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EVALUATION OF PSEUDOTHROMBOCYTOPENIA ON AUTOMATED HEMATOLOGY ANALYZER USING VARIOUS ANTICOAGULANTS

Original Research

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ABSTRACT

Background: Pseudothrombocytopenia (PTCP) is an in vitro artifact primarily caused by anticoagulant-induced platelet clumping, leading to falsely low platelet counts and potential misdiagnosis. Ethylenediaminetetraacetic acid (EDTA) is frequently implicated due to its conformational effects on platelet membrane proteins, exposing hidden epitopes that trigger aggregation. Accurate platelet enumeration is essential for clinical decision-making, and the use of alternative anticoagulants may improve reliability, particularly in patients with suspected PTCP.

Objective: To compare platelet counts and platelet indices among different anticoagulated blood samples using EDTA, Heparin, Sodium Citrate, and Magnesium Sulfate.

Methods: This cross-sectional study included blood samples collected via antecubital venipuncture using strict aseptic techniques. Samples were added to BD Vacutainer tubes containing EDTA, Heparin, Sodium Citrate (3.2%), and custom-prepared Magnesium Sulfate (4.060 mOsmol/mL). All samples were analyzed within one hour using a Sysmex XP-100 three-part hematology analyzer and examined microscopically with Leishman-stained peripheral blood smears. Parameters included platelet count, mean platelet volume (MPV), platelet distribution width (PDW), red blood cell count (RBC), white blood cell count (WBC), and hemoglobin (Hb). Statistical analysis was performed using SPSS version 26. One-way ANOVA and Chi-square tests were applied with $p < 0.05$ considered statistically significant.

Results: EDTA showed the lowest mean platelet count (92.1 ± 36.3 $\times 10^9$/L) and the highest MPV (12.0 ± 1.3 fL) and PDW (18.4 ± 3.5 fL), indicating pronounced platelet aggregation. Magnesium Sulfate exhibited the highest mean platelet count (274.4 ± 57.6 $\times 10^9$/L) with 100.0% of samples in the normal range. Heparin (176.1 ± 55.9 $\times 10^9$/L) and Sodium Citrate (196.2 ± 60.4 $\times 10^9$/L) showed intermediate performance. Statistically significant differences were observed across all parameters ($p = 0.000$).

Conclusion: EDTA significantly underestimates platelet count due to PTCP. Magnesium Sulfate proved to be the most reliable anticoagulant for accurate platelet enumeration, while EDTA remained suitable for RBC and hemoglobin analysis.

Keywords: Anticoagulants, Ethylenediaminetetraacetic acid, Hematology, Magnesium Sulfate, Mean Platelet Volume, Platelet Count, Pseudothrombocytopenia.

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INTRODUCTION

Megakaryocytes, the large progenitor cells found within the bone marrow, give rise to platelets—small, anucleate cytoplasmic fragments essential for maintaining vascular integrity through their central role in hemostasis. These circulating elements, with a typical lifespan of seven to ten days, are replenished continuously through bone marrow production. Notably, platelet levels and functions are not static; they exhibit circadian variation, with activation patterns more prominent in the morning and a gradual increase in platelet count, approximately 5%, throughout the day (1). Hemostasis, the primary function of platelets, depends not only on their quantity but also on their ability to respond to vascular injury. When activated by stimuli such as von Willebrand factor (vWF) and collagen, platelets adhere to disrupted endothelial surfaces to initiate clot formation (2,3). Thrombocytopenia, defined as a platelet count below 150×10^9/L, is a frequent finding among critically ill patients and is associated with adverse clinical outcomes, including an elevated risk of bleeding (4). However, in some instances, a deceptively low platelet count is not due to true thrombocytopenia but rather to pseudothrombocytopenia (PTCP), an in vitro phenomenon caused by platelet clumping that interferes with automated platelet enumeration. PTCP arises due to pre-analytical and analytical factors, including the use of certain anticoagulants such as ethylenediaminetetraacetic acid (EDTA), heparin, sodium citrate, and magnesium sulfate (5). Among these, EDTA is most commonly implicated, leading to significant platelet aggregation by inducing conformational changes in platelet surface glycoproteins. These changes expose cryptic epitopes, prompting agglutination when autoantibodies of IgG, IgM, or IgA subclasses are present (6).

Although PTCP is often benign and not indicative of any underlying pathology, its occurrence has been documented in association with various conditions, including autoimmune disorders, pregnancy, viral infections, and adverse drug reactions. Importantly, PTCP can also be observed in otherwise healthy individuals (7). The clinical significance of PTCP lies in the potential for misdiagnosis and overtreatment. Failure to recognize PTCP can result in unnecessary interventions, such as platelet transfusions, bone marrow biopsies, or even erroneous suspicion of hematological malignancy (8). The challenge is further complicated in patients with cold agglutinin disease, where low-temperature-induced platelet clumping may skew results and mask the true platelet count (8,9). Laboratory identification of PTCP relies on detecting platelet clumps in a peripheral blood smear. If pseudothrombocytopenia is suspected, the use of an alternative anticoagulant—less likely to promote clumping—should be considered for repeat analysis. This step is crucial in differentiating PTCP from true thrombocytopenia and ensuring appropriate patient management (10). Despite the recognized clinical implications of PTCP, local data on this issue remain scarce. Therefore, the current study was designed to compare platelet counts and platelet indices across samples treated with different anticoagulants, with the objective of improving diagnostic accuracy and avoiding mismanagement due to artifactual thrombocytopenia.

METHODS

This cross-sectional analytical study was conducted on routine blood samples anticoagulated with tripotassium ethylenediaminetetraacetic acid (K_3EDTA) that showed positive flagging for platelet aggregates and thrombocytopenia on initial screening by an automated hematology analyzer (Sysmex XP-100). Confirmation of platelet clumping was performed through microscopic evaluation of Leishman-stained peripheral blood smears. Upon smear confirmation, additional blood samples were collected following informed consent. The study protocol was reviewed and approved by the institutional ethical committee in accordance with the Declaration of Helsinki. Participants were selected based on strict inclusion and exclusion criteria. Only those with EDTA-induced platelet aggregation and without any known cause of thrombocytopenia—determined through medical history, clinical examination, and review of medical records—were included. Patients with any identifiable cause of thrombocytopenia were excluded. Blood sampling was performed via antecubital venipuncture using standard aseptic technique, and samples were drawn into commercially available BD Vacutainer tubes prefilled with 3.2% sodium citrate and lithium heparin as anticoagulants. An additional tube was prepared using magnesium sulfate ($MgSO_4$), in which 0.3 mL of sterile $MgSO_4$ solution at a standardized concentration of 4.060 mOsmol/mL was preloaded under sterile conditions into a plain tube, followed by the addition of 2.7 mL of freshly drawn whole blood.

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All blood samples were processed within one hour of collection to prevent time-related changes. Hematological parameters including platelet count, mean platelet volume (MPV), platelet distribution width (PDW), red blood cell (RBC) count, white blood cell (WBC) count, and hemoglobin (Hb) levels were analyzed using the Sysmex XP-100 hematology analyzer, a three-part differential automated system. The analyzer was routinely calibrated, and all quality control (QC) requirements were fulfilled in compliance with manufacturer recommendations throughout the study period. Internal QC protocols were followed, and only validated samples were included in the analysis. Peripheral blood smears from all anticoagulant tubes were examined microscopically to assess the presence or resolution of platelet aggregates and to corroborate the analyzer findings. This review ensured differentiation between true thrombocytopenia and pseudothrombocytopenia induced by various anticoagulants. The methodology adhered to established scientific protocols, incorporating minor modifications based on prior literature (7,8). Data were analyzed using IBM SPSS Statistics version 26. Descriptive statistics such as mean and standard deviation were computed for continuous variables. One-way analysis of variance (ANOVA) was used to assess differences in hematological parameters across the different anticoagulants. Categorical variables such as platelet count categories were expressed as frequencies and percentages. The Chi-square test was applied to evaluate associations between anticoagulant types and platelet category distributions. A p-value less than 0.05 was considered statistically significant.

RESULTS

The comparison of anticoagulants—EDTA, Heparin, Sodium Citrate, and Magnesium Sulfate—revealed statistically significant differences ($p < 0.05$) across all hematological parameters evaluated. Red blood cell (RBC) count varied significantly among anticoagulants ($p = 0.000$), with EDTA and Heparin both showing the highest mean values (4.9 ± 0.6 and $4.9 \pm 0.5 \times 10^{12}$/L, respectively). Magnesium Sulfate demonstrated a slightly lower count ($4.8 \pm 0.5 \times 10^{12}$/L), while Sodium Citrate showed the lowest RBC count ($4.4 \pm 0.5 \times 10^{12}$/L), likely due to dilutional effects. White blood cell (WBC) counts were also significantly different ($p = 0.009$). EDTA yielded the highest mean WBC count ($8.7 \pm 2.1 \times 10^{9}$/L), followed by both Sodium Citrate and Magnesium Sulfate (7.7 ± 2.0 and $7.7 \pm 1.8 \times 10^{9}$/L, respectively). Heparin resulted in the lowest mean WBC count ($6.9 \pm 3.2 \times 10^{9}$/L), possibly indicating some degree of leukocyte degradation or clumping. Hemoglobin (Hb) levels differed significantly ($p = 0.000$), with the highest values seen in Heparin (14.4 ± 1.5 g/dL) and EDTA (14.2 ± 1.8 g/dL), followed closely by Magnesium Sulfate (14.1 ± 1.7 g/dL). Sodium Citrate demonstrated the lowest Hb concentration (12.8 ± 1.7 g/dL), reinforcing its dilutional effect. Platelet counts showed highly significant differences across anticoagulants ($p = 0.000$). EDTA demonstrated the lowest mean platelet count ($92.1 \pm 36.3 \times 10^{9}$/L), consistent with EDTA-induced pseudothrombocytopenia. Heparin ($176.1 \pm 55.9 \times 10^{9}$/L) and Sodium Citrate ($196.2 \pm 60.4 \times 10^{9}$/L) showed notably higher values, while Magnesium Sulfate recorded the highest mean platelet count ($274.4 \pm 57.6 \times 10^{9}$/L), suggesting superior prevention of in vitro platelet aggregation. Platelet distribution width (PDW) was significantly elevated in EDTA (18.4 ± 3.5 fL), followed by Heparin (15.7 ± 2.5 fL), Sodium Citrate (14.3 ± 2.4 fL), and was lowest in Magnesium Sulfate (12.2 ± 1.8 fL), indicating reduced platelet activation and clumping in the latter. Mean platelet volume (MPV) followed a similar trend, with EDTA again showing the highest mean (12.0 ± 1.3 fL), followed by Heparin (10.6 ± 1.2 fL), Sodium Citrate (9.8 ± 1.1 fL), and Magnesium Sulfate demonstrating the lowest MPV (8.9 ± 1.0 fL), further supporting its role in minimizing platelet activation.

Analysis of platelet category distribution revealed a statistically significant difference across anticoagulants ($p = 0.000$). EDTA showed the highest rate of underestimation, with only 5.0% of samples categorized as having normal platelet counts, while 47.5% were classified as mild, 32.5% as moderate, and 15.0% as severe thrombocytopenia. In contrast, Heparin demonstrated normal platelet levels in 75.0% of cases, followed by Sodium Citrate (82.5%) and Magnesium Sulfate (100.0%), which had no cases of thrombocytopenia, confirming its reliability for accurate platelet estimation. To address subgroup-level analysis, the distribution of platelet categories across the four anticoagulants—EDTA, Heparin, Sodium Citrate, and Magnesium Sulfate—was quantitatively compared. This subgroup breakdown revealed that EDTA had the highest proportion of falsely categorized thrombocytopenia cases, with only 5.0% of samples within the normal range, while the majority exhibited mild to severe underestimation of platelet counts. In contrast, Magnesium Sulfate showed 100.0% of samples falling within the normal range, followed by Sodium Citrate at 82.5% and Heparin at 75.0%. This stratified analysis further validated the superior performance of Magnesium Sulfate in preventing platelet clumping and pseudothrombocytopenia. Although smear review was performed in all cases, quantitative scoring of platelet aggregates or interobserver variability assessment was not included in the study design. Inclusion of such metrics could enhance reproducibility and the objectivity of morphological assessment in future investigations.

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Table 1: Distribution of Platelet Categories across Different Anticoagulants

Anticoagulant		PLT CAT				p-value
		Normal	Mild	Moderate	Severe	
EDTA	n	2	19	13	6	0.000*
	%	5.0%	47.5%	32.5%	15.0%	
Hep	n	30	7	2	1	
	%	75.0%	17.5%	5.0%	2.5%	
NaCit	n	33	5	1	1	
	%	82.5%	12.5%	2.5%	2.5%	
MgSo4	n	40	0	0	0	
	%	100.0%	0.0%	0.0%	0.0%	
Total	n	105	31	16	8	
	%	65.6%	19.4%	10.0%	5.0%	

Table 2: Comparison of Blood Parameters across Different Anticoagulants

Blood parameters	Anticoagulant	Mean	SD	Minimum	Maximum	p-value
RBC	EDTA	4.9	0.6	3.5	6.1	0.000*
	Hep	4.9	0.5	3.6	6.1	
	NaCit	4.4	0.5	2.8	5.5	
	MgSo4	4.8	0.5	3.5	6.1	
WBC	EDTA	8.7	2.1	5.0	13.3	0.009*
	Hep	6.9	3.2	2.9	23.2	
	NaCit	7.7	2.0	4.5	11.9	
	MgSo4	7.7	1.8	4.5	11.9	
HB	EDTA	14.2	1.8	10.4	17.6	0.000*
	Hep	14.4	1.5	10.8	17.6	
	NaCit	12.8	1.7	6.5	14.9	
	MgSo4	14.1	1.7	10.5	16.5	
PLT	EDTA	92.1	36.3	22.0	150.0	0.000*
	Hep	176.1	55.9	40.0	300.0	

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Blood parameters	Anticoagulant	Mean	SD	Minimum	Maximum	p-value
	NaCit	196.2	60.4	45.0	300.0	
	MgSo4	274.4	57.6	170.0	499.0	
PDW	EDTA	18.4	3.5	12.4	25.1	0.000*
	Hep	15.7	2.5	10.0	19.1	
	NaCit	14.3	2.4	10.2	18.3	
	MgSo4	12.2	1.8	9.5	15.5	
MPV	EDTA	12.0	1.3	9.6	13.9	0.000*
	Hep	10.6	1.2	7.8	12.6	
	NaCit	9.8	1.1	7.3	11.9	
	MgSo4	8.9	1.0	6.7	10.8	

Table 3: Platelet Category Distribution by Anticoagulant

Anticoagulant	Normal	Mild	Moderate	Severe	Total Samples
EDTA	5	47.5	32.5	15	40
Heparin	75	17.5	5	2.5	40
Sodium Citrate	82.5	12.5	2.5	2.5	40
Magnesium Sulfate	100	0	0	0	40

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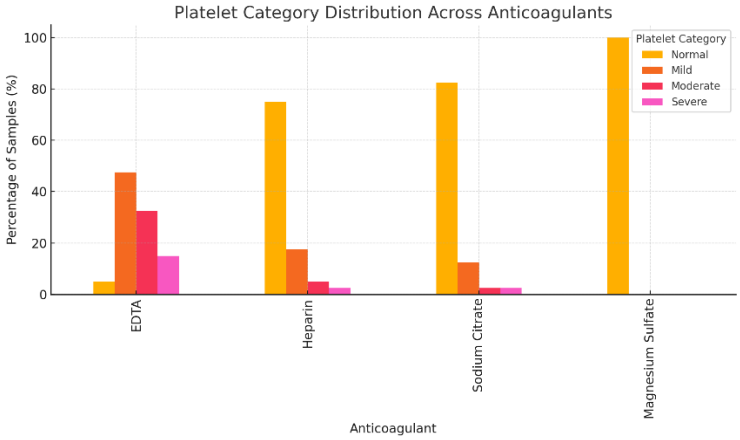

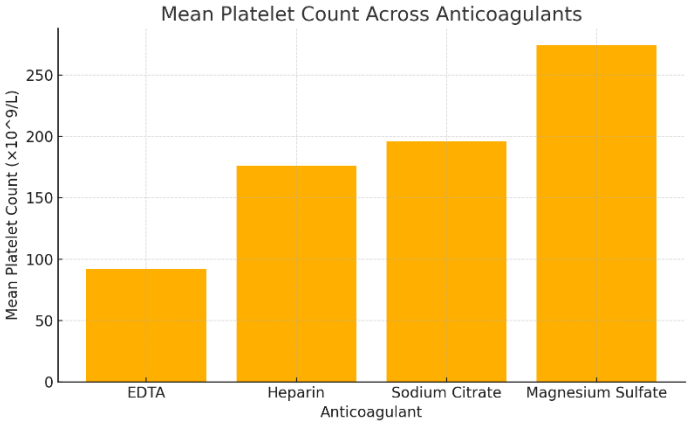

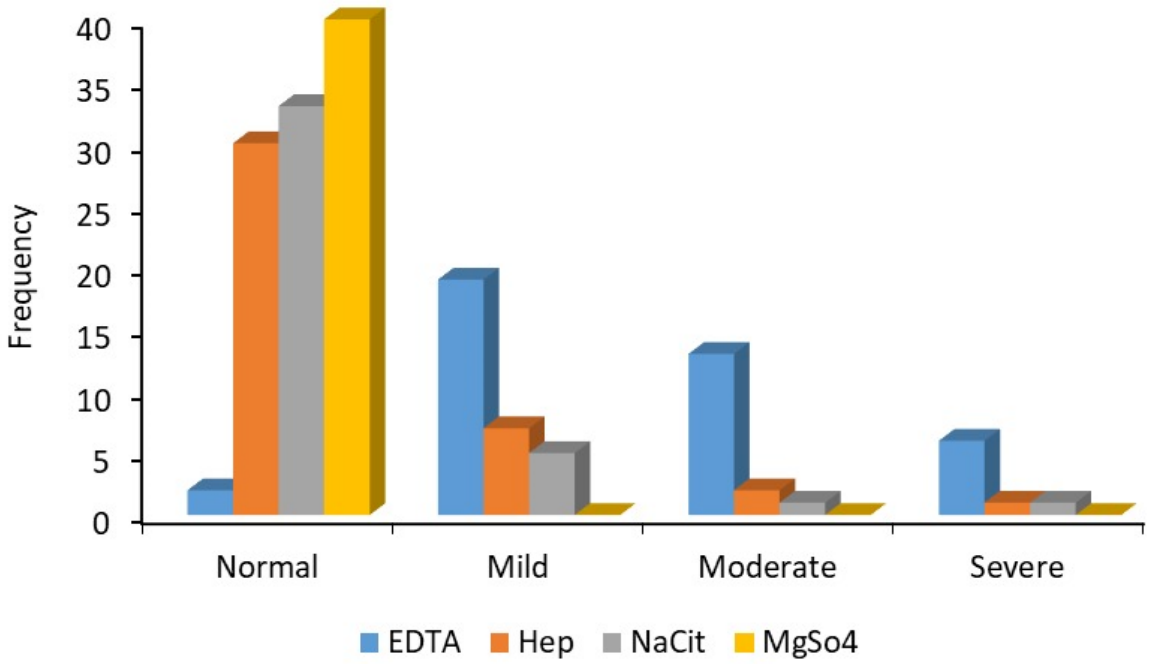

DISCUSSION

The present study demonstrated significant variability in hematological parameters, particularly platelet indices, across different anticoagulants. EDTA, widely used in routine hematology, was associated with the most profound alterations in platelet count, platelet distribution width (PDW), and mean platelet volume (MPV), consistent with the phenomenon of pseudothrombocytopenia (PTCP). These findings align with existing evidence that EDTA can induce conformational changes in platelet membrane glycoproteins, unmasking cryptic epitopes that bind naturally occurring or acquired antibodies, leading to in vitro platelet clumping (11). The significantly lower mean platelet count observed with EDTA (92.1 ± 36.3) compared to Heparin, Sodium Citrate, and Magnesium Sulfate underscores its unreliability for platelet estimation in susceptible individuals. Similar patterns were observed in earlier investigations, which identified EDTA as the most common causative agent for PTCP, while Heparin and Citrate demonstrated a markedly lower incidence of such artifacts (12). Among all anticoagulants tested, Magnesium Sulfate yielded the highest mean platelet count (274.4 ± 57.6), the lowest PDW (12.2 ± 1.8), and the lowest MPV (8.9 ± 1.0), suggesting optimal preservation of platelet morphology and function (13). These observations support findings from prior studies that also recognized Magnesium Sulfate as an effective alternative for

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preventing in vitro platelet aggregation and mitigating PTCP. The absence of any cases of thrombocytopenia in $MgSO_4$-anticoagulated samples further reinforces its potential as a superior anticoagulant, particularly in patients where EDTA-induced PTCP is suspected (14).

Sodium Citrate performed well in preserving platelet count and indices, with 82.5% of samples falling within the normal platelet range. Although Citrate has been occasionally associated with PTCP in previous reports, its performance in this study supports its recommendation as a valid alternative to EDTA, especially when processed promptly to minimize dilutional effects (15). The addition of Tris-buffer or pyridoxal 5-phosphate to Citrate has been explored in literature to further neutralize its adverse interactions, though this was beyond the scope of the current study (16). EDTA also showed the highest PDW and MPV values, reflecting increased platelet activation and heterogeneity. These parameters, when elevated, are indicative of altered platelet morphology, which may be misleading in clinical decision-making (17). The high frequency of mild to severe thrombocytopenia observed in EDTA samples (95.0%) reaffirms its role in falsely categorizing patients under thrombocytopenic states, potentially leading to unnecessary investigations or interventions. Additionally, EDTA and Heparin exhibited the highest mean RBC and WBC counts, consistent with prior findings that these anticoagulants exert minimal dilutional impact on cellular elements (18). Sodium Citrate, on the other hand, showed a relatively lower RBC count and hemoglobin levels, likely due to its volume-dependent dilutional property. While not affecting platelet morphology, this characteristic may influence red cell parameters and should be taken into account when interpreting results from Citrate-anticoagulated blood (19).

One of the strengths of this study lies in its direct comparison of commonly used anticoagulants under standardized conditions, with parallel analysis of both automated results and peripheral smear morphology. The addition of Magnesium Sulfate, although not routinely employed in clinical practice, offered valuable insights into its diagnostic potential. However, limitations were also present. The study did not evaluate the time-dependent effects on platelet aggregation, which could influence the degree of PTCP, particularly in EDTA samples. Furthermore, no alternative analytical techniques, such as fluorescence-based platelet enumeration or flow cytometry, were employed to validate findings, primarily due to financial constraints. The study also lacked a formal assessment of interobserver variability or quantitative scoring in smear review, which could have strengthened morphological evaluations. Future research should consider longitudinal sampling to assess the temporal dynamics of platelet aggregation and explore advanced analytical platforms for more accurate platelet quantification. Incorporating subgroup analyses based on patient demographics and clinical characteristics could provide further insight into individual susceptibility to PTCP. Moreover, standardizing smear grading and involving multiple blinded reviewers would improve the reproducibility and diagnostic reliability of smear-based evaluations (20). Overall, the findings affirm that while EDTA remains standard in hematology, its limitations in certain clinical contexts necessitate consideration of alternative anticoagulants. Magnesium Sulfate, in particular, emerges as a promising option for accurate platelet assessment, followed by Sodium Citrate and Heparin, each with their respective advantages and limitations.

CONCLUSION

This study concluded that EDTA, although widely used, leads to significant underestimation of platelet counts due to pseudothrombocytopenia, as evidenced by elevated platelet indices suggestive of in vitro aggregation. Among the anticoagulants evaluated, magnesium sulfate emerged as the most effective in providing accurate and stable platelet estimations, making it a reliable alternative in cases where EDTA-induced artifacts are suspected. While EDTA remained dependable for red blood cell and hemoglobin measurements, white blood cell counts were best preserved in EDTA samples. These findings emphasize the importance of selecting an appropriate anticoagulant tailored to the parameter being assessed, and support the use of magnesium sulfate in clinical scenarios requiring accurate platelet evaluation.

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Saima Zahir	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Qurrat-Ul-Ain	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Rimsha Bashir	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Shumaila Asghar	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published

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