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Comparison of non-fasting LDL-C levels calculated by Friedewald formula with those directly measured in Chinese patients with coronary heart disease after a daily breakfast☆

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ABSTRACT

Background: LDL-C level can be measured by direct methods (LDL-C_M) or calculated by Friedewald formula (LDL-C_C). The aim of this study was to investigate the difference between LDL-C_M and LDL-C_C after a daily breakfast in Chinese patients with coronary heart disease (CHD).

Methods: Three hundred and three inpatients, including 203 CHD patients (CHD group) and 100 non-CHD controls (CON group), were enrolled in this study. Serum levels of blood lipid parameters, including LDL-C_C and LDL-C_M, at 0, 2 and 4 h (h) were monitored after a daily breakfast in all subjects.

Results: LDL-C_M was significantly higher than LDL-C_C in fasting state in each group and at 4 h postprandially in CHD group ($P < .05$). Postprandial LDL-C_M and LDL-C_C significantly decreased in each group ($P < .05$). Postprandial decline in LDL-C_M was significantly greater than that of LDL-C_C ($P < .05$). For CHD patients taking statins for ≥1 month before admission, non-fasting percent attainment of LDL-C_M or LDL-C_C was significantly higher than its fasting value, especially at 4 h ($P < .05$). The percent deviation of LDL-C_M from 1.8 mmol/L at 4 h was significantly different from its fasting value. However, there was no significant difference in percent deviation of LDL-C_C from 1.8 mmol/L between fasting and non-fasting states.

Conclusions: It indicated that the clinical monitoring of non-fasting LDL-C level in CHD patients could be relatively complex, and the judgement may depend not only on the method to acquire LDL-C level, but also on the evaluation method.

1. Introduction

Elevated low-density lipoprotein cholesterol (LDL-C) level is considered as a causal factor of coronary heart disease (CHD) since the release of the first National Cholesterol Education Program Adult Treatment Panel guidelines in 1988 [1,2]. It is well-known that reducing LDL-C level can lower morbidity and mortality of CHD [3–6]. Thus, it is very important to monitor the change in LDL-C level after the treatments of statins and other lipid-lowering medications. Since 2009, according to recommendations from the Danish Society for Clinical Biochemistry, non-fasting blood lipids testing at any random time-point after a daily meal was carried out in all laboratories in Denmark [7]. Similarly, non-fasting lipids detection was endorsed in the primary prevention since 2014 by the NICE guidelines [8]. Now it has been recommended to detect LDL-C level in both fasting and non-fasting states in European countries and the United States [7–11]. At present, LDL-C level is still routinely measured in the fasting state and there is no recommendation for non-fasting lipid testing in China yet.

As a reference method to measure serum LDL-C level, the β-quantification method is seldom used because ultracentrifugation is not available in most clinical laboratories [12]. Since 1995, a 'homogeneous' or 'direct' assay has become an alternative to the β-quantification method to measure serum LDL-C level [13–15] and this alternation has been recommended by an expert panel of NCEP [16]. Currently, LDL-C level can be acquired by Friedewald calculation (calculated LDL-C level, LDL-C_C) at triglyceride (TG) < 4.5 mmol/L

☆ All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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[17] or measured by the commercially available direct methods (measured LDL-C level, LDL-C_M) regardless of TG level in many clinical laboratories. A lot of evidence shown that Friedewald calculation might underestimate LDL-C level at relative low concentrations when compared with the direct measurement in the fasting state [18–22]. However, there was rare study to compare LDL-C_M with LDL-C_C in the non-fasting state [23]. As we know that studies about predictive effect of non-fasting blood lipids after a daily meal on cardiovascular diseases in Chinese subjects were very rare [24], especially about the comparison of fasting and non-fasting blood lipids within a same day [25]. Furthermore, there was no study to compare non-fasting LDL-C_M with non-fasting LDL-C_C in Chinese CHD patients after a daily meal.

Thus, the aim of this study was to compare LDL-C_C with LDL-C_M in Chinese patients with CHD after a daily breakfast, and to evaluate goal attainment of LDL-C controlling according to non-fasting LDL-C_M or LDL-C_C, respectively, in CHD patients.

2. Methods

2.1. Subjects

Three hundred and three inpatients, including 203 documented CHD patients (CHD group) and 100 non-CHD subjects (CON group), were recruited in this study from March 2017 to June 2018 in the Department of Cardiovascular Medicine of the Second Xiangya Hospital, Central South University. CHD was defined as a history of myocardial infarction (MI) and/or angiographically proven coronary atherosclerosis in patients with angina pectoris (AP). Contemporaneous subjects who had no clinical history and manifestation of CHD were classified into CON group.

Next, to evaluate the goal attainment of LDL-C controlling in CHD patients, some cases in CHD group were excluded, including those with a new onset of MI within recent one month (1 m) for potential impact of stress response on blood lipids (subgroup 1, n = 45), as well as those with AP but without taking any statin or taking statins for < 1 m before admission (subgroup 2, n = 82). Thus, evaluation of goal attainment was carried out in those with AP and taking statins for ≥1 m before admission (subgroup 3, n = 76).

The study was approved by the Ethics Committee of the Second Xiangya Hospital of Central South University and informed consent was gained from all participants.

2.2. Laboratory examinations

After fasting for at least 8 h, venous blood samples were collected in all subjects before (i.e. 0 h) and at 2 h, 4 h after a breakfast according to their daily diet habits. Serum levels of total cholesterol (TC) and TG were measured by automated enzymatic assays, and those of LDL-C_M and high-density lipoprotein cholesterol (HDL-C) were measured by a commercially available direct method (Wako, Japan), i.e. selective protection method and antibody blocking method, respectively, on a HITACHI 7170A analyzer (Instrument Hitachi Ltd., Tokyo, Japan) [26–28] by a laboratory technician who had no idea of this study [29]. LDL-C_C = TC − HDL-C − TG/2.2 (in mmol/L) only when TG was < 4.5 mmol/L, otherwise it was substituted by LDL-C_M.

2.3. Evaluation of goal attainment of LDL-C controlling in CHD patients

Due to the lack of baseline LDL-C level in a considerable number of patients in subgroup 3, goal attainment of LDL-C controlling was evaluated as percent attainment of LDL-C goal (i.e. < 1.8 mmol/L) and the percent deviation of LDL-C from 1.8 mmol/L. The latter was calculated as (LDL-C − 1.8)/1.8 × 100%.

2.4. Statistical analysis

Statistical analysis was performed on Statistical Package for Social Sciences version 20.0. Data drawing was completed by Origin Pro 8.0 software. Quantitative variables were expressed as mean ± standard deviation (SD), and qualitative variables were expressed as numbers and percentages. TG level not conforming to a normal distribution was log transformed for comparison and presented as median with 25th and 75th percentiles. Differences between the intra- and inter-group means were analyzed by *t*-test or one-way ANOVA. Categorical variables were compared using chi-squared statistic tests. All P values were 2-tailed, and P < .05 was considered statistically significant.

3. Results

3.1. Clinical characteristics and fasting blood lipids of two groups

The baseline characteristics, including sex, age, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP) between CHD group and CON group were roughly matched. CHD group had higher percentages of current smoking, diabetes and statins intake (P < .05) (Table 1).

There was no significant difference in TG level between two groups, except that levels of TC, HDL-C and LDL-C_M were significantly lower in CHD group (P < .05), which could be associated with higher percentage of cases taking statins or the potential impact of stress response on blood lipids from a recent MI in 22.2% cases in CHD group (Table 1).

Fasting LDL-C_M was significantly higher than fasting LDL-C_C in each group (P < .05) (Fig. 1A and B) and exceeded fasting LDL-C_C in 85.1% CHD patients and 85.0% non-CHD controls, respectively.

3.2. Non-fasting changes in both LDL-C_M and LDL-C_C in CHD group and CON group

Levels of TC, HDL−C, LDL-C_M and LDL-C_C significantly decreased while that of TG significantly increased from baseline to 4 h in each group (P < .05) (Fig. 1A and B, Supplementary Fig. S1A−C).

The non-fasting difference between LDL-C_C and LDL-C_M seemed to be almost due to the fasting difference between them. Non-fasting LDL-C_M exceeded non-fasting LDL-C_C in about 70% CHD patients (at 2 h: 74.2%, at 4 h: 69.7%) and in > 60% non-CHD controls (at 2 h: 71.0%, at 4 h: 62.0%). Although non-fasting LDL-C_M seemed higher than non-fasting LDL-C_C in each group, the difference only reached statistical significance at 4 h in CHD group (P < .05) (Fig. 1A). Postprandial

Table 1
Clinical characteristics of CHD group and CON group.

	CHD (n = 203)	CON (n = 100)
Male, n(%)	147(72.4)	68(68.0)
Age, y	60 ± 10	58 ± 7
BMI, kg/m^2	24.9 ± 3.6	24.2 ± 2.9
SBP, mm Hg	131.9 ± 20.6	132.8 ± 19.5
DBP, mm Hg	79.0 ± 13.5	79.8 ± 12.2
Current smoking, n(%)	99(48.8)*	33(33.0)
Statins treatment, n(%)	117(57.6)*	17(17.0)
Diabetes, n(%)	50(24.6)*	12(12.0)
TG, mmol/L	1.41(1.05, 2.06)	1.48(1.10, 2.04)
TC, mmol/L	3.95 ± 0.98*	4.32 ± 0.88
HDL-C, mmol/L	1.04 ± 0.28*	1.13 ± 0.26
LDL-C_M, mmol/L	2.47 ± 0.86*	2.74 ± 0.76

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglyceride; TC, total cholesterol; HDL−C, high-density lipoprotein cholesterol; LDL-C_M, measured low-density lipoprotein cholesterol level. Lipids was directly measured at fasting state. Continuous variables were presented as mean ± SD except for TG.

* P < .05 when compared with CON group.

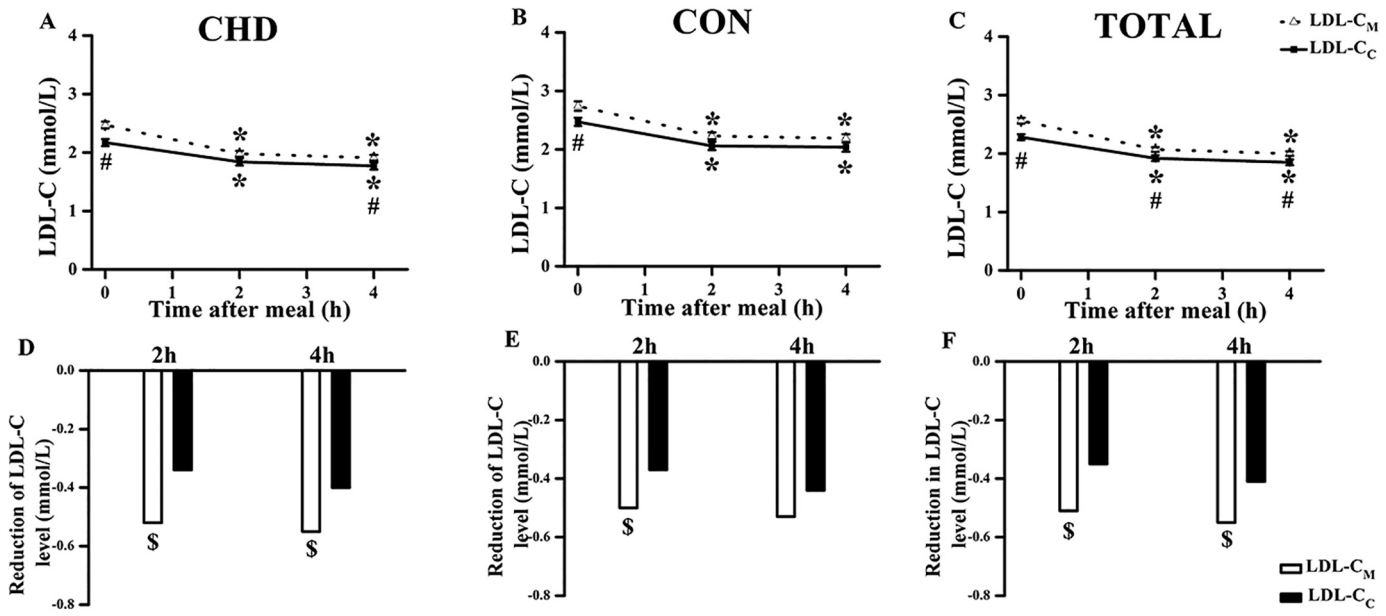

Fig. 1. Comparison between non-fasting changes in LDL-C_M and LDL-C_C in two groups. (A–C) Comparison between LDL-C_M and LDL-C_C in non-fasting state in CHD group (n = 203) (A), CON group (n = 100) (B) and total subjects (n = 303) (C). (D–F) Comparison between postprandial reductions in LDL-C_M and LDL-C_C in each group (D, E) and total subjects (F) after a daily breakfast. $^{\#}P < .05$ when compared with LDL-C_M at the same time point. $^{*}P < .05$ when compared with the fasting LDL-C level acquired by the same method. $^{\$}P < .05$ when compared with postprandial reduction in LDL-C_C at the same time point.

reduction in LDL-C_M was significantly greater than that in LDL-C_C at 2 h or 4 h in CHD group, while only at 2 h in CON group ($P < .05$) (Fig. 1D and E).

When taking all subjects as a whole (n = 303), LDL-C_M were significantly higher than LDL-C_C in both fasting and non-fasting states. Moreover, postprandial decline in LDL-C_M was significantly greater than that in LDL-C_C at both 2 h and 4 h (Fig. 1C and F).

3.3. Clinical characteristics and fasting blood lipids of three subgroups

There was no difference in sex, age, BMI, SBP, DBP, percentages of current smoking and diabetes, and fasting HDL-C level among three subgroups. The highest fasting TG level was found in subgroup 1 ($P < .05$), which may be related to the impact of stress response on blood lipids from a recent MI within 1 m. The lowest fasting levels of TC and LDL-C_M were found in subgroup 3, which was associated with high rate and long time of statin treatment ($P < .05$) (Table 2). The difference between fasting LDL-C_M and LDL-C_C reached statistic significance in subgroup 1 and 2 ($P < .05$) but not in subgroup 3 with the lowest LDL-C_M (Fig. 2A–C).

3.4. Non-fasting changes in both LDL-C_M and LDL-C_C in three subgroups

Non-fasting LDL-C_M and LDL-C_C showed significant decline in each subgroup ($P < .05$), except for non-fasting LDL-C_C at 2 h in subgroup 1 (Fig. 2A–C). Fasting LDL-C_M in subgroup 3 was the lowest among subgroups. Therefore, non-fasting LDL-C_M in subgroup 3 were significantly lower than those in other subgroups ($P < .05$) (Fig. 2D). Similarly, difference in LDL-C_C between subgroup 2 and 3 reached statistical significance in fasting as well as non-fasting state ($P < .05$) (Fig. 2E). It indicated that non-fasting difference in LDL-C level among/between subgroups could depend on its fasting difference.

Postprandial reduction in LDL-C_M was significantly higher than that in LDL-C_C in subgroup 1 or 2 but not in subgroup 3 ($P < .05$) (Fig. 2F and G), suggesting that difference in postprandial decline between LDL-C_M and LDL-C_C tended to smaller when LDL-C level was relatively low, such as in subgroup 3. The greatest postprandial reduction in LDL-C_M and the most obvious difference in postprandial decline between LDL-C_M and LDL-C_C was found in subgroup 1, which could be relevant to higher non-fasting TG level (Supplementary Fig. S2) in this subgroup.

Table 2
Clinical characteristics of CHD patients among three subgroups.

	Subgroup 1 (n = 45)	Subgroup 2 (n = 82)	Subgroup 3 (n = 76)
Male, n(%)	37(82.2)	55(67.1)	55(76.4)
Age,y	59 ± 10	60 ± 11	61 ± 9
BMI, kg/m^2	24.9 ± 3.5	25.3 ± 4.0	24.7 ± 2.8
SBP, mm Hg	127.1 ± 23.1	135.0 ± 20.4	131.5 ± 18.8
DBP, mm Hg	77.6 ± 11.7	80.7 ± 12.9	78.1 ± 15.0
Current smoking, n(%)	25(55.6)	40(48.8)	34(44.7)
Diabetes, n(%)	12(26.7)	17(20.7)	21(27.6)
Statins treatment, n(%)	19(42.2)*	22(26.8)*	76(100)
TG, mmol/L	1.86(1.28,2.26)*	1.39(0.92,2.13)	1.33(1.02,1.87)
TC, mmol/L	4.11 ± 1.04*	4.12 ± 0.94*	3.67 ± 0.95
HDL-C, mmol/L	1.00 ± 0.37	1.08 ± 0.27	1.02 ± 0.23
LDL-C_M, mmol/L	2.65 ± 0.86*	2.58 ± 0.81*	2.24 ± 0.86

subgroup 1: CHD patients with a new onset of myocardial infarction (MI) within recent one month; subgroup 2: those with AP but without taking any statin or taking statins for < 1 m before admission; subgroup 3: those with AP and taking statins ≥ for 1 m before admission.

* $P < .05$ when compared with subgroup 3.

3.5. Evaluation of goal attainment of LDL-C controlling in subgroup 3

For CHD patients taking statins for ≥1 m (i.e. subgroup 3), percent attainment of LDL-C_C < 1.8 mmol/L seemed higher than that of LDL-C_M < 1.8 mmol/L in both fasting and non-fasting states, although the difference did not reach statistical significance. Moreover, non-fasting percent attainment of LDL-C_M significantly increased at 2 h or 4 h when compared with its fasting percent attainment ($P < .05$). Percent attainment of LDL-C_C showed a similar increase in non-fasting state but the difference reached statistical significance only at 4 h ($P < .05$) (Fig. 3A).

Percent deviation of LDL-C_M from 1.8 mmol/L was significantly different from that of LDL-C_C at each time point ($P < .05$). The percent

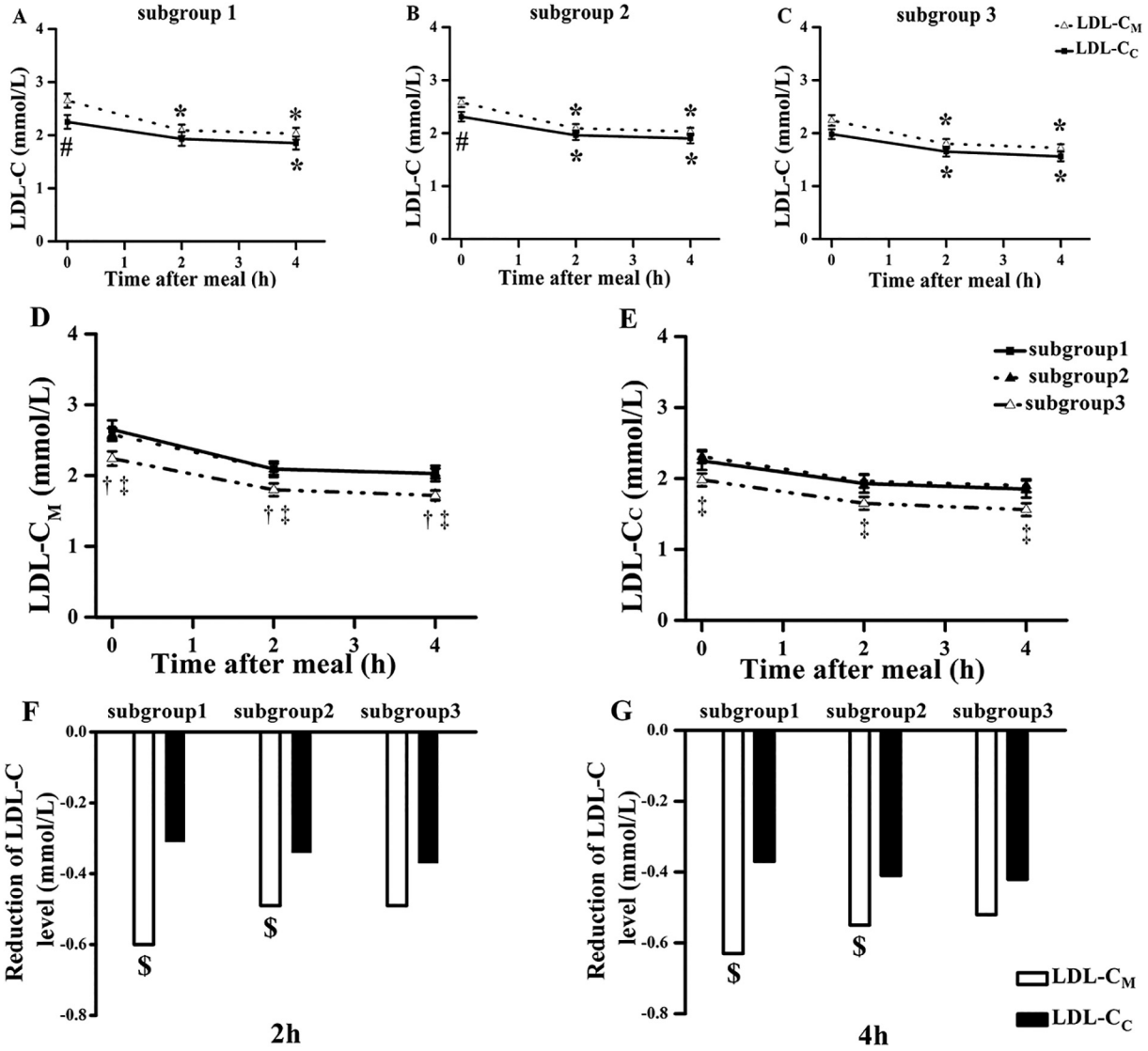

Fig. 2. Comparison between non-fasting changes in LDL-C_M and LDL-C_C in three CHD subgroups. (A–C) Comparison between LDL-C_M and LDL-C_C in non-fasting state in subgroup 1 (n = 45) (A), subgroup 2 (n = 82) (B) and subgroup 3 (n = 76) (C). (D, E) Comparison of non-fasting LDL-C_M (D) or non-fasting LDL-C_C (E) among three subgroups. (F, G) Comparison between postprandial reductions in LDL-C_M and LDL-C_C in three subgroups at 2 h (F) or 4 h (G) after a daily breakfast. $^{\#}P < .05$ when compared with LDL-C_M at the same time point. $^{*}P < .05$ when compared with the fasting LDL-C level acquired by the same method. $^{\dagger}P < .05$ when compared with subgroup 1 at the same time point. $^{\ddagger}P < .05$ when compared with subgroup 2 at the same time point. $^{\$}P < .05$ when compared with postprandial reduction in LDL-C_C within the same subgroup.

deviation of LDL-C_M at 4 h was significantly different from its fasting value ($P < .05$). However, there was no significant difference in percent deviation of LDL-C_C between fasting and non-fasting states (Fig. 3B).

4. Discussion

Non-fasting levels of blood lipids can better reflect the real situation of blood lipids within human body because people spend most of the

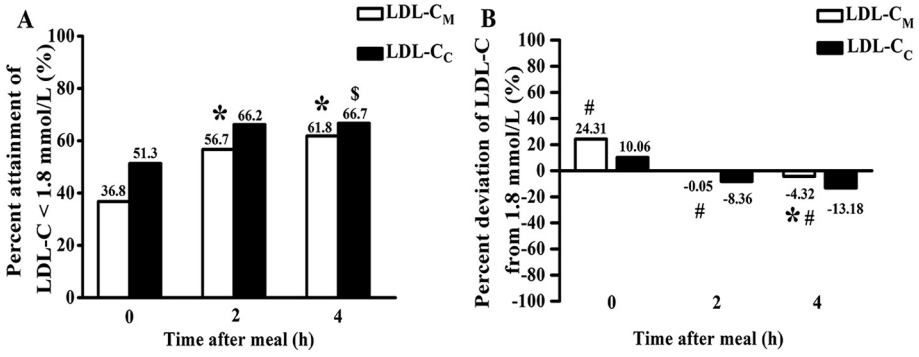

Fig. 3. Comparison of percent attainment of LDL-C < 1.8 mmol/L and percent deviation of LDL-C from 1.8 mmol/L in fasting and non-fasting states in subgroup 3. (A) Comparison of percent attainment of LDL-C (LDL-C_M vs. LDL-C_C) < 1.8 mmol/L in fasting and non-fasting states. (B) Comparison of percent deviation of LDL-C (LDL-C_M vs. LDL-C_C) from 1.8 mmol/L in fasting and non-fasting states. $^{*}P < .05$ when compare with fasting percent attainment or deviation of LDL-C_M. $^{\$}P < .05$ when compare with fasting percent attainment of LDL-C_C. $^{\#}P < .05$ when compare with percent deviation of LDL-C_C at the same time point.

day in a non-fasting state. Studies about detection of non-fasting blood lipids after a daily diet in China were quite few [24,25], although those after a high-fat diet had been widely reported [30–33]. In this investigation, it was firstly reported that measured LDL-C level was significantly different from calculated LDL-C level in both fasting and non-fasting states. Prominent reductions in both LDL-C_C and LDL-C_M were found in non-fasting state. Moreover, non-fasting percent attainment of LDL-C level, either LDL-C_M or LDL-C_C, < 1.8 mmol/L was significantly higher than its fasting percent attainment, especially at 4 h. However, when percent deviation of LDL-C from 1.8 mmol/L was assessed, only percent deviation of LDL-C_M from 1.8 mmol/L at 4 h was significantly different from that at fasting state, while that of LDL-C_C showed borderline variations around the goal in fasting and non-fasting states. Thus, it could be necessary to carefully analyze which LDL-C level (LDL-C_C or LDL-C_M) should be selected in the clinical monitoring of non-fasting LDL-C levels in Chinese patients with CHD.

Study about inconsistency between LDL-C_C and LDL-C_M was exactly scarce in China, especially in non-fasting state. In this study, non-fasting LDL-C_M exceeded non-fasting LDL-C_C in most subjects. However, the discordance between LDL-C_M and LDL-C_C was significant only at 4 h in CHD group after a daily breakfast. Interestingly, there was a peak of TG level in Chinese patients at about 4 h after a low-fat or high-fat breakfast in our previous studies [30–33]. It suggested that TG level could be one of important clinical parameters that affect the accuracy of Friedewald formula [34–37]. Some studies showed that Friedewald calculation underestimates fasting LDL-C_M level when TG ≥ 1.7 mmol/L and the discrepancy between the two LDL-C levels increased linearly as TG level increased [34,37]. On the contrary, another investigation presented that fasting Friedewald LDL-C_C was significantly higher than fasting and non-fasting LDL-C_M in American participants with TG ≤ 2.26 mmol/L (200 mg/dL) [35]. In addition, Chung and et al. [34] found that relative low or high TG level could affect the accuracy of Friedewald formula in Korean. Although those data were somewhat contradictory and complicated, all of them implied that the accuracy of Friedewald LDL-C remained to be discussed [35,38,39].

However, the observed discrepancies between LDL-C_C and LDL-C_M could also simply reflect variation among different commercially available direct methods used to measure LDL-C and HDL-C levels (used in the Friedewald calculation) [13]. Miller et al. [40] compared four different homogeneous or direct methods for LDL-C detection with the β-quantification reference method and found that those methods had progressively poor performance as TG level elevated moderately when Friedewald calculation was not unsuitable. Direct assays based on different principles may select different subclasses of LDL or HDL that may or may not be equally quantified, depending on the assay procedure and reagents. Test results differ substantially between the various direct methods from different manufacturers, particularly in hypertriglyceridemic (TG > 2 mmol/L or 175 mg/dL) samples [13]. Thus, discrepancies in terms of percent difference in LDL-C (measured vs. calculated) may be less or more seen when using another direct method.

Additionally, LDL-C accuracy by Friedewald calculation with a fixed ration had been questioned [17,36,37,39,41]. LDL-C_C level was estimated according to Friedewald equation that was derived since 1970s and used a fixed ration of 2.2:1 in mmol/L or 5:1 in mg/dL between TG and very low density lipoprotein-cholesterol (VLDL-C) in fasting state [17]. However, the ration of TG to VLDL-C will vary with TG increasing in either non-fasting or fasting state, which induces significant errors in LDL-C estimation [17,39,41]. When taking the β-quantification method as a reference, accuracy was higher with estimated LDL-C level using an adjustable factor for the TG:VLDL-C ratio compared with the fixed Friedewald LDL-C_C in both fasting and non-fasting states [39]. It suggested that Friedewald LDL-C_C with a adjustable or flexible ration seems to be a better choice in non-fasting state [17,39,41].

Non-fasting change in LDL-C level (most of which is LDL-C_C) was considered slight according to several studies with large population in Denmark [7,9,42,43]. In this study, the postprandial changes in LDL-C level were more obvious. Moreover, postprandial reductions in LDL-C_M were more prominent than those of LDL-C_C. The underling mechanisms for the decline of LDL-C level after meals are not yet fully elucidated. Postprandial reduction in LDL-C level was thought to due to fluid intake but not food intake and thus adjusting the data for albumin concentration was recommended [10,42]. Langsted et al. [42] observed that non-fasting LDL-C level did not change after adjusting for plasma albumin concentration as a marker of water intake. Therefore, the only way to prevent this drop in LDL-C level using either fasting or non-fasting samples was recommended to minimize water intake before lipid testing by Nordestgaard et al. [9]. It was pity that non-fasting albumin concentration was not detected in this study, so it is not clear whether hemodilution would be the only cause of postprandial decline in LDL-C level. Additionally, it was reported that LDL-C_M and LDL-C_C correlated well in general population statistics [9], but not in an individual subject [13,14]. Thus, whether postprandial reduction in LDL-C level is obvious or not could be related to sample size. In addition, postprandial high TG level may also affect the accuracy of LDL-C_M detection by a certain commercially direct method. As for if there are other more complex mechanisms, further research is needed to explore.

Fasting LDL-C level in CHD patients should be below a target of 1.8 mmol/L (70 mg/dL) (or by reducing ≥50% if this target cannot be attained) [44–46]. Recently, a new guideline suggests that achievement of a > 50% reduction in fasting LDL-C level is paramount for CHD patients irrespective of baseline LDL-C level [47]. However, the baseline LDL-C levels in a considerable number of patients were unavailable in this study. Therefore, percent attainment of LDL-C goal < 1.8 mmol/L and percent deviation of LDL-C from 1.8 mmol/L was evaluated. The difference between percent attainment of LDL-C_M and that of LDL-C_C did not reach statistic significance in non-fasting state in subgroup 3, indicating that either non-fasting LDL-C_M or non-fasting LDL-C_C could be used to evaluate the goal attainment of LDL-C controlling in CHD patients. It was worth noting that percent attainment of LDL-C, either LDL-C_M or LDL-C_C, < 1.8 mmol/L in non-fasting state was higher than that in fasting state in subgroup 3, especially at 4 h. However, the difference in percent deviation of LDL-C_C, but not LDL-C_M, from 1.8 mmol/L between fasting and non-fasting states did not reach statistic significance. It suggested that postprandial percent deviation of LDL-C_C was not large enough to translate to a different treatment choice for a physician. Considering the cost of direct measurement of LDL-C_M, it seems no substantial advantage for using LDL-C_M when compared with LDL-C_C to evaluate percent deviation of LDL-C_C from 1.8 mmol/L.

There were several limitations in this study. Firstly, the number of cases in this study was relatively small when compared with other similar studies [34,35]. Secondly, non-fasting albumin concentration was not detected in the subjects. Thirdly, no reference method (the β-quantification method) was used in this study. Fourthly, non-fasting lipids detection can be recommended in patients on stable drug therapy according to a European consensus statement [9]. Obviously, most of CHD patients in subgroup 3 did not meet this condition. In the future, we would further explore the application of non-fasting LDL-C detection in CHD patients in a prospective study with a large sample size.

5. Conclusions

There was a significant difference between LDL-C_M and LDL-C_C levels as well as their postprandial drops in non-fasting state in Chinese subjects. The latter induced elevated percent attainment of LDL-C (LDL-C_M or LDL-C_C) < 1.8 mmol/L in non-fasting state, but not significant difference in percent deviation of LDL-C_C from 1.8 mmol/L between fasting and non-fasting states. It indicated that the clinical monitoring of non-fasting LDL-C level in CHD patients could be relatively complex, and the judgement may depend not only on the method to acquire LDL-C level, but also on the evaluation method.

Supplementary data to this article can be found online at https://

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Conflict of interest

The authors report no relationship that could be construed as a conflict of interest.

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