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Hypertension in the Elderly: A Link Between High-density Lipoprotein Cholesterol and Renal Function

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ABSTRACT

Background: Few studies have looked into the possible link between renal function and high-density lipoprotein cholesterol (HDL-C) in elderly people with primary hypertension. As a result, the current study's goal was to assess the link between HDL-C and renal function in senior hypertension patients.

Methods: In our cross-sectional study, a total of 14,644 senior hypertension individuals were included. The patients were divided into two groups based on their serum HDL-C levels and their glomerular filtration rate (GFR) values. To compare the parameters among the groups, a one-way analysis of variance was employed. For multiple comparisons, the Bonferroni correction was used. Confounding influences were controlled using an analysis of covariance. The chi-square test for categorical data was used to examine the significance of differences between three or more groups.

Results: According to tertiles of HDL-C and tertiles of HDL-C/total cholesterol ratio (all P for trends <0.05), serum creatinine and uric acid were adversely associated to HDL-C level, but GFR was positively connected to HDL-C level in elderly hypertensive patients. The link between HDL-C and renal function was stronger in male elderly hypertension patients than in female elderly hypertensive patients. In hypertensive older people, low HDL-C was linked to renal insufficiency and proteinuria (P<0.05). When compared to the "normal renal-filtrator," the elderly "renal-hyperfiltrator" had a lower HDL-C level (P<0.05). By GFR stratum, there was an inverted "V" shape between HDL-C and GFR.

Conclusion: Low HDL-C levels are linked to poor kidney function in elderly hypertension patients, according to our findings. Glomerular hyperfiltration may also alter HDL-C levels, and sex may have a role in the relationship between HDL-C and renal function in senior hypertension patients.

INTRODUCTION

The elderly are vulnerable to aging-related dyslipidemia and renal failure. Chronic kidney disease can be accelerated by lipid disorders (CKD). [1] According to new findings, the cardiovascular risk profile of the elderly differs from that of the young. [2,3] A link between high blood cholesterol and an elevated risk of cardiovascular disease in young males and middle-aged people has been established. [4] However, prospective studies on the link of total serum cholesterol levels with cardiovascular events and kidney disease progression in the

elderly have shown mixed results. [5–7] Further research into the link between dyslipidemia and the advancement of renal disease in the elderly is required.By extracting tissue cholesterol, high-density lipoprotein cholesterol (HDL-C) promotes kidney function and the cardiovascular system. HDL-C level was connected with kidney function in general population-based studies, and the link between the two was stronger when the estimated glomerular filtration rate (GFR) fell. [8,9] However, it appears that renal function status influences HDL-C levels and function. Patients on hemodialysis with end-stage renal disease (ESRD) have defective HDL-C, which is significantly less effective in accepting cholesterol from macrophages. [10] ESRD reduced the amounts of apolipoprotein A-I (ApoA-I) and HDL-C, as well as changed the composition of HDL, according to clinical and laboratory research. [11,12]Even minor renal impairment was linked to a low HDL-C level, according to a recent study. In a recent study, we discovered that the elderly with hypertension had a greater rate of dyslipidemia and renal impairment. [13] As a result, the current study was started to see if there was a link between HDL-C and kidney function in elderly hypertensive people.

METHODS

Age >60 years and hypertension were used as inclusion criteria in this study. No data on blood pressure (BP) value, glucose concentration, cholesterol level, blood urea nitrogen (BUN) level, uric acid (UA) level, serum creatinine (Scr) level, urine analysis, body weight, or height; diabetes; obesity; immune diseases; malignant diseases; use of lipid-lowering agents; history of primary renal diseases; renal artery stenosis; and duplicate cases were among the exclusion criteria.There were 28,258 people left after the subjects with missing data and duplicate instances were removed. Hypertension was detected in 19,276 of the 28,258 participants who took part in the study. Those with diabetes, obesity, immunological disorders, malignant diseases, primary renal diseases, or renal artery stenosis were removed, and patients taking lipid-lowering medicines were excluded. Finally, 14,644 senior hypertension participants were screened and found to be eligible for the study. According to the Joint National Committee (JNC) 7 report, hypertension was defined. [14]People who had previously been diagnosed with hypertension were likewise deemed to have hypertension, even if their blood pressure had returned to normal after treatment with antihypertensive medicines. Fasting blood glucose of 7.0 mmol/L or casual blood glucose of 11.1 mmol/L were used to diagnosis diabetes. [15] Patients who had previously been diagnosed with diabetes were likewise regarded to have diabetes, even if their blood glucose levels returned to normal after therapy. Obesity was defined as a body mass index (BMI) of 30 kg/m^2 or higher. [16]

MEASUREMENTS AND RENAL FUNCTION ESTIMATES

The examination took place at an outpatient visit and included an interview to identify the patient's history of hypertension, diabetes, hyperlipidemia, medication use, and other factors. After a 10-minute rest, blood pressure was taken with a suitably sized cuff and a mercury column sphygmomanometer in the sitting posture. Height and weight were measured, and BMI was computed by dividing the body weight by the square of height. All of the subjects had their venous blood samples taken. Standard laboratory procedures were used to evaluate serum glucose, total cholesterol (TC), HDL-C, low-density lipoprotein cholesterol (LDL-C), triglyceride (TG), BUN, Scr, and UA levels.The sulfosalicylic acid method was used to determine the qualitative analysis of urine protein. The simplified modification of diet in renal disease (MDRD)[17] equations and the chronic kidney disease epidemiology collaboration were used to calculate GFR (CKD-EPI). [18]TheGFR_{MDRD}wascalculatedas 186 $Scr^{-1.154}\times Age^{-0.203}$ (×0.742 iffemale). The $GFR_{CKD\text{-}EPI}$ was calculated as recommended: for women with a Scr ≤0.7, $144 \times (Scr/0.7)^{-0.329} \times 0.993^{age}$; for women with an Scr>0.7, 144 ×

$(Scr/0.7)^{-1.209} \times 0.993^{age}$; for men with a Scr≤0.9, $141 \times (Scr/0.9)^{-0.411} \times 0.993^{age}$; for men with a Scr>0.9, $141 \times (Scr/0.9)^{-1.209} \times 0.993^{age}$.

PROTOCOL

The 14,644 senior hypertension participants were split into three groups based on their serum HDL-C and HDL-C/TC ratios. The patients were then divided into six groups based on their GFR_{MDRD} values and five groups based on their $GFR_{CKD-EPI}$ values. The prevalence of renal insufficiency and proteinuria, as well as BMI, BP, TC, TG, HDL-C, LDL-C, glucose concentration, GFR, Scr, UA, and BUN, were also compared between groups.

STATISTICAL ANALYSIS

Unless otherwise noted, continuous data were given as mean standard deviation (SD) and categorical variables as a percentage. The difference in means of continuous variables among three or more groups was compared using a one-way analysis of variance. For multiple comparisons, the Bonferroni correction was used. Confounding factors were controlled using the analysis of covariance (ANCOVA). The chi-square test for categorical data was used to examine the significance of differences between three or more groups. Statistical significance was defined as a P value of less than 0.05. The analysis was carried out using the statistical software package SPSS16.0 for Windows (SPSS Inc, Chicago, IL).

RESULTS

SUMMARY OF STUDY SUBJECTS

Table 1 shows the clinical and analytical characteristics of study participants. A total of 14,644 participants, 67 percent of whom were male and aged 70±6 years, were studied. Among the hypertensive elderly, isolated systolic hypertension predominated. The ratio of HDL-C to TC was 0.3±0.1. The MDRD formula estimated GFR was higher than the CKD-EPI equation calculated GFR ($P<0.01$). GFR_{MDRD} and $GFR_{CKD-EPI}$ had a good correlation ($r=0.92$, $P<0.01$).

RENAL FUNCTION ACCORDING TO TERTILES OF HDL-C

Table 2 shows the parameters of three groups based on the tertiles of serum HDL-C levels. The individuals in tertiles 1 and 2 were younger than those in tertile 3. Females had a higher HDL-C level than males, who had a lower amount. TC levels increased when HDL-C levels grew from tertile 1 to tertile 3, although TG and BMI levels declined. Tertile 2 had the highest LDL-C level, while tertile 1 had the lowest. Tertile 1 exhibited a higher glucose level than tertile 2 and tertile 3. The three groups did not differ in terms of systolic blood pressure (SBP), diastolic blood pressure (DBP), or pulse pressure (PP).Figure 1 shows the GFR, Scr, and UA values for three groups. Scr and UA levels were highest in tertile 1 and lowest in tertile 3, respectively, whereas GFR_{MDRD} and $GFR_{CKD-EPI}$ levels were lowest in tertile 1 and highest in tertile 3. The BUN levels in the three groups were identical ($P<0.06$, data not shown). The prevalence of renal insufficiency and proteinuria

TABLE 1. Clinical and Laboratory Characteristics of Subjects

Variables	Values
N	14644
Age, y	70 ± 6
Male, %	66.7%
BMI, kg/m^2	24.2 ± 2.8
SBP, mmHg	148 ± 18
DBP, mmHg	81 ± 12
PP, mmHg	67 ± 16
TC, mmol/L	5 ± 0.9
TG, mmol/L	1.6 ± 1
HDL-C, mmol/L	1.3 ± 0.3
LDL-C, mmol/L	3 ± 0.8
Glucose, mmol/L	5.1 ± 0.7
BUN, mmol/L	5.4 ± 1.6
Scr, μmol/L	77.8 ± 27.7
UA, μmol/L	329.3 ± 88.4
$GFR_{CKD-EPI}$, mL/min/1.73m^2	81.7 ± 14.2
GFR_{MDRD}, mL/min/1.73m^2	88.4 ± 19.7
Renal insufficiency, %	6.1%
Proteinuria, %	14.2%
Hyperlipidemia, %	41.6%

BMI = body mass index, BUN = blood urea nitrogen, CKD-EPI = chronic kidney disease epidemiology collaboration, DBP = diastolic blood pressure, GFR = glomerular filtration rate, HDL-C = high-density lipoprotein cholesterol, LDL-C = low-density lipoprotein cholesterol, MDRD = simplified modification of diet in renal disease, PP = pulse pressure, SBP = systolic blood pressure, Scr = serum creatinine, SD = standard deviation, TC = total cholesterol, TG = triglyceride, UA = uric acid.

prevalence was lower in tertile 2 and tertile 3, compared with that in tertile 1 (P<0.05, Figures 2A and 3A). We further divided tertiles of HDL-C into female and male groups because sex was a role in lipid and renal function. To investigate the influence of HDL-C on kidney function, we used ANCOVA with age, BMI, TC, TG, LDL-C, and glucose concentration as confounding factors (data shown in Table 3). Scr and UA levels reduced when HDL-C levels climbed from tertile 1 to tertile 3, but GFR increased in male individuals. For female participants, there was no difference in BUN, Scr, or GFR between tertiles of HDL-C.

RENAL FUNCTION ACCORDING TO TERTILES OF HDL-C/TC RATIO

Figure 4 shows the GFR, Scr, and UA values for three groups based on the HDL-C/TC ratio. GFR_{MDRD} rose and UA level decreased when the HDL-C/TC ratio grew from tertile 1 to tertile3. However, tertile 2 exhibited the greatest $GFR_{CKD-EPI}$ and the lowest Scr. Between the three groups, there was no significant difference in BUN levels (P=0.052, data not shown). When compared to tertile 1, the prevalence of renal insufficiency and proteinuria was reduced in tertile 2 and tertile 3 (P<0.05, Figures 2B and 3B).Table 4 shows the BUN, Scr, UA, and GFR values of male and female participants from tertile 1 to tertile 3. Scr and UA levels reduced as the HDL-C/TC ratio grew from tertile 1 to tertile 3, but GFR increased in male individuals. The UA level in female patients fell gradually from tertile 1 to tertile 3, whereas Scr, GFR, and BUN values did not differ between tertiles.

HDL-C LEVELS IN DIFFERENT GFR STRATA

According to MDRD formula, GFR increased from sextile 1 to sextile 5 (all P for the trends < 0.05, Figure 5) and then eGFR decreased from sextile 5 to sextile 6 (P < 0.05, Figure 5). The $GFR_{CKD-EPI}$ increased from quintile 1 to quintile 4 (all P for the trends <0.05, Figure 5), and then $GFR_{CKD-EPI}$ decreased from quintile 4 to quintile 5 (P < 0.05, Figure 5). The inverse ‘‘V’’

shape was observed between GFR and HDL-C (Figure 5).Either the MDRD formula or CKD-EPI equation was used to calculate GFR.

DISCUSSION

There are few studies to date that are specifically directed at determining the relationship between blood HDL-C levels and renal function in elderly hypertension patients. In geriatric hypertension, our population-based investigation revealed a complicated link between HDL-C and kidney function. Our findings corroborate several findings from earlier research. HDL-C was found to be linked to renal function in general population-based studies, and the link grew stronger as GFR decreased. [8]

TABLE 2. Parameters Among 3 Groups According to High-density Lipoprotein Cholesterol Level

Parameters	Tertile 1	Tertile 2	Tertile 3	*P*
N	2241	11783	620	
Age, y	69.7 ± 6	69.8 ± 6.2	71 ± 6.7*,#	<0.01
Male, %	82.4%	64.8%*	47.6%*,#	<0.01
BMI, kg/m^2	25.2 ± 2.4	24.1 ± 2.7*	21.6 ± 3*,#	<0.01
SBP, mmHg	148 ± 18	148 ± 18	147 ± 19	0.37
DBP, mmHg	81 ± 12	81 ± 12	80 ± 13	0.07
PP, mmHg	67 ± 16	67 ± 16	68 ± 17	0.72
TC, mmol/L	4.6 ± 0.9	5.1 ± 0.9*	5.6 ± 1*,#	<0.01
TG, mmol/L	2.3 ± 1.7	1.5 ± 0.8*	1 ± 0.5*,#	<0.01
HDL-C, mmol/L	0.9 ± 0.1	1.4 ± 0.2*	2.2 ± 0.2*,#	<0.01
LDL-C, mmol/L	2.7 ± 0.8	3 ± 0.8*	2.9 ± 0.9*,#	<0.01
Glucose, mmol/L	5.2 ± 0.7	5.1 ± 0.6*	5.1 ± 0.6*	<0.01

The HDL-C ranges in tertile 1, tertile 2, and tertile 3 were <1, 1–2, >2 mmol/L, respectively. Compared with tertile 1. BMI = body mass index, DBP = diastolic blood pressure, HDL-C = high-density lipoprotein cholesterol, LDL-C = low-density lipoprotein cholesterol, PP = pulse pressure, SBP = systolic blood pressure, TC = total cholesterol, TG = triglyceride.

* $P < 0.05$; Compared with tertile 2.

# $P < 0.05$.

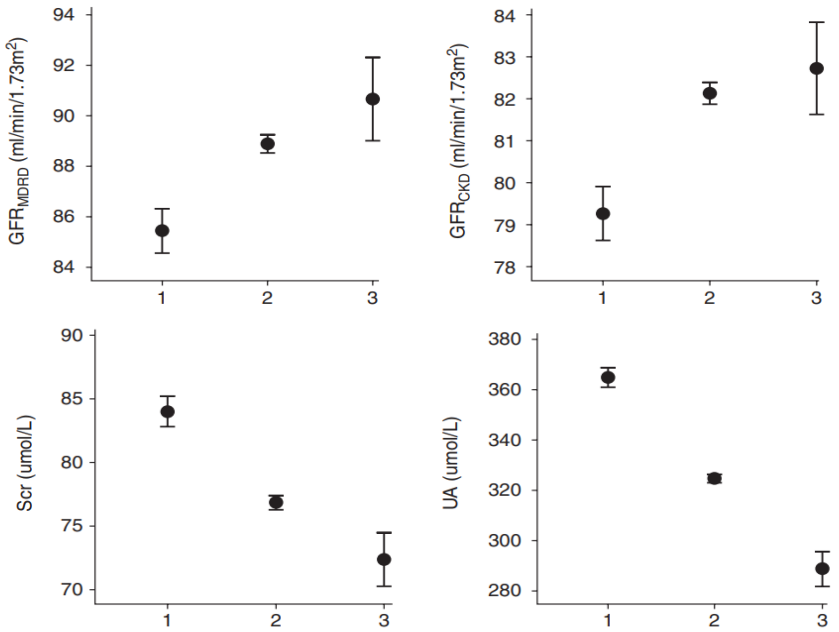

FIGURE 1. GFR$_{MDRD}$, GFR$_{CKD-EPI}$, Scr, and UA levels according to tertiles of HDL-C. Range 1, 2, and 3 of HDL-C in increasing tertiles were <1, 1–2, >2 mmol/L, respectively. The vertical bars represent 95% confidence interval for mean. All *P* for trends <0.05. CKD-EPI = chronic kidney disease epidemiology collaboration, GFR = glomerular filtration rate, HDL-C = high-density lipoprotein cholesterol, MDRD = simplified modification of diet in renal disease, Scr = Serum creatinine, UA = uric acid. n = 2241 for tertile 1, 11783 for tertile 2, and 620 for tertile 3.

Low HDL-C levels were associated with early dialysis or doubling of the Scr level, according to longitudinal follow-up research, and were the only lipid parameter that impacted the course

of CKD irrespective of diabetes. [19] In CKD patients with low HDL-C levels, serum cholesterol efflux mediated by scavenger receptor class B member 1 (SR-B1) was dramatically reduced, and HDL functioning was similarly affected. [19] According to several research, renal dysfunction is related to a drop in HDL-C or ApoA-I levels, which may be linked to the downregulation of ApoA-I synthesis in the liver. [20–24]According to the tertiles of HDL-C, Scr and UA are adversely associated with HDL-C levels, whereas GFR is positively related to HDL-C levels in senior hypertension patients.

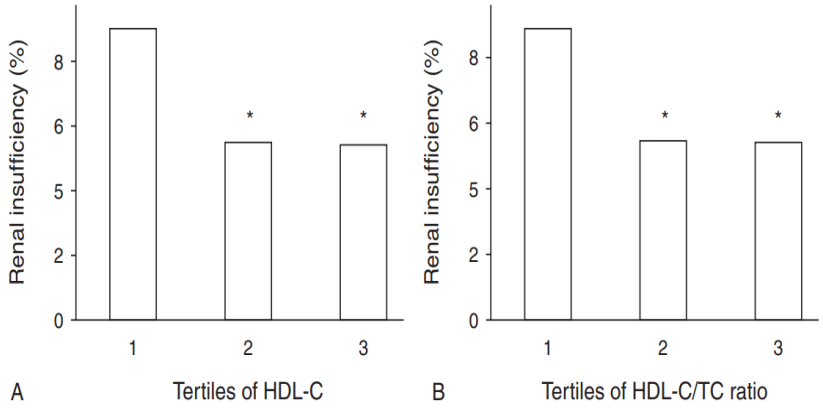

FIGURE 2. (A) Prevalence of renal insufficiency according to tertiles of HDL-C. Range 1, 2 and 3 of HDL-C in increasing tertiles of HDL-C were <1, 1–2, >2 mmol/L, respectively. (B) Prevalence of renal insufficiency according to tertiles of HDL-C/TC Ratio. Range 1, 2, and 3 of HDL-C/TC ratio in increasing tertiles of HDL-C/TC ratio were <0.2, 0.2–0.4, >0.4, respectively. Renal insufficiency was defined as GFR <60 mL/min/1.73m^2 by MDRD equation. $^*P<0.05$ vs tertile 1. HDL-C = high-density lipoprotein cholesterol, MDRD = simplified modification of diet in renal disease, TC = total cholesterol. n = 2241 for tertile 1, 11783 for tertile 2 and 620 for tertile 3 according to HDL-C level, n = 2458 for tertile 1, 11418 for tertile 2, and 768 for tertile 3 according to HDL-C/TC Ratio.

The connection between HDL-C and renal function is higher in male elderly hypertension patients than in female elderly hypertensive patients. In the aged, low HDL-C is linked to renal insufficiency and proteinuria. In the current study, however, older patients with excessively high GFR (GFR_{MDRD}>140 mL/min/1.73m^2 or $GFR_{CKD\text{-}EPI}$>120 mL/min/1.73m^2) had significantly lower HDL-C values by both GFR_{MDRD} and $GFR_{CKD\text{-}EPI}$ stratum.Our findings also reveal that in the elderly hypertensive population, there is an inverse "V" association between GFR and HDL-C based on GFR strata. It demonstrates that glomerular hyperfiltration, which has been hypothesised in young healthy males, may lower HDL-C levels in the elderly. [25]
Glomerular hyperfiltration occurs in a variety of clinical situations, including hypertension and kidney disease. There is no precise definition of glomerular hyperfiltration as of yet. According to a recent analysis, 88.4 percent of glomerular hyperfiltration threshold studies employed a single threshold, whereas 11.6 percent used multiple thresholds to define glomerular hyperfiltration. [26] The threshold for glomerular hyperfiltration varied from 90.7 to 175 mL/min/1.73 m^2. The pathophysiology of glomerular hyperfiltration, which varies depending on the underlying illnesses, has not been well investigated. Renal damage can be both a cause and a result of glomerular hyperfiltration.Obesity and metabolic syndrome have both been linked to renal hyperfiltration. [25,27–30] In a broad sample of people aged 40 to 72, the "hyperfiltrators" were really at an increased risk of cardiovascular disease. Regardless of whether GFR was calculated using the CKD-EPI method or the MDRD equation, glomerular hyperfiltration predicted the combined outcomes of overall death and cardiovascular events at a 3-fold relative risk. [31] In comparison to the "non-hyperfiltrators," the "hyperfiltrators" had a greater plasma atherogenic index (TG/HDL-C ratio), but lower HDL-C and UA levels. [31]Sextile 6 (GFR_{MDRD}>140 mL/min/1.73m2) and quintile 5 ($GFR_{CKD\text{-}EPI}$>120 mL/min/1.73m2) showed higher GFR values in this study, although their HDL-C levels declined considerably.

TABLE 3. Kidney Function Among Tertiles of High-density Lipoprotein Cholesterol in Male and Female Subjects

Variables		Tertile 1	Tertile 2	Tertile 3	*P*
BUN, mmol/L	Male	5.5 ± 1.6	5.5 ± 1.6	5.8 ± 1.8*,†	0.03
	Female	5 ± 1.5	5.1 ± 1.5	5.2 ± 1.3	0.81
Scr, μmol/L	Male	87.8 ± 29.5	83.9 ± 26.6*	80.9 ± 20.4*	0.03
	Female	65.2 ± 18.7	63.9 ± 23.3	64.5 ± 28.6	0.63
UA, μmol/L	Male	375.8 ± 85.4	348.4 ± 82.8*	331.8 ± 82.9*,†	0.00
	Female	313.5 ± 77.5	280.9 ± 76*	249.6 ± 72.1*,†	0.01
$GFR_{CKD-EPI}$, mL/min/1.73m^2	Male	78.7 ± 15.6	80.9 ± 13.8*	82 ± 14*	0.00
	Female	82.9 ± 15.1	84.3 ± 13.6	83.3 ± 13.8	0.16
GFR_{MDRD}, mL/min/1.73m^2	Male	85.1 ± 20.6	88.2 ± 19.2*	91.6 ± 21.8*,†	0.01
	Female	88 ± 21.2	90.2 ± 19.7	89.8 ± 20	0.32

Statistical significances were obtained by analysis of covariance. The covariates included age, BMI, TC, TG, LDL-C, and glucose concentration. The HDL-C ranges in tertile 1, tertile 2, and tertile 3 were <1, 1–2, >2 mmol/L, respectively. Compared with tertile 1. BMI = body mass index, BUN = blood urea nitrogen, CKD-EPI = chronic kidney disease epidemiology collaboration, GFR = glomerular filtration rate, HDL-C = high-density lipoprotein cholesterol, LDL-C = low-density lipoprotein cholesterol, MDRD = simplified modification of diet in renal disease, Scr = serum creatinine, TC = total cholesterol, TG = triglyceride, UA = uric acid.

* $P < 0.05$; compared with tertile 2.

† $P < 0.05$.

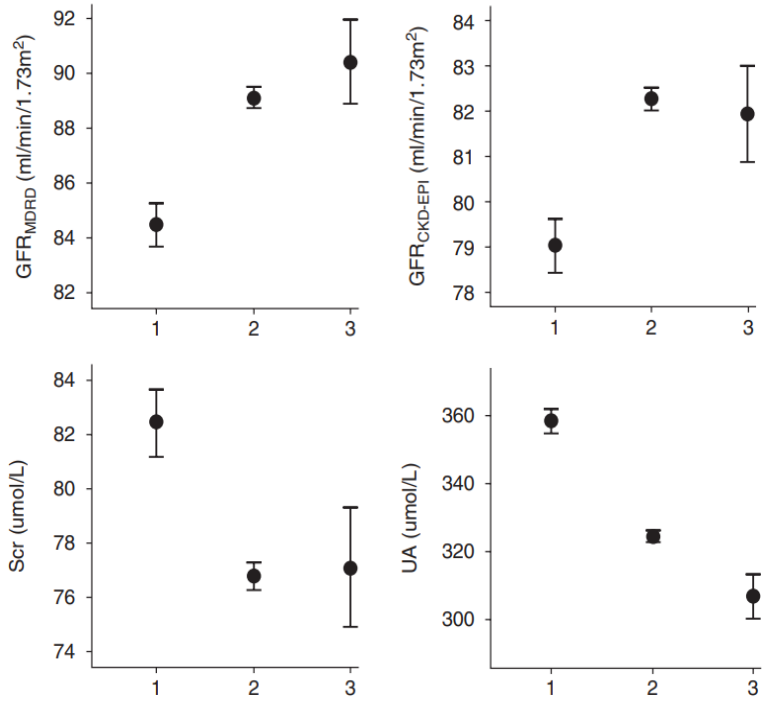

FIGURE 4. GFR_{MDRD}, $GFR_{CKD-EPI}$, Scr, and UA levels according to tertiles of HDL-C/TC Ratio. Range 1, 2, and 3 of HDL-C/TC ratio in increasing tertiles were <0.2, 0.2–0.4, >0.4, respectively. The vertical bars represent 95% confidence interval for mean. All *P* for trends <0.05. CKD-EPI = chronic kidney disease epidemiology collaboration, GFR = glomerular filtration rate, HDL-C = high-density lipoprotein cholesterol, MDRD = simplified modification of diet in renal disease, Scr = Serum creatinine, TC = total cholesterol, UA = uric acid. n = 2458 for tertile 1, 11418 for tertile 2, and 768 for tertile 3.

Hypertensive patients are known to have a high blood volume and increased vasoconstriction, both of which lead to an increase in glomerular pressure. [32] Increased glomerular pressure may signal the onset of hypertension and contribute directly to the progression of CKD. [33] Glomerular hypertension has been shown in experiments to facilitate progressive kidney injury after a variety of starting lesions. [34–36]However, the pathophysiological mechanism of low HDL-C related with glomerular hyperfiltration is yet unknown, despite several studies finding lower HDL-C levels in "hyperfiltrators." The involved mechanism remains to be elucidated in future studies to develop new therapeutic strategies.

The recent research has a number of advantages. Because there are more elderly hypertensive people in the study, the results are more dependable and repeatable. The MDRD equation was

created to estimate renal function in people with a GFR of more than 60 mL/min/1.73m^2.

TABLE 4. Kidney Function Among Tertiles of High-density Lipoprotein Cholesterol to Total Cholesterol Ratio in Male and Female Subjects

Variables		Tertile 1	Tertile 2	Tertile 3	*P*
BUN, mmol/L	Male	5.5 ± 1.6	5.5 ± 1.6	5.7 ± 1.9	0.08
	Female	5.1 ± 1.5	5.1 ± 1.4	5.1 ± 1.6	0.80
Scr, μmol/L	Male	88.1 ± 24.6	83.8 ± 27.6*	83 ± 26*	0.00
	Female	67.6 ± 18.8	63.5 ± 18.2	64.6 ± 22.6	0.10
UA, μmol/L	Male	378.3 ± 84	348.5 ± 82.5*	332.6 ± 90.2*,†	0.00
	Female	306.1 ± 79.6	279.1 ± 75.4*	252.3 ± 75.7*,†	0.00
$GFR_{CKD-EPI}$, mL/min/1.73m^2	Male	78 ± 14.9	81.1 ± 13.9*	81.5 ± 14.9*	0.00
	Female	81.7 ± 14.8	84.4 ± 13.5	84.3 ± 14.5	0.10
GFR_{MDRD}, mL/min/1.73m^2	Male	83.8 ± 19.3	88.5 ± 19.4*	90 ± 21.8*	0.00
	Female	86.3 ± 20.3	90.4 ± 19.7	91.8 ± 21	0.47

Statistical significances were obtained by analysis of covariance (ANCOVA). The covariates included age, BMI, TG, LDL-C, and glucose concentration. The HDL-C ranges in tertile 1, tertile 2 and tertile 3 were <0.2, 0.2–0.4, >0.4, respectively. Compared with tertile 1. BMI = body mass index, BUN = blood urea nitrogen, CKD-EPI = chronic kidney disease epidemiology collaboration, GFR = glomerular filtration rate, HDL-C = high-density lipoprotein cholesterol, LDL-C = low-density lipoprotein cholesterol, MDRD = simplified modification of diet in renal disease, Scr = serum creatinine, TG = triglyceride, UA = uric acid.

* $P < 0.05$; compared with tertile 2.

† $P < 0.05$.

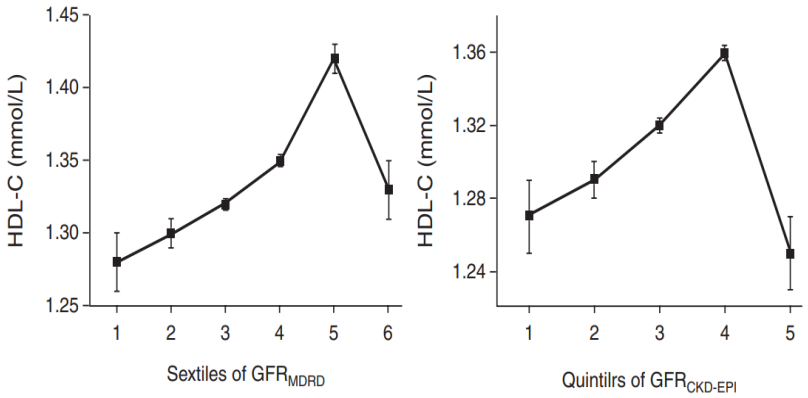

FIGURE 5. Inverse "V" shape between GFR and HDL-C. Range 1, 2, 3, 4, 5, and 6 of GFR_{MDRD} in increasing sextiles of GFR_{MDRD} were <30, 30–60, 60–90, 90–120, 120–140, >140 mL/min/1.73m^2, respectively. Range 1, 2, 3, 4, and 5 of $GFR_{CKD-EPI}$ in increasing quintiles of $GFR_{CKD-EPI}$ were <30, 30–60, 60–90, 90–120, >120 mL/min/1.73m^2, respectively. All *P* for trends <0.05. CKD-EPI = chronic kidney disease epidemiology collaboration, GFR = glomerular filtration rate, HDL-C = high-density lipoprotein cholesterol, MDRD = simplified modification of diet in renal disease. n = 79 for sextile 1, 807 for sextile 2, 7166 for sextile 3, 5831 for sextile 4, 602 for sextile 5, and 159 for sextile 6 according to GFR_{MDRD}, n = 90 for quintile1, 1073 for quintile 2, 8763 for quintile 3, 4629 for quintile 4, and 89 for quintile 5 according to $GFR_{CKD-EPI}$.

In comparison to the MDRD equation, the CKD-EPI formula was designed to assess a higher GFR. [17,18] We employed both of them to estimate GFR in our study because of their accuracy in varied GFR ranges.Diabetes and obesity have been found to have an impact on renal function. As a result, participants with diabetes and obesity were excluded from this study. We also omitted participants who took lipid-lowering drugs to reduce the number of confounding factors.

There are certain limitations to our research. The study's main weakness is that cross-sectional data does not allow us to track the effects of risk factors over time and discern between cause and effect. Another problem is that we don't know how long high blood pressure and low HDL-C levels last. The older participants are more likely to have a longer history of risk factors, which could affect the relationship between HDL-C and renal function. Furthermore, in the current investigation, information on some chronic conditions, such as recent infection and chronic inflammatory diseases, was not enquired about or documented.Confounding factors such as recent infection and chronic inflammatory illnesses can affect HDL-C levels and renal function. In the future, chronic disease conditions should be excluded from further research. Finally, rather than using directly measured GFR, we used eGFR in our study. However, in

clinical practice, direct GFR measurement is rarely employed. Given these limitations, further longitudinal cohort studies are needed to investigate and analyse the causative link between HDL-C and renal function in the elderly with hypertension.

CONCLUSION

In conclusion, our findings confirm the hypothesis that a low HDL-C level is linked to renal function deterioration in senior hypertension patients. The relationship between HDL-C and GFR may be influenced by gender. Glomerular hyperfiltration (GFR_{MDRD}>140 mL/min/1.73m^2 or $GFR_{CKD\text{-}EPI}$>120 mL/min/1.73m^2) can influence HDL-C levels in the blood. Based on GFR stratification in senior hypertension, GFR and HDL-C have an inverse "V" shape.

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