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FREQUENCY OF VENTRICULAR ARRHYTHMIAS AFTER ACUTE MYOCARDIAL INFARCTION AND ITS ASSOCIATION WITH HYPOKALEMIA

Original Research

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ABSTRACT

Background: Ventricular arrhythmias are among the most life-threatening complications following acute myocardial infarction (AMI), frequently contributing to early mortality. Hypokalemia, a common electrolyte disturbance during AMI, may further destabilize cardiac electrical activity and predispose to fatal dysrhythmias. Early recognition and correction of hypokalemia are therefore critical in minimizing arrhythmic events and improving patient outcomes.

Objective: To determine the frequency of ventricular arrhythmias in patients presenting with acute myocardial infarction and to assess their association with hypokalemia.

Methods: A cross-sectional comparative study was conducted in the Coronary Care and Intensive Care Units of Gulab Devi Teaching Hospital, Lahore. A total of 100 patients with confirmed AMI, irrespective of gender, were enrolled through purposive sampling. Demographic details, type of myocardial infarction, serum potassium levels, and predisposing factors were recorded using a structured proforma. Electrocardiograms were evaluated to identify ventricular tachycardia (VT), ventricular fibrillation (VF), and premature ventricular contractions (PVCs). Data were analyzed using SPSS version 26. Quantitative variables such as age, height, weight, and serum potassium were expressed as mean ± SD, while qualitative variables were presented as frequencies and percentages. The Chi-square test was applied to assess the association between hypokalemia and ventricular arrhythmias, considering $p < 0.05$ statistically significant.

Results: Of the 100 AMI patients, 66 were male and 34 female, with a mean age of 57.82 ± 11.21 years. The mean potassium level was 4.13 ± 0.86 mmol/L. Hypokalemia (serum potassium < 3.4 mmol/L) was found in 13% of patients. Ventricular arrhythmias were observed in 20% of participants, including VT (9%), VF (6%), and PVCs (5%). A significant association was found between hypokalemia and ventricular arrhythmias ($p = 0.000$), while gender showed no significant relationship ($p = 0.916$). Current smoking also demonstrated a strong correlation ($p = 0.002$).

Conclusion: The study concluded that hypokalemia was significantly associated with the development of ventricular arrhythmias in patients with acute myocardial infarction, underscoring the importance of routine electrolyte monitoring and timely correction to prevent life-threatening cardiac events.

Keywords: Acute Myocardial Infarction, Electrolyte Imbalance, Hypokalemia, Myocardial Ischemia, Potassium, Ventricular Arrhythmia, Ventricular Fibrillation.

INTRODUCTION

Acute Myocardial Infarction (AMI) represents a critical manifestation of ischemic heart disease (IHD), resulting from the necrosis of myocardial tissue due to prolonged ischemia and hypoxia of the coronary arteries (1). It is characterized by elevated serum myocardial enzyme levels, persistent electrocardiographic (ECG) abnormalities, and complications such as arrhythmia, cardiogenic shock, and heart failure, which contribute substantially to its fatality. Despite advances in diagnostic and interventional strategies, AMI remains a leading cause of morbidity and mortality worldwide, particularly due to life-threatening ventricular arrhythmias, including ventricular tachycardia (VT) and ventricular fibrillation (VF). These disturbances are responsible for 20–50% of sudden cardiac deaths in the acute phase of infarction and often develop within 48 hours of symptom onset (2,3). Ventricular arrhythmias arise as a consequence of post-infarction ventricular remodeling, where necrotic myocardium is replaced by fibrotic scar tissue, disrupting the uniform propagation of electrical impulses. Such structural heterogeneity fosters re-entrant circuits that precipitate abnormal automaticity and triggered activity (4). Various clinical determinants, such as atrial fibrillation, cardiogenic shock, chronic kidney disease, left main stenosis, low serum potassium, and incomplete ST-segment resolution, have been identified as independent predictors of ventricular arrhythmogenesis following AMI (5). However, the precise mechanisms underlying these arrhythmias and their interaction with metabolic disturbances remain incompletely understood. Among the biochemical factors implicated in arrhythmogenesis, hypokalemia has received significant attention for its direct influence on myocardial excitability. Potassium, an essential intracellular cation, plays a pivotal role in maintaining resting membrane potential and electrical stability of cardiac tissue. In myocardial ischemia, excessive catecholamine discharge stimulates Na^+/K^+-ATPase activity, driving potassium influx into cells and leading to a transient decline in serum potassium concentration (6,7). Hypokalemia—defined as serum potassium below 3.5 mEq/L—reduces cardiac repolarization reserve and elevates intracellular calcium, thereby predisposing the myocardium to premature depolarizations and potentially fatal ventricular arrhythmias (8).

Clinical investigations have consistently demonstrated a strong association between low serum potassium and the occurrence of ventricular arrhythmias during AMI. In a retrospective study, patients with hypokalemia exhibited a 27.3% incidence of ventricular arrhythmias within the first 24 hours of infarction, compared to only 7.5% among normokalemic individuals ($p<0.001$) (9). Similarly, a study reported that potassium levels below 3.5 mEq/L were independently correlated with both short- and long-term mortality after AMI (10). These findings support the recommendation that serum potassium levels should be maintained at or above 4.0 mEq/L in patients with acute coronary syndromes to mitigate arrhythmic risks (11). The electrophysiological basis of these disturbances lies in ischemia-induced depolarization, impaired impulse conduction, and increased heterogeneity in repolarization. QT interval prolongation and dispersion on ECG have emerged as key non-invasive indicators of ventricular electrical instability, correlating with an elevated risk of VT/VF and sudden cardiac death (12,13). Moreover, reperfusion therapy, while life-saving, may transiently exacerbate arrhythmias due to rapid ionic shifts and catecholamine surges. Hence, timely recognition and management of electrolyte imbalances remain integral to post-AMI care. Despite the wealth of evidence linking hypokalemia to arrhythmic complications, the true burden of ventricular arrhythmias among hypokalemic versus normokalemic AMI patients, particularly in South Asian populations, remains underexplored. Most available studies focus narrowly on VT or VF rather than encompassing all forms of ventricular dysrhythmias, leaving a significant gap in understanding the broader arrhythmic spectrum and its prognostic implications (14,15). Furthermore, variations in dietary potassium intake, diuretic use, and delayed hospital presentation in low- and middle-income regions may further influence these outcomes. Therefore, this study aims to determine the frequency of ventricular arrhythmias in patients presenting with acute myocardial infarction and to evaluate their association with hypokalemia. By delineating this relationship, the research intends to aid clinicians in early identification of high-risk individuals and implement preventive strategies to reduce mortality associated with post-infarction arrhythmias.

METHODS

The present investigation was designed as a cross-sectional comparative study conducted in the Department of Cardiology at Gulab Devi Teaching Hospital, Lahore, over a duration of six months following approval from the Institutional Review Board (IRB). The study population comprised patients admitted to the coronary care unit (CCU) who presented with acute myocardial infarction (AMI). A non-

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probability purposive sampling technique was employed to recruit participants meeting the inclusion and exclusion criteria. Patients of either gender aged between 18 and 60 years who were diagnosed with AMI based on clinical assessment, ECG findings, and biochemical markers were included. Individuals with congenital heart disease, valvular or pericardial diseases, cardiomyopathies, or any form of renal impairment were excluded to minimize confounding influences on serum electrolyte levels and cardiac rhythm interpretation. The sample size was calculated using Cochran's formula ($n = Z^2Pq/d^2$), where $Z = 1.96$, $P = 16.5\%$ (taken from a parent study), $q = 1 – P$, and $d = 0.05$, yielding a required sample size of 211 participants. However, the number of patients enrolled during the data collection period was 100. Data were collected using a pre-structured proforma developed for the study. Patient information, including demographic variables, clinical details, and laboratory parameters, was extracted from hospital records. Electrocardiograms (ECGs) were reviewed to identify ventricular arrhythmias, while serum potassium levels were obtained from the patients' electrolyte profiles documented in their files. Predisposing factors for arrhythmias were also recorded. Data collection was carried out by the investigator under clinical supervision to ensure accuracy and consistency. The study variables included age, gender, type of myocardial infarction, potassium concentration, and the presence or absence of ventricular arrhythmias. The research adhered to ethical principles outlined in the Declaration of Helsinki. Ethical approval was obtained from the Ethical Review Committee of Gulab Devi Teaching Hospital, Lahore and informed written consent was obtained from all participants prior to inclusion in the study. Confidentiality of all patient data was maintained throughout the research process. Data analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 26. Descriptive statistics, including mean, median, mode, and standard deviation, were computed for quantitative variables, while qualitative variables were expressed as frequencies and percentages. Comparative and frequency analyses were conducted using appropriate statistical tests, with results presented in the form of tables, charts, and graphs to facilitate interpretation and visualization.

RESULTS

A total of 100 patients with acute myocardial infarction were analyzed. Males comprised 66.0% (n=66) and females 34.0% (n=34). Age ranged from 35 to 80 years (mean 57.82 ± 11.21; median 58.5; mode 60). Height ranged from 152.88 to 190.10 cm (mean 173.52 ± 10.48; median 177.49; mode 182.88). Weight ranged from 54.00 to 78.00 kg (mean 65.97 ± 5.57; median 67.50; mode 69.00). Body mass index (BMI) ranged from 17.70 to 29.10 kg/m² (mean 21.96 ± 2.02; median 21.50), with multiple modes; 77.0% fell between 17.70–23.39 kg/m² and 23.0% between 23.40–29.10 kg/m². Regarding myocardial infarction subtype, 45.0% (n=45) had ST-elevation MI (STEMI) and 55.0% (n=55) had non-ST-elevation MI (NSTEMI). Cardiac troponin I was raised in 100% of cases (n=100). Serum potassium ranged from 2.60 to 6.90 mmol/L (mean 4.13 ± 0.87; median 3.90; mode 3.60). Based on distribution classes, 79.0% (n=79) were between 2.60–4.74 mmol/L, 20.0% (n=20) between 4.75–6.89 mmol/L, and 1.0% (n=1) at ≥6.90 mmol/L. By categorical status, hypokalemia was present in 13.0% (n=13) and absent in 87.0% (n=87). For predisposing factors, current smoking was reported in 60.0% (n=60), diabetes in 67.0% (n=67), hypertension in 79.0% (n=79), electrolyte imbalance (overall, as recorded by clinicians) in 21.0% (n=21), heart failure in 42.0% (n=42), and hyperlipidemia in 8.0% (n=8). Ventricular arrhythmias occurred in 20.0% (n=20) of patients; 80.0% (n=80) had no ventricular arrhythmia. Among arrhythmia phenotypes, ventricular tachycardia accounted for 9.0% (n=9), ventricular fibrillation 6.0% (n=6), and premature ventricular contractions 5.0% (n=5); 80.0% (n=80) had none. The distribution of ventricular arrhythmias by sex showed 13 males and 7 females among the 20 arrhythmic cases, yielding a non-significant association (χ^2=0.011, df=1, p=0.916). The distribution by hypokalemia demonstrated that all hypokalemic patients (n=13/13; 100%) had ventricular arrhythmias, whereas among normokalemic patients 8.0% (n=7/87) had ventricular arrhythmias and 92.0% (n=80/87) did not; the association was statistically significant (χ^2=59.770, df=1, p<0.001). The distribution by current smoking showed 18 smokers and 2 non-smokers among the 20 arrhythmic cases; the association was statistically significant (χ^2=9.375, df=1, p=0.002).

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Table 1: Descriptive and Frequency Distribution of Demographic and Anthropometric Characteristics of Myocardial Infarction Patients (n = 100)

Variable	Category / Statistical Parameter	Frequency	Percent (%)	Mean	Median	Mode	Std. Deviation	Skewness	Std. Error of Skewness	Range	Minimum	Maximum
Gender	Male	66	66.0									
	Female	34	34.0									
	Total	100	100.0									
Age (years)	—			57.82	58.5	60.00	11.21091	0.227	0.241	45	35	80
	35 – 49	26	26.0									
	50 – 64	47	47.0									
	65 – 79	23	23.0									
	80.00+	4	4.0									
Height (cm)	—			173.5166	177.4900	182.88	10.48197	-0.272	0.241	37.22	152.88	190.10
	152 – 171	41	41.0									
	171 – 191	59	59.0									
	Total	100	100.0									
Weight (kg)	—			65.9700	67.5000	69.00	5.56950	-0.481	0.241	24.00	54.00	78.00
	54 – 65	39	39.0									
	66 – 78	60	60.0									
	78.00+	1	1.0									
	Total	100	100.0									
Body Mass Index (kg/m²)	—			21.9619	21.5000	21.10[a]	2.01728	0.929	0.241	11.40	17.70	29.10
	17.70 – 23.39	77	77.0									
	23.40 – 29.10	23	23.0									
	Total	100	100.0									

[a]Multiple modes exist; the smallest value (21.10) is shown.

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Table 2: Frequency Distribution of Myocardial Infarction Type, Cardiac Marker (Trop I), and Statistical Analysis of Serum Potassium Levels in Myocardial Infarction Patients (n = 100)

Variable	Category / Statistical Parameter	Frequency	Percent (%)	Median	Mode	Std. Deviation	Skewness	Std. Error of Skewness	Range	Minimum	Maximum
Type of Myocardial Infarction	STEMI	45	45.0								
	NSTEMI	55	55.0								
	Total	100	100.0								
Cardiac Marker (Trop I)	Raised	100	100.0								
Serum Potassium Level (mmol/L)	—	—	—	3.9000	3.60	0.86637	1.032	0.241	4.30	2.60	6.90

Table 3: Frequency Distribution of Clinical Characteristics, Predisposing Factors, and Outcome Variables in Myocardial Infarction Patients (n = 100)

Variable	Category	Frequency	Percent (%)
Serum Potassium Level (mmol/L)	2.60 – 4.74	79	79.0
	4.75 – 6.89	20	20.0
	6.90+	1	1.0
	Total	100	100.0
Current Smoker	Yes	60	60.0
	No	40	40.0
	Total	100	100.0
Diabetic Patients	Yes	67	67.0
	No	33	33.0
	Total	100	100.0
Hypertensive Patients	Yes	79	79.0
	No	21	21.0
	Total	100	100.0
Electrolyte Imbalance	Yes	21	21.0
	No	79	79.0

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Variable	Category	Frequency	Percent (%)
	Total	100	100.0
Heart Failure	Yes	42	42.0
	No	58	58.0
	Total	100	100.0
Hyperlipidemia	Yes	8	8.0
	No	92	92.0
	Total	100	100.0
Hypokalemic Patients (Outcome Variable)	Yes	13	13.0
	No	87	87.0
	Total	100	100.0
Ventricular Arrhythmias (Outcome Variable)	Yes	20	20.0
	No	80	80.0
	Total	100	100.0
Type of Ventricular Arrhythmias	Ventricular Tachycardia (VT)	9	9.0
	Ventricular Fibrillation (VF)	6	6.0
	Premature Ventricular Contraction (PVC)	5	5.0
	No Ventricular Arrhythmia	80	80.0
	Total	100	100.0

Table 4: Association Between Gender and Ventricular Arrhythmias in Myocardial Infarction Patients (n = 100)

Variable	Ventricular Arrhythmias (Yes)	Ventricular Arrhythmias (No)	Total
Male	13	53	66
Female	7	27	34
Total	20	80	100
Pearson Chi-Square			
Statistical Test	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	0.011[a]	1	0.916

Table 5: Association Between Ventricular Arrhythmias and Hypokalemia in Myocardial Infarction Patients (n = 100)

Variable	Ventricular Arrhythmias (Yes)	Ventricular Arrhythmias (No)	Total
Hypokalemia: Yes	13	0	13
Hypokalemia: No	7	80	87

Variable	Ventricular Arrhythmias (Yes)		Ventricular Arrhythmias (No)	Total
Total	20		80	100
Pearson Chi-Square				
Statistical Test	Value	Df	Asymp. Sig. (2-sided)	
Pearson Chi-Square	59.770[a]	1	0.000	

[a]Indicates a statistically significant association between hypokalemia and ventricular arrhythmias ($p < 0.05$).

Table 6: Association Between Ventricular Arrhythmias and Current Smoking Status in Myocardial Infarction Patients (n = 100)

Variable	Ventricular Arrhythmias (Yes)		Ventricular Arrhythmias (No)	Total
Current Smoker: Yes	18		42	60
Current Smoker: No	2		38	40
Total	20		80	100
Pearson Chi-Square				
Statistical Test	Value	Df	Asymp. Sig. (2-sided)	
Pearson Chi-Square	9.375[a]	1	0.002	

[a]Indicates a statistically significant association between current smoking and ventricular arrhythmias ($p < 0.05$).

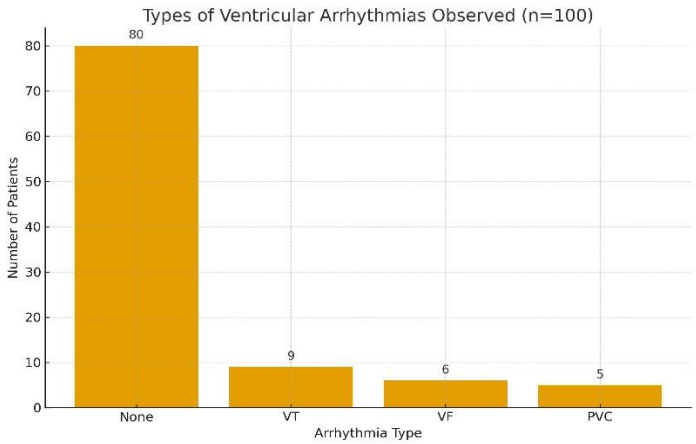

Figure 2 Types of Ventricular Arrhythmias Observed (n=100)

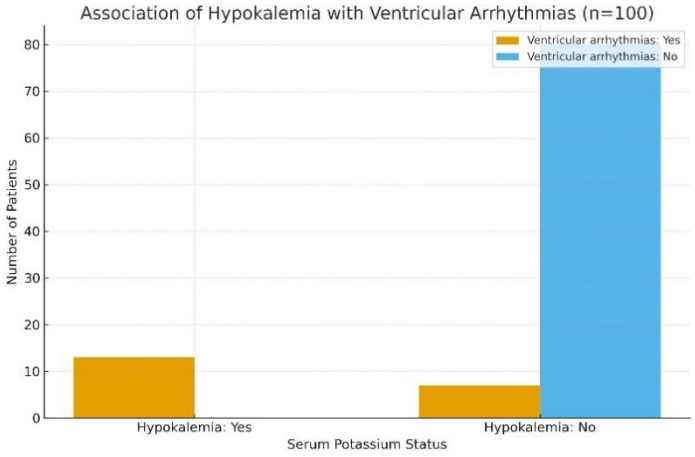

Figure 2 Association of Hypokalemia with Ventricular Arrhythmias (n=100)

DISCUSSION

The study demonstrated that ventricular arrhythmias occurred in one-fifth of patients with acute myocardial infarction, with ventricular tachycardia being the most frequent phenotype, followed by ventricular fibrillation and premature ventricular contractions. Hypokalemia was present in 13% of the cohort and showed a strong bivariate association with ventricular arrhythmias, whereas sex was not associated and current smoking was positively associated. These observations aligned with prior evidence that malignant ventricular tachyarrhythmias constitute a principal mechanism of early mortality after myocardial infarction and that low serum potassium

exacerbates electrical instability in the infarcted myocardium (11). The direction and magnitude of the observed association between hypokalemia and arrhythmias were concordant with multiple clinical investigations reporting higher odds of ventricular ectopy and sustained ventricular tachyarrhythmias at potassium concentrations below normal physiological targets, particularly in the first 24–48 hours after symptom onset (12). The pattern of arrhythmia burden in the present cohort paralleled historical coronary care unit series in which ventricular tachycardia and ventricular fibrillation clustered early after infarction and contributed disproportionately to in-hospital deaths, with risk further amplified among those arriving within the first hour of symptoms and in those requiring resuscitative transport (13). The finding that smokers were overrepresented among patients with ventricular arrhythmias supported prior reports linking adrenergic drive and ischemia-related dispersion of repolarization to ventricular instability, while the null association with sex echoed studies in which sex differences diminished after accounting for ischemic substrate and autonomic tone (14). Although not measured here, prior literature has emphasized that reduced left ventricular ejection fraction remains a dominant substrate for re-entrant circuits in the late post-infarct period, and that electrolyte perturbations, particularly hypokalemia, can lower the threshold for triggered activity and facilitate re-entry by prolonging repolarization and slowing conduction (15).

Clinical implications in resource-constrained settings were direct. First, routine early electrolyte surveillance with expedited correction to contemporary targets—often at or above 4.0 mmol/L for potassium in acute coronary syndromes—appeared justified to mitigate arrhythmic risk, particularly within the first 24–48 hours when autonomic activation and reperfusion shifts are most pronounced (16,17). Second, telemetry prioritization for hypokalemic and actively smoking patients may improve detection of actionable rhythm disturbances, while integration of smoking-cessation counseling into post-infarct care may reduce recurrent adrenergic triggers. Third, standardized order sets that pair potassium repletion with magnesium assessment are sensible, given the synergistic role of magnesium in stabilizing myocardial membranes and the frequent co-occurrence of deficits during acute ischemia (18,19). The study possessed notable strengths. It focused on clinically pragmatic variables obtainable at admission, used uniform ECG review for arrhythmia ascertainment, and reported a complete distribution of arrhythmia phenotypes pertinent to bedside decision-making. At the same time, several limitations tempered inference. The design was cross-sectional and unadjusted; as such, temporality and causality could not be established, and confounding by indication remained possible. The achieved sample of 100 patients was smaller than the calculated sample size of 211, which likely widened variance and reduced precision of estimates. A zero cell in the hypokalemia–no-arrhythmia category inflated the chi-square statistic and precluded stable effect-size estimation without continuity corrections, underscoring the need for exact or penalized methods in sparse tables. Important covariates—left ventricular ejection fraction, infarct size surrogates, door-to-balloon time, beta-agonist or diuretic exposure, serum magnesium, and use of beta-blockers or antiarrhythmics—were not recorded, limiting confounder control. Potential misclassification of “electrolyte imbalance” as a composite variable could have obscured specific contributions of potassium versus other ions. Finally, single-center, purposive sampling constrained generalizability.

Future investigations should adopt prospective cohort designs with serial electrolyte measurements, protocolized repletion to predefined targets, and time-to-event analyses to delineate early versus late arrhythmic windows. Multivariable logistic regression or competing-risk models should adjust for age, sex, infarct phenotype (STEMI/NSTEMI), ejection fraction, autonomic markers, and medication exposures, with interaction testing for hypokalemia by smoking status. Randomized or stepped-wedge implementation studies could evaluate standardized potassium-magnesium repletion bundles on arrhythmic endpoints and telemetry alarms. Inclusion of continuous ECG metrics such as QTc dispersion and premature ventricular complex burden may refine risk prediction and bridge mechanistic insights with clinical triage (20,21). In summary, ventricular arrhythmias were common after myocardial infarction in this cohort, and hypokalemia was strongly associated with their occurrence, consistent with established electrophysiologic principles and prior clinical data (22). While the unadjusted nature of the analysis and limited sample size cautioned against causal claims, the findings supported immediate, actionable practices: early electrolyte surveillance, prompt potassium optimization, and targeted monitoring of high-risk profiles to reduce preventable arrhythmic events in the acute phase of infarction (23).

CONCLUSION

The study concluded that hypokalemia had a significant association with the development of ventricular arrhythmias among patients presenting with acute myocardial infarction. The findings emphasized that electrolyte imbalance, particularly low serum potassium, acts as a critical precipitating factor for rhythm disturbances following myocardial injury. Identifying and correcting hypokalemia early in the course of treatment may therefore play a vital role in reducing the occurrence of life-threatening ventricular arrhythmias and improving survival outcomes in patients with acute coronary syndromes.

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AUTHOR CONTRIBUTION

Author	Contribution
Aqsa Azhar*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Zarina Naz	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
Muhammad Ali	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Muhammad Yasir	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Atif Mughal	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Arham Hayat	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published

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