Mortality Risk Score for Critically Ill Patients with Viral or Unspecified Pneumonia: Assisting Clinicians with COVID-19 ECMO Planning

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Abstract. Respiratory complications due to coronavirus have claimed hundreds of thousands of lives in 2020. Extracorporeal membrane oxygenation (ECMO) is a life-sustaining oxygenation and ventilation therapy that may be used when mechanical ventilation is insufficient. While early planning and surgical cannulation for ECMO can increase survival, clinicians report the lack of a risk score hinders these efforts. We develop the PEER score to highlight critically ill patients with viral or unspecified pneumonia at high risk of mortality in a subpopulation eligible for ECMO. The score is validated across two critical care datasets, and predicts mortality at least as well as other existing risk scores.

Keywords: mortality risk score · pneumonia · COVID-19 · ARDS.

1 Introduction

Coronavirus disease COVID-19 has infected millions globally. Many cases progress from Severe Acute Respiratory Syndrome (SARS-CoV-2) with viral pneumonia to acute respiratory distress syndrome (ARDS) to death. ECMO can temporarily sustain patients with severe ARDS when mechanical ventilation fails to facilitate with oxygenation via lungs. However, ECMO is costly and applicable only for patients healthy enough to recover and return to a high functional status.

While ECMO is more effective when planned in advance [7], applicable risk scores remain unavailable [2, 17]. This paper introduces the Viral or Unspecified **P**neumonia **E**CMO-**E**ligible **R**isk (PEER) Score, using measurements from the time of would-be planning—early in the critical care stay. In contrast to existing pneumonia risk scores [6, 8, 18, 19], the PEER score targets those with viral or unspecified pneumonia in the critical care setting, for a cohort potentially eligible for ECMO. Unspecified pneumonia is included since the infectious etiology of pneumonia often cannot be determined, and it broadens the study population.

Though limited by geographic availability, ECMO usage has increased 4-fold in the last decade [22]. COVID-19 guidelines suggest ECMO as a late option in

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escalation of care for severe ARDS secondary to SARS-CoV-2 infection [1, 17]. However, early epidemiological studies of coronavirus [27, 30, 31] have yet to establish ECMO's utility. A pooled analysis of four studies [13] showed mortality rates of 95% with ECMO vs. 70% without, but the number of ECMO recipients was small, and no studies described a protocol specifying indications for ECMO.

To better understand the role of ECMO as a rescue for ventilation non-responsive, SARS-CoV-2 ARDS, we study its broader use in ARDS. Treatment guidelines suggest ECMO use in severe ARDS alongside other advanced ventilation strategies [20, 28], with the World Health Organization citing effectiveness for ARDS and reducing mortality of the Middle East Respiratory Syndrome (MERS). Despite these recommendations and allocated ECMO resources [22], risk scores tailored to ECMO consideration are lacking. Our study addresses this by drawing from viral and source unidentified cases of pneumonia that escalate to critical care admissions, guided by the intuition that ARDS from these pneumonia are expected to better resemble COVID-19 ARDS than all-comer ARDS.

Related Work There are a number of pneumonia [6, 8, 11, 18, 26], COVID-19 [9, 10, 15], hospitalization mortality [32], and ECMO risk scores [23], but none center on the time of risk evaluation for ECMO candidacy. The pneumonia and COVID-19 risk scores are assessed on populations with lower acuity, while APACHE is not focused on respiratory illness. Our risk score is meant for use in ECMO planning rather than predicting outcomes among patients already receiving ECMO. Registry-based studies have also compared SARS-CoV-2 outcomes to that of other viral infections, including MERS, H1N1 flu, and seasonal flu. One MERS-related ARDS study of critically ill patients demonstrated higher mortality than those in studies on COVID-related ARDS, but may be attributed to sicker patients at enrollment [4]. A similar H1N1 study reported lower mortality (12-17%), albeit considering a younger population (average age 40) [3].

Physiologic concerns have also been raised about the use of ECMO for SARS-CoV-2. One argues that while ECMO is primarily beneficial for respiratory recovery, a spike in all-cause death but not ARDS-related death could indicate a limited role of ECMO[14]. Others point out that COVID-associated lymphopenia might be exacerbated by ECMO-induced lymphopenia which could mechanistically affect a healthy immune response to infection. Inflammatory cytokines and specifically interleukin 6 elevation is associated with COVID-19 mortality and rises with the use of ECMO [5, 13]. These expert voices do not argue for the avoidance of ECMO, but rather call for additional study.

2 Data

The eICU Collaborative Research Database [21] contains 200,859 admissions to intensive care units (ICU) across multiple centers in the United States between 2014 and 2015. The MIMIC-III clinical database [16] consists of data from 46,476 patients who stayed in critical care units of the Beth Israel Deaconess Medical Center between 2001 and 2012. Model development and in-domain validation primarily use data from eICU, and out-of-domain validation uses MIMIC-III.

Cohort Selection Inclusion criteria for the study cohort are delineated in Figure 1. The population of interest is among patients with viral or otherwise unspecified non-bacterial, non-fungal, non-parasitic, and non-genetic pneumonia. While there are no absolute contraindications of ECMO, the therapy is reserved for patients likely to have functional recovery. Patients over 70 years old would not be good candidates for ECMO, and SARS-CoV-2 pneumonia progressing to hypoxic respiratory failure is exceedingly rare in patients under 18. Other relative contraindications to ECMO are also listed in Figure 1. We select the first ICU stay within each patient's hospital stay, and exclude patients who died or were discharged within the first 48 hours of being admitted. This is done to focus on the stage of critical care after initial entry when lower-risk oxygen supplementation strategies (*e.g.*, ventilation) are being performed, and, methodologically, to provide a richer set of features for prediction. Table 1 and Appendix Table 4 summarize characteristics of the cohorts.

Data Extraction The study cohorts are extracted using string matching on diagnosis codes and subsequent clinician review. Features are merged through a process of visualization, query, and physician review. This includes harmonizing feature units, removing impossible values, and merging redundant data fields. Additional details are in Appendix B. All features are combined into a fixed-length vector, using the most recent value prior to 48 hours after ICU admission. Before imputation, approximately half of the features had missingness below 5%, and 80% of the features had missingness below 30%, however multiple variables had high missingness (Appendix B). Missing values are imputed using MissForest [25], which we find PEER is insensitive to (Appendix B).

Features Features are extracted from demographics, comorbidities, vitals, physical exams, and lab findings routinely collected in critical care settings. Numerical features are normalized, and categorical features are converted with dummy variables. All variables in Tables 1 and 4 are provided to the model.

Outcomes Our primary outcome of interest is in-ICU mortality. Secondary outcomes indicating decompensation are vasopressor use and mechanical ventilation use. For each outcome, we define the time to event as the time to first outcome or censorship, where censorship corresponds to discharge from the ICU.

3 Methods

Lasso-Cox To predict patient survival, we use the Cox proportional hazards model with L1 regularization, referred to as *Lasso-Cox* [24]. Lasso-Cox is chosen for its ease of interpretation and calculation, owing to its selection of sparse models.* For a patient with covariates $\mathbf{x} \in \mathbb{R}^d$, the predicted log hazard is $\beta^\top \mathbf{x}$, (higher hazard implies shorter survival time), where $\beta \in \mathbb{R}^d$ are coefficients that can be interpreted as log hazard ratios. L1 regularization $\lambda \sum_{j=1}^{d} |\beta_j|$ is used to encourage sparsity in β, where $\lambda > 0$ is a user-specified hyperparameter.

*We also tried the Cox model with elastic-net regularization (combined L1 and L2 regularization) but found little to no gain in cross-validation concordance.

Table 1: Demographics and outcomes of patients with viral or unspecified pneumonia in eICU and MIMIC-III cohorts. Data are median (Q1-Q3) or count (% out of n).

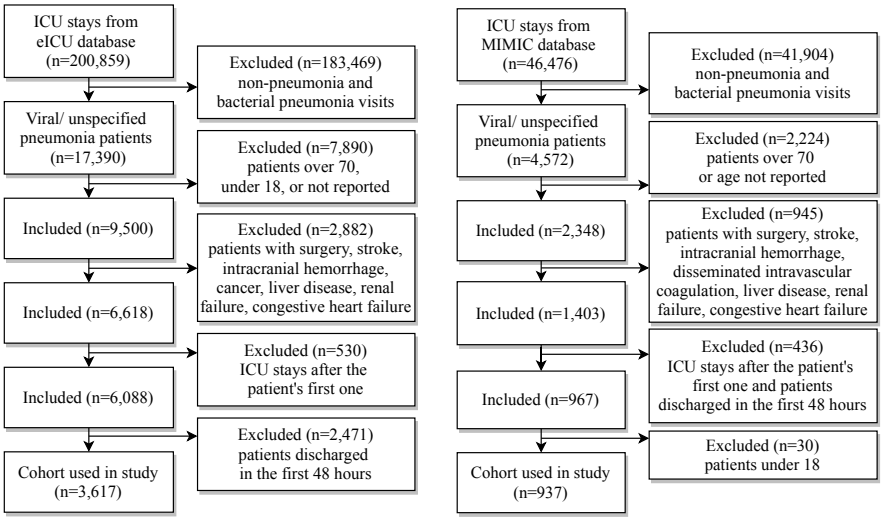
	Variable	eICU (n = 3617)	MIMIC (n = 937)
Demographics	Age, years	58.0 (48.0-64.0)	54.5 (44.1-62.7)
	18-30	225 (6.2%)	83 (8.9%)
	30-39	277 (7.7%)	94 (10.0%)
	40-49	500 (13.8%)	159 (17.0%)
	50-59	1064 (29.4%)	281 (30.0%)
	60-70	1546 (42.7%)	320 (34.2%)
	Male	1949 (53.9%)	542 (57.8%)
	Female	1663 (46.0%)	395 (42.2%)
Out.	Deceased	270 (7.5%)	94 (10.0%)
	Vasopressors administered	589 (16.3%)	389 (41.5%)
	Ventilator used	1835 (50.7%)	758 (80.9%)

Evaluation Metrics To evaluate model performance, we consider concordance and calibration. *Concordance* (c-index) is a common measure of goodness-of-fit in survival models [12], defined as the fraction of pairs of subjects whose survival times are correctly ordered by a prediction algorithm, among all pairs that can be ordered. Confidence intervals are computed using 1000 bootstrapped samples. We evaluate *calibration* by plotting the Kaplan-Meier observed survival probability versus the predicted survival probability. We construct our calibration plots (Figure 3) [29] with 1000 bootstrap resamplings for internal calibration. Both internal and external calibrations use 5 groups for 7 days.[†]

Experimental Setup The eICU cohort is divided into a training set (70% of the data, n=2537) and test set (30%, n=1080). The eICU training set is used for model development, whereas the eICU test set and entirety of the MIMIC cohort are used for model evaluation. Throughout our evaluation, we compare our risk score (PEER) to three pneumonia risk scores: CURB-65 [26], PSI/PORT [8], and SMART-COP [6]; and one COVID-19 risk score: GOQ [10].

Model selection We select λ via 10-fold cross validation and grid search on the eICU training set to maximize concordance subject to sufficient sparsity. We observe that $\lambda = 0.01$ gives the best trade-off between concordance (0.73) and number of features selected (18), as a 0.01 increase in concordance corresponds to 10 additional non-zero features. To check the stability of this hyperparameter choice, we impute our data using ten random seeds and run 10-fold cross validation on the resulting datasets. Across all runs, $\lambda = 0.01$ achieves concordance of approximately 0.73 and selects similar features and coefficients. Additional details about grid search, the concordance and sparsity tradeoff, and robust selection of coefficients can be found in Appendix B. Code for data extraction and all model results is available at https://github.com/hlzhou/peer-score.

[†]We plot at day 7 instead of 30 because censorship level is too high beyond a week.



(a) eICU cohort selection (b) MIMIC-III cohort selection

Fig. 1: Inclusion and exclusion criteria for cohorts extracted from eICU and MIMIC. Disseminated intravascular coagulation was highly missing from eICU.

4 Results

The hazard ratios from Lasso-Cox with $\lambda = 0.01$ are displayed in Table 2. For easy calculation of the PEER score, we also provide a nomogram (Figure 2)[‡].

The PEER score achieves concordance greater than or comparable to that of existing risk scores on all datasets (Table 3). On the eICU test set, PEER achieves the highest concordance among the risk scores, 0.77. On MIMIC, the maximum concordance degrades to 0.66, achieved by PEER and SMART-COP. The PEER calibration curves (Figure 3) show one high risk group separate from low risk groups. While predicted survival of the high risk group is overestimated in the training set, it is within confidence intervals in both test sets.

We define low and high risk subpopulations by thresholding our model's predicted risks on the training set at the 90th percentile. Each group's Kaplan-Meier survival curves are plotted over a 30-day period (Figure 4). For the first week, the low and high risk curves are clearly distinct (Figure 4), with respective survival proportions 0.68 and 0.95 on eICU test, and 0.75 and 0.95 on MIMIC. Beyond the first week, censorship grows quickly and there is less data, resulting in increased uncertainty. Compared to low and high risk curves derived from related risk scores, those of the PEER score are the most separated (Appendix B). Secondary indicators of decompensation (i.e. ventilator and vasopressor use) are also more common in the high risk group than the low risk group (Figure 5).

[‡]To compute risk, look up a patient's values in the nomogram, match it to points listed across the top, add them up, and look up the total in the scale across the bottom.

Table 2: Hazard ratios (HR) for the Lasso-Cox model, i.e. the PEER score. HR and 95% confidence intervals (CI) are reported on normalized data. Means and standard deviations used for scaling are included for reference.

Feature	**HR (95% CI)**	mean	std. dev.
Age (years)	1.22 (1.04 – 1.43)	54.5	12.5
Heart rate (beats per minute)	1.13 (0.984 – 1.3)	89.4	17.8
Systolic blood pressure (mmHg)	0.928 (0.755 – 1.14)	122	22
Diastolic blood pressure (mmHg)	0.996 (0.745 – 1.33)	67.7	15.1
Mean arterial pressure (mmHg)	0.926 (0.673 – 1.27)	83.7	17.9
Glasgow Coma Scale	0.93 (0.803 – 1.08)	11.3	3.26
White blood cells (thousands/μL)	0.984 (0.871 – 1.11)	12.9	8.91
Platelets (thousands/μL)	0.924 (0.79 – 1.08)	208	108
Red blood cell dist. width (%)	1.24 (1.08 – 1.43)	15.8	2.47
Neutrophils (%)	0.972 (0.853 – 1.11)	79.1	13
Blood urea nitrogen (mg/dL)	1.07 (0.937 – 1.23)	25.1	19.5
Aspartate aminotransferase (units/L)	1.12 (1.06 – 1.18)	143	774
Direct bilirubin (mg/L)	1.03 (0.935 – 1.13)	0.385	0.816
Albumin (g/dL)	0.954 (0.82 – 1.11)	2.65	0.636
Troponin (ng/mL)	1.06 (0.985 – 1.14)	1.07	3.85
Prothrombin time (sec)	1.05 (0.909 – 1.2)	16.6	6.75
pH	0.856 (0.75 – 0.977)	7.38	0.0713
Arterial oxygen saturation (mmHg)	0.787 (0.723 – 0.856)	95.8	4.12

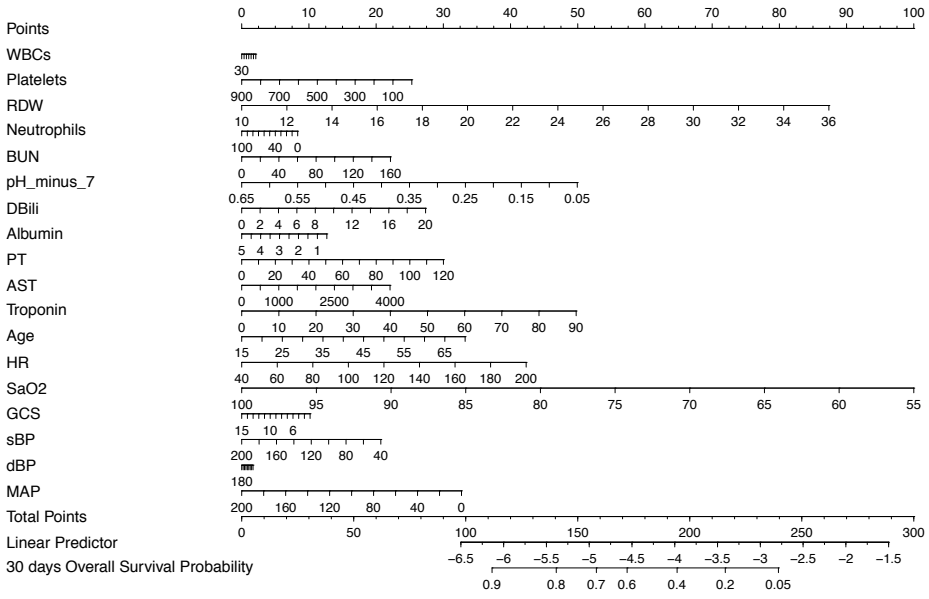
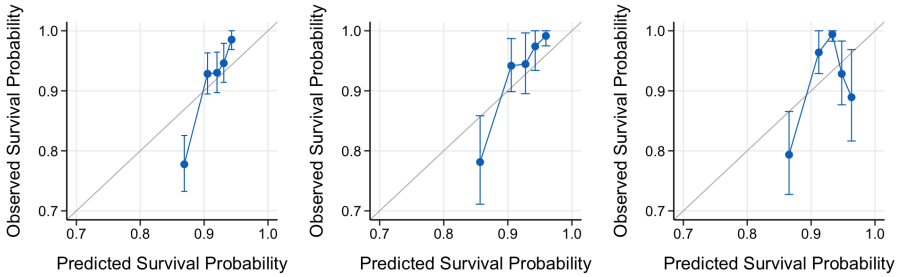


Fig. 2: Nomogram for manual calculation of the PEER score.

Table 3: Concordances (and 95% confidence intervals) of the PEER score, CURB-65, PSI/PORT, SMART-COP, and GOQ.

Score	Train eICU	Test eICU	MIMIC
PEER (ours)	**0.77 (0.72 - 0.81)**	**0.77 (0.69 - 0.83)**	**0.66 (0.57 - 0.74)**
CURB-65 [26]	0.66 (0.61 - 0.70)	0.62 (0.55 - 0.69)	0.59 (0.52 - 0.66)
PSI/PORT [8]	0.71 (0.66 - 0.76)	0.71 (0.63 - 0.78)	0.62 (0.55 - 0.69)
SMART-COP [6]	0.69 (0.64 - 0.73)	0.73 (0.67 - 0.80)	**0.66 (0.59 - 0.72)**
GOQ [10]	0.67 (0.63 - 0.71)	0.62 (0.54 - 0.70)	0.58 (0.50 - 0.66)



(a) Train eICU (b) Test eICU (c) MIMIC

Fig. 3: Calibration plots with 95% confidence intervals.

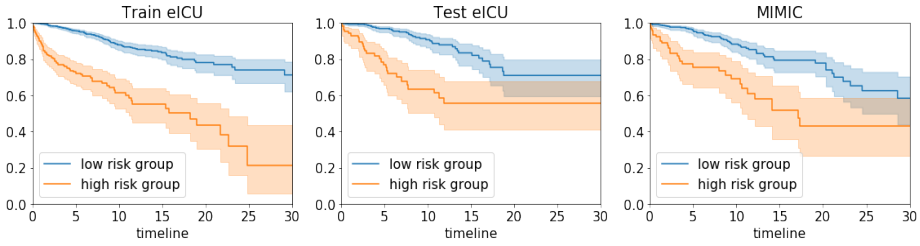
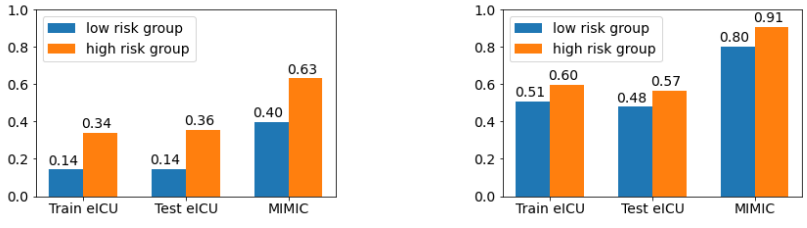


Fig. 4: Kaplan-Meier survival curves of high vs. low risk groups in train eICU, test eICU, and MIMIC. Shaded regions are the 95% confidence intervals.



(a) vasopressor (b) ventilator

Fig. 5: Proportion of each subgroup that received vasopressors or ventilators.

5 Discussion

The PEER score achieves greater or comparable concordance to baselines on the eICU (in-domain) and MIMIC (out-of-domain) test sets. Lasso-Cox selects 18 features, making for easy computation. Qualitatively, the score is consistent with clinical intuition. SaO2, associated with poorer oxygenation status, is predictive of decompensation. Old age is predictive of death. Red blood cell distribution width, associated with expanded release of immature red blood cells in response to insufficient oxygen delivery to tissues, is also a strong risk factor for death with COVID-19 [9]. However, the hazard ratios themselves should be interpreted with caution as three variables (pH, prothrombin time, and age) violate the proportional hazards assumption, and L1 regularization shrinks coefficients towards 0.

Stratifying each cohort into high and low risk subpopulations based the PEER score, we observe a clear separation in their survival curves (Figure 4) across all three datasets. Additionally, secondary indicators of decompensation (e.g. vasopressor and ventilator use) are more prevalent in the high risk group (Figure 5). Calibration plots for PEER also show a high risk group separated from the rest (Figure 3). While the survival probability of the high risk group is overestimated on the eICU training set, it is within error bars on all test sets.

For ECMO allocation, practically, accurate *ranking* of risk, as measured by concordance, may be more important than the precise probabilities predicted. The PEER score outperforms other risk scores on the eICU test set, but there is a decline in performance on the MIMIC test set, and the performance of PEER becomes comparable to that of SMART-COP. One possible reason for this decline is that in MIMIC, an important feature for PEER, the arterial oxygen saturation (SaO2), has 72.6% missingness. In contrast, it has 1.5% missingness in eICU. This demonstrates the importance of thinking critically about how our risk score, which was trained on the eICU cohort and depends on 18 specific features, generalizes to the population to which the score is being applied.

Limitations and Future Work Importantly our cohort is defined not by COVID-19 positive pneumonia patients but instead by viral or unspecified pneumonia patients who are ECMO-eligible. While our risk score demonstrates good discriminative ability and is interpretable, there are several additional decision-making considerations beyond the scope of this paper. Clinicians interested in applying the risk score to COVID-19 pneumonia should consider how representative this population is of their own. Because ECMO is a constrained resource, there are also ethical questions about who should get treatment. This risk score does not attempt to address these questions, but simply provides relevant information to those making such decisions. More broadly, we hope to provide this risk score as a potential resource for future SARS-like diseases that require ECMO consideration.

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A Summary Characteristics

Table 4: Summary characteristics per cohort, with median (Q1-Q3) or count (% of n).

		Variable	eICU (n = 3617)	MIMIC (n = 937)
Physical exam findings		Orientation		
		oriented	1121 (31.0%)	411 (43.9%)
		confused	1287 (35.6%)	76 (8.1%)
		Temperature (°C)	36.9 (36.6-37.3)	37.2 (36.6-37.7)
		Heart rate (beats per minute)	89.0 (77.0-101.0)	90.0 (78.0-104.0)
		Respiratory rate (breaths per minute)	20.0 (17.0-25.0)	20.0 (16.0-25.0)
		Systolic blood pressure (mmHg)	120.0 (106.0-136.0)	118.0 (104.0-134.0)
		Diastolic blood pressure (mmHg)	66.0 (57.0-76.0)	63.0 (54.0-72.0)
		Mean arterial pressure (mmHg)	81.0 (72.0-93.0)	79.0 (71.0-90.0)
		Glasgow Coma Scale	14.0 (10.0-15.0)	14.0 (9.0-15.0)
Laboratory findings (Abbrevations: Coagulation as Coag. and Blood Gas as B.G.)	Hemotology	Red blood cells (millions/μL)	3.5 (3.0-4.0)	3.4 (3.0-3.8)
		White blood cells (thousands/μL)	11.0 (7.9-15.6)	11.0 (8.0-15.1)
		Platelets (thousands/μL)	193.0 (136.0-261.0)	199.0 (128.8-276.0)
		Hematocrit (%)	31.1 (27.2-35.6)	30.2 (27.0-33.6)
		Red blood cell dist. width (%)	15.2 (14.0-16.8)	14.8 (13.8-16.4)
		Mean corpuscular volume (fL)	90.4 (86.0-95.0)	89.0 (85.0-93.0)
		Mean corpuscular hemoglobin/ MCH (pg)	29.7 (27.9-31.2)	30.2 (28.7-31.6)
		MCH concentration (g/dL)	32.7 (31.7-33.6)	33.8 (32.8-34.8)
		Neutrophils (%)	82.0 (73.3-89.0)	82.3 (73.8-88.5)
		Lymphocytes (%)	8.4 (5.0-14.0)	9.5 (5.8-15.7)
		Monocytes (%)	6.0 (3.7-8.6)	4.0 (2.7-5.9)
		Eosinophils (%)	0.1 (0.0-1.0)	0.4 (0.0-1.2)
		Basophils (%)	0.0 (0.0-0.3)	0.1 (0.0-0.3)
		Band cells (%)	8.0 (3.0-17.0)	0.0 (0.0-5.0)
	Chemistry	Sodium (mmol/L)	139.0 (136.0-142.0)	139.0 (136.0-142.0)
		Potassium (mmol/L)	3.9 (3.6-4.3)	3.9 (3.6-4.3)
		Chloride (mmol/L)	105.0 (101.0-109.0)	105.0 (101.0-109.0)
		Bicarbonate (mmol/L)	25.0 (22.0-28.0)	26.0 (23.0-29.0)
		Blood urea nitrogen (mg/dL)	19.0 (12.0-33.0)	17.0 (11.0-28.0)
		Creatinine (mg/dL)	0.8 (0.6-1.4)	0.8 (0.6-1.3)
		Glucose (mg/dL)	131.0 (105.0-165.0)	124.0 (104.5-151.5)
		Aspartate aminotransferase (units/L)	30.0 (19.0-57.0)	37.0 (22.0-70.0)
		Alanine aminotransferase (units/L)	27.0 (16.0-47.0)	28.0 (18.0-52.0)
		Alkaline phosphatase (units/L)	84.0 (62.0-117.0)	85.0 (62.0-121.0)
		Direct bilirubin (mg/L)	0.2 (0.1-0.5)	0.6 (0.2-2.2)
		Total bilirubin (mg/L)	0.5 (0.3-0.8)	0.6 (0.4-1.1)
		Total protein (g/dL)	6.0 (5.3-6.7)	6.1 (5.3-7.0)
		Calcium (mg/dL)	8.2 (7.7-8.6)	8.2 (7.8-8.6)
		Albumin (g/dL)	2.6 (2.2-3.1)	3.0 (2.6-3.5)
		Troponin (ng/mL)	0.1 (0.0-0.2)	0.0 (0.0-0.3)
	Coag.	Prothrombin time (sec)	14.5 (12.7-16.7)	13.9 (13.0-15.3)
		Partial thromboplastin time (sec)	33.0 (28.5-41.0)	30.2 (26.6-36.9)
	B.G.	pH	7.39 (7.33-7.43)	7.41 (7.36-7.45)
		Partial pressure of oxygen (mmHg)	83.0 (68.0-111.0)	97.0 (73.5-127.5)
		Arterial oxygen saturation (mmHg)	96.0 (94.0-99.0)	97.0 (95.0-98.0)

B Extended Version

Additional details can be found at https://arxiv.org/abs/2006.01898.