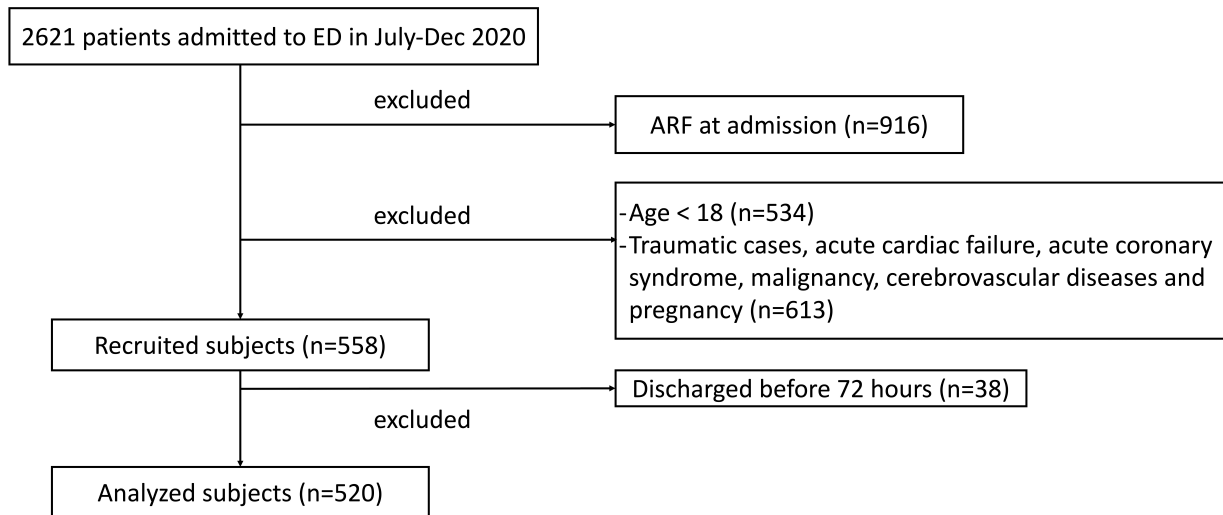


FIGURE 1. Flow chart of subjects. ARF: Acute Respiratory Failure; ED: Emergency Department.

TABLE 1. Clinical characteristics of COVID-19 pneumonia patients admitted to emergency ward (n = 520).

Variable	Acute Respiratory Failure in 72 hours		p-value
	Yes (n = 71) n (%)	No (n = 449) n (%)	
Age			
>65 years old	23 (20.9)	87 (79.1)	
≤65 years old	48 (11.7)	362 (88.3)	
Gender			
Male	44 (14.8)	254 (85.2)	0.393
Female	27 (12.2)	195 (87.8)	
Cardiac disease	17 (26.2)	48 (73.8)	0.002
Diabetes	24 (15.9)	127 (28.3)	0.061
CKD	1 (8.3)	11 (91.7)	0.587
Hypertension	54 (76.0)	292 (65.0)	0.108

CKD: Chronic Kidney Disease.

ARF group were significantly higher than in the non-ARF group, while the SpO₂ was lower in the ARF group than in the non-ARF group. The mean SpO₂ of the ARF group was found to be 4% lower than that of the group without ARF. Most of the patients were admitted to the ED without fever, with a mean body temperature of 36.8 °C in both groups (Table 2). The proportions of nasal flaring and contractions of the sternocleidomastoid muscle and abdominal muscles were higher in the ARF group than in the non-respiratory group (Table 3).

In our finding, the cut-off value of RR >23 x/minute showed the highest sensitivity and specificity for predicting ARF, at a sensitivity of 83.1% and a specificity of 86%. In room air, pulse oxygen saturation was <93% at a sensitivity of 80.5% and a moderate specificity of 75.2%. The HR cut-off value of 92 x/minute and MAP cut-off of 93.5 showed moderate sensitivity and specificity in predicting ARF at 71.8% sensitivity and 75.2% specificity, and at 64.8% sensitivity and 60.4%

specificity, respectively (Table 4).

4. Discussion

Our study was conducted in a tertiary national referral hospital. Most of the patients were already hospitalized in other hospitals and had more severe diseases. A high proportion of ARF patients were admitted. Our result showed that ARF in the COVID-19 pneumonia patients developed on the 9th–10th day of onset, and most patients were admitted to the ED on the 7th day of onset. Previous studies have reported various timings of ARDS, in which the median time from the first symptom to the onset of ARF was related to ARDS at 8–14 days [30–33]. This finding is in contrast to the Berlin ARDS criteria, which specify that ARDS onset is within seven days of COVID-19 symptoms, or new or worsening respiratory symptoms. COVID-19 patients are still at risk of ARF at more than seven days. Therefore, patients should be monitored

TABLE 2. Vital signs and the work of breathing in COVID-19 pneumonia patients admitted to the emergency ward (n = 520).

Variable	Acute Respiratory Failure in 72 hours		p-value
	Yes (n = 71) Median (Min–Max)	No (n = 449) Median (Min–Max)	
Respiratory rate	26 (19–32)	20 (17–28)	<0.001
Heart rate	102 (68–138)	87 (60–131)	<0.001
Temperature	36.8 (36.5–38.8)	36.8 (36.5–40.0)	0.413
Mean arterial pressure	100.00 (73.33–126.67)	91.83 (71.00–156.67)	<0.001
SpO ₂ room air	92 (91–99)	96 (91–100)	<0.001

SpO₂: Pulse Oxygen Saturation.

TABLE 3. Work of breathing in COVID-19 pneumonia patients admitted to the emergency ward (n = 520).

Variable	Acute Respiratory Failure in 72 hours		p-value
	Yes (n = 71) n (%)	No (n = 449) n (%)	
Nasal flaring	6 (100.0%)	0 (0.0%)	<0.001
Contraction of sternocleidomastoid muscle	19 (86.4%)	3 (13.6%)	<0.001
Activation of abdominal muscles	25 (67.6%)	12 (32.4%)	<0.001

TABLE 4. Cut-off of vital signs value based on the Youden index.

Vital signs	Cut-off value	Sensitivity	Specificity	Youden index
Respiratory rate	23.0	0.831	0.860	1.672
Heart rate	93.0	0.718	0.752	1.471
Mean arterial pressure	93.5	0.648	0.604	1.251
SpO ₂ room air	93.0	0.805	0.776	1.564

SpO₂: Pulse Oxygen Saturation.

for the development of ARF as their pneumonia persists or progresses [34].

Several studies reported the occurrence of COVID-16-related acute respiratory distress syndrome (ARDS) vs. non-COVID-19 has some similarities and differences in characteristics. Therefore, the rearrangement of some variables can be considered to predict ARF in detecting the likelihood of mortality COVID-19 induced ARDS [8, 17]. Bain *et al.* [17] reported a longer duration of mechanical ventilation in COVID-19-induced ARDS. Moreover, it was found that the level of IL-6 in COVID-19-related ARDS was lower compared to ARDS mediated by bacterial infection and in the culture-negative groups [17]. These findings have led to controversy regarding the concept of IL-6-related cytokine storm and support the study finding indicating the administration of IL-6 inhibitors does not provide benefits in preventing hypoxemia patients. Yet, the reduction of IL-6 levels in COVID-19-related ARDS does not completely exclude the possibility of micro-inflammation mediated by IL-6 in lung parenchyma. Moreover, there may be subpopulations that demonstrate good clinical outcomes with the administration of IL-6 inhibitors [17, 35].

In our study, out of the 520 patients, a total of 71 patients

(13.65%) who experienced respiratory failure within 72 hours were majority males, elderly and had a medical history of hypertension. Caillon *et al.* [36] showed that elevated systolic blood pressure upon admission was a major factor in the mortality prediction models. However, it remains unclear whether these findings reflect the burden of uncontrolled hypertension in the elderly population or whether it is arisen due to systemic inflammation. Others results have suggested that the mechanism of enzymatic activity of angiotensin-converting enzyme 2 (ACE-2) disruption is caused by SARS-CoV-2 [36].

Previous studies have reported that the relationship between hypertension and poor outcome in COVID-19 infection among the elderly remains controversial [7]. Lee *et al.* [37] found that hypertension was not significantly associated with COVID-19 mortality in elderly patients. Increased mortality due to hypertension may be potentially associated with the risk of kidney damage or acute kidney injury [38]. Furthermore, Ramos JM *et al.* [39] reported that limited studies are confirming the association between hypertension and an increased risk of COVID-19 mortality in the older population.

Several studies have reported findings related to a higher incidence and mortality of COVID-19 in elderly men compared with elderly women [7, 40]. These findings have been

linked to various mechanisms, including a decrease in the total number of B and T cells, as well as the aging of Cluster of Differentiation (CD8⁺) T effector memory cells in older men compared with the older women. In addition, a greater reduction in cytokine secretion and T cell proliferation activity was observed in older men compared with women [40].

Several studies have demonstrated associations among reduced oxygen saturation, increased glucose levels, and elevated respiratory rate on admission with an unfavourable outcome in patients with COVID-19 [41–44]. RR was found to be an important parameter for predicting ARF in COVID-19 pneumonia patients within 72 hours. RR is regulated by the central nervous system based on input from various central and peripheral chemoreceptors to maintain oxygen and carbon dioxide levels in the blood by maintaining their near-normal levels [45]. Adults normally take 12–20 breaths per minute, and an increase in RR indicates the need for more oxygen or less carbon dioxide. RR can also be an indicator of physiological conditions, such as hypoxia, hypercapnia and metabolic and respiratory acidosis [46]. An increase in RR, especially in hypovolemia, is an early marker of acidosis. The compensatory mechanism will work by increasing RR and HR without significant changes in blood vessels [47]. A previous study based on the work of breathing scale showed that there was a low incidence of accessory respiratory muscle use when the RR was ≤ 20 , however, as the RR increased, the use of accessory respiratory muscles increased proportionally [29].

Our study succeeded in determining the RR cut-off value. Based on this finding, clinicians can predict the risk of ARF and provide further evaluation and appropriate treatment interventions. In previous studies, the failure to monitor RR was associated with an increased risk of death. An RR > 36 breaths/minute and an HR > 140 pulse/minute were related to cardiac arrest [48]. Gibson *et al.* [49] considered RR ≥ 30 breaths/minute and SpO₂ $\leq 92\%$ as early signs of ARF. In our study, we found that a lower cut-off value of RR > 23 breaths/minute was a predictor of ARF in COVID-19 pneumonia patients. This finding indicates the possibility of “happy hypoxia” in COVID-19, where hypoxemia occurs without a proportional increase in RR, and rapid deterioration can occur [50].

Fever is a common symptom among most COVID-19 patients who need hospitalisation. Moreover, body temperature may indicate the severity of inflammation [51]. Our findings revealed that the majority of the patients in this study arrived at the ED without experiencing fever, and the average body temperature in both groups was recorded as 36.8 °C. At the present, there is a lack of published studies that have examined body temperature as a potential prognostic indicator. However, Tharakan S *et al.* [51] reported that inadequate control of body temperature during the COVID-19 infection is indicative of an unfavourable prognosis, suggesting that body temperature can be used as a valuable easy-obtained prognostic marker.

In our study, the cut-off value of SpO₂ $< 93\%$ was in line with the target oxygen level in COVID-19 guidelines [52, 53]. SpO₂ $< 93\%$ is considered to indicate a high patient risk of developing more severe disease and mortality. The LOCO-2 trial, which compared a conservative target with SpO₂ 88–92% and a liberal target of SpO₂ $\geq 96\%$ reported that at day 90,

more patients in the conservative-oxygen group had died than in the liberal-oxygen group [54]. The intensive monitoring of pulse oximetry can detect a sudden drop in SpO₂ of 3–4% [55]. In 2002, Ikram AS *et al.* [56] revealed that a lower oxygen saturation level on admission was identified as an independent risk factor contributing to a higher mortality rate in COVID-19 patients. Furthermore, for every 1% increase in the initial oxygen saturation level, the likelihood of mortality decreased by 7.8%. This finding suggests that the delayed presentation of patients with COVID-19 [56]. Ambulatory oxygen saturation monitoring has also been showed to be useful in identifying patients who may require high levels of oxygen [57]. Moreover, in South Africa, Nematswerani *et al.* [58] reported that patients who utilised a pulse oximeter to monitor their oxygen saturation levels at home experienced considerably lower mortality rates compared with those who did not use this monitoring method.

We acknowledge several limitations of this study. Firstly, we recognize the potential for measurement bias, which may impact the reliability of our measurements. Nevertheless, we made efforts to minimize this bias by conducting measurement training before the commencement of the study and providing regular training sessions in every month throughout the study duration. Secondly, we emphasize that while numerous COVID-19 risk prediction models have been established, the specific cut-off values of vital signs obtained in this research require validation in the context of COVID-19 pneumonia and other clinical settings. The applicability of these cut-off values in developing countries with limited resources remains uncertain and warrants further investigation. Additionally, further studies should be conducted with a larger sample sizes to evaluate the performance of a prediction model including vital signs at admission and appropriate external validation. Moreover, the implementation and innovation of COVID-19 risk prediction models based on easily obtainable parameters, could serve as a crucial tool for addressing future epidemics wave of infection in resource-limited settings.

5. Conclusions

The assessment of vital signs and the work of breathing within the first hour in the ED can predict ARF within 72 hours in COVID-19 pneumonia patients. This model has the potential to serve as a cost-effective method for predicting the prognosis of the course of COVID-19 infections in resource-limited settings and in developing countries. However, its applicability in the resource-limited health centres needs to be validated in order to ascertain the relevance of vital sign cut-off values in COVID-19 pneumonia and other clinical scenarios. Furthermore, further research with a larger sample sizes is crucial to be conducted to assess the effectiveness of this model in a broader population.

ABBREVIATIONS

ARDS, Acute Respiratory Distress Syndrome; ARF, Acute Respiratory Failure; PaO₂, Arterial Oxygen Pressure; CI, confidence interval; CKD, Chronic kidney disease; COVID-19, Coronavirus disease 2019; DP, diastolic pressure; ED, Emer-

gency Department; HR, Heart Rate; MAP, Mean Arterial Pressure; SpO₂, Pulse Oxygen Saturation; RR, Respiratory Rate.

AVAILABILITY OF DATA AND MATERIALS

Data that support the findings in this study are available from the corresponding author upon reasonable request. The data are not publicly available due to privacy and ethical reasons.

AUTHOR CONTRIBUTIONS

ME and MR—designed the research study. ME and RA—performed the research; wrote the manuscript. MR and PP—provided help and advice for the research. RA—analysed the data. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The Medical Ethics Committee of Universitas Indonesia and Persahabatan Hospital gave approval for the study (registration number: KET-977/UN2.F1/ETIK/PPM.00.02/2020). All patients in this study or their closest relatives gave written consent to participate in this study.

ACKNOWLEDGMENT

We thank the 6th ICE on the IMERI committee, who had supported the manuscript preparation. We also thank to Aria Kekalih for assistance in conducting the statistical analysis.

FUNDING

This research received no external funding.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

- [1] Meyer NJ, Gattinoni L, Calfee CS. Acute respiratory distress syndrome. *The Lancet*. 2021; 398: 622–637.
- [2] Aslan A, Aslan C, Zolbanin NM, Jafari R. Acute respiratory distress syndrome in COVID-19: possible mechanisms and therapeutic management. *Pneumonia*. 2021; 13: 14.
- [3] Elhidsi M, Fachruha F, Irawan RY. N-Acetylcysteine for COVID-19: a potential adjuvant therapy. *Journal of Health Sciences*. 2021; 11: 1–6.
- [4] Sankey CB, McAvay G, Siner JM, Barsky CL, Chaudhry SI. “Deterioration to door time”: an exploratory analysis of delays in escalation of care for hospitalized patients. *Journal of General Internal Medicine*. 2016; 31: 895–900.
- [5] Yi P, Yang X, Ding C, Chen Y, Xu K, Ni Q, *et al*. Risk factors and clinical features of deterioration in COVID-19 patients in Zhejiang, China: a single-centre, retrospective study. *BMC Infectious Diseases*. 2020; 20: 943.
- [6] Bellani G, Laffey JG, Pham T, Fan E, Brochard L, Esteban A, *et al*. Epidemiology, patterns of care, and mortality for patients with acute respiratory distress syndrome in intensive care units in 50 countries. *JAMA*. 2016; 315: 788–800.
- [7] Dadras O, SeyedAlinaghi S, Karimi A, Shamsabadi A, Qaderi K, Ramezani M, *et al*. COVID-19 mortality and its predictors in the elderly: a systematic review. *Health Science Reports*. 2022; 5: e657.
- [8] Tzotzos SJ, Fischer B, Fischer H, Zeitlinger M. Incidence of ARDS and outcomes in hospitalized patients with COVID-19: a global literature survey. *Critical Care*. 2020; 24: 516.
- [9] Cummings MJ, Baldwin MR, Abrams D, Jacobson SD, Meyer BJ, Balough EM, *et al*. Epidemiology, clinical course, and outcomes of critically ill adults with COVID-19 in New York City: a prospective cohort study. *The Lancet*. 2020; 395: 1763–1770.
- [10] Montrieff T, Ramzy M, Long B, Gottlieb M, Hercz D. COVID-19 respiratory support in the emergency department setting. *The American Journal of Emergency Medicine*. 2020; 38: 2160–2168.
- [11] Bellani G, Pham T, Laffey JG. Missed or delayed diagnosis of ARDS: a common and serious problem. *Intensive Care Medicine*. 2020; 46: 1180–1183.
- [12] Kellett J, Sebat F. Make vital signs great again—a call for action. *European Journal of Internal Medicine*. 2017; 45: 13–19.
- [13] Gazmuri RJ, Apigo M, Fanapour P, Nadeem A. Abstract 108: work of breathing scale to assess need of intubation in Covid-19 pneumonia. *Circulation*. 2020; 142: A108.
- [14] Barrett NA, Hart N, Camporota L. Assessment of work of breathing in patients with acute exacerbations of chronic obstructive pulmonary disease. *COPD: Journal of Chronic Obstructive Pulmonary Disease*. 2019; 16: 418–428.
- [15] Leuvan CHV, Mitchell I. Missed opportunities? An observational study of vital sign measurements. *Critical Care and Resuscitation*. 2008; 10: 111–115.
- [16] Chua WL, Mackey S, Ng EKC, Liaw SY. Front line nurses’ experiences with deteriorating ward patients: a qualitative study. *International Nursing Review*. 2013; 60: 501–509.
- [17] Bain W, Yang H, Shah FA, Suber T, Drohan C, Al-Yousif N, *et al*. COVID-19 versus non-COVID-19 acute respiratory distress syndrome: comparison of demographics, physiologic parameters, inflammatory biomarkers, and clinical outcomes. *Annals of the American Thoracic Society*. 2021; 18: 1202–1210.
- [18] Zhao X, Xu X, Yin H, Hu Q, Xiong T, Tang Y, *et al*. Clinical characteristics of patients with 2019 coronavirus disease in a non-Wuhan area of Hubei province, China: a retrospective study. *BMC Infectious Diseases*. 2020; 20: 311.
- [19] World Health Organization. Therapeutics and COVID-19: living guideline. 2022. Available at: <https://www.who.int/publications-detail-redirect/WHO-2019-nCoV-therapeutics-2022.4> (Accessed: 04 September 2022).
- [20] Elhidsi M, Rasmin M, Prasenoahadi. In-hospital mortality of pulmonary tuberculosis with acute respiratory failure and related clinical risk factors. *Journal of Clinical Tuberculosis and other Mycobacterial Diseases*. 2021; 23: 100236.
- [21] Rasmin M, Elhidsi M, Prasenoahadi, Putra Yahya WS, Sutanto YS, Setijadi AR, *et al*. Underlying diseases and in-hospital mortality of acute respiratory failure patients: Indonesian prospective cohort study. *Journal of Natural Science, Biology and Medicine*. 2021; 12: 22–26.
- [22] Rasmin M, Elhidsi M, Yahya WS. Characteristics and outcome of acute respiratory failure patients: a cross-sectional study from the national referral hospital for respiratory diseases. *Pneumologia*. 2018; 67: 77–81.
- [23] McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, *et al*. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *European Heart Journal*. 2021; 42: 3599–3726.
- [24] Kidney Disease: Improving Global Outcomes (KDIGO) Blood Pressure Work Group. KDIGO 2021 clinical practice guideline for the management of blood pressure in chronic kidney disease. *Kidney International*. 2021; 99: S1–S87.
- [25] DeMers D, Wachs D. Physiology, Mean Arterial Pressure. *StatPearls*. StatPearls Publishing: Treasure Island (FL). 2023.
- [26] Drummond GB, Fischer D, Arvind DK. Current clinical methods of measurement of respiratory rate give imprecise values. *ERJ Open Research*. 2020; 6: 00023–02020.

- [27] American Heart Association. All about heart rate (pulse). 2022. Available at: <https://www.heart.org/en/health-topics/high-blood-pressure/the-facts-about-high-blood-pressure/all-about-heart-rate-pulse> (Accessed: 04 September 2022).
- [28] Boyer J, Eckmann J, Strohmayer K, Koele W, Federspiel M, Schenk M, *et al.* Investigation of non-invasive continuous body temperature measurements in a clinical setting using an adhesive axillary thermometer (SteadyTemp®). *Frontiers in Digital Health.* 2021; 3: 794274.
- [29] Apigo M, Schechtman J, Dhliwayo N, Al Tameemi M, Gazmuri RJ. Development of a work of breathing scale and monitoring need of intubation in COVID-19 pneumonia. *Critical Care.* 2020; 24: 477.
- [30] Chen SL, Feng HY, Xu H, Huang SS, Sun JF, Zhou L, *et al.* Patterns of deterioration in moderate patients with COVID-19 from Jan 2020 to Mar 2020: a multi-center, retrospective cohort study in China. *Frontiers in Medicine.* 2020; 7: 567296.
- [31] Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, *et al.* Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *The Lancet.* 2020; 395: 497–506.
- [32] Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, *et al.* Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus—infected pneumonia in Wuhan, China. *JAMA.* 2020; 323: 1061–1069.
- [33] Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z, *et al.* Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. *The Lancet.* 2020; 395: 1054–1062.
- [34] Li X, Ma X. Acute respiratory failure in COVID-19: is it “typical” ARDS? *Critical Care.* 2020; 24: 198.
- [35] Sinha P, Matthay MA, Calfee CS. Is a “cytokine storm” relevant to COVID-19? *JAMA Internal Medicine.* 2020; 180: 1152–1154.
- [36] Caillon A, Zhao K, Klein KO, Greenwood CMT, Lu Z, Paradis P, *et al.* High systolic blood pressure at hospital admission is an important risk factor in models predicting outcome of COVID-19 Patients. *American Journal of Hypertension.* 2021; 34: 282–290.
- [37] Lee JY, Kim HA, Huh K, Hyun M, Rhee JY, Jang S, *et al.* Risk factors for mortality and respiratory support in elderly patients hospitalized with COVID-19 in Korea. *Journal of Korean Medical Science.* 2020; 35: e223.
- [38] Trecarichi EM, Mazzitelli M, Serapide F, Pelle MC, Tassone B, Arrighi E, *et al.* Clinical characteristics and predictors of mortality associated with COVID-19 in elderly patients from a long-term care facility. *Scientific Reports.* 2020; 10: 20834.
- [39] Ramos-Rincon J, Buonaiuto V, Ricci M, Martín-Carmona J, Paredes-Ruiz D, Calderón-Moreno M, *et al.* Clinical characteristics and risk factors for mortality in very old patients hospitalized with COVID-19 in Spain. *The Journals of Gerontology.* 2021; 76: e28–e37.
- [40] Perrotta F, Corbi G, Mazzeo G, Boccia M, Aronne L, D’Agnano V, *et al.* COVID-19 and the elderly: insights into pathogenesis and clinical decision-making. *Aging Clinical and Experimental Research.* 2020; 32: 1599–1608.
- [41] Youssef Ali Amer A, Wouters F, Vranken J, Dreesen P, de Korte-de Boer D, van Rosmalen F, *et al.* Vital signs prediction for COVID-19 patients in ICU. *Sensors.* 2021; 21: 8131.
- [42] Singh AK, Singh R. Hyperglycemia without diabetes and new-onset diabetes are both associated with poorer outcomes in COVID-19. *Diabetes Research and Clinical Practice.* 2020; 167: 108382.
- [43] Sands KE, Wenzel RP, McLean LE, Korwek KM, Roach JD, Miller KM, *et al.* Patient characteristics and admitting vital signs associated with coronavirus disease 2019 (COVID-19)—related mortality among patients admitted with noncritical illness. *Infection Control & Hospital Epidemiology.* 2021; 42: 399–405.
- [44] Elhidsi M, Kusumoputri Buwono DA, Musridharta E, Soehardiman D, Prasenohadi P. Non-invasive ventilation in neuromuscular disease with acute respiratory failure: a narrative review. *Romanian Journal of Neurology.* 2022; 21: 219–224.
- [45] Marjanovic N, Mimoz O, Guenezan J. An easy and accurate respiratory rate monitor is necessary. *Journal of Clinical Monitoring and Computing.* 2020; 34: 221–222.
- [46] Rolfe S. The importance of respiratory rate monitoring. *British Journal of Nursing.* 2019; 28: 504–508.
- [47] Carrara M, Ferrario M, Bollen Pinto B, Herpain A. The autonomic nervous system in septic shock and its role as a future therapeutic target: a narrative review. *Annals of Intensive Care.* 2021; 11: 80.
- [48] Chatterjee NA, Jensen PN, Harris AW, Nguyen DD, Huang HD, Cheng RK, *et al.* Admission respiratory status predicts mortality in COVID-19. *Influenza and other Respiratory Viruses.* 2021; 15: 569–572.
- [49] Gibson PG, Qin L, Puah SH. COVID-19 acute respiratory distress syndrome (ARDS): clinical features and differences from typical pre-COVID-19 ARDS. *The Medical Journal of Australia.* 2020; 213: 54–56.e1.
- [50] Dhont S, Derom E, Van Braeckel E, Depuydt P, Lambrecht BN. The pathophysiology of ‘happy’ hypoxemia in COVID-19. *Respiratory Research.* 2020; 21: 198.
- [51] Tharakan S, Nomoto K, Miyashita S, Ishikawa K. Body temperature correlates with mortality in COVID-19 patients. *Critical Care.* 2020; 24: 298.
- [52] World Health Organization. Clinical management of severe acute respiratory infection (SARI) when COVID-19 disease is suspected. Interim guidance. *Pediatrics i Medycyna Rodzinna.* 2020; 16: 9–26.
- [53] Elhidsi M, Rasmin M, Prasenohadi, Aniwidyansih W, Desianti GA, Alatas MF, *et al.* Rational supplemental oxygen therapy in COVID-19. *Sahel Medical Journal.* 2020; 23: 201–205.
- [54] Barrot L, Asfar P, Mauny F, Winiszewski H, Montini F, Badie J, *et al.* Liberal or conservative oxygen therapy for acute respiratory distress syndrome. *The New England Journal of Medicine.* 2020; 382: 999–1008.
- [55] Joshi LR. Principles, utility and limitations of pulse oximetry in management of COVID-19. *Journal of Lumbini Medical College.* 2020; 8: 105–110.
- [56] Ikram AS, Pillay S. Admission vital signs as predictors of COVID-19 mortality: a retrospective cross-sectional study. *BMC Emergency Medicine.* 2022; 22: 68.
- [57] Akhavan AR, Habboushe JP, Gulati R, Iheagwara O, Watterson J, Thomas S, *et al.* Risk stratification of COVID-19 patients using ambulatory oxygen saturation in the emergency department. *The Western Journal of Emergency Medicine.* 2020; 21: 5–14.
- [58] Nematswerani N, Collie S, Chen T, Cohen M, Champion J, Feldman C, *et al.* The impact of routine pulse oximetry use on outcomes in COVID-19-infected patients at increased risk of severe disease: a retrospective cohort analysis. *South African Medical Journal.* 2021; 111: 950–956.

How to cite this article: Mia Elhidsi, Menaldi Rasmin, Riana Agustin, Prasenohadi Pradono. Vital signs and work of breathing assessment in the emergency department as predictor for acute respiratory failure in COVID-19 pneumonia. *Signa Vitae.* 2024; 20(2): 63-69. doi: 10.22514/sv.2024.017.