

Original Article

Role of bempedoic acid in secondary prevention of atherosclerotic cardiovascular disease patients: A prospective observational study

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ABSTRACT

Background and Objective: Atherosclerotic cardiovascular disease (ASCVD) is one of the leading causes of global mortality. Many patients with ASCVD fail to achieve the target lipid profile despite being on maximally tolerated statins. Bempedoic acid (BA) is a novel oral adenosine triphosphate-citrate lyase inhibitor, introduced as an effective therapy for managing hyperlipidaemia. This study evaluates its efficacy and safety as an add-on therapy in ASCVD patients. The objective of the study is to evaluate the efficacy and safety of BA as an add-on therapy to maximally tolerated statin therapy in patients with ASCVD for secondary prevention.

Material and Methods: The study was a prospective, observational study conducted at King George's Medical University, Lucknow. We included 113 patients with ASCVD between 18 and 70 years old. All patients were already on their maximum tolerated statin doses with low-density lipoprotein (LDL) levels >55 mg/dL. BA (180 mg once daily) was added to all ASCVD patients to achieve target LDL goals (as per European Society of Cardiology [ESC] guidelines 2019). The following biochemical parameters were evaluated: lipid profile, kidney and liver function tests; creatine kinase (CK); serum uric acid; estimated glomerular filtration rate (eGFR); haemoglobin A1c (HbA1c), at baseline and at 12 weeks.

Results: All the enrolled patients completed the full 12-week follow-up. Comparing from baseline, after 12 weeks, the mean LDL cholesterol dropped significantly from 93.21 to 64.22 mg/dL (30% reduction), total cholesterol, triglycerides, non-high-density lipoprotein (HDL) cholesterol also decreased significantly from 187 to 147 mg/dL (20% reduction), 153 to 139 mg/dL (8% reduction), 138 to 92 mg/dL (32% reduction), respectively. HDL (good cholesterol) also increased by 11%, which is beneficial. There was a small but statistically significant drop in HbA1c levels (1.9% reduction) also in 65 ASCVD patients who were diabetic, indicating improvement in glycaemic control. Kidney and liver function remained mostly stable. There was a significant increase in creatinine levels by 7% and a drop in eGFR by 6%. Uric acid was increased by 9.2%. There were no significant changes in CK at the end of 12 weeks.

Conclusion: Adding BA to maximally tolerated statin therapy in ASCVD patients led to better control of LDL cholesterol and other lipid parameters without major side effects. It was generally well-tolerated and showed potential as a helpful option for patients who have not reached their cholesterol goals with statins alone. These findings support the use of BA in real-world settings as part of secondary prevention strategies for heart disease.

Keywords: Atherosclerotic cardiovascular disease, Bempedoic acid, Dyslipidaemia, Low-density lipoprotein-cholesterol, Secondary prevention, Statins

INTRODUCTION

Atherosclerotic cardiovascular disease (ASCVD) is the leading cause of global mortality, responsible for about one-third of all deaths. It results from arterial plaque buildup and manifests as coronary heart disease, cerebrovascular disease, peripheral artery disease and aortic atherosclerosis.^[1] Prevention strategies include primary prevention to delay onset and secondary prevention to reduce recurrent events.^[2] Among modifiable risk factors, hyperlipidaemia, particularly elevated low-density lipoprotein cholesterol (LDL-C), is central, with recent Indian studies showing hypercholesterolaemia in 25–30% of urban and 15–20% of rural populations.^[3,4]

Statins remain the cornerstone of LDL-C lowering, yet their use is limited by intolerance (10–15% in real-world settings) and the inability of maximally tolerated doses to consistently achieve guideline-recommended targets.^[3] The term ‘Maximally tolerated statin therapy’ refers to the highest intensity statin regimen that a patient can continue without adverse effects.^[5] The 2018 AHA/ACC guidelines recommend <70 mg/dL for high-risk ASCVD patients, while the 2019 European society of cardiology/ European atherosclerosis society (ESC/EAS) guidelines recommend ≥50% reduction from baseline and targets <55 mg/dL for very high-risk groups.^[3] Despite additional options such as ezetimibe and PCSK9 inhibitors, many patients fail to achieve these goals, highlighting the need for novel, well-tolerated adjunct therapies to further reduce residual cardiovascular risk.^[6,7]

Bempedoic acid (BA) is a novel, oral, once-daily, adenosine triphosphate-citrate lyase (ACL) inhibitor, developed as a promising option for managing hyperlipidaemia.^[8]

Mechanism of action

BA is a pro-drug that remains inactive and gets converted into its active form, bempedoyl-coenzyme (bempedoyl CoA or ETC-1002-CoA) by a substrate-specific isozyme acyl-CoA synthetase (ACSVL1). In the cholesterol biosynthesis pathway, bempedoyl-CoA acts as a competitive inhibitor of ACL, which acts upstream of HMG-CoA reductase, differentiating it from other lipid-lowering therapies.^[9] Inhibition of ACL leads to a decrease in cholesterol synthesis in the liver and also increases the expression of LDL receptors that resulting in enhanced clearance of LDL-C from the bloodstream.^[10]

An important key feature of BA that differentiates it from statins is its liver-specific action. This is due to the enzyme ACSVL1, which is expressed in the liver and not in muscle or other peripheral tissues, that activates BA and converts it into its active form.^[10] Hence, the action of BA is primarily limited to the liver, reducing the risk of statin-like muscle-related side effects.^[11] In addition, BA has high bioavailability due

to its small size and rapid intestinal absorption.^[3] Clinical studies have shown that when BA is treated for 12 weeks in patients, it is significantly effective in reducing LDL-C levels while maintaining a good safety profile.^[5] Improved lipid parameters were observed when BA was given as a standalone treatment or in combination with other lipid-modifying therapies.^[12] Unlike statins which have shown ability to induce new-onset diabetes mellitus (NODM), in high-risk individuals prone to diabetes: Obesity, females, elderly age group, Asian descent and those with pre-diabetes or metabolic syndrome, in previous clinical studies;^[13] BA has demonstrated significant reductions in haemoglobin A1c (HbA1c) by –0.12% and –0.06% with no worsening of fasting blood sugar levels in diabetic or pre-diabetic patients. It showed a significant and consistent lowering of LDL-C with no role in worsening of glycaemic variables or increasing the incidence of NODM.^[14] Furthermore, BA used in hypercholesterolaemic patients resulted in a lower risk of cardiovascular events and diabetes mellitus.^[15]

The present study was undertaken because many ASCVD patients fail to achieve target LDL-C levels despite receiving maximally tolerated statin therapy, leaving them at elevated risk for future cardiovascular events.^[3] BA represents a potential adjunctive option in such patients. This study, therefore, evaluates its efficacy in further lowering LDL-C when added to statins in an ASCVD cohort from Northern India. The findings are expected to provide valuable insights for optimising secondary prevention strategies and improving the quality of care in this high-risk population.

MATERIALS AND METHODS

Study design

The study was a prospective observational study, conducted in the Department of Pharmacology in collaboration with the Department of Cardiology and the Department of Neurology, King George’s Medical University, Lucknow. Institution’s ethics committee approval was obtained. All ASCVD patients attending Cardiology and Neurology out-patient departments (OPDs), receiving BA plus Statin as part of lipid-lowering therapy and satisfying inclusion criteria, were enrolled, and written informed consent was obtained.

Duration of study

The study duration was 12 months.

Tool used in the study

A case report form designed as per the study protocol was filled out according to the prescription of the patient. It included sociodemographic details and clinical variables such as the patient’s name, age, sex, personal history, family

history, associated comorbidities, side effect profile including clinical and biochemical parameters, investigations related to diagnosis, ongoing drugs and concomitant medications. Data were collected on the basis of enrolled patients satisfying the inclusion criteria, who received BA. The prescribed dose was 180 mg OD for all the enrolled cases.

Biochemical analysis

Further in this study, clinical laboratory samples for the analysis of primary end points, i.e., basic fasting lipids (total cholesterol [TC], calculated LDL-C, high-density lipoprotein-cholesterol (HDL-C), non-HDL-C and triglycerides [TGs]), HbA1c levels were collected at the screening visit (Day 1) and week 12. Secondary end points included safety and tolerability of BA, which was assessed by evaluating results from clinical laboratory tests, including serum uric acid levels, serum creatinine, alanine and aspartate aminotransferase levels, and creatinine kinase levels on the screening day (day 1) and at week 12. The relationship was assessed between all these parameters and BA.

Inclusion criteria

All the patients of ASCVD aged 18–70 years, attending OPD in the Cardiology and Neurology department, KGMU. Patients receiving stable maximally tolerated or the maximally defined statin dose required additional LDL-C lowering. Patients taking a stable or the same dose of statins for the last 4–8 weeks before enrolling in the study. No history of acute cerebrovascular and cardiovascular emergencies within 1 month of study or any other disease interfering with the study. Patients having LDL-C levels >55 mg/dL at the time of screening.

Exclusion criteria

Patients who were not willing to provide written informed consent. Pregnant or lactating female patients or those of childbearing age not using an adequate method of birth control. History of acute cerebrovascular and cardiovascular emergencies, including recent (within 1 month) before the screening visit-myocardial infarction (MI), unstable angina leading to hospitalisation, uncontrolled, symptomatic cardiac arrhythmia (or medication for an arrhythmia that was started or dose changed within 1 month of screening), cerebrovascular accident, transient ischaemic attack. Patients were excluded from the study if they had experienced or planned to undergo any surgical procedure or medical intervention in the cardiovascular/cerebrovascular/endovascular/part for peripheral vascular disease within 1 month before screening or during the study duration. (e.g., percutaneous coronary intervention), coronary artery bypass grafting, surgery or stenting, carotid or peripheral revascularisation). Uncontrolled hypertension, defined as

sitting systolic blood pressure ≥ 160 mmHg and/or diastolic blood pressure ≥ 100 mmHg measured according to local standards. Uncontrolled hypothyroidism; liver disease or dysfunction with deranged liver enzymes (3-fold rise in upper limit of normal [ULN] range). Renal dysfunction (estimated glomerular filtration rate [eGFR] 3 times the ULN range or glomerular filtration rate [GFR] <30 mL/min) or chronic kidney disease patients on dialysis. Gastrointestinal conditions or procedures that may affect drug absorption were done within 3 months of the study at the time of screening/day 1. Haematologic or coagulation disorders; active malignancy; unexplained creatine kinase (CK) elevation >3 times the ULN at the time of screening/day 1. Any additional lipid-lowering agent other than statins, within 4 months before screening.

Sample size calculation

The sample size was calculated based on 'CLEAR Outcome Trials' [5,8,12,16] assuming a minimum 4% change in LDL before and after treatment, and an expected population standard deviation (SD) to be 15% and a 99% confidence interval. Keeping a provision of data loss at 10% the proposed sample size was 113.

Statistical analysis

Categorical variables were presented in numbers and percentages (%). Continuous variables were presented as mean and SD. Descriptive statistics (Mean \pm SD, Frequency [%]) were used to depict baseline characteristics of the study population. Paired *t*-test was used to assess pre- and post-change in clinical variables. The Shapiro–Wilk test was used to assess the normality of data at baseline. The Shapiro–Wilk $P > 0.05$ data distribution was considered normal. The data were entered in MS-Excel, and analysis was done using the Statistical Package for the Social Sciences (SPSS 24.0) software. $P < 0.05$ has been taken as statistically significant.

Study flowchart

Patients with ASCVD on maximally tolerated doses of statins and baseline LDL >55 mg/dL were enrolled from Cardiology and Neurology OPD after satisfying inclusion criteria and written informed consent to receive Bempedoic acid plus statin. Baseline lipid profile, HbA1c, and other biochemical parameters were evaluated, followed by repeat evaluation at 12 weeks to assess efficacy, safety and proportional changes from baseline [Figure 1].

RESULTS

296 patients were screened, of whom 113 ASCVD patients were enrolled. All completed the 12-week follow-up period. In baseline demographics, the mean age of the patients was 55.16 ± 11.35 years, and the male/female ratio was 84/29,

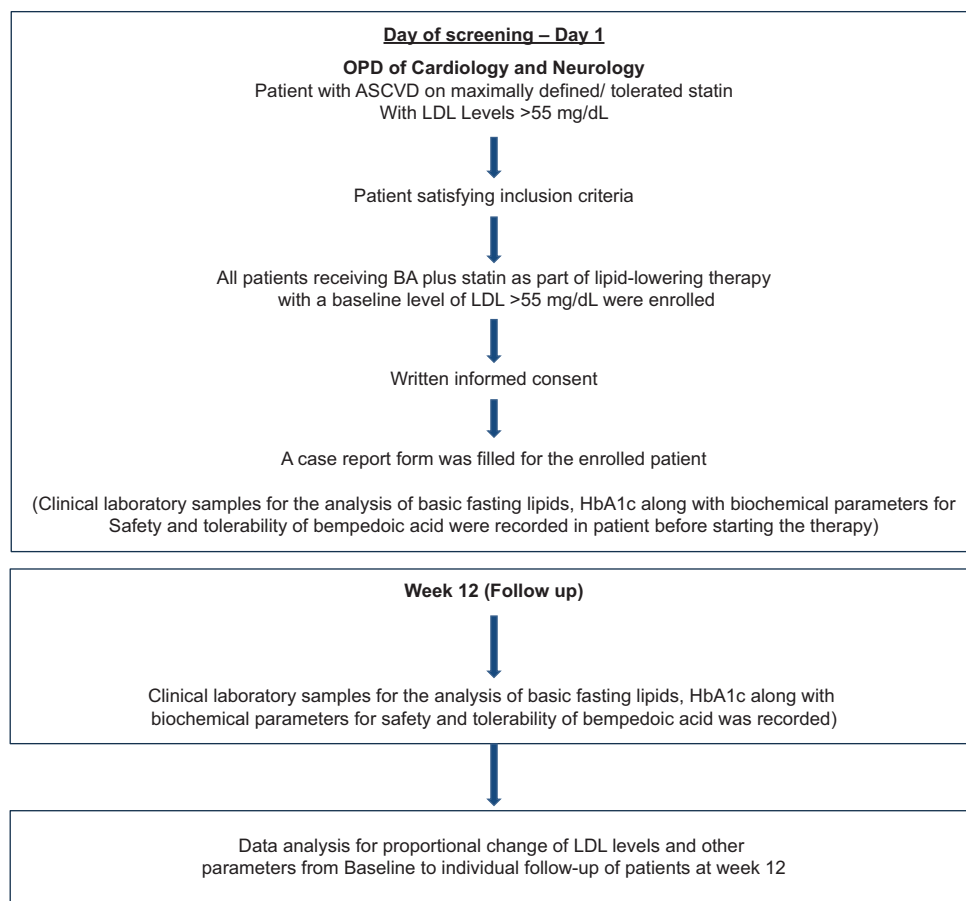


Figure 1: Study flowchart. OPD: Outpatient Department, ASCVD: Atherosclerotic Cardiovascular Disease, LDL: Low Density Lipoprotein

eGFR category (mL/min/1.73 m ²)	Number of patients at baseline (%)	Number of patients at week 12 (%)
>90	35 (31.53)	25 (22.52)
60–89	59 (53.15)	66 (59.46)
45–59	17 (15.32)	18 (16.22)
30–44	–	2 (1.80)

eGFR: Estimated glomerular filtration rate, ASCVD: Atherosclerotic cardiovascular disease

indicating a higher prevalence of ASCVD among males in the study population. 65 patients were diabetic, and the following was eGFR distribution among ASCVD patients.

At baseline, most patients (84.68%) had normal to mildly decreased kidney function (eGFR \geq 60). Overall, renal function remained largely stable in the majority, with only minor downward shifts seen [Table 1].

Lipid profile assessment

These results indicate a progressive and statistically significant improvement in all lipid parameters over 12 weeks of therapy [Table 2].

Changes in other laboratory parameters

For liver function tests (LFTs), none of the above changes were statistically significant ($P > 0.05$), suggesting no hepatotoxic effect of BA in combination with statins.

For renal parameters, findings indicate a mild but statistically significant increase in serum creatinine and uric acid, accompanied by a modest decline in eGFR. However, serum urea did not change significantly.

CK levels remained stable with no statistically significant changes. This suggests no significant muscle toxicity attributable to BA therapy over 12 weeks [Table 3].

Changes in HbA1c levels

Table 4 compares the effect of BA (180 mg) added to statin therapy on HbA1c levels in ASCVD patients with and without diabetes over 12 weeks.

In non-diabetic patients ($n = 48$), there was no significant glycaemic effect of BA. No patient developed NODM since HbA1c levels were in the normal range for all patients at week 12.

Table 2: Assessment of baseline and follow-up lipid profile parameters of ASCVD patients over 12 weeks.

Lipid profile	Baseline (mean±SD)	Week 12 (mean±SD)	Change (mean±SD)	% change (mean±SD)	P-value
TC (mg/dL)	187.70±36.61	147.01±23.87	-40.68±18.90	-20.90±6.94	<0.001
HDL (mg/dL)	49.27±11.10	54.48±12.55	+5.20±4.99	+11.04±11.42	<0.001
LDL (mg/dL)	93.21±30.14	64.22±20.38	-28.98±15.26	-30.51±8.77	<0.001
Non-HDL (mg/dL)	138.43±35.89	92.53±25.37	-45.89±18.93	-32.77±10.12	<0.001
TG (mg/dL)	153.66±48.49	139.55±40.73	-14.11±17.11	-8.08±10.72	<0.001

Paired t test was used to evaluate pre- (baseline) and post- changes in clinical variables (at week 12) in same group. P value <0.05 is considered Statistically significant. ASCVD: Atherosclerotic cardiovascular disease, TC: Total cholesterol, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, TG: Triglycerides, SD: Standard deviation

Table 3: Assessment of laboratory parameters in ASCVD patients.

Variable	Baseline (Mean±SD)	Week 12 (Mean±SD)	Change (mean±SD)	% Change (mean±SD)	P-value
Serum bilirubin total (mg/dL)	0.69±0.27	0.71±0.29	0.02±0.28	2.9±40.61	0.058
Serum bilirubin direct (mg/dL)	0.26±0.09	0.27±0.12	0.01±0.11	3.85±40.79	0.071
Serum bilirubin indirect (mg/dL)	0.430±0.27	0.431±0.31	0.001±0.29	0.23±67.60	0.9158
SGOT (IU/L)	28.83±16.27	30.42±11.40	1.59±14.05	5.52±48.73	0.062
SGPT (IU/L)	34.73±14.24	36.02±15.45	1.29±14.86	3.71±42.78	0.064
ALP (IU/L)	89.86±36.37	93.30±48.68	3.44±42.86	3.83±47.64	0.059
Serum urea (mg/dL)	31.08±8.20	32.30±7.74	1.22±7.97	3.93±25.65	0.057
Serum creatinine (mg/dL)	1.03±0.23	1.11±0.21	0.08±0.22	7.77±21.36	<0.001*
Uric acid (mg/dL)	5.10±1.21	5.57±1.43	0.47±1.32	9.22±25.84	<0.001*
eGFR (mL/min/1.73 m ²)	79.92±17.79	74.60±16.96	-5.32±17.38	-6.66±21.75	<0.001*
Creatine kinase (U/L)	113.16±55.07	114.70±52.57	1.54±53.86	1.36±47.56	0.241

Paired t test was used to evaluate pre- (baseline) and post- changes in clinical variables (at week 12) in same group. *Statistically significant (<0.05). SD: Standard deviation, ASCVD: Atherosclerotic cardiovascular disease, SGOT: Aspartate aminotransferases, SGPT: Serum alanine aminotransferases, ALP: Alkaline phosphatase, eGFR: Estimated glomerular filtration rate

Table 4: Change in HbA1c levels based on type 2 DM status in ASCVD patients.

DM status	Number of patients	Baseline Mean±SD	Week 12 Mean±SD	Change (0-12) (mean±SD)	% Change (mean±SD)	P-value
No	48	5.25±0.52	5.21±0.46	-0.04±0.26	-0.601±5.06	0.24
Yes	65	8.42±1.24	8.25±1.23	-0.16±0.30	-1.90±3.73	<0.001

Paired t test was used to evaluate pre- (baseline) and post- changes in clinical variables (at week 12) in same group. p value <0.05 is considered Statistically significant. ASCVD: Atherosclerotic cardiovascular disease, DM: Diabetes mellitus, SD: Standard deviation, HbA1c: Haemoglobin A1c

In diabetic patients ($n = 65$), mean HbA1c reduction was statistically significant, suggesting that BA has a favourable impact on glycaemic control in diabetics.

DISCUSSION

Managing ASCVD in high-risk patients remains a significant challenge, particularly in secondary prevention. The INTERHEART study confirmed that LDL-C is a critical risk factor among Indians, comparable to other ethnic groups.^[17] Although statins are the cornerstone of lipid-lowering therapy, many patients fail to achieve target LDL-C levels due to statin intolerance and muscle-related adverse

effects.^[18] This highlights the need for additional therapies such as BA, PCSK9 inhibitors and ezetimibe, especially as add-on treatments in patients already on maximally tolerated statins.

In our study, 47% of patients achieved the LDL-C target of <55 mg/dL at 12 weeks, as per ESC/EAS 2019 guidelines for very high-risk ASCVD patients, underscoring the effectiveness of BA in this setting. These results are comparable to Indian data, such as Manoj *et al.*, where LDL-C goals (<70 mg/dL) were achieved in 40–50% of patients.^[19]

The most notable finding was the significant LDL-C reduction of 28.98 mg/dL (30.51%) from baseline (93.21 ± 30.14

to 64.22 ± 20.38 mg/dL, $P < 0.001$). This reduction was greater than that reported in the CLEAR Harmony trial (18.1% reduction) and the CLEAR Wisdom trial (17.4% reduction).^[20] Trials in statin-intolerant patients, such as CLEAR Serenity and CLEAR Tranquility, showed LDL-C reductions of 21.4% and 28.5%, respectively.^[16,20]

Other lipid parameters also showed favourable changes. TC decreased by 20.9% (187.70 ± 36.61 to 147.01 ± 23.87 mg/dL, $P < 0.001$), consistent with reductions reported in CLEAR Harmony (15.4%) and CLEAR Tranquility (18.8%). HDL-C increased by 11.04% (49.27 ± 11.10 to 54.48 ± 12.55 mg/dL, $P < 0.001$), a more pronounced rise compared to increases observed in CLEAR Serenity and Tranquility trials (2.3–2.7 mg/dL). Non-HDL cholesterol fell by 32.77% (138.43 ± 35.89 to 92.53 ± 25.37 mg/dL, $p < 0.001$), a reduction comparable to CLEAR Tranquility (~24%). TGs declined by 8.08% (153.66 ± 48.49 to 139.55 ± 40.73 mg/dL, $P < 0.001$), consistent with modest but similar reductions observed in the CLEAR trials.^[20]

Glycaemic outcomes showed an interesting trend. In patients without diabetes ($n = 48$), HbA1c changes were insignificant ($P = 0.24$), consistent with CLEAR Serenity and Tranquility, confirming that BA does not worsen glycaemic control in non-diabetics. In contrast, patients with type 2 diabetes ($n = 65$) experienced a mean HbA1c reduction of 1.9% ($P < 0.001$), suggesting a moderate glycaemic benefit, consistent with Leiter *et al.*, who reported HbA1c reductions over 1 year.^[14] These findings contrast with CLEAR Harmony, where glycaemic effects were neutral or slightly unfavourable and with Manoj *et al.*^[19,20] who reported HbA1c worsening. Importantly, no new-onset diabetes was observed in our study.

Renal safety outcomes revealed statistically significant changes. Serum creatinine increased by 7.7%, eGFR declined by 6.6% ($P < 0.001$), and uric acid increased by 9.92% ($P < 0.001$). These findings are consistent with the CLEAR trials and Indian studies, which reported modest, clinically insignificant renal changes.^[14,16,19-21] Notably, these changes were mild, reversible and did not necessitate discontinuation of therapy. LFTs also remained stable, aligning with CLEAR trials.^[20]

Overall, the safety profile of BA in this study was favourable, with most changes being arbitrate and clinically manageable. These findings reinforce global and Indian evidence that BA is effective, safe and well-tolerated.

Limitations

This single-centre, observational study with a modest sample size may not allow evaluation of long-term outcomes due to the lack of a control arm.

Larger, multicentre prospective studies are required to confirm these findings and establish the long-term safety and utility of bempedoic acid in cardiovascular care.

CONCLUSION

This 12-week study from a Northern Indian cohort shows that BA, when added to maximally tolerated statins in high-risk ASCVD patients, effectively lowered LDL-C and was well-tolerated without major side effects. Diabetic patients showed modest HbA1c reduction, while non-diabetics had no glycaemic impact. Safety outcomes, including liver and muscle enzymes, remained stable. Given limited access to PCSK9 inhibitors, BA offers a practical, cost-effective alternative for residual risk reduction. Larger multicentre studies are needed to confirm long-term cardiovascular benefits.

Ethical approval: The research/study was approved by the Institutional Review Board at King George's Medical University, approval number XXII-PGTSC-IIA/P18, dated 15th December 2023.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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