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Meta-Analysis

Role of Inflammatory Markers in Severity, ICU Admission, and Mortality in COVID-19: A Systematic Review and Meta-analysis of 79,934 Patients

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Abstract

Introduction

Despite extensive investigations into the roles of inflammatory biomarkers in the prognosis of COVID-19 through systematic reviews and meta-analyses, they are limited by small sample sizes and focus on a specific marker. This meta-analysis investigated the role of 11 inflammatory biomarkers in severity, intensive care unit (ICU) admission, and mortality among COVID-19 patients.

Methods

Studies up to October 25, 2023, were identified through a search of Google Scholar, limited to human studies published in English. Inclusion criteria required confirmed COVID-19 cases diagnosed via reliable laboratory methods, original articles from eligible journals, proper grouping of severity status, ICU admission, or mortality outcomes, and presentation of continuous data in mean and standard deviation, median with range, or interquartile range.

Results

A total of 241 studies, comprising 79,934 cases of COVID-19, were included in this study. Albumin levels significantly declined in severe, ICU, and dead cases compared to mild, moderate, non-ICU, and survived cases ($p<0.001$). C-reactive protein (CRP), D-dimer, erythrocyte sedimentation rate (ESR), ferritin, fibrinogen, Interleukin-6 (IL-6), lactate dehydrogenase (LDH), neutrophil-to-lymphocyte ratio (NLR), procalcitonin, and white blood cell (WBC) were all significantly ($p<0.001$) increased and correlated with the severity of COVID-19. CRP, D-dimer, ferritin, fibrinogen, IL-6, LDH, NLR, procalcitonin, and WBC were all significantly ($p<0.05$) elevated and correlated with the risk of ICU admission (except fibrinogen) and mortality in COVID-19 in both fixed and random effects.

Conclusion

Inflammatory biomarkers like albumin, CRP, D-dimer, ferritin, IL-6, LDH, NLR, procalcitonin, and WBC all significantly impact severity status, ICU admission, and mortality in COVID-19.

1. Introduction

The coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has emerged as a global pandemic with significant morbidity and mortality [1]. As of March 17, 2024, there were over 774 million confirmed COVID-19 cases, with seven million deaths. In February 2024 alone, 252,585 new cases were reported, with 5,272 reported deaths [2]. This infectious disease presents a spectrum of clinical manifestations, ranging from asymptomatic or mild symptoms to severe respiratory distress and multi-organ failure [3]. Approximately 70–80% of patients are expected to experience a variety of short- or long-term post-infection complications, particularly in cases of severe COVID-19 [3,4]. Inflammatory biomarkers (IBs) have garnered significant attention in understanding the pathogenesis and prognosis of COVID-19 [5]. Biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and ferritin play crucial roles in reflecting the inflammatory response and disease severity [6]. Other markers like D-dimer, neutrophil-to-lymphocyte ratio (NLR), and procalcitonin have shown promise in predicting disease progression and outcomes [7]. The prognosis of COVID-19 is influenced by various factors, including patient demographics, comorbidities, and disease severity [8]. Identifying risk factors associated with disease severity and mortality is essential for guiding clinical management and resource allocation [9]. Investigating IBs in COVID-19 patients can be vital for early risk stratification, informing treatment decisions, and improving patient outcomes [10]. Increasing evidence suggests that systemic inflammation contributes to COVID-19 progression by triggering the release of pro-inflammatory cytokines. Consequently, interventions aimed at suppressing inflammatory responses may hold promise in the management of severe cases of COVID-19. Despite extensive investigations into the roles of IBs in the prognosis of COVID-19 through systematic reviews and meta-analyses, they are limited by small sample sizes and focus on a specific marker [11-15]. This meta-analysis investigated the role of 11 IBs in severity, intensive care unit (ICU) admission, and mortality among COVID-19 patients.

2. Methods

2.1 Data sources and search strategy

The systematic review and meta-analysis adhered to the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines. Studies published until October 25, 2023, were identified via a comprehensive search of Google Scholar. The studies should have investigated the correlation between levels of IBs and COVID-19 severity, ICU admission, and mortality. The search strategy included the following keywords: ("Inflammatory biomarker" OR "Inflammatory biomarkers" OR "Inflammatory marker" OR "Inflammatory markers" OR "C reactive protein" OR "C reactive proteins" OR CRP OR "Erythrocyte sedimentation rate" OR ESR OR Procalcitonin OR PCT OR "Serum amyloid" OR "Serum amyloids" OR Cytokines OR Cytokine OR "Alpha 1 acid glycoprotein" OR "Plasma viscosity" OR Ceruloplasmin OR Hepcidin OR Haptoglobin OR "Tumor Necrosis Factor" OR "Tumor Necrosis Factors" OR TNFα OR Interleukin OR Interleukins OR IL OR "Interferon gamma" OR "IFN γ" OR Fibrinogen OR Immunoglobulin OR Immunoglobulins OR IgM OR Albumin OR "White blood cell" OR "White blood cells" OR WBC OR Eosinophils OR Eosinophil OR Basophils OR Basophil OR Neutrophil OR Neutrophils OR "complete blood count" OR "complete blood counts" OR CBC OR "lactate dehydrogenase" OR LDH OR "D dimer" OR "D dimers" OR Ferritin OR Ferritins OR Calprotectin OR Interferon OR Interferons) AND ("SARS CoV 2" OR "COVID-19" OR "Coronavirus 2019" OR "2019-nCoV") AND (mortality OR morbidity OR outcome OR survival OR survive OR analysis OR evaluation OR severity OR complication OR complications OR hospitalization OR ICU OR "intensive care unit" OR death OR fatality OR comorbidity OR comorbidities OR risk OR risks). The search was restricted to human studies published in the English language.

2.2 Eligibility criteria

The inclusion criteria of the study encompassed: 1) Confirmed COVID-19 cases diagnosed via reverse transcriptase-polymerase chain reaction (RT-PCR) or other reliable laboratory methods, 2) Original articles published in eligible journals, 3) Assessment of IBs such as albumin, CRP, D-dimer, erythrocyte sedimentation rate (ESR), ferritin, fibrinogen, IL-6, lactate dehydrogenase (LDH), NLR, procalcitonin, serum amyloid, and white blood cell (WBC), 4) Identification and proper grouping of severity status, ICU admission, or mortality outcomes, 5) Presentation of quantitative or continuous data in mean and standard deviation, median with range, or interquartile range. Excluded studies involved pediatric populations, investigating other diseases besides COVID-19, review articles, studies deriving sample sizes from datasets or popular databases, and those published in warning journal lists.

2.3 Study selection process

Three researchers initially reviewed the titles and abstracts of the identified studies, followed by full-text screening based on predetermined eligibility criteria. Subsequently, eligible studies were chosen for inclusion. In cases of disagreement, a fourth

author intervened to facilitate resolution through deliberation and discourse.

2.4 Data extraction

Two authors extracted data, which was blindly reviewed by a third author for accuracy. The extracted data from each study encompassed various parameters, including the first author's surname, publication year, study country, design, sample size, demographic characteristics (such as mean age and gender distribution), prevalent comorbidities, extent of clinical severity, ICU admission status, outcomes, and levels of IBs presented as mean with standard deviation, median with range, or interquartile ranges for different groups (mild, moderate, severe, ICU, non-ICU, survived, and deceased). Median with range or interquartile ranges were transformed into means and standard deviation according to the calculation method proposed by Lou et al. and Wan et al. [16,17]. The IBs were extracted in standard measurement units as follows: albumin (g/L), CRP (mg/L), NLR (ratio), D-Dimer (µg/ml), ESR (mm/hr), ferritin (ng/mL), fibrinogen (g/L), IL-6 (pg/mL), LDH (UL), procalcitonin (ng/mL), WBC (10^9/L).

2.5 Statistical Analysis

All statistical analyses were performed using MedCalc Statistical Software (version 22.021, MedCalc Software Ltd, Belgium). Meta-analysis (continuous measure) was utilized to analyze continuous data, and the Chi-squared test was used to measure categorical data. A p-value < 0.05 was considered statistically significant. Forest plots were generated to depict binary data of included markers for mild vs severe cases, moderate vs severe cases, ICU vs non-ICU cases, and survived vs deceased cases of COVID-19, utilizing standardized mean difference (SMD) and 95% confidence intervals (95% CI). A random-effects model was applied to calculate the SMD for the severity, ICU admission, and mortality risks of COVID-19. Heterogeneity among studies was assessed by the Q test ($p < 0.1$), with I^2 values of 50% and 70% indicating moderate and high heterogeneity, respectively. Publication bias was evaluated using Begg's rank correlation test and Egger's regression asymmetry test via funnel plots. In cases of significant publication bias, studies with substantial standard errors (SE) and SMD were excluded iteratively to make the bias insignificant.

3. Results

3.1 Study search and selection

The systematic search initially yielded a total of 2,770 articles. Following the removal of duplicates (311), abstracts (143), and non-English articles (39), the titles and abstracts of 2,277 articles were screened. Of these, 1,485 were excluded due to irrelevance. Full-text screening was then conducted for 792 articles, resulting in the exclusion of 469. The remaining 323 articles were assessed against the full eligibility criteria, with 82 excluded due to their publication in predatory journals as identified by the Kscien list [18]. Finally, 241 eligible articles were included in the systematic review and meta-analysis [19-259]. A detailed PRISMA flow chart of the process is presented in (Figure 1).

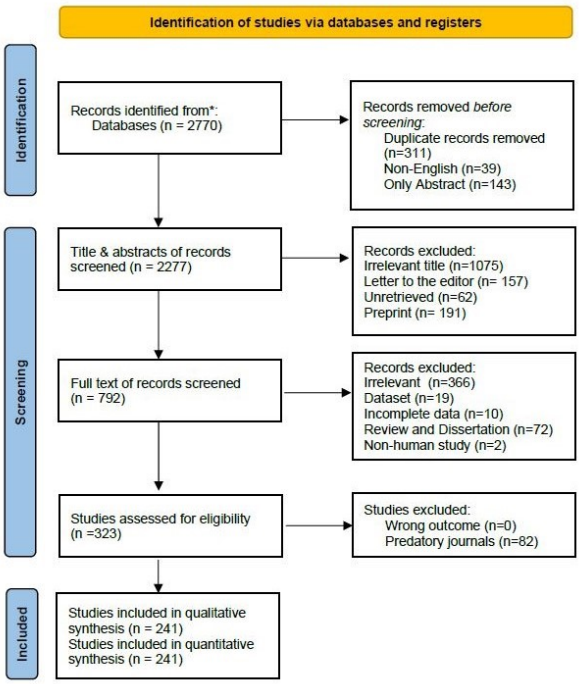

Figure 1. Prisma Flow Chart

3.2 Characteristics of the Studies

A total of 241 studies, comprising 79,934 cases of COVID-19, were included in this study. The majority of the studies were conducted in Turkey (25.3%), followed by Indonesia (10%), India (8.7%), and China (7.9%). Almost all of the studies were observational, with 73% being cohort studies, 21.2% cross-sectional studies, 4.6% case-control studies, and 0.8% case series. Additionally, one study was identified as a randomized controlled trial (0.4%) (Table 1). The raw data for each included study can be found in the supplementary material (Table 1 supplementary).

3.3 Main findings

Males accounted for 55.1% of the cases, while females represented 38.7%, with gender unspecified in 6.2% of cases. The mean age (means of mean) of patients was 58.2 ± 8.5 years, ranging from 41.2 to 86.6 years. Commonly reported comorbidities included hypertension (18.3%), diabetes mellitus (13.1%), and respiratory diseases (4.2%). Disease severity was reported in 29,490 cases (36.9%), categorized as mild in 10,551 cases (13.2%), moderate in 9,903 cases (12.4%), and severe in 9,036 cases (11.3%). ICU admission status was defined for 38,940 cases (48.7%), with 9,234 cases (11.5%) admitted to the ICU. In total, 43,051 cases (53.9%) survived the disease, while 11,937 cases (14.9%) died. Survival status was not defined in 31.2% of cases (Table 1).

Table 1. Baseline characteristics of the included studies.

Variables	Frequency / Percentage
Country of studies	
Turkey	61 (25.3%)
Indonesia	24 (10.0%)
India	21 (8.7%)
China	19 (7.9%)
Iran	16 (6.6%)
Pakistan	16 (6.6%)
Egypt	15 (6.2%)
Romania	8 (3.3%)
Iraq	7 (2.9%)
Mexico	6 (2.5%)
Italy	5 (2.1%)
Republic of Korea	5 (2.1%)
Spain	5 (2.1%)
Brazil	4 (1.7%)
Others	29 (12.0%)
Study design	
Cohort	176 (73.0%)
Cross-sectional	51 (21.2%)
Case-control	11 (4.6%)
Case Series	2 (0.8%)
Randomized controlled trial	1 (0.4%)
Gender	
Male	44043 (55.1%)
Female	30944 (38.7%)
N/R	4947 (6.2%)
Age* (year), mean of means ± SD, (min-max)	58.2 ± 8.5 (41.2-86.6)
Covid-19 severity	
Mild	10551 (13.2%)
Moderate	9903 (12.4%)
Severe	9036 (11.3%)
N/R	50444 (63.1%)
Comorbidities#	
Diabetes mellitus	10459 (13.1%)
Hypertension	14635 (18.3%)
Respiratory diseases	3336 (4.2%)
ICU admission	
Yes	9234 (11.5%)
No	29706 (37.2%)
N/R	40994 (51.3%)
Survival status	
Survived	43051 (53.9%)
Died	11937 (14.9%)
N/R	24946 (31.2%)

SD: Standard deviation, ICU: Intensive care unit, UAE: United Arab Emirates, N/R: not-reported
**The age belongs to 53196 cases.*
#Other comorbidities might be present but only the most common have been included.

3.4 Meta-analysis of COVID-19 severity with age and gender

A total of 37 studies, comprising 5,029 patients for mild vs severe cases and 64 studies with 9,849 cases for moderate vs severe cases, were included in the analysis of disease severity based on age. Severity demonstrated a significant correlation with age in both groups—mild vs severe and moderate vs severe (SMD = 0.884; 95% CI, 0.667-1.101; $p < 0.001$; $I^2 = 88.79\%$; $P_{heterogeneity} < 0.0001$ and SMD = 0.497; 95% CI, 0.349-0.645; $p < 0.001$; $I^2 = 89.86\%$; $P_{heterogeneity} < 0.0001$, respectively). Statistically significant heterogeneity across the selected studies was observed, as evidenced by the I^2 statistics for age and severity of COVID-19. Begg's test ($p = 0.0797$ for mild vs severe, $p = 0.5409$ for moderate vs severe) and Egger's test ($p = 0.0886$ for mild vs severe, $p = 0.0588$ for moderate vs severe) for age were not significant (Table 2-A and B supplementary, Figure 2). Funnel plots for the mild vs severe and moderate vs severe cases according to age suggested no potential publication bias (Figure 2). Disease severity was significantly associated with male gender in the analysis of 20,793 patients ($p < 0.05$) (Table 2).

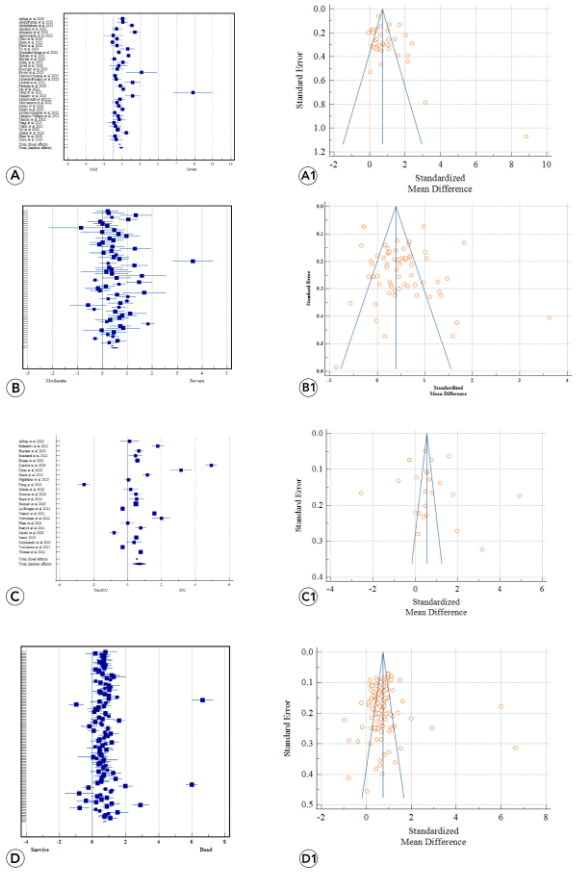

Figure 2. Forest plots depict standardized mean differences (SMD) and 95% confidence intervals, indicating the association between age and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots illustrate publication bias in studies examining the relationship between age and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

3.5 Meta-analysis of COVID-19 ICU admission with age and gender

Twenty-four studies, with a sample size of 19,216, were analyzed to reveal the role of age and gender in ICU admission among COVID-19 patients. Age demonstrated a significant correlation in increasing the risk of ICU admission (SMD = 0.685; 95% CI, 0.274 to 1.096; $p < 0.001$; $I^2 = 98.75\%$; $P_{heterogeneity} < 0.0001$). Begg's test ($p = 0.44$) and Egger's test ($p = 0.68$) for age in the ICU and non-ICU groups were not significant (Table 2-C supplementary, Figure 2). The funnel plot revealed no potential publication bias (Figure 2). Gender showed no impact on admission to the ICU in the analysis of 25,489 patients ($p = 0.26$) (Table 2).

Table 2. The role of gender in the severity, ICU admission, and mortality in COVID-19 patients.

Gender	Severity			ICU status		Outcome	
	Mild	Moderate	Severe	ICU	Non-ICU	Dead	Survived
Female	3051 (38.8%)	2954 (41.4%)	2296 (39.7%)	2129 (36%)	7210 (36.8%)	3401 (39.6%)	12889 (45.8%)
Male	4816 (61.2%)	4188 (58.6%)	3488 (60.3%)	3781 (64%)	12369 (63.2%)	5188(60.4%)	15280 (54.2%)
Total	7867 (37.8%)	7142 (34.3%)	5784 (27.8%)	5910 (23.2%)	19579 (76.8%)	8589 (23.4%)	28169 (76.6%)
P-value		0.005		0.262		<0.001	

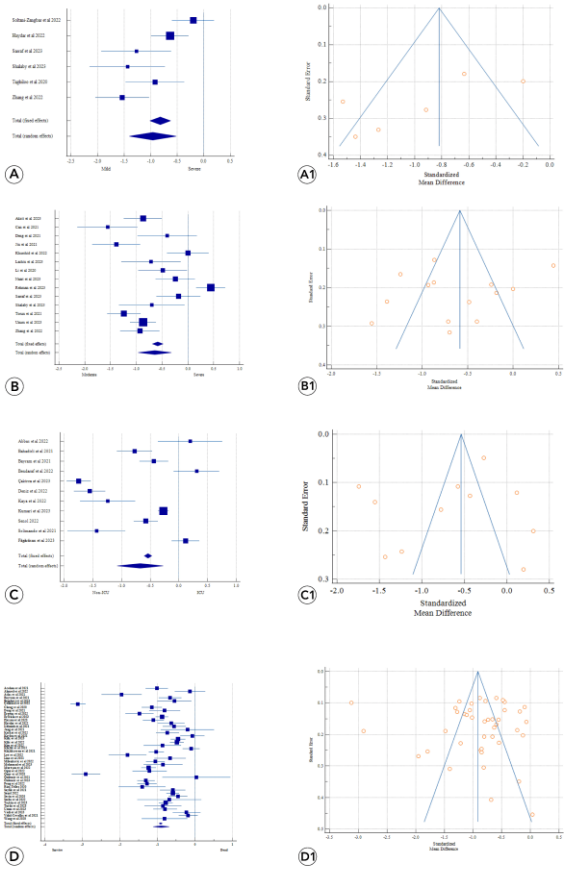

Figure 3. Forest plots illustrate the standardized mean difference (SMD) and 95% confidence intervals regarding the association between Albumin levels and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots delineate publication bias in studies examining the relationship between Albumin levels and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

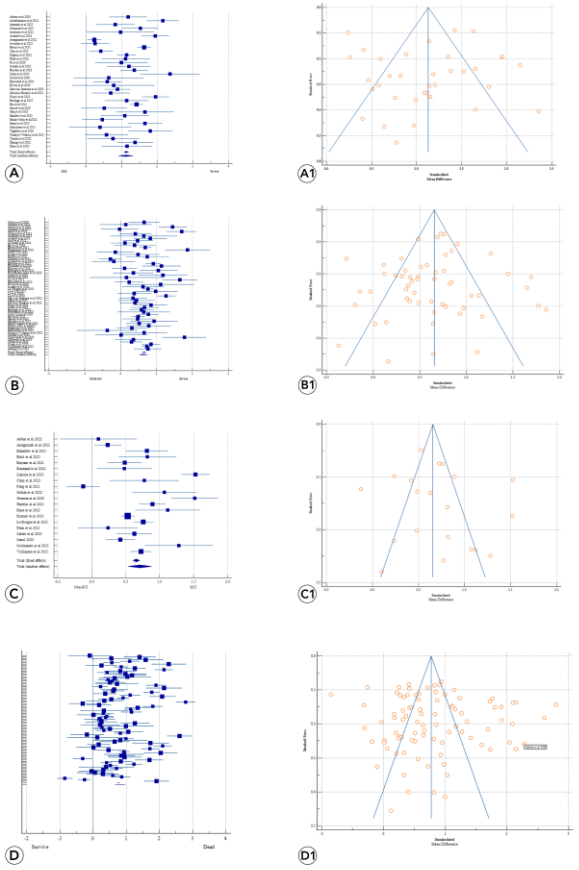

Figure 4. Forest plots display the standardized mean difference (SMD) and 95% confidence intervals for the association between CRP levels and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots delineate publication bias in the studies examining the association between CRP levels and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

3.6 Meta-analysis of COVID-19 mortality with age and gender

Across 41,320 patients in 115 studies, age again showed a significant correlation with mortality (SMD = 0.741; 95% CI, 0.626 to 0.857; $p < 0.001$; $I^2 = 94.49\%$; $P^{heterogeneity} < 0.0001$). Begg's test ($p = 0.87$) and Egger's test ($p = 0.89$) for age in the survived and dead groups were not significant (Table 2-D supplementary, Figure 2). The funnel plot also showed no significant publication bias (Figure 2). The male gender was highly vulnerable to mortality when analyzing 36,758 cases ($p < 0.0001$) (Table 2).

3.7 Meta-analysis of inflammatory markers and COVID-19 severity

Among both the mild vs severe groups and moderate vs severe groups, all studied IBs were significantly varied. Albumin level was significantly decreased in severe cases compared to mild and moderate cases (SMD = -0.959; 95% CI, (-1.399) – (-0.520); $p < 0.001$; $I^2 = 78.90\%$; $P^{heterogeneity} = 0.0002$ and SMD = -0.798; 95% CI, (-1.033) – (-0.563); $p < 0.001$; $I^2 = 73.48\%$; $P^{heterogeneity} < 0.0001$, respectively) (Table 2-E, F supplementary, Figure 3). Begg's test ($p = 0.188$ for mild vs severe, $p = 0.68$ for moderate vs severe) and Egger's test ($p = 0.0959$ for mild vs severe, $p = 0.749$ for moderate vs severe) for albumin were not significant and there was no publication bias (Figure 3). CRP

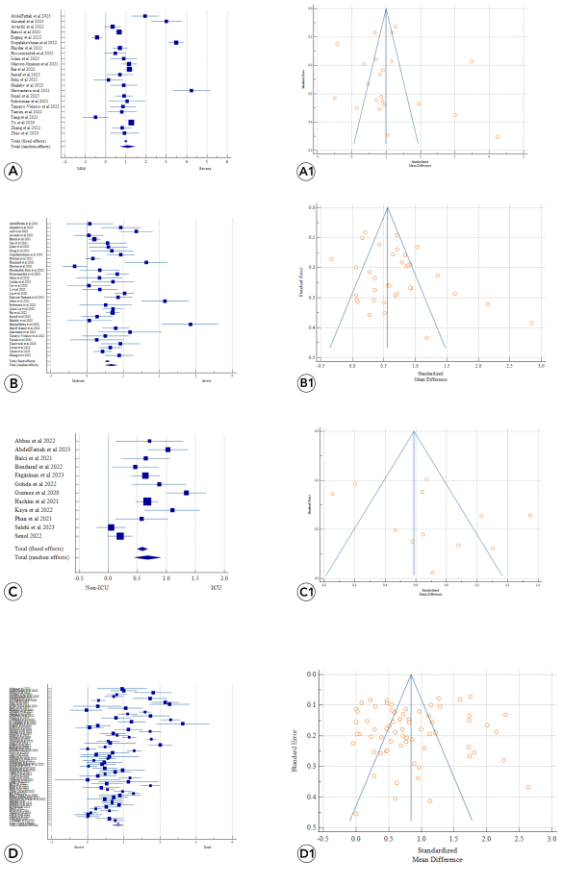

Figure 5. Forest plots depict the standardized mean difference (SMD) and 95% confidence intervals for the association between D-dimer levels and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots detail publication bias in the studies investigating the association between D-dimer levels and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

(SMD=1.118; 95% CI, 0.913 - 1.323; p<0.001; I^2 = 87.56%; P $_{heterogeneity}$ < 0.0001; Begg's test p =0.34; Egger's test p =0.87 for mild vs severe groups, SMD=0.630;95% CI, 0.520 - 0.740; p<0.001; I^2 = 78.29%; P $_{heterogeneity}$ < 0.0001; Begg's test p =0.943; Egger's test p =0.519 for moderate vs severe groups) (Table 2-G,H supplementary, Figure 4), D-dimer (SMD=1.086; 95% CI, 0.740 -1.432; p<0.001; I^2 = 95.49%; P $_{heterogeneity}$ < 0.0001; Begg's test p =0.178; Egger's test p =0.834 for mild vs severe groups, SMD=0.665; 95% CI, 0.506 -0.824; p<0.001; I^2 = 83.26%; P $_{heterogeneity}$ < 0.0001; Begg's test p =0.659; Egger's test p =0.059 for moderate vs severe groups) (Table 2-I,J supplementary, Figure 5), ESR (SMD=0.713; 95% CI, 0.424 - 1.001; p<0.001; I^2 = 0.0%; P $_{heterogeneity}$ = 0.392; Begg's test p =0.174; Egger's test p =0.131 for mild vs severe groups, SMD=0.806; 95% CI, 0.434 - 1.178; p<0.001; I^2 = 71.86%; P $_{heterogeneity}$ = 0.0016; Begg's test p =0.293; Egger's test p =0.109 for moderate vs severe groups) (Table 2-K,L supplementary, Figure 6), Ferritin (SMD=1.221; 95% CI, 0.961 - 1.481; p<0.001; I^2 = 87.84%; P $_{heterogeneity}$ < 0.0001; Begg's test p = 0.812; Egger's test p =0.248 for mild vs severe groups, SMD=0.563; 95% CI, 0.404 - 0.721; p<0.001; I^2 = 83.62%; P $_{heterogeneity}$ <0.0001; Begg's test p =0.345; Egger's test p =0.199

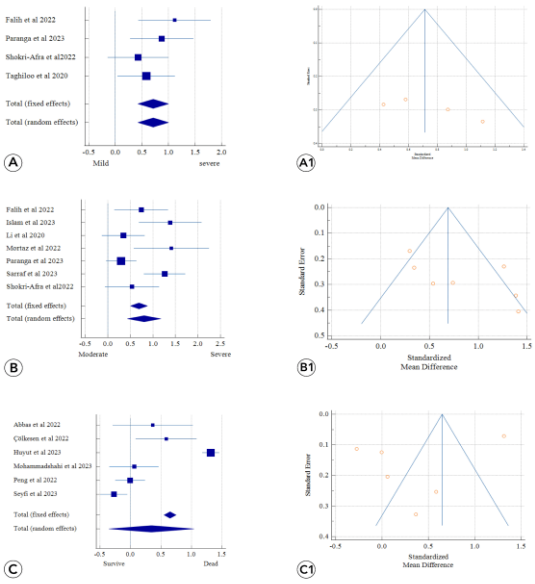

Figure 6. Forest plots present the standardized mean difference (SMD) and 95% confidence intervals for the association between ESR and severity (A and B) and mortality in COVID-19 (C). Random-effects funnel plots provide details on publication bias in studies examining the association between ESR and severity (A1 and B1) and mortality in COVID-19 (C1).

for moderate vs severe groups) (Table 2-M,N supplementary, Figure 7) Fibrinogen (SMD=0.750; 95% CI, 0.333 - 1.167; p<0.001; I^2 = 74.36%; P $_{heterogeneity}$ = 0.0036; Begg's test p = 0.327; Egger's test p =0.464 for mild vs severe groups, SMD=0.387; 95% CI, (-0.0569) – (0.831); p=0.087; I^2 = 84.17%; P $_{heterogeneity}$ <0.0001; Begg's test p =0.347; Egger's test p =0.50 for moderate vs severe groups) (Table 2-O,P supplementary, Figure 8), IL-6 (SMD=1.185; 95% CI, 0.774 - 1.595; p<0.001; I^2 = 90.02%; P $_{heterogeneity}$ < 0.0001; Begg's test p = 0.564; Egger's test p =0.0834 for mild vs severe groups, SMD=0.877; 95% CI, 0.633 – 1.122; p<0.001; I^2 = 87.45%; P $_{heterogeneity}$ <0.0001; Begg's test p =0.404; Egger's test p =0.545 for moderate vs severe groups) (Table 2-Q,R supplementary, Figure 9), LDH (SMD=1.186; 95% CI, 0.894 - 1.478; p<0.001; I^2 = 90.87%; P $_{heterogeneity}$ < 0.0001; Begg's test p = 0.506; Egger's test p =0.332 for mild vs severe groups, SMD=0.735; 95% CI, 0.560 – 0.910; p<0.001; I^2 = 77.90%; P $_{heterogeneity}$ <0.0001; Begg's test p =0.119; Egger's test p =0.695 for moderate vs severe groups) (Table 2-S,T supplementary, Figure 10), NLR (SMD=1.188; 95% CI, 0.305 - 2.070; p<0.001; I^2 = 98.56%; P $_{heterogeneity}$ < 0.0001; Begg's test p = 0.471; Egger's test p =0.450 for mild vs severe groups, SMD=0.858; 95% CI, 0.679 – 1.037; p<0.001; I^2 = 88.29%; P $_{heterogeneity}$ <0.0001; Begg's test p =0.870; Egger's test p =0.871 for moderate vs severe groups) (Table 2-U,V supplementary, Figure 11), procalcitonin (SMD=0.606; 95% CI, 0.333 - 0.880; p<0.001; I^2 = 60.86%; P $_{heterogeneity}$ = 0.0369; Begg's test p = 0.624; Egger's test p =0.244 for mild vs severe groups, SMD=0.441; 95% CI, 0.225 – 0.657; p<0.001; I^2 = 84.55%; P $_{heterogeneity}$ <0.0001; Begg's test p = 0.741; Egger's test p =0.345 for moderate vs severe groups) (Table 2-W,X supplementary, Figure 12), and WBC (SMD=0.693; 95% CI, 0.400 - 0.985; p<0.001; I^2 =

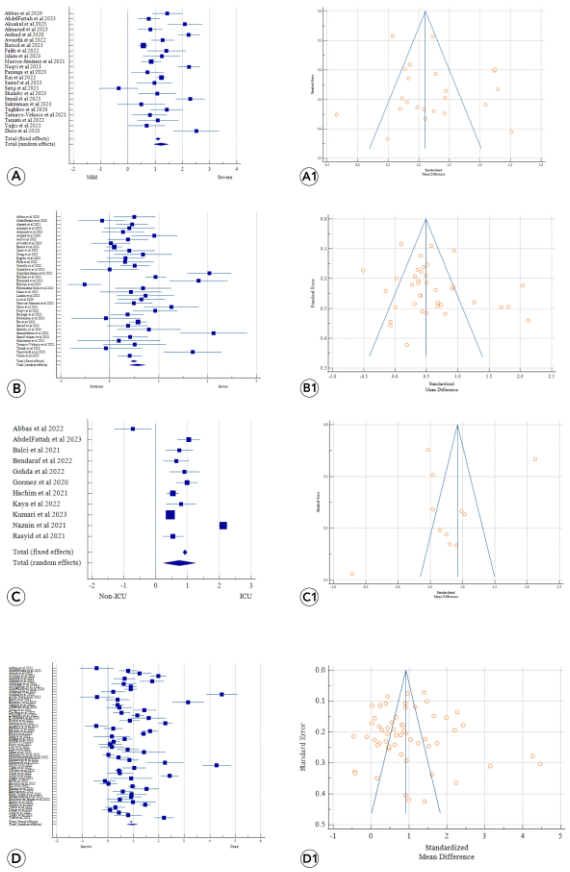

Figure 7. Forest plots illustrate the standardized mean difference (SMD) and 95% confidence intervals for the association between Ferritin levels and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots detail publication bias in the studies investigating the association between Ferritin levels and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

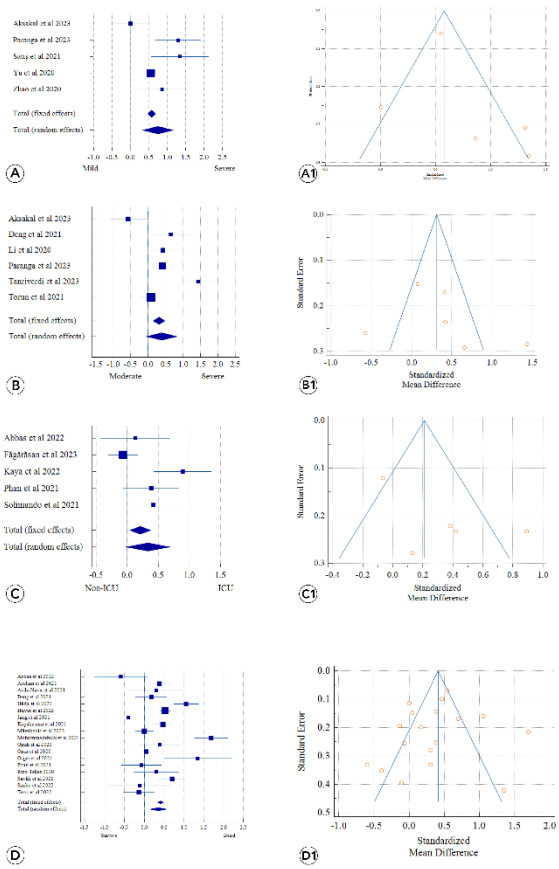

Figure 8. Forest plots depict the standardized mean difference (SMD) and 95% confidence intervals for the association between Fibrinogen levels and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots delineate publication bias in the studies investigating the association between Fibrinogen levels and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

88.66%; P $_{heterogeneity}$ < 0.0001; Begg's test p = 0.397; Egger's test p =0.240 for mild vs severe groups, SMD=0.438; 95% CI, 0.316 – 0.560; p<0.001; I^2 = 71.57%; P ^{heterogeneity} <0.0001; Begg's test p = 0.142; Egger's test p =0.104 for moderate vs severe groups) (Table 2-Y,Z supplementary, Figure 13), were all significantly increased and correlated with the severity of COVID-19 in both fixed and random effects except for fibrinogen in which reached significant level only in fixed effects.

3.8 Meta-analysis of inflammatory markers and ICU admission and Mortality in COVID-19

All IBs were significantly different between the non-ICU vs ICU groups and survived vs dead groups. Albumin level significantly declined in ICU and dead cases compared to non-ICU and survived cases (SMD = -0.674; 95% CI, (-1.072) – (- 0.276); p < 0.001; I^2 = 96.51%; P ^{heterogeneity} < 0.0001 and SMD = -0.902; 95% CI, (-1.112) – (-0.692); p < 0.001; I^2 = 95.58%; P ^{heterogeneity} < 0.0001, respectively) (Table 3-A, B supplementary, Figure 3).

Begg's test (p = 0.585 for non-ICU vs ICU groups, p = 0.441 for survived vs dead groups) and Egger's test (p = 0.389 for non-ICU vs ICU groups, p = 0.897 for survived vs dead groups) for albumin were not significant and there was no publication bias (Figure 3). CRP (SMD=0.708; 95% CI, 0.534 - 0.881; p<0.001; I^2 = 90.37%; P $_{heterogeneity}$ < 0.0001; Begg's test p =0.436; Egger's test p =0.505 for non-ICU vs ICU groups, SMD=0.815;95% CI, 0.670 - 0.959; p<0.001; I^2 = 94.86%; P $_{heterogeneity}$ < 0.0001; Begg's test p =0.648; Egger's test p =0.324 for survived vs dead groups) (Table 3-C,D supplementary, Figure 4), D-dimer (SMD=0.677; 95% CI, 0.453 - 0.900; p<0.001; I^2 = 82.96%; P $_{heterogeneity}$ < 0.0001; Begg's test p =0.583; Egger's test p =0.166 for non-ICU vs ICU groups, SMD=0.833; 95% CI, 0.686 -0.981; p<0.001; I^2 = 94.21%; P $_{heterogeneity}$ < 0.0001; Begg's test p =0.286; Egger's test p =0.759 for survived vs dead groups) (Table 3-E,F supplementary, Figure 5), ESR (SMD= 0.342; 95% CI, (-0.352) – (1.037); p

=0.334; I^2 = 97.38%; P $_{heterogeneity}$ < 0.0001; Begg's test p =0.573; Egger's test p =0.301 for survived vs dead groups (Table 3-G supplementary, Figure 6), Ferritin (SMD=0.745; 95% CI, 0.251 - 1.238; p =0.003; I^2 = 97.90%; P $_{heterogeneity}$ < 0.0001; Begg's test

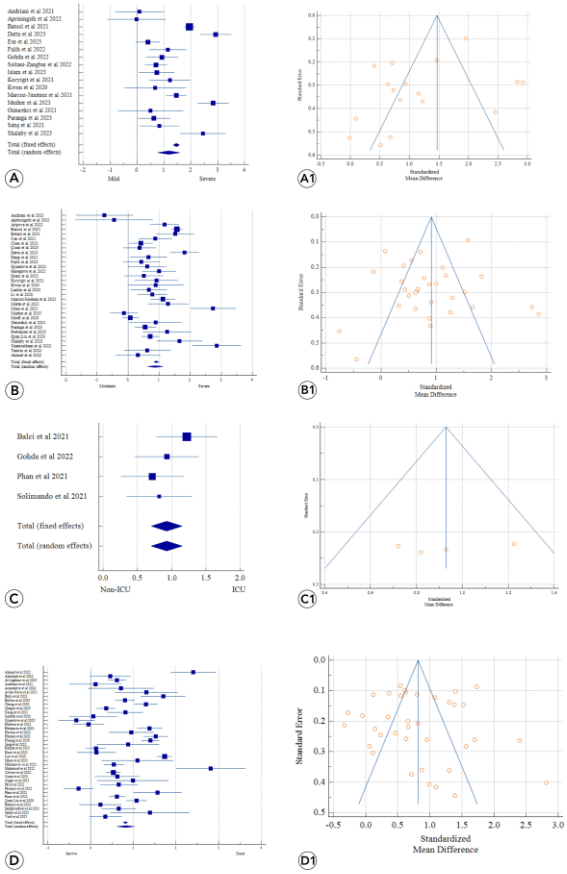

Figure 9. Forest plots display the standardized mean difference (SMD) and 95% confidence intervals for the association between IL-6 levels and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots outline publication bias in the studies investigating the association between IL-6 levels and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

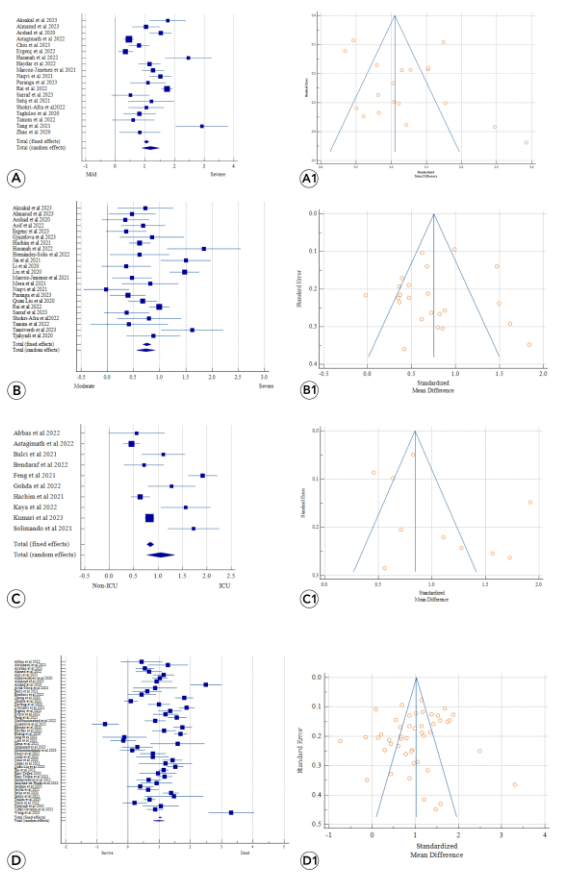

Figure 10. Forest plots illustrate the standardized mean difference (SMD) and 95% confidence intervals for the association between LDH levels and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots provide insights into publication bias in the studies investigating the association between LDH levels and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

p = 0.697; Egger's test p =0.718 for non-ICU vs ICU groups, SMD=0.956; 95% CI, 0.759 - 1.154; p<0.001; I^2 = 95.06%; P $_{heterogeneity}$ <0.0001; Begg's test p =0.330; Egger's test p =0.824 for survived vs dead groups) (Table 3-H,I supplementary, Figure 7) Fibrinogen (SMD=0.333; 95% CI, (-0.0242) – (0.691); p=0.068; I^2 = 73.85%; P $_{heterogeneity}$ = 0.0041; Begg's test p = 0.624; Egger's test p =0.158 for non-ICU vs ICU groups, SMD=0.357; 95% CI, 0.147 – 0.566; p=0.001; I^2 = 84.70%; P $_{heterogeneity}$ <0.0001; Begg's test p =0.569; Egger's test p =0.530 for survived vs dead groups) (Table 3-J,K supplementary, Figure 8), IL-6 (SMD=0.928; 95% CI, 0.702 - 1.155; p<0.001; I^2 = 0.0%; P $_{heterogeneity}$ = 0.421; Begg's test p = 0.496; Egger's test p =0.460 for non-ICU vs ICU groups, SMD=0.823; 95% CI, 0.625 – 1.022; p<0.001; I^2 = 92.63%; P ^{heterogeneity} <0.0001; Begg's test p =0.150; Egger's test p =0.918 for survived vs dead groups) (Table 3-L,M supplementary, Figure 9), LDH (SMD=1.057; 95% CI, 0.775 - 1.340; p<0.001; I^2 = 91.23%; P $_{heterogeneity}$ < 0.0001; Begg's test p = 0.179; Egger's test p =0.204 for non ICU vs ICU groups, SMD=0.978; 95% CI, 0.806 – 1.151; p<0.001; I^2 = 91.75%; P ^{heterogeneity} <0.0001; Begg's test p =0.373; Egger's test p =0.438 for survived vs dead groups) (Table 3-N,O supplementary, Figure 10), NLR (SMD=1.231; 95% CI, 0.967 - 1.496; p<0.001; I^2 = 89.69%; P $_{heterogeneity}$ < 0.0001; Begg's test p = 0.654; Egger's test p =0.970 for non-ICU vs ICU groups, SMD=1.013; 95% CI, 0.828 – 1.197; p<0.001; I^2 = 95.96%; P ^{heterogeneity} <0.0001; Begg's test p =0.499; Egger's test p =0.587 for non-ICU vs ICU groups) (Table 3-P,Q supplementary, Figure 11), procalcitonin (SMD=0.348; 95% CI, 0.055 - 0.640; p =0.020; I^2 = 53.53%; P $_{heterogeneity}$ = 0.116; Begg's test p = 0.601; Egger's test p =0.893 for non-ICU vs ICU groups, SMD=0.826; 95% CI, 0.627 – 1.025; p<0.001; I^2 = 93.28%; P ^{heterogeneity} <0.0001; Begg's test p = 0.753; Egger's test p =0.609 for survived vs dead groups) (Table 3-R,S supplementary, Figure 12) , and WBC (SMD=0.598; 95% CI, 0.324 - 0.872; p<0.001; I^2 = 91.23%; P $_{heterogeneity}$ < 0.0001; Begg's test p = 0.902; Egger's test p =0.356 for non-ICU vs ICU groups, SMD=0.676; 95% CI, 0.568 – 0.784; p<0.001; I^2 = 89.10%; P ^{heterogeneity} <0.0001; Begg's test p = 0.289; Egger's test p =0.556 for survived vs dead groups) (Table 3-T,U supplementary, Figure 13) , were all significantly elevated and correlated with the risk of ICU admission and mortality in COVID-19 in both fixed and random effects except for fibrinogen in which reached significant level only in fixed effects for ICU admission (p=0.014). In addition, ESR only correlated with mortality in

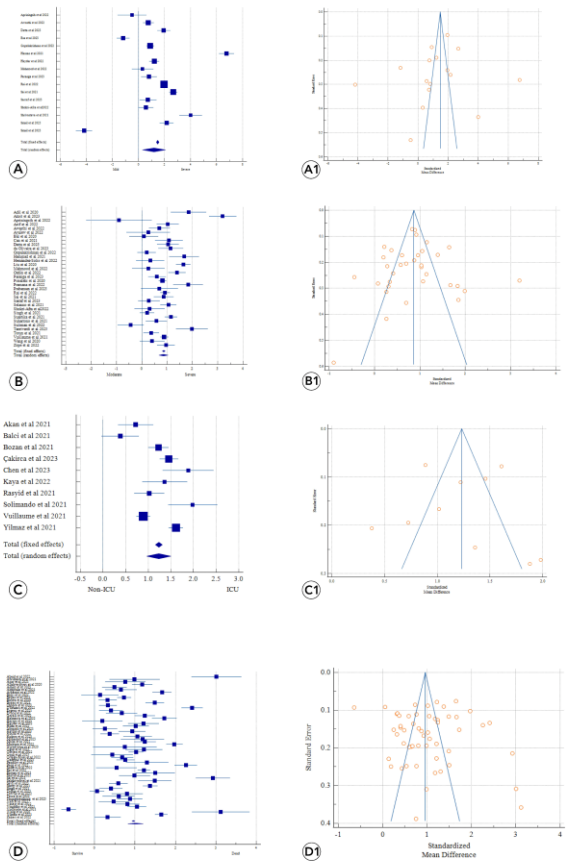

Figure 11. Forest plots display the standardized mean difference (SMD) and 95% confidence intervals for the association between NLR (Neutrophil-to-Lymphocyte Ratio) and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots provide insights into publication bias in the studies investigating the association between NLR and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1)..

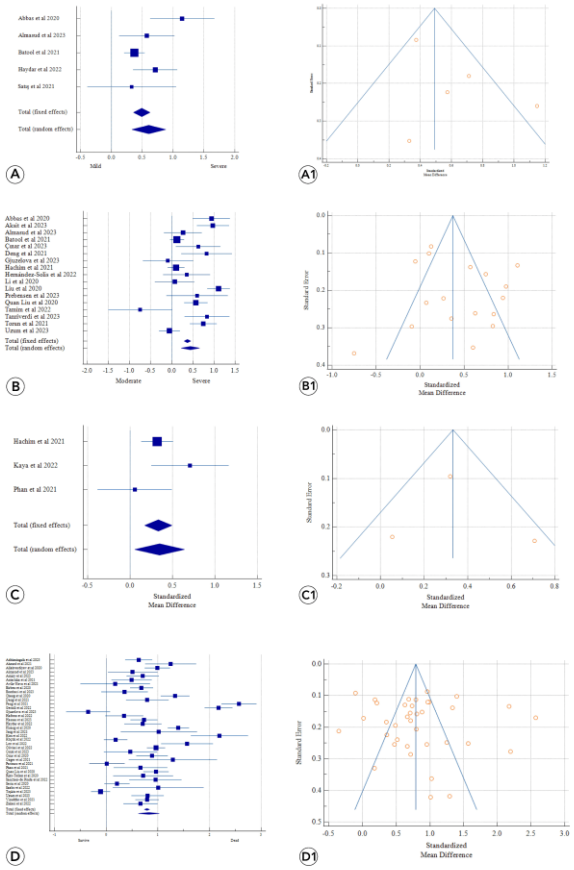

Figure 12. Forest plots depict the standardized mean difference (SMD) and 95% confidence intervals for the association between Procalcitonin levels and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots provide insights into publication bias in the studies investigating the association between Procalcitonin levels and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

fixed effects ($p<0.001$), and no effect on ICU admission was generated due to a lack of proper data.

4. Discussion

While the majority of COVID-19 cases manifest with mild or moderate symptoms, prompt identification of critical cases is crucial to mitigate prolonged hospitalization and higher mortality rates. Hematological and biochemical indicators play a significant role in early detection and prognosis assessment. Severe COVID-19 patients frequently exhibit systemic inflammation [92]. Treatment approaches diverge significantly between mild and severe COVID-19 cases. Mild cases often require minimal intervention, with some patients recovering without any treatment. In contrast, severe cases may necessitate various measures, including mechanical ventilation, extracorporeal membrane oxygenation, and continuous renal replacement therapy [8]. It has been indicated that heightened levels of inflammatory cytokines in the bloodstream are associated with liver and pulmonary damage in COVID-19 infection [13]. An expedient and early COVID-19 diagnostic test is imperative as a biomarker to aid in predicting and mitigating associated morbidity and mortality. Additionally, routine blood tests have been proposed as a more acceptable method for screening asymptomatic or mildly symptomatic individuals and could be utilized for screening purposes in outbreak areas [12]. This study represents the most extensive and comprehensive systematic review and meta-analysis, at least to the best of our knowledge, elucidating the roles of several common IBs in determining severity, ICU admission, and mortality among COVID-19 patients.

Regarding gender in COVID-19 patients, Ahmed et al. observed a male predominance of 69%. Similarly, a retrospective cohort study of 239 hospitalized COVID-19 cases in Lombardy, Italy, reported 71% of cases being male, while another report from Wuhan, China, noted a rate of 75% male cases [30, 260,261]. However, a meta-analysis conducted by Zavalaga-Zegarra et al. found that among 12,245 cases, the male population constituted 56.5% [15]. In the current meta-analysis, disease severity among 20,793 patients and mortality among 36,758 cases were significantly associated with the male gender ($p < 0.05$ and $p<$

0.0001, respectively). However, gender had no impact on ICU admission in the analysis of 25,489 patients (p = 0.26).

Increasing patient age independently predicted mortality, alongside significant associations with severity, with median ages of 65.5 years and 56 years observed in non-survivors and severe cases, respectively [30]. Luo Xiaomin et al.'s study on 298 COVID-19 cases from China also found a higher mortality proportion among those aged above 60, indicating age-related disease progression [262]. This can be attributed to impaired innate and adaptive immunity in elderly individuals. In healthy individuals, innate immunity typically neutralizes the virus early in the disease process, preventing it from reaching the alveoli. However, in elderly patients, innate immunity may fail to do so, allowing the virus to reach and replicate in the alveoli extensively. This triggers a robust response from macrophages and lymphocytes to eliminate virally infected cells, leading to elevated levels of cytokines [11]. In the present study, severity showed a significant correlation with age in both groups, mild vs severe and moderate vs severe (SMD = 0.884; 95% CI, 0.667-1.101; p < 0.001 and SMD = 0.497; 95% CI, 0.349-0.645; p < 0.001, respectively). Age also demonstrated a significant correlation with an increased risk of ICU admission (SMD = 0.685; 95% CI, 0.274 to 1.096; p < 0.001) and mortality (SMD = 0.741; 95% CI, 0.626 to 0.857; p < 0.001).

Hypoalbuminemia indicates malnutrition, hepatic and renal impairment, and diminished survival among critically ill patients. Furthermore, it has been identified as an independent risk factor linked to unfavorable outcomes in COVID-19 patients [92]. Reduced albumin levels may heighten mortality risk in COVID-19 patients. Yet, besides its anti-inflammatory role, albumin is suggested to have antioxidative, antithrombotic properties, and potential antiviral effects by binding to the SARSCoV-2 spike protein S1 subunit [263]. Baig et al. observed ICU patients initially with normal albumin levels, which rapidly decreased within 24 hours alongside increased oxygen needs. These patients lacked typical causes (proteinuria or chronic liver disease) for low albumin. The decline correlated with rising CRP, suggested as a hallmark of COVID-19 pneumonia. Furthermore, discharged ICU patients showed improved serum albumin levels, while those who succumbed to COVID-19 pneumonia had persistently low serum albumin levels [264]. Huang et al. suggested hypoalbuminemia in COVID-19 may arise from systemic inflammation. Serum albumin levels below 35 g/L were associated with a six-fold increase in COVID-19-related mortality risk (Odds ratio: 6.394, 95% CI: 1.316-31.092) [265]. Albumin levels, in this meta-analysis, were significantly decreased in severe cases compared to mild and moderate cases (SMD = -0.959; 95% CI, -1.399 to -0.520; p < 0.001 and SMD = -0.798; 95% CI, -1.033 to -0.563; p < 0.001, respectively). They also significantly declined in ICU and deceased cases compared to non-ICU and survived cases (SMD = -0.674; 95% CI, -1.072 to -0.276; p < 0.001 and SMD = -0.902; 95% CI, -1.112 to -0.692; p < 0.001, respectively).

In severe cases of COVID-19, elevated levels of CRP and other IBs including IL-6, IL-8, IL-2R, and IL-10 were observed compared to non-severe cases. The heightened levels of cytokines, chemokines, and NLR in severe cases indicate a potential role of hyperinflammatory response in the

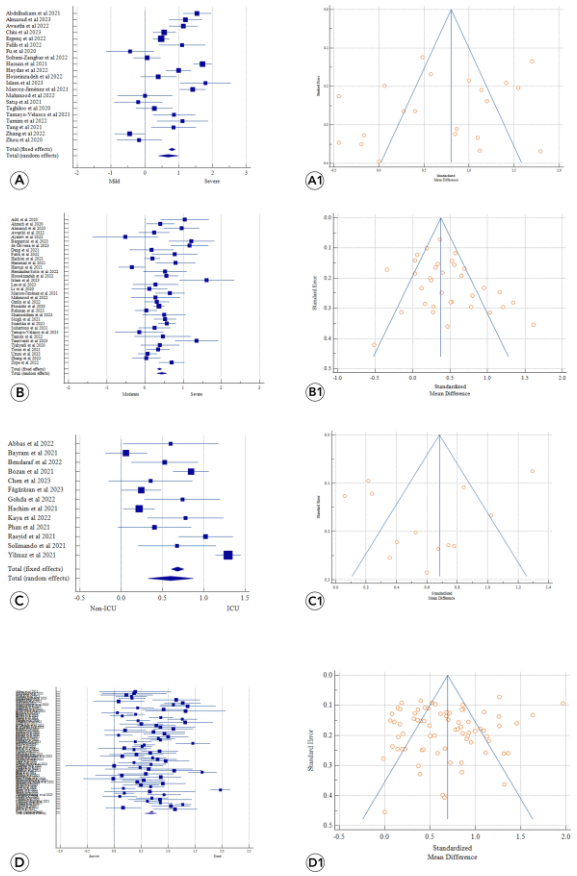

Figure 13. Forest plots illustrate the standardized mean difference (SMD) and 95% confidence intervals for the association between WBC (White Blood Cell count) and severity (A and B), ICU admission (C), and mortality in COVID-19 (D). Random-effects funnel plots provide insights into publication bias in the studies investigating the association between WBC and severity (A1 and B1), ICU admission (C1), and mortality in COVID-19 (D1).

pathogenesis of COVID-19 [11,13]. In a meta-analysis of 38 articles involving 5,699 patients with severity outcomes and 6,033 patients with mortality outcomes, it was found that severe cases and non-survivors of COVID-19 had higher NLR levels upon admission compared to non-severe cases and survivors (SMD 0.88; 95%CI 0.72–1.04; I^2=75.52% and 1.87; 95%CI 1.25–2.49; I^2=97.81%, respectively) [10]. Ali et al.'s meta-analysis, comprising 11 studies and 2,437 COVID-19 patients, found significantly elevated serum levels of CRP (SMD = 3.363, P value<.001), D-Dimer (SMD = 1.073, P value <.001), and LDH (SMD = 3.345, P value <.001) in severe cases of COVID-19 [11]. Another study revealed a positive correlation between CRP levels and the diameter of lung lesions, suggesting CRP is a potential indicator of disease severity [266]. Furthermore, other scholars observed that common markers, including WBC count, neutrophil count, lymphocyte count, CRP, and D-dimer levels, showed trends in predicting disease severity and mortality outcomes [218,257]. Nugroho et al.'s meta-analysis of 29 studies (4,328 patients) revealed higher D-dimer levels on admission in severe cases compared to non-severe cases (MD = 0.95, 95% CI: 0.61-1.28, P < .05; I^2 = 90%). Non-survivors showed significantly higher D-dimer values (MD = 5.54, 95%

CI: 3.40-7.67, P < .05; I^2 = 90%), while ICU admission did not significantly affect D-dimer levels (MD = 0.29, 95% CI: -0.05 to 0.63, P = 0.10; I^2 = 71%) [9].

IL-6 plays a key role in maintaining immunocompetence, defined as the host's ability to respond to infections [267]. However, elevated levels have been linked to increased disease severity. These findings suggest high IL-6 levels during viral infections may promote virus survival and worsen the disease [268]. Amiri-Dashatan et al. found in 23 studies that elevated CRP, TNF-a, and IL-6 levels were linked to severe COVID-19 and potential liver damage [13]. Aziz et al. analyzed nine studies, showing significantly higher IL-6 levels in severe cases, also associated with increased mortality risk [269]. Ahmed et al. reviewed 157 COVID-19 cases and found ferritin levels significantly associated with disease severity and mortality [30]. In another study, hyperferritinemia (> 400 µg/L) was observed in severe cases, 1.5 to 5.3 times higher than non-severe cases. Non-survivors had ferritin levels around 1400 ng/mL, 3 to 4 times higher than survivors [270]. A pooled analysis showed that patients with elevated LDH faced a 16-fold higher mortality rate and over 6-fold increased risk of severe COVID-19 illness. Additionally, in all included studies reporting mortality, elevated LDH levels were observed in 95% of non-survivors compared to 60% of survivors [6]. Henry et al. analyzed 21 studies involving 2,984 patients and found that markers such as D-dimer, CRP, ferritin, and procalcitonin were significantly elevated in patients with severe COVID-19. Furthermore, in the mortality cohort comprising three studies, these markers were significantly higher in non-survivors compared to survivors [271]. Other scholars, in a multivariate analysis of 271 patients, reported that elevated levels of procalcitonin and CRP were significantly associated with mortality, even after adjusting for age, sex, and race or ethnicity [97]. In this meta-analysis, all IBs, such as CRP, D-dimer, ferritin, IL-6, LDH, NLR, procalcitonin, and WBC, were significantly associated with severity, ICU admission, and mortality in COVID-19 patients. However, ESR only showed a correlation with the severity of COVID-19 and had no impact on mortality (p = 0.334). Fibrinogen was significantly associated with severe cases compared to mild cases (p < 0.001); however, it did not significantly differ between moderate and severe cases (p = 0.087). It showed a significant increase in deceased cases (p = 0.001) but did not affect ICU admission (p = 0.068).

Although this study boasts the largest sample size and includes the largest number of IBs, it encounters several limitations. Firstly, the search was exclusively conducted on Google Scholar, potentially overlooking studies not indexed in the search. Additionally, most of the included studies were observational, contributing to the high statistical heterogeneity observed post-meta-analysis. This heterogeneity stems from clinical and methodological variations among the included studies but could also be influenced by geographical differences and risk of bias. Moreover, estimated effect measures were calculated as mean differences without adjusting for potential confounders such as age, sex, or comorbidities, which may influence inflammatory processes. Therefore, we could not definitively claim that any marker is superior to others in assessing the prognosis of patients with COVID-19.

5. Conclusion

The IBs can play a pivotal role in determining the clinical course and outcomes among patients with COVID-19. Albumin, CRP, D-dimer, ferritin, IL-6, LDH, NLR, procalcitonin, and WBC all significantly impact severity status, ICU admission, and mortality. However, ESR and fibrinogen cannot reliably reflect all three situations among COVID-19 patients.

Declarations

Conflicts of interest: The author(s) have no conflicts of interest to disclose.

Ethical approval: Not applicable.

Patient consent (participation and publication)**:** Not applicable.

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Authors' contributions: FHK and HOA were major contributors to the conception of the study and the literature search for related studies. AMM, DSH, BAA, HOA, and SHM were involved in the literature review, manuscript writing, and data analysis and interpretation. SAB, MAR, DMA, DQH and SMA Literature review, final manuscript approval, and the Figures' processing. FA, DKA, YMM and KKM were involved in the literature review, the study's design, and the manuscript's critical revision. HOA and FHK Confirmation of the authenticity of all the raw data All authors approved the final version of the manuscript.

Use of AI: AI was not used in the drafting of the manuscript, the production of graphical elements, or the collection and analysis of data.

Data availability statement: Not applicable.

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