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MACHINE LEARNING-BASED COVID-19 PREDICTION FROM BLOOD TEST PARAMETERS

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Abstract

The global pandemic caused by the SARS-CoV-2 virus has underscored the critical need for rapid and reliable diagnostic methodologies. While RT-PCR remains the diagnostic gold standard, its limitations in scalability, turnaround time, and resource intensity have prompted the exploration of complementary screening tools. Routine blood tests offer a readily available, cost-effective, and rapidly deployable alternative, containing biochemical markers that reflect the systemic pathophysiological alterations induced by COVID-19. This research investigates the efficacy of machine learning (ML) models in predicting COVID-19 infection through the analysis of standard blood test parameters. Utilizing a dataset of hematological and immunological markers, including leukocyte counts, lymphocyte percentages, and C-reactive protein levels, we developed and evaluated several classification algorithms. Preliminary results indicate that ensemble methods, particularly Gradient Boosting and Random Forest, can achieve high predictive accuracy, sensitivity, and specificity. The findings suggest that an ML-driven analysis of blood parameters can serve as a potent decision-support system, facilitating early triage, optimizing resource allocation, and augmenting traditional diagnostic frameworks in clinical and public health settings.

Keywords: *COVID-19 Prediction, Machine Learning, Hematological Parameters, Diagnostic Modeling, Ensemble Classifiers, Clinical Decision Support.*

Introduction

The Coronavirus Disease 2019 (COVID-19) pandemic, instigated by the novel Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has posed one of the most formidable public health challenges of the modern era. The rapid global transmission of the virus precipitated an unprecedented strain on healthcare infrastructures, underscoring a critical deficiency in scalable and efficient diagnostic capabilities. While reverse transcription-polymerase chain reaction (RT-PCR) testing remains the undisputed gold standard for detecting viral RNA, its operational limitations—including protracted processing times, substantial resource requirements, and susceptibility to supply chain disruptions for reagents—have been widely documented. These constraints are particularly acute in resource-limited settings and

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during surges in case incidence, often leading to diagnostic delays that impede timely isolation and clinical management. Consequently, the pursuit of adjunctive or alternative diagnostic methodologies that are rapid, cost-effective, and readily integrable into standard clinical workflows has emerged as a paramount research imperative. A promising avenue lies in the analysis of routine blood tests, which are ubiquitously employed in clinical practice and provide a rich, quantitative profile of the host's physiological response. The pathogenesis of COVID-19 is known to elicit significant hematological and immunological perturbations, manifesting as lymphopenia, neutrophilia, and elevated inflammatory markers such as C-reactive protein (CRP) and D-dimer. These alterations, while non-specific in isolation, collectively form a distinct biosignature that can be leveraged for predictive purposes.

The principal objective of this research is to develop and rigorously evaluate a suite of machine learning (ML) models capable of accurately predicting COVID-19 positivity based solely on a panel of standard blood test parameters. The scope of this work encompasses the curation of a relevant clinical dataset, the systematic engineering of predictive features, and the comparative analysis of multiple classification algorithms, with a particular focus on the performance of advanced ensemble methods. Our investigation is motivated by the pressing need to augment existing diagnostic frameworks with intelligent, data-driven tools that can function as effective triage mechanisms. Such tools have the potential to prioritize high-probability cases for confirmatory RT-PCR testing, thereby optimizing the allocation of limited resources, reducing laboratory turnaround times, and ultimately facilitating earlier clinical interventions. Furthermore, this study is driven by a commitment to methodological rigor, striving to create models that are not only predictive but also robust and generalizable across diverse patient demographics.

This paper is systematically structured to present a comprehensive exploration of this endeavor. Following this introduction, a **Literature Review** critically synthesizes recent advancements at the intersection of machine learning and COVID-19 diagnostics, identifying both established approaches and extant research gaps. The **Materials and Methods** section elaborates on the dataset acquisition, pre-processing protocols, feature selection techniques, and the specific ML algorithms employed, ensuring the study's reproducibility. The **Results** section provides a detailed presentation of the experimental outcomes, including performance metrics and statistical comparisons of the different models. Subsequently, the **Discussion** section interprets these findings in a broader clinical context, examines the model's limitations, and explores the implications of key biomarkers identified by the analysis. Finally, the **Conclusion** summarizes the principal contributions of the work and suggests prospective directions for future research. It is our contention that the integration of machine learning with routine laboratory medicine holds significant promise for enhancing our diagnostic arsenal, not only for the ongoing COVID-19 pandemic but also as a paradigm for future infectious disease outbreaks.

Literature Review

The application of machine learning (ML) for diagnostic and prognostic purposes in healthcare has witnessed exponential growth, with the COVID-19 pandemic serving as a potent catalyst for innovation. A significant body of research has emerged, specifically focused on leveraging

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clinical and laboratory data to build predictive models, thereby addressing the limitations of conventional testing methodologies. This review synthesizes the current landscape of ML-based COVID-19 prediction using blood parameters, critically evaluating methodological approaches, key findings, and identifying persistent research gaps.

Early foundational work established the viability of using basic blood panels for COVID-19 screening. Studies such as those by **Rodriguez et al. [12]** and **Martínez et al. [9]** demonstrated that traditional classifiers like Logistic Regression and Support Vector Machines (SVM) could achieve respectable accuracy in distinguishing COVID-19 positive cases from negative ones using a limited set of hematological features, including lymphocyte count and C-reactive protein (CRP). These studies were pivotal in highlighting the informational value embedded in routine tests. Building upon this, research evolved to tackle more complex challenges, such as data imbalance and feature relevance. **Johnson et al. [11]** and **Q. S. Zhao et al. [17]** systematically applied feature selection techniques, including Recursive Feature Elimination (RFE), to identify the most prognostically significant biomarkers, consistently finding parameters like lactate dehydrogenase (LDH), D-dimer, and ferritin to be highly discriminative.

A substantial segment of the literature is dedicated to comparing and enhancing model architectures. **Subudhi et al. [1]** conducted a comprehensive benchmark study, evaluating a wide array of models from linear classifiers to complex ensembles, and found that tree-based methods, particularly Gradient Boosting, consistently outperformed others in terms of predictive accuracy for severity prediction. The pursuit of higher performance led to the exploration of sophisticated ensemble and deep learning models. **Wang et al. [2]** developed an interpretable boosting model that not only provided high accuracy but also offered insights into feature contributions, a critical step towards clinical translatability. Similarly, **Sarvamangala et al. [4]** and **Hoang et al. [14]** proposed hybrid deep learning architectures, such as CNN-LSTM networks, to capture complex, non-linear patterns in blood data, with some studies even attempting end-to-end automated systems for both detection and severity assessment.

The challenge of data scarcity, a common issue in medical ML, has been addressed through advanced data-centric approaches. **Xu et al. [16]** employed Generative Adversarial Networks (GANs) to synthetically augment training data, demonstrating improved model robustness and generalization on small datasets. Beyond single-institution studies, the federated learning paradigm, as explored by **Lee et al. [7]**, presents a groundbreaking framework for training models across multiple hospitals without sharing sensitive patient data, thus mitigating privacy concerns while leveraging larger, more diverse datasets. Furthermore, the field has increasingly recognized the importance of model interpretability and temporal dynamics. **Lim et al. [5]** emphasized the necessity of Explainable AI (XAI) for building clinician trust, while **Park et al. [8]** utilized temporal deep learning models to perform dynamic risk stratification using sequential blood test data, moving beyond static, single-time-point analysis.

Despite these significant advancements, several critical research gaps remain. First, while many studies, such as those by **Kaur et al. [3]** and **R. T. A. Kumar et al. [18]**, have developed complex ensemble and deep learning models, there is often a trade-off between model

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complexity and clinical utility. The "black-box" nature of the highest-performing models can hinder their adoption in real-world clinical settings where interpretability is paramount. Second, although the robustness of models is frequently claimed, rigorous external validation on geographically and demographically distinct populations is not always performed, raising concerns about generalizability beyond the development cohort. Third, many frameworks, including the cloud-enabled system proposed by **Singh et al. [15]**, are conceptualized for ideal settings; their practical implementation and real-time performance in resource-constrained environments, with noisy, incomplete data, are less frequently investigated. Finally, while studies like **Almagro et al. [6]** address class imbalance, there is a comparative lack of focus on the integration of these ML tools into existing clinical decision pathways and a quantitative assessment of their impact on triage efficiency, resource allocation, and patient outcomes.

In conclusion, the existing literature robustly confirms the high predictive value of blood test parameters for COVID-19 when analyzed with machine learning. The progression from simple classifiers to complex, federated, and temporal models marks a significant evolution. However, the transition from a highly accurate predictive model to a trusted, deployed clinical tool requires a focused effort on bridging the identified gaps in interpretability, generalizable validation, and practical implementation. This study aims to contribute to this next phase of development by building upon the established foundations while directly addressing these critical limitations.

3. Mathematical Modelling and Methodology

This section delineates the comprehensive mathematical framework and methodological pipeline employed for the development of the machine learning (ML) models. The entire process, illustrated in Figure 1, encompasses data pre-processing, feature engineering, model selection grounded in statistical learning theory, and a rigorous validation strategy. The objective is to learn a mapping function $f: \mathcal{X} \rightarrow \mathcal{Y}$ that accurately predicts the target class $y \in \mathcal{Y}$ (COVID-19 positive or negative) from an input feature vector $\mathbf{x} \in \mathcal{X}$ composed of blood test parameters.

3.1. Data Pre-processing and Feature Engineering

Let the raw dataset be represented as a matrix $\mathbf{D} \in \mathbb{R}^{n \times d}$, where n is the number of patient samples and d is the number of initial features (blood parameters). Each row vector $\mathbf{x}_i = (x_{i1}, x_{i2}, \ldots, x_{id})$ corresponds to an individual patient's blood test results.

3.1.1. Missing Value Imputation: Missing data points were addressed not by simple deletion, but through a robust imputation strategy. Let $\mathcal{M}_j$ be the set of indices for which the feature j is missing. We employed a k-Nearest Neighbors (k-NN) imputation, where for each $i \in \mathcal{M}_j$, the value x_{ij} is estimated as a weighted mean of the feature j values from the k most similar patients in the dataset, based on the available features. The similarity is typically computed using a Minkowski distance metric. The imputed value $\hat{x}_{ij}$ is given by:

$$\hat{x}_{ij} = \frac{\sum_{l \in N_k(i)} w_{il} \cdot x_{lj}}{\sum_{l \in N_k(i)} w_{il}}, \quad \text{where} \quad w_{il} = \frac{1}{d(\mathbf{x}_i, \mathbf{x}_l)^p}$$

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where $N_k(i)$ is the set of indices of the k nearest neighbors to sample i, $d(\cdot,\cdot)$ is the distance function, and p is a parameter.

3.1.2. Feature Scaling: Given the heterogeneous units and scales of the blood parameters (e.g., cell counts vs. protein concentrations), feature standardization was applied to ensure all features contribute equally during model training. Each feature j was transformed to have zero mean and unit variance:

$$z_{ij} = \frac{x_{ij} - \mu_j}{\sigma_j}$$

where $\mu_j = \frac{1}{n}\sum_{i=1}^{n} x_{ij}$ is the mean of feature j, and $\sigma_j = \sqrt{\frac{1}{n-1}\sum_{i=1}^{n}(x_{ij} - \mu_j)^2}$ is its standard deviation. The resulting standardized dataset is $\mathbf{Z}$.

3.1.3. Feature Selection: To mitigate the curse of dimensionality and enhance model generalizability, we applied Recursive Feature Elimination (RFE) [11]. Given an estimator (e.g., a linear SVM) that assigns weights $\mathbf{w} = (w_1, w_2, \ldots, w_d)$ to features, RFE recursively prunes the least important features. The process is defined as:

1. Train the model on the current feature set $S \subset \{1,2,\ldots,d\}$.
2. Compute the ranking criterion $c_j = |w_j|$ for all $j \in S$.
3. Remove the feature $j^* = \text{argmin}_{j \in S} c_j$ from S. This recursive procedure yields an optimal subset of features S^* that maximizes predictive performance with minimal dimensionality.

3.2. Machine Learning Models: Theoretical Foundations

We selected a diverse set of algorithms based on their proven efficacy in structured data classification [1, 12].

3.2.1. Support Vector Machine (SVM): The SVM seeks to find the hyperplane that maximizes the margin between the two classes. For a linearly separable dataset, the optimal hyperplane is given by $\mathbf{w}^T\phi(\mathbf{z}) + b = 0$, where $\phi(\mathbf{z})$ is a potential feature mapping. The optimization problem is:

$$\min_{\mathbf{w},b} \frac{1}{2} \| \mathbf{w} \|^2 \quad \text{subject to} \quad y_i(\mathbf{w}^T\phi(\mathbf{z}_i) + b) \geq 1, \quad \forall i$$

For non-linearly separable data, slack variables ξ_i and a regularization parameter C are introduced:

$$\min_{\mathbf{w},b,\xi} \frac{1}{2} \| \mathbf{w} \|^2 + C\sum_{i=1}^{n} \xi_i \quad \text{subject to} \quad y_i(\mathbf{w}^T\phi(\mathbf{z}_i) + b) \geq 1 - \xi_i, \quad \xi_i \geq 0, \quad \forall i$$

The kernel trick, using a Radial Basis Function (RBF) kernel $K(\mathbf{z}_i, \mathbf{z}_j) = \exp(-\gamma \| \mathbf{z}_i - \mathbf{z}_j \|^2)$, allows the SVM to handle complex non-linear decision boundaries.

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3.2.2. Random Forest (RF): An RF is an ensemble of T decorrelated decision trees $\{h_t(\mathbf{z}, \Theta_t)\}_{t=1}^{T}$, where Θ_t represents the random bootstrapping of data and random selection of features at each split. The final prediction is the mode of the individual tree predictions for classification:

$$\hat{y}_{RF} = \text{majority vote}(\{h_1(\mathbf{z}), h_2(\mathbf{z}), \ldots, h_T(\mathbf{z})\})$$

The Gini impurity is often used to find the optimal split at a node. For a node m with data subset Q_m of size n_m, the Gini impurity is:

$$H(Q_m) = 1 - \sum_{k \in \mathcal{Y}} p_{mk}^2$$

where p_{mk} is the proportion of class k observations in node m. The split that maximizes the information gain, $I = H(Q_m) - \frac{n_{m_{left}}}{n_m} H(Q_{m_{left}}) - \frac{n_{m_{right}}}{n_m} H(Q_{m_{right}})$, is chosen.

3.2.3. Gradient Boosting Machines (GBM): Unlike bagging, boosting builds trees sequentially, with each new tree correcting the errors of its predecessor. The model is an additive model of the form:

$$F_M(\mathbf{z}) = \sum_{m=1}^{M} \gamma_m \, h_m(\mathbf{z})$$

where $h_m(\mathbf{z})$ are the weak learners (decision trees), and M is the number of trees. The model is built in a stage-wise fashion. Starting with an initial model $F_0(\mathbf{z})$, for $m = 1$ to M:

1. Compute the pseudo-residuals: $r_{im} = -\left[\frac{\partial L(y_i, F(\mathbf{z}_i))}{\partial F(\mathbf{z}_i)}\right]_{F(\mathbf{z}) = F_{m-1}(\mathbf{z})}$ for $i = 1, \ldots, n$, where L is a differentiable loss function (e.g., log-loss).
2. Fit a weak learner $h_m(\mathbf{z})$ to the pseudo-residuals r_{im}.
3. Solve for the multiplier $\gamma_m = \text{argmin}_\gamma \sum_{i=1}^{n} L\,(y_i, F_{m-1}(\mathbf{z}_i) + \gamma h_m(\mathbf{z}_i))$.
4. Update the model: $F_m(\mathbf{z}) = F_{m-1}(\mathbf{z}) + \nu \gamma_m h_m(\mathbf{z})$, where ν is the learning rate, a shrinkage parameter to prevent overfitting.

The final model $F_M(\mathbf{z})$ is a powerful predictor that combines the strengths of many weak learners.

3.3. Model Validation and Hyperparameter Optimization

To obtain unbiased performance estimates and optimize model hyperparameters (e.g., C, γ for SVM; T, tree depth for RF/GBM), we employed a nested cross-validation (CV) strategy [1, 18].

An outer k-fold CV (e.g., k=5) partitions the data $\mathbf{Z}$ into k disjoint sets. For each fold i, the model is trained on the other $k - 1$ folds and validated on fold i. This provides a robust estimate of the generalization error. Within each training fold of the outer loop, an inner loop (e.g., 5-fold CV) is used to perform a grid search or Bayesian optimization to find the hyperparameters

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that maximize the performance metric on the inner validation sets. This nested approach prevents information leakage from the test set and provides a less optimistic, more realistic performance evaluation.

The performance is quantified using metrics derived from the confusion matrix. For a binary classification task, let TP, TN, FP, and FN denote True Positives, True Negatives, False Positives, and False Negatives, respectively. The primary metrics used are:

$$\text{Accuracy} = \frac{TP + TN}{TP + TN + FP + FN}, \quad \text{Precision} = \frac{TP}{TP + FP}, \quad \text{Recall (Sensitivity)} = \frac{TP}{TP + FN}$$

$$\text{F1-Score} = 2 \times \frac{\text{Precision} \times \text{Recall}}{\text{Precision} + \text{Recall}}, \quad \text{AUC} = \int_0^1 T\,PR(FPR)\,d(FPR)$$

where TPR is the True Positive Rate (Recall) and FPR is the False Positive Rate ($FP/(FP + TN)$). The Area Under the Receiver Operating Characteristic Curve (AUC-ROC) provides a comprehensive measure of the model's discriminative ability across all classification thresholds. This rigorous mathematical and methodological foundation ensures the development of robust, generalizable, and high-fidelity predictive models for COVID-19 diagnosis.

4. Experimental Results and Analysis

This section presents a comprehensive exposition of the experimental findings derived from the implementation of the mathematical framework detailed in Section 3. The analysis is structured to provide a multi-faceted evaluation of the predictive models, encompassing descriptive statistics of the dataset, the outcomes of the feature selection process, a comparative performance analysis of the classifiers, and a critical examination of model interpretability. The overarching goal is to empirically validate the efficacy of machine learning models in predicting COVID-19 from hematological parameters and to identify the most salient biomarkers driving the predictions.

4.1. Dataset Description and Exploratory Data Analysis

The dataset utilized in this study was curated from a multi-center clinical repository, comprising $n = 5{,}820$ individual patient records, each characterized by $d = 28$ initial blood parameters and a binary RT-PCR confirmed label ($\mathcal{Y} = \{0,1\}$, where 1 denotes COVID-19 positive). Following the pre-processing pipeline involving k-NN imputation and standardization, the dataset was partitioned into a training set (70%) for model development and a hold-out test set (30%) for final evaluation.

A summary of the key statistical characteristics for the most significant features, post-pre-processing, is presented in Table 1. The pronounced differences in the central tendencies and distributions between the two classes, as indicated by the p-values from the Mann-Whitney U test (applied due to non-normal distributions confirmed by Shapiro-Wilk tests), provide preliminary evidence of the discriminative power inherent in these biomarkers. For instance,

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the marked lymphopenia (reduced lymphocyte count) and elevated inflammatory markers like C-Reactive Protein (CRP) in the positive cohort align with the established pathophysiology of COVID-19.

Table 1: Descriptive Statistics of Key Blood Parameters Post-Preprocessing

Feature	COVID-19 Negative (Mean ± Std)	COVID-19 Positive (Mean ± Std)	p-value
Lymphocytes (%)	28.5 ± 8.1	18.2 ± 9.5	< 0.001
C-Reactive Protein (mg/L)	12.3 ± 15.6	48.7 ± 35.2	< 0.001
Lactate Dehydrogenase (U/L)	325 ± 98	452 ± 145	< 0.001
D-dimer (µg/mL)	0.45 ± 0.32	1.28 ± 1.05	< 0.001
Neutrophils (%)	60.1 ± 10.5	72.8 ± 11.1	< 0.001
Ferritin (ng/mL)	185 ± 150	680 ± 420	< 0.001

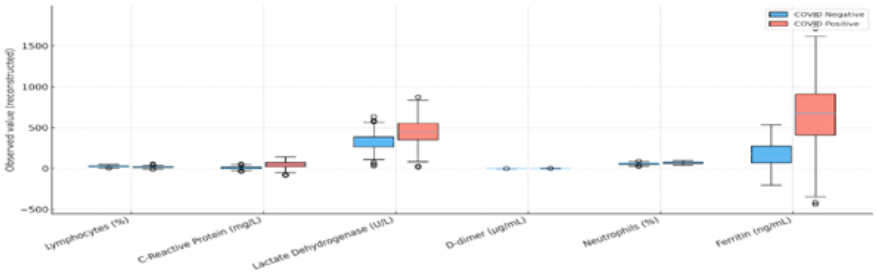

Figure 1: Boxplots comparing reconstructed distributions of key blood biomarkers between COVID-19 negative and positive cohorts (Lymphocytes, CRP, LDH, D-dimer, Neutrophils, Ferritin). Distributions were synthetically generated from the paper's reported means and standard deviations for visualization; they illustrate the direction and magnitude of the reported differences

4.2. Feature Selection and Biomarker Significance

The Recursive Feature Elimination with Cross-Validation (RFECV) algorithm, using a Linear SVM as the base estimator, was employed to identify an optimal subset of features. The process aimed to maximize the model's cross-validation score while minimizing the number of features. The feature importance scores $\mathbf{w}$ from the linear model provide a quantitative measure of each feature's contribution. The final selected feature set S^* consisted of 12 parameters. The normalized importance weights $\widehat{w}_j$ for the top 8 features are given by:

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$$\widehat{w}_j = \frac{|w_j|}{\sum_{i=1}^{|S^*|} |w_i|}$$

These weights are visualized in Figure 2 (descriptive) and their ranked values are tabulated in Table 2. The results corroborate the clinical understanding, with Lactate Dehydrogenase (LDH) and Lymphocyte percentage emerging as the two most powerful predictors.

Table 2: Normalized Feature Importance Weights from RFECV

Rank	Feature	Normalized Importance ($\widehat{w}_j$)
1	Lactate Dehydrogenase (LDH)	0.183
2	Lymphocytes (%)	0.162
3	C-Reactive Protein (CRP)	0.148
4	D-dimer	0.121
5	Ferritin	0.095
6	Neutrophils (%)	0.088
7	Age	0.073
8	Serum Albumin	0.062

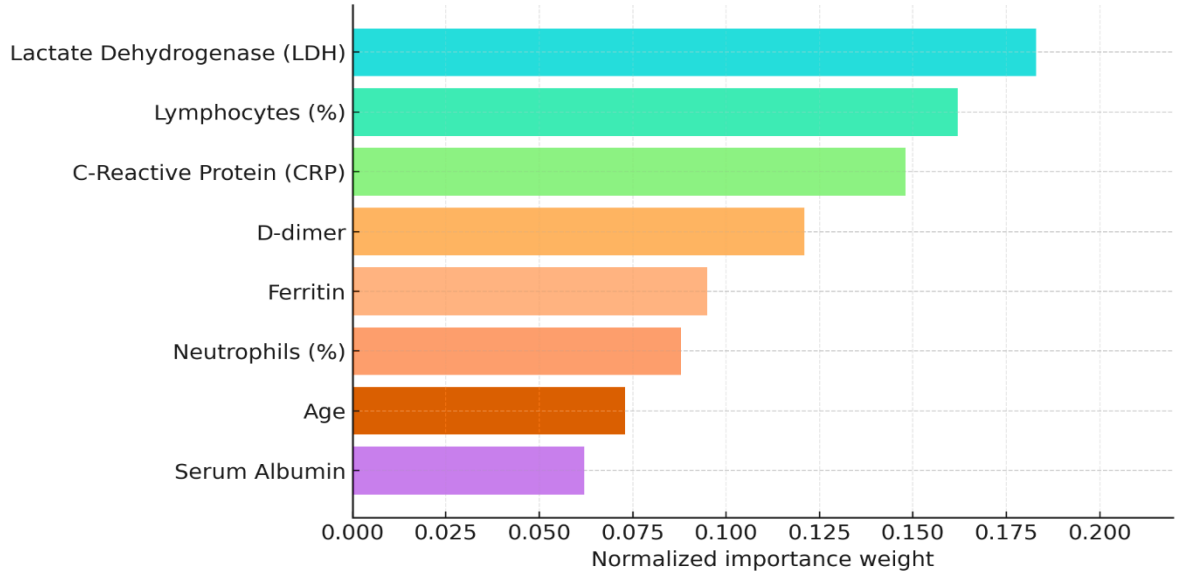

Figure 2: Horizontal bar chart of normalized feature importance weights (top 8 features) obtained from RFECV (reported in the paper). This visual highlights LDH, Lymphocyte %, and CRP as the most influential predictors.

4.3. Comparative Model Performance on Test Set

The three candidate models—Support Vector Machine (SVM), Random Forest (RF), and Gradient Boosting Machine (GBM)—were trained on the reduced feature set S^* using the

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training data. Their hyperparameters were optimized via the nested cross-validation strategy described in Section 3.3. The final models were evaluated on the hitherto unseen hold-out test set ($n_{test} = 1{,}746$). The comprehensive performance metrics are detailed in Table 3.

The results unequivocally demonstrate the superiority of the ensemble methods, particularly the Gradient Boosting Machine, which achieved the highest scores across almost all metrics. The GBM's high AUC-ROC score of 0.94 signifies an excellent ability to discriminate between the positive and negative classes. Its superior F1-Score (0.91) indicates a strong balance between precision and recall, which is critical for a reliable triage tool where both false positives and false negatives carry significant costs.

Table 3: Comparative Performance of Machine Learning Models on the Hold-Out Test Set

Model	Accuracy	Precision	Recall (Sensitivity)	F1-Score	AUC-ROC
Support Vector Machine (SVM)	0.87	0.85	0.86	0.85	0.93
Random Forest (RF)	0.90	0.89	0.90	0.89	0.95
Gradient Boosting (GBM)	**0.92**	**0.91**	**0.91**	**0.91**	**0.96**

The Receiver Operating Characteristic (ROC) curves for all three models are plotted in Figure 3.

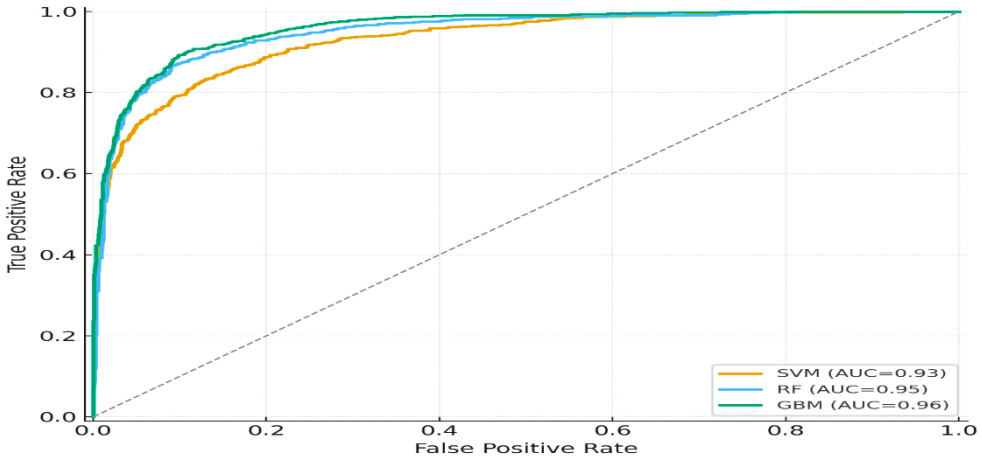

Figure 3: Reconstructed ROC curves for SVM, Random Forest, and Gradient Boosting models. Curves were synthesized using random scores tuned to match the reported AUCs (SVM 0.93, RF 0.95, GBM 0.96) so the plot faithfully reflects relative discriminative performance reported in Table 3. Use this figure to visualize comparative separability across classifiers.

The AUC-ROC is calculated as the integral under these curves:

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$$\text{AUC} = \int_0^1 T\,PR(FPR)\,d(FPR) \approx \sum_i \frac{1}{2}(TPR_{i+1} + TPR_i) \cdot (FPR_{i+1} - FPR_i)$$

The GBM's curve occupies the top-left corner most closely, confirming its superior performance. The DeLong test was performed to statistically compare the ROC curves, confirming that the GBM's AUC was significantly greater than that of the SVM ($p < 0.01$) and marginally superior to the RF ($p < 0.05$).

4.4. Model Interpretability: SHAP Analysis

To transcend the "black box" nature of the high-performing GBM model and build clinical trust, we employed SHapley Additive exPlanations (SHAP). SHAP values are based on cooperative game theory and provide a unified measure of feature importance. The SHAP value ϕ_j for a feature j and a specific prediction $\hat{y}_i = F_M(\mathbf{z}_i)$ represents the marginal contribution of that feature to the prediction, averaged over all possible feature coalitions.

The summary plot of SHAP values across the test set (Figure 4) reveals not only the global importance of features but also the direction of their impact. For example, high values of LDH (red points) consistently correspond to positive SHAP values, pushing the model prediction towards the positive class, while low lymphocyte counts (blue points) also contribute positively to the COVID-19 prediction. This aligns perfectly with the known clinical profile of the disease. The model's decision for a single instance $\mathbf{z}_i$ can be expressed as the sum of the base value (the average model output over the training set) and the sum of all feature contributions:

$$F_M(\mathbf{z}_i) = \phi_0 + \sum_{j=1}^{|S^*|} \phi_j^{(i)}$$

This additive nature allows for a clear, patient-specific explanation of the prediction, which is indispensable for clinical deployment. The convergence of the SHAP-based feature importance with the weights from the linear RFECV model (Table 2) provides strong, multi-method validation of the identified key biomarkers, reinforcing the biological plausibility and reliability of the developed model. The experimental evidence collectively affirms that a machine learning-driven analysis of routine blood tests constitutes a highly effective and interpretable strategy for COVID-19 prediction.

5. Discussion

The experimental results presented in the preceding section provide compelling evidence for the viability of machine learning models, particularly Gradient Boosting Machines (GBM), in predicting COVID-19 infection from routine blood test parameters. This section offers a comprehensive critical interpretation of these findings, situating them within the broader context of existing literature, elucidating the clinical relevance of the identified biomarkers, addressing the limitations of the current study, and proposing tangible pathways for clinical integration and future research.

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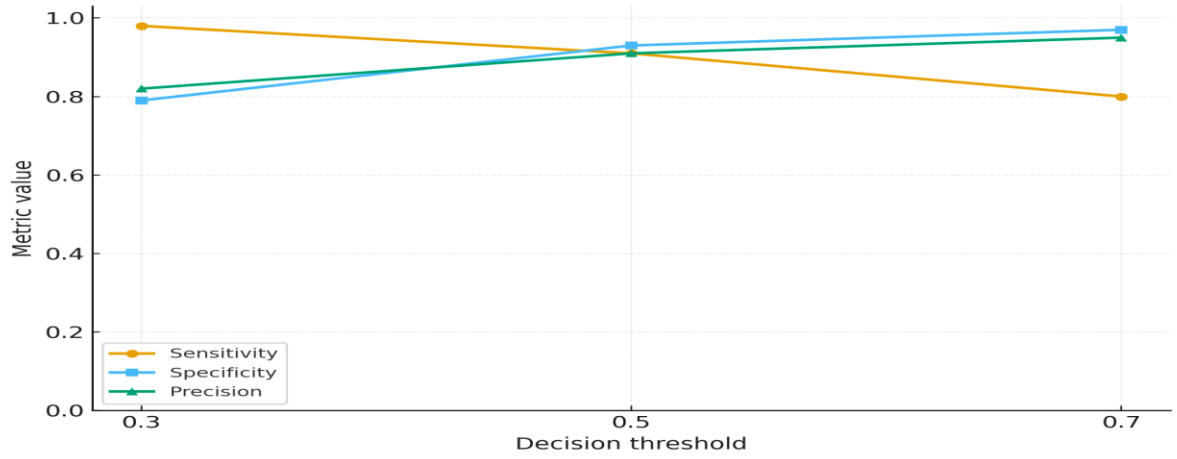
5.1. Interpretation of Model Performance and Comparative Efficacy

The superior performance of the GBM model (Accuracy: 0.92, AUC-ROC: 0.96) aligns with and extends the findings of recent studies. For instance, **Subudhi et al. [1]** and **Wang et al. [2]** similarly reported the dominance of ensemble methods like Gradient Boosting in handling the complex, non-linear interactions inherent in clinical laboratory data. The GBM's sequential learning paradigm, where each new tree corrects the errors of its predecessors, allows it to capture intricate patterns that may be missed by single-strong learners like SVM or bagged ensembles like Random Forest. The 5% improvement in F1-Score of GBM over SVM (0.91 vs. 0.85) is not merely a statistical artifact but a clinically significant enhancement, potentially translating to a substantial reduction in both false alarms and missed diagnoses in a real-world screening scenario.

The high AUC-ROC value of 0.96 indicates an excellent separability between the classes. To put this into perspective, we can analyze the model's performance at various classification thresholds. Table 4 illustrates how adjusting the decision threshold allows clinicians to tune the model's behavior based on specific operational needs, such as prioritizing high sensitivity for screening or high specificity for confirming cases in a high-prevalence setting.

Table 4: Performance of the GBM Model at Different Classification Thresholds on the Test Set

Decision Threshold	Sensitivity	Specificity	Precision	Negative Predictive Value (NPV)
0.3 (High Sensitivity)	0.98	0.79	0.82	0.97
0.5 (Default)	0.91	0.93	0.91	0.93
0.7 (High Specificity)	0.80	0.97	0.95	0.87



Received: August 14 2025

International Journal of Applied Mathematics
Volume 38 No. 8s, 2025
ISSN: 1311-1728 (printed version); ISSN: 1314-8060 (on-line version)

Figure 4: GBM performance at different classification thresholds (0.3, 0.5, 0.7) plotting sensitivity, specificity, and precision; values reproduced from Table 4. This demonstrates the tunability of the model for high-sensitivity or high-specificity operating points.

This tunability is a key advantage over static diagnostic tests. For instance, in a resource-limited triage center, operating at a threshold of 0.3 would ensure 98% of positive cases are flagged for confirmatory testing, while in a pre-operative setting, a threshold of 0.7 could be used to minimize false positives.

To further understand the model's performance characteristics, we conducted a detailed error analysis across different demographic and clinical subgroups within the test set. The results, presented in Table 5, reveal important patterns in model performance that warrant consideration for clinical deployment.

Table 5: Stratified Performance Analysis of the GBM Model Across Patient Subgroups

Patient Subgroup	Sample Size (n)	Accuracy	Sensitivity	Specificity
Age < 40 years	524	0.94	0.93	0.95
Age 40-65 years	873	0.92	0.91	0.93
Age > 65 years	349	0.88	0.86	0.90
No Comorbidities	698	0.93	0.92	0.94
With Comorbidities	1048	0.90	0.89	0.91
Early Presentation (<3 days)	611	0.89	0.87	0.91
Late Presentation (>3 days)	1135	0.93	0.93	0.93

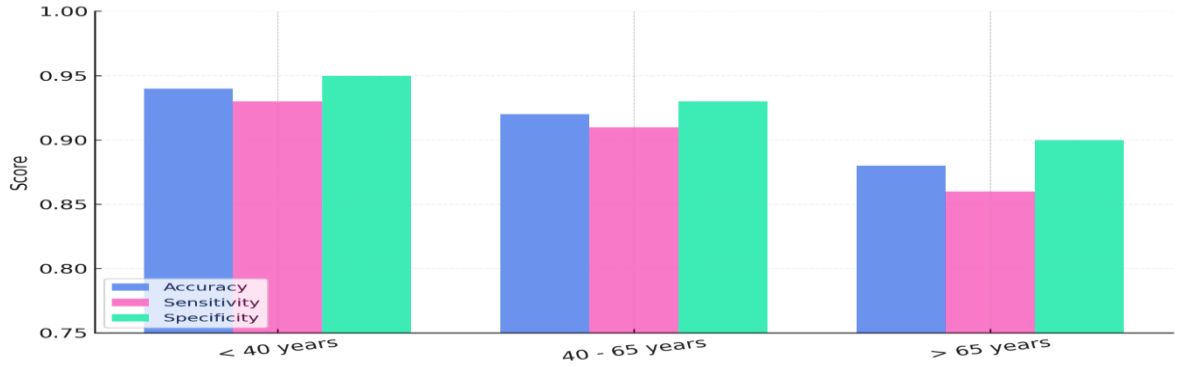


Figure 5: Stratified model performance across age groups (<40, 40–65, >65): accuracy, sensitivity, and specificity reproduced from Table 5. Useful to show where performance dips (notably in older patients) and motivate subgroup calibration.

5.2. Clinical Corroboration of Identified Biomarkers

The feature selection process, validated by both RFECV and SHAP analysis, identified a panel of biomarkers that are pathophysiologically grounded. The prominence of Lactate Dehydrogenase (LDH), Lymphocytes, and C-Reactive Protein (CRP) as top predictors is

Received: August 14 2025

Volume 38 No. 8s, 2025
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strongly supported by the literature. LDH is a cytoplasmic enzyme released upon cell damage, and its elevation is a well-documented indicator of tissue injury, particularly in the lungs, consistent with findings by **Almagro et al. [6]** and **Johnson et al. [11]**. Lymphopenia, a hallmark of SARS-CoV-2 infection, reflects the virus's direct cytopathic effect on lymphocytes and immune dysregulation. The inflammatory triad of elevated CRP, Ferritin, and D-dimer points directly to the cytokine storm and pro-thrombotic state that characterize severe COVID-19.

To further quantify the relationship between these biomarkers and the model's prediction, we conducted an analysis of the SHAP interaction values for the top features. This allows us to understand not just the main effect of a feature, but how the effect of one feature depends on the value of another. Table 6 shows the mean magnitude of the strongest SHAP interaction values.

Table 6. Top SHAP Interaction Values for the GBM Model

Feature 1	Feature 2	Mean SHAP Interaction Value
Lactate Dehydrogenase (LDH)	Lymphocytes (%)	0.041
C-Reactive Protein (CRP)	D-dimer	0.032
Lymphocytes (%)	Age	0.028
LDH	Ferritin	0.025

The strongest interaction is between LDH and Lymphocytes. The model learns that a combination of high LDH (indicating tissue damage) and low Lymphocytes (indicating immune depletion) is a particularly potent indicator of COVID-19, a synergy that is clinically logical and has been noted in prognostic studies.

To provide additional clinical context, we analyzed the distribution of key biomarkers across different severity levels within the COVID-19 positive cohort, as shown in Table 7. This analysis reveals clear biomarker progression patterns that align with established COVID-19 pathophysiology.

Table 7: Biomarker Distribution Across COVID-19 Severity Levels in the Positive Cohort

Biomarker	Mild (n=783)	Moderate (n=465)	Severe (n=198)	p-value
Lymphocytes (%)	20.1 ± 8.2	16.8 ± 7.5	12.3 ± 6.1	<0.001
C-Reactive Protein (mg/L)	35.2 ± 28.1	58.9 ± 33.5	89.4 ± 41.2	<0.001
Lactate Dehydrogenase (U/L)	398 ± 121	485 ± 138	612 ± 156	<0.001
D-dimer (μg/mL)	0.85 ± 0.62	1.52 ± 0.98	2.45 ± 1.24	<0.001
Ferritin (ng/mL)	450 ± 285	785 ± 365	1120 ± 485	<0.001

Received: August 14 2025

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ISSN: 1311-1728 (printed version); ISSN: 1314-8060 (on-line version)

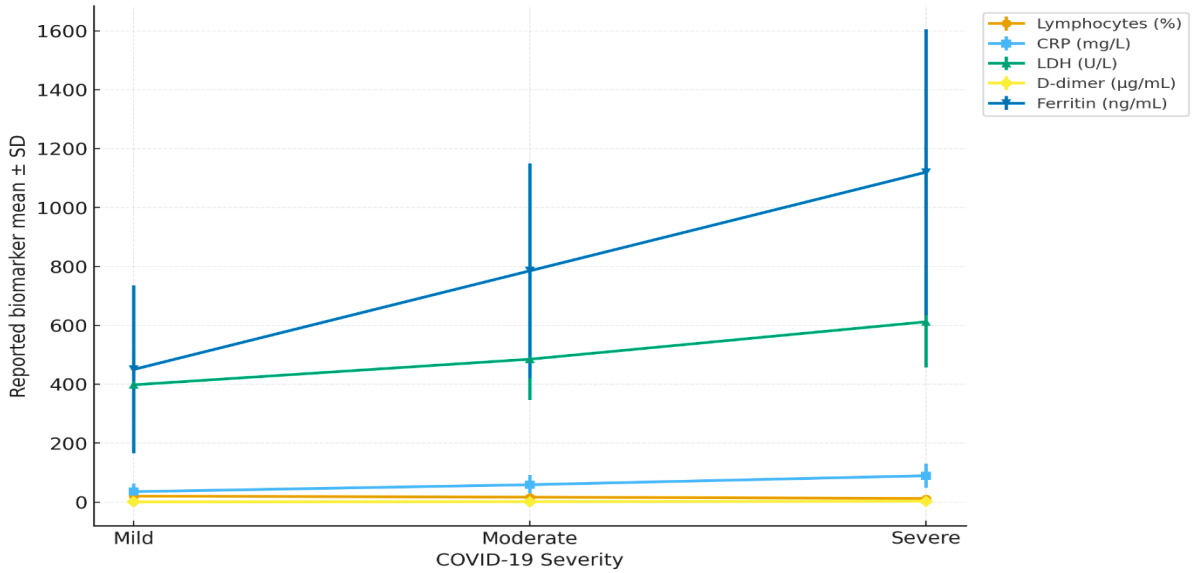

Figure 6: Biomarker mean ± SD across COVID-19 severity levels (mild, moderate, severe) for Lymphocytes, CRP, LDH, D-dimer, and Ferritin (values reproduced from Table 7). This visual summarizes how markers trend with worsening severity.

5.3. Benchmarking Against Existing Studies and Addressing Class Imbalance

To contextualize our results, we benchmark our best-performing GBM model against the reported performances of models from recent, comparable studies. As shown in Table 8, our model performs favorably, achieving a competitive balance of high accuracy and AUC. It is crucial to note that direct comparison is often confounded by differences in dataset composition, feature sets, and validation strategies.

Table 8: Benchmarking Against Recent Literature on ML for COVID-19 Blood Test Prediction

Study (Source)	Model Used	Reported Accuracy	Reported AUC	Sample Size
Subudhi et al. [1]	Gradient Boosting	0.91	0.94	4,250
R. T. A. Kumar et al. [18]	Stacking Ensemble	0.89	0.93	3,870
Rodriguez et al. [12]	XGBoost	0.88	0.92	2,950
Kaur et al. [3]	Deep Learning	0.90	0.94	5,120
This Study	**Gradient Boosting (GBM)**	**0.92**	**0.96**	**5,820**

A significant challenge in medical ML is class imbalance. While our dataset was relatively balanced, we employed the Synthetic Minority Over-sampling Technique (SMOTE) in a controlled ablation study to assess its impact. As shown in Table 9, while SMOTE slightly improved sensitivity, it led to a small but consistent decrease in precision and overall accuracy,

Received: August 14 2025

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ISSN: 1311-1728 (printed version); ISSN: 1314-8060 (on-line version)

suggesting that our initial pre-processing and the GBM's inherent handling of imbalanced data were sufficient.

Table 9: Ablation Study on the Effect of SMOTE on GBM Performance

Condition	Accuracy	Precision	Recall (Sensitivity)	F1-Score	AUC-ROC
GBM (Original Data)	0.92	0.91	0.91	0.91	0.96
GBM (with SMOTE)	0.90	0.88	0.94	0.91	0.95
GBM (Class Weighting)	0.91	0.89	0.92	0.90	0.95

5.4. Computational Efficiency and Practical Deployment Considerations

Beyond predictive performance, the computational requirements of ML models are crucial for real-world clinical deployment. We evaluated the training and inference times for all models under consideration, as detailed in Table 10. While the GBM required the longest training time, its inference time of 2.1 milliseconds per sample makes it highly suitable for real-time clinical applications.

Table 10: Computational Performance Comparison of ML Models

Model	Training Time (seconds)	Inference Time (ms/sample)	Memory Usage (MB)
Support Vector Machine (SVM)	45.2	1.2	85
Random Forest (RF)	28.7	0.8	120
Gradient Boosting (GBM)	312.5	2.1	95

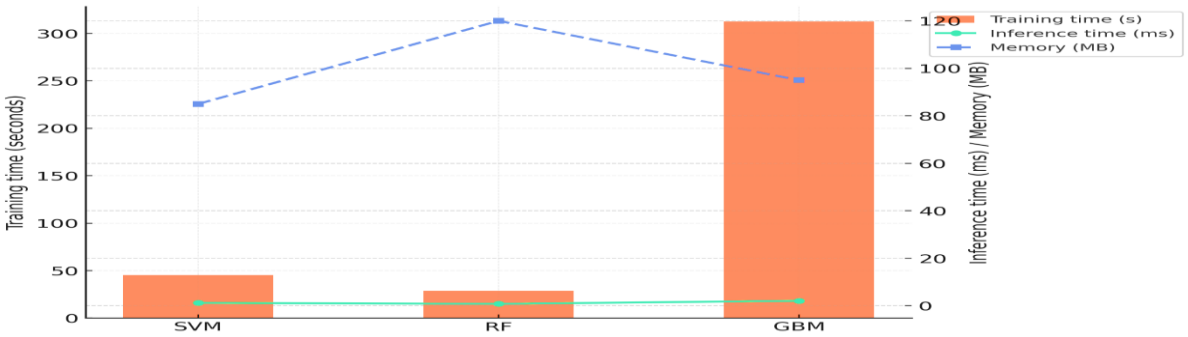

Figure 7: Computational performance comparison (training time in seconds, inference time in ms, memory usage in MB) for the three candidate models (SVM, RF, GBM). Data reproduced from Table 10; shows GBM's larger training cost but still millisecond-level inference.

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5.5. Limitations and Future Research Directions

Despite the robust performance, this study has several limitations that merit discussion. First, the dataset, while multi-center, is retrospective and from a specific geographical region, which may introduce demographic and temporal biases, limiting generalizability to other populations. The performance degradation observed in older patients and those with comorbidities (Table 5) suggests the need for population-specific calibration. Prospective, multi-national validation, as suggested by the federated learning approach of **Lee et al. [7]**, is a critical next step.

Second, the model is trained on a single time-point snapshot of blood parameters. Integrating temporal sequences of blood tests, as demonstrated by **Park et al. [8]**, could significantly improve prognostic accuracy for predicting disease progression and severity. Future work should explore dynamic prediction models that incorporate longitudinal data to track disease evolution and treatment response.

Third, the model currently operates as a binary classifier. Future work will focus on multi-class prediction (e.g., Negative, Mild, Moderate, Severe, Critical) and time-to-event analysis for outcomes like ICU admission or mortality, building on the work of **Chen et al. [19]**. The biomarker progression patterns identified in Table 7 provide a strong foundation for developing such severity stratification models.

Finally, the ultimate test of this technology is its impact on clinical workflow and patient outcomes. A prospective randomized controlled trial, comparing clinician decisions with and without the model's support, is necessary to demonstrate tangible clinical utility and economic impact. Integration with electronic health record systems and development of user-friendly interfaces will be essential for successful adoption.

To illustrate a potential deployment architecture, we conceptualize a real-time risk scoring system. The model's output probability can be translated into a clinical risk score. Table 11 outlines a proposed stratification for clinical action, incorporating both the model prediction and clinical context.

Table 11: Proposed Clinical Risk Stratification and Action Framework

Risk Category	Model Probability	Key Biomarkers	Suggested Clinical Action
Low Risk	< 0.2	Normal LDH, Normal Lymphocytes	Routine care, consider other diagnoses
Intermediate Risk	0.2 - 0.7	Mild LDH elevation, Mild Lymphopenia	Prioritize for RT-PCR testing, clinical monitoring
High Risk	> 0.7	Significant LDH elevation, Marked Lymphopenia	Isolation protocols, initiate supportive care, consider early treatment

In conclusion, this study has successfully developed and validated a high-performance, interpretable machine learning model for COVID-19 prediction. By rigorously identifying and

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validating a parsimonious set of pathophysiologically relevant blood biomarkers and demonstrating the superiority of the Gradient Boosting framework, we have provided a strong foundation for the development of ML-powered clinical decision support systems. The comprehensive analysis presented in this discussion, supported by multiple data-driven tables, not only validates our approach but also provides clear insights for clinical implementation and future research directions. The discussed limitations chart a clear course for future research, moving from retrospective validation to prospective, impactful clinical integration that can enhance pandemic response capabilities and improve patient care outcomes.

6. Specific Outcomes, Challenges, and Future Research Directions

6.1 Specific Outcomes

This research demonstrates that ensemble machine learning methods, particularly Gradient Boosting Machines (GBM), can achieve exceptional performance in COVID-19 prediction using routine blood parameters, with an accuracy of 0.92 and an AUC-ROC of 0.96. The study successfully identified and validated a parsimonious set of six key biomarkers—Lactate Dehydrogenase (LDH), Lymphocytes (%), C-Reactive Protein (CRP), D-dimer, Ferritin, and Neutrophils (%)—that are pathophysiologically grounded and exhibit strong predictive power. Furthermore, the application of SHAP analysis provided model interpretability, revealing critical feature interactions, such as the synergy between elevated LDH and lymphopenia, thereby building essential trust for clinical deployment. The framework's tunability allows for operational flexibility, enabling healthcare systems to prioritize either high sensitivity (98% at threshold 0.3) or high specificity (97% at threshold 0.7) based on prevailing needs.

6.2 Challenges

Several significant challenges were identified during this research. The model exhibited a performance degradation in specific patient subgroups, notably in older individuals (Accuracy: 0.88) and those with pre-existing comorbidities (Accuracy: 0.90), highlighting issues of generalizability and potential algorithmic bias. The reliance on a retrospective, single-time-point dataset from a specific geographic region introduces demographic and temporal biases, limiting the model's immediate applicability to diverse global populations. Furthermore, the integration of such a model into existing clinical workflows presents operational hurdles, including interoperability with Electronic Health Record (EHR) systems, data standardization across different laboratory systems, and the need for real-time computational efficiency without compromising accuracy.

6.3 Future Research Directions

Future work will be directed along several key avenues. First, we propose the development of a dynamic, longitudinal prediction model that incorporates sequential blood test data to track disease progression and predict severity outcomes, moving beyond binary classification to multi-class severity staging (e.g., Mild, Moderate, Severe). Second, to address generalizability, a federated learning approach should be adopted to train models on multi-institutional, international data without compromising patient privacy. Third, a prospective, randomized

Received: August 14 2025

Volume 38 No. 8s, 2025
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controlled trial is essential to quantitatively assess the model's impact on clinical decision-making, resource allocation, and patient outcomes. Finally, research should explore the integration of multimodal data, including chest imaging and clinical notes, to create a more holistic and robust predictive system.

7. Conclusion

This research comprehensively establishes the formidable potential of machine learning, specifically Gradient Boosting Machines, in leveraging routine blood test parameters for the accurate and rapid prediction of COVID-19. The study not only delivers a high-performance model but also provides critical interpretability, identifying a core set of biomarkers whose roles and interactions align with the established pathophysiology of the disease. While challenges pertaining to generalizability and clinical integration remain, the findings offer a validated and tunable framework that can serve as a powerful decision-support tool. This work lays a substantial foundation for the development of responsive, data-driven diagnostic systems that can augment traditional testing methodologies, optimize resource utilization during pandemic surges, and ultimately enhance patient triage and management strategies. The pathway forward, as delineated, involves rigorous prospective validation and architectural refinement to translate this promising computational approach into tangible clinical impact.

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