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Clinical characteristics of a COVID-19 cohort treated at UCLA Ronald Reagan Medical Center during the breaking phase of the pandemic: A retrospective study

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A B S T R A C T

To this date, COVID-19 remains an unresolved pandemic, and the impairment of redox homeostasis dictates the severity of clinical outcomes. Here we examined initial UCLA cohort of 440 COVID-19 patients hospitalized between March 1st and April 1st, 2020, representing the first wave of the pandemic. The mean age was 58.88 ± 21.12, among which males were significantly more than females (55.5 % vs. 44.5 %), most distinctively in age group of 50–69. The age groups of 50–69 (33.6 %) and ≥70 (34.8 %) dominated. The racial composition was in general agreement with Census data with slight under-representation of Hispanics and Asians, and over-representation of Caucasians. Smoking was a significant factor (28.8 % vs. 11.0 % in LA population), likewise for obesity (BMI ≥30) (37.4 % vs. 27.7 % in LA population). Patients suffering from obesity or BMI<18.5 checked into ICU at a significantly higher rate. A 74.5 % of the patients had comorbidities including diabetes, chronic kidney disease, chronic pulmonary disease, congestive heart failure and peripheral vascular disease. The levels of d-dimer were drastically upregulated (1159.5 ng/mL), indicating hypercoagulative state. Upregulated LDH (328 IU/L) indicated significant tissue damages. A distorted redox hemeostasis is a common trait associated with these risk factors and clinical markers. A quarter of the patients received antivirals, among which Remdesivir most prescribed (23.6 %). Majority received antithrombotics (75 %), and antibiotics. Upon admission, 67 patients were intubated or received CPR; 177 patients eventually received intensive care (40.2 %). While 290 were discharged alive, 10 remained hospitalized, 73 were transferred, and 36 died with 3 palliatively discharged. In summary, our data fully characterized a Californian cohort of COVID-19 at the breaking phase of the pandemic, indicating that population demographics, biophysical characters, comorbidities and molecular pathological parameters have significant impacts on the evolvement of a pandemic. These provide critical insights into effective management of COVID-19, and future break from another pathogen.

1. Introduction

The coronavirus disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), led to a deluge of positive cases in the United States (US) that suffocated many millions of households [1]. The first confirmed case in California was reported on Jan 26th, 2020 [2], just five days after the 1st reported case in the US in the State of Washington [3]. During the 1st quarter of 2020, the first wave was gaining in strength, and an immenent surge was all but inevitable. On March 11th, 2020, WHO officially declared COVID-19 as a global pandemic [4].

Redox imbalance is key mediator of COVID-19 pathophysiolgy [5], which leads to exacerbated inflammation (e.g., cytokine storm) and accelerated replication and entrance of SARS-COV-2 into host cells [6]. ACE2-dependent NOX2 activation in the endothelial cells is an early trigger for the subsequent hikes of oxidative stress [7]. The severity of redox imbalance/disruption of redox homeostasis correlated with magnitude of heightened inflammary (e.g., interleukin-6,

pro-coagulant (e.g., D-dimer) responses and tissue damages, ultimately predicted mortality and chronic disabilities (i.e. Long COVID) [5,8–10] among infected population. This evolving situation posed an urgent as well as long-lasting threat to the humanity, alerting to better characterization of the patient population especially focusing on factors revealing oxidative and inflammatory stress.

On the verge of a global pandemic, several unique characteristics of Californian population make this state decisive for the magnitude and duration of the national ramification. As the most populous state, the congregation of people obviously facilitate the spread of viruses. Moreover, California has long been known as a "majority minority" state, as about 62.5 % of the population is reported to be ethnic minorities [11] with family roots around the world. Of note, SARS-CoV-2 propogated at varying kinetics across different populations [12,13]. From the perspectives of flatten-the-curve and reaching-herd-immunity, a distinctive blend of these demographic features presented unique sets of challenges and opportunities.

Facing the onslaught of the fierce 1st wave, the California healthcare system, as well as the whole nation, learned as it scrambled to provide the best available remedies to all, in the midst of an acute shortage of medical supplies [14,15]. Patients admitted during this period were also generally presenting severe symptoms. The dynamics of this early cohort primed and set the trajectory of the pandemic. Meanwhile, earlier experience from China indicated beneficial effects of treating COVID-19 with Traditional Chinese Medicine [16,17]; but there was limited opportunies to have those applied to treatment of patients in other territories though exported to some. Amid difficulties, the countermeasures by the UCLA Medical Center, the highest ranked hospital at the time in the West, provided pivotal references. Moreover, as the attention of the public and scientific community has been shifting toward long COVID symptoms, the threats of acute COVID infections have ramped up again exactly this season. In fact, waves of acute infections blew through every winter since 2020. The magnitude and impact of the 2023 winter wave urged HHS to approve the delivery of free antigen test kits to once again all American households in September 2023 and in November 2023 via USPS [18]. The aim of this study was to delineate the correlations and associations among demographics, clinical manifestations, laboratory workups, treatment regimes and outcomes of the cohort treated at UCLA Medical Center in the leading edge of the 1st wave, which remain critically important for the current management of the disease and of future outbreaks of pandemics of any new pathogens.

2. Methods

2.1. Study population

This study was approved by the Institutional Review Board at UCLA Medical Center.

We performed a retrospective observational cohort study of inpatients with the diagnosis of COVID-19 hospitalized between March 1st and April 1st, 2020 at the UCLA Ronald Regan Medical Center (n = 440). Diagnosis was made using a RT-PCR test with a nasopharyngeal swab specimen. The medical records of patients were retrospectively reviewed and relevant data was extracted and collected by the study investigators through a clinical research form (CRF, UCLA).

2.2. Data collection

Data were collected on the following patient's demographic characteristics: age, sex, ethnicity, smoking history, body mass index (BMI), blood types, vital signs (i.e., heart rate, body temperature, respiratory rate and blood pressures) and a full scale laborarotary workups (e.g., coagulation factors, cytokines, tissue injury markers). In parallel, data on the following comorbidities was collected: myocardial infarction, congestive heart failure, peripheral vascular disease, cerebrovascular disease, dementia or chronic cognitive deficit, chronic pulmonary

disease, rheumatologic disorder, peptic ulcer disease, liver disease, diabetes with end-organ damage, diabetes, paraplegia or hemiplegia, chronic kidney disease, active cancer, and HIV. Data on the following primary outcomes was traced and collected up to 13 May 2020, including hospital admission, ICU admission, duration of stay at ICU, oxygen saturation status at ICU, usage of ECMO, transplant needed and survival or discharge options.

2.3. Statistical analysis

Data in the eMR database was de-identified of protected health information (PHI) per HIPAA guideline and then tabulated into a worksheet of SPSS (version 28.0). Categorical variables were presented in the form of counts of patient numbers and percentages. They were then analyzed using χ2 test; when sample size being small (n < 5), Fisher's exact test was used, instead. The comparison in distribution between hospitalized patients and the general population in LA was conducted using the binomial distribution method. The population distribution data for LA was sourced from the United States Census Reporter [19]. The BMI data was sourced from the Adult Obesity Prevalence Maps [20]. The data on prevalence of smoking in LA population was quoted from California Tobacco Facts and Figures 2019 [21]. Clinical lab results were examined by Mann-Whitney U test. A p < 0.05 was considered statistically significant. All analyses were performed using the built-in statistics package of SPSS (IBM SPSS Statistics Premium Campus Edition).

3. Results

During the period of data collection (March 1st to April 1st, 2020), a total of 440 patients was diagnosed, treated at UCLA Ronald Reagan Medical Center, and included in this restrospective cohort. All these patients had a positive COVID-19 diagnosis based on RT-PCR results.

3.1. Demographics

The mean age of the cohort was 58.88 years (Table 1), which was markedly higher than the average of 37.6 years for the general population of LA (2020 Census data [19]). Further analysis revealed that the cohort represented a significant deviation from LA population in all age groups: with higher proportions of senior [50–69 (33.6 % vs. 24 %) and ≥70 (34.8 % vs. 10 %)] and lower proportions of youngsters and adults [0–18 (3.6 % vs. 22 %) and 19–49 (28.0 % vs. 44 %)] (Fig. 1). These data seem to be consistent with the notion that aged people are more prone to COVID-19 [22]. However, the age group 0–18 in this corhort had the highest probability ($p = 0.018$) in need of intensive care (Table 2).

The statistics also illustrated that males were more vulnerable than females (55.5 % vs. 44.5 %), with most striking distinction observed at the age group of 50–69 [23,24]. After receiving treatments, the chances of needing intensive care did not show significant differences between genders in this cohort (Table 2).

The racial composition of the cohort were in general agreement with the most recent Census data of LA with slight under-representation of Hispanic (40.8 % vs. 48.1 %) and Asian (8.1 % vs. 11.5 %) ethnicities and over-representation of Caucasian (35.6 % vs. 28.1 %) (Fig. 1). Interestingly, females with both Hispanic and Caucasian heritages represented a significantly higher proportion over their male counterpart, which was not observed in any other races. After treatment, Caucasians turned out to be the group with the least chance in need of intensive care (Table 2).

Another demographic feature that affected the vulnerability greatly was body mass index (BMI). According to CDC reports, the mean BMI of LA population was 27.5 [20], which is significantly below National average. The matrix of this cohort's BMI, on the other hand, was significantly skewed upward to 29.02. Remarkably, people in the obese category (BMI ≥30) accounted for 37.4 % of this cohort in comparison to 27.7 % of LA population ($p < 0.000$) [25]. Moveover, patients with a

Table 1
Demographics.

Gender	Male (%)	Female (%)	P value	Total (%)	Census [male%]	P value
Count	244 (55.5)	196 (44.5)		440 (100)	50	0.025*
Age	**Male**	**Female**	**P value**	**Total**	**Census (%)**	**P value**
Mean	57.62 ± 19.47	60.43 ± 22.97	0.173	58.88 ± 21.12	37.6	0.000*
0–18 yr	9 (3.7)	7 (3.6)	0.948	16 (3.6)	22	0.000*
19–49 yr	63 (25.8)	60 (30.6)	0.266	123 (28.0)	44	0.000*
50–69 yr	98 (40.2)	50 (25.5)	0.001*	148 (33.6)	24	0.000*
≥70 yr	74 (30.3)	79 (40.3)	0.029*	153 (34.8)	10	0.000*
Ethnicity	**Male (239)**	**Female (191)**	**P value**	**Total (430)**	**Census**	**P value**
Hispanic				175 (40.8)	48.1	0.001*
Hispanic alone	3 (1.2)	1 (0.5)	0.633	4		
Hispanic + African American	2 (0.8)	0 (0)	0.505	2		
Hispanic + Asian	1 (0.4)	0 (0)	1.000	1		
Hispanic + American Indian	1 (0.4)	0 (0)	1.000	1		
Hispanic + Caucasian	22 (9.1)	33 (17.1)	0.013*	55		
Hispanic + Other	64 (26.4)	48 (24.9)	0.699	112		
African American	20 (8.3)	15 (7.8)	0.846	35 (8.1)	7.8	0.422
Asian	18 (7.4)	17 (8.8)	0.606	35 (8.1)	11.5	0.014*
Caucasian	88 (36.4)	65 (33.7)	0.548	153 (35.6)	28.1	0.000*
Hawaiian & Pacific Islander	0 (0)	1 (0.5)	0.444	1 (0.2)	0.2	0.577
Other	20 (8.3)	11 (5.7)	0.299	31 (7.2)	4.3	0.004*
BMI	**Male (187)**	**Female (147)**	**P value**	**Total (334)**	**CDPH Data**	**P value**
Mean (kg/m²)	28.77 ± 6.83	29.32 ± 9.03	0.813	29.02 ± 7.87	27.5	0.000*
Distribution						
BMI <18.5	6 (3.2)	7 (4.8)	0.466	13 (3.9)	2.5	0.08
18.5 ≤ BMI <25	56 (29.9)	48 (32.7)	0.596	104 (31.1)	35.9	0.038*
25 ≤ BMI <30	57 (30.5)	35 (23.8)	0.175	92 (27.5)	33.9	0.008*
BMI ≥30	68 (36.4)	57 (38.8)	0.651	125 (37.4)	27.7	0.000*
Smoking History	**Male (206)**	**Female (166)**	**P value**	**Total (372)**	**CDPH Data**	**P value**
Total smoker	66 (32.0)	41 (24.7)	0.120	107 (28.8)	11.0	0.000*
Current	9 (4.4)	7 (4.2)	0.943	16 (4.3)		
Former	57 (27.7)	34 (20.5)	0.109	91 (24.5)		
Never smoker	140 (68.0)	125 (75.3)	0.120	265 (71.2)		

healthy BMI (18.5 ≤ BMI <25) were less likely ($p = 0.019$) checked into ICU, while patients suffering from obesity (Table 2) or with BMI <18.5 (Fig. 2) checked in at a significantly higher rate ($p = 0.041$).

Smoking history was also a significant risk factor. Among 372 patients responded to this survey, 28.8 % had a smoking history compared

to LA population average at 11.0 % reported by the California Department of Public Health (CDPH) [21].

3.2. Comorbidities and mortalities

Most patients within this cohort checked in with comorbidities (74.5 %) (Table 3); some of them had multiple. Only 112 out of 440 did not report known comobidities. The most common comorbidities were diabetes (n = 175), chronic kidney disease (n = 136), chronic pulmonary diasese (n = 122), congestive heart failure (n = 90) and peripheral vascular disease (n = 80). There were no significant gender-based differences of having them. The relative influences attributed by comorbidities were dissected by further stratification of the data. Among patients with no more than 2 comorbidities, the top four complicating factors ranked as diabetes (67/440 or 15.2 %), chronic pulmonary diasese (33/440 or 7.5 %), chronic kidney disease (24/440 or 5.45 %) and active cancer (21/440, or 4.77 %).

3.3. Vitals and labs

Vitals and laboratory workup sheets of all patients were recorded (Table 4). The body temperature was elevated (37.33°C ± 0.94°C) beyond the reference range of healthy population (36°C–37°C). Meanwhile, the means of respiration rate (19.91 ± 4.55) were close to the upper limit of the reference range (12–20 per min). Upon admission, 104 (42.6 %) males and 78 (39.8 %) females recorded an oxygen saturation levels less than 95 %; during hospitalization, the oxygen saturation levels of 232 (95.1 %) males and 173 (88.3 %) females dropped below 95 %, while 148 (60.7 %) males and 118 (60.2 %) females dropped below 90 %. Nevertheless, only 14 (5.7 %) males and 13 (6.6 %) females recorded lactic acidosis (>25.7 mg/dL). Systolic blood pressure (SBP, 129.24 ± 23.66) was also elevated (90–120), while diastolic blood pressure (DBP, 76.71 ± 16.06) did not show a statistically significant increase (60–80) with the cohort as a whole. Taking a closer look, we were able a discern a mildly higher DBP (78.05 ± 16.11) for the males over that (75.04 ± 15.87) of females ($p = 0.025$).

CBC result revealed that the means of all tested blood cell characteristics were within the normal ranges. It is well established that the male population has higher levels of hemoglobin and hematocrit, while the female population has a higher level of platelet, which was consistent with observed values in the chart.

The means of d-dimer were drastically upregulated (1159.5 ng/mL) beyond reference range (0.00–500 ng/mL) (Fig. 3). This is consistent with the notion that COVID-19 patients have high thrombotic profiles [26,27]. Meanwhile, the means of prothrombin time (PT, 13.55 s), international normalized ratio (INR, 1.10), activated particle thromboplastin time (aPTT, 31.7 s), Fibrinogen (579 mg/dL) were within their respective normal reference ranges. This was in agreement with that this cohort was eligible to receive antithrombotic therapy and it was important for this cohort of patients, like for most if not all severe cases of COVID-19 patients, to receive antithrombotic therapies. (Table 5).

The means of positive acute phase proteins, C-reactive protein (7.2 mg/L), procalcitonin (0.1 ng/mL), Ferritin (589 ng/mL), Fibrinogen (579 mg/dL) were all upregulated; while the means of negative acute phase protein, albumin (3.6 g/dL), were downregulated. Furthermore, the rise of ferritin level was higher in males (756.5 ng/mL) than females (355.5 ng/mL). The mean of pro-inflammatory cytokine, interleukin-6 (7.0 pg/mL) were within its reference range, consistent with anti-inflammary therapy that this cohort had received (Table 5). These inflammatory markers increased further for patients in need of intensive care, so did the marker for blood clots, d-dimer (Fig. 3).

The increase in the circulating level of LDH (328 IU/L) indicated that this cohort generally suffered significant tissue damages. Conversely, the means of CK (95.5 IU/L), AST (35 IU/L), ALT (27 IU/L), BNP (75.5 pg/mL), bilirubin total (0.4 mg/dL) and bilirubin direct (0.2 mg/dL) were all within each respective reference range. Taken together, it
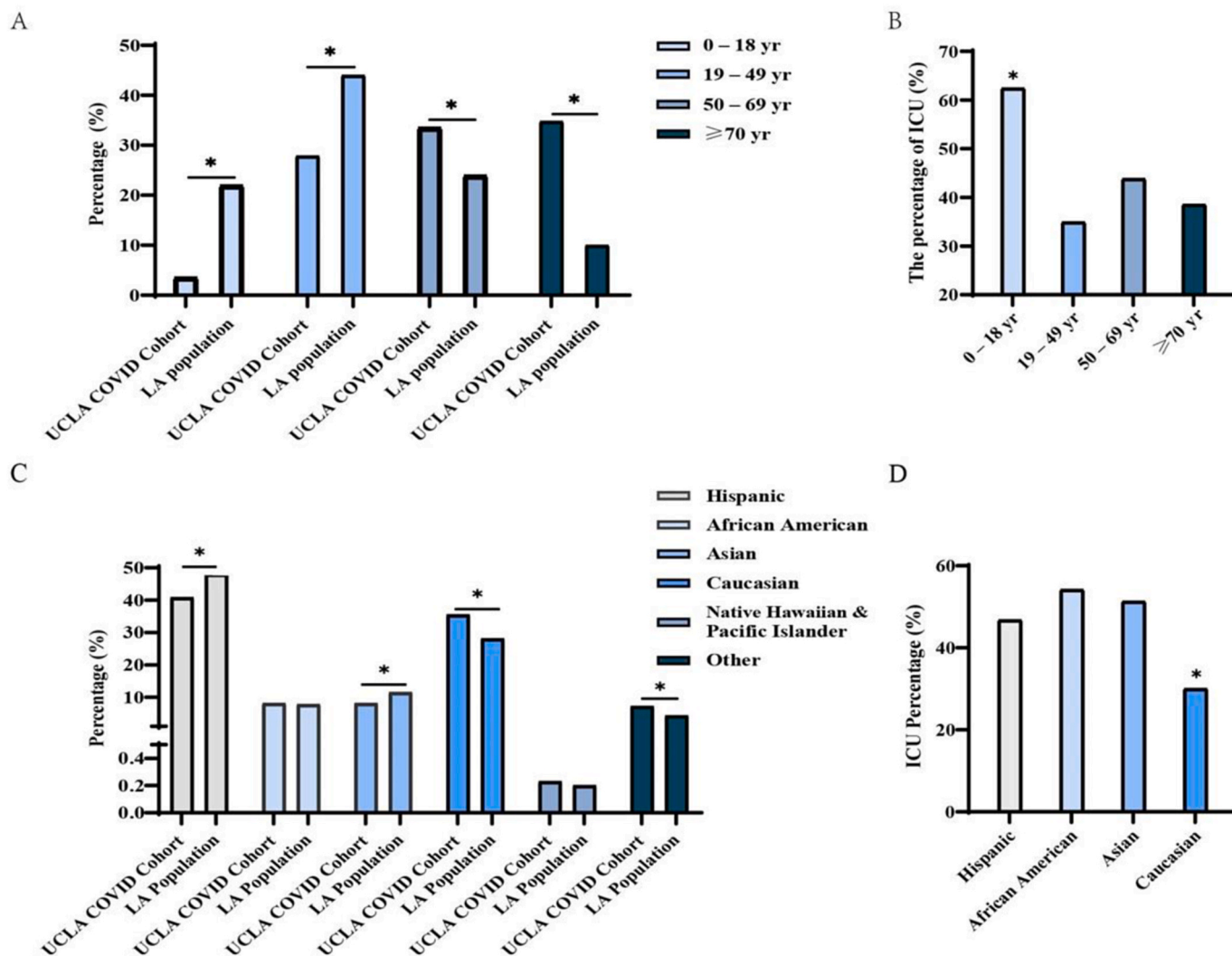
Fig. 1. Age Distribution of the UCLA Cohort in Comparision with Los Angeles Population

The composition of the UCLA cohort was dissected and compared to the general population in Los Angeles (LA). **Panel A**. Four age groups were denominated for patients receiving treatment of COVID-19 at UCLA Ronald Reagent Medical Center: 0–18 years old, 19–49 years old, 50–69 years old and older than 70 years. **Panel B**. The UCLA cohort in need of intensive care were counted by age groups. **Panel C**. Six racial heritages were denominated: Hispanic, African American, Asian, Caucasian, Native Hawaiian & Pacific Islanders as well as Other. **Panel D**. The UCLA cohort in need of intensive care were counted by racial groups. Statistical analysis was conduced to examine whether the makeup of this cohort significantly deviated from the distributions among the LA local population. * represents p ≤ 0.05.

illustrated that tissue damages in this cohort had been systemic and heterogeneous. Interestingly, all but BNP exhibited significantly higher means in males versus females (LDH $p = 0.048$, CK $p = 0.003$, AST $p = 0.009$, ALT $p = 0.000$, total bilirubin $p = 0.000$). The values of creatinine, urine nitrogen and eGFR were within normal reference ranges. Creatinine levels were significantly higher in males than in females ($p = 0.000$).

3.4. Medications

Fighting COVID-19 back in March 2020 when targeted remedies remained elusive, a combination of medication (Table 5) were formulated to quash the acute assults by the pathogens and to mitigate secondary injuries.

A quarter of the cohort received antivirals, Ganciclovir (2), Oseltamivir (7), Remdesivir (104), Ribavirin (1) and Valganciclovir (6). Remdesivir has been reported to be effective to combat COVID-19 at the time [28]. Thus, it was most frequently prescribed (23.6 % of the cohort). There was no gender-based preference in antiviral applications,

expect Valganciclovir. It was used exclusively for males, with known side effects to fetus. Convalescent plasma was provided to 42 patients to eradicate lingering virus. Working in concert with antivirals, ACE inhibitor (40) and angiotensin receptor blocker (33) were prescribed to manage hypentensive comorbidities.

The majority of this cohort was in need of and prescribed with antithrombotics (75 %), in the forms of continuous infusion of heparin (69 patients), subcutaneous unfractionated heparin (129 patients), low molecular weight heparin (226 patients), or argatroban (4 patients). The course of antithrombotics patients received, progressed as their symptom evolved, as severe cases received more aggressive intervention (Table 6). Managing inflammatory response, steroids were provided with caution due to their known side-effects. About 10 % of the cohort did receive steroids. On the contrary, NSAIDs were dispensed to approximately half of the cohort (43 %). In general, there were no gender-based preference, except asprin. As the most widely used NSAIDs, Asprin is unique in its antithrombotic activities. It was prescribed less frequently to the female patients ($p = 0.023$). A total of fifteen forms of immunosuppressants were provided.

Table 2
Demographics of patients received intensive care.

	NO ICU (%)	ICU (%)	Total (%)	P value
Count	263 (59.8)	177 (40.2)	440 (100)	
Age				
Mean	59.38 ± 21.24	58.12 ± 20.97	58.88 ± 21.12	0.777
0–18 yr	5 (1.9)	11 (6.2)	16 (3.6)	0.018*
19–49 yr	81 (30.8)	42 (23.7)	123 (28.0)	0.105
50–69 yr	83 (31.6)	65 (36.7)	148 (33.6)	0.261
≥70 yr	94 (35.7)	59 (33.3)	153 (34.8)	0.603
Gender				
Male	141 (53.6)	103 (58.2)	244 (55.5)	0.343
Female	122 (46.4)	74 (41.8)	196 (44.5)	
Oxygen saturation status at ICU				
Room air	231 (90.9)	118 (74.2)	349 (84.5)	0.000
Nasal cannula	22 (8.7)	18 (11.3)	40 (9.7)	0.374
Invasive ventilation	1 (0.4)	23 (14.5)	24 (5.8)	0.000
Ethnicity				
Hispanic				
Hispanic alone	2 (0.8)	2 (1.1)	4 (0.9)	1.000
Hispanic + African American	1 (0.4)	1 (0.6)	2 (0.5)	1.000
Hispanic + Asian	0 (0)	1 (0.6)	1 (0.2)	0.407
Hispanic + American Indian	1 (0.4)	0 (0)	1 (0.2)	1.000
Hispanic + Caucasian	28 (10.8)	27 (15.3)	55 (12.6)	0.175
Hispanic + other	61 (23.6)	51 (29.0)	112 (25.7)	0.226
African American	16 (6.2)	19 (10.8)	35 (8.0)	0.088
Asian	17 (6.6)	18 (10.2)	35 (8.0)	0.178
Caucasian	107 (41.3)	46 (26.1)	153 (35.2)	0.001*
Native Hawaiian & Pacific Islander	0 (0)	1 (0.6)	1 (0.2)	0.407
Other	22 (8.5)	9 (5.1)	31 (7.1)	0.170
BMI				
Mean	28.52 ± 7.99	29.75 ± 7.66	29.02 ± 7.87	0.046*
BMI< 18.5	7 (3.5)	6 (4.5)	13 (3.9)	0.651
18.5 ≤ BMI <25	72 (36.0)	32 (23.9)	104 (31.1)	0.019*
25 ≤ BMI <30	55 (27.5)	37 (27.6)	92 (27.5)	0.982
BMI ≥30	66 (33.0)	59 (44.0)	125 (37.4)	0.041*

Hydroxychloroquine and leronlimab each was provided to 11 % of the cohort. Leronlimab ($p = 0.038$) and sarilumab ($p = 0.037$) were prescribed more frequently among males, while methotrexate ($p = 0.039$) more among females.

To manage symptoms, 163 patients (male: 95, 38.9 %; female 68, 34.7 %) received bronchodilator in the form of inhaled beta-agonist and half of the cohort received vasopressors. In parallel, antibiotics was provided to the majority of the cohort to prevent consequent/co-existing bacterial infection or bacterial sepsis. No gender-based preference was practiced on these medications.

For patients in need of intensive care, there were some significant changes in medication regime (Table 6) compared to those with milder ailment. Antivirals was prescribed more aggressively, particularly Remdesivir ($p = 0.003$), so was convalescent plasma ($p = 0.000$). Antithrombotics was ramped up favoring continuous infusion. Potent antiinflammatry steroid and immunosuppressants were used to replace NSAIDs such as ketorolac. Bronchodilator and vasopressors were dispensed more to have a stronger grip on vital symptoms for these

Table 3
Comorbidities.

| Comorbidities	Male (%)	Female (%)	Total	P value
Myocardial Infarction	38 (15.6)	19 (9.7)	57 (13.0)	0.068
Congestive Heart Failure	51 (20.9)	39 (19.9)	90 (20.5)	0.795
Peripheral Vascular Disease	48 (19.7)	32 (16.3)	80 (18.2)	0.366
Cerebrovascular Disease	47 (19.3)	31 (15.8)	78 (17.7)	0.347
Dementia or Chronic Cognitive Deficit	32 (13.1)	37 (18.9)	69 (15.7)	0.098
Chronic Pulmonary Disease	60 (24.6)	62 (31.6)	122 (27.7)	0.101
Rheumatologic Disorder	12 (4.9)	17 (8.7)	29 (6.6)	0.115
Peptic Ulcer Disease	12 (4.9)	9 (4.6)	21 (4.8)	0.873
Liver Disease	42 (17.2)	30 (15.3)	72 (16.4)	0.591
Diabetes	101 (41.4)	74 (37.8)	175 (39.8)	0.438
Paraplegia or Hemiplegia	13 (5.3)	7 (3.6)	20 (4.5)	0.379
Chronic Kidney Disease	80 (32.8)	56 (28.6)	136 (30.9)	0.342
Active Cancer	39 (16.0)	31 (15.8)	70 (15.9)	0.962
HIV	3 (1.2)	1 (0.5)	4 (0.9)	0.632
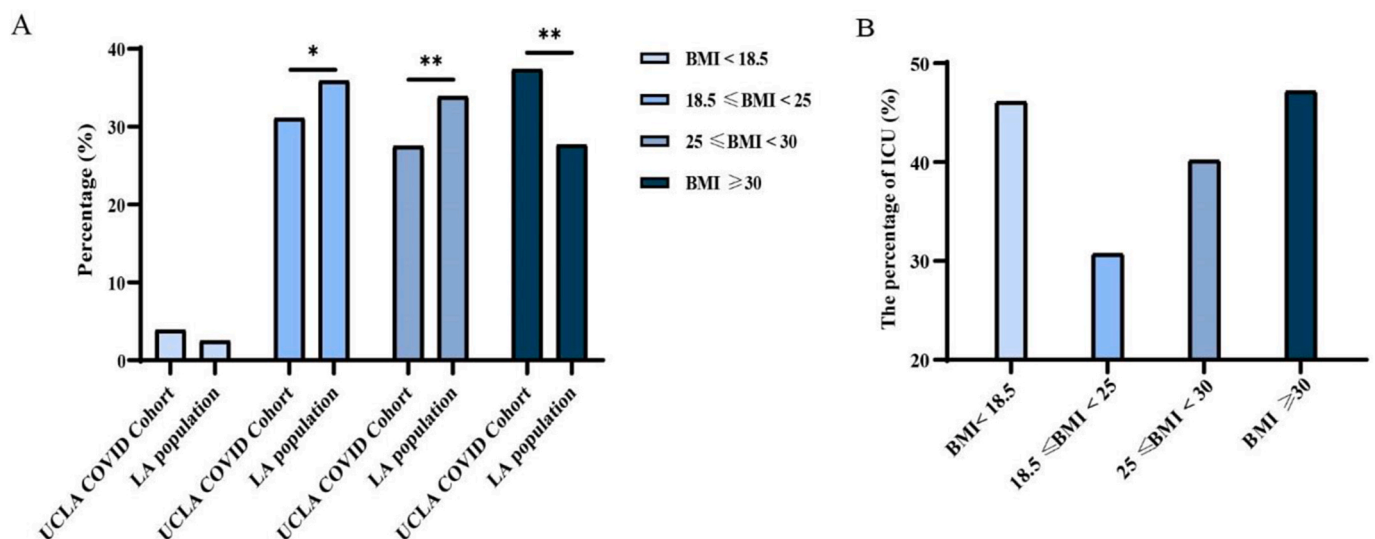
Fig. 2. BMI Distribution of the UCLA Cohort in Comparision with Los Angeles Population
The BMI of patients in the UCLA cohort was partitioned into four groups: Less than 18.5, between 18.5 and 25, between 25 and 30, as well as greater than 30. **Panel A**. The percentage of patients in each group of the cohort was charted in comparison with population percentage in Los Angeles (LA). **Panel B**. The chance of needing intensive care was charted against BMI of patients. The group with BMI within 18.5–25 had the lowest chance in need of intensive care, while either increase or decrease of BMI outside of normal range exhibited a higher vulnerability.

Table 4
Vital signs and clinical laboratory workups.

Vital Signs	Male (95 % CI)	Female (95 % CI)	P value	Total (95 % CI)	Reference
Heart Rate (/min)	91 (58–133)	93 (60–146)	0.223	92 (60–138)	60–100
Temperature (°C)	37.2 (35.9–39.4)	37.0 (36.0–39.3)	0.067	37.1 (35.9–39.3)*	36–37
Respiratory Rate (/min)	18 (14–30)	18 (15–33)	0.489	18 (15–32)	12–20
SBP (mmHg)	130 (82–181)	128 (88–174)	0.606	128 (86–177)*	90–120
DBP (mmHg)	77 (47–114)	73 (45–114)	0.025*	76 (46–114)	60–80
Laboratory Workups	**Male (95 % CI)**	**Female (95 % CI)**	**P value**	**Total (95 % CI)**	**Reference**
White Cell ($\times 10^9$/L)	7.05 (1.81–22.07)	7.02 (2.95–22.59)	0.298	7.03 (2.14–21.91)	3.4–10.8
Hemoglobin (g/dL)	13.50 (7.66–17.32)	12.10 (6.92–15.48)	0.000*	12.7 (7.29–16.66)	11.1–15.9
Platelet ($\times 10^9$/L)	187.50 (54.83–450.95)	227.00 (82.00–455.40)	0.000*	207 (64.40–452.60)	150–450
Hematocrit (%)	41.20 (24.18–51.92)	37.20 (22.30–48.08)	0.000*	39.2 (23.64–50.16)	34.0–46.6
Lymphocyte ($\times 10^9$/L)	0.94 (0.20–41.04)	1.08 (0.26–3.51)	0.058	0.99 (0.20–3.69)	0.7–3.1
Neutrophil ($\times 10^9$/L)	5.22 (0.66–18.41)	5.12 (1.43–19.95)	0.655	5.17 (0.96–19.77)	1.4–7.0
Eosino ($\times 10^9$/L)	0.01 (0.00–0.33)	0.01 (0.00–0.29)	0.679	0.01 (0.00–0.30)	0.0–0.4
d-dimer (ng/mL FEU)	1141.00 (249.80–10000.00)	1195.00 (370.00–10000.00)	0.766	1159.5 (331.38–10000.00)*	0.00–500
PT (sec)	13.55 (12.00–22.28)	13.55 (11.71–24.77)	0.784	13.55 (11.93–22.37)*	9.1–12.0
INR	1.10 (0.90–2.11)	1.10 (0.90–2.36)	0.604	1.10 (0.90–2.12)	0.9–1.2
APTT (sec)	32.00 (25.08–60.93)	30.90 (24.00–89.76)	0.089	31.7 (24.02–75.58)	24–33
Fibrinogen (mg/dL)	548.5 (137.0–888.5)	588.0 (107.0–882.4)	0.969	579 (116.0–881.0)*	233–496
C-Reactive Protein (mg/L)	7.20 (0.30–34.69)	7.20 (0.30–19.96)	0.133	7.20 (0.30–28.98)*	0.00–3.00
Procalcitonin (ng/mL)	0.11 (0.10–16.09)	0.10 (0.10–14.99)	0.456	0.1 (0.10–14.64)*	0.00–0.08
Ferritin (ng/mL)	756.50 (74.50–5494.25)	355.50 (24.50–3996.00)	0.000*	589 (37.45–5129.88)*	15–150
Albumin (g/dL)	3.70 (2.17–4.64)	3.60 (2.21–4.60)	0.166	3.6 (2.20–4.60)*	4.0–5.0
Interleukin-6 (pg/mL)	7.0 (2.0–618.84)	6.6 (2.0–837.05)	0.796	7.0 (2.0–504.6)	0.0–13.0
LDH (IU/L)	352.50 (156.40–1119.60)	302.00 (136.50–658.50)	0.048*	328 (152.50–1066.30)*	121–224
CK (IU/L)	109.00 (24.58–2532.95)	74.00 (14.88–1539.00)	0.003*	95.5 (18.88–1912.38)	24–173
AST (IU/L)	37.00 (15.40–224.00)	33.00 (12.00–218.98)	0.009*	35.00 (13.00–218.00)	0–40
ALT (IU/L)	31.00 (6.70–152.50)	23.00 (5.68–132.65)	0.000*	27.00 (6.00–141.15)	0–44
Troponin (ng/mL)	0.04 (0.04–0.97)	0.04 (0.04–1.20)	0.217	0.04 (0.04–1.07)	0–22
BNP (pg/mL)	74.00 (15.45–4283.90)	81.00 (15.00–620.65)	0.823	75.5 (15.00–1973.73)	0.0–100.0
Direct bilirubin (mg/dL)	0.20 (0.20–0.95)	0.20 (0.2–1.03)	0.010*	0.20 (0.20–0.89)	0.0–0.4
Total bilirubin (mg/dL)	0.50 (0.20–3.21)	0.40 (0.20–2.35)	0.000*	0.4 (0.20–2.86)	0.0–1.2
Creatinine (mg/dL)	1.02 (0.45–10.25)	0.79 (0.34–6.96)	0.000*	0.94 (0.39–9.06)	0.57–1.00
Urea nitrogen (mg/dL)	18.00 (6.30–105.40)	16.00 (5.00–91.18)	0.143	17 (6.00–99.45)	6–20
eGFR (mL/min/1.73)	76.00 (5.75–89.00)	76.00 (6.00–89.00)	0.999	76 (6.00–89.00)	>59

The lab results were presented in the format of median (95 % CI). 95 % confidence interval was presented as the values corresponding to the 2.5th and 97.5th percentiles, respectively.

patients. The application of antibiotics was increased for ICU patients to keep secondary infections in check.

3.5. Outcomes

Upon admission, 67 patients were intubated or received CPR (Table 7). After checked in, 177 patients eventually received intensive care (40.2 %) and the chances were not different between males and females. Of this cohort, 290 were discharged alive, 10 remained hospitalized during the phase of this study, 73 were transferred to other facilities, and 36 did not survive with 3 palliatively discharged. The medium of measured IL-6 values of the cohort falls within reference range, however, individual's IL-6 exhibited significant correlation with the necessity of intense care ($p = 0.000$) and final outcome (Table 8). Although a signficiantly higher percentage of males were admitted with SARS-CoV-2 infection (Table 1), after clinical intervention, the proportion of male patients that were in need of intensive care or perished showed no significant difference to the female patients in this cohort.

4. Discussion

The sudden emergence and rapid spread of COVID-19 caught the world offguard [29]. In the first quarter of 2020, the absence of targeted medication, lack of standard treatment protocol, shortage of medical supplies including even PPE, brought upon panic in advent of a rising pandemic [30,31]. Based on historical data, the world has a good chance of confronting another pandemic in our life time [32,33]. In 2024, acute COVID-19 morbidity and mortality comes back again while we are shifting our focus to long COVID syndromes. In light of this fact, HHS has authorized $600 million to urgently manufacture test kits to contain the

2023 winter episode of large scale infections [18]. In the context of a grueling pandemic, geology matters. Los Angeles, as one of the few major hubs of the global village, had far-reaching impacts, well beyond its metropolitan boundaries. Understanding its relevant demographic characteristics, identifying vulnerable communities, recognizing effective intervention approaches will enable us to better seize the opportunities breaking the chain of spread at the present time as well as when "the next big one" comes [34].

Comparing with other major US metro areas, Los Angeles has the highest Hispanic population at 48.1 %, while New York at 29 %, Seattle (reported 1st US COVID-19 case) at 7.5 %, and Washington DC at 11.7 %; relatively high Asian population at 11.8 %, while New York at 14.5 %, Seattle at 16.8 %, Washington DC at 4.7 %; and relatively lower fraction of Causation population than the rest (www.Census.gov). Our data (1 March to 1 April 2020) and data from New York (29 February to 1 June 2020) [35], Washington DC (4 March to 24 April 2020) [36] all illustrated that racial demographics and potentially associated biophysical, socioeconomical and behavior factors were significant features in the context of susceptibility to the original strain of SARS-CoV-2. Despite the differences, there were consistent findings. Seniors were clearly more vulnerable in Los Angeles (58.88 vs 37.6, mean vs Census) (Table 1), Seattle (64 vs 35.9) [37], New York (62 vs 38.4) [38] and Washington DC (63 vs 34.3) [36]. In this cohort, although 0–18 year age group represented the smallest fraction of admitted patients, the chance of these patients in this group in need of intense care was the highest. This might be due to the higher chances of exposure to SARC-CoV-2 and higher viral loads for this age group, which warrant future analyses. On a separate note, Caucasian represented the highest racial group admitted, yet the least proportion of which was in need of intensive care. This findings might be partially attributed to the notion that this group of patients had a statistically healthier body-mass index.
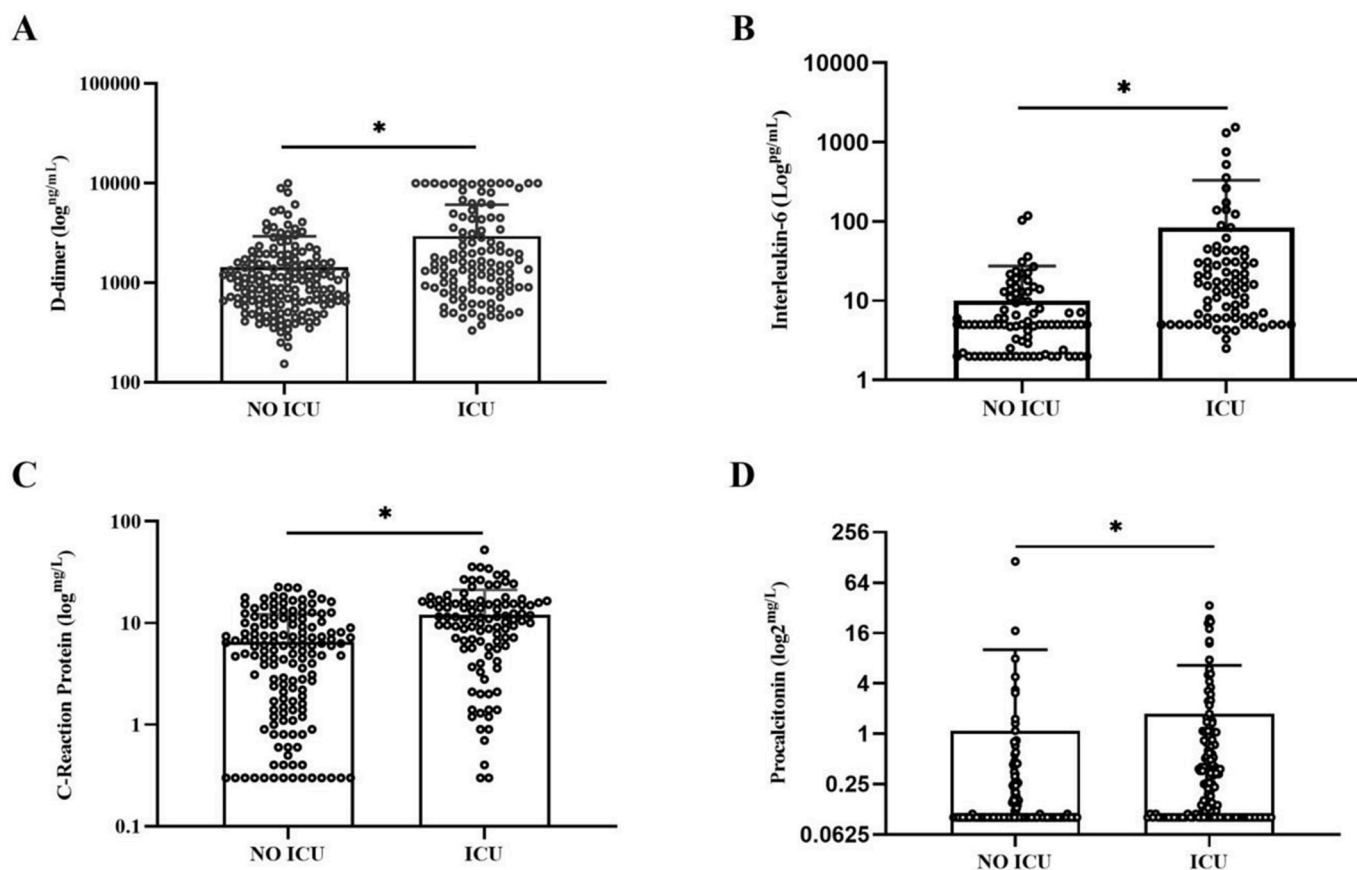
Fig. 3. Clinical Workups of Patients Receiving Intensive Care
The clinical lab test results on four parameters were charted for patients needing or not needing intensive care. Each dot represented the reading for one individual patient. Log scales were used for the y axis. Statitically significant difference was denoted with a *. **Panel A.** D-dimer. **Panel B.** Interleukin-6. **Panel C.** C-Reactive Protein. **Panel D.** Procalcitonin.

More males were confirmed with a positive diagnosis in New York (67 %) [38], Washington DC (53 %) [36], Seattle (63 %) [37], in general agreement with the stats in this cohort (55.5 %, $p = 0.025$ compared to Los Angeles population) [7,24]. Of note, the percentages of males in the New York and Seattle cohorts were the highest, and more males were critically ill. In Washington DC, 56 % of the severe cases were males patients. In our cohort, males accounted for 58.2 % of those in need of intensive care ($p = 0.343$ compared to this cohort).

Smoking history, current or former, contributed to the vulnerability. 28.8 % of patients in this cohort has smoking history, which is markedly higher than the 11 % reported by CDPH for general population in LA. The Washington DC cohort had an even higher percentage at 33 % [36]. The New York cohort was at 15.6 % [39], which is still significantly higher than the 10.1 % rate for the local population [40]. Overweight is a risk factor. This was seen in New York cohort (BMI 30.8 over 25.4 for the local population) and Washington DC cohort (29 over 24.3), in parallel with this cohort (29.02 over 27.5). Besides overweight, data also suggest that underweight (BMI <18.5) is a potential risk factor in this cohort, as higher proportion of patients in this weight group were pre-scribed with intensive care (Table 6).

Comorbidities predisposed patients with elevated risk in the context of viral infection. The leading comorbidities varied among different co-horts. The top three comobidies were heart disease (73 %), diabetes (58 %) and chronic kidney disease (23 %) [35] in New York; diabetes (58 %), chronic kidney disease (21 %) and obstructive sleep apnea (21 %) in Seattle [37]; hypertension (47 %), coronary artery disease (32 %) and diabetes (30 %) in Washington DC [36]. In this cohort, the top como-bidies were diabetes (39.8 %), chronic kidney disease (30.9 %) and chronic pulmonary disease (27.7 %). Diabetes, cardiovascular diseases

and chronic kidney diseases were top compounding factors in all these cohorts, but their percentages and rankings differed. The demographic variances may be a root cause for this deviation. Environmental factors may play a role as well contributing to the higher rate of chronic pul-monary disease in this LA cohort.

With a positive COVID-19 diagnosis, the majority of the patients showed compelling tissue damages, featuring a marked increase in circulating LDH level. This is in agreement with the observations in the cohorts of New York [39] and Washington DC [36]. However, the me-dians of tissue specific markers (troponin, BNP, AST, ALT, CK) were within normal reference range, not pinpointing a particular organ as the common primary target. Both the New York and the Washington DC cohorts recorded increase in median BNP levels and increase in AST median for the New York cohort. The differences in comorbidity affected the pathology of each patient. The most striking lab result was the drastic increase in d-dimer level median over the normal reference range. The medians of New York and Washington DC cohorts were elevated as well, but not as high as in this UCLA cohort. Disregulation of coagulation factors seemed to be a common attributes. In addition, the median of fibrinogen was significantly elevated in this cohort, while PT, INR, APTT levels still fell within the normal range. This is in line with the anti-coagulation prescriptions made available to this cohort.

Vital meaturements showed that the redox homeostasis for majority of this cohort were under assults, as indicated by blood oxygen satua-ration levels. The impact of the hypoxic state is far-reaching. In fact, most of the aforementioned risk factors and comorbidities involved perturbation of the delicate redox systems. The effects of these factors can be cumulative. Despite that endothelial injury and bursts in ROS production are well known to manifest early to mediate pathological

Table 5
Treatment medications.

Antivirals	Male (%)	Female (%)	Total (%)	P value
Ganciclovir	2 (0.8)	0 (0.0)	2 (0.5)	0.505
Oseltamivir	4 (1.6)	3 (1.5)	7 (1.6)	1.000
Remdesivir	58 (23.8)	46 (23.5)	104 (23.6)	0.941
Valganciclovir	6 (2.5)	0 (0.0)	6 (1.4)	0.036*
Ribavirin	1 (0.4)	0 (0.0)	1 (0.2)	1.000
Hyperimmune Plasma				
Convalescent Plasma	19 (7.8)	23 (11.9)	42 (9.6)	0.151
RAS blockers				
ACE Inhibitor	29 (11.9)	11 (5.6)	40 (9.1)	0.023*
Angiotensin Receptor Blocker	20 (8.2)	13 (6.6)	33 (7.5)	0.536
Antithrombotics				
Argatroban	1 (0.4)	3 (1.5)	4 (0.9)	0.328
Continuous infusion of heparin	40 (16.5)	29 (14.9)	69 (15.8)	0.667
Low molecular heparin	124 (50.8)	102 (52.6)	226 (51.6)	0.715
Subcutaneous unfractionated heparin	73 (29.9)	56 (28.9)	129 (29.5)	0.810
Steroids				
Fludrocortisone Acetate	3 (1.2)	4 (2.1)	7 (1.6)	0.705
Inhaled Corticosteroid	13 (5.3)	15 (7.7)	28 (6.4)	0.321
Prednisolone	0 (0.0)	2 (1.0)	2 (0.5)	0.198
Non-Steroid Anti-Inflammatory drugs (NSAIDs)				
Aspirin	80 (32.8)	45 (23.0)	125 (28.4)	0.023*
Celecoxib	0 (0)	1 (0.5)	1 (0.2)	0.445
Diclofenac	3 (1.2)	2 (1.0)	5 (1.1)	1.000
Ibuprofen	23 (9.4)	21 (10.7)	44 (10.0)	0.654
Ketorolac Tromethamine	12 (4.9)	11 (5.6)	23 (5.2)	0.745
Meloxicam	1 (0.4)	0 (0.0)	1 (0.2)	1.000
Naproxen	1 (0.4)	1 (0.5)	2 (0.5)	1.000
Immunosuppressants				
Adalimumab	0 (0)	1 (0.5)	1 (0.2)	0.445
Azathioprine	0 (0)	1 (0.5)	1 (0.2)	0.445
Basiliximab	1 (0.4)	0 (0)	1 (0.2)	1.000
Chloroquine	2 (0.8)	0 (0)	2 (0.5)	0.505
Etanercept	1 (0.4)	0 (0)	1 (0.2)	1.000
Hydroxychloroquine	32 (13.1)	20 (10.2)	52 (11.8)	0.347
Leronlimab	34 (13.9)	15 (7.7)	49 (11.1)	0.037*
Methotrexate	0 (0)	4 (2.0)	4 (0.9)	0.039*
Mofetil	10 (4.1)	6 (3.1)	16 (3.6)	0.564
Mycophenolate	11 (4.5)	6 (3.1)	17 (3.9)	0.434
Rituximab	0 (0)	2 (1.0)	2 (0.5)	0.198
Sarilumab	10 (4.1)	2 (1.0)	12 (2.7)	0.049*
Sirolimus	2 (0.8)	0 (0)	2 (0.5)	0.505
Tacrolimus	19 (7.8)	13 (6.6)	32 (7.3)	0.643
Tocilizumab	19 (7.8)	7 (3.6)	26 (5.9)	0.062
Bronchodilator				
Inhaled beta-agonist	95 (38.9)	68 (34.7)	163 (37.0)	0.360
Vasopressors				
Dobutamine	0 (0)	1 (0.5)	1 (0.2)	0.445
Dopamine	7 (2.9)	6 (3.1)	13 (3.0)	0.906
Epinephrine	57 (23.4)	42 (21.4)	99 (22.5)	0.630
Milrinone	1 (0.4)	0 (0)	1 (0.2)	1.000
Phenylephrine	29 (11.9)	13 (6.6)	42 (9.5)	0.062
Vasopressin	25 (10.2)	15 (7.7)	40 (9.1)	0.347
Antibiotics				
Amikacin	6 (2.5)	1 (0.5)	7 (1.6)	0.139
Amoxicillin/Clavulanate	3 (1.2)	7 (3.6)	10 (2.3)	0.182
Amphotericin	2 (0.8)	0 (0.0)	2 (0.5)	0.505
Ampicilin/Sulbactam	5 (2.0)	3 (1.5)	8 (1.8)	1.000
Azithromycin	115 (47.1)	80 (40.8)	195 (44.3)	0.185

Table 5 (*continued*)

Antivirals	Male (%)	Female (%)	Total (%)	P value
Aztreonam	0 (0)	1 (0.5)	1 (0.2)	0.443
Cefazolin	11 (4.5)	11 (5.6)	22 (5.0)	0.597
Cefepime	45 (18.4)	30 (15.3)	75 (17.0)	0.385
Cefoxitin	1 (0.4)	0 (0)	1 (0.2)	1.000
Ceftriaxone	0 (0)	1 (0.5)	1 (0.2)	0.445

Table 6
Medications for patients received intense care.

	NO ICU (%)	ICU (%)	P Value
Antivirals			
Valganciclovir	1 (0.4)	5 (2.8)	0.042*
Remdesivir	49 (18.6)	55 (31.1)	0.003*
Hyperimmune Plasma			
Convalescent Plasma	5 (1.9)	37 (20.9)	0.000*
Antithrombotics			
Subcutaneous unfractionated heparin	55 (21.1)	74 (41.8)	0.000*
Continuous infusion of unfractionated heparin	17 (6.5)	52 (29.5)	0.000*
Low molecular heparin	125 (47.9)	101 (57.1)	0.060
Steroids			
Fludrocortisone acetate	1 (0.4)	6 (3.4)	0.019*
Non-steroid anti-inflammatory drugs (NSAIDS)			
Ketorolac tromethamine	19 (7.2)	4 (2.3)	0.022*
Immunosuppressants			
Hydroxychloroquine	21 (8.0)	31 (17.5)	0.002*
Tocilizumab	1 (0.4)	25 (14.1)	0.000*
Sarilumab	0 (0)	12 (6.8)	0.000*
Leronlimab	15 (5.7)	34 (19.2)	0.000*
Mycophenolate	5 (1.9)	12 (6.8)	0.009*
Mofetil	5 (1.9)	11 (6.2)	0.018*
Bronchodilator			
Inhaled Beta-Agonist	71 (27.0)	92 (52.0)	0.000*
Vasopressors			
Dopamine	0 (0)	13 (7.3)	0.000*
Epinephrine	9 (3.4)	90 (50.8)	0.000*
Phenylephrine	9 (3.4)	33 (18.6)	0.000*
Vasopressin	0 (0)	40 (22.6)	0.000*
Antibiotics			
Amikacin	1 (0.4)	6 (3.4)	0.019*
Azithromycin	97 (36.9)	98 (55.4)	0.000*
Cefepime	20 (7.6)	55 (31.1)	0.000*

progression of COVID-19 [7,16,24], direct, robust and clinically informative assays are to be established in the midst of respiratory pandemic now and beyond.

Immunosuppressants, NSAIDs and steroid were provided to rein in inflammatory disorders. The median of IL-6 levels remained in the normal range, while a significantly higher IL-6 was observed in the Washington cohort [36]. Of note, IL-6 plays an important role in the feed-forward activation of endothelial NADPH oxidase isoform 2 (NOX2), resulting in impairment of redox homeostasis and excessive superoxide production, and cascaded inflammatory responses [7]. The stats suggested that most COVID-19 patients sought professional cares days after initial onset of symptoms, so that effective care was given to potentially break the vicious cycles of cytokine storm. In addition, off-label use of immunosuppressants were practiced on a case by case basis. This included hydroxychloroquine, which had EUA at the time by FDA [41]. Precautions were also practiced to avoid potential tolerance (Leronlimab) or fertility impacts (Methotrexate, Sarilumab) of individual drugs. For patients with milder symptoms, argatroban and NSAIDs were selected as alternatives for the more aggressive approaches. The prescription on antivirals and the usage of covalescent plasma followed the same paradigm of prudence, including the compassionate use of

Table 7
Clinical outcomes.

At hospital admission	Male (%)	Female (%)	Total (%)	P value
CPR or intubation at hospital admission	40 (16.4)	27 (13.8)	67 (15.2)	0.477
No CPR and no intubation	204 (83.6)	169 (86.2)	373 (84.8)	
ICU admission				
ICU admission	103 (42.2)	74 (37.8)	177 (40.2)	0.343
No ICU	141 (57.8)	122 (62.2)	263 (59.8)	
Outcome				
Discharged alive	160 (65.6)	130 (66.3)	290 (65.9)	0.869
Hospitalized	6 (2.5)	4 (2.0)	10 (2.3)	1.000
Transfer to other facility	41 (16.8)	32 (16.3)	73 (16.6)	0.894
Death	22 (9.0)	14 (7.1)	36 (8.2)	0.476
Palliative discharge	1 (0.4)	2 (1.0)	3 (0.7)	0.588
Unkonwn	14 (5.7)	14 (7.1)	28 (6.4)	0.548
Transplant needed				
Heart	2 (8.7)	2 (13.3)	4 (10.5)	
Lung	6 (26.1)	2 (13.3)	8 (21.1)	
Kidney	7 (30.4)	9 (60.0)	16 (42.1)	
Liver	4 (17.4)	1 (6.7)	5 (13.2)	
Bone marrow or stem cell	1 (4.3)	1 (6.7)	2 (5.3)	
Other	1 (4.3)	0 (0)	1 (2.6)	
Multiple	2 (8.7)	0 (0)	2 (5.3)	
Total	23 (60.5)	15 (39.5)	38 (100)	0.510
ECMO	5 (2.0)	1 (0.5)	6 (1.4)	0.232

Table 8
IL-6 and clinical outcome.

	Normal IL-6	High IL-6	Total	P value
Count	381 (86.6)	59 (13.4)	440	
ICU				
NO ICU	247 (64.8)	16 (27.1)	263 (59.8)	0.000*
ICU	134 (35.2)	43 (72.9)	177 (40.2)	
Outcome				
Discharged alive	261 (68.5)	29 (49.2)	290 (65.9)	0.004*
Hospitalization	6 (1.6)	4 (6.8)	10 (2.3)	0.043*
Transfer to other facility	66 (17.3)	7 (11.9)	73 (16.6)	0.294
Death	24 (6.3)	12 (20.3)	36 (8.2)	0.001*
Palliative discharge	2 (0.5)	1 (1.7)	3 (0.7)	0.351
Unknown	22 (5.7)	6 (10.2)	28 (6.3)	0.317

Remdesivir [42].

Of note, 40.2 % of patients of this cohort were in need to intensive care. Seniors (over 70 years) faced significantly higher risks compared to the adult group (19–49 years) (percentage of prevalence: 28.0 % vs. 44 % for cohort patients vs. LA population for 19–49 years group, but 34.8 % vs. 10 % for over 70 years group respectively, Table 1). It was also noted that even though children (0–18 years) represents the smallest fraction of the cohort, a much higher proportion of it checked into ICU. This suggested that perhaps not all children received professional medical care before their symptoms turned severe. Public broadcasting at the time indicated that *'coronavirus was mysteriously sparing kids'* [43]. This could have been misleading, discouraging children from getting timely treatment when actually needed. BMI had a significant impact on the severity as well. Compared to patients with BMI ranging 18.5–25 (32/104, or 30.7 %), the overweight group (BMI 25–30) had a higher rate of ICU admission (37/92, or 40.2 %) and the obese group (BMI ≥30) had an even higher rate (59/125, 47.2 %) (Fig. 1B). It was noted that the underweight group (BMI <18.5) also had a higher rate of ICU admission (6/13, or 46.2 %). Among all ethnicities, Caucasians had a ICU admission rate at 30.1 % compared to 40.2 % of the whole cohort (Fig. 1D). The BMI mean of the Caucasian patients was at 25.75, significantly lower than the cohort mean of 28.75. Furthermore, the medians of AST, ALT and CK levels of Caucasian groups were at (31.0 IU/L, 24.0 IU/L and

84.5 IU/L) compared to the levels of non-Caucasian at (38.0 IU/L, 29.0 IU/L and 103.5 IU/L). In this cohort, Caucasian patients statistically had a better BMI profiles, which was associated with less significant tissue injuries and less severe overall symptoms.

In summary, our data fully characterize a local Californian cohort of COVID-19 at the initial phase of the break of the pandemic. Our observations indicate that population demographics as well as associated biophysical characters, comorbidities and resulting molecular pathological parameters have significant impacts on the etiology of a pandemic. As a novel virus metastasizes, patients will benefit greatly with early intervention, even before targeted medications or vaccines become available. In depth understanding of local population is the first step. Another COVID-like pandemic will certainly return [34]. Effectively managing acute injuries and re-purposing drugs will still be indispensable in the next campaign dealing with the new rising trend of COVID-19 in this season, and future break from another pathogen.

CRediT authorship contribution statement

Bo Zhou: Validation, Formal analysis, Data curation. **Nobel Chenggong Zong:** Writing – original draft, Validation, Formal analysis, Data curation. **Yuhan Zhang:** Writing – original draft. **Yuanli Huang:** Writing – original draft. **Ji-Youn Youn:** Formal analysis, Data curation. **Hua Cai:** Writing – review & editing, Writing – original draft, Validation, Supervision, Resources, Project administration, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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