Title

Novel use of home pulse oximetry monitoring in COVID-19 patients discharged from the emergency department identifies need for hospitalization

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ABSTRACT

Objectives

Our objective was to evaluate patient-reported oxygen saturation (SpO_2) using pulse oximetry as a home monitoring tool for patients with initially non-severe COVID-19 to identify need for hospitalization.

Methods

Patients were enrolled at the emergency department (ED) and outpatient testing centers. Each patient was given a home pulse oximeter and instructed to record their SpO_2 every eight hours. Patients were instructed to return to the ED for sustained home SpO_2 <92% or if they felt they needed emergent medical attention. Relative risk was used to assess the relation between hospitalization and home SpO_2 <92% in COVID-19 positive patients.

Results

We enrolled 209 patients with suspected COVID-19, of which 77 patients tested positive for COVID-19 and were included. Subsequent hospitalization occurred in 22/77 (29%) patients. Resting home SpO_2 <92% was associated with an increased likelihood of hospitalization compared to SpO_2 >92% [RR 7.0 (95% CI 3.4 – 14.5), p-value <0.0001]. Home SpO2 <92% was also associated with increased risk of ICU admission, ARDS and septic shock. In our cohort, 50% of patients who ended up hospitalized only returned to the ED for incidental finding of low home SpO_2 without worsening of symptoms. One-third (33%) of non-hospitalized patients stated they would have returned to the ED if they did not have a pulse oximeter to reassure them at home.

Conclusions

This study found that home pulse oximetry monitoring identifies need for hospitalization in initially non-severe COVID-19 patients when a cut off of SpO_2 92% is used. Half of patients who ended up hospitalized had SpO_2 <92% without worsening symptoms. Home SpO_2 monitoring also reduces unnecessary ED revisits.

INTRODUCTION

Background

In December 2019, a novel coronavirus called SARS-CoV-2 appeared in Wuhan city, Hubei Province, China and rapidly spread across the rest of the world. This virus causes a disease known as COVID-19. Most patients with this infection recover after experiencing mild flu-like symptoms, but 20% of patients clinically deteriorate, requiring hospitalization and critical care[1]. This deterioration can be quite rapid at times, resulting in patients requiring intubation and other advanced life support measures before or at arrival to the hospital.

One of the challenges of the COVID-19 pandemic in the United States is the strain it is placing on healthcare resources. Drastic measures have been taken to rapidly increase healthcare resources and reallocate healthcare workers to meet the needs during the pandemic. Given the severity of the ongoing global pandemic, the ability to remotely monitor patients who do not require hospitalization is essential for optimal utilization of healthcare resources.

Importance

A reasonable concern brought forward by emergency medicine physicians discharging initially non-severe patients with COVID-19 is that these patients could potentially decompensate at home after discharge. Home pulse oximetry has been proposed as a way to monitor disease progression in such patients. However, there are currently no data to guide the use of home pulse oximetry in COVID-19 patients or its validity in identifying disease progression.

Additionally, while it is generally known that patients with advanced age, comorbidities or certain laboratory findings are at increased risk for worse clinical outcomes, specific predictors for who will require hospitalization are not known at this time[2, 3].

Goal of Investigation

Our objective was to evaluate patient-reported oxygen saturation using pulse oximetry as a home monitoring tool for patients with initially non-severe COVID-19 to identify need for hospitalization.

METHODS

Study design and setting

This prospective, uncontrolled open-label study took place at Swedish Hospital, part of NorthShore University HealthSystem in Chicago, IL between March 20 and April 22, 2020. The institutional review board approved the study and all patients consented to participate in the study. This study was registered with ClinicalTrials.gov (NCT04373161).

Study population

All patients were older than 18 years of age. Patients were enrolled if they had suspected COVID-19 as defined by the World Health Organization (WHO)[1]. Testing for COVID-19 was performed using reverse-transcriptase polymerase-chain-reaction (RT-PCR) of an oropharyngeal or nasopharyngeal swab. Patient testing locations included the emergency department (ED) or Swedish Hospital affiliated testing centers, including outpatient and employee testing sites for symptomatic individuals. For patients seen in the ED, only those being discharged to home were included. All patients had resting SpO_2 >92% on discharge from the ED. Patients being admitted to the hospital or discharged to a nursing facility were excluded. Other exclusion criteria included pregnancy and home oxygen use. Patients were not included if they were unable to be reached after enrollment.

Not all patients with suspected COVID-19 were tested due to ongoing test kit shortages during the time of this study. Only patients with positive COVID-19 testing were included in our outcome measures and analysis. Patients with suspected COVID-19 who did not undergo initial testing were still enrolled in case they were tested at a later time. ED physicians were not blinded to potential patient enrollment, but they were not specifically made aware of which patients were being enrolled into the study, or if patients were already enrolled upon return to the ED.

Study protocol

Upon discharge to home from the ED or testing site, patients were provided with an FDA approved Concord Health Supply EAD Fingertip Pulse Oximeter ^{TM} (Skokie, IL, USA) at no

cost to the patient. Patients had their resting oxygen saturation (SpO_2) checked using this pulse oximeter at time of enrollment and this measurement was recorded as Day 0. For seven days, patients checked their SpO_2 using the pulse oximeter at 6:00 AM, 2:00 PM, and 10:00 PM. Seven day follow up was selected given the duration from symptom onset to hospitalization has been reported as 4, interquartile ratio (IQR) 2-7 days[2]. Investigators on the research study team called patients daily to collect data in real time.

In the study protocol provided to patients, they were instructed to return to the ED if: 1) their resting SpO_2 dropped below 92% and was confirmed with a separate reading ten minutes later or 2) they felt they needed emergent medical attention. During these calls, patients were also surveyed on whether use of home pulse oximetry prevented further ED visits. The standardized script used for patient calls is available as supplemental methods material accompanying the online article. After the home pulse oximeter-monitoring period, patients returned the pulse oximeter along with a standardized form detailing their measurements.

The decision to hospitalize on subsequent return to the ED was left to the discretion of the ED physician evaluating the patient, independent of this study.

Measurements

Patients' charts were reviewed to identify prior medical problems, laboratory values on preliminary ED visit, laboratory values on subsequent return to the ED or hospitalization and outcomes of hospitalization. Obesity was defined as body mass index (BMI) > 30 kg/m^2 and lymphopenia was defined as lymphocyte count <1,500 cells/μL.

Outcomes

The primary outcome was hospitalization in patients with resting home SpO_2 below 92%. Other outcomes measured included trend in resting home pulse oximetry readings, timing of SpO_2 <92%, if home pulse oximeter use decreased subsequent ED visits, and outcomes of hospitalization such as length of stay and transfer to the intensive care unit (ICU). We also measured time to drop (TTD), defined as time from symptom onset to SpO_2 <92%, to see if this predicted admission to the ICU, development of acute respiratory distress syndrome (ARDS),

septic shock or mortality. Finally, we collected data on demographics, past medical history and laboratory values.

Statistical Analysis

The relative risk (RR) of hospitalization for COVID-19 positive patients with resting home SpO_2 below 92% was calculated, with p-value and associated 95% confidence interval determined using the Wald method. An a priori power analysis indicated a sample size of 76 to provide 80% power to detect a relative risk of 2.75 between hospitalizations and resting home SpO_2 below 92%. Differences in SpO_2 trends by time of day were compared with a linear mixed effects model with an unstructured covariance matrix. The covariates considered were time of day and hospitalizations with a patient-specific intercept specified as a random effect. Differences between lab values for patients with both initial visit measurements and measurements at hospitalization were analyzed with a Wilcoxon signed rank test. We ran univariate logistic regression to identify predictors of ICU admission, development of ARDS, septic shock or mortality. We considered running multivariate analysis but given the small sample size of our study, this was not considered to be statistically relevant and was not included. Statistical significance was set at the 0.05 level and analysis was performed using R version 3.6.2.

RESULTS

Characteristics of study subjects

A total of 209 patients with suspected COVID-19 were enrolled in our study. Of patients enrolled, 119 (57%) underwent RT-PCR testing and 79 (38%) tested positive for COVID-19. Patients who tested negative, withdrew consent or were unable to be contacted after enrollment were excluded. A total of 77 COVID-19 positive patients were ultimately included and analyzed in our study (Figure 1). Of these 77 patients, 9 patients were not initially tested on enrollment but tested positive at a subsequent ED visit. Enrollment locations included 61 (79%) patients enrolled from the emergency department, 9 (12%) from employee testing, and 7 (9%) from the outpatient testing center.

Demographic and baseline characteristics in COVID-19 positive patients are summarized in Table 1. Median age was 44 (IQR 19), 43 (56%) were male and median BMI was 29.7 (IQR 7.9) mg/kg^2. Patients were Hispanic (57%), Asian (27%), African American (8%), and Caucasian (8%). In our cohort, 20 (26%) were healthcare workers. There were 32 (42%) patients with no medical problems, 20 (25%) with one medical comorbidity, 11 (14%) with two comorbidities, and 14 (18%) with three or more comorbidities. The most common medical comorbidities were obesity (27%), hypertension (26%), diabetes (16%), hyperlipidemia (13%) and asthma (9%). There were 10 (13%) patients on ACE inhibitor or angiotensin II receptor blockers.

Baseline laboratory values in patients at time of enrollment and subsequent laboratory values for hospitalized patients are summarized in Table 2. Patients had lymphopenia, elevated lactate dehydrogenase, C-reactive protein, liver enzymes, ferritin and d-dimer on initial visit to the ED and upon hospitalization. Not all patients had laboratory studies drawn on enrollment as the decision to do so was left to the evaluating provider independent of this study. Laboratory values on day of admission to the hospital were not available for six patients as they were hospitalized at other institutions.

Main Results

There were 19/77 patients (25%) with home SpO_2 <92%. Of these, 17 came back to the ED and 16 were hospitalized. Remarkably, 8 of these 16 patients (50%) only returned to the ED for incidental finding of low home SpO_2 without worsening symptoms. The single patient with SpO2 <92% who returned to the ED and was not hospitalized had an oxygen saturation of 94% in the ED and was discharged to home. Of the 58 patients who maintained SpO_2 >92%, 11 (19%) returned to the ED, where 5 patients were discharged and 6 patients were hospitalized (Figure 2). Resting home SpO_2 <92% was strongly associated with hospitalization compared to home SpO_2 >92% [RR 7.0 (95% CI 3.4 – 14.5), p-value <0.0001] (Figure 3).

Symptoms were present for a median of 5 (IQR 4) days prior to enrollment and 6 (IQR 2) days prior to hospitalization. The median length of stay for hospitalization was 8 (IQR 6) days. Of hospitalized patients, 8 (36%) were transferred to the ICU. Within the ICU cohort, 6/8 (75%) patients had home SpO_2 <92% and 2/8 (25%) had home SpO_2 >92%. Of this ICU cohort, 4/8 (50%) only came to the ED for incidental finding of low home pulse oximetry readings. Both patients within the ICU cohort with home SpO2 >92% had downtrending oxygen saturation with last reported reading of 93% prior to hospitalization. While in the ICU, 7 patients developed ARDS requiring mechanical ventilation and 6 patients developed septic shock requiring vasopressors. There were two patients who died in the ICU. Resting home SpO2 <92% was associated with increased risk of ICU admission [RR 9.8 (95% CI 2.2 – 44.6) p<0.002], ARDS [RR 8.2 (95% CI 1.7 – 38.7,) p<0.007] and septic shock [RR 6.6 (95% CI 1.3 – 32.9), p=0.02]. Resting home SpO_2 <92% was not associated with increased mortality (p=0.5). There were 5 (23%) patients still hospitalized at the time of data censoring.

There was no specific time of day that had higher likelihood of SpO_2 <92% (p=0.09). Presented in Figure 4 are longitudinal home pulse oximetry readings in patients who ended up hospitalized and patients who were not hospitalized. All hospitalizations occurred within 5 days of enrollment. The median time to drop (TTD) was 6 (IQR 2) days. TTD was not associated with ICU admission (p=0.3), ARDS (p=0.5), septic shock (p=0.7) or mortality (p=0.7).

Trending laboratory values in patients who ended up hospitalized demonstrated significant increase in lactate dehydrogenase (p=0.03) from initial ED visit to return ED visit for

hospitalization. See Table 3. Of COVID-19 positive patients who did not return to the ED, 16/49 (33%) stated they would have returned to the ED if they did not have the pulse oximeter to reassure them at home.

Univariate logistic regression found that lower initial pulse oximetry reading was associated with increased odds of hospitalization [OR 1.7 (95% CI 1.2 – 2.4), p<0.004)]. See Table 4. Although lower platelet count [OR 0.98 (95% CI 0.96 – 0.99), p=0.03] and lower albumin levels [OR 0.5 (95% CI 0.26 - 0.83), p=0.03] were associated with hospitalization, the median levels were within the normal range. Asthma [OR 9.5, 95% CI (1.53 – 56.8), p=0.01] and albumin [OR 0.6, (95% CI 0.35 – 0.91), p=0.03] were associated with a composite outcome of ICU admission, ARDS and septic shock (Table 5).

Demographic data and prior medical history in patients with suspected COVID-19 who did not undergo testing are summarized in Supplemental Table S1. Initial laboratory values on enrollment in this cohort is summarized in Supplemental Table S2. Longitudinal home pulse oximetry readings in these patients are presented in Supplemental Figure S1.

DISCUSSION

In this study, we assessed the utility of home pulse oximetry monitoring in patients with initially non-severe COVID-19. Our study was designed to be a practical approach to monitor suspected and confirmed COVID-19 patients remotely and reduce in-person healthcare utilization. Our results found that pulse oximetry as a home monitoring tool identifies need for hospitalization in initially non-severe COVID-19 patients when a cut off of SpO_2 92% is used.

We selected SpO_2 <92%, a measure of peripheral oxygen saturation, because this indicates the presence of hypoxemia, a measure of oxygen pressure in arterial blood (PaO_2). A recent multicenter, prospective study found SpO_2 <92% had 95% sensitivity and 90% specificity for detecting PaO_2 <60 mmHg[3]. PaO_2 less than 60 mmHg defines hypoxemic respiratory failure[4]. On the oxygen-dissociation curve, there is a steep drop in oxygen saturation as PaO_2 approaches 60 mmHg known as the "slippery slope". Below this level, small reductions in PaO_2 correlate with disproportionately large reductions in oxygen saturation and thereby oxygen delivery[5]. In a cohort study of 2,923 patients seen in the ED with pneumonia, hospitalizing patients for SpO_2 <92% was associated with improved mortality compared to hospitalizing patients with SpO_2 <90%[6]. This data supports an intervention using SpO_2 <92% as the cutoff to identify patients who may clinically deteriorate.

Over half of hospitalized patients in our cohort presented to the ED due to an incidental finding of low home SpO_2 without change in symptoms. A similar pattern has emerged recently whereby hypoxemia precedes severe symptoms in some patients with COVID-19, termed "silent hypoxemia"[7]. Pathophysiology to explain this phenomenon is still being debated. Histologic evaluation on autopsy in a COVID-19 positive patient demonstrated diffuse alveolar damage, pulmonary edema, lymphocytic inflammatory infiltrate and hyaline membrane formation, consistent with ARDS[8]. A recent publication suggests that while ARDS is present in COVID-19, there appears to be heterogeneity in clinical presentation suggesting two disease phenotypes. They propose a varying combination of increasing inflammation and edema from patient self-inflicted lung injury related to increased negative intra-thoracic pressure against the otherwise compliant lung[9]. The use of supplemental oxygen improves hypoxemia and decreases work of

breathing, which may reduce the risk of lung injury. It is plausible that outcomes could be improved with early intervention. Based on our findings, home pulse oximetry may identify these "silent hypoxemia" patients in the outpatient setting prior to onset of severe symptoms and respiratory failure. A randomized controlled trial of pulse oximetry in the patient population that we studied will be required to test that hypothesis.

In our cohort, most patients who had SpO_2 <92% experienced an abrupt drop in SpO_2 rather than a gradual decline. This is consistent with emerging findings of certain patients rapidly deteriorating within a matter of hours[10]. The underlying physiology for this sudden change in clinical status is attributed to a surge in pro-inflammatory molecules including IL-1β, IL-6, CCL-2, CCL-3, CCL-5, TNF, and has been termed the "cytokine storm" phase of COVID-19[11]. It is plausible that "cytokine storm" contributes to this drop in SpO_2.

Lactate dehydrogenase (LDH) increased in patients who had labs drawn on enrollment and then were subsequently hospitalized after returning to the ED. Our findings are concordant with recent data demonstrating elevated LDH as a predictor of more severe-COVID-19 disease [12]. This laboratory value could be useful in assessing disease progression in COVID-19 patients who return to the ED. While platelet and albumin were inversely associated with odds of hospitalization, the median levels were within the normal ranges, so these findings may not be clinically relevant.

While recent literature suggests a low prevalence of asthma in patients with severe COVID-19, we found asthma to be associated with ICU admission, ARDS and septic shock in our cohort[12,13]. There are proposed mechanisms to account for a potential increased risk of severe disease in some patients with asthma including increased expression of angiotensin converting enzyme 2 and transmembrane protease serine 2. Further investigation into the outcomes of asthma patients with COVID-19 will be needed to better risk stratify these patients.

Our patient cohort differs in several characteristics compared to other published studies. Most studies evaluate the hospitalized COVID-19 population, which is comprised of patients who are more likely to be older and have more comorbid disease[14,15]. In contrast, our patient population

was younger and almost half had no chronic medical problems. Additionally, while our hospital serves a community that is 72% non-Hispanic, our hospitalized cohort was predominantly Hispanic. Despite this, Hispanic ethnicity did not emerge as a factor associated with hospitalization in our univariate analysis. It is unknown if our findings will translate similarly to other patient populations.

This intervention was also successful in reassuring patients who may not require hospitalization, which in turn reduces ED utilization. This finding has two important benefits. Reducing ED utilization may reduce the risk of exposure to COVID-19 in healthcare workers in the emergency department. Additionally, this intervention may reduce unnecessary personal protective equipment (PPE) use. Globally, there is a PPE shortage including medical masks, respirators, gloves, gowns and eye protection. The WHO has released guidelines that call for minimizing the need for PPE in healthcare settings given the global shortage[16]. Our study found that providing home pulse oximeters to those with suspected or confirmed COVID-19 made patients feel more comfortable not returning to the ED as long as their oxygen saturation remained appropriate.

Home pulse oximetry is made less accurate by nail polish, severe anemia, hyperbilirubinemia, hemoglobinopathies, or poor peripheral perfusion from severe vasoconstriction or poor cardiac output[17]. While none of these conditions were present in our patients, it is important to note if applying to a larger patient population.

Limitations

Given that one-quarter of our COVID-19 positive patients were healthcare workers, it is possible our cohort was easier to train in using the home pulse oximeter and had better follow up than the general population. Two patients withdrew from the study due to difficulty understanding how to use the pulse oximeter. Some patients could not be reached after enrollment. These occurrences emphasize the importance of patient selection and patient education when utilizing this intervention.

We standardized the home pulse oximeter used in our study to avoid variability between different brands. If multiple brands of pulse oximeters are used, the findings could be more

heterogeneous with variability between home pulse oximeter readings. In a study of three different commercially available pulse oximeters, good correlation was observed for each of the finger pulse oximeters when compared to arterial blood gas samples in 94 patients[18]. However, agreement may vary from device to device.

Patients were called once per day to collect data in real time. It is possible these patient callbacks highlighted the importance of SpO_2 below 92%, which may have increased likelihood of patients returning to the ED. The use of home pulse oximetry monitoring may perform better when paired with some form of telemedicine.

Given the need to censor data in order to be shared, outcomes of patients may underrepresent ICU status, ARDS, septic shock or mortality. Hospital length of stay is likely skewed lower as five patients remained hospitalized at time of data censoring. Additionally, our study is a small sample size, and larger scale studies need to be conducted to further investigate the utilization of home pulse oximetry monitoring to identify robust predictors of hospitalization. Such future studies should consider using known risk factors for poor outcomes in COVID-19 including age, gender, pre-existing hypertension, diabetes, chronic lung disease, cardiovascular disease, low albumin, elevated CRP and lymphopenia[19-22]. Finally, we opted to exclude patients who tested negative for COVID-19, however it should be noted that there is a significant false negative rate with the current iteration of the RT-PCR test[23]. There may be some utility to providing pulse oximeters to patients with high index of suspicion for COVID-19 who test negative, however we did not investigate this.

Conclusions

This study found that home pulse oximetry monitoring identifies need for hospitalization in initially non-severe COVID-19 patients when resting home oxygen saturation drops below 92%. Half of patients who ended up hospitalized had SpO_2 <92% without worsening symptoms. Home pulse oximetry monitoring reduces ED utilization, which in turn reduces exposure risk to frontline healthcare workers and conserves PPE.

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Supplemental Information linked to the online version of the paper at Wiley-Blackwell:

- Methods Supplement
- Table S1, S2
- Figure S1

Table 1: Patient characteristics in COVID-19 positive patients*

Variable	All Patients N = 77	Hospitalized Patients n = 22
Median age (IQR) — yr	44 (19)	49 (19)
Male sex — no. (%)	43 (56%)	16 (73%)
Ethnicity— no. (%)†		
Hispanic	44 (57%)	16 (73%)
Asian	21(27%)	5 (23%)
Caucasian	6 (8%)	1 (5%)
African American	6 (8%)	0 (0%)
Healthcare worker§	20 (26%)	4 (18%)
Median BMI (IQR) —%	29.7 (7.9)	30.1 (7.8)
Obesity — no. (%)¶	21 (27%)	9 (43%)
Hypertension— no. (%)	20 (26%)	6 (27%)
Diabetes mellitus— no. (%)	12 (16%)	5 (23%)
Hyperlipidemia— no. (%)	10 (13%)	4 (18%)
ACEI or ARB use — no. (%)**	10 (13%)	4 (18%)
Asthma— no. (%)	7 (9%)	3 (14%)
Deep venous thromboembolism/ Pulmonary embolism— no. (%)	3 (4%)	2 (9%)
Coronary artery disease— no. (%)	3 (4%)	2 (9%)
Human Immunodeficiency Virus— no. (%)	3 (4%)	1 (5%)
Chronic kidney disease— no. (%)	2 (7%)	1 (5%)

Chronic obstructive pulmonary disease— no. (%)	2 (7%)	0 (0%)
Heart Failure— no. (%)	2 (7%)	1 (5%)
Autoimmune Disease— no. (%)	1 (1%)	1 (5%)
History of malignancy— no. (%)	1 (3%)	1 (5%)
Hepatitis B virus— no. (%)	1 (1%)	1 (5%)
Other— no. (%)[† †]	0 (0%)	0 (0%)

[*]The above characteristics are based upon self-reported information and chart review of all patients who underwent confirmatory testing for COVID-19 represented by either IQR (interquartile range) or nominal value

† Ethnicity determined by patient or family member report

§ Healthcare worker status determined by patient report

¶ Obesity determined by BMI ≥ 30 mg/kg^2

** ACEI = angiotensin-converting-enzyme inhibitor, ARB = angiotensin II receptor blocker

† † Other comorbidities include cerebrovascular accident, cirrhosis, active malignancy, hepatitis C virus

Table 2: Laboratory values in COVID-19 positive patients*

Laboratory Variable	**Initial Visit (n = 28)**	**Day of hospital admission (n = 16)**
Hemoglobin (g/dl)	14.1 ± 1.8	13.9 ± 1.8
White cell count (per μl)	6.6 ± 2.8	6.4 ± 2.1
Lymphocyte count (cells/μl)	1,226 ± 562	1,206 ± 764
Neutrophil count (cells/μl)	4,754 ± 2,844	4,860 ± 2,004
Platelet Count (per μl)	226,000 ± 61,000	229,000 ± 77,000
Blood urea nitrogen (mg/dl)	14 ± 11	18 ± 21
Creatinine (mg/dl)	1.3 ± 2.0	2.0 ± 3.0
Albumin (g/dl)	4.5 ± 0.3	4.3 ± 0.3
AST (U/liter)	40 ± 21	73 ± 79
ALT (U/liter)	47 ± 31	75 ± 56
Total Bilirubin (mg/dl)	0.6 ± 0.4	0.6 ± 0.2
C-Reactive Protein (mg/liter)	70 ± 78	103 ± 81
Lactate Dehydrogenase (U/liter)	267± 68	430 ± 200

Ferritin (ng/ml)	516 ± 323	1,097 ± 1,273
Creatine kinase (U/liter)	164 ± 135	174 ± 134
Troponin (ng/dl)	<0.03 ± 0	0.04 ± 0.1
D-Dimer (mcg/ml)	0.3 ± 0.3	1.0 ± 0.9
Procalcitonin (n/ml)	0.2 ± 0.6	0.4 ± 1.0

*Plus-minus values are means ±SD. Laboratory values not available on all patients on initial visit due to enrollment in non-emergency department locations, or due to no laboratory studies ordered by emergency department provider. Laboratory values not available on all patients on day of admission to the hospital if they were hospitalized at another institution.

Table 3: A direct comparison of laboratory values on enrollment to laboratory values on day of subsequent hospitalization in COVID-19 patients *

Laboratory Variable	On enrollment (n=11)	Hospitalization (n=11)	Paired p-value
White cell count (per µl)	5.8 (2.5)	5.7 (2.3)	0.742
Lymphocyte count (cells/µl)	875 (296)	718 (488)	0.547
Neutrophil count (cells/µl)	4,333 (2457)	4,387 (1964)	0.641
Hemoglobin (g/dl)	13.9 (3.2)	13.7 (4.0)	0.310
Platelet count (per µl)	284,000 (153,000)	196,000 (78,000)	0.233
Blood urea nitrogen (mg/dl)	12 (9)	12 (7)	0.999
Creatinine (mg/dl)	0.8 (0.3)	0.8 (0.3)	0.999
AST(U/liter)	46 (24)	41 (22)	0.400
ALT (U/liter)	53 (39)	57 (46)	0.674
Total bilirubin (mg/dl)	0.7 (0.3)	0.8 (0.3)	0.462
Albumin (g/dl)	4.4 (0.2)	4.1 (0.4)	0.075
C-reactive protein (mg/liter)	30 (14)	63 (39)	0.125
Lactate dehydrogenase (U/liter)	291 (97)	379 (72)	**0.031**
Creatine kinase (U/liter)	103 (40)	117 (49)	0.313
D-dimer (mcg/ml)	0.3 (0.2)	0.3 (0.1)	0.999
Procalcitonin (n/ml)	0.2 (0.8)	0.2 (1.1)	0.625

*Plus-minus values are median (IQR). Laboratory values not available on all patients on initial visit due to enrollment in non-emergency department locations, or due to no laboratory studies ordered by emergency department provider. Laboratory values not available on all patients on day of admission to the hospital if they were hospitalized at another institution. Hence data is available for 11 out of 22 patients who ended up hospitalized.

Table 4: Univariate logic regression of factors associated with hospitalization in COVID-19 patients

Variable	OR (95% CI)	P-value
Age	1.0 (0.99 – 1.08)	0.084
Male sex	2.8 (0.98 – 8.68)	0.064
Body mass index	1.1 (0.95 – 1.23)	0.2420
Lower SpO_2 at enrollment	1.7 (1.20 – 2.40)	**0.004**
Ethnicity*		
Hispanic	2.6 (0.91 – 8.07)	0.086
Asian	0.7 (0.21 – 2.18)	0.572
Healthcare worker†	0.5 (0.14 – 1.73)	0.328
Hypertension	1.1 (0.33 – 3.20)	0.90
Hyperlipidemia	1.8 (0.43 – 7.27)	0.385
Obesity§	1.5 (0.49 – 4.58)	0.473
Diabetes	1.9 (0.51 – 6.90)	0.311
Asthma	1.9 (0.35 – 9.58)	0.415
ACEI or ARB use¶	1.7 (0.41 – 6.83)	0.430
White blood cell count	0.9 (0.58 – 1.17)	0.423
Lymphocyte count	1.0 (0.996 – 1.001)	0.066
Lymphopenia	8.9 (1.27 – 182.2)	0.058
Neutrophil count	1.0 (0.996 – 1.00)	0.958
Hemoglobin	1.0 (0.65 – 1.54)	0.992
Platelet count	0.98 (0.96 – 0.99)	**0.032**
Blood urea nitrogen	1.1 (0.99 – 1.32)	0.312
Creatinine	1.4 (0.85 – 2.26)	0.449
AST	1.0 (0.99 – 1.08)	0.149
ALT	1.0 (0.99 – 1.04)	0.255
Total bilirubin	0.8 (0.08 – 6.78)	0.839
Albumin	0.5 (0.26 - 0.83)	**0.029**
C-reactive protein	1.0 (0.98 – 1.01)	0.362
Lactate dehydrogenase	1.0 (0.98 – 1.01)	0.779
Ferritin	1.0 (099 – 1.02)	0.579
Creatinine kinase	1.0 (0.92 – 1.01)	0.104
D-dimer	0.007 (0 – 1.76)	0.191

* Ethnicity determined by patient or family member report

† Healthcare worker status determined by patient report

§ Obesity determined by BMI ≥ 30 mg/kg^2

¶ ACEI = angiotensin-converting-enzyme inhibitor – ARB = angiotensin II receptor blocker

Table 5: Univariate logic regression of factors associated with composite outcome of ICU admission, ARDS and septic shock in COVID-19 patients*

	All COVID-19 patients (N = 77)		COVID-19 patients who were hospitalized (n = 22)	
Variable	**OR (95% CI)**	**p-value**	**OR (95% CI)**	**p-value**
Age	1.036 (0.98 – 1.10)	0.200	1.014 (0.95 – 1.09)	0.676
Male Gender	1.360 (0.31 – 7.05)	0.689	0.455 (0.06 – 3.22)	0.420
BMI	1.028 (0.86 – 1.22)	0.753	0.971 (0.77 – 1.18)	0.775
SpO_2 at enrollment§	0.771 (0.50 – 1.18)	0.222	1.185 (0.68 – 2.17)	0.553
Home SpO2 <92%	14.25 (2.90 – 105.8)	**0.002**	1.667 (0.26 – 14.42)	0.605
Hypertension	0.926 (0.13 – 4.45)	0.929	0.833 (0.09 – 5.78)	0.857
Hyperlipidemia	1.074 (0.05 – 7.39)	0.950	0.611 (0.03 – 6.09)	0.696
Obesity	1.88 (0.40 – 8.89)	0.410	1.600 (0.27 – 10.01)	0.605
Diabetes	1.900 (0.25 – 9.71)	0.469	1.222 (0.13 – 9.56)	0.848
Asthma	9.450 (1.53 – 56.79)	**0.012**	3.24 (0.0 – 35.0)	0.995
ACEI or ARB use**	0.921 (0.05 – 6.12)	0.942	0.52 (0.02 – 5.09)	0.605
White blood cell count	0.748 (0.32 – 1.18)	0.373	0.439 (0.09 – 1.34)	0.201
Lymphocyte count	0.998 (0.996 – 1.00)	0.201	0.999 (0.995 – 1.002)	0.732
Neutrophil count	0.999 (0.993 – 1.002)	0.628	0.999 (0.997 – 1.001)	0.220
Hemoglobin	0.621 (0.31 – 1.08)	0.118	0.524 (0.19 – 1.03)	0.112
Platelet count	0.999 (0.99 – 1.00)	0.327	1.00 (0.99 – 1.01)	0.678
Blood urea nitrogen	1.10 (0.99 – 1.40)	0.288	1.071 (0.97 – 1.42)	0.455
AST	1.036 (0.99 – 1.09)	0.115	1.027 (0.97 – 1.11)	0.373
ALT	1.008 (0.98 – 1.04)	0.590	0.996 (0.95 – 1.04)	0.860
Total bilirubin	0.959 (0.04 – 11.76)	0.976	1.401 (0.01 – 173.4)	0.884
Albumin	0.0098 (0.0002 – 0.38)	**0.042**	0.070 (0.0001 – 4.36)	0.266
C-reactive protein	0.988 (0.95 – 1.01)	0.346	0.987 (0.94 – 1.01)	0.432
Lactate dehydrogenase	0.997 (0.98 – 1.01)	0.697	0.997 (0.98 – 1.01)	0.697
Creatinine kinase	0.974 (0.93 – 1.01)	0.183	0.986 (093 – 1.04)	0.576
D-dimer	0.045 (0 – 9.46)	0.368	2.539 (0.73 – 14.64)	0.202

* Laboratory values not available on all patients on initial visit due to enrollment in non-emergency department locations, or due to no laboratory studies ordered by emergency department provider. Laboratory values not available on all patients on day of admission to the hospital if they were hospitalized at another institution. Hence data is available for 11 out of 22 patients who ended up hospitalized.

§ SpO_2 = home pulse oximeter oxygen saturation

¶ Obesity determined by BMI ≥ 30 mg/kg^2

** ACEI = angiotensin-converting-enzyme inhibitor, ARB = angiotensin II receptor blocker

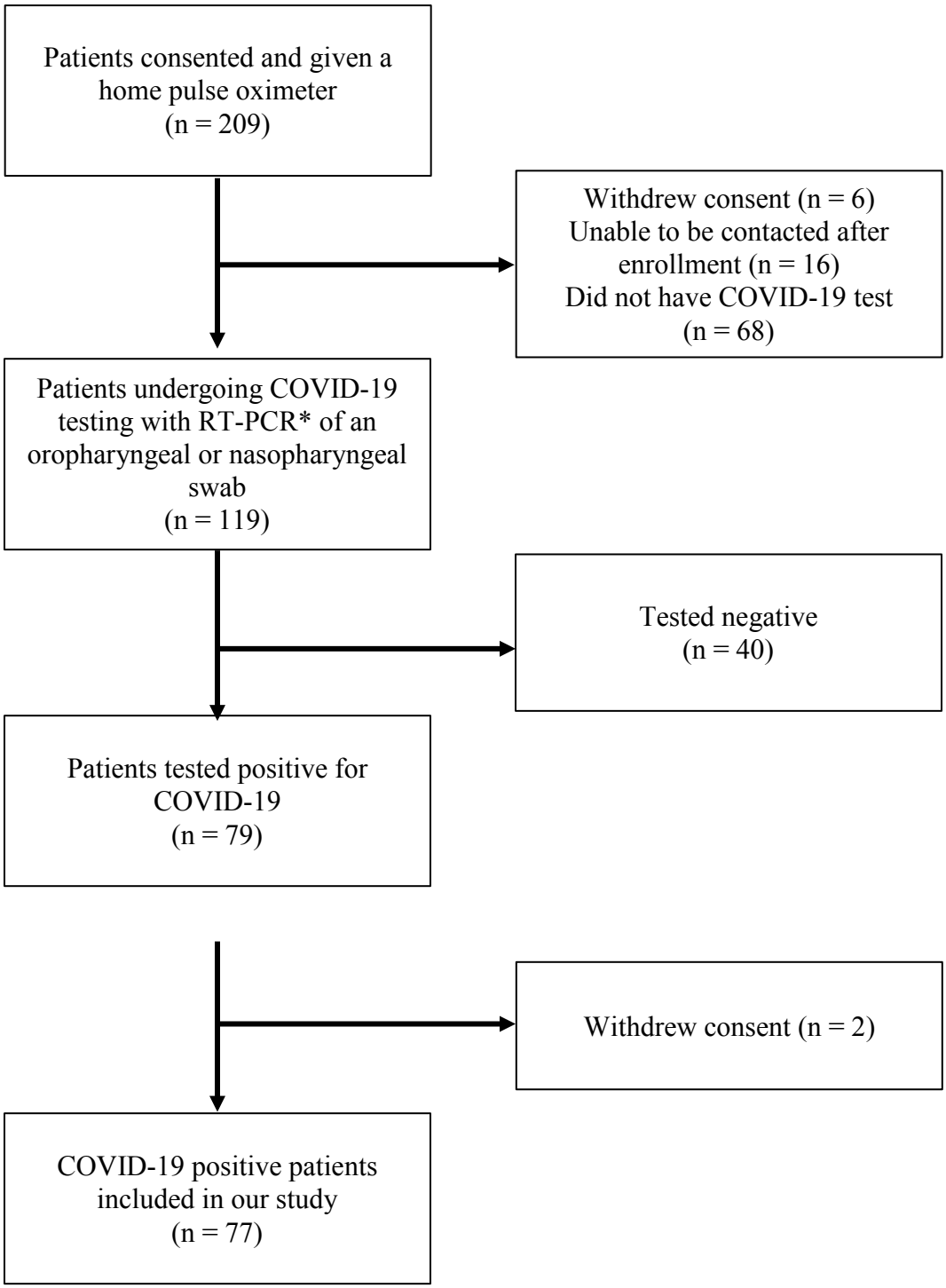


Figure 1: Patient Enrollment
In accordance with our institutional review board, patients who withdrew consent or met exclusion criteria were not included. 209 were enrolled, 77 were ultimately included in our study.
* RT-PCR = reverse-transcriptase polymerase-chain-reaction

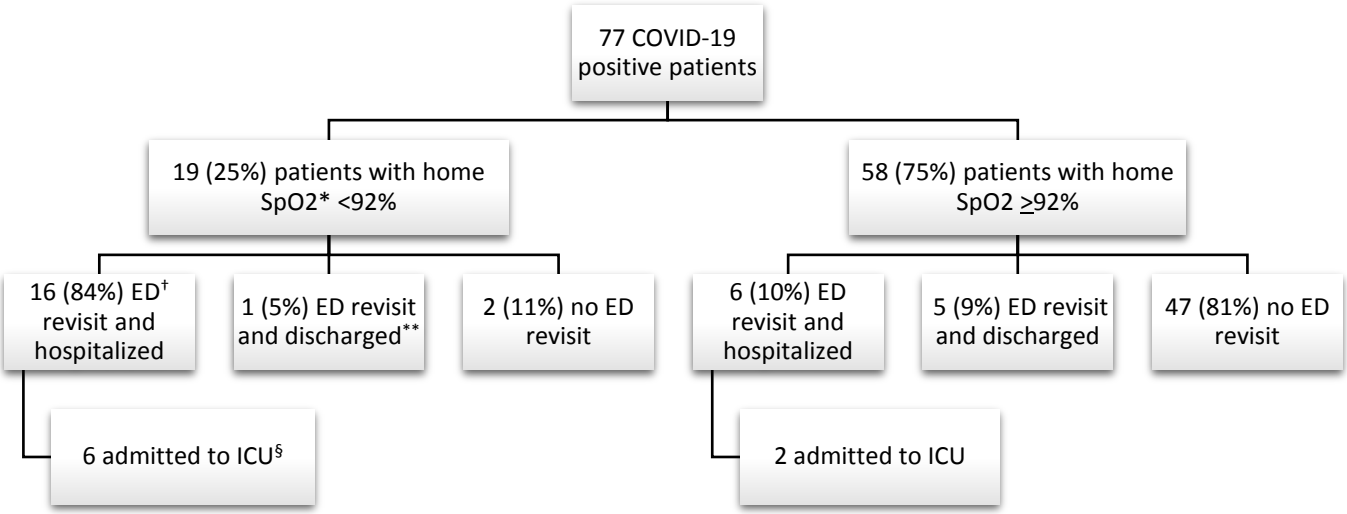


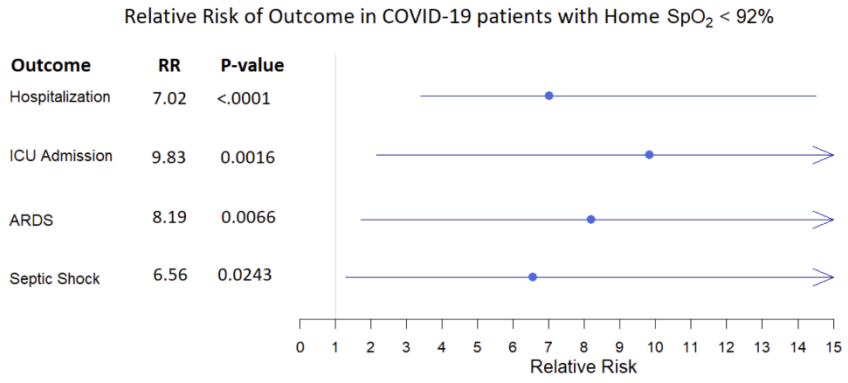
Figure 2: Outcomes of COVID-19 positive patients

*SpO2 = home pulse oxygen saturation

†ED = emergency department

§ICU = intensive care unit

**This patient had resting SpO_2 of 94% in the emergency department and was discharged to home.



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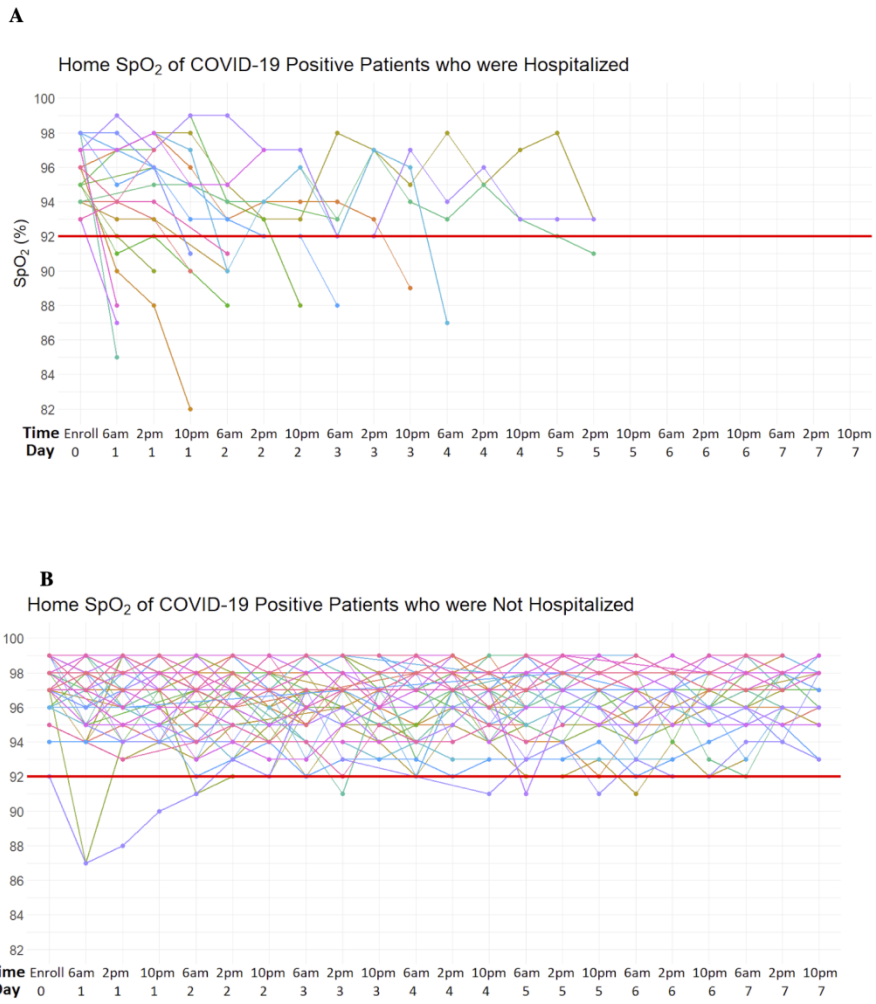

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