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ORIGINAL RESEARCH

# Patterns of Dyslipidemia in the Anemic and Nonanemic Hypertensive Saudi Population: A Cross-Sectional Study

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**Background:** Risk factors of cardiovascular disease include dyslipidemia, hypertension (HTN), and anemia. Our objective is to assess the patterns of dyslipidemia in the anemic and non-anemic hypertensive Saudi population.

**Methods:** A retrospective, cross-sectional study of the gender, blood pressure, lipid markers, and CBC parameters of 3111 subjects, which were retrieved from the database of Al-Borg Medical Laboratories over a six-year period (2014–2019), was carried out. Means were compared among study groups and the prevalence, association, and diagnostic accuracy of lipid markers for HTN were evaluated.

**Results:** TG, LDL/HDL, and TG/HDL were significantly higher ($P < 0.0001$) in hypertensives. Anemia reduces TC and LDL ($P < 0.0001$) in both genders, and reduces all markers and increases HDL ($P < 0.01$) in male hypertensives. HTN was more prevalent in anemics with high TC than normal TC (38.23% vs 11.17%, $P < 0.001$) and in non-anemics with high TG than normal TG (56.31% vs 21.22%, $P < 0.001$). Furthermore, non-anemics with high TG/HDL had the highest risk for HTN (RR = 1.20, 95% CI = 1.1551–1.2473, $P < 0.0001$). Elevated TC ($P = 0.0142$), TG ($P < 0.0001$), TC/HDL ($P < 0.0001$), LDL/HDL ($P < 0.0001$), and TG/HDL ($P < 0.0001$), and low HDL ($P < 0.0001$) were risk factors for HTN as shown by ORs. In anemics, high TC/HDL, LDL/HDL, and TG/HDL were not. Importantly, only TG and TG/HDL had a discriminating capacity for HTN.

**Conclusion:** The anemic state of hypertensive Saudi patients influences dyslipidemia which warrants further investigation.

**Keywords:** hypertension, dyslipidemia, anemia, prevalence, Saudi Arabia

## Plain Language Summary

The risk for cardiovascular disease significantly increases with increased blood lipids, blood pressure (hypertension), and anemia. This study investigates changes in blood levels of different lipids in relation to hypertension in the Saudi population. The isolated and combined effect of anemia and hypertension on blood lipids was also examined. Gender, age, and laboratory results of 3111 subjects were retrieved from the database of Al-Borg Medical Laboratories and analyzed. We found that distinct lipid markers are differentially altered based on gender, anemic state, and/or hypertension. In particular, HDL, TG, LDL/HDL, and TG/HDL were significantly higher in hypertensive subjects. In males with hypertension, the presence of anemia reduces TG, TC/HDL, LDL/HDL, and TG/HDL and increases HDL. Hypertension was more common in subjects with high LDL (60.95% vs 15.22%), LDL/HDL (62.82% vs 25.43%), and TG/HDL (43.52% vs 25.26%) compared to those with normal results. Hypertension was also more common in subjects with anemia who have high TC (38.23%) compared to those with normal TC (11.17%). In non-anemic subjects, hypertension was more common with high TG than normal TG (56.31% vs 21.22%), and subjects who had high TG/HDL had the highest risk for hypertension. We identified increased TC, TG, TC/HDL, LDL/HDL, and TG/HDL, and low HDL as risk factors for hypertension. Among all lipid

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markers, only TG and TG/HDL may be useful to diagnose hypertension. Altogether, our findings highlight the importance of assessing the anemic state of hypertension patients along with measuring blood lipids in order to reduce the burden of cardiovascular disease.

# Introduction

Cardiovascular disease (CVD) is the leading cause of death worldwide with an estimated total of 18 million deaths in 2019 making up 32% of the global death toll.[1] CVD accounts for 50% of deaths in the United States and causes around 4 million annual deaths in Europe representing 45% of all mortalities.[2] In Saudi Arabia, 42% of deaths were due to CVD whose prevalence is projected to increase by 2035 with concomitant rise in costs to approximately $10 billion.[3] Established risk factors for CVD include poor dietary habits, sedentary lifestyle, obesity, dyslipidemia, hypertension (HTN), tobacco smoking, alcohol consumption, and anemia.[4,5] In fact, anemia is detected in one in three individuals with acute coronary syndrome,[6] and one in two cases of chronic heart failure.[7] More alarmingly, the presence of anemia increases the risk of death from chronic heart failure from 16% to 28%.[8]

Dyslipidemia is defined as an abnormal blood level of any lipid species brought about by either genetic or lifestyle factors. These include obesity, tobacco use, physical inactivity, and bad dietary habits. Obesity, as a predisposing factor for dyslipidemia, is projected to exert an enormous national and personal economic burden given the projected increase in prevalence in Western and Eastern countries.[9] In a systematic review, it was revealed that the prevalence of dyslipidemia had a median of 43.5% and 63% in middle- and low-income countries, respectively. Conceivably, childhood obesity-related HTN was more prevalent in middle- and low-income countries in comparison to high-income countries (35.6% vs 12.7%). Moreover, Asia had the highest prevalence of childhood obesity-related HTN at 38.6% followed by South America (25.3%) and Europe (20.1%).[10] For instance, in Jordan, Vietnam, and China, the odds of having dyslipidemia are higher in overweight individuals than in those with normal weight.[9] In a case-control study of Vietnamese children, Hanh et al found that obesity significantly increased the risk of hypertriglyceridemia with prenatal nutrition, parental BMI, and high birth weight acting as precipitating factors.[11] The differential diagnosis of dyslipidemia must include common comorbidities such as nephrotic syndrome, biliary obstruction, and hypothyroidism.[12] Dyslipidemia is complicated by CVD whose risk could be significantly attenuated by lipid-lowering medications and lifestyle improvement.[13]

HTN is a global pandemic affecting 1.28 billion individuals aged 30–79 years, 46% of whom are unaware of their condition, and 79% has it uncontrolled. A principal cause of mortality, HTN is complicated by brain, heart, kidney, and multiple organ dysfunction.[14] In addition to CVD risk factors, genetic predisposition, aging, diabetes, and kidney disease also contribute to HTN development and prognosis. Since only 42% of HTN cases are diagnosed and treated, measures must be taken to improve screening and detection efforts. Thus, understanding the biochemical determinants of HTN and the shared and distinct patterns of comorbidities is of utmost importance. In this study, we aim to examine the prevalence of HTN in the Saudi population in association with dyslipidemia and anemia.

# Materials and Methods

## Study Population

This study was conducted according to the Declaration of Helsinki and approved by the Biomedical Ethics Unit of Al-Borg Medical Laboratories. Consent for participation was waived by the Biomedical Ethics Unit due to the retrospective nature of the study and lack of access to participant identifiers. Laboratory results (2014–2019) were extracted from the database for 3111 subjects. Males and females were separated and age groups were formed as shown in Table 1. Young (<17 years), young adults (18–39 years), adults (40–64 years), and elderlies (≥65 years) were included. Dyslipidemia was defined by a TC of ≥200 mg/dl, LDL of ≥100 mg/dl, HDL of <40 mg/dl, or TG of ≥150 mg/dl.[15] Consequently, a TC/HDL ratio of ≥6, a LDL/HDL ratio of >2.5, and a TG/HDL ratio of >2 were considered high. HTN was defined, according to the most recent guidelines set forth by the American College of Cardiology, by systolic or diastolic blood pressure (BP) of ≥120 and ≥80 mmHg, respectively.[16] If systolic BP is ≥130 mmHg, then diastolic BP of >80 mmHg is diagnostic.[17,18] Anemia was defined by a hemoglobin (Hgb) level of <12 g/dl.[19] Subjects with missing data necessary for a particular analysis were excluded.

**Table 1** Distribution of Study Subjects

| Gender | No. of Subjects (%) |
|---|---|
| Male | 1336 (42.94) |
| Young | 56 (1.80) |
| Young adults | 458 (14.72) |
| Adults | 681 (21.89) |
| Elderlies | 141 (4.53) |
| Female | 1751 (56.28) |
| Young | 62 (1.99) |
| Young adults | 777 (24.97) |
| Adults | 723 (23.24) |
| Elderlies | 189 (6.07) |
| Unknown | 24 (0.77) |

## Statistical Analysis

Data are shown as means ± confidence interval (95% CI). Two-tailed, unpaired Student's *t*-test and one-way ANOVA were used to statistically compare two and three or more groups, respectively. Association among variables was assessed by the relative risk (RR) and odds ratio (OR). GraphPad Prism v9.2.0 (GraphPad Software, Inc., San Diego, CA, USA) was used for analysis, and statistical significance was set at a $P$ value of <0.05.

# Results

Our analysis revealed differential influence of isolated and combined presence of HTN and anemia on lipid markers. When both genders were considered together, hypertensives were found to have significantly elevated TG, TC/HDL, LDL/HDL, and TG/HDL (Figure 1A). In the normotension group (NTN), LDL/HDL was significantly lower in anemics, whereas in HTN, all markers were significantly reduced in anemics except HDL (Figure 1B). In male hypertensive patients, HDL was significantly lower but all other markers were significantly elevated (Figure 2A). The presence of anemia in male normotensives caused HDL to be significantly higher and LDL/HDL to be significantly lower compared to non-anemics (Figure 2B). Additionally, all markers were significantly reduced except HDL which was significantly higher in the HTN anemic group (Figure 2B). In hypertensive females, only HDL was significantly reduced whereas TG, TC/HDL, LDL/HDL, and TG/HDL were significantly increased (Figure 3A). The combined presence of anemia and HTN resulted in significantly lower TC, LDL, and TG values compared to HTN alone (Figure 3B).

Moreover, although TC (Figure 4A), LDL (Figure 4B), and HDL (Figure 4C) displayed modest diagnostic ability for HTN, TG showed the best performance (AUC = 0.612, $P < 0.0001$) in discriminating individuals with NTN and those with HTN (Figure 4D). Similar to TG, TG/HDL had the highest discriminating capacity among lipid ratios for NTN and HTN (AUC = 0.621, $P < 0.0001$) compared to TC/HDL (Figure 4E) and LDL/HDL (Figure 4F).

Notably, HTN was more prevalent in anemics with elevated TC (Table 2) although hypertensive anemics had lower mean TC compared to non-anemics (Figures 1B, 2B, and 3B). Surprisingly, all abnormal markers except high LDL were found to be risk factors for HTN (Table 3) despite it being more prevalent in those with elevated LDL (Table 2).

Among all lipid markers, TG exhibited the most consistent alterations as it was higher in hypertensive males (Figure 2B) and females (Figure 3B) which was significantly lowered in presence of anemia. In congruence, HTN was more prevalent in non-anemics with hypertriglyceridemia (Table 2). Importantly, elevated TG/HDL, along with low HDL, carried the greatest risk for HTN in non-anemics as revealed by calculated ORs (Table 3).

# Discussion

The prevalence of HTN has alarmingly increased by 81% from 594 million to 1.13 billion over the period 1975–2015, constituting a major threat to public health particularly in low- and middle-income countries. This geographical disparity may be attributed to varying levels of both physician[20] and patient[14,21] awareness and instituted preventive measures of modifiable risk factors in developed countries.[22] In this report, we highlight the differential dysregulation of lipid

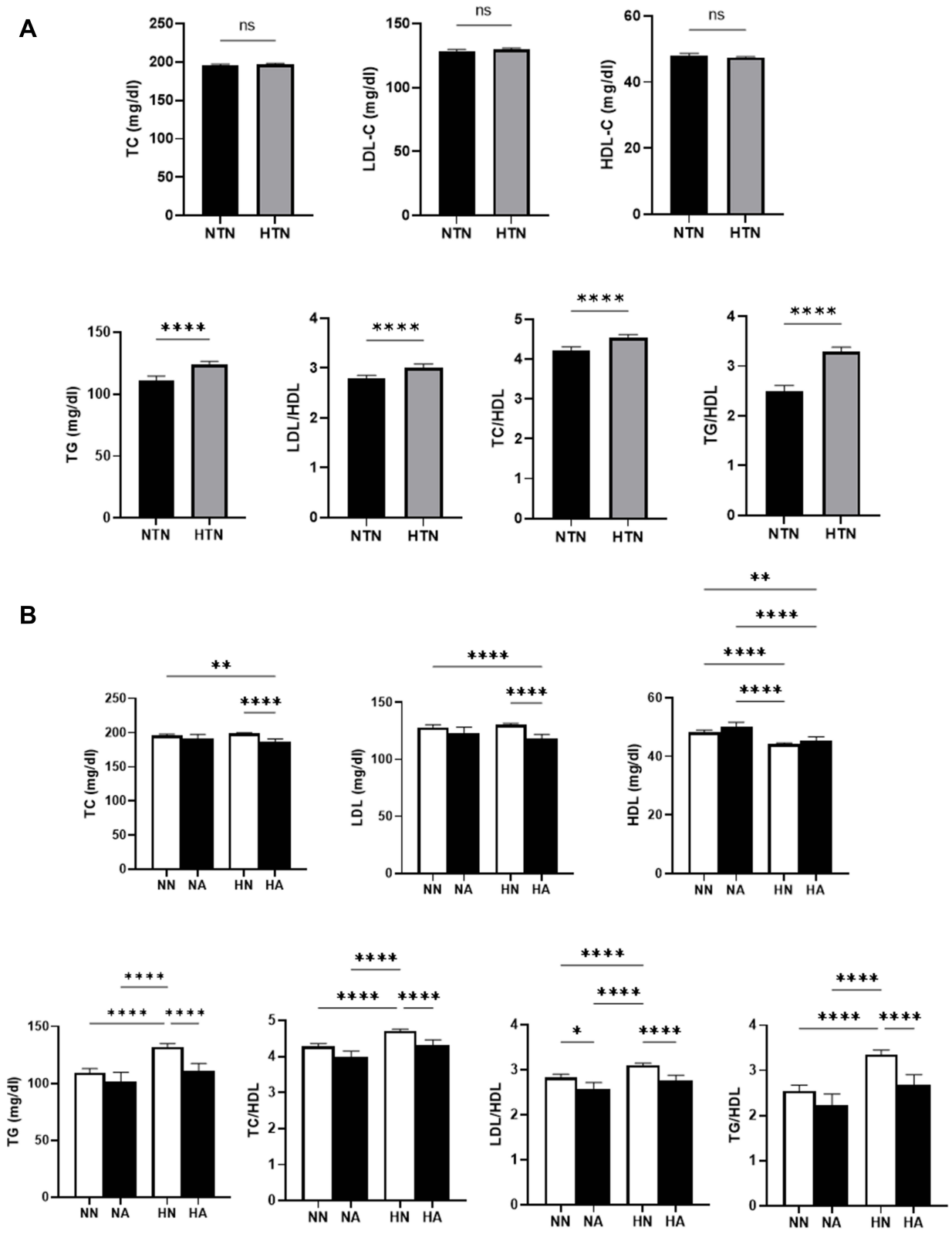


**Figure 1** Distribution of lipid markers in both genders. (**A**) Comparison of lipid markers in NTN and HTN (mean ± 95% CI). (**B**) Comparison of lipid markers in NTN and HTN with and without anemia. Data are shown as mean ± 95% CI, *($P < 0.05$), **($P < 0.01$), and ****($P < 0.0001$).

**Abbreviations**: NN, normotensive non-anemics; NA, normotensive anemics; HN, hypertensive non-anemics; HA, hypertensive anemics; ns, not significant.

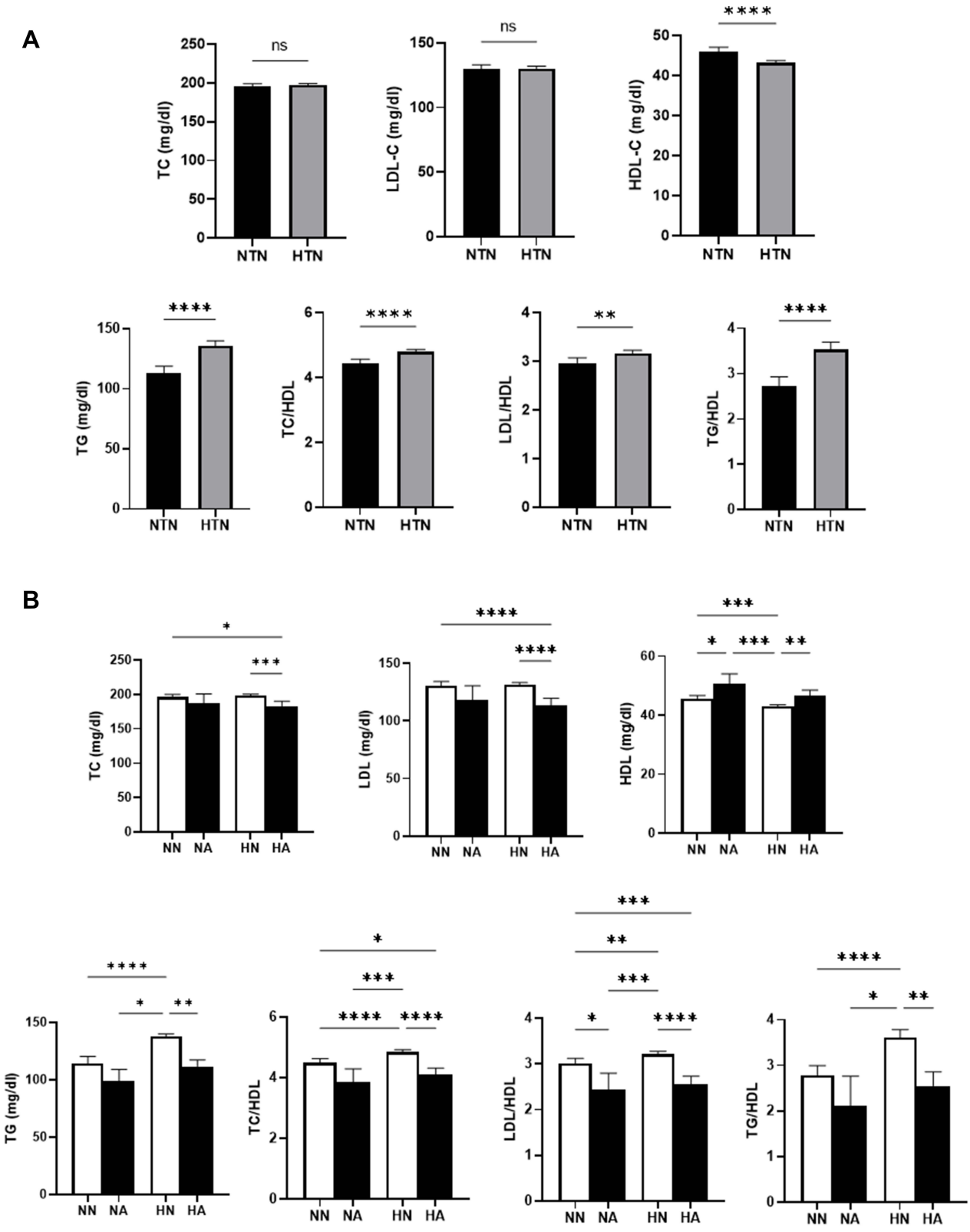


**Figure 2** Distribution of lipid markers in males. (**A**) Comparison of lipid markers in NTN and HTN. (**B**) Comparison of lipid markers in NTN and HTN with and without anemia. Data are shown as mean ± 95% CI, *($P < 0.05$), **($P < 0.01$), ***($P < 0.001$), and ****($P < 0.0001$).

**Abbreviations**: NN, normotensive non-anemics; NA, normotensive anemics; HN, hypertensive non-anemics; HA, hypertensive anemics; ns, not significant.

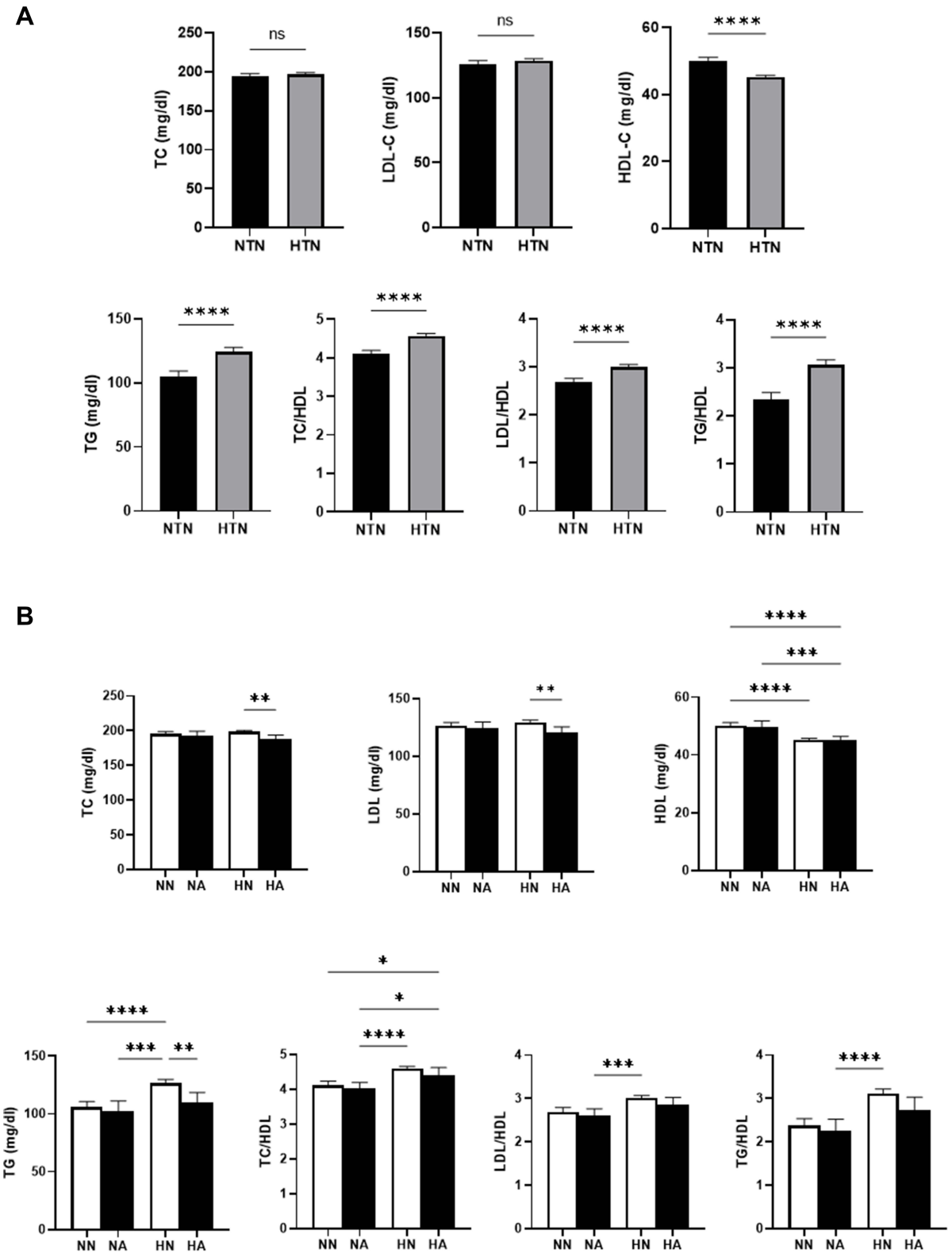


**Figure 3** Distribution of lipid markers in females. (**A**) Comparison of lipid markers in NTN and HTN. (**B**) Comparison of lipid markers in NTN and HTN with and without anemia (mean ± 95% CI). Data are shown as mean ± 95% CI, *($P < 0.05$), **($P < 0.01$), ***($P < 0.001$), and ****($P < 0.0001$).
**Abbreviations**: NN, normotensive non-anemics; NA, normotensive anemics; HN, hypertensive non-anemics; HA, hypertensive anemics; ns, not significant.

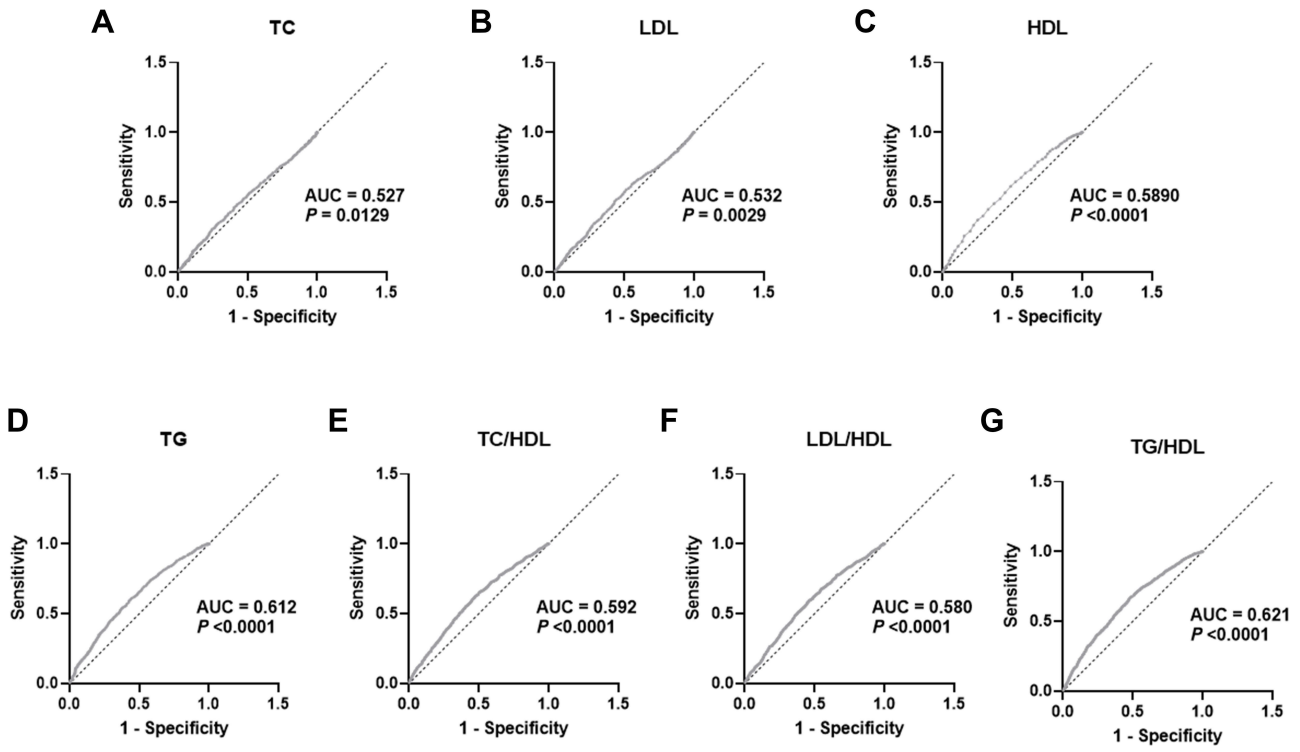


**Figure 4** ROC curve analysis of the diagnostic accuracy of lipid markers for HTN in both genders. Area under the curve (AUC) and *P* value of the diagnostic accuracy of (**A**) TC, (**B**) LDL, (**C**) HDL, (**D**) TG, (**E**) TC/HDL, (**F**) LDL/HDL, and (**G**) TG/HDL.

metabolism in light of HTN and anemic status in the Saudi population and notably underscore the association of hypertriglyceridemia and elevated TG/HDL, but not TC, with HTN.

A recent study reported a prevalence of 61.6% for HTN and 51.4% for abnormal BMI in Saudi young adults, and, compared to lean children, the obese were almost six times more likely to develop HTN.[18] Dyslipidemia and tobacco smoking were more common in men than women,[23,24] but anemia was consistently more prevalent in women especially those with low income.[19] In contrast, unhealthy dietary habits and physical inactivity show no significant gender disparity.[25] It was, however, evident in the awareness to and prevalence of HTN in favor of females.[26,27] Response to stress and anxiety differ between genders with females being more susceptible,[28] necessitating a multidisciplinary approach to study HTN. Furthermore, whereas males in an Indian urban setting had higher odds of HTN,[29] being a female increased the risk of HTN in Bangladesh.[30] The impact of HTN and diabetes mellitus on the risk of myocardial infarction was more pronounced in females.[31] Because BP rises after menopause, premenopausal females had a lower prevalence of HTN than age-matched males.[32] Beyond 65 years of age, HTN is more prevalent in females.[33] These differences strongly point to the involvement of distinct BP regulatory mechanisms in men and women. Sex steroids are particularly involved in the differential susceptibility of males and females to chronic conditions,[34] along with the renin-angiotensin system.[27] Differences in the efficacy of preventive measures and in the response to treatment remain, nonetheless, to be investigated. Intriguingly, tobacco smoking was significantly lower in Saudis with CVD,[22] further highlighting the ethnic-specific patterns of CVD risk factors.

In one study, it was found that over half of the Saudi population had hypercholesterolemia[35] at least in part attributed to unhealthy dietary habits.[4,36] In adult Saudi diabetics, 66.5% had dyslipidemia[15] and correcting dyslipidemia and HTN has been demonstrated to improve bone markers by promoting osteoblast growth and function as well as preventing excess urinary loss of calcium.[37]

Age-standardized disability-adjusted life years caused in particular by high LDL were highest in Europe and are rising in Saudi Arabia[38] in parallel to the annual increase in CVD risk factors in that country.[39] For instance, hypertriglyceridemia was common in 40.3% of Saudis.[35] Furthermore, the PURE-Saudi study revealed that 32.1% of

**Table 2** Prevalence (%) of HTN in Studied Population

| | All Subjects | Non-Anemics | Anemics | *P** |
|---|---|---|---|---|
| Unadjusted | 76.15 | 77.5 | 49.41 | |
| High TC | 35.12 | 36.91 | 38.23 | <0.001 |
| Normal TC | 41.03 | 40.62 | 11.17 | |
| Unadjusted | 76.17 | 77.54 | 49.41 | |
| High LDL | 60.95 | 62.82 | 43.52 | 0.290 |
| Normal LDL | 15.22 | 14.72 | 5.88 | |
| Unadjusted | 76.17 | 77.54 | 49.41 | |
| Low HDL | 27.60 | 28.72 | 10.0 | <0.05 |
| Normal HDL | 48.56 | 48.81 | 39.41 | |
| Unadjusted | 76.15 | 77.54 | 49.41 | |
| High TG | 20.20 | 56.31 | 15.29 | <0.001 |
| Normal TG | 55.94 | 21.22 | 34.11 | |
| Unadjusted | 76.15 | 77.54 | 49.41 | |
| High TC/HDL | 11.51 | 12.24 | 7.05 | 0.724 |
| Normal TC/HDL | 64.64 | 65.30 | 42.35 | |
| Unadjusted | 76.15 | 77.54 | 49.41 | |
| High LDL/HDL | 50.71 | 52.75 | 32.35 | 0.962 |
| Normal LDL/HDL | 25.43 | 24.79 | 17.05 | |
| Unadjusted | 76.15 | 77.5 | 49.41 | |
| High TG/HDL | 50.89 | 52.77 | 29.41 | 0.329 |
| Normal TG/HDL | 25.26 | 24.76 | 20.0 | |

**Note**: *Effect of marker on the prevalence in non-anemics vs anemics.

2047 participants had dyslipidemia, and 30.3% had HTN, which was more common in elderlies and in rural as opposed to urban areas.[4] In contrast, hypertriglyceridemia and smoking were more prevalent in urban Saudis,[35,40] as was HTN in a larger study.[41] Altogether, these observations corroborate our findings that TG and TG/HDL may serve as the best predictors of HTN in our population.

It was recently reported that impaired renal function and anemia cooperatively increase CVD risk.[5] Anemia in CVD is caused by malnutrition, bone marrow suppression, impaired renal function leading to low erythropoietin, medications (eg, acetylcholinesterase inhibitors), and aging.[5,42] It remains elusive, however, whether anemia reflects disease severity or precipitates outcomes. In any case, inflammation and related oxidative stress observed in anemic states[5,43] may account for the vascular injury typical of CVD. Indeed, the hematocrit was significantly higher in hypertensive than in normotensive subjects (40.79%, 95% CI 40.47–41.12 vs 42.46%, 95% CI 42.25–42.68, P < 0.0001), but after adjustment for Hgb, anemic hypertensives had significantly lower hematocrit than non-anemics (43.68% 95% CI 43.54–43.84 vs 32.91%, 95% CI 32.45–33.38, $P < 0.0001$). Therefore, reduced red cell mass may explain at least in part the apparent protective role of anemia against HTN observed in the current study (Figures 1–3, Table 3). In fact, nitric oxide-induced peripheral vasodilation seen in anemia contributes to reduced BP,[42] but this, however, does not explain why anemics also had significantly lower lipid markers irrespective of their BP status (Figures 1–3) nor does it demonstrate how anemia seemed to displace the association of abnormal lipid markers with HTN (Table 3). Since an equilibrium between serum and red cell TC does exist,[43] diminished serum lipids are more likely a result rather than an impetus of anemia.

Along those lines, it is worth noting that diminished HDL was the only marker resistant to the protective effect of anemia as it was consistently positively associated with HTN regardless of the anemic status (Table 3). Also unique to this marker was that it was significantly improved by anemia only in hypertensive males (Figure 2), whereas TC, LDL, and TG were improved in hypertensive females (Figure 3). In fact, HTN was more prevalent in anemics with hypercholesterolemia and in non-anemics with hypertriglyceridemia (Table 2). Differential regulation of specific lipid species has been observed in previous studies. For example, TC, LDL, and HDL were significantly lowered in sickle cell disease patients compared to healthy controls, whereas TG was significantly increased and even positively correlated with the degree of hemolysis and pulmonary HTN. Similarly, elevated TG/HDL positively correlated with endothelial

**Table 3** Risk Assessment of Dyslipidemia for HTN

| Parameter | | RR | 95% CI | *P* | OR | 95% CI | *P* |
|---|---|---|---|---|---|---|---|
| High TC | | | | | | | |
| | All subjects | 1.04 | 1.0087–1.0783 | 0.0136 | 1.19 | 1.0363–1.3768 | 0.0142 |
| | Non-anemics | 1.04 | 1.0083–1.0795 | 0.0150 | 1.20 | 1.0368–1.4098 | 0.0155 |
| | Anemics | 1.15 | 0.7950–1.6877 | 0.4442 | 1.32 | 0.6608–2.6540 | 0.4285 |
| High LDL | | | | | | | |
| | All subjects | 0.98 | 0.9454–1.0266 | 0.4772 | 0.93 | 0.7850–1.1214 | 0.4873 |
| | Non-anemics | 0.98 | 0.9465–1.0317 | 0.5890 | 0.94 | 0.7784–1.1541 | 0.5939 |
| | Anemics | 1.04 | 0.6474–1.6801 | 0.8628 | 1.08 | 0.4349–2.7088 | 0.8607 |
| Low HDL | | | | | | | |
| | All subjects | 1.15 | 1.1227–1.1972 | <0.0001 | 1.99 | 1.6898–2.3451 | <0.0001 |
| | Non-anemics | 1.14 | 1.1057–1.1810 | <0.0001 | 1.92 | 1.6126–2.2891 | <0.0001 |
| | Anemics | 1.01 | 0.6962–1.4796 | 0.9386 | 1.77 | 1.0584–2.9663 | 0.0296 |
| High TG | | | | | | | |
| | All subjects | 1.13 | 1.0984–1.1751 | <0.0001 | 1.83 | 1.5315–2.2071 | <0.0001 |
| | Non-anemics | 1.12 | 1.0885–1.1658 | <0.0001 | 1.82 | 1.5003–2.2192 | <0.0001 |
| | Anemics | 1.28 | 0.9414–1.7505 | 0.1145 | 1.69 | 0.8446–3.3954 | 0.1377 |
| High TC/HDL | | | | | | | |
| | All subjects | 1.12 | 1.0845–1.1736 | <0.0001 | 1.82 | 1.4411–2.3084 | <0.0001 |
| | Non-anemics | 1.11 | 1.0705–1.1598 | <0.0001 | 1.76 | 1.3748–2.2589 | <0.0001 |
| | Anemics | 1.18 | 0.7880–1.7746 | 0.4182 | 1.42 | 0.5671–3.5855 | 0.4507 |
| High LDL/HDL | | | | | | | |
| | All subjects | 1.13 | 1.0952–1.1807 | <0.0001 | 1.67 | 1.4508–1.9308 | <0.0001 |
| | Non-anemics | 1.12 | 1.0818–1.1697 | <0.0001 | 1.64 | 1.4078–1.9229 | <0.0001 |
| | Anemics | 1.14 | 0.8268–1.5859 | 0.4148 | 1.30 | 0.6985–2.4253 | 0.4066 |
| High TG/HDL | | | | | | | |
| | All subjects | 1.20 | 1.1551–1.2473 | <0.0001 | 2.06 | 1.7920–2.3833 | <0.0001 |
| | Non-anemics | 1.19 | 1.1454–1.2409 | <0.0001 | 2.08 | 1.7867–2.4359 | <0.0001 |
| | Anemics | 1.13 | 0.8290–1.5500 | 0.4322 | 1.27 | 0.6964–2.3482 | 0.4278 |

dysfunction.[43] In light of current evidence, alterations in lipoprotein metabolism and dynamics and the potential modulatory effect of sex steroid hormones, nitric oxide, and fatty acid mobilization, in relation to BP and anemic status deserve further investigation.

Prevention of HTN relies on several factors. These include physician and patient awareness, provision of medications, enforcing dietary restrictions, and promoting physical activity, alongside surveillance programs to facilitate early intervention and management. The need for physician education regarding appropriate clinical management of HTN is highlighted in several studies. Concerns were specifically raised regarding lack of adherence to published guidelines, use of incorrect reference values, and following inappropriate therapeutic approaches.[44–46] Inordinate sodium intake and insufficient potassium in the diet are also known to contribute to the development of HTN. The Dietary Approaches to Stop Hypertension (DASH); a dietary regimen rich in whole grains, protein, and low-fat dairy foods, combined with lowered sodium, is effective for reducing BP.[47,48] Another important lifestyle modification is increased physical activity as it is consistently associated with reduced BP. In particular, improved renal and endothelial function, angiogenesis, and insulin sensitivity, along with reduced inflammation, oxidative stress, and psychosocial stress are some of the proposed mechanisms through which energy expenditure through physical activity lowers BP.[49]

Strengths of this study include the natural sampling design, large sample size, limited analytical variability due to automated data acquisition, suitability for chronic conditions such as HTN, and identification of associated risk of multiple forms of dyslipidemia in relation to HTN and anemia. Limitations include missing data regarding anthropometric variables, lifestyle habits, personal and family history of disease, and supplement and medication intake. Also,

given the cross-sectional design of the study, it was unable to derive the incidence of or the cause and effect relationship among dyslipidemia, HTN, and anemia.

## Conclusion

In summary, this report shows that TG and TG/HDL perform better than other lipid markers in discriminating hypertensive from normotensive subjects. Both parameters also resist the masking effect of anemia more strongly than other markers, further arguing for their clinical value in management of HTN and related conditions. CVD is the leading cause of death in Saudi Arabia, precipitated by high BMI, hyperglycemia, and HTN.[29] The high prevalence of risk factors has led to an earlier onset of CVD in the Saudi population[4] which necessitates stringent adherence to preventive measures.

## Abbreviations

CVD, cardiovascular disease; HTN, hypertension; TC, total cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein; TG, triglycerides; BP, blood pressure; Hgb, hemoglobin; CI, confidence intervals; RR, relative risk; OR, odds ratio; NTN, normotension.

## Data Sharing Statement

Data is available from the corresponding author upon reasonable request, and with permission of Al-Borg Medical Laboratories.

## Consent to Participate

Consent for participation was waived by the Biomedical Ethics Unit due to the retrospective nature of the study and lack of access to participant identifiers.

## Consent to Publish

Consent to publish was not required by the Biomedical Ethics Unit as no participant identifiers were used in the study.

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## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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## Disclosure

Mohammed R. Algethami serves as Vice Chairman of the Biomedical Ethics Unit at Al-Borg Medical Laboratories. The authors report no other conflicts of interest in this work.

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