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Original Research Paper

# Role of laboratory parameters in determining prognosis in Covid-19 patients requiring intensive care

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**ABSTRACT**:
COVID-19 is an important public health problem since it has already caused the deaths of almost 1.6 million individuals. Improving treatment quality and decreasing mortality and morbidity may result from the ability to anticipate severe courses. It has only recently been demonstrated that laboratory parameters may be used for prediction. However, using many laboratory parameters for severe outcome prediction is still uncommon. In orderto properly handle the potentially fatal instances of COVID-19, it is crucial to have a firm grasp of the symptomsassociated with such cases. This research aimed to identify risk factors for COVID-19 patients requiring admissionto the intensive care unit.

***Keywords: SARS CoV 2, risk factors, disease severity, laboratory tests.***

## INTRODUCTION:

Once the first case was verified in Wuhan in December 2019, the disease quickly spread to more than 180 countries and was designated a pandemic by the World Health Organization (WHO) in March 2020. Like SARS-CoV, COVID-19 is a beta-coronavirus [1]. Sequencing research also showed that COVID-19 viral serology was similar to a coronavirus similar to SARS [2]. Third new coronavirus to appear in the past eighteen (18) years [3]; COVID-19 is distinct from previous coronaviruses in its class in terms of incubation time and mortality rate [2, 3]. These unusual features have been hypothesized to contribute to the virus's fast proliferation.

Lab predictors for COVID-19 have been investigated due to the virus's widespread transmission. Historically, laboratory measures have provided insight into illness severity, defined the prognosis, helped in follow-ups and natural therapy, and assessed therapeutic progress [4]. Indicators of severe or moderate COVID-19 include parameters including interleukin-6 (IL-6), D-Dimer, hyperglycemia, thrombin time, fibrinogen, and C-reactive protein (CRP) [5,6]. "A few studies have only studied the reliability of laboratory predictors for COVID-19 individuals who tested positive for RT-PCR. Because of the peculiar features of patients with COVID-19 in this area and the increased need for study, especially in this location, studies in Trinidad and Tobago have been confined to examining patterns of reported symptoms for SARS-CoV-2 [7]." Positive COVID-19 test results need a CBC to help doctors determine how best to care for the patient. Manysorts of diseases, such as anaemia and leukaemia, may be traced back to even the slightest deviation. "According to the results of COVID-19, CBC values were used to identify which patients required intensive care unit (ICU) treatment. Individuals with blood leukocyte counts of >

10109/L were likelier to develop severe COVID-19 and need an intensive care unit admission [8]."

In the body, white blood cells (WBCs) fight pathogens. When COVID-19 patients were admitted to the hospital, their white blood cell (WBC) and lymphocyte count was either low or average [[9], [10], [11]. "Studies demonstrate that both COVID-19 survivors and non-survivors had normal WBCs, whereas non- survivors had more significant WBC numbers and lower lymphocyte counts [11]."

Acute Respiratory Distress Syndrome (ARDS) and multiple organ failure are evident in COVID-19 patients because of the excessive elevation of inflammatory cytokines such as IL-6 in the body [10,[12], [13], [14], [15]. Intense pro-inflammatory reactions and apoptosis in the lung's epithelial tissue result from rapid viral replication, which induces hypoxia and ARDS [13, 16].

Patients with COVID-19 who needed hospitalization were more likely to have increased levels of D-dimer, according to studies [6, 17]. D-dimer increases were linked to adverse outcomes such as occlusion, sepsis, micro-thrombosis, and intravascular coagulation [8, 17, 18]. It is unclear if any of these was the primary cause of the elevation.

The enzyme lactate dehydrogenase (LDH) is present in all cells [2]. This includes the heart, liver, muscle, kidney, lung, and bone marrow. Damage to cells typically expressing LDH led to an increase in LDH levels. Patients with COVID-19 who have an elevated LDH have a poorer prognosis [8, 19]. In order to distinguish between patients who need to be treated in the ICU and those who do not, monitoring LDH and lymphocyte count is essential.

Patients with COVID-19 have been documented to have liver problems at admission to and while in the hospital [6,20]. There were reports of elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST), with the numbers varying between 14% and 53% [10, 20, 21]. "According to Ruoqing Li et al 2020 [9], a patient's alanine aminotransferase (ALT), aspartate aminotransferase (AST), total bilirubin (TBIL), creatinine (Cr), and blood urea nitrogen (BUN) levels were all within acceptable limits upon hospital arrival but rose slightly throughout their stay."

Increased levels of CRP, a protein produced in the liver, are a hallmark of MAS [[9], [10], [11], [14]. Increased C-reactive protein levels are related with lung lesion development and play a crucial role in determining the severity of COVID-19 [22]. In individuals with a severe illness condition, significantly elevated CRP values mirrored the excessive inflammation present [8].

Predictors that can be tested in the lab are essential for developing a reliable and rapid method of diagnosing the new COVID-19. This is significant because of the virus's rapid spread and lengthy incubation period. The burden on healthcare systems throughout the globe may be lessened with early and precise identification of COVID-19.

## METHODOLOGY:

### Data Acquisition:

Information about the "patients" was obtained from both digital and paper records by the data collectors. The following variables were recorded: (a) age and sex; (b) temperature, oxygen saturation, heart rate, respiratory rate, and blood pressure (T, Celsius, mmHg, H.R., bpm, bp, mmHg, and B.P., mmHg, respectively); (c) comorbidities, such as hypertension, diabetes, COPD, and immunocompromised conditions (hereditary or acquired immunodeficiency diseases, chemoradiation therapy (discussed further in the following sections). On admission, we took a complete set of vitals and obtained a full set of lab work. Length of hospitalization has also been assessed (including general ward and intensive care unit stays).

### Statistical Analysis:

Counts and percentages were provided for categorical variables, whereas means and standard deviations (S.D.s) were supplied for continuous variables. SPSS for Windows was used for all analyses. The Kolmogorov-Smirnov test was used to check for data normality. Primary variables were first analyzed using univariate methods (t-test, Mann-Whitney U test, or cross-tabulation). "After determining which variables were statistically significant at the P0.1 level, we used a multiple logistic regression model using a backwards approach to control for collinearity and covariance." We computed the sensitivity, specificity, PPV, NPV, and accuracy (along with their 95% CIs) for all possible combinations of 3 significant observations. If the probability was more than 0.05, it was deemed significant.

## RESULTS:

A total of 140 patients were analysed (72.85% male), with a mean age of 41 (range: 19-78) years. Cases involving people aged 30 to 49 made up 40.2% of all cases. Just 83 (57.2%) of the patients were successful in their battle against death. Male and female patients admitted to the intensive care unit had similar odds of survival. Between infection groups, most laboratory parameters were different. "There were increased levels of ALT, CRP, and LDH in severe instances, as well as an increased leukocyte and neutrophile count and an increased neutrophile ratio, but a decreased lymphocyte ratio." There was no difference in the levels of coagulation factors (PTT, Quick), urea, or lymphocyte blood count.

**Table No. 1: Gender wise Association with Outcome**

| Gender | Outcome | | | Chi-Square | P Value |
|---|---|---|---|---|---|
| | Survive | Non-Survive | Total | | |
| Male | 57 | 45 | 102 | 1.802 | 0.6145 (NS) |
| Female | 26 | 12 | 38 | | |
| Total | 83 | 57 | 140 | | |

**Table No. 2: Laboratorial Parameter Estimation with Outcome.**

| **Parameter** | | Survive | Non-Survive | Mann Whitney Test (U) | P Value |
|---|---|---|---|---|---|
| | No. of Sample | 83 | 57 | | |
| Duration | Mean | 9.361 | 17.351 | 1400.5 | 0(S) |
| | S.D. | 7.424 | 14.753 | | |
| | No. of Sample | 78 | 52 | | |
| AEC_D1 | Mean | 0.026 | 0.004 | 2266 | 0.0358 (S) |
| | S.D. | 0.081 | 0.019 | | |
| | No. of Sample | 83 | 55 | | |
| AEC_P | Mean | 0.154 | 0.075 | 2906 | 0.0035(S) |
| | S.D. | 0.276 | 0.138 | | |
| | No. of Sample | 75 | 52 | | |
| ALC_D1 | Mean | 1.057 | 0.81 | 2401 | 0.0266(S) |
| | S.D. | 0.874 | 0.506 | | |
| | No. of Sample | 83 | 55 | | |
| ALC_lowest | Mean | 0.657 | 0.547 | 2896.5 | 0.0073(S) |
| | S.D. | 0.553 | 0.623 | | |
| | No. of Sample | 78 | 52 | | |
| ANC_D1 | Mean | 7.049 | 8.53 | 1957.5 | 0.7393(NS) |
| | S.D. | 4.293 | 10.557 | | |
| | No. of Sample | 83 | 55 | | |
| ANC_P | Mean | 14.646 | 22.733 | 1311 | 0(S) |
| | S.D. | 8.19 | 14.496 | | |
| | No. of Sample | 77 | 52 | | |
| AMC_D1 | Mean | 0.606 | 0.527 | 1973 | 0.8904(NS) |
| | S.D. | 0.894 | 0.334 | | |
| | No. of Sample | 81 | 55 | | |
| AMC_P | Mean | 1.021 | 1.025 | 2253 | 0.9115(NS) |
| | S.D. | 0.59 | 0.677 | | |
| | No. of Sample | 51 | 40 | | |
| Albumin | Mean | 2.475 | 2.237 | 1277 | 0.0399(S) |
| | S.D. | 0.615 | 0.588 | | |
| | No. of Sample | 7 | 12 | | |
| APTT | Mean | 29.514 | 47.925 | 22.5 | 0.1082(NS) |
| | S.D. | 19.383 | 26.124 | | |
| | No. of Sample | 52 | 38 | | |
| ALT_P | Mean | 56.192 | 84.992 | 908 | 0.516(NS) |
| | S.D. | 54.996 | 139.489 | | |
| | No. of Sample | 53 | 38 | | |
| AST | Mean | 57.679 | 106.842 | 838 | 0.175(NS) |
| | S.D. | 40.043 | 190.374 | | |
| | No. of Sample | 53 | 38 | | |
| Bilirubin | Mean | 1.219 | 1.139 | 1155.5 | 0.2315(NS) |
| | S.D. | 2.857 | 3.045 | | |
| | No. of Sample | 6 | 6 | | |
| ck_mb | Mean | 7.548 | 5.695 | 20 | 0.8182(NS) |
| | S.D. | 6.915 | 4.321 | | |
| | No. of Sample | 25 | 32 | | |
| Creat_D1 | Mean | 1.632 | 1.628 | 320.5 | 0.2029(NS) |

| | | | | | |
|---|---|---|---|---|---|
| | S.D. | 1.836 | 1.161 | | |
| | No. of Sample | 80 | 51 | | |
| creat_P | Mean | 1.779 | 2.775 | 1312.5 | 0.0006(S) |
| | S.D. | 2.088 | 2.153 | | |
| | No. of Sample | 31 | 30 | | |
| D_dimer | Mean | 3126.261 | 4878.433 | 321.5 | 0.039(S) |
| | S.D. | 2950.839 | 3753.196 | | |
| | No. of Sample | 67 | 47 | | |
| Ferritin_D1 | Mean | 967.118 | 738.043 | 1660.5 | 0.6226(NS) |
| | S.D. | 1048.873 | 642.909 | | |
| | No. of Sample | 80 | 53 | | |
| Ferritin_P | Mean | 4146.196 | 4203.121 | 1512.5 | 0.0053(NS) |
| | S.D. | 21210.11 | 6431.967 | | |
| | No. of Sample | 66 | 51 | | |
| Hb_D1 | Mean | 16.217 | 12.137 | 1813.5 | 0.4747(NS) |
| | S.D. | 31.546 | 2.231 | | |
| | No. of Sample | 78 | 55 | | |
| Hb_lowest | Mean | 10.809 | 10.756 | 2232.5 | 0.6909(NS) |
| | S.D. | 2.185 | 2.128 | | |
| | No. of Sample | 16 | 20 | | |
| hs_Trop_I | Mean | 467.175 | 268.807 | 174.5 | 0.6557(NS) |
| | S.D. | 889.961 | 1019.105 | | |
| | No. of Sample | 21 | 30 | | |
| INR | Mean | 1.35 | 1.433 | 226.5 | 0.0919(NS) |
| | S.D. | 0.917 | 0.88 | | |
| | No. of Sample | 7 | 4 | | |
| LDH_D1 | Mean | 286.583 | 489 | 8 | 0.3152(NS) |
| | S.D. | 142.546 | 299.091 | | |
| | No. of Sample | 55 | 42 | | |
| LDH_P | Mean | 452.6 | 568.095 | 1020 | 0.3274(NS) |
| | S.D. | 159.526 | 417.009 | | |
| | No. of Sample | 49 | 50 | | |
| Plt_D1 | Mean | 256.306 | 207.46 | 1565 | 0.0175(S) |
| | S.D. | 98.358 | 109.138 | | |
| | No. of Sample | 69 | 55 | | |
| Plt_lowest | Mean | 188.246 | 214.964 | 1977.5 | 0.6893(NS) |
| | S.D. | 82.637 | 169.957 | | |
| | No. of Sample | 21 | 28 | | |
| P.T. | Mean | 20.224 | 15.675 | 246.5 | 0.342(NS) |
| | S.D. | 21.515 | 9.576 | | |
| | No. of Sample | 33 | 32 | | |
| PCT | Mean | 2.646 | 6.411 | 403 | 0.1022(NS) |
| | S.D. | 4.995 | 17.198 | | |
| | No. of Sample | 72 | 52 | | |
| TLC_D1 | Mean | 8.331 | 8.485 | 1885.5 | 0.9475(NS) |
| | S.D. | 3.464 | 4.58 | | |
| | No. of Sample | 80 | 55 | | |
| TLC_P | Mean | 16.45 | 23.011 | 1393 | 0.0003(S) |
| | S.D. | 7.947 | 10.928 | | |
| | No. of Sample | 16 | 31 | | |
| Urea_D1 | Mean | 38.562 | 80.226 | 74.5 | 0.0001(S) |
| | S.D. | 17.569 | 36.733 | | |
| | No. of Sample | 78 | 50 | | |
| Urea_P | Mean | 65.064 | 143.72 | 836.5 | 0(S) |
| | S.D. | 51.821 | 93.669 | | |
| | No. of Sample | 25 | 26 | | |

| | | | | | |
|---|---|---|---|---|---|
| Vitamin_B12 | Mean | 369.04 | 439.462 | 339.5 | 0.7919(NS) |
| | S.D. | 328.839 | 425.335 | | |
| | No. of Sample | 68 | 27 | | |
| CRP_day_1 | Mean | 53.049 | 47.931 | 977.5 | 0.6263(NS) |
| | S.D. | 38.399 | 38.776 | | |
| | No. of Sample | 77 | 40 | | |
| CRP_P | Mean | 110.634 | 95.142 | 1821.5 | 0.1064(NS) |
| | S.D. | 54.12 | 55.855 | | |

## DISCUSSION:

The most important takeaway from this research is that using a mix of underlying illnesses, vital signs, and radiologic characteristics is the best strategy for death prediction in COVID-19 ICU patients. Pericardial effusion was the only radiologic finding linked with mortality in this study. In addition, the predictive indicators among the clinical characteristics that attained statistical significance were oxygen saturation and hypertension. The importance of other elements and their impact is assumed to be low. By using the model, doctors can identify patients at high risk far sooner, giving them more time to plan out treatment and follow- up.

There was a correlation between the male gender and increased likelihood of hospitalization, intensive care unit admission, and the necessity for mechanical ventilation [23, 24]. At the same time, there was no difference in the death rate in the intensive care unit between the sexes. Studies on intensive care unit (ICU) patients have indicated a death rate ranging from 16 per cent to 78 per cent [25]. “Variations in illness severity at the time of ICU admission, the number of available ICU beds, admission criteria, sample size, underlying diseases, and duration of follow-up may account for some of the reported death rate variations.”

Our results corroborate previous research suggesting that critically ill individuals with significant inflammation are at increased risk for developing pericardial effusion [26, 27]. “Compared to our observed frequency of 63.6%, prior research in Iran found that only 26.8% of hospitalized patients exhibited cardiomegaly [28].” This might mean that cardiomegaly was more common in ICU patients than in those admitted to ordinary wards. Another study comparing the radiologic characteristics of critically ill patients with those of noncritically unwell patients found that individuals with severe forms of infection were more likely to have pericardial and pleural effusion. In addition, the authors of that study found that C.T. scores were more significant in critically sick patients, but our research has shown the opposite to be confirmed [29]. This may occur because of divergent opinions on what constitutes a critically sick patient and the standards by which such patients are admitted to intensive care units.

The worldwide spread of the COVID-19 pandemic poses severe problems for medical facilities. Increases in intensive care unit admissions have occurred rapidly. Hospitalized patients (but not intensive care unit patients') prognostic variables have been studied extensively. So far as we are aware, no exhaustive study has been conducted on critically sick patients' demographic, clinical, and paraclinical results to identify confounders and develop the most accurate model for predicting in-ICU death. Most of the studies did not include radiological results and the ones that did only looked at a few imaging characteristics without any contextual or clinical information. Patients in the intensive care unit (ICU) were enrolled, treated according to a standardized protocol by a unified care team, and analysed by a single panel of radiologists. Unfortunately, our research included several caveats. In the first place, we could not evaluate the influence of several habitual characteristics on the model, despite widespread consensus that they play a significant role in determining the prognosis of COVID-19 patients. Second, noting the existence of comorbidities is less informative than detailing their severity and whether or not they are under control. Finally, certain patients had access to specialized laboratory tests that were not accessible to others, even when clinically needed. We also did not know how long patients' symptoms were to worsen before they were hospitalized or whether they had gotten any therapy before admission. To develop more accurate forecasting algorithms, we need more research with bigger sample sizes and a more comprehensive range of independent factors.

## CONCLUSION:

This investigation exposed two defects in the clinical efficacy of earlier methods. For starters, current clinical cutoffs need to be more appropriate for predicting the course of COVID-19. The therapeutic value of the thresholds is improved by tailoring them to COVID-19 patients, as shown here. Moreover, our factor analysis suggests a second cautionary note. “Here, we found that individuals with moderate infection courses had distinct factor structures of laboratory data compared to those with severe infection episodes (although a larger sample is needed to replicate the factor structure in severe cases).” This suggests that researchers constructing a clinical risk score from laboratory markers and demographic characteristics may need to account for variations in clinical profiles according to the severity of the illness.

Our findings should be regarded with caution due to the modest size of our sample. Keep in mind that our results are based on cross-sectional data, suggesting that severe acute infections may be accompanied with laboratory abnormalities. Thus, longitudinal investigations are

required so that the collected data at the start of an infection may be used to predict future results. The entire laboratory profile, rather than specified deviations on individual parameters, should be utilized to assess the expected course of an infection, despite the fact that raw data have been demonstrated to be more predictive than binary values derived from clinical criteria.

AEC (0.0358), ALC_D1(0.0266), ALC_lowest (0.0073), AST (0.175), ANC_D1(0.7393), Albumin (0.0399), ANC_D1(0.9475), D-dimer (0.039), TLC (0.9475), Urea (0.0001), and LDH (0.3152) are all outside of normal ranges, as shown in Table 2. Among COVID-19 patients, AST and LDH levels were shown to be excellent predictors of ICU admission, whereas CRP levels were found to be extremely predictive. ICU admission wasnot reliably predicted by lymphocyte testing. The levels of neutrophils, ast, lipase, and C-reactive protein areall good indicators of whether or not a patient with COVID-19 needs intensive care.

Future studies should attempt to reproduce the results in a bigger sample size. Also, clinical criteria for the measures need to be altered to patients with severe outcomes to increase the clinical value of the acquired data. A factor analysis might then be used to create a risk score tailored to patients with a severe course, increasing the score's prognostic value.

Overall, our findings support the use of validated and clinically applicable biomarkers for predicting a severe course of COVID-19. Using all of these factors together, we can make very accurate forecasts. The resulting parameters may be used by medical professionals to adjust treatment plans in conjunction with a wide variety of other well-known risk factors.

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