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The association between mortality due to COVID-19 and coagulative parameters: a systematic review and meta-analysis study

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Abstract

Aims and objectives This systematic review and meta-analysis study evaluated the association between mortality due to COVID-19 and coagulative factors.

Methods A systematic search was conducted on electronic databases including PubMed, Scopus, and the Web of Science from the beginning of the pandemic until October 2024 to identify relevant studies on COVID-19 patients and their laboratory findings related to coagulation markers and mortality outcome. Eligibility criteria were defined based on the PICO framework, and data extraction was performed by two authors independently using a standardized sheet. Statistical analysis was accomplished using the random effects model, and heterogeneity among studies was assessed using the I^2 test. R and RStudio were used for statistical analysis and visualization.

Results Our systematic literature search yielded 6969 studies, with 48 studies meeting the inclusion criteria for our meta-analysis. The mean platelet count was significantly lower in deceased COVID-19 patients compared to survivors (20.58), while activated partial thromboplastin time (aPTT) and fibrinogen levels did not show significant differences. The pooled mean difference of D-Dimer, International Normalized Ratio (INR), and prothrombin time (PT) were significantly lower in survived patients (-2.45, -0.10, and -0.84, respectively). These findings suggest that platelet count, D-Dimer, INR, and PT may serve as potential indicators of mortality in COVID-19 patients.

Conclusion The results of our systematic review and meta-analysis revealed a significant reduction in the pooled platelet count among deceased individuals when compared to survivors. However, no significant distinctions were observed in the pooled mean activated aPTT and fibrinogen levels between the deceased and survivor groups. On the other hand, there were noticeable variations in the pooled estimated mean of INR, PT, and D-Dimer levels, with significantly higher values in the deceased group compared to those who survived.

Keywords COVID-19, Mortality, Coagulopathy, D-dimers, Fibrinogen, Prothrombin time, Meta-analysis

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Background

The global impact of the COVID-19 pandemic caused by the SARS-CoV-2 virus has been profound, resulting in a significant loss of lives in a relatively short period. SARS-CoV-2 which belongs to the coronavirus family, renowned for its ability to induce respiratory infections. Notably, COVID-19 has been linked to coagulopathy, leading to the development of intravascular thrombi and fibrinogen deposition [1–3]. Extensive research has demonstrated that COVID-19 infection triggers the activation and dysregulation of the coagulation system, leading to abnormalities in various coagulation parameters. Crucially, COVID-19-associated coagulopathy exhibits unique characteristics that distinguish it from other coagulation disorders. Initial reports from Wuhan, China, have highlighted elevated levels of D-dimers and fibrinogen, accompanied by alterations in platelet activation. These findings suggest a state of hypercoagulability in individuals with COVID-19 [4–10].

Severe cases of COVID-19 have been found to fulfil the criteria for disseminated intravascular coagulation (DIC), signifying a serious and potentially life-threatening complication associated with the disease [11–14]. However, recent investigations have revealed that COVID-19-associated coagulopathy differs from coagulopathic disorders caused by bacterial infections and other disorders. Specifically, individuals with COVID-19 often exhibit increased D-dimer and fibrinogen levels, while prothrombin time and platelet count may not exhibit significant changes during the early stages of the disease. Given the observed coagulation abnormalities, the early detection of elevated coagulation biomarkers becomes paramount for risk stratification in COVID-19 patients [15–20]. Thromboembolic complications have emerged as potential causes of clinical deterioration in severe cases, even after patients have been discharged from the hospital. This underscores the significance of vigilant monitoring of coagulation status in individuals with COVID-19 [21–23].

To assess the reliability of risk stratification based on early coagulation parameters and its impact on mortality and disease outcome, we conducted a comprehensive meta-analysis of published research worldwide. By scrutinizing data from various clinical studies, we aim to gain deeper insights into coagulation parameters in individuals with COVID-19.

Methods and materials

The present study was conducted based on the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guideline 2020 [24] (see Supplemental Table 1).

Search strategy

A systematic search was accomplished on electronic databases such as Web of Science, Scopus, and PubMed from the beginning of the pandemic until October 2024. The search strategy included a combination of relevant medical subject heading (MeSH) terms and relevant keywords for ("Covid-19" OR "Sars-Cov-2" OR "Coronavirus") AND ("Laboratory findings" OR "Coagulation" OR "Biochemical markers") AND ("Mortality", "Survival", "Death", "Outcome") (see supplemental Table 2).

Eligibility criteria

We defined our eligibility criteria based on the PICO framework: (P) Population: COVID patients (confirmed by serum antibody tests, polymerase chain reaction, or clinical symptoms after close exposure to proven cases of COVID) who deceased (I) Intervention/Exposure: Laboratory and coagulative measures. (C) Control: COVID patients who survived (O) Outcome: Mortality. The exclusion criteria were defined as: absence of laboratory findings, not reporting mortality as outcome, lack of individual data, reporting only patients with confounding conditions such as warfarin usage, pregnant women, cancer, coagulopathy disorder. Furthermore, articles in non-English language were excluded. The kappa statistic was used to measure inter-rater reliability in screening and selecting the eligible studies and the results were interpreted as less than 0.2 represents no agreement; 0.21–0.29 represents minimal agreement; 0.40–0.59 represents weak agreement; 0.60–0.79 represents moderate agreement; 0.80–0.90 represents strong agreement; greater than 0.9 represents almost perfect agreement [25].

Data extraction and outcome measures

The methodological quality evaluation of the included articles was assessed by the Newcastle–Ottawa Scale (NOS) checklist [26]. The quality scores were classified as follows: very good (score of 9–10), good (score of 7–8), satisfactory (score of 5–6), or unsatisfactory (score of 0–5). The data extraction using a standardized sheet was performed by two independent authors. A third-party discussion was done when any disagreement occurred. The standardized sheet included: authors' name, year of publication, total number of participants, mean age of the participants, design of the study, and country of the study along with mean platelet count and its standard deviation (SD) and total number, mean prothrombin time (PT) and its standard deviation (SD) and total number, mean activated partial thromboplastin time (aPTT) and its standard deviation (SD) and total number, mean D-Dimer level and its standard deviation (SD) and total number, mean fibrinogen level and its standard deviation (SD) and total

number, and mean International Normalized Ration (INR) and its standard deviation (SD) and total number. The aforementioned variables were extracted in two groups: the survivors and the deceased.

Statistical analysis and data synthesis

The pooled mean difference was calculated using random effects model and Hedges' g along with SD estimation. For assessing the heterogeneity of the included studies, the I^2 (I square) test was used. Moreover, < 25% considered low, 25–75% considered moderate, and > 75% considered high heterogeneity. Additionally, the leave-one-out sensitivity analysis was conducted to assess robustness of findings. The Mantel–Haenszel method and random effects model was used for pooling the effect sizes and SD was consequently calculated. For testing the overall significance of the random model, *z*-test was performed Potential publication bias was graphically assessed by creating funnel plots for each of the aforementioned groups. Furthermore, subgroup analysis based on patients' average age (> 60 or ≤ 60 years) and meta-regression was performed to find the source of heterogeneity. Two independent reviewers evaluated the certainty of synthesized evidence using GRADE guidelines [27]. R (R Foundation for Statistical Computing, Vienna, Austria) and RStudio (RStudio, Inc., Boston, MA) were used for the statistical analysis and creating forest and funnel plots.

Results

Study selection and characteristics

Our systematic search of the literature obtained 6969 studies, primarily. After removing the duplicates ($n = 2580$), 4389 studies were screened based on their title and abstract. Finally, 189 studies were included for full-text evaluation. Based on our inclusion and exclusion criteria, 48 studies [22, 28–74] were included in our final meta-analysis model (Fig. 1). The agreement between

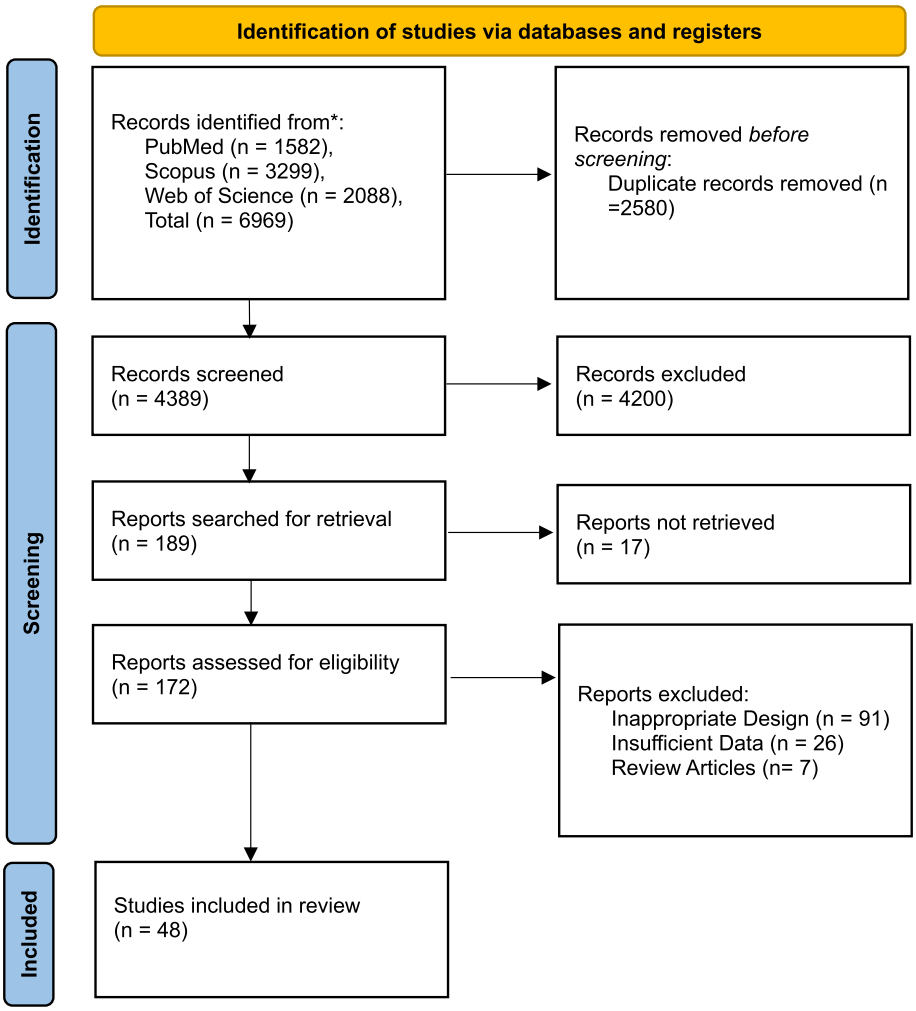

Fig. 1 PRISMA flow diagram of the systematic search and study selection

authors was moderate for the screening and selection process (κ = 0.70). The detailed quality assessment of the included studies is presented in supplemental Table 3. Supplemental Tables 4–6 represent characteristics of included articles and exact values of coagulative parameters of survived and deceased cases.

Results of syntheses

Based on our meta-analysis, the mean difference between Platelet count among the survivors and the deceased was 20.58 (95% CI: 12.28 to 28.88; $P = < 0.01$) based on the random effects model and was significantly (*p-value* < 0.01) lower among the deceased (Fig. 2). The between-study heterogeneity was high ($I^2 = 80\%$). Results of the Leave-one-out by sensitivity analysis method showed no difference from excluding any studies (Supplemental Fig. 1). Current meta-analysis indicated the mean difference between aPTT count among the survivors and the deceased was not statistically significant (MD: -0.609; 95% CI: -1.66 to 0.45; $P = 0.24$) based on the random effects model (Fig. 3). High heterogeneity ($I^2 = 95.3\%$) was observed between the included studies. Leave-one-out analysis indicated no significant difference by and studies' omission (Supplemental Fig. 2). Meta-analysis of D-dimer levels revealed that the average D-dimer counts were significantly lower in the survived compared to non-survived patients (MD: -2.45; 95% CI: -3.24 to -1.66; $P < 0.01$) (Fig. 4). The included studies were highly heterogeneous ($I^2 = 92.5\%$). The pooled effect and heterogeneity were not statistically changed by removing any studies using the leave-one-out method (Supplemental Fig. 3). Meta-analysis of the difference in fibrinogen levels among survival and deceased showed a non-significant mean difference between the two groups (MD: -0.25; 95%

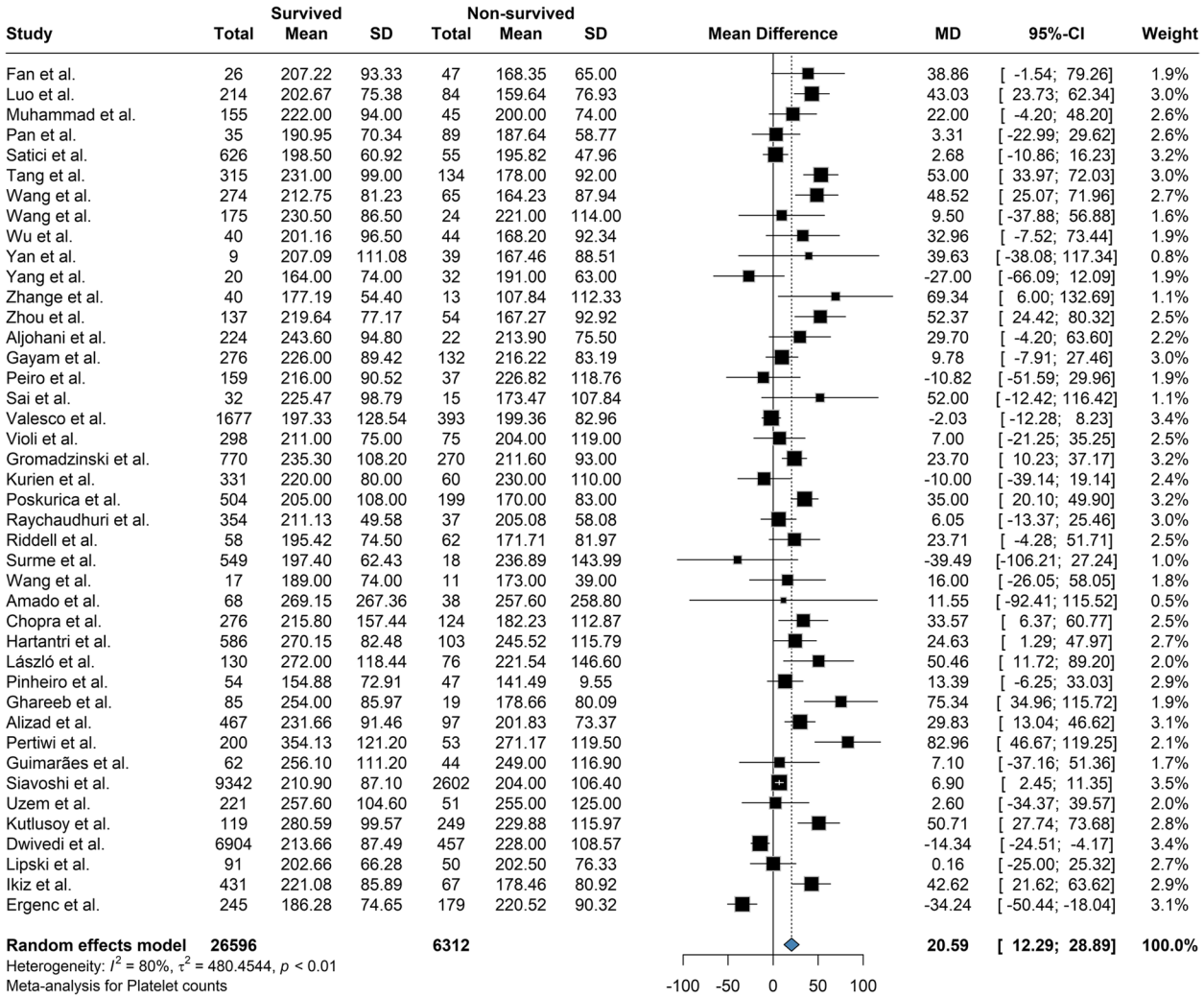

Fig. 2 The mean difference between Platelet count among the survivors and the deceased was 20.58 and was significantly higher among the survivors

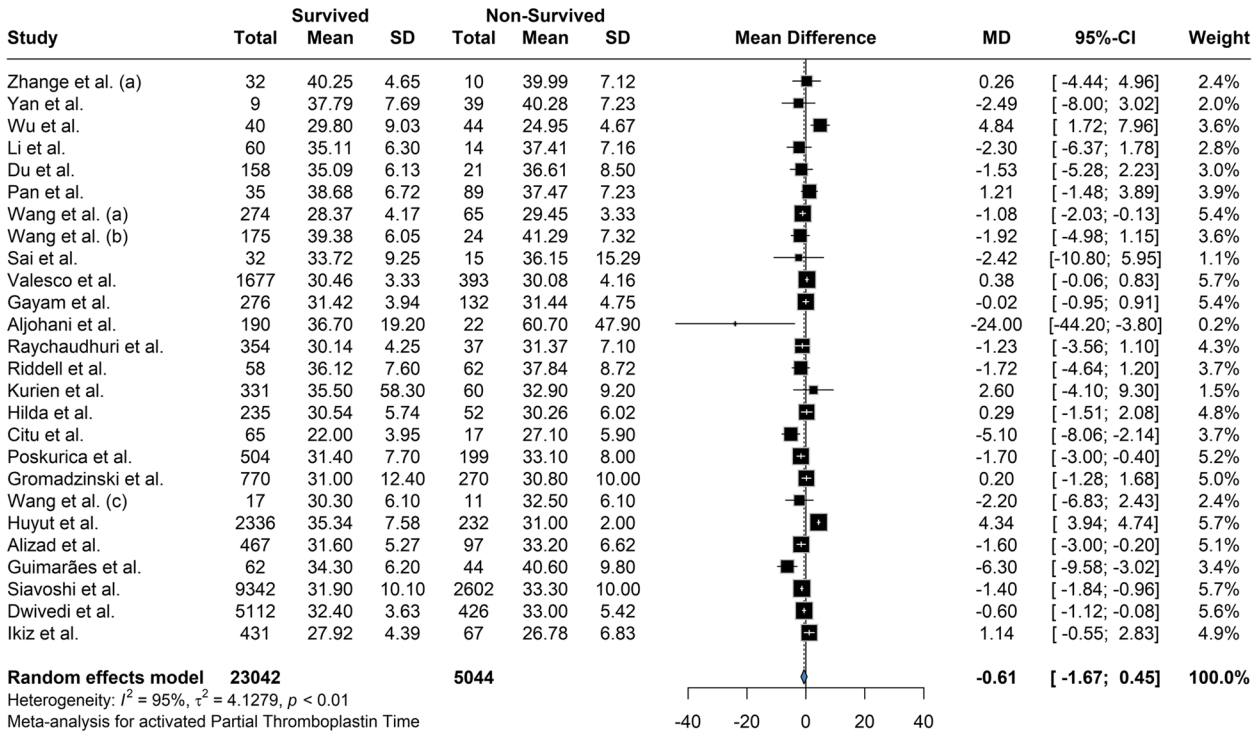

Fig. 3 The mean difference between aPTT count among the survivors and the deceased was -0.60 and was not significantly lower among the deceased

CI: -0.62 to 0.11; $P=0.16$) (Fig. 5). The between-study heterogeneity was markedly high ($I^2=91\%$). Further sensitivity analysis using the leave-one-out method indicated that removing any studies did not significantly alter the polled results (Supplemental Fig. 4). The meta-analysis results based on the random effects showed significantly higher INR count in deceased than in the survived patients (MD: -0.10; 95% CI: -0.14 to -0.05; $P<0.01$) (Fig. 6). High between-study heterogeneity ($I^2=93.9\%\%$) in the included studies was observed. Furthermore, the Leave-one-out sensitivity analysis resulted in no significant difference by removing any selected studies (Supplemental Fig. 5). The findings of the meta-analysis indicated significantly lower PT levels in the survived compared to the deceased (MD: -0.84; 95% CI: -1.09 to -0.59; $P<0.01$) (Fig. 7). The included articles were moderately heterogeneous ($I^2=73.5\%$). Sensitivity analysis by the leave-one-out method did not reveal any significant change in pooled estimate results or heterogeneity by any studies' omission (Supplemental Fig. 6).

Subgroup analyses

Subgroup analyses were performed based on age (Figure Supplemental 7–12). The tests of between-group differences were non-significant for all variables except for fibrinogen (P value$=0.02$) and INR (P value<0.01). In both Fibrinogen and INR analyses, only a subgroup of studies with an average age of less than 60 years showed significant mean differences between survived and non-survived cases (Figure Supplemental 10 and 11). Furthermore, the between-study heterogeneity within each group remained in the same category as the overall heterogeneity for all variables.

Meta-regression

Meta-regression analysis with year of publication, sample size, and age was performed for all variables and presented in supplemental Table 4. Findings of platelet, PT, D-dimer, INR and aPTT meta-regression were non-significant. However, the age in fibrinogen analysis significantly accounted for heterogeneity.

Certainty of evidence

Supplemental Table 5 shows a detailed investigation of the quality of polled results. The certainty was low in platelet, aPTT, INR, and PT results and very low in D-dimer and fibrinogen findings.

Risk of publication bias

The risk of publication bias was assessed by visual investigation and statistical tests (Figure Supplemental 13–18). The funnel plots of platelet, aPTT, Fibrinogen, and INR

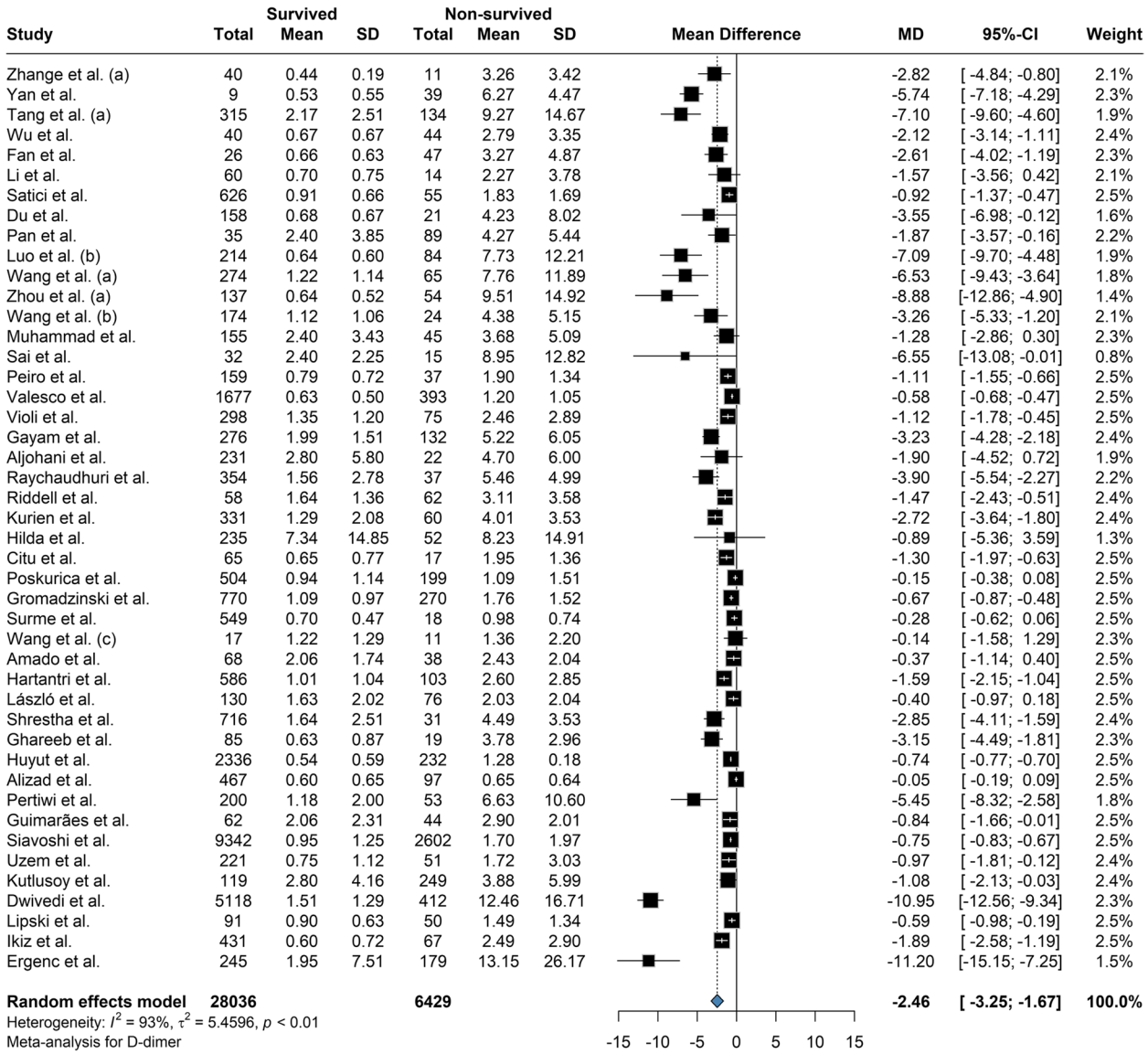

Fig. 4 The mean difference between D-Dimer count among the survivors and the deceased was -2.45 and was significantly lower among the survived

meta-analyses were symmetrical, and statistical tests did not demonstrate a significant risk of publication bias. In contrast, funnel plots of DD and PT analyses were asymmetrical, representing probable risk of publication bias confirmed by the Begg test (P values < 0.05).

Discussion

According to the results of our systematic review and meta-analysis study, the estimated pooled platelet count was significantly lower among the deceased compared to the survivors. Also, the pooled mean aPTT and fibrinogen levels did not show any significant difference among the deceased and the survivors. The pooled estimated mean of INR, PT, and D-Dimer was significantly higher among the deceased compared to those who survived.

Platelets play a critical role in maintaining haemostasis and contribute to thrombo-inflammatory processes during viral infections. Changes in platelet production or destruction at various stages of viral infection can lead to coagulation imbalances, resulting in pro-thrombotic events or platelet dysfunction and bleeding risks. Thrombocytopenia (low platelet count) has emerged as a significant marker of COVID-19 severity and mortality [75–78]. Thrombocytopenia has diverse reasons and may

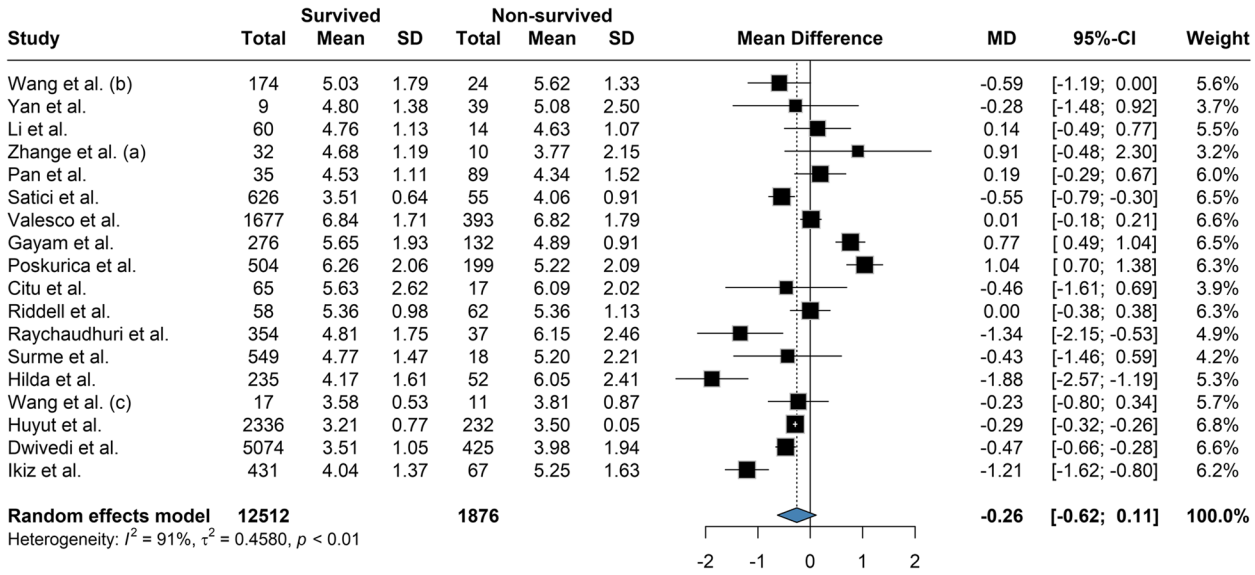

Fig. 5 The mean difference between Fibrinogen count among the survivors and the deceased was -0.25 and was not significantly lower among the deceased

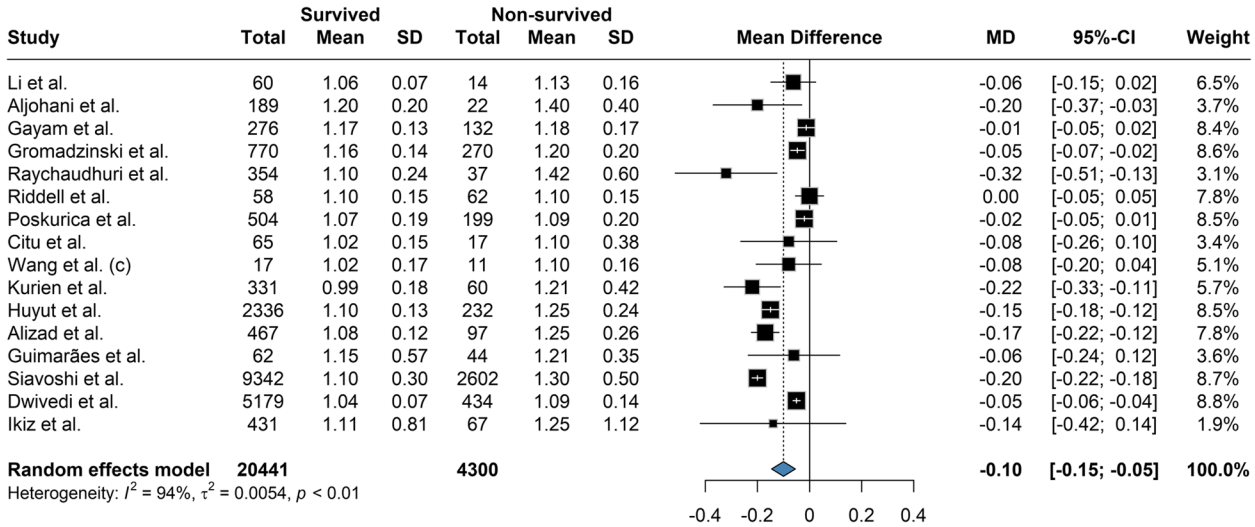

Fig. 6 The mean difference between INR count among the survivors and the deceased was -0.10 and was significantly lower among the survived

include early suppression of platelet production, damage to bone marrow, and hemo-phagocytosis [79–83].

In the convalescent phase of COVID-19, some patients may experience reactive thrombocytosis following initial thrombocytopenia. The increase in immature reticulated platelets observed in COVID-19 patients may impact the effectiveness of anti-platelet therapies [84–88]. Changes in platelet parameters and reactivity have been associated with severe COVID-19 infections. Moreover, studies have reported dysfunction of megakaryocytes (platelet progenitors) in COVID-19 patients, with elevated and abnormal megakaryocytes found in various organs, including the lungs, heart, brain, and bone marrow. The lungs appear to play a significant role in platelet biogenesis, and hypoxia-induced thrombocytopenia is linked to reduced lung megakaryocytes and impaired platelet generation in the lungs [89–95].

The SARS-CoV-2 virus has been implicated in up-regulating megakaryocyte progenitors and elevating circulating megakaryocytes in severe COVID-19, possibly through infection of early megakaryocyte progenitors. This suggests the possibility of viral RNA transfer from

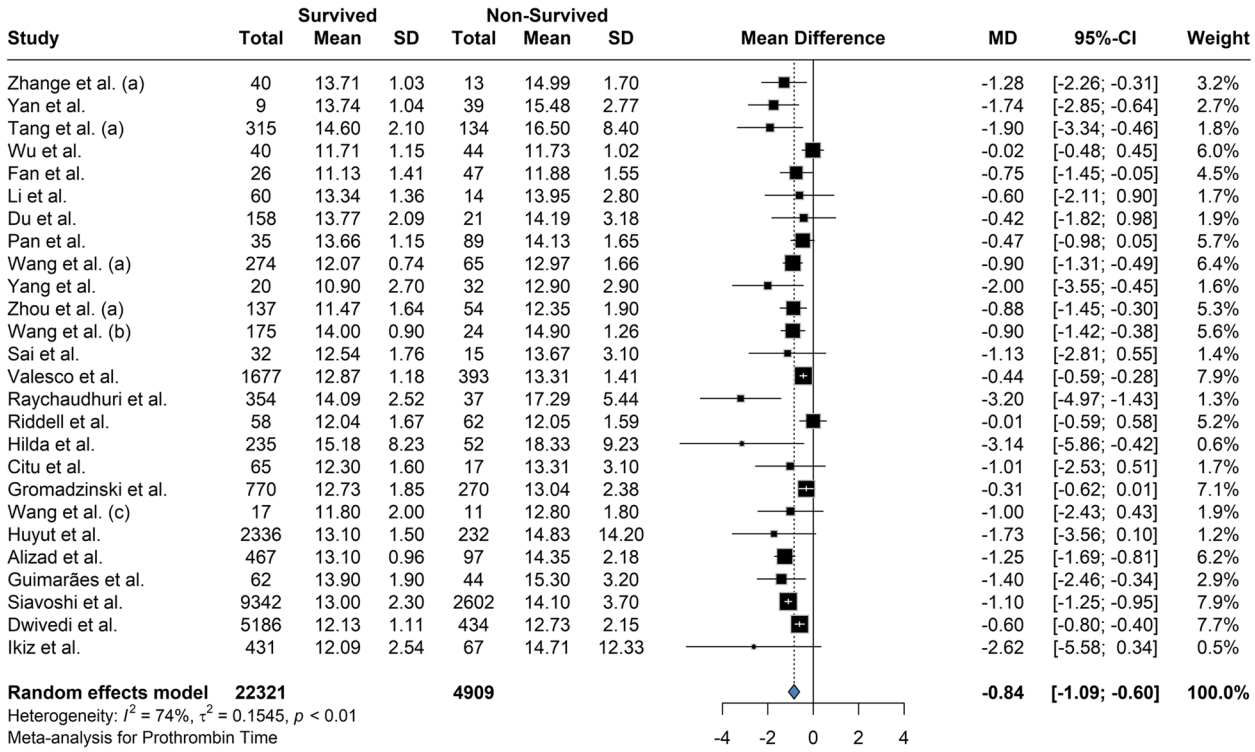

Fig. 7 The mean difference between PT count among the survivors and the deceased was -0.84 and was significantly lower among the survived

megakaryocytes to platelets and circulating plasma extracellular vehicles (EVs) [96–100]. Notably, plasma SARS-CoV-2 RNA has been strongly associated with increased mortality in COVID-19 patients. However, there is a gap in our knowledge of platelet non-canonical function and regeneration, so other research is essential to comprehensively grasp COVID-19 pathogenesis and identify increased thromboembolic risk [96–106].

Publication bias and variability in cut-off values for D-dimer (DD) measurement across studies may influence reported outcomes. Haemostasis tests, including D-dimers, may have limited usefulness because there is some differences in methodologies, antibody origin, detection methods, calibrators, and diagnostic thresholds among different laboratories.

Autopsy findings have shown that increased DD levels can be linked to fibrin deposits in the pulmonary extravascular space and alveoli. However, these elevations may not always be specific to intravascular fibrin formation. Elevated DD levels, along with increased neutrophil counts, have been identified as predictors of pulmonary embolism in COVID-19 patients. DD and fibrinogen (FIB) levels are elevated in both COVID-19 and thromboembolism, making them non-specific and unhelpful as single tests to distinguish between these conditions [107–110]. The lack of specificity might be attributed to the involvement of various systems, such as endothelial cells, complement activation, and hypo-fibrinolysis, in the abnormal coagulation processes during COVID-19, leading to changes that routine tests may not capture. Initial reports from China indicated a decreased activated partial thromboplastin time (APTT) as a marker of hypercoagulation [111–114]. However, later studies reported prolonged APTT, which could suggest deficiencies in clotting factors or the presence of inhibitors, such as heparin therapy. Another explanation could be the presence of antiphospholipid antibodies (aPL) or lupus anticoagulant (LA) which is observed in patients affected by COVID-19. Prolonged APTT may also be influenced by increased heparin therapy usage, especially in severe cases of COVID-19 [55, 115–117].

There are conflicting reports regarding fibrinogen levels and COVID-19 severity. Some studies show high fibrinogen levels in COVID-19 patients, while others report alternative findings. Severe COVID-19 infection may induce fibrinolysis shutdown, contributing to high levels of D-dimers and fibrinogen. However, other reports suggest increased plasmin-antiplasmin complexes and mild consumption coagulopathy in COVID-19 patients [118–123]. Tissue-type plasminogen activator elevation has also been found in severe COVID-19 cases. Fibrinogen plays a crucial role in linking coagulation, complement

system, and inflammation in COVID-19. Recent post-mortem studies have identified microvascular thrombi in multiple organs, indicating a hypercoagulable state with impaired fibrinolysis. However, the association between abnormal visco-elastometric testing (VET) patterns and clinical outcomes has not been demonstrated in patients affected by COVID-19 [124–128].

Routine coagulation tests which are taken at the time of admission can be helpful in differentiating severe and non-severe COVID-19 patients. The mechanisms of fibrinolysis shutdown may involve reduced fibrinolysis factors (plasminogen) and elevated inhibitors of fibrinolysis (such as α2-antiplasmin and plasminogen activator inhibitor PAI-1). Longitudinal observational studies are crucial for understanding COVID-19 infection dynamics and disease outcomes. Daily changes in relevant haemostasis parameters, including D-dimers and PT, have been observed in severe COVID-19 patients. However, it's essential to consider the variability in disease progression and admission timepoints when interpreting study results.

The current study has certain constraints. Included studies may have used different methods and kits for performing coagulation tests. Furthermore, the timing of tests could vary from study to study, while coagulation parameters may change throughout the disease. Additionally, the vaccination may affect the coagulation tests [129], and since study publications varied from 2020 to 2023, some of the differences may have contributed to vaccinations. Likewise, several underlying conditions, such as cancers, pregnancy, and receiving anticoagulants, may alter the results of coagulation tests. Unfortunately, detailed analyses on each condition were not allowed due to the incomplete reports of patients' conditions in the included articles, and readers should interpret our findings with caution. Further investigation, considering all the confounding factors mentioned, should be necessary to evaluate the findings of the current study.

Conclusion

The outcomes of our systematic review and meta-analysis indicate a significant decrease in the estimated pooled platelet count in deceased individuals compared to survivors. However, no significant differences were found in the pooled mean aPTT and fibrinogen levels between the deceased and survivor groups. Conversely, there were notable variations in the pooled estimated mean of INR, PT, and D-Dimer levels, which were significantly higher in the deceased group compared to those who survived.

Abbreviations

DIC	Disseminated intravascular coagulation
PRISMA	Preferred Reporting Items for Systematic reviews and Meta-Analyses
MeSH	Medical subject heading
PICO	Population, Intervention, Comparison, Outcome
SD	Standard deviation
aPTT	Activated partial thromboplastin time
PT	Prothrombin time
INR	International Normalized Ration
DD	D-dimer
EVs	Extracellular vehicles
FIB	Fibrinogen
aPL	Antiphospholipid antibodies
LA	Lupus anticoagulant
VET	Visco-elastometric testing

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12879-024-10229-y.

Supplementary Material 1.

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Not applicable.

Authors' contributions

S.N. and Z.P. designed the study and participated in the search and screening sections. A.A., S.H.I., and S.M.P. extracted and analysed data. S.S. and S.M.P. wrote the manuscript. All authors revised and modified the final manuscript.

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Not received.

Data availability

Data would be available based upon an eligible request to the corresponding author.

Declarations

Ethics approval and consent to participate

The protocol for this systematic review was approved by the ethics committee at IKHC ethics committee (Ethics code: IR.TUMS.IKHC.REC.1399.102).

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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