

# Association with Non-HDL Cholesterol and the Chronic Kidney Disease

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## Abstract

### Background

Chronic Kidney Disease (CKD) has been reported to be a risk factor for cardiovascular diseases as well as for dialysis. In effect, it has been reaffirmed that the presence of CKD causes the prognosis of patients of cardiovascular disease (CVD) to deteriorate. However, no studies on the association between CKD and non-HDL cholesterol (non-HDL) have been reported to the best of our knowledge. Therefore, in this report we investigated the relationship between non-HDL and the development and progress of CKD.

### Methods

The subjects were 8,677 inhabitants who underwent comprehensive medical examinations at the Kasugai City Medical Center between April 2006 and March 2008. The odds ratio to CKD of age, sex (males), hyperglycemia, hypertension, obese, and dyslipidemia was calculated as a cross-sectional study.

Non-HDL-cholesterol (non-HDL) was calculated by subtracting HDL-C from TC. The quartile for TC, TG and non-HDL was calculated for 7,725. Moreover, respectively the odds ratio to CKD of each groups were compared on the basis of the first group for quartile of TC, TG and non-HDL.

Furthermore, for 1,584 subjects who the medical checkups was received in this center also five years ago, and were not dyslipidemia at the time, it divided into that in which non-HDL was quartile group for five years afterward, and compared in the amount of reduction of each eGFR.

### Results

Significantly high odds ratios were found between CKD and each factor: 1.07 (95% confidence interval: 1.06-1.08) for age, 1.82 (1.62-2.04) for sex (male), 1.24 (1.07-1.43) for hyperglycemia, 1.23 (1.08-1.40) for hypertension, 1.49 (1.30-1.71) for obesity and 1.42 (1.24-1.63) for dyslipidemia.

TC, TG and non-HDL was set to Group 1 to Group 4 by dividing in quartiles, respectively.

As a result, odds ratios of non-HDL were 1.21 (1.01-1.45) in Group 2 ( $116 \leq \text{non-HDL} < 137$ ), 1.34 (1.12-1.60) in Group 3, and 1.61 (1.35-1.92) in Group 4, all being significant values with strong linearity. (p for trend <0.01)

Amount of reduction of eGFR over five years after adjusting for sex and age in each quartiles of non-HDL demonstrated significant differences between Group 1 and Groups 2 to 4. ( $p<0.01$ )

## Conclusion

Our study suggests that non-HDL is involved in the development and progression of CKD because elevated non-HDL is a CKD risk factor and rises in non-HDL lead to decreased eGFR and increased incidences of CKD.

## 1. Introduction

Chronic Kidney Disease (CKD), which was proposed by the National Kidney Foundation, has been garnering attention in Japan and preventative measures are starting to be taken against it as research regarding the disease progresses<sup>1)</sup>. CKD is diagnosable if either of the following continue for over three months: findings that suggest nephropathy (abnormal urinalysis, abnormal imaging, pathological findings) or estimated glomerular filtration rate (eGFR) of less than 60ml/min/1.73m<sup>2</sup> (unit abbreviated hereafter) for Japanese patients<sup>1)</sup>. As the number of patients with terminal renal failure increases worldwide and CKD emerges as a serious health-threatening risk factor, the remission and regression of CKD via early detection and treatment has been made possible<sup>1,2)</sup>.

With the highest dialysis patient density per unit of population, Japan is a dialysis superpower. In 2010, the number of dialysis patients was 297,000 and a vast number of people at risk for CKD are thought to exist<sup>3)</sup>. The Japanese Society of Nephrology initiated a CKD Committee in 2004 that aims to reduce the number of dialysis patients by furthering awareness of the concept of CKD and conducting preventive measures. The number of Japanese CKD patients inferred epidemiologically from data taken during health checkups based on a GFR prediction equation modified for Japanese patients is approximately 13,300,000<sup>3)</sup>. This has led to CKD being called our “new national affliction”.

Furthermore, CKD is also strongly related to lifestyle diseases such as arteriosclerosis caused by aging and hypertension<sup>4-6)</sup> and it has been reported to be a risk factor for cardiovascular diseases as well as for dialysis<sup>7-11)</sup>. Numerous epidemiological surveys have shown impaired renal function to be involved in an increase in cardiovascular events and the concept of a heart-kidney relationship has led CKD to be recognized as a new risk factor for cardiovascular disease (CVD)<sup>7-11)</sup>. In effect, it has been reaffirmed that the presence of CKD causes the prognosis of patients of CVD to deteriorate.

Although conventional markers of arteriosclerotic diseases include LDL cholesterol (LDL) and triglycerides (TG), discrepancy has been observed between the kits concerning direct methods for LDL as a philosophical issue<sup>12)</sup>, and TG is difficult to manage as it is susceptible to the diet. Therefore in recent years, non-HDL-cholesterol, calculated by deducting HDL-cholesterol (HDL) from total cholesterol (TC), is attracting attention as a new indicator of coronary artery disease<sup>13, 14)</sup>. Non-HDL is regarded as an indicator capable of comprehensive evaluation of arteriosclerosis provocative lipoproteins such as LDL, IDL, VLDL as well as remnant and small dense LDL since

the influence of HDL with anti-atherogenic action is eliminated. Furthermore, Cui et al.<sup>15)</sup> has reported non-HDL to be a stronger predictive factor of CVD compared to LDL.

However, no studies on the association between CKD and non-HDL have been reported to the best of our knowledge. Therefore, in this report we investigated the relationship between non-HDL cholesterol (non-HDL) and CKD. Non-HDL is attracting attention as a new indicator for coronary artery disease and non-HDL is calculated by deducting HDL-cholesterol (HDL-C) from TC.

## 2. Methods

### 2.1. Subjects and Analyses methods

First, the subjects were 8,677 (4,383 males and 4,294 females) who underwent comprehensive medical examinations at the Kasugai City Medical Center between April 2006 and March 2008. The odds ratio to CKD of age, sex (males), hyperglycemia (fasting plasma glucose  $\geq 126$ mg/dl or hemoglobin A1c  $\geq 6.1\%$ ), hypertension (systolic blood pressure  $\geq 140$ mm/Hg, diastolic blood pressure  $\geq 90$ mm/Hg), obese (body mass index  $\geq 25$ ), and dyslipidemia (triglyceride  $\geq 150$ mg/dl or HDL-cholesterol  $\leq 40$ mg/dl or Total-cholesterol  $\geq 220$ mg/dl) was calculated as a cross-sectional.

Second, non-HDL-cholesterol (non-HDL) was calculated by subtract HDL-C from TC. The quartile for TC, TG and non-HDL was calculated for 7,725 (3,983 men, 3,742 women) excluded what is treating dyslipidemia. Moreover, respectively the odds ratio to CKD of each groups were compared on the basis of the first group for quartile of TC, TG and non-HDL.

Furthermore, for 1,584 subjects (964 men, 620 women) who the medical checkups was received in this center also five years ago, and were not dyslipidemia at the time, it divided into that in which non-HDL was quartile group for five years afterward, and compared in the amount of reduction of each eGFR.

### 2.2. CKD diagnostic criteria

It was diagnosed as CKD that the protein urine positivity or eGFR was less than 60ml/min/1.73m<sup>2</sup>. At this time, GFR was estimated by using the following Japanese formula<sup>16)</sup>.

$$\text{GFR (ml/min/1.73m}^2\text{)} = 194 \times \text{serum creatinine}^{-1.094} \text{ (mg/dl)} \times \text{age}^{-0.287} (\times 0.739 \text{ for women})$$

### 2.3. Laboratory measurements

Plasma high-density lipoprotein cholesterol (HDL-C), total lipoprotein cholesterol (TC), triglyceride (TG) and creatinine (CRE) were measured by the direct enzymatic method. All venous blood samples were obtained in the morning from subjects after a fasting period of 12 h. Their concentrations were measured using an automated analyzer (model 7180; Hitachi, Japan).

Albuminuria was detected by using urine test paper "Pre-test (Multi II)" and conducting automatic judgment using automatic urine analyzer "Pre-tester (RM6050)" made by Wako Pure Chemical Industries, Ltd.

### 2.4. Statistical analyses

Statistical analyses were performed using the SAS system for Windows (release 9.13; SAS Institute, Cary, NC). Differences between two groups were evaluated by Student t-test. All statistical tests were two-sided, and a P value <0.05 was considered to be significant.

### 3. Results

#### 3.1. The characteristics and the odds ratio of cause of disease by the all subjects

Table 1 shows the characteristics of the all subjects. The characteristic features (mean value  $\pm$  standard deviation) were as follows: the age of males were  $61.6 \pm 11.7$  years while that of females were  $59.8 \pm 10.6$  years; the body mass index (BMI) values of males were  $23.1 \pm 2.9$  while those of females were  $21.9 \pm 3.0 \text{ kg/cm}^2$ ; the non-HDL values of males were  $137.3 \pm 31.8$  while those of females were  $138.8 \pm 33.8 \text{ mg/dl}$ ; the creatine values of males were  $0.89 \pm 0.26$  while those of females were  $0.64 \pm 0.14 \text{ mg/dl}$ ; the eGFR values of males were  $71.2 \pm 14.5$  while those of females were  $74.6 \pm 14.4 \text{ mL/min/1.73m}^2$ .

Table 1. The characteristics of subjects

Characteristics	Males (n = 4,383)	Females (n = 4,294)
Age (years)	61.6 $\pm$ 11.7	59.8 $\pm$ 10.6
Body Mass Index ( $\text{kg/m}^2$ )	23.1 $\pm$ 2.9	21.9 $\pm$ 3.0
Abdomen circumference (cm)	83.2 $\pm$ 8.0	80.4 $\pm$ 8.9
Systolic blood pressure (mmHg)	124.7 $\pm$ 16.5	122.0 $\pm$ 17.6
Diastolic blood pressure (mmHg)	74.0 $\pm$ 9.5	71.3 $\pm$ 9.6
Total-cholesterol (mg/dl)	197.8 $\pm$ 31.0	210.1 $\pm$ 32.1
HDL-cholesterol (mg/dl)	60.1 $\pm$ 15.6	71.2 $\pm$ 16.6
Non-HDL cholesterol (mg/dl)	137.8 $\pm$ 31.7	138.9 $\pm$ 33.2
Triglyceride(mg/dl)	118.6 $\pm$ 74.2	95.1 $\pm$ 50.7
Fasting plasma glucose (mg/dl)	99.0 $\pm$ 20.6	92.5 $\pm$ 15.3
Hemoglobin A1c(%)	5.5 $\pm$ 0.7	5.4 $\pm$ 0.5
Creatinine (mg/dl)	0.89 $\pm$ 0.26	0.64 $\pm$ 0.14
eGFR( $\text{mL/min/1.73m}^2$ )	71.2 $\pm$ 14.5	74.6 $\pm$ 14.4
mean $\pm$ SD		

Table 2 shows the odds ratio of cause of disease by the all subjects. Significantly high odds ratios were found between CKD and each factor: 1.07 (95% confidence interval: 1.06-1.08) for age, 1.82 (1.62-2.04) for sex (male), 1.24 (1.07-1.43) for hyperglycemia, 1.23 (1.08-1.40) for hypertension, 1.49 (1.30-1.71) for obesity and 1.42 (1.24-1.63) for dyslipidemia.

Table 2. The odds ratio to CKD of multiple analyses

Risk factors	Odds ratio	95%CI		P-value
Age	1.07	1.06	- 1.07	<0.001
Male	1.82	1.62	- 2.04	<0.001
Dyslipidemia	1.42	1.24	- 1.63	<0.001
Obesity	1.49	1.30	- 1.71	<0.001
Hyperglycemia	1.24	1.07	- 1.43	0.0034
Hypertension	1.23	1.08	- 1.40	0.0021

### 3.2. Epidemiological features of CKD and odds ratio for lipid items in data excluding cases under treatment for dyslipidemia

Table 3 shows the epidemiological features depending on the presence of CKD. Group with CKD was significantly high in all items except HDL-C and significantly low in HDL-C compared to the group without CKD in both male and female subjects.

Table 3. Clinical characteristics by CKD

variable	Males (3,983)				Females (3,742)			
	normal (3,024)		CKD (959)		normal (3,287)		CKD (455)	
	Average	SD	Average	SD	Average	SD	Average	SD
Age	59.5 ±	12.20	66.9 ±	8.84 §	58.2 ±	10.80	64.5 ±	9.25 §
Body Mass Index	22.8 ±	2.82	23.5 ±	2.97 §	21.7 ±	3.02	22.2 ±	3.14 §
Abdomen circumference	82.5 ±	7.80	84.5 ±	8.10 §	79.7 ±	8.86	81.4 ±	8.81 §
Systolic blood pressure	122.8 ±	15.77	129.4 ±	17.54 §	120.6 ±	17.28	125.1 ±	18.91 §
Diastolic blood pressure	73.5 ±	9.33	75.5 ±	9.86 §	70.9 ±	9.46	72.5 ±	10.44 §
Total-cholesterol	196.7 ±	30.64	200.5 ±	32.49 *	209.6 ±	32.73	214.3 ±	33.08 §
HDL-cholesterol	60.9 ±	15.82	58.4 ±	15.54 §	71.7 ±	16.54	69.0 ±	16.98 §
non-HDL cholesterol	134.7 ±	31.47	140.0 ±	32.31 §	136.8 ±	33.65	142.2 ±	33.85 §
Fasting plasma glucose	97.3 ±	17.43	102.4 ±	26.42 §	91.8 ±	15.17	93.8 ±	15.27 §
Hemoglobin A1c	5.4 ±	0.63	5.6 ±	0.87 §	5.4 ±	0.51	5.4 ±	0.48 *
Creatinine	0.82 ±	0.10	1.08 ±	0.44 §	0.62 ±	0.08	0.82 ±	0.31 §
eGFR	76.4 ±	11.49	57.0 ±	13.15 §	78.0 ±	12.84	56.4 ±	11.21 §

\* p&lt;0.05

§ p&lt;0.01

Table 4 shows the results of lipid items TC, TG, and non-HDL cholesterol demonstrated in quartiles.

Table 4. The value of quartile for lipid parameters

parameters	Group 1	Group 2	Group 3	Group 4
Total cholesterol	$\leq 182$	$182 \leq 203$	$203 \leq 225$	$> 225$
Triglycerides	$\leq 66$	$66 \leq 90$	$90 \leq 125$	$> 125$
non-HDL-cholesterol	$\leq 116$	$116 \leq 137$	$137 \leq 160$	$> 160$

Table 5 shows the odds ratio of each lipid items demonstrated in quartiles. Group 2 and Group 3 showed insignificant results while Group 4 alone showed significant results in TC and TG. On the other hand, odds ratios of non-HDL were 1.21 (1.01-1.45) in Group 2 ( $116 \leq \text{non-HDL} < 137$ ), 1.34 (1.12-1.60) in Group 3, and 1.61 (1.35-1.92) in Group 4, all being significant values with strong linearity. (p for trend <0.01) The prevalence rate of CKD of males were 24.1% while that of females were 12.2%, and this difference reach statistical significantly.

Table 5. Relationship between quartile of lipid parameters for CKD

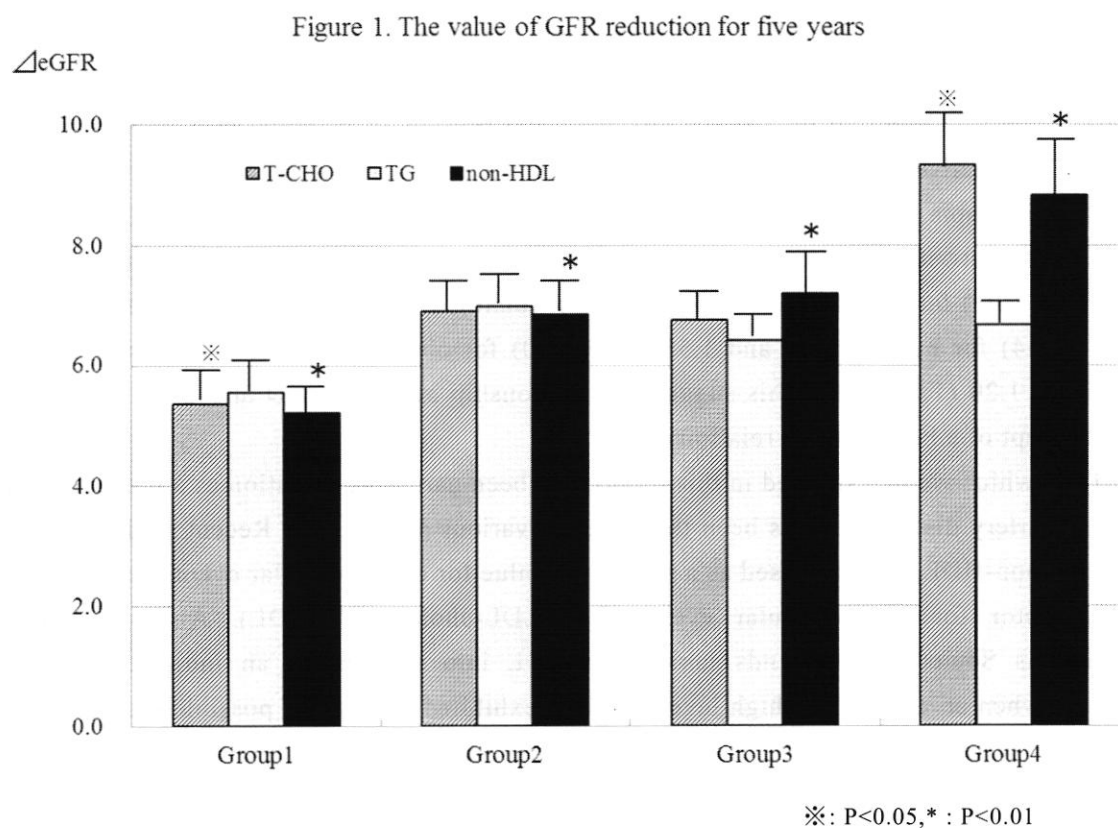
parameters	Total cholesterol		Triglycerides		non-HDL cholesterol	
	OR	95%CI	OR	95%CI	OR	95%CI
Group 1	1.00	(reference)	1.00	(reference)	1.00	(reference)
Group 2	0.98	0.82 - 1.16	1.02	0.87 - 1.19	1.21	1.01 - 1.45
Group 3	0.95	0.80 - 1.13	1.25	0.56 - 2.79	1.34	1.12 - 1.60
Group 4	1.27	1.07 - 1.51	1.54	1.34 - 1.78	1.61	1.35 - 1.92

We also investigated CKD odds ratios when 116mg/dl or less (25% of non-HDL when divided into quartiles as shown in table 4) was taken as a reference point: Group 2 was 1.21 ( $116 \leq \text{non-HDL} < 137$ ), Group 3 was 1.34 ( $137 \leq \text{non-HDL} < 160$ ) and Group 4 was 1.61 (1.35-1.92) with non-HDL over 160mg/dl. Thus, strong linearity was exhibited. (p for trend <0.01).

3.3 Longitudinal results

The amount of reduction of eGFR ( $\Delta$ eGFR) over five years after adjusting for sex and age in

each quartiles of non-HDL shown in Figure 1 were as follows: -5.21 in Group 1, -6.85 in Group 2, -7.20 in Group 3, and -8.82 in Group 4, demonstrating significant differences between Group 1 and Group 2 to 4. ( $p<0.01$ )



#### 4. Discussion

In this research, we have conducted a retrospective cohort study using data obtained from comprehensive medical examinations at Kasugai City Medical Center. We examined the relation between non-HDL, which is thought to be the latest indicator of coronary artery disease capable of comprehensive evaluation of arteriosclerosis provocative lipoproteins, and CKD, which is drawing attention in Japan in recent years with prevention measures being taken with the progress of research.

Research into the relationship between CKD and coronary artery disease has shown that even mildly impaired renal function is a CVD risk factor independent of existing CVD risk factors such as hypertension, diabetes or a history of myocardial infarction. The HOPE (Heart Outcomes Prevention Evaluation) Study<sup>17)</sup> revealed, for example, that a serum creatinine value of over 1.4mg/dl doubles the risk of cardiovascular mortality and all-cause mortality. The VALIANT study<sup>18)</sup>, which targeted patients after an acute myocardial infarction (AMI), also showed that the presence of mildly impaired renal function led to increased recurrence of infarctions, cardiovascular mortality and heart failure. Furthermore, a cohort study targeting 1,120,000 US



medical insurance subscribers that showed impaired renal function to also be a CKD risk in the general population reported that the risk of cardiovascular events, hospitalization and death increased each time GFR decreased by 15 points. In Japan, a study conducted by Irie et al.<sup>19)</sup> in Ibaraki Prefecture reported that cerebral stroke, cardiovascular events and all-cause mortality increased in both males and females when eGFR was less than 60ml/min/1.73m<sup>2</sup> and that risk of mortality due to cardiovascular disease doubled in males and increased four-fold in females when albuminuria was taken into account. In addition, the Framingham Heart Study<sup>20,21)</sup> and the Hisayama Study<sup>22-24)</sup> reported on risk factors for CVD present in patients with impaired renal function. All predictive factors for CKD onset such as old age, hypertension, glucose intolerance, diabetes, dyslipidemia, obesity and metabolic syndrome were all also risk factors for CVD onset. The odds ratios between CKD and each factor in our study were identical to other studies: 1.07 (95% CI 1.06-1.07) for age, 1.91 (1.67-2.17) for sex (male), 1.26 (1.06-1.50) for hyperglycemia, 1.18 (1.04-1.34) for hypertension and 1.55 (1.33-1.80) for obesity. Non-HDL results were also significant, at 1.20 (1.05-1.36). This suggests a relationship between CKD and CVD and fits in with the concept of a heart-kidney relationship.

Non-HDL, which we investigated in this report, has been garnering attention as a new indicator for coronary artery disease and has been the target of various studies<sup>13,14)</sup>. Recent studies<sup>15)</sup> have reported that non-HDL, which is used as a predictive value for cardiovascular events, is a stronger predictive factor for cardiovascular events than LDL-cholesterol (LDL). Also, the Japan Atherosclerosis Society recommends taking non-HDL into account as an indicator for risk management when an abnormally high level of TG is exhibited because of possible inconsistency between kits when measuring LDL directly<sup>12)</sup>. Atherogenic lipoproteins such as remnant lipoprotein and small dense LDL are known to increase when hypertriglyceridemia occurs. However, because TG is easily influenced by diet and fluctuates readily, it is difficult to use as a control target value. Therefore, a sound indicator for hypertriglyceridemia was needed and non-HDL, which is HDL-C deducted from TC, was able to fulfill this role. Non-HDL is a value that encompasses all malignant lipoproteins including atherogenic lipoproteins present in hypertriglyceridemia such as remnant lipoproteins and small dense LDL, as well as LDL. Non-HDL is not influenced by diet because neither TC nor HDL-C, which are necessary in its calculation, are easily influenced by diet. It is also highly useful because it uses previously measured test data (therefore not requiring any new testing) and because it is a rational indicator. Therefore, it is currently anticipated to be used at clinical sites and for health checkups and has been the target of numerous studies. The Japan Arteriosclerosis longitudinal study (JALS)<sup>25)</sup>, which was conducted domestically within Japan, divided non-HDL into quartiles as we did in this study, and a relationship with myocardial infarction (MI) was observed. As a result, each quartile exhibited results almost identical to those in our study: Group 1 – 117mg/dl, Group 2 – 118-141 mg/dl, Group 3 – 142-166mg/dl and Group 4 – 160mg/dl. When the lowest group was subsequently used as a reference point, it was observed that the higher the higher the group's rank, the stronger the relationship with MI risk. Just as in this study, the CKD odds ratio increased concurrently with non-HDL when non-HDL<116mg/dl (lowest quartile group) was used as a



reference point: 1.21 (1.01-1.45) when  $116 \leq \text{non-HDL} < 137 \text{ mg/dl}$ , 1.34 (1.12-1.60) when  $137 \leq \text{non-HDL} < 160 \text{ mg/dl}$  and 1.58 (1.32-1.90) when  $160 \leq \text{non-HDL} < 200 \text{ mg/dl}$ . The odds ratio was 1.79 (1.30-2.48) when  $\text{non-HDL} \geq 200 \text{ mg/dl}$ . (p for trend  $< 0.01$ ). Furthermore, the longitudinal analysis showed a significantly large reduction in eGFR for the non-HDL groups over five years after adjusting for sex and age. This indicated a relationship between non-HDL and CKD. These results are identical to those reported by Holzman et. al<sup>26)</sup> and suggest that both CKD and CVD are caused by changes in lifestyle habits and the aging of the population that has occurred in recent years. They also suggest that CKD is vascular pathology caused by lifestyle habits and aging and that CKD and CVD are therefore closely related to one another.

## 5. Conclusion

Our study suggests that non-HDL is involved in the development and progression of CKD because elevated non-HDL is a CKD risk factor and rises in non-HDL lead to decreased eGFR and increased incidences of CKD. A future topic of debate is likely to be whether CKD prognosis can be improved by treating non-HDL.

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## Non-HDL コレステロールと慢性腎臓疾患との関連性

柴田 清

【目的】 米国腎臓財団により提唱された慢性腎臓疾患 (CKD) は、近年、日本でも注目され、研究が進み予防対策がとられてきている。そこで、今回われわれは、冠動脈疾患の新たな指標として注目されている総コレステロール (TC) から HDL-cholesterol (HDL-C) を差し引いた non-HDL-コレステロール (non-HDL) と慢性腎臓疾患 (CKD) との関連を調べ検討したので報告する。

【対象および方法】 平成 18 年 4 月から平成 20 年 3 月までに春日井市健康管理センターの人間ドックを受診した受診者のうち、脂質代謝異常症を治療中のものを除いた 7,725 名 (男性 3,983 名、女性 3,742 名) を対象とした。

まず、CKD の判定基準を蛋白尿陽性もしくは推算糸球体濾過量 (eGFR: mL/min/1.73 m<sup>2</sup>, 以下単位略) が 60 未満とし、性別および年代別における CKD の有病率および non-HDL の四分位を算出した。

次に、non-HDL の四分位の 25% 未満 (Q1) を基準として、四分位の各群 (Q2~Q4) の CKD に対するオッズ比を各々求め比較検討した。

さらに、5 年前にも同センターにて健診を受け、5 年前の時点において脂質異常症でなかった 1,584 名 (男性 964 名、女性 620 名) を対象に、non-HDL を四分位に分け各々の eGFR の減少量 ( $\Delta$ eGFR) を求め比較検討した。

【結果】 対象者全体の特性 (平均値  $\pm$  標準偏差) は、年齢は男性  $61 \pm 11.9$  歳、女性  $59 \pm 10.8$  歳、CKD の指標である eGFR は男性  $71.7 \pm 14.5$ 、女性  $75.3 \pm 14.5$ 、蛋白尿陽性率は男性 6.6%、女性 1.9%、CKD 有病率は全体で男性 24.1%、女性 12.2% であった。

non-HDL の四分位は Q1:  $\sim 116$ , Q2:  $116 \sim 137$ , Q3:  $137 \sim 160$ , Q4:  $160 \sim$  となった。

次に四分位の Q1 を基準とした各オッズ比は Q2: 1.07 (95%CI: 1.06-1.08), Q3: 1.58 (1.40-1.79), Q4: 1.37 (1.18-1.59) となった。

また、5 年間の縦断的结果では、性、年齢補正後の  $\Delta$ eGFR がそれぞれ Q1: -5.21, Q2: -6.85, Q3: -7.20, Q4: -8.82 となり、Q1 と Q2~Q4 の間には有意な差が認められた ( $p < 0.01$ )。

【まとめ】 今回のわれわれの研究において、non-HDL が高くなるほど CKD に対するオッズ比が有意に高まることが明らかとなった。さらに、CKD の指標である eGFR の低下量においても non-HDL の高い群のほうが有意に高い結果となった。これは non-HDL と CKD との間に高い関連性がみられ、冠動脈疾患の指標とされている non-HDL は、CKD の発症進展においても重要であることが示唆された。