SERUM SODIUM AND POTASSIUM LEVELS IN NEWLY DIAGNOSED ESSENTIAL HYPERTENSION: A COMPARATIVE ANALYSIS WITH

HEALTHY CONTROLS

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ABSTRACT:

BACKGROUND: Primary hypertension, accounting for over 90% of hypertension cases, is a major risk factor for coronary, cerebral, and peripheral vascular diseases. While its exact etiology remains unclear, serum sodium and potassium levels significantly influence blood pressure

regulation.

AIM: To study the levels of serum sodium and potassium in newly diagnosed essential

hypertension and healthy individuals and to correlate it with risk factors.

MATERIALS AND METHODS: This cross-sectional analytical study included 100 individuals aged above 18 years, divided into Group-A (50 newly diagnosed essential hypertensives) and Group-B (50 healthy controls). Hypertension was diagnosed if SBP>140mmHg and DBP>90mmHg on three separate readings. Individuals with comorbidities or on medications

affecting blood pressure were excluded.

RESULTS: The mean age of Group-A and Group-B was 55.54±11.94 and 52.82±10.84 years, respectively. Group-A and Group-B included 27 and 26 males, 23 and 24 females, respectively. Group-A and Group-B included 38% and 16% smokers, 42% and 14% alcoholics, 64% and 24% obese, 16% and 8% had a family history of hypertension, respectively. There was a statistically significant association between hypertension with smoking (p<0.05), alcohol consumption (p<0.001), and obesity (p<0.01). Hypertensives had statistically significantly higher mean serum sodium levels (148.06±2.75mEq/L) and lower serum potassium levels (3.55±0.29mEq/L) compared to normotensives (p<0.0001). There was no significant association between serum sodium and potassium levels with risk factors (p>0.05).

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CONCLUSION: A significant association between serum electrolyte imbalances and primary

hypertension indicates that dietary interventions to reduce sodium and increase potassium intake

could be vital strategies for preventing and managing hypertension.

KEY WORDS: Serum Sodium, Serum Potassium, Essential Hypertension, Blood Pressure

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INTRODUCTION:

Hypertension is a leading cause of morbidity and mortality worldwide, significantly contributing to the burden of cardiovascular diseases, including coronary artery disease, stroke, and peripheral vascular disease. Primary hypertension, which accounts for more than 90% of all hypertension cases, has become an emerging health issue in India, mirroring trends seen globally. Unfortunately, many individuals with hypertension are unaware of their condition until it leads to irreversible damage to vital organs, such as the kidneys, heart, and brain, resulting in life-threatening complications like renal failure, myocardial infarction, and stroke. Alarmingly, in developed countries like the United States, despite 70% of the population being aware of their hypertension status, only 50% receive appropriate treatment, and only 20% achieve adequate control over their blood pressure.^{1,2}

Among the multiple factors contributing to the development of hypertension, hereditary predispositions, excessive sodium intake, inadequate potassium consumption, and an overactive sympathetic nervous system are frequently implicated. In countries like India, where dietary habits often include high sodium and low potassium intake, the risk of hypertension is exacerbated. Several studies have demonstrated a positive correlation between low serum potassium levels and

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elevated blood pressure, suggesting that dietary modifications, including the reduction of sodium

intake and the enhancement of potassium intake, may play a critical role in both the prevention

and management of hypertension.³

Despite the growing prevalence of hypertension in India, limited independent studies have been

conducted to examine the specific relationship between serum sodium and potassium levels and

blood pressure regulation in the Indian population. This study aims to address this gap by

investigating the serum sodium and potassium levels in hypertensive individuals and exploring

their potential role as biomarkers in the development and management of hypertension.

MATERIALS AND METHODS:

This cross-sectional analytical study was conducted over a period of six months at a tertiary care

teaching hospital. A total of 100 individuals were enrolled, comprising 50 newly diagnosed

primary hypertensive patients (study group) and 50 normotensive individuals (control group). The

hypertensive participants were recruited from the outpatient and inpatient units in the department

of General Medicine after obtaining informed consent. Controls were selected from caregivers of

hypertensive patients residing in the same environment, excluding first-degree relatives to

minimize genetic overlap.

The study included adults over the age of 18 who had been newly diagnosed with essential

hypertension and were not currently on any antihypertensive medications. Individuals were

excluded from the study if they had a diagnosis of diabetes mellitus, renal failure, secondary

hypertension, or peripheral vascular disease. Additional exclusion criteria included the presence

of acute diarrheal illnesses, current use of antihypertensive or non-steroidal anti-inflammatory

drugs, pregnancy, or the use of oral contraceptive pills.

To rule out secondary hypertension, a detailed clinical history was obtained, and all participants

underwent a comprehensive physical examination and relevant biochemical investigations.

Anthropometric data, including height and weight, were collected. Body mass index (BMI) was

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calculated as weight in kilograms divided by height in meters squared (kg/m²). Fundoscopy was

performed to assess for hypertensive retinopathy.

Blood pressure was measured using a standard mercury sphygmomanometer. Participants were

advised to abstain from smoking, tea, or coffee for at least 30 minutes before measurement.

Readings were taken with the patient in a seated position, after five minutes of rest. Blood pressure

was recorded on three separate occasions at one-week intervals. A diagnosis of hypertension was

made if systolic blood pressure (SBP) was ≥140 mmHg and/or diastolic blood pressure (DBP) was

≥90 mmHg, based on the average of the three readings.

Fasting venous blood samples were drawn under aseptic conditions and analyzed for serum sodium

and potassium using an automated electrolyte analyzer based on the ion-selective electrode (ISE)

method.

Data were entered into Microsoft Excel and analyzed using SPSS software. Descriptive statistics

were used for demographic and clinical variables. t-test and Chi-square test were employed to

compare continuous and categorical variables, respectively. Pearson's correlation coefficient was

used to assess the relationship between serum electrolyte levels, BMI, and blood pressure. A p-

value < 0.05 was considered statistically significant.

AIM OF THE STUDY:

1. To study the levels of serum sodium and potassium in newly diagnosed primary hypertension

patients.

2. To correlate the sodium and potassium levels with risk factors.

RESULTS:

This cross-sectional analytical study included 100 individuals aged above 18 years, divided into

two groups: Group A (50 newly diagnosed, untreated primary hypertensives) and Group B (50

normotensive healthy controls). The overall mean age was 54.18 ± 11.39 years, with Group A

having a mean age of 55.54 ± 11.94 years and Group B 52.82 ± 10.84 years. The majority of

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participants (31%) were aged \geq 61 years, followed by 28% in the 51–60-year age group. Gender distribution was similar between groups, with no statistically significant differences (p > 0.05).

Regarding lifestyle and risk factors, smoking, alcohol consumption, and obesity were more prevalent among hypertensives compared to controls. Specifically, 19 (38%) hypertensives were smokers, 21 (42%) consumed alcohol, and 22 (44%) were obese. In contrast, 8 (16%) controls were smokers, 7 (14%) consumed alcohol, and 10 (20%) were obese. These differences were statistically significant (p < 0.05 for smoking, p < 0.001 for alcohol, and p < 0.01 for obesity). A family history of hypertension was reported in 8 (16%) hypertensives and 4 (8%) controls, but this was not statistically significant (p > 0.05).

Body Mass Index (BMI) analysis showed that 50 individuals had a normal BMI (18.5–22.9), and 32 were categorized as obese (BMI \geq 25). The mean BMI among hypertensives was significantly higher at 24.28 \pm 4.11 compared to 22.12 \pm 2.71 in the control group (p < 0.01).

Biochemical analysis revealed that the mean serum sodium level in hypertensives was significantly elevated (148.06 ± 2.75 mEq/L) compared to normotensives (139.28 ± 4.06 mEq/L), with high statistical significance (p < 0.0001). Conversely, serum potassium levels were significantly lower in hypertensives (3.55 ± 0.29 mEq/L) than in controls (4.60 ± 0.41 mEq/L), also with high statistical significance (p < 0.0001). There was no significant association found between serum sodium and potassium levels with other risk factors such as smoking, alcohol use, BMI, or family history of hypertension (p > 0.05).

DISCUSSION:

Hypertension remains one of the most significant risk factors for cardiovascular diseases, including stroke, myocardial infarction, and kidney failure.⁴ The role of electrolyte imbalances, particularly in serum sodium and potassium, has garnered attention in understanding the pathophysiology of primary hypertension.⁵ Given the vital roles of potassium and sodium in maintaining cellular function, the imbalances in these electrolytes can adversely affect health, further complicating the pathophysiology of hypertension.⁶ In this study, we aimed to investigate the serum sodium and potassium levels in newly diagnosed hypertensive patients and to explore their correlation with blood pressure and risk factors for hypertension. The findings indicate a significant alteration in

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the serum sodium and potassium levels in hypertensive individuals compared to normotensive

controls, which is consistent with the findings of previous studies.^{7,8}

Several studies have indicated that an elevated serum sodium level correlates with an increased

risk of hypertension.^{6,9} In this study, the mean sodium levels in hypertensive patients (Group A)

were significantly higher than in the normotensive controls (Group B), which is in line with prior

research.^{7,9} Elevated serum sodium levels may increase blood pressure by influencing renal sodium

handling and altering the sensitivity of vascular smooth muscle to vasopressors, thus contributing

to vascular stiffness and hypertension.^{5,11} Furthermore, the relationship between sodium intake and

hypertension risk has been well documented. 10 Although some studies suggest the regulation of

blood pressure through sodium intake may be independent of serum sodium levels, others show a

clear association, particularly when dietary sodium intake is high.^{4,5}

On the other hand, serum potassium levels were found to be significantly lower in hypertensive

patients compared to the control group in our study, which mirrors findings from previous

research. 9,12 Potassium plays a crucial role in maintaining normal cellular function, particularly in

the regulation of vascular tone and blood pressure. A lower potassium intake has been shown to

lead to increased vascular resistance, contributing to hypertension. 11 Potassium deficiency may

cause alterations in vascular smooth muscle cell function and sodium retention, which can elevate

blood pressure. 13 Moreover, studies have suggested that low potassium levels in the blood are

associated with a higher risk of developing hypertension. 10

The sodium-to-potassium ratio has also emerged as an important factor in hypertension risk, with

some studies showing a stronger association between this ratio and blood pressure outcomes than

sodium or potassium alone. 10 This may reflect the interplay between sodium retention and

potassium excretion, which together affect blood pressure regulation. Our study also showed a

significant inverse relationship between serum potassium and blood pressure, supporting previous

studies that have indicated that higher potassium levels may have a protective effect against

hypertension. 11,13

In addition to electrolyte imbalances, several modifiable and non-modifiable risk factors contribute

to hypertension. Our study found that obesity, smoking, and alcohol consumption were

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significantly associated with hypertension, which corroborates the findings of other studies. ^{14,15} Obesity, particularly abdominal obesity, has long been recognized as a major risk factor for hypertension, with numerous studies showing that overweight individuals are more likely to develop high blood pressure due to metabolic and hormonal changes associated with excess adiposity. ¹⁴ Similarly, smoking has been shown to acutely increase blood pressure, potentially through sympathetic nervous system activation and vasoconstriction. ^{16,17} Alcohol consumption also plays a significant role in hypertension development, with excessive intake being linked to higher blood pressure levels. ¹⁸ Our study found significant associations between these risk factors and hypertension, emphasizing the importance of lifestyle modifications in preventing and managing high blood pressure.

The relationship between serum sodium and potassium levels and blood pressure regulation is complex, and it is important to note that other factors such as diet, renal function, and genetic predispositions could further modulate these electrolyte levels. A study demonstrated that dietary potassium intake, rather than serum potassium levels alone, is an important determinant of hypertension risk. Additionally, the impact of kidney function on sodium and potassium regulation cannot be overlooked, as renal dysfunction is closely tied to the development of hypertension. The study's exclusion of patients with renal failure or secondary hypertension helps to isolate the effects of primary hypertension on electrolyte imbalances.

Despite these valuable insights, our study has limitations. The sample size, although sufficient for initial findings, may not provide enough power to detect smaller associations, particularly for more complex relationships between electrolytes and blood pressure. Furthermore, serum sodium and potassium levels measured at a single time point may not fully capture the long-term fluctuations in these electrolytes. Longitudinal studies with repeated measurements of serum electrolytes and 24-hour urinary sodium and potassium excretion would provide a more comprehensive understanding of these relationships. Moreover, this study did not account for dietary sodium and potassium intake, which is a crucial determinant of serum electrolyte levels.¹¹

Our study adds to the growing body of evidence suggesting that altered serum sodium and potassium levels are associated with primary hypertension. Elevated serum sodium and decreased

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serum potassium levels may contribute to the pathophysiology of hypertension, and their balance

may be a useful target for intervention. Further research, particularly in diverse populations with

larger sample sizes and longitudinal data, is necessary to clarify the mechanisms underlying these

associations and to explore potential therapeutic strategies.

CONCLUSION:

The present study highlights the significant association between serum electrolyte levels and the

development of primary hypertension, with positive correlation for serum sodium levels and

negative correlation for serum potassium levels, further supporting the importance of maintaining

a proper electrolyte balance in blood pressure regulation. Our study also emphasizes the role of

body mass index (BMI) as a significant factor in the development of hypertension, independent of

gender. The increasing prevalence of high-sodium, low-potassium diets, coupled with rising

obesity rates, may contribute to the global hypertension epidemic, especially in genetically

predisposed populations. These findings suggest that dietary interventions aimed at reducing

sodium intake and increasing potassium consumption may be crucial strategies for preventing and

managing hypertension.

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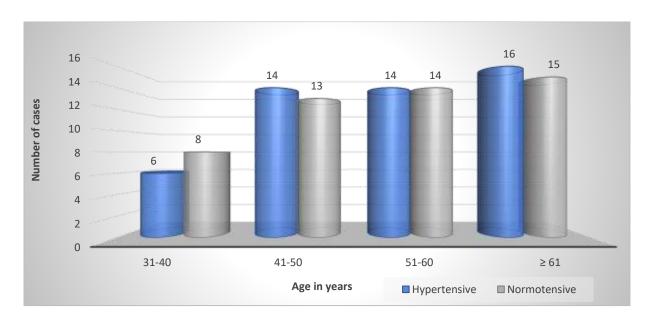
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RESULTS AND OBSERVATIONS

| TABLE 1. BASIC CHARACTERISTICS | | | | | |
|--------------------------------------|------------------|--------------------|---------------------------------|--|--|
| CHARACTERISTICS Age in years (mean) | | HYPERTENSIVES | NORMOTENSIVES 51.74 ± 10.84 | | |
| | | 55.4 ± 11.94 | | | |
| Sex | Male | 20 | 19 | | |
| (in numbers) | Female | 15 | 16 | | |
| | Smoker | 19 | 8 | | |
| | Alcoholic | 21 | 7 | | |
| Risk factor (in numbers) | Family History | 8 | 4 | | |
| | BMI more than 25 | 22 | 10 | | |
| | BMI less than 25 | 28 | 40 | | |
| Serum Sodium mEq/L (mean) | | 148.06 ± 2.75 | 139.28 ± 4.06 | | |
| Serum Potassium mEq/L(mean) | | 3.55 ± 0.29 | 4.60 ± 0.41 | | |
| Systolic BP | (mean) mmHg | 164.60 ± 18.25 | 112.92 ± 7.56 | | |
| Diastolic BF | (mean) mmHg | 95.14 ± 12.76 | 72.04 ± 6.10 | | |

| TABLE 2. DISTRIBUTION OF CASES AND CONTROLS WITH RESPECT TO AGE | | | | | | |
|---|---|---------|---------------|---------------|---------------|------|
| ACE in moons | HYPERTENSIVES | | NORMOT | NORMOTENSIVES | | OTAL |
| AGE in years | No. | % | No. | % | No. | % |
| 31-40 | 6 | 12.0 | 8 | 16.0 | 14 | 14.0 |
| 41-50 | 14 | 28.0 | 13 | 26.0 | 27 | 27.0 |
| 51-60 | 14 | 28.0 | 14 | 28.0 | 28 | 28.0 |
| ≥ 61 | 16 | 32.0 | 15 | 30.0 | 31 | 31.0 |
| Total | 50 | 100.0 | 50 | 100.0 | 100 | 100 |
| Mean ± SD | 55.54 = | ± 11.94 | 52.82 ± 10.84 | | 54.18 ± 11.39 | |
| P – value | t = 1.192 $P = 0.236$, Not Significant | | | | | |

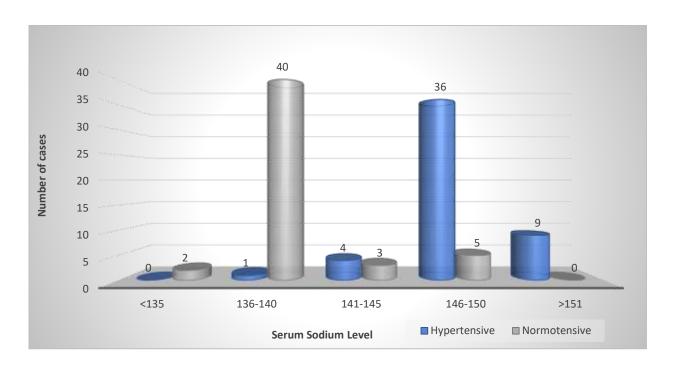


| TABLE 3. DISTRIBUTION OF CASES AND CONTROLS WITH RESPECT TO RISK FACTORS | | | | | | | |
|--|----------------------|----------------------|---------------|------|-------|-----------------|---|
| Risk | | Evocuoney | Hypertensives | | Normo | tensives | Test value |
| Factors | Categories | Frequency | No. | % | No. | % | and p-value |
| C 1 | Smoker | 27 | 19 | 38.0 | 8 | 16.0 | $X^{2} = 6.139,$ P = 0.013, Significant |
| Smoking | Non-smoker | 73 | 31 | 62.0 | 42 | 84.0 | |
| A111 | Alcoholic | Alcoholic 28 21 42.0 | 42.0 | 7 | 14.0 | $X^2 = 9.722$, | |
| Alcohol | Non-alcoholic | 72 | 29 | 58.0 | 43 | 86.0 | P = 0.001, Highly significant |
| Family | Family history | 12 | 8 | 16.0 | 4 | 8.0 | $X^2 = 1.51,$ |
| history | No-family history | 88 | 42 | 84.0 | 46 | 92.0 | P = 0.218, Significant |

| TABLE 4. DISTRIBUTION OF CASES AND CONTROLS WITH RESPECT TO BMI | | | | | | |
|---|-------|---------------|-------|---------------|--------|--|
| PMI ka/m² | Total | HYPERTENSIVES | | NORMOTENSIVES | | |
| BMI kg/m ² | Total | No. | % | No. | % | |
| Underweight < 18.5 | 8 | 3 | 6.0 | 5 | 10.0 | |
| Normal weight 18.5 - 22.9 | 50 | 19 | 38.0 | 31 | 62.0 | |
| Overweight 23.0 - 24.9 | 10 | 6 | 12.0 | 4 | 8.0 | |
| Obese ≥ 25 | 32 | 22 | 44.0 | 10 | 20.0 | |
| Total | 100 | 50 | 100.0 | 50.0 | 100.0 | |
| Mean ± SD | | 24.28 ± 4.11 | | 22.12 = | ± 2.71 | |

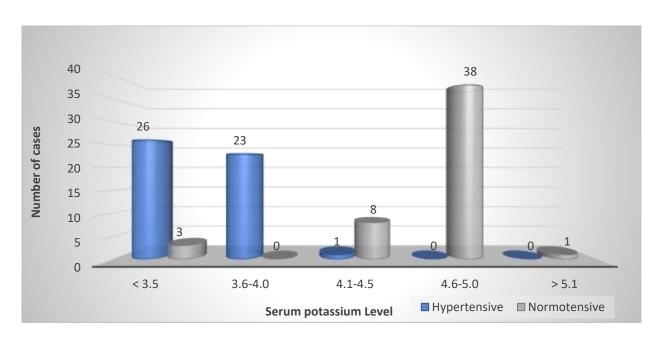
| t test value and D value | t = 2.092 D = 0.002 Highly significant | |
|--------------------------|---|--|
| t-test value and P-value | t = 3.083, $P = 0.003$, Highly significant | |
| | | |

| CD CODUM LEVEL | HYPER | TENSIVES | NORMO | TENSIVES |
|------------------|--|-----------|--------|----------|
| SR. SODIUM LEVEL | No. | % | No. | % |
| <135 | 0 | 0.0 | 2 | 4.0 |
| 136-140 | 1 | 2.0 | 40 | 80.0 |
| 141-145 | 4 | 8.0 | 3 | 6.0 |
| 146-150 | 36 | 72.0 | 5 | 10.0 |
| >151 | 9 | 18.0 | 0 | 0.0 |
| Total | 50 | 100.0 | 50 | 100.0 |
| Mean ± SD | 148.0 | 06 ± 2.75 | 139.28 | 3 ± 4.06 |
| t-test, P-value | t = 12.657, $P = 0.000$, Highly Significant | | | |



| HYPERT | TENSIVES | NORMO | TENSIVES |
|--------|-----------------|-----------------------------------|--|
| No. | % | No. | % |
| 26 | 52.0 | 3 | 6.0 |
| 23 | 46.0 | 0 | 0.0 |
| 1 | 2.0 | 8 | 16.0 |
| 0 | 0.0 | 38 | 76.0 |
| 0 | 0.0 | 1 | 2.0 |
| 50 | 100.0 | 50 | 100.0 |
| | No. 26 23 1 0 0 | 26 52.0 23 46.0 1 2.0 0 0.0 0 0.0 | No. % No. 26 52.0 3 23 46.0 0 1 2.0 8 0 0.0 38 0 0.0 1 |

| 1 | t-test, P-value | t = 14.685, | P = 0.000, | Highly Significant | | |
|---|-----------------|-------------|------------|--------------------|--|--|
|---|-----------------|-------------|------------|--------------------|--|--|



| TA | BLE 7. COMPARISO | ON OF SERUM SODI | UM WITH RISK FAC | ΓORS |
|-----------------|-------------------|----------------------------|----------------------------|---------------------------|
| RISK FACTORS | CATEGORIES | HYPERTENSIVES Mean ± SD | NORMOTENSIVES Mean ± SD | Test Value and P-Value |
| SMOKING | Smoker | 147.52 ± 3.71 | 139.28 ± 4.06 | t = 1.076, P = 0.287 |
| SMOKING | Non-smoker | 148.38 ± 1.94 | 140.15 ± 3.89 | Not Significant |
| ALCOHOL | Alcoholic | 147.71 ± 3.53 | 138.72 ± 4.21 | t = 0.753, P = 0.455 |
| ALCOHOL | Non-alcoholic | 148.31 ± 2.03 | 139.95 ± 3.78 | Not Significant |
| FAMILY | Family History | 148.75 ± 2.66 | 137.88 ± 4.32 | t = 0.771, P = 0.445 |
| HISTORY | No-family History | 147.92 ± 2.77 | 140.67 ± 4.10 | Not Significant |
| | Under weight | 147.33 ± 2.08 | 138.45 ± 3.95 | |
| BMI | Normal weight | 147.94 ± 3.25 | 139.12 ± 4.25 | F = 1.573, P = 0.209 |
| BMI | Overweight | 146.17 ± 3.12 | 141.03 ± 3.84 | Not Significant |
| | Obese | 148.77 ± 2.04 | 138.98 ± 4.00 | |

| TAB | TABLE 8. COMPARISON OF SERUM POTASSIUM WITH RISK FACTORS | | | | | | | |
|-----------------|--|----------------------------|----------------------------|---|--|--|--|--|
| RISK FACTORS | CATEGORIES | HYPERTENSIVES Mean ± SD | NORMOTENSIVES Mean ± SD | Test Value and P-Value | | | | |
| SMOKING | Smoker | 3.57 ± 0.26 | 4.60 ± 0.41 | t = 0.448, P = 0.656 | | | | |
| SMORING | Non-smoker | 3.53 ± 0.30 | 4.58 ± 0.39 | Not Significant | | | | |
| ALCOHOL | Alcoholic | 3.62 ± 0.31 | 4.63 ± 0.42 | t = 1.448, P = 0.154 | | | | |
| ALCOHOL | Non-alcoholic | 3.50 ± 0.27 | 4.55 ± 0.40 | Not Significant | | | | |
| FAMILY | Family History | 3.62 ± 0.19 | 4.67 ± 0.43 | t = 0.795, P = 0.431 | | | | |
| HISTORY | No-family History | 3.53 ± 0.30 | 4.61 ± 0.38 | Not Significant | | | | |
| | Under weight | 3.53 ± 0.32 | 4.59 ± 0.41 | | | | | |
| BMI | Normal weight | 3.64 ± 0.33 | 4.62 ± 0.44 | E 1500 D 0225 | | | | |
| BMI | Overweight | 3.58 ± 0.29 | 4.56 ± 0.40 | F = 1.508, P = 0.225 Not Significant | | | | |
| | Obese | 3.45 ± 0.23 | 4.64 ± 0.39 | | | | | |