RESEARCH PAPER

To Analyze Respiratory and Metabolic Imbalances; The Prospective Role of ABG analysis in therapeutics

Mr. Ravindra Dattatray Waingade[1], Mr. Bipin Kumar[1], Ms. Shweta Avinash Sandbhor[1], Ms. Midhuna Sudhir Nair[1], Dr. Abhay R. Shinde[1], Dr. Dipak Ravindra Pawar[1], Dr. Ganesh Vijaykumar Kumbhar[1]

[1]Doctor Of Pharmacy (Pharm.D) Dr. D Y Patil College of Pharmacy, Akurdi, Pune-411044.
Affiliated to Savitribai Phule Pune University, Pune

Mail IDs: [1]raviwaingade71157@gmail.com, [2]Krbipin.167@gmail.com, [3]Shweta.sandbhor02@gmail.com, [4]midhuna644@gmail.com, [5]abhayshinde434@gmail.com, [6]drpawar779825@gmail.com, [7]ganeshkumbhar477@gmail.com

ABSTRACT

Background and Rationale: Acid-base disorders (ABD) are commonly encountered in intensive care units (ICUs) and significantly influence clinical outcomes. Arterial Blood Gas (ABG) analysis remains a critical tool for diagnosing and managing these imbalances by providing real-time data on pH, $PaCO_2$, HCO_3^-, and other gasometric parameters crucial for evaluating respiratory and metabolic conditions.

Objectives:

1) To check the influence of ABG analysis on therapeutic choices: Assess how ABG results influence clinical decisions regarding the treatment of acid-base disorders.
2) Evaluate the prevalence and severity of acid-base disorders in ICU patients.
3) To Investigate the Correlation Between ABG Parameters and Other Clinical Markers of Acid-Base Balance, Such as Lactate Levels and Urine Output.
4) To evaluate the quality of life of patients suffering from acid-base disorders: Examine how acid-base disorders affect the overall well-being and daily functioning of patients.

Methodology: A prospective randomized observational study was conducted over six months (Nov 2024–Apr 2025) in Lokmanya Hospital and Ruby Hall Clinic, Pune. Data from 138 patients aged ≥18 with suspected or confirmed ABD were collected using a structured patient information form. ABG values, clinical presentations, treatments, and outcomes were recorded and statistically analyzed. Patients were assessed for severity of illness using the APACHE II (Acute Physiology and Chronic Health Evaluation) score at admission. Health-related quality of life (QoL) was evaluated using the SF-36 survey, which covers eight domains and generates Physical Component Summary (PCS) and Mental Component Summary (MCS) scores. Descriptive statistics were applied to summarize baseline characteristics. Independent samples t-test and ANOVA were used to compare continuous variables between groups. Correlation analysis (Pearson's r) assessed relationships between ABG parameters and clinical markers. Logistic regression was used to identify predictors of ICU outcomes and duration of stay.

Results: Among the 138 ICU patients studied, the most prevalent acid-base disorder was metabolic acidosis (36.23%), followed by mixed acid-base disturbances and Respiratory Acidosis in 18.84 % of cases respectively. These mixed patterns were associated with higher disease severity, as evidenced by elevated APACHE II scores, indicating a more critical clinical status and higher risk for adverse outcomes. Strong correlations were found between ABG parameters, such as pH with lactate and bicarbonate. Patients managed with ABG guidance showed better outcomes and improved quality of life, as reflected in SF-36 scores. These findings highlight the clinical value of ABG in critical care.

Discussion: The findings reaffirm the diagnostic and prognostic significance of ABG analysis in ICU care. Early identification and interpretation of ABD via ABG allowed tailored treatment strategies, positively influencing patient outcomes.

Conclusion: ABG analysis is an indispensable tool in critical care, offering timely insights into a patient's acid-base status and guiding effective therapeutic interventions. Its integration into routine ICU protocols enhances

clinical decision-making and patient management.

Keywords: Acid-Base Disorder, ABG Analysis, ICU, Metabolic Acidosis, Respiratory Alkalosis, Metabolic Alkalosis, Respiratory Acidosis.

How to cite this article: Waingade RD, Kumar B, Sandbhor SA, Nair MS, Shinde AR, Pawar DR, Kumbhar GV. To Analyze Respiratory and Metabolic Imbalances; The Prospective Role of ABG analysis in therapeutics. Int J Drug Deliv Technol. 2026;16(57s): 1349-1370. DOI: 10.25258/ijddt.16.57s.135

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Acidosis: A pH-lowering process. This may be caused by reduced sSr. bicarbonate levels and/or a rise in the PaCO2.
Alkalosis: A pH-raising process. This might occur due to reduced Sr. bicarbonate levels and/or elevated PaCO2. [1]

Disorders associated with acid-base equilibrium involve the coordination of multiple organ systems, including the liver, kidneys, lungs, and brain. [2] Acid-base diseases are categorised into two primary types: metabolic and respiratory. Primary changes in blood bicarbonate concentration indicate MA and alkalosis, whereas respiratory diseases primarily alter PaCO2. There are still many clinical scenarios where multiple single or simple diseases coexist and result in mixed acid-base disorders. [3]

A blood gas analysis frequently utilises diagnostic techniques for assessing the partial pressure of gases and the acid-base equilibrium in blood. A crucial diagnostic method for assessing a patient's oxygen levels, ventilation, and Acid-base level is the analysis of ABG.[4] It helps establish a diagnosis, determines a treatment plan, improves ventilator management, improves acid-base control for improved drug performance, and focuses on how the patient's acid/base balance can impact electrolyte levels.[5]

Sampling site for ABG analysis: Identifying a discernible artery is the first step in performing a percutaneous needle puncture. Typical places for arterial access consist of the radial, femoral, brachial, dorsalis pedis, or axillary arteries. Nonetheless, the radial artery is utilised more frequently than the other locations due to its accessibility and patient comfort. [6]

Respiratory Acidosis (Primary Hyper-Capnia): RA (pH<7.35) is caused by a rise in CO2 Concentration in body fluids. It's stated that an elevation in the arterial carbon dioxide partial pressure (PaCO2 > 45 mm Hg) results from a decrease in alveolar ventilation (primary ↑CO_2). [7]

Respiratory Alkalosis (Primary ↓CO_2): Alkalemia (a pH level>7.45) resulting from a drop in arterial CO2 concentration (PaCO2 <35 mm Hg) is known as RAlk. Primary ↓CO_2 results from the alveolar ↑Vent about the production of carbon dioxide. [8]

Metabollic Acidosis: A first ↓ Sr. HCO3- concentration, a secondary ↓PaCO2 of approximately 1 mmHg for each 1 mmol/l drop in Sr. HCO3- concentration levels, and a decrease in blood pH are the hallmarks of MA. [9]

Metabolic Alkalosis: The onset of MAlk is triggered by a rise in Sr. HCO3-. The reduction of breathing that follows the rise in Sr. (HCO3-), which raises PaCO2, occurs next.

Mixed acid-base disorders

Triple acid-base disturbances consist of two metabolic disturbances, a metabolic and respiratory disturbance, or two metabolic and one respiratory disturbance, which are frequently seen, especially in individuals who are very sick. Aside from modest persistent ↓CO_2, the pH level of the blood is abnormal in most disorders. A mixed acid-base problem is thus suggested by any divergence from normal Sr. (HCO3-) or PaCO2 levels linked to a normal plasma pH. [10]

Aim: To Analyse Respiratory and Metabolic Imbalances; The Prospective Role of ABG analysis in therapeutics.

Objectives:

To check the influence of ABG analysis on therapeutic choices: Assess how ABG results influence clinical decisions regarding the treatment of acid-base disorders.

Evaluate the prevalence and Severity of acid-base disorders in ICU patients.

To Investigate the Correlation Between ABG Parameters and Other Clinical Markers of Acid-Base Balance, Such as Lactate Levels and Urine Output.

To evaluate the quality of life of patients suffering from acid-base disorders: Examine how acid-base disorders affect the overall well-being and daily functioning of patients.

METHODOLOGY

The current study was conducted in the Intensive Care Unit at **the Lokmanya Hospital (Nigdi and Chinchwad) and Ruby Hall Clinic, Hinjewadi, Pune.** The study was executed for 6 months, and a total of 138 patients were

closely observed for the systematic data collection. Prior permission from the hospital administration and consent from the patient's legally acceptable representative were obtained for a case study and case report.

Plan of work

The study includes following steps: -

1. Identification of question.
2. Literature survey.
3. Preparation of data collection, interview forms with consent.
4. Collection of data from ICU patients in selected population by case papers, treatment charts and nursing chart
5. Interpretation and preparation of database from data collection form
6. Analysis of data for recovery and outcomes.
7. Statistical evaluation for probable outcomes.

Study Design-Prospective observational study

Study Setting-The study was conducted in the Intensive Care Unit at the Lokmanya Hospital (Nigdi and Chinchwad) and Ruby Hall Clinic, Hinjewadi, Pune.

Study Duration-The study is conducted for a period of 6 months from November 2024 to April 2025

Sample Size-Total 138 patients in the age group of 18 to 95 years were enrolled in this study.

Study Criteria:

Inclusion Criteria:-

1. The study included adults (≥18 years) who had acid-base imbalances or abnormal ABG findings.
2. Patients with acute or chronic conditions associated with acid-base imbalance were also enrolled.
3. In addition, those who required clinical management based on ABG analysis were considered eligible.
4. Patients who were undergoing or had received mechanical ventilation were also included in the study

Exclusion Criteria:-

1. The study excluded patients who were less than 18 years of age.
2. Pregnant patients were not enrolled.
3. Non-ICU patients with stable conditions were excluded from participation.

Statistical Analysis:-

Software: Excel / SPSS (better mention SPSS for journals)

Tests:

- Descriptive statistics
- Pearson correlation
- Linear regression
- t-test

Significance level: $p < 0.05$

Study Method:-

The Data Collection form includes various clinical and non-clinical parameters. It included closed questions about the signs and symptoms of the patients, Past Medical History, detailed information about the ABG parameters, Treatment and Other Clinical Markers of Acid-Base Balance, Such as Lactate Levels and Urine Output. The data collection form also incorporated standardised assessment tools, including the SF-36 scale and the APACHE scoring system. The SF-36 scale was used to evaluate patients' overall health-related quality of life across multiple domains. The APACHE score was applied to assess the severity of illness and predict clinical outcomes in critically ill patients.

STATISTICAL ANALYSIS AND RESULTS:

Gender Distribution of Study Population:

The pie chart presents the gender-wise distribution of the population enrolled in the study **titled "Analyzing Respiratory and Metabolic Imbalances; The Prospective Role of ABG Analysis in Therapeutics."** Among the 138 patients assessed in the ICU setting:

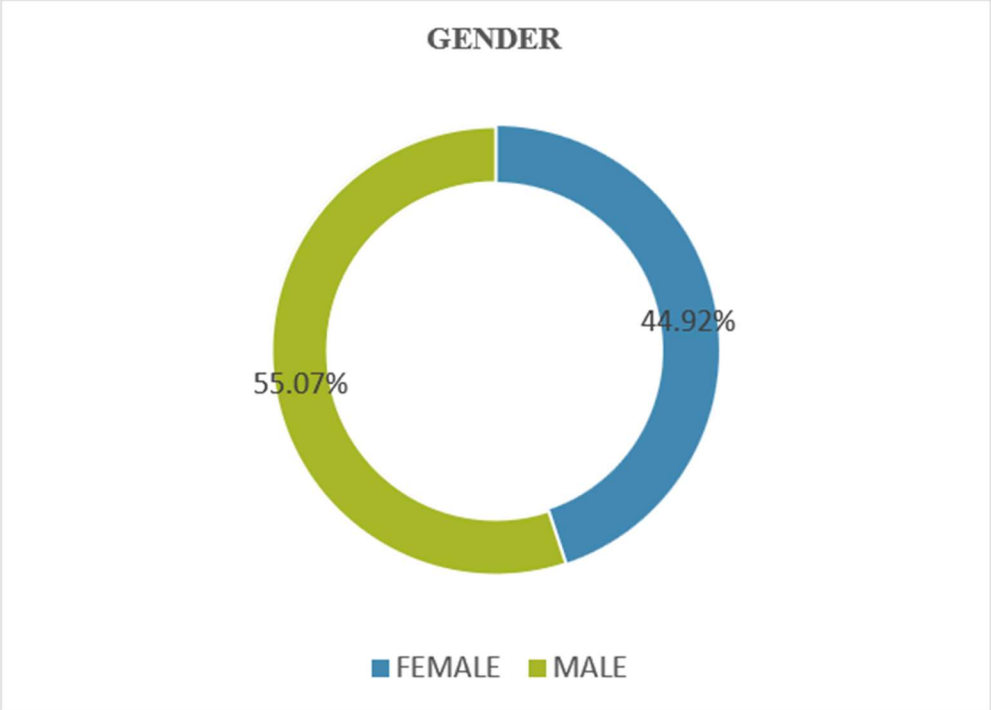

Fig.1: Gender distribution of study population

N = 138

This equates to:

76 males (around 55.07% of 138)

62 women (around 44.92% of 138)

Age Distribution of the Study

138 participants were recruited for the study. The participants were divided into four age groups to determine the distribution of acid-base disturbances by age group.

Age Group	Number of Participants	Percentage of Total Sample (%)
Age Group: 21–40 years	18	13
Age Group: 41–60 years	27	19.6
Age Group: 61–80 years	81	57.8
Age Group:81–100 years	12	8.7

Table 1: Age distribution

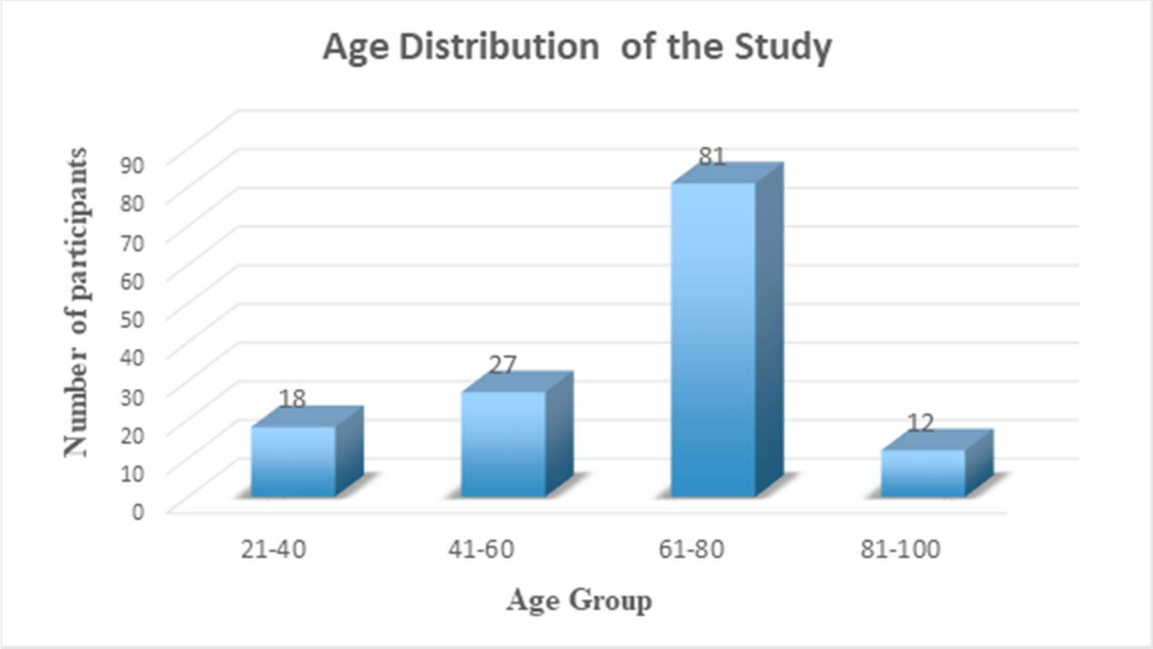

Fig.2: Age distribution of study population

Distribution of Acid-Base Disorders:

ABG Diagnosis	NO. OF CASES	%
RESPIRAORY ACIDOSIS	26	18.84%
RESPIRATORY ALKALOSIS	18	13.04%
METABOLIC ACIDOSIS	50	36.23%
METABOLIC ALKALOSIS	18	13.04%
OTHERS	26	18.84%
TOTAL	138	100%

Table No.2: Distribution of Acid-Base Disorders

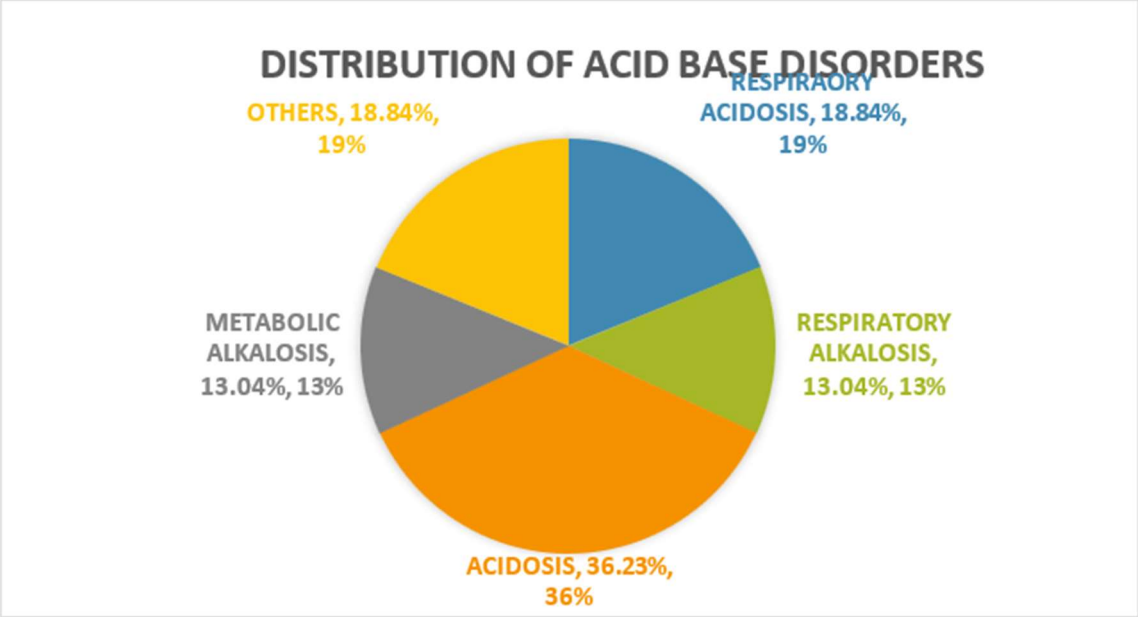

Fig no.3: Distribution Of Acid Base Disorder Influence Of ABG Analysis On Therapeutic Choices

This study provides a concise summary of the therapeutic approaches to respiratory acidosis based on the pattern of interventions observed. The study revealed a strong connection between ABG reports and therapy responses. Out of 138 patients studied, ABG results directly influenced the therapeutic decisions in all cases (100%). Acid-base derangement distribution and related therapeutic interventions are shown in:

Respiratory Acidosis (18.84%): O2 therapy was initiated in 92 % of cases, and Bronchodilators were administered to 77 % and 0.9 % NaCl were administered to 100% of cases, and the remaining patients were managed conservatively with NaHCO3, Diuretic, Ventillator, and K+ Replacement

Therapy	% of Therapy Administered
O2 Therapy	92%
NaHCO3	23%
Diuretic	12%
Bronchodilator	77%
Ventillator	38%
K+ Replacement	19%
0.9 % NaCl	100%

Table No. 3: Therapeutic Choices for Respiratory Acidosis

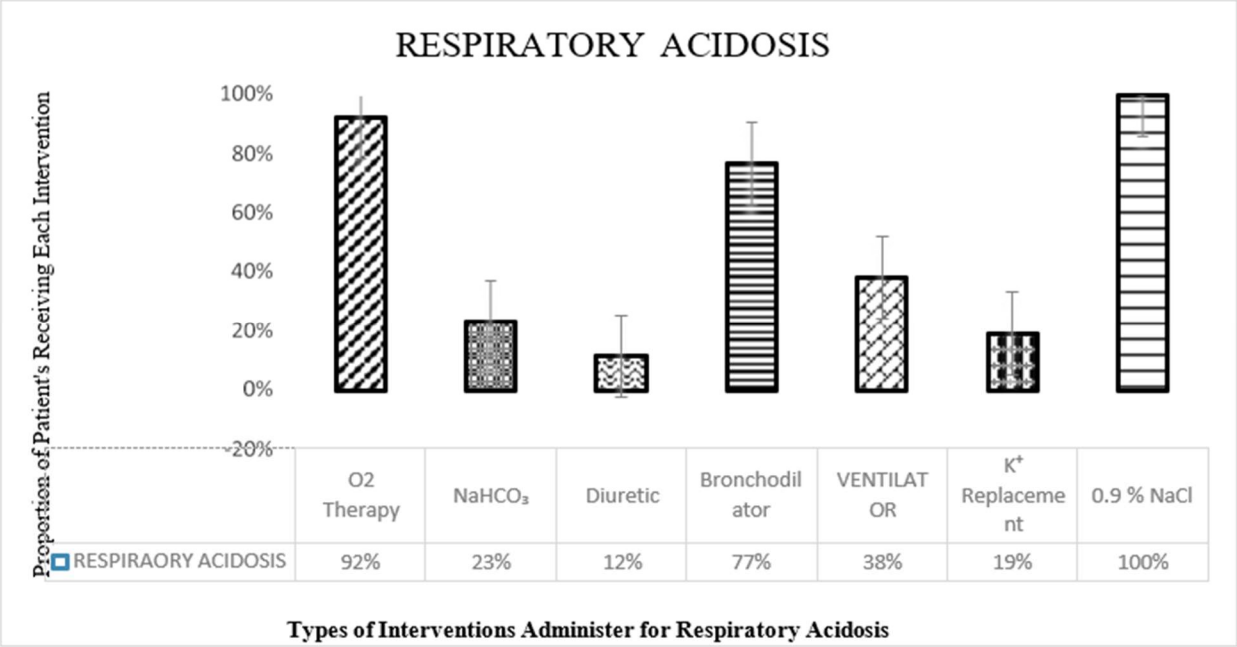

Fig. no.4: Therapeutic intervention for respiratory acidosis

The bar chart indicates the incidence of all of the interventions administered to the patients with respiratory acidosis.

Respiratory Alkalosis (13.04%):
O2 therapy was initiated in 83% of cases, and Bronchodilators were administered to 66.66 % and 0.9 % NaCl was administered to 100% of cases, and the remaining patients were managed conservatively with NaHCO3, Diuretic, Ventilator, and K+ Replacement

Therapy	% of Therapy Administered
O2 Therapy	83%
NaHCO3	11.11%
Diuretic	55.55%
Bronchodilator	66.66%
Ventilator	38.88%
K+ Replacement	16.66%
0.9 % NaCl	100%

Table No.4: Therapeutic Choices for Respiratory Alkalosis

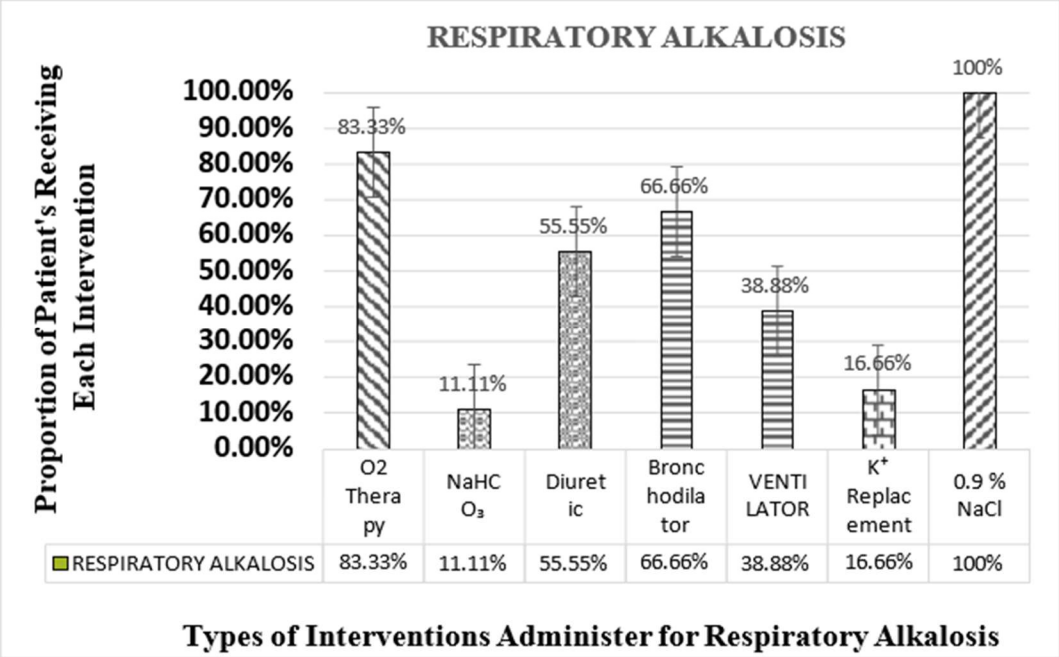

Fig. no.5: Therapeutic interventions administered for respiratory alkalosis

Metabolic Acidosis (36.23%):
Most patients with metabolic acidosis were treated with bicarbonate therapy (100%) and 0.9% NaCl (100%), while the remaining were treated with diuretics, ventilators, bronchodilators, and K+ Replacement therapy.

Therapy	% of therapy administered
O2 Therapy	76 %
NaHCO3	100%
Diuretic	40%
Bronchodilator	24%
Ventillator	20%
K+ Replacement	60%
0.9 % NaCl	100%

Table No.5: Therapeutic Choices for Metabolic Acidosis

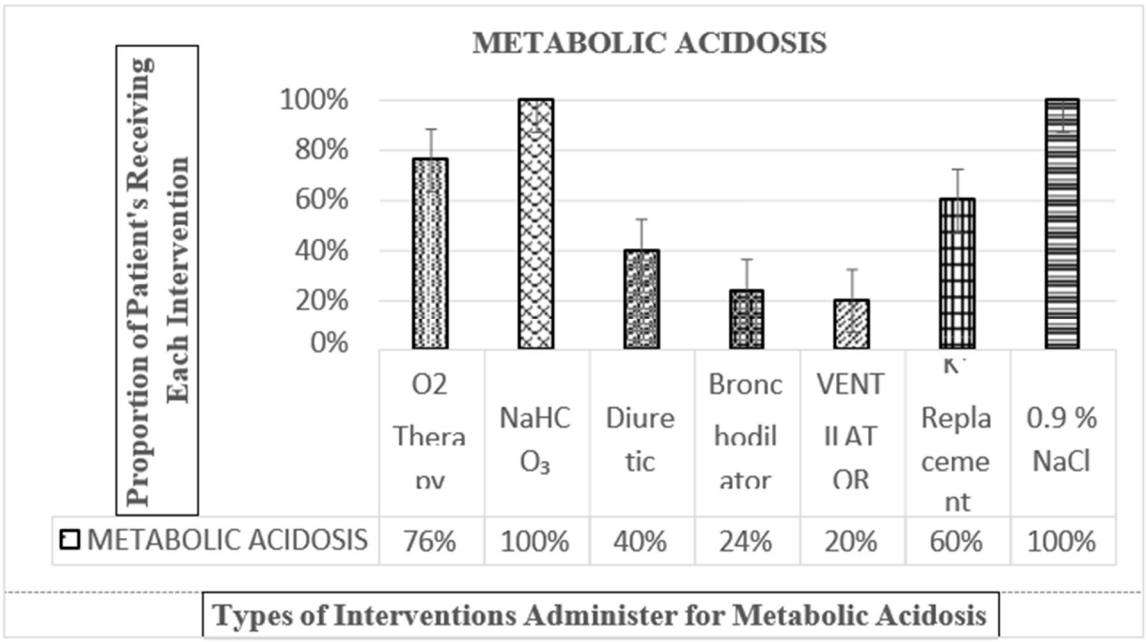

Fig no. 6: Therapeutic interventions administered for metabolic acidosis

Metabolic Alkalosis (13.04%):
The majority of patients with metabolic alkalosis were treated with K+ Replacement therapy (83.33%) and 0.9% NaCl (100%), while the remaining were treated with diuretic, ventilator, bronchodilator, Bicarbonate and O2 therapy.

Therapy	% of therapy administered
O2 Therapy	44.44%
NaHCO3	27.77%
Diuretic	78%
Bronchodilator	22.22%
Ventillator	27.77%
K+ Replacement	83.33%
0.9 % NaCl	100%

Table No. 6: Therapeutic Choices for Metabolic Alkalosis

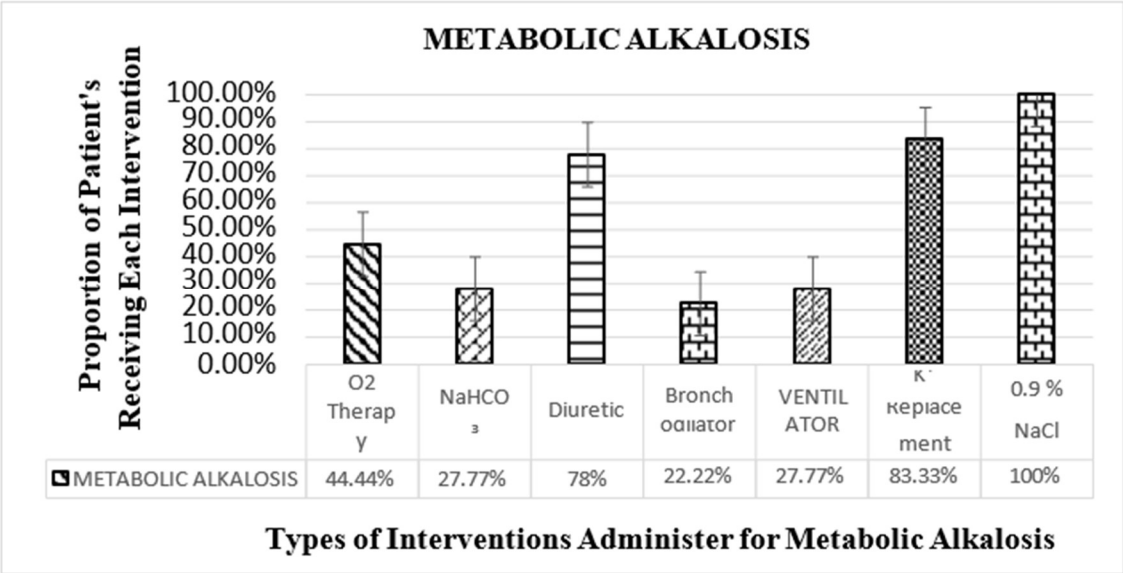

Fig. No.7: Therapeutic interventions administered for metabolic alkalosis

Others (18.84%):
Others include the four main types of acid-base disorders, i.e., RA, RAlk, MA, MAlk, along with partially compensated, fully compensated, and uncompensated and mixed acid-base disorders.

The majority of patients with other categories were treated with O2 therapy (76.92%) and 0.9% NaCl (100%), while the remaining were treated with diuretic, ventilator, bronchodilator, Bicarbonate, and K+ repletion therapy.

Therapy	% of therapy administered
O2 Therapy	76.92%
NaHCO3	46.15%
Diuretic	69.23%
Bronchodilator	30.76%
Ventillator	23.07%
K+ Replacement	46.15%
0.9 % NaCl	100%

Table No.7: Therapeutic Choices for Others

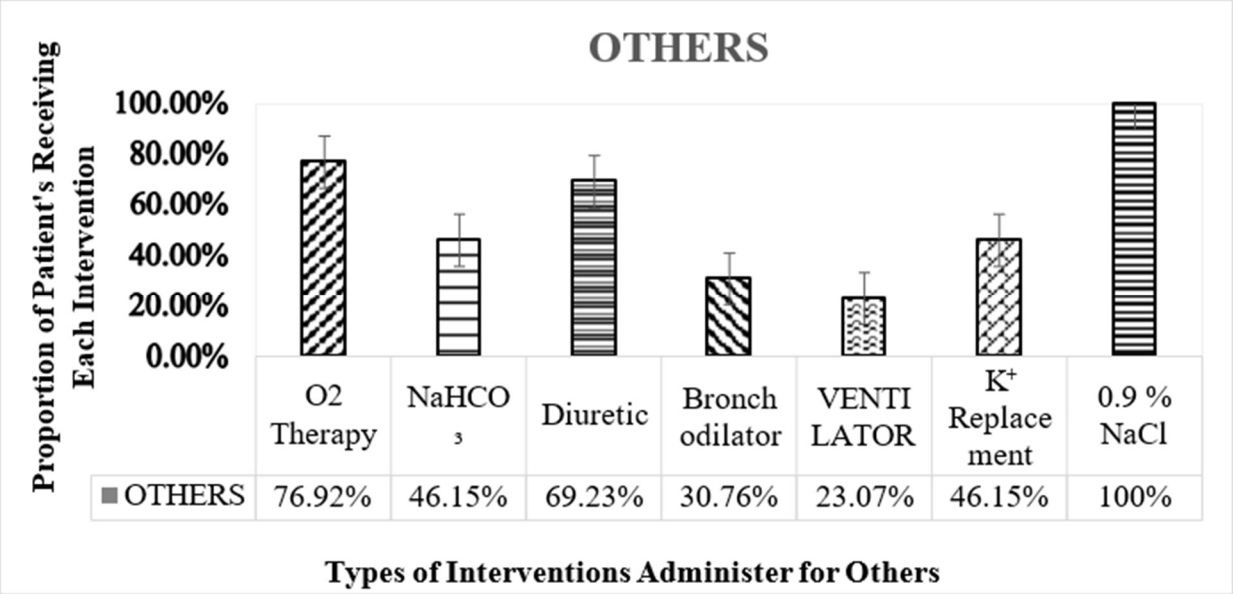

Fig. No.8: Therapeutic interventions administered for others

Prevalance Of Acid base Disorder

Age Distribution of the Study

138 participants were recruited for the study and divided into two age groups: 21-60 years and 61-100 years, to analyse the distribution of acid-base disturbances.Age Group: 21–60 years have total Number of Participants is 45 with Percentage of Total Sample is 32.6%.This group comprises the study's younger adult and middle-aged participants. And the Age Group 61–100 years have total Number of Participants is 93 with Percentage of Total Sample 67.4%.This group represents the older adult and elderly participants in the study. The higher percentage in this group indicates a larger representation of older individuals in the study population.

The overall prevalence of acid-base disturbances in the study population was determined. The prevalence of specific acid-base disorders is shown below:

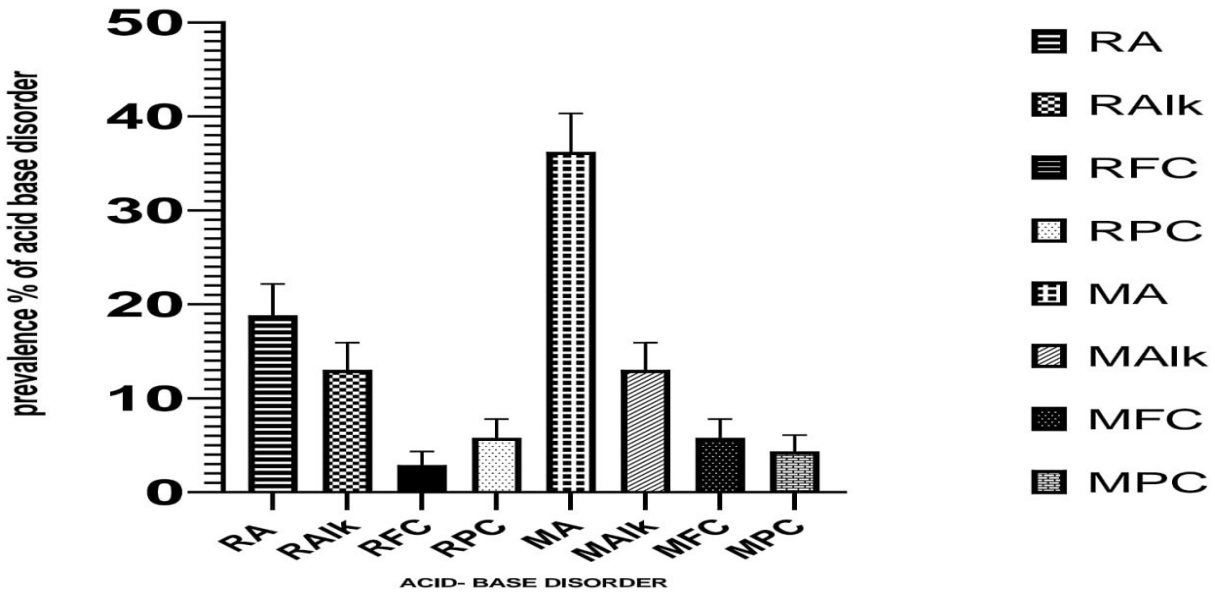

Fig 9: Prevalence of Acid-Base Disorders

The table below shows the prevalence (%) of each acid-base disorder with standard error (SE) and 95% confidence intervals (CI):

Sr.no.	PARAMETER	PREVALENCE	STANDARD ERROR	LOWER 95% CI	UPPER 95% CI
1.	Respiratory Acidosis	18.84%	3.33%	12.32%	25.36%
2.	Respiratory Alkalosis	13.04%	2.87%	7.42%	18.66%
3.	Respiratory Fully Compensated	2.90%	1.43%	0.10%	5.70%
4.	Respiratory Partially Compensated	5.80%	1.99%	1.90%	9.70%
5.	Metabolic acidosis	36.23%	4.09%	28.21%	44.25%
6.	Metabolic Alkalosis	13.04%	2.87%	7.42%	18.66%
7.	Metabolic Partially Compensated	5.80%	1.99%	1.90%	9.70%
8.	Metabolic fully Compensated	4.35%	1.74%	0.95%	7.75%

Table No.8: Prevalence of Acid-Base Disorders

As shown in table 8 Metabolic Acidosis was the most prevalent disorder (36.23%), while Respiratory Fully Compensated disorder showed the lowest prevalence (2.90%).

Relative Risk of Acid-Base Disturbances:
Age Group Comparison (21-60 vs. 61-100 years)

The relative risk (RR) of developing specific acid-base disturbances was compared between the two age groups: 21-60 years and 61-100 years. The 21-60 age group served as the reference group (RR = 1.0).

The relative risk for each acid-base disturbance in the 61-100 age group compared to the 21- 60 age group is shown below:

1	Respiratory Acidosis	1.77%
2	Respiratory Alkalosis	3.00%
3	Respiratory Fully Compensated	0.04%
4	Respiratory Partially Compensated	1.91%
5	Metabolic Acidosis	1.42%
6	Metabolic Alkalosis	1.70%
7	Metabolic Fully Compensated	0.06%
8	Metabolic Partially Compensated	2%

Table No.9: Relative Risk of ABD in the age group

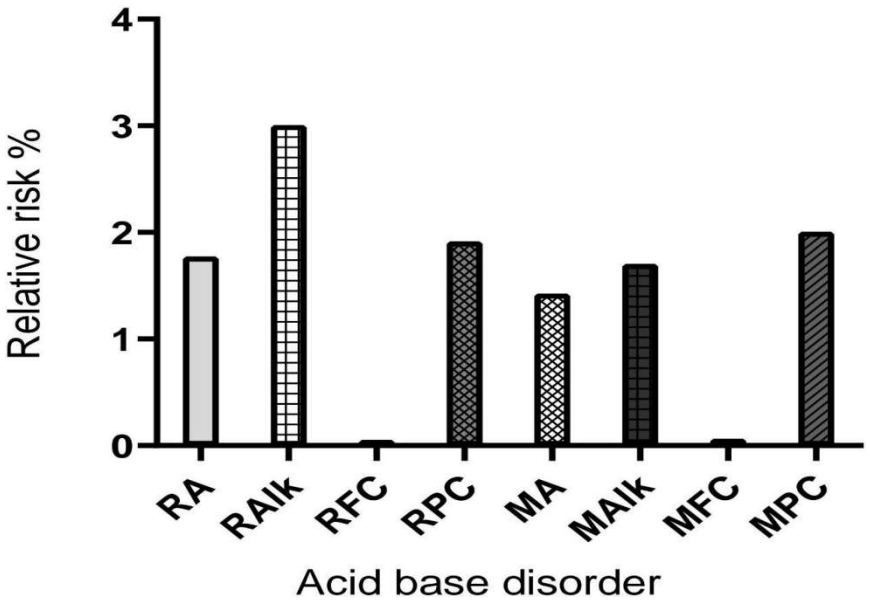

Fig. no.10: Relative Risk of Acid-Base Disturbances (61-100 years vs. 21-60 years)

Overall, the relative risk analysis indicates that older patients (61-100 years) have a generally higher risk of developing most acid-base disturbances compared to younger patients (21-60 years).

Specifically,The relative risk of Respiratory Acidosis was 1.77, indicating a 77% higher risk in the older age group and The relative risk of Respiratory Alkalosis was 3.00, showing a threefold increased risk in the older age group and The relative risk of Respiratory Fully Compensated was 0.04, suggesting a substantially lower risk of this specific disturbance in the older group and The relative risk of Respiratory Partially Compensated was 1.91, indicating a 91% higher risk in the older group and The relative risk of Metabolic Acidosis was 1.42, showing a 42% higher risk in the older group and The relative risk of Metabolic Alkalosis was 1.70, indicating a 70% higher risk in the older group and The relative risk of Metabolically Compensated was 0.06, suggesting a markedly lower risk of this disturbance in the older group and The relative risk of Metabolically Partially Compensated was 2.00, indicating a two fold increased risk in the older group.

DISEASES SEVERITY BASED ON AGE GROUPS IN ACID BASE DISORDER

AGE GROUP	NO PARTICIPANT	MEAN (APACHE II)	STD D	MEDIAN
21-40	18	20.55555556	8.060230782	19
41-60	27	14.34615385	7.155095011	11.5
61-80	81	18.425	7.163152902	17
81-100	12	23.25	4.634357855	23.5

Table No.10: Disease severity based on age group in ABD

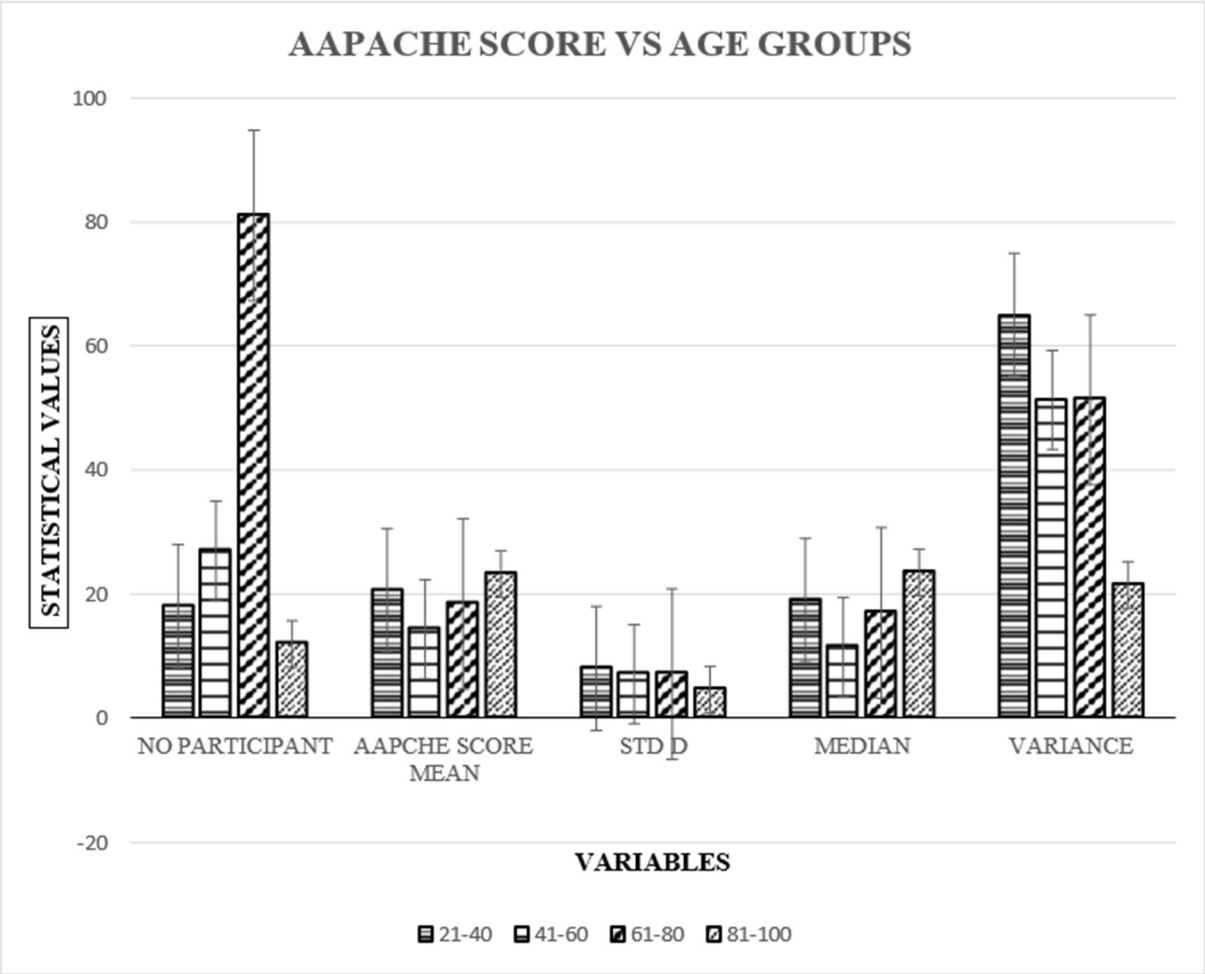

Fig. no.11: Disease severity in various age groups

The highest severity is in the 81–100 group (Mean APACHE IS 23.25), indicating more critical illness in the elderly. The lowest APACHE score is in the 41–60 group, suggesting milder cases or better outcomes of this group. Despite being younger, the 21–40 group has a higher APACHE score than the 41–60 and 61–80 groups, indicating individual variability or possibly more acute cases.

The youngest group (21–40) has the highest variability (SD = 8.06), indicating a wide spread in patient severity. The oldest group (81–100) has the lowest variability (SD = 4.63), suggesting more consistent severity in this age group.

Sr.no	Abg parameter	Clinical marker	Correlation coefficient ®	R^2	CI(95%)	P-value	Interpretation
1.	PH	Lactate	-0.8162	0.6661	-0.8653 to -0.7515	<0.0001	Strong negative correlation
2.	PH	Bicarbonate	0.3083	0.09502	0.1488 to 0.4521	0.0002	Weak positive correlation

Table No.11: Correlation Analysis PH VS LACTATE

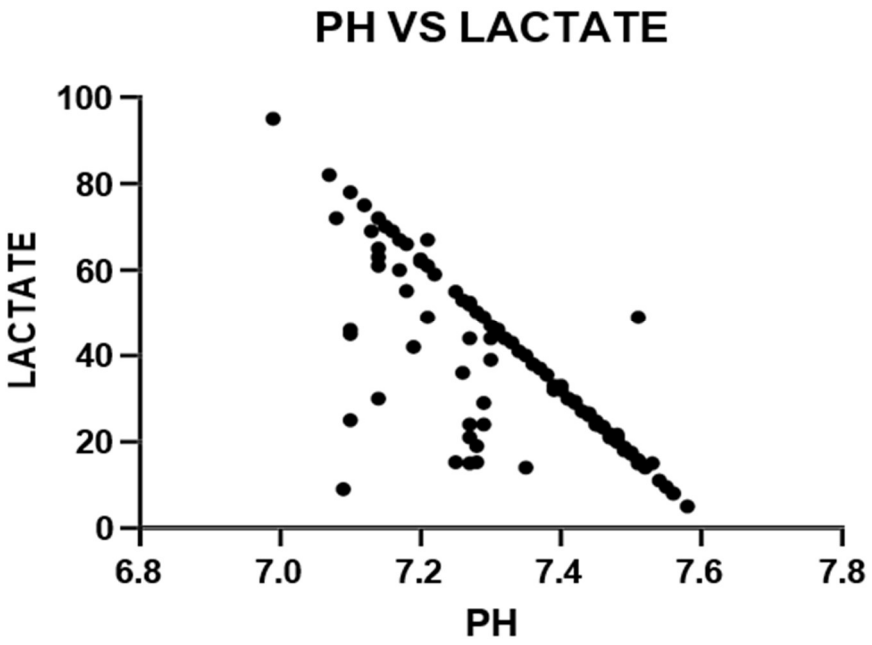

Fig.no.12: Correlation Analysis of PH Vs Lactate

The results disclosed a strong negative correlation between pH and lactate, with a Pearson correlation measure (r) of-0.8162. This suggests that as lactate situations increase, pH values tend to drop significantly, indicating worsening acidosis with elevated lactate.

The 95% confidence interval for the correlation coefficient ranged from -0.8653 to -0.7515, reinforcing the reliability

of the observed negative association. The measure of determination (R^2) was 0.6661, which implies that roughly 66.6% of the variability in pH values can be explained by changes in lactate situations.

The correlation was set up to be statistically significant, with a p-value < 0.0001(two-tailed), attesting that the observed relationship isn't due to arbitrary chance.

The scatter plot easily illustrates this inverse relationship, with data points showing a downcast trend from high lactate concentrations at lower pH values toward lower lactate concentrations at higher pH values. The concentration of data points near the trend line further reinforces the robustness of this association.

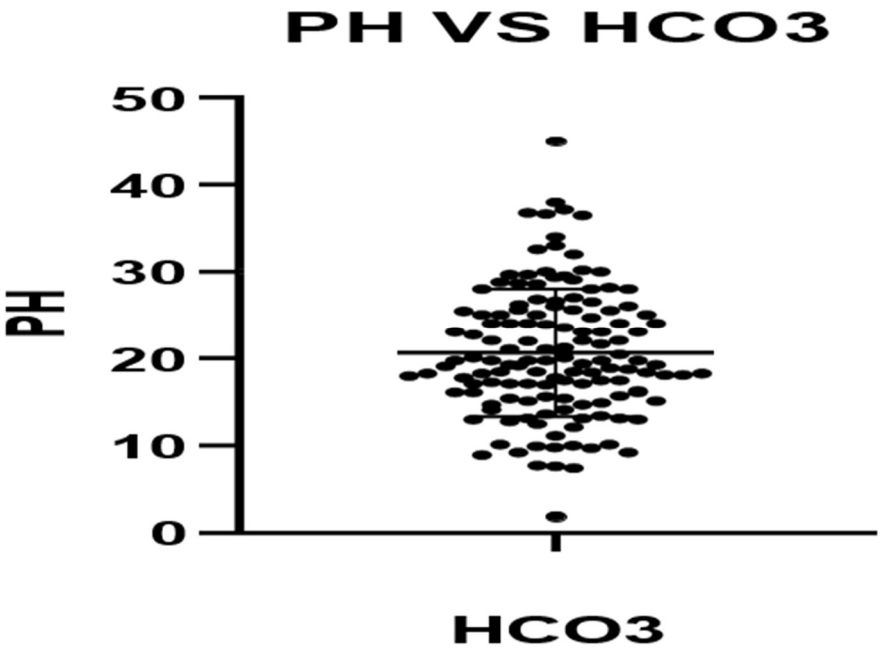

Fig. No.13: Correlation analysis of pH vs HCO3

The Pearson correlation coefficient (r) was calculated as 0.3083, indicating a weak positive correlation between pH and bicarbonate levels. This suggests that as bicarbonate levels increase, there is a slight tendency for the pH to increase as well—consistent with the physiological buffering role of bicarbonate in maintaining acid-base balance.

The 95% confidence interval for this correlation ranged from 0.1488 to 0.4521, confirming that the association, although modest, is statistically reliable. The coefficient of determination (R^2) was 0.09502, indicating that only about 9.5% of the variation in pH values can be explained by changes in bicarbonate levels.

The relationship was found to be statistically significant, with a two-tailed p-value of 0.0002, which is well below the threshold of significance (alpha = 0.05).

The accompanying scatter plot visually represents the data, showing a dispersed but slightly upward trend between bicarbonate values and pH levels. Despite the weak strength of the relationship, the statistical significance supports the biological plausibility of bicarbonate contributing to pH regulation.

This result reflects the expected physiological interaction where bicarbonate acts as a primary buffer in blood, and its levels generally correlate with the degree of metabolic compensation in acid-base disorders.

All statistical analyses were conducted using Microsoft Excel with the Data Analysis Tool Pack and were supplemented where necessary with manual calculations using standard epidemiological formulas.

Descriptive statistics were used to summarise the demographic data and baseline characteristics of the study population. Measures included frequency, percentage, mean, and standard deviation for variables such as age, quality of life (QOL), and types of acid-base disorders.

GENDER	NO OF PARTICIPANTS	MEAN	MEDIAN	STD D(σ)	VARIANCE	RANGE
M	76	69.23	70	±9.03	81.61	0-92
F	62	66.56	65	±9.05	81.98	0-95
M+F	138	68.28	68	±9.06	82.08	0-95

Table No.12: Descriptive Statistics

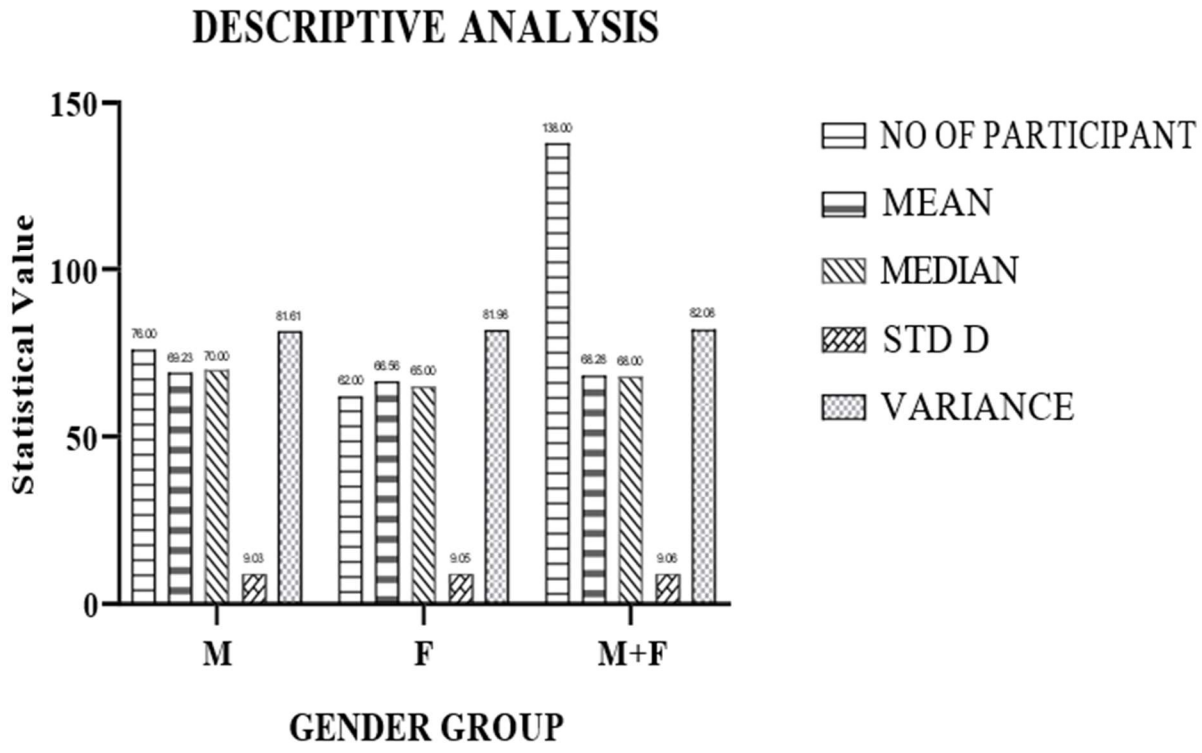

Fig. no.14: Quality of Life in Gender Groups

Demographic Characteristics Of The Study Population
Age Distribution-

The participants in this study were categorised into four continuous age groups to evaluate age-related trends and associations with acid-base disorders and quality of life (QOL). A total of **138 patients** were enrolled in the study. Among them:**18 participants (13.0%)** were in the **21–40 years** age group,**27 participants (19.6%)** belonged to the **41–60 years** age group,**81 participants (58.7%)** were within the **61–80 years** age group,**12 participants (8.7%)** were aged **81–100 years.**

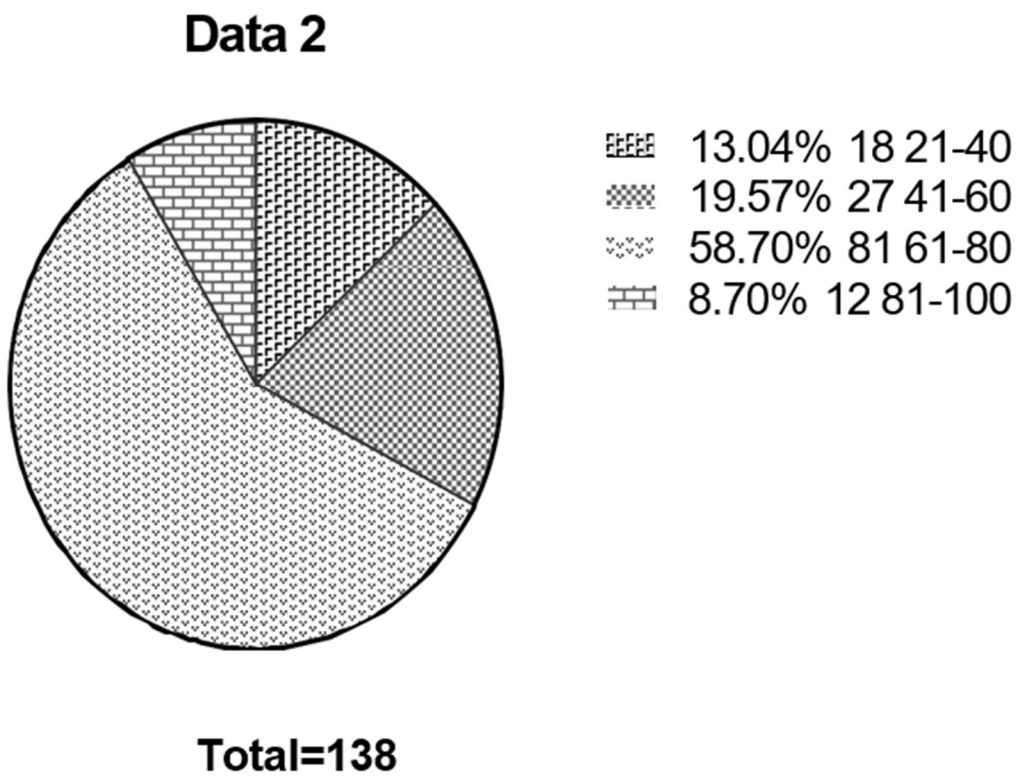

Fig. No.15: Quality of life in age groups

LINEAR REGRESSION ANALYSIS OF AGE VS. QOL:

A simple linear regression model was used to examine the relationship between **age** (independent variable) and **QOL score** (dependent variable). The regression output included:Coefficients (slope and intercept),R-squared value (indicating model fit),p-value (for significance) and 95% confidence intervals.

Coefficients	**Standard Error**	**t Stat**	**P-value**	**Lower 95%**	**Upper 95%**
Intercept	95.39	2.05	46.50	91.33	99.44
Age	-0.43	0.03	-13.67	-0.49	-0.37

Regression Statistics	
Multiple R	0.7619
R Square	0.5805

Adjusted R Square	0.5774
Standard Error	5.9526

ANOVA	df	SS	MS	F	Significance F
Regression	1	6618.53	6618.53	186.79	3.08×10^{-27}
Residual	137	4783.46	35.43		
Total	138	11401.99			

Table No.13: Regression analysis of AGE vs QOL

- **R^2 = 0.5805** indicates that 58.05% of the variability in QOL is explained by age.
- The **negative coefficient for age (-0.43)** suggests that as age increases by one year, QOL decreases on average by 0.43 units.
- **P-value < 0.0001** shows a highly significant association between age and QOL.
- The **95% confidence interval** does not cross zero, confirming statistical significance.
- There is a **strong, statistically significant negative correlation** between age and QOL.

LINEAR REGRESSION ANALYSIS OF MALES VS. FEMALES QOL

The unpaired t-test produced a **t value of 2.027** with **136 degrees of freedom**, and a **two-tailed P value of 0.0446**, indicating a **statistically significant difference** between males and females at the 0.05 significance level.

Table No.14: Linear Regression analysis of Male Vs Female QOL

B) REGRESSION **MALE QOL VS FEMALE QOL**	
Unpaired t-test	
P value	0.0446
Significantly different (P < 0.05)	Yes
One- or two-tailed P value?	Two-tailed
t, df	t=2.027, df=136
How big is the difference?	
Mean of MALE	69.68
Mean of FEMALE	66.56
Difference between means (F - M) ± SEM	-3.120 ± 1.539

95% confidence interval	-6.163 to -0.07665
R squared (eta squared)	0.02934
F test to compare variances	
F, DFn, Dfd	1.026, 61, 75
P value	0.9104
Significantly different (P < 0.05)	No
Data analyzed	
Sample size, MALE	76
Sample size, FEMALE	62

The unpaired t-test produced a **t value of 2.027** with **136 degrees of freedom**, and a **two-tailed P value of 0.0446**, indicating a **statistically significant difference** between males and females at the 0.05 significance level.

The mean value for males was 69.68, while the mean for females was 66.56, resulting in a mean difference of -3.120 ± 1.539. The 95% confidence interval for the difference ranged from -6.163 to -0.07665

An unpaired (independent samples) t-test was conducted to compare the mean values between male and female groups.

The test assumes equal variances, as supported by an F-test for equality of variances, which yielded an F ratio of 1.026 ($df_1 = 61$, $df_2 = 75$) with a P value of 0.9104, indicating no significant difference in variance between the two groups ($P > 0.05$).

DISCUSSION:

This six-month prospective observational study assessed 138 ICU patients with documented acid-base disturbances by ABG analysis. The patient cohort, most of whom were elderly (57.8% were aged 61–80 years), had a high prevalence of metabolic acidosis (36.23%), followed by respiratory and mixed disturbances. These results underscore the clinical importance of acid-base disturbances in critical care and their age-related predilection.

Therapeutic treatment in this study was primarily ABG result-driven. In respiratory acidosis (18.84%), oxygen in 92%, bronchodilators in 77%, and sodium bicarbonate in 23% patients. This indicates the application of ABG as an active guide in directing evidence-based treatment. These trends are in line with the prevailing patterns in treating hypercapnic respiratory failure as per Palmer et al.'s core curriculum for respiratory disorders.

From the pathophysiologic standpoint, the study proved statistically significant negative correlation between lactate and pH (r = -0.8162), indicating prognostic relevance of lactate in assessment of severity of acidosis. The evidence is underpinned by results presented by Rubavathy et al., which emphasized the most important determinant of poor prognosis for ICU patients with combined derangements in acid-base status.

Weak but significant positive correlation was also observed between pH and bicarbonate (r = 0.3083), and this supported the buffering action of bicarbonate in metabolic derangement. Regression analysis also showed that variables such as pH and lactate played an important role on outcomes such as ICU stay and post-discharge quality of life.

Interestingly enough, the therapeutic choices were regulated by ABG results in 100% of the cases, a figure which agrees with the findings of Reddy et al., who had reported that therapy with ABG at the appropriate time optimizes outcomes in respiratory ICU settings.

In comparison with Wargo and Center's original article in "ABCs of ABGs," the current study reflects the same emphasis on systematic evaluation of pH, $PaCO_2$, and HCO_3^- to classify acid- base disorders and guide therapy.

This study not only confirms previous evidence but also contributes to the evidence base by quantifying the relation between ABG values and intervention patterns and trajectories of recovery. These findings confirm the pivotal function of ABG in diagnosis, prognosis, and therapeutic management in intensive care practice.

The APACHE II scoring system used in our research further supported the correlation between acid-base severity and risk of death, as has been demonstrated with its established use as a prognostic tool in the ICU environment.

Patient well-being as measured by SF-36 Health Survey following treatment demonstrated significant improvement in both physical and mental components, attesting to the whole-person benefit of ABG-guided therapy.

CONCLUSION

This prospective observational study, conducted over six months at two tertiary care hospitals in Pune, India, evaluated the clinical impact of Arterial Blood Gas (ABG) analysis on patients with acid-base disorders admitted to the Intensive Care Unit (ICU). The findings underscore the pivotal role of ABG as both a diagnostic and therapeutic tool in critical care medicine.

The study enrolled 138 patients with abnormal ABG profiles and ICU-level clinical needs. A majority of patients presented with mixed acid-base disorders, reflecting the complex interplay of underlying pathologies such as sepsis, chronic obstructive pulmonary disease (COPD), diabetic ketoacidosis (DKA), renal dysfunction, and drug-induced respiratory alterations. Among isolated disorders, respiratory alkalosis and respiratory acidosis were most frequently observed, emphasizing the dominance of ventilatory abnormalities in critically ill individuals.

ABG analysis directly influenced therapeutic decisions, including ventilator settings, fluid management, and electrolyte corrections. For instance, patients with respiratory acidosis received targeted ventilatory support, while metabolic derangements guided interventions like sodium bicarbonate infusion or insulin therapy in DKA. The study highlighted how real-time

ABG interpretation helped clinicians implement personalized treatment protocols, improving patient outcomes and decision-making efficiency.

Furthermore, the study established significant correlations between ABG parameters (especially pH, bicarbonate (HCO_3^-), and lactate) and markers of disease severity, including the APACHE II score. These correlations validated ABG as an effective prognostic tool for early risk stratification in ICU settings. High lactate levels were strongly associated with metabolic acidosis and increased morbidity, reinforcing the need for early intervention in lactic acidosis cases.

The quality of life (QoL) assessment conducted post-treatment showed that ABG-directed interventions positively impacted patients' physical, respiratory, and overall functional status. Statistical models such as linear and logistic regression further affirmed that timely correction of ABG abnormalities significantly reduced ICU stay duration, complications, and facilitated better long-term recovery.

REFERENCE

1. Hamilton PK, Morgan NA, Connolly GM, Maxwell AP. Understanding acid-base disorders. The Ulster Medical Journal. 2017 Sep 12;86(3):161.
2. Seifter JL, Chang HY. Disorders of acid-base balance: new perspectives. Kidney Diseases. 2017 Dec 10;2(4):170-86.
3. NARINS RG, EMMETT M. Simple and mixed acid-base disorders: a practical approach. Medicine. 1980 May 1;59(3):161-82.
4. Karunarathna I, Bandara S, Perera N, Ekanayake U, Godage S, Hapuarachchi T, Gunasena P, Gunathilake S, De Alvis K, Nawarathna C, Gunawardana K. ABG Interpretation: Clinical Significance and Common Errors.
5. Cowley NJ, Owen A, Bion JF. Interpreting arterial blood gas results. Bmj. 2013 Jan 16;346.
6. Theodore AC, Manaker S, Hollingsworth H. Arterial blood gases. Assessment. 2013;2:2.
7. Hamm LL, DuBose TD. Disorders of Acid-Base Balance. In: Yu ASL, Chertow GM, Luyckx, VA, et al. (eds) Brenner & Rector's The Kidney. Philadelphia: Elsevier Inc. (2020): 496-536.
8. Adrogué HJ, Madias NE. Respiratory Acidosis, Respiratory Alkalosis, and Mixed Disorders. In: Feehally J, Floege J, Tonelli M, et al. (eds) Comprehensive Clinical Nephrology. Philadelphia: Elsevier Inc. (2018): 170-183.
9. Kraut JA, Madias NE. Metabolic acidosis: pathophysiology, diagnosis and management. Nature Reviews Nephrology. 2010 May;6(5):274-85.
10. Rodríguez-Villar S, Do Vale BM, Fletcher HM. The arterial blood gas algorithm: Proposal of a systematic approach to analysis of acid-base disorders. Revista Española de Anestesiología y Reanimación (English Edition). 2020 Jan 1;67(1):20-34.
11. Epstein SK, Singh N. Respiratory acidosis. Respir Care 46 (2001): 366-383.
12. Brijker F, Heijdra YF, Van den Elshout FJJ, Hans Th M Folgering. Discontinuation of furosemide decreases PaCO2 in patients with COPD. Chest 121 (2002): 377-382.

13. Roy G Brower, Michael A Matthay, Alan Morris, David Schoenfeld, B Taylor Thompson, Arthur Wheeler, et al. Ventilation with Lower Tidal Volumes as Compared with Traditional Tidal Volumes for Acute Lung Injury and the Acute Respiratory Distress Syndrome. N Engl J Med 342 (2000): 1301-1308.

14. Christophe Faisy, Ferhat Meziani, Benjamin Planquette, Marc Clavel, Arnaud Gacouin, Caroline Bornstain, et al. Effect of acetazolamide vs placebo on duration of invasive mechanical ventilation among patients with chronic obstructive pulmonary disease: A randomized clinical trial. JAMA - J Am Med Assoc 315 (2016): 480-488.

15. Banga A, Khilnani GC. Post-hypercapnic alkalosis is associated with ventilator dependence and increased ICU stay. COPD J Chronic Obstr Pulm Dis 6 (2009): 43744.

16. Tinawi M. Pathophysiology, Evaluation, and Management of Metabolic Alkalosis. Cureus 13 (2021): e12841.

17. Tinawi M. Disorders of Calcium Metabolism: Hypocalcemia and Hypercalcemia. Cureus 13 (2021): e12420.

18. Madias NE, Adrogué HJ. Respiratory alkalosis. In: DuBose TD, Hamm LL (eds) Acid-Base and Electrolyte Disorders: A Companion to Brenner and Rector's The Kidney. Philadelphia: Saunders (2002): 147-164.

19. Laffey JG, Kavanagh BP. Hypocapnia. N Engl J Med 347 (2002): 43-53.

20. Palmer BF, Clegg DJ. Respiratory acidosis and respiratory alkalosis: core curriculum 2023. American Journal of Kidney Diseases. 2023 Sep 1;82(3):347-59.

21. Kraut JA, Madias NE. Metabolic acidosis: pathophysiology, diagnosis and management. Nature Reviews Nephrology. 2010 May;6(5):274-85. Madias NE, Kraut JA. Metabolic acidosis: overview 2010 May;7(9):284-75.

22. Tinawi M. Pathophysiology, Evaluation and Management of Metabolic Acidosis. Archives of Clinical and Biomedical Research. 2021;5(1):85-109.

23. Matyukhin I, Patschan S, Ritter O, Patschan D. Etiology and management of acute metabolic acidosis: an update. Kidney and Blood Pressure Research. 2020 Jul 30;45(4):523-31.

24. Kraut JA, Madias NE. Treatment of acute metabolic acidosis: a pathophysiologic approach. Nature Reviews Nephrology. 2012 Oct;8(10):589-601.

25. Webster NR, Kulkarni V. Metabolic alkalosis in the critically III. Critical reviews in clinical laboratory sciences. 1999 Jan 1;36(5):497-510.

26. Khanna A, Kurtzman NA. Metabolic alkalosis. Journal of Nephrology. 2006 Mar 1;19:S86-96.

27. Emmett M. Metabolic alkalosis: a brief pathophysiologic review. Clinical Journal of the American Society of Nephrology. 2020 Dec 1;15(12):1848-56.

28. Pahari DK, Kazmi W, Raman G, Biswas S. Diagnosis and management of metabolic alkalosis. Journal of the Indian Medical Association. 2006 Nov 1;104(11):630-4.

29. Friedman BS, Lumb PD. Prevention and management of metabolic alkalosis. Journal of Intensive Care Medicine. 1990 Dec;5(1_suppl):S22-7.

30. Siwali M, Satyawali VN, Joshi SC, Kumar A. ACID BASE DISORDERS IN MEDICAL ICU AND THEIR RELATION WITH THE PATIENT OUTCOME IN A TERTIARY HEALTH CARE CENTRE. Int J Acad Med Pharm. 2023;5(3):1871-6.

31. Musleh BA, Abd-ElAziz MA, Mehany MM. Respiratory Acid-Base Disorders and Related Risk Factors in Critically Ill Patients. Assiut Scientific Nursing Journal. 2020 Feb 1;8(20.00):69-79.

32. Reddy BM, Viritha P, Reddy VV. Role of Arterial Blood Gases in Determining the Treatment and Prognosis of Patients Admitted in Respiratory Intensive Care Unit (RICU). Journal of Evolution of Medical and Dental Sciences. 2019 Nov 4;8(44):3314-8.

33. Wargo KA, Centor RM. ABCs of ABGs: a guide to interpreting acid-base disorders. Hospital pharmacy. 2008 Oct;43(10):808-18.

34. Rubavathy P, Chinnusamy M, Janakiraman S. STUDY AMONG PATIENTS ADMITTED IN ICU TO DETERMINE THE PROPORTION OF MIXED ACIDBASE DISORDER AND THE PROPORTION OF LACTIC ACIDOSIS. Int J Acad Med Pharm. 2023;5(6):976-83.

35. Gennari FJ. Metabolic alkalosis. Core concepts in the disorders of fluid, electrolytes, and acid-base balance. 2012 Jun 14:275-96.]

36. Ware JE, Snow KK, Kosinski M, Gandek B. SF-36 health survey. Manual and interpretation guide. 1993;2.