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RESEARCH ARTICLE

# Lipid profile of patients with uncontrolled type 2 diabetes mellitus at a tertiary South African hospital

**A Parak[1,2*], R Daya[1,2] and Z Bayat[1,2]**

[1]*Division of Endocrinology and Metabolism, Department of Internal Medicine, Helen Joseph Hospital, Johannesburg, South Africa*
[2]*Division of Endocrinology and Metabolism, Department of Internal Medicine, Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, South Africa*
**Correspondence: amirahparak@gmail.com*



**Introduction:** Patients with type 2 diabetes mellitus (T2DM) are at very high risk of cardiovascular disease (CVD). All modifiable risk factors for CVD, especially dyslipidaemia, need to be screened for and managed.
**Objective:** The primary objective was to determine lipid profiles of patients with uncontrolled T2DM. The secondary objectives was to determine whether patients were achieving a target low density lipoprotein cholesterol (LDL-C) level, and whether statin choice and dosing were appropriate.
**Methods:** A retrospective review of files at Helen Joseph Hospital diabetic clinic.
**Results:** 229 patients with poorly controlled T2DM ($HbA_{1c}$ > 8%) were studied. The prevalence of dyslipidaemia was 93.01% ($n$ = 213/229). In the primary prevention group, 61.84% of females and 60.47% of males did not achieve target LDL-C < 1.8 mmol/L. In the secondary prevention group, 66.67% of females and 83.33% of males did not achieve target LDL-C < 1.4 mmol/L. Statin therapy was prescribed for 86% ($n$ = 197) of patients. No correlation was found between $HbA_{1c}$ and different lipid parameters.
**Conclusion:** This study highlights the suboptimal lipid targets achieved by patients at a specialised diabetic clinic in South Africa. A combination of factors including clinical inertia, clinician knowledge of lipid targets, and a lack of second line lipid therapy needs to be addressed.

**Keywords:** dyslipidaemia, diabetes, lipid targets, LDL-C targets, statin therapy

## Introduction

According to the 2021 International Diabetes Federation (IDF) statistics, 1 in 22 adults in Africa is living with diabetes. More than half (54%) of people living with diabetes (PLWD) in Africa are undiagnosed. The IDF estimates the prevalence of diabetes in South Africa (SA) to be 10.8%. In 2021, diabetes was responsible for 6.7 million deaths globally, and 416 000 deaths in Africa.[1] Cardiovascular disease (CVD) is the leading cause of death in PLWD.[1]

The treatment of diabetes starts with optimising glycaemic control.[2] This is defined by the Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA) as a glycated haemoglobin ($HbA_{1c}$) target of < 7% in most patients, to prevent the development of microvascular disease.[3] There is an incremental increase in microvascular disease, CVD, and all-cause mortality for each 1% increase in $HbA_{1c}$ above 7%.[2]

As CVD is the leading cause of mortality in PLWD, the risk factors that are associated with CVD need to be modified.[2] In PLWD, the risk of CVD is increased by 2–3 fold in males and 3–5 fold in females compared with non-diabetics.[3] Traditional risk factors include smoking, hypertension, dyslipidaemia, and increased waist circumference.[2]

Dyslipidaemia in diabetes is characterised by increased total cholesterol (TC) and triglycerides (TG) and decreased high-density lipoprotein cholesterol (HDL-C).[4] Low-density lipoprotein cholesterol (LDL-C) is usually not markedly elevated; however, there is an increase in small dense LDL-C particles, which are highly atherogenic. The clinical consequence of dyslipidaemia in PLWD is an increased risk of atherosclerotic cardiovascular disease (ASCVD).[4,5]

The goal of lipid-lowering therapy is based on the patient's ASCVD risk. Risk scoring is useful in PLWD to obtain an objective risk estimate; however, it is not needed to decide on initiation of lipid-lowering therapy as diabetes is considered to be a coronary risk equivalent.[3,6]

The following patients with T2DM are considered as having the highest risk:[3,7]

- existing atherosclerotic disease;
- age > 40 years;
- T2DM > 10 years plus one or more risk factors (smoking, hypertension, family history of early coronary artery disease (CAD), any albuminuria, dyslipidaemia);
- chronic kidney disease.

There is a weak association between raised TG and ASCVD, whereas there is an inverse relationship between HDL-C and ASCVD.[4] The ratio of TC to HDL-C can be used to predict CVD.[5] However, the best proven primary predictor of ASCVD is LDL-C.[4] Therapy is therefore aimed at lowering the LDL-C, as lowering of LDL-C by 1 mmol/L causes:[3,5,7]

- 10% reduction in all-cause mortality;
- 20% reduction in deaths due to coronary artery disease;

- 24% reduction in major coronary events;
- 15% reduction in stroke.

The 2017 SEMDSA guidelines recommend that all T2DM patients should have a target LDL-C of < 1.8 mmol/L.[3] The American Diabetes Association (ADA) has different targets for primary and secondary prevention. For primary prevention, the ADA standard of care guidelines 2024 recommends that patients with T2DM have an LDL-C target of < 1.8 mmol/L and a ≥ 50% drop from baseline.[8] For secondary prevention, in T2DM patients with established ASCVD, the ADA recommends a target LDL-C of < 1.4 mmol/L and a reduction of ≥ 50% from baseline.[8]

There are no treatment target values for HDL-C or TG. However, guidelines recommend the following ideal lipid profile: HDL-C > 1.0 mmol/L in men and > 1.2 mmol/L in women, and TG < 1.7 mmol/L.[3,7]

The first step in treatment of dyslipidaemia is dietary changes, weight loss, and smoking cessation.[7] Adequate glycaemic control is needed to control diabetic dyslipidaemia.[3] Statins are the first-line pharmacological therapy for treating patients with hypercholesterolaemia.[3,7] The statins available in South African public hospitals are simvastatin, atorvastatin, and rosuvastatin. Simvastatin is a moderate-intensity statin, whereas atorvastatin and rosuvastatin, in higher doses, are high-intensity statins.[7] Simvastatin is widely available, but atorvastatin is restricted to secondary and tertiary hospitals. Rosuvastatin 10 mg can be prescribed at selected hospitals in some provinces.

Second-line therapy for hypercholesterolaemia includes ezetimibe and proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors.[7]

Dietary TG restriction is the cornerstone of therapy for hypertriglyceridaemia. When TG are > 10 mmol/L a fibrate is recommended.[3,7]

The purpose of this study is to assess the lipid profile, treatment of dyslipidaemia, and achievement of LDL-C targets, in patients with uncontrolled T2DM, at a specialist diabetic clinic in South Africa. The glycaemic control of this patient cohort will be addressed in a different paper.

## Methodology

### Study setting

This study was a retrospective review of patients attending the diabetic clinic at Helen Joseph Hospital. A patient list was retrieved from the Guidepost programme (a telemedicine study conducted during the COVID-19 pandemic, to assess the effect of telemedicine on $HbA_{1c}$, in people living with type 2 diabetes who have an $HbA_{1c}$ > 8%). The guidepost programme enrolled 261 patients and was representative of the patient population. The period of assessment was from 1 January 2020–31 December 2021. Diabetic clinic files were reviewed to obtain the relevant data on demographics, comorbidities, lipid profiles, treatment and dosages.

### Definitions

1. Uncontrolled diabetes: $HbA_{1c}$ > 8%.[3]
2. Obesity: body mass index ≥ 30 kg/m.[2,3]
3. Dyslipidaemia: abnormal serum cholesterol (total cholesterol, LDL-C or HDL-C), triglycerides, or both.[3]
4. TC target: TC < 4.5 mmol/L.[3,7]
5. HDL-C target: HDL-C > 1.0 mmol/L in men, and > 1.2 mmol/L in women.[3,7]
6. TG target: TG < 1.7 mmol/L.[3,7]
7. LDL-C target:
   (a) Primary prevention: LDL-C < 1.8 mmol/L.[3,8]
   (b) Secondary prevention: LDL-C < 1.4 mmol/L.[8]

### Statistical analysis

The data were entered onto an Excel spread sheet (Microsoft Corp, Armonk, NY, USA) and analysed using descriptive statistical methods. The Shapiro–Wilk test was used to assess whether data was normally distributed. The data are presented as mean and standard deviation if normally distributed or median and range if not normally distributed. Multiple linear regression analysis was done to assess the relationship of $HbA_{1c}$ to the individual lipid parameters.

### Ethics

Permission for the study was granted by the medical Human Research Ethics Committee of the University of the Witwatersrand (Ref: M220404).

## Results

The guidepost study enrolled a total of 261 patients. Duplicated patients, and patients with no lipid results, were excluded and a total of 229 patients were included in this study. The mean age was 59 years (SD ± 11) and 59% ($n$ = 136) were female. Patient demographics are summarized in Table 1.

Data on comorbidities were available for 221 patients, of whom 83% ($n$ = 184/221) were found to be hypertensive. Data on body mass index (BMI) was available for 167 patients of whom 67% ($n$ = 95/167) were found to be obese.

## Diabetes

The mean $HbA_{1c}$ at the time of lipid testing was 10.8% (SD ± 1.9). The maximum $HbA_{1c}$ recordable in our laboratory is 15.4%. Eight of the patients had an $HbA_{1c}$ above 15.4% and this could therefore affect the mean value. From the study cohort 86% ($n$ = 197) of patients were on human insulin as well as metformin, 13% ($n$ = 30) of patients were on human insulin alone, one patient was on human insulin and a sulphonylurea, and only one patient was on oral hypoglycaemic agents alone.

## Dyslipidaemia

The prevalence of dyslipidaemia was 93.01% ($n$ = 213/229). At the time of the study, the laboratory calculated LDL-C using the Friedewald equation. In 7 patients the TG were > 4.5 mmol/L and therefore the LDL-C could not be calculated. The TG levels in these patients were between 5.32 and 7.92 mmol/L.

There were 6.55% ($n$ = 15/229) of PLWD with documented ASCVD as evidenced by a previous myocardial infarction or cerebrovascular accident. These patients were classified into the secondary prevention group. The rest of the 93.45% ($n$ = 214/229) of patients were classified into the primary prevention group. Of note, no imaging or further

**Table 1:** Patient demographics

| **Total number of patients** | | **229** |
|---|---|---|
| Gender | | |
| | Males *n* (%) | 93 (41%) |
| | Females *n* (%) | 136 (59%) |
| Age (years) | | |
| | Mean ± SD | 59 ± 11 |
| Body mass index (kg/m$^2$) | | |
| | *N* | 167 |
| | Mean ± SD | 31.32 ± 6,69 |
| | Underweight (< 18.5 kg/m$^2$) *n* (%) | 3 (2%) |
| | Normal (18.5–24.9 kg/m$^2$) *n* (%) | 25 (15%) |
| | Overweight (25–29.9 kg/m$^2$) *n* (%) | 44 (26%) |
| | Class 1 obesity (30–34.9 kg/m$^2$) *n* (%) | 46 (28%) |
| | Class 2 obesity (35–39.9 kg/m$^2$) *n* (%) | 34 (20%) |
| | Class 3 obesity (> 40 kg/m$^2$) *n* (%) | 15 (19%) |
| Duration of diabetes (years) | | |
| | *N* | 148 |
| | median (IQR) | 14 (9–20) |
| Creatinine clearance* | | |
| | *N* | 213 |
| | Males (mL/min) median (IQR) | 100 (76.65–122.55) |
| | Females (mL/min) median (IQR) | 95 (67.58–121.46) |

*Creatinine clearance calculated using the Cockcroft–Gault formula.

investigation was done to determine the presence of undiagnosed ASCVD.

The lipid profiles of the two groups of patients are shown in Tables 2 and 3. Across all groups, the commonest lipid abnormality was an LDL-C greater than target. In the primary prevention group, 61.84% of females and 60.47% of males did not achieve the target LDL-C < 1.8 mmol/L. In the secondary prevention group, 66.67% of females and 83.33% of males did not achieve the target LDL-C < 1.4 mmol/L. Figures 1 and 2 show the percentage of dyslipidaemia in patients in the primary prevention group for females and males respectively. Only 15.7% ($n = 19/121$) of females and 16.47% ($n = 14/85$) of males had all 3 parameters (TG, HDL-C, and LDL-C) at target.

Amongst the obese patients (BMI ≥ 30 kg/m$^2$) who had LDL-C levels available, 67.03% ($n = 61/91$) of patients did not meet LDL-C targets. Hypothyroidism was present in 13 patients, of whom 5 patients were biochemically hypothyroid despite treatment (TSH levels > 5.5 mIU/L). Of these five patients, four did not meet the LDL-C target. Other secondary causes of dyslipidaemia were not assessed.

Statin therapy was prescribed for 86% ($n = 197$) of patients. Doses of statin therapy and lipid targets are presented in Table 4. Statins were not prescribed for 14% ($n = 32$) of patients. Of these patients, 2 patients' LDL-C could not be calculated, and 16 patients had an LDL-C above target. Therefore, statin treatment was warranted in these 16 patients. In the group receiving simvastatin 40 mg, 60.98% ($n = 25/41$) were on amlodipine 10 mg. Due to drug interactions, the maximum dose of simvastatin that should be prescribed with amlodipine is 20 mg.[7] From the study cohort, one patient was on a fibrate and a statin and one patient was on a fibrate alone. Both these patients did not reach TG or LDL-C targets.

**Table 2:** Lipid profile of patients with no proven ASCVD (primary prevention)

| **Lipids** | **Females** | | **Males** | |
|---|---|---|---|---|
| | **Mean ± SD** | **% not at target** | **Mean ± SD** | **% not at target** |
| TC mmol/L | 4.37 ± 1.1 | 30.99 | 3.79 ± 0.92 | 17.24 |
| HDL mmol/L | 1.29 ± 0.35 | 54.72 | 1.08 ± 0.29 | 43.02 |
| LDL mmol/L* | 2.27 ± 0.89 | 61.84 | 1.96 ± 0.67 | 60.47 |
| | **Median (IQR)** | **% not at target** | **Median (IQR)** | **% not at target** |
| TG mmol/L | 1.63 (1.06–2.32) | 40.19 | 1.42 (0.97–2) | 31.03 |

TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol. *LDL-C target < 1.8 mmol/L.

**Table 3:** Lipid profile of patients with proven ASCVD (secondary prevention)

| **Lipids** | **Females** | | **Males** | |
|---|---|---|---|---|
| | **Mean ± SD** | **% not at target** | **Mean ± SD** | **% not at target** |
| TC mmol/L | 4.07 ± 1.14 | 33.33 | 3.68 ± 0.52 | 16.67 |
| HDL mmol/L | 1.10 ± 0.24 | 66.67 | 0.97 ± 0.03 | 66.67 |
| LDL mmol/L* | 2.17 ± 1.07 | 66.67 | 1.67 ± 0.71 | 83.33 |
| | **Median (IQR)** | **% not at target** | **Median (IQR)** | **% not at target** |
| TG mmol/L | 1.45 (1.26–2.39) | 33.33 | 1.93 (1.69–2.5) | 66.67 |

TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol.
*LDL-C target < 1.4 mmol/L.

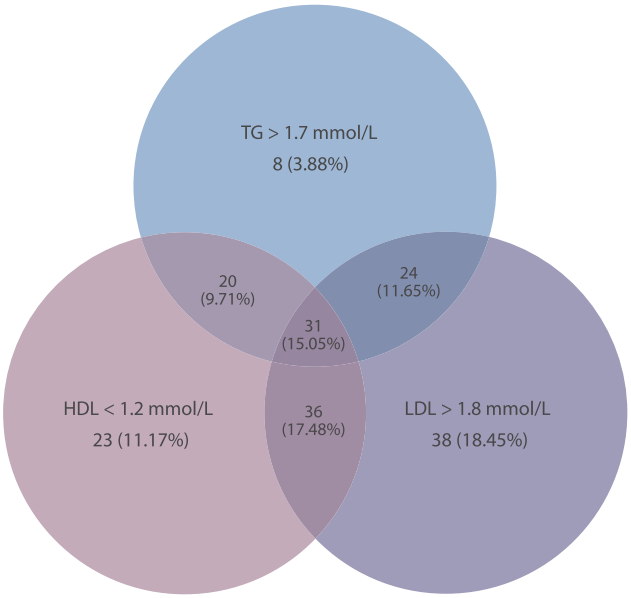


**Figure 1:** Percentage of females in the primary prevention group not achieving lipid targets.

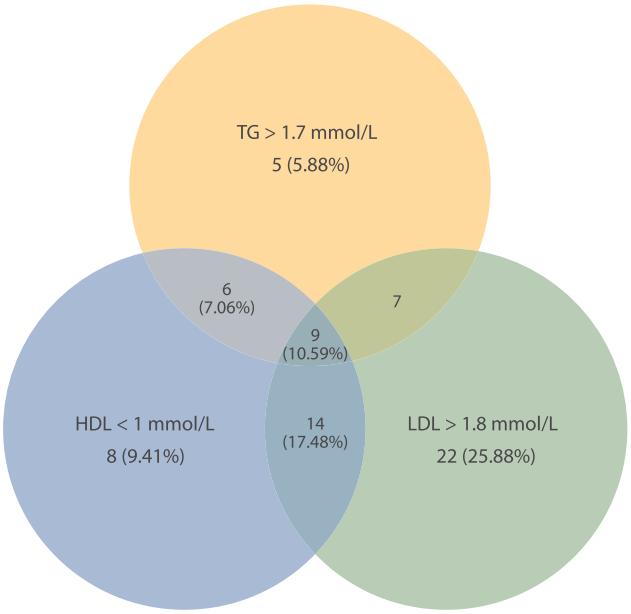


**Figure 2:** Percentage of males in the primary prevention group not achieving lipid targets.

Multiple linear regression models were generated in order to determine whether $HbA_{1C}$ correlated with any of the lipid parameters. There were no significant correlations between $HbA_{1C}$ and any of the lipid parameters. The results are summarized in Table 5.

## Discussion

ASCVD is the leading cause of death in PLWD.[4] In addition to glycaemic control, other modifiable risk factors for ASCVD including hypertension, smoking, and obesity should be addressed.[3] In this study of patients with uncontrolled diabetes, 67% of patients were obese, and of these patients, most did not achieve LDL-C target goals. This is concerning for the prevention of ASCVD.

**Table 4:** Statin therapy and LDL-C targets

| Dose | | Simvastatin | Atorvastatin |
|---|---|---|---|
| 10 mg | Total number of patients receiving dose | 3 | 1 |
| | Number not achieving target LDL-C for primary prevention | 2 (66%) | 1 (100%) |
| 20 mg | Total number of patients receiving dose | 77 | 22 |
| | Number not achieving target LDL-C for primary prevention | 35 (51.47%) | 15 (68.18%) |
| | Number not achieving target LDL-C for secondary prevention | 6 (66.67%) | |
| 40 mg | Total number of patients receiving dose | 41 | 40 |
| | Number not achieving target LDL-C for primary prevention | 25 (65.79%) | 29 (74.36) |
| | Number not achieving target LDL-C for secondary prevention | 2 (66.67%) | 1 (100%) |
| 80 mg | Total number of patients receiving dose | | 13 |
| | Number not achieving target LDL-C for primary prevention | | 7 (70%) |
| | Number not achieving target LDL-C for secondary prevention | | 2 (100%) |

The prevalence of diabetic dyslipidaemia globally is 72–85%.[4] Our study found a higher prevalence of diabetic dyslipidaemia, which was similar to the 2012 Helen Joseph study (93% prevalence).[9] The reason for the higher prevalence could be multifactorial, including poor socioeconomic setting and poor glycaemic control.

Data obtained from Helen Joseph Hospital in 2012 showed that 76% of PLWD did not achieve the guideline specified LDL-C target on simvastatin alone.[9] A study done in 2017 at Charlotte Maxeke Academic Hospital (a tertiary South African Hospital) showed that 73.5% of PLWD did not achieve SA LDL-C targets on either simvastatin or atorvastatin.[10] Using the same targets, 61.84% of females and 60.47% of males in the primary prevention group did not achieve LDL-C targets. This shows that a higher percentage of patients had achieved targets in our study, which is possibly due to the availability of atorvastatin at the diabetic clinic.

Lifestyle intervention focusing on weight loss, increasing physical activity, and healthy eating habits should be instituted for all PLWD.[3,8] In addition, for primary prevention, the ADA guidelines recommends initiation of moderate-intensity statin therapy (simvastatin 20–40 mg, atorvastatin 10–20 mg, or rosuvastatin 5–10 mg) for PLWD who are ≥ 40 years of age.[8] High-intensity statin therapy (atorvastatin 40–80 mg or rosuvastatin 20–40 mg) is recommended for patients with additional ASCVD risk factors.[8] For secondary prevention in PLWD who have established ASCVD, high-intensity statin therapy is recommended.[8] The risk of side effects increases with higher doses of simvastatin. Patients who do not reach LDL-C targets

Table 5: Correlations between $HbA_{1C}$ and lipid parameters

| Item | $HbA_{1C}$ / TC | $HbA_{1C}$ / TG | $HbA_{1C}$ / HDL-C | $HbA_{1C}$ / LDL-C |
|---|---|---|---|---|
| *F* | 3.08 | 2.6 | 0.60 | 4.08 |
| Significance *F* (*p*-value) | 0.08 ($p > 0.05$) | 0.11 ($p > 0.05$) | 0.44 ($p > 0.05$) | 0.06 ($p > 0.05$) |
| $r^2$ | 0.01 | 0.01 | 0.01 | 0.02 |
| Multiple *R* | 0.11 | 0.11 | 0.05 | 0.13 |

with 40 mg simvastatin, or are on other drugs that interact with simvastatin (e.g., amlodipine or anti-retroviral therapy), should be switched to atorvastatin.[7]

Statin therapy was prescribed in a significant (86%) number of patients. High-intensity statins (atorvastatin 40–80 mg) can decrease LDL-C by up to 50%.[4] However, only 26.9% ($n = 53/197$) were on a high-intensity statin, with only 6.59% of patients being on a maximum dose of atorvastatin. This implies that there is significant clinical inertia in dose titration of statins to reach lipid targets. The CEPHEUS SA study showed that 72.2% of patients were still on the same lipid-lowering drug as when first prescribed pharmacotherapy, 63.5% of all patients were still taking the initial starting dose, while the dose had been increased in 8.7% of patients.[11] Clinical inertia is a severe limitation to treatment, and education of doctors needs to be addressed.

Statin treatment was indicated for 16 of the 32 patients who were not on treatment. This is concerning as these patients are at high CVS risk. The reason for statins not being prescribed was not assessed in this study.

If the target LDL-C is not achieved with maximum tolerated statin therapy, addition of ezetimibe or a PCSK9 inhibitor is recommended. In the IMPROVE-IT study ezetimibe in combination with simvastatin was shown to decrease LDL-C.[12] Ezetimibe is restricted to a named patient basis at select lipid clinics in South Africa and is not available at Helen Joseph Hospital.

Patient factors contributing to poor lipid control may include poor education regarding lipid targets and the consequences of dyslipidaemia, suboptimal adherence to medication, and the side effect profile of statins. Clinician factors may include unawareness of lipid targets, clinical inertia, concerns about drug side effects, and failure to monitor lipids and titrate to target.[11] In the state sector, staff and medication limitation or shortage plays a role in suboptimal management of patients.

### *Limitations*

All required data were not recorded for every patient. Duration of statin therapy, side effects and compliance with statin therapy, as well as secondary causes for dyslipidaemia, were not assessed. These could have been confounding factors in patients not achieving lipid targets. Absolute LDL-C targets were assessed and reduction in baseline was not assessed as baseline LDL-C data were not available. Adherence to treatment and reasons for potential non-adherence were not addressed in this study.

The presence of underlying ASCVD was not adequately established. Patients with a previous MI or CVA were classified as requiring a lower LDL-C target. However, no imaging was done on the rest of the patients. The ESC guidelines recommend coronary artery calcium scores, assessment of atherosclerotic plaques (of the carotid and femoral vessels), and testing of Lp(a) to assess ASCVD risk.[13] However, in a resource-limited setting such as South Africa, these tests are not performed. Therefore, patients who had ASCVD (and who require lower LDL-C targets) could have been underestimated.

## Conclusion

The results of this study highlight the suboptimal management of dyslipidaemia in T2DM patients at a specialised diabetic centre in South Africa. The percentage of patients on statins was high. However, the dose and statin intensity were not escalated appropriately to achieve the target LDL-C. In addition, the lack of high-potency statins and second-line therapy contributes to poor management of patients. Ultimately, a combination of patient and clinician factors, together with poor resources, contributes to lipid targets not being achieved. Further prospective studies are required to assess to assess factors affecting optimal lipid management.

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## ORCID

*A Parak* http://orcid.org/0000-0001-7464-8860

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