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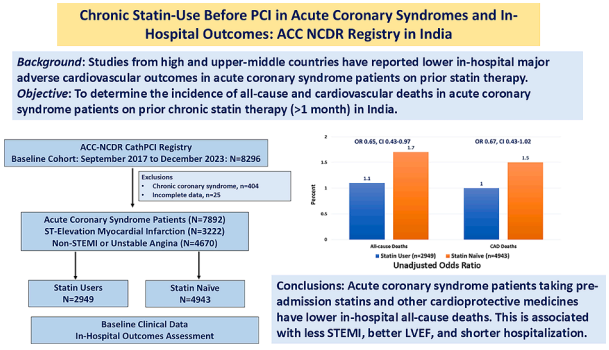
Chronic statin-use before PCI in acute coronary syndromes and in-hospital outcomes: ACC—NCDR registry in India

Rajeev Gupta [a,*], Prashant Dwivedi [b], Krishna K Sharma [c], Sanjeev K Sharma [b], Jitender S Makkar [b], Atul Kasliwal [b], Vishnu Natani [a], Raghubir S Khedar [a], Samin K Sharma [d], Soneil Guptha [e]

[a] Department of Medicine, Eternal Heart Care Centre & Research Institute, Jaipur, India
[b] Department of Cardiology, Eternal Heart Care Centre & Research Institute, Jaipur, India
[c] Department of Pharmacology, LBS College of Pharmacy, Rajasthan University of Health Sciences, Jaipur, India
[d] Department of Cardiology, Mount Sinai Hospital & Icahn School of Medicine at Mount Sinai, NY, NY, USA
[e] Department of Medicine, Rocky Vista University, Ivins, UT, USA

GRAPHICAL ABSTRACT



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ABSTRACT

Objective: To compare in-hospital major cardiovascular adverse outcomes among chronic statin-user and statin-naïve acute coronary syndrome(ACS) patients following percutaneous coronary intervention(PCI).
Methods: Successive patients with ACS who underwent PCI from Sep'17 to Dec'23 were enrolled in a prospective registry. Details of risk factors, presentation, angiography, interventions, and in-hospital outcomes were recorded. Chronic statin use was defined as > 1-month intake before presentation. Primary outcomes were in-hospital all-cause and cardiovascular deaths. Univariate and multivariate odds ratios(OR) and 95 % confidence intervals (CI) were calculated.
Results: 8296 patients were enrolled, and ACS was in 7892(STEMI-ST elevation myocardial infarction 3222, non-STEMI/unstable angina 4670). Prior chronic statin use was in 2949(37.4 %), and 4943(62.6 %) were statin naïve. Statin-user vs. statin-naïve patients were older(62±10 vs. 60±11y), with more hypertension(61 vs. 48 %), diabetes(36 vs. 32 %), prior PCI(20 vs 8 %), CABG(5 vs 2 %), beta-blockers(61.7 vs 8.3 %), anti-platelets(92.8 vs

* Correspondence author at. Department of Medicine, Eternal Heart Care Centre & Research Institute, Jawahar Circle, Jaipur, 302017, India.
E-mail address: drrajeev.gupta@eternalheart.org (R. Gupta).

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5.3 %), and lower mean total-, LDL-, and non-HDL-cholesterol($p < 0.001$); chronic statin users had less STEMI(30 % vs 47 %) and better LVEF(46.5 ± 10 vs 44.5 ± 10 %) at presentation and median hospitalization was shorter (66.3 vs 68.6 h)($p < 0.001$). In statin-user vs. statin-naïve groups, the incidence of all-cause deaths: 33(1.12 %) vs 85(1.72 %) (OR 0.65, CI 0.43–0.97) and CV deaths: in 29(0.98 %) vs 73(1.47 %) (OR 0.67, CI 0.43–1.02) were lower. The ORs attenuated following multivariate adjustments for risk factors, previous treatments, clinical features, angiographic findings and interventions.

Conclusions: Acute coronary syndrome patients taking pre-admission statins and other cardioprotective medicines have lower in-hospital all-cause deaths. This is associated with less STEMI, better LVEF, and shorter hospitalization in prior statin users.

1. Introduction

Statins (HMG-CoA reductase inhibitors) reduce plasma total, low-density lipoprotein (LDL), and non-high-density lipoprotein (non-HDL) cholesterol and are widely used for coronary artery disease (CAD) primary and secondary prevention [1,2]. Statins have pleiotropic effects beyond LDL lowering, including plaque stabilisation and vascular benefits [3]. Large meta-analyses and umbrella reviews have reported that statin use is associated with a significant decrease in cardiovascular disease (CVD) events in primary and secondary prevention populations and decreased incidence of CVD and all-cause deaths in secondary prevention [4]. All the national and international guidelines recommend that statins, particularly moderate to high intensity, should be prescribed to all individuals at intermediate to high risk for primary prevention and to all patients for secondary prevention [1,2,5–7].

Multiple studies from Europe, North America, and East Asia have reported beneficial outcomes of prior statin use in patients with acute coronary events. Only a few studies have reported insignificant benefits [8–15]. South Asian countries harbor the largest population of patients with CAD and dyslipidemias in the world [16,17]. There are no large studies on cardiovascular outcomes in patients taking statins before acute coronary syndrome (ACS), defined as acute ST-elevation myocardial infarction (STEMI), non-STEMI, or unstable angina, in India. We prospectively obtain data of all patients who undergo percutaneous coronary intervention (PCI) at our hospital as part of the American College of Cardiology (ACC) National Cardiovascular Disease Registry (NCDR) CathPCI Registry [18–22]. The present study has been performed to evaluate the association of prior statin use in the ACS patients who underwent PCI with risk factors, clinical presentation, disease severity, and in-hospital outcomes.

2. Methods

This single-center registry-based prospective study has been conducted in India. Our hospital participates in the Cath-PCI Registry of ACC—NCDR Centre of Excellence program [23]. Study protocol has been approved by the institutional ethics committee of the hospital registered with the Government of India (CDSCO Registration No ECR/615/Inst/RJ/2014/RR-20). Informed consent was obtained from each participant in the registry, with specific consent for including anonymized data. The study protocol and all the data are available on the ACC—NCDR website [23].

Patients: Successive patients who underwent PCI at the hospital over 63 months (from September 2017 to December 2023) have been included. Clinical data were prospectively obtained at admission, coronary intervention, and hospital discharge and entered into the NCDR database by research assistants. Details of the methodology have been previously reported [18–22]. Briefly, we obtained details regarding age and sex, risk factors- hypertension, diabetes, dyslipidemias, tobacco use, and chronic kidney disease, previous medication use (specifically aspirin or other antiplatelets, statins, beta-blockers or renin-angiotensin system blockers), other laboratory investigations, clinical presentation (ST-segment myocardial infarction (STEMI), non-STEMI (NSTEMI)/unstable angina or chronic coronary disease. Chronic statin use was defined as the consumption of any statin (rosuvastatin or atorvastatin) at least 30 days prior to admission. Dose of the statin was not inquired. We also obtained data on echocardiographic left ventricular ejection fraction (LVEF), angiographic details of the location and extent of CAD, and the number of stents deployed (>99 % drug-eluting stents, DES).

Details of in-hospital management with a focus on pharmacological vasopressors (noradrenaline, dopamine, vasopressin, etc.), cardiac-support devices (intra-aortic balloon pump (IABP) and miniature ventricular assist devices (mVAD), and post-discharge evidence-based medications were also recorded. Duration of hospitalization and clinical outcomes was available for all the patients. The primary outcomes were in-hospital deaths, either all-cause deaths or cardiovascular deaths. Other in-hospital events were also enumerated and recorded. All the outcomes have been adjudicated using a validated protocol [24].

Statistical analyses: All the data are continuously uploaded to the ACC—NCDR website [22], data were downloaded from the website and transferred to MS Excel worksheets for the present analyses. Data analyses have been performed using SPSS software (Version 23). Continuous variables are reported as mean ± 1 SD and categorical variables as a per cent. Non-normal data are presented as median with 25–75th percentile interquartile (IQR). Inter-group differences have been determined using *t*-test or ANOVA for continuous variables, χ^2 test for categorical variables, and Kruskall-Wallis test for non-normal data. To identify the magnitude of inter-group difference in the in-hospital outcomes, we calculated unadjusted, age-sex adjusted, and multivariate-adjusted (age-sex, risk factors, presentation, left ventricular ejection fraction, extent of CAD, coronary stents, in-hospital therapies including vascular support, and duration of hospitalization) odds ratio (OR) and 95 % confidence intervals (CI) using stepwise logistic regression.

3. Results

8296 successive patients (men 6563, women 1733) have been enrolled in the study period. ACS was in 7892 patients (95.1 %) (STEMI 3222, NSTEMI, or unstable angina 4670). Prior chronic statin use was in 2949 patients (37.4 %), while 4943 (62.6 %) were statin naïve. Demographic, risk factor, and clinical data in statin user and statin naïve groups are in Table 1. Statin-user vs. statin-naïve patients were older (61.7 ± 10 vs. 59.7 ± 11y), with more hypertension (61 vs. 48 %), diabetes (36 vs. 32 %), chronic kidney disease (2.6 vs. 1.8 %), family history of CAD (15.6 vs. 14.1 %), past PCI (19.9 vs 8.2 %), and past CABG (5.2 vs 2.3 %). In chronic statin users, use of other cardioprotective medicines- beta-blockers (61.7 vs 8.3 %) and anti-platelet drugs (92.8 vs 5.3 %)- was also significantly more ($p < 0.001$). The mean total-, non-HDL-, and LDL cholesterol levels were lower in the statin-user group ($p < 0.001$). The prevalence of raised total-, non-HDL- and LDL-cholesterol and triglycerides was also lower ($p < 0.001$). Symptom-to-door time and door-to-balloon times (STEMI) were not significantly different in the two groups. Statin users had less STEMI (30.3 % vs 47.1 %) and more non-STEMI (69.7 vs 52.9 %) ($p < 0.001$). The mean LVEF was higher in prior statin users (46.5 ± 9.9 vs 44.5 ± 10.2 %) ($p < 0.001$). Statin use was significantly greater among the patients with previous PCI and CABG surgery, but more than a third of these patients

were not on statins at presentation.

Angiographic findings and details of interventions and management are in Table 2. In the prior statin-user vs statin-naïve groups, there were insignificant differences in the severity of CAD. In statin users, the left anterior descending artery was less involved. Significantly more stents were deployed in statin-users than statin-naïve patients, while pharmacological vasopressors and mechanical support were significantly more in statin-naïve patients. The median duration of hospitalisation (hours) was lower in the statin-user group (66.3, IQR 51.0–79.6 vs 68.6, IQR 51.4–85.1 h, $p < 0.001$).

In statin-users vs. statin-naïve patients, the incidence of in-hospital all-cause deaths was 33 (1.12 %) vs 85 (1.72 %) (OR 0.65, CI 0.43–0.97), and CAD deaths were 29 (0.98 %) vs 73 (1.47 %) (OR 0.67, CI 0.43–1.02), and (Fig. 1). The odds ratios attenuated following multivariate adjustments for risk factors, prior medications (beta-blockers and antiplatelets), clinical presentation, and interventions (Table 3). The secondary outcomes of recurrent ACS, kidney-related, or infection-related deaths were not significantly different.

4. Discussion

This large prospective coronary intervention registry in India shows that acute syndrome patients (STEMI, NSTEMI/unstable angina) taking statins and other cardioprotective therapies (beta-blockers, antiplatelets) before the event have lower in-hospital all-cause deaths. These patients had lower total, LDL, and non-HDL cholesterol, less STEMI, better LVEF, lesser need for vascular supportive therapy, and shorter in-hospital stay.

Several interventions to prevent adverse outcomes following acute coronary syndrome have been evaluated [25,26]. The most important measures to restore myocardial blood supply are primary PCI, pharmacologic fibrinolysis, or pharmaco-invasive therapy [26]. Other measures to reduce infarct size include oxygenation, beta-blockers, RAAS blockers, and anti-platelet drugs such as aspirin and P2Y12 inhibitors [27]. Loading with a high dose of statins has been used but the outcomes are not uniformly beneficial [28]. High-dose statin pretreatment could benefit due to their pleiotropic properties and modulation of mechanisms involved in the pathogenesis of ACS, including a reduction in inflammation, oxidative stress, endothelial dysfunction, and thrombosis, and have been postulated to reduce periprocedural myocardial infarction and 30-day adverse events in patients undergoing PCI [29,30]. Experiments have demonstrated a reduction in infarct size after myocardial reperfusion in models pre-treated with statins [31,32], but clinical studies have not shown consistently such a beneficial effect among patients with ACS [33], and studies have reported variable results following statin pre-treatment in STEMI before PCI on indices of myocardial perfusion or infarct size in randomized clinical trials [34–39]. We did not load these patients with pre-procedural high-dose statins, and this mechanism is not likely.

Previous studies and meta-analyses from Europe, North America, and East Asia have reported that chronic statin use at baseline is associated with lower in-hospital and long-term CAD events and deaths. A 2011 meta-analysis of 13 randomized trials [40], suggested that high-dose statin pretreatment led to a significant reduction in periprocedural myocardial infarction and 30-day adverse events in patients undergoing percutaneous coronary intervention. On the other hand, a 2020 meta-regression analysis [41], of 26,497 patients reported a decreasing trend in the risk of MCE between 30 days and 12 months in patients on prior high-intensity statin therapy. Furthermore, the more recent publication of a retrospective analysis of prior (>6 months) statin therapy suggested better in-hospital clinical outcomes in patients on statins with STEMI undergoing PCI than those without prior statins

Table 1
Risk factors and clinical characteristics among statin-user and statin-naïve groups at presentation.

Variable	Total cohort ($N = 7892$)	Statin User group ($N = 2949$)	Statin Naïve Group ($N = 4943$)	Chi-square statistic	P Value
Age (mean+SD, years)	60.4 ± 10.9	61.7 ± 10.5	59.7 ± 11.1	63.6	<0.001
Age <50 years	1194(15.1)	353(12.0)	841(17.0)	36.6	<0.001
Men (%)	6231(79.1)	2273(77.1)	3958(80.1)	9.97	0.002
Risk factors					
• Hypertension	4171(52.9)	1788(60.6)	2383(48.2)	114.3	<0.001
• Diabetes	2611(33.1)	1051(35.6)	1560(31.6)	13.9	<0.001
• Cholesterol ≥170mg/dl	2913(36.9)	831(28.2)	2082(42.1)	154.1	<0.001
• Non-HDL cholesterol ≥100mg/dl	4814(61.0)	1530(51.9)	3284(66.4)	164.5	<0.001
• LDL cholesterol ≥70mg/dl	5582(70.7)	1861(63.1)	3721(75.3)	132.2	<0.001
• Triglyceride≥150 mg/dl	3166(40.1)	1128(38.3)	2038(41.2)	6.8	0.009
• HDL cholesterol <50 mg/dl	6633(84.0)	2462(83.5)	4171(84.4)	1.1	0.293
• Smoking/Tobacco(ever)	697(8.8)	216(7.3)	481(9.7)	13.3	<0.001
• CKD, creatinine ≥2mg/dl	168(2.1)	77(2.6)	91(1.8)	5.2	0.022
• BMI≥25kg/m2	5035(63.8)	1890(64.1)	3145(63.6)	0.17	0.678
• CAD family history	1155(14.6)	460(15.6)	695(14.1)	3.5	0.061
Previous cardiovascular status					
• Coronary intervention (PCI)	990(12.5)	586(19.9)	404(8.2)	230.4	<0.001
• Coronary bypass surgery	269(3.4)	153(5.2)	116(2.3)	45.3	<0.001
Past Medications:					
• Antiplatelets	2998(38.0)	2737(92.8)	261(5.3)	6007.3	<0.001
• Beta blocker	2231(28.3)	1820(61.7)	411(8.3)	2597.5	<0.001
Presentation					
• STEMI	3222(40.8)	895(30.3)	2327 (47.1)	213.9	<0.001
• NSTEMI	4670(59.2)	2054(69.7)	2616(52.9)	213.9	<0.001
Symptom to door time (hour): median, IQR	6.7(3.2–10.0)	7.2(3.3–11.0)	6.6(3.2–10.2)	1.7	0.186
Door to balloon time (STEMI, min): median, IQR	62.0(46.0–84.0)	62.0(44.0–85.0)	61.5(47.0–82.0)	0.02	0.889
Left ventricular EF (mean)	45.3 ± 10.1	46.5 ± 9.9	44.5 ± 10.2	71.6	<0.001
• EF <30 % ($n = 8267$)	368(4.7)	121(4.1)	247(5.0)	3.3	0.068
• EF 30–49 %	4103(52.0)	1353(45.9)	2750(55.6)	70.4	<0.001
• EF ≥50 %	3396(43.0)	1459(49.5)	1937(39.2)	79.7	<0.001

Numbers ± indicate 1 SD. Numbers in parenthesis are percent.
CAD coronary artery disease; CKD chronic kidney disease; EF ejection fraction; HDL high density lipoprotein; IQR interquartile range 25–75; LDL low density lipoprotein; LMCA left main coronary artery; NSTEMI non ST segment elevation myocardial infarction; STEMI ST segment elevation myocardial infarction.

Table 2
In-hospital management in statin-user and statin-naïve patients.

Variable	Total cohort ($N = 7892$)	Statin User group ($N = 2949$)	Statin Naïve Group ($N = 4943$)	Chi-square statistic	P Value
Coronary angiography data					
• Left main	419(5.3)	185(6.3)	234(4.7)	8.7	0.003
• Right	4075(51.6)	1554(52.7)	2521(51.0)	2.1	0.145
• Left anterior descending	6289(79.7)	2314(78.5)	3975(80.4)	4.3	0.037
• Left circumflex	3945(50.0)	1509(51.2)	2436(49.3)	2.6	0.105
• Single-vessel disease	3106(39.8)	1127(39.5)	1979(40.0)	2.5	0.109
• Double vessel disease	2588(33.2)	975(34.2)	1613(32.6)	0.15	0.694
• Triple vessel disease	1923(24.7)	730(25.2)	1193(24.1)	0.38	0.535
Stents deployed					
• Nil	175(2.2)	56(1.9)	119(2.4)	2.2	0.138
• 1 stent	5232(66.3)	1849(62.7)	3383(68.4)	27.2	<0.001
• 2 stents	1954(24.8)	825(28.0)	1129(22.8)	26.1	<0.001
• ≥3 stents	531(6.7)	219(7.4)	312(6.3)	3.6	0.056
Specific management					
• Pharmacological vasopressor	883(11.2)	300(10.2)	583(11.8)	1.7	0.194
• Mechanical support (IABP, Impella, ECMO)	263(3.3)	70(2.4)	193(3.9)	6.9	0.008
Duration of hospitalization (hr): median, IQR	68.1(51.2–83.0)	66.3(51.0–79.6)	68.6(51.4–85.1)	15.1	0.001
Discharge medications					
• Statins	7737(98.0)	2908(98.6)	4829(97.7)	8.03	0.002
• Beta-blockers	5557(70.4)	2061(69.9)	3496(70.7)	0.62	0.430
• ACEI/ARB/ARNI	3357(42.5)	1255(42.6)	2102(42.5)	0.01	0.978
• Antiplatelets	7736(98.0)	2906(98.5)	4830(97.7)	6.50	0.011

Numbers ± indicate 1 SD. Numbers in parenthesis are percent.
ACEI angiotensin converting enzyme inhibitors; ARB angiotensin receptor blockers; ARNI angiotensin receptor neprilysin inhibitor; CAD coronary artery disease; CKD chronic kidney disease; ECMO extracorporeal membrane oxygenation; HDL high density lipoprotein; IQR interquartile range 25–75; LDL low density lipoprotein; NSTEMI non ST segment elevation myocardial infarction; STEMI ST segment elevation myocardial infarction.

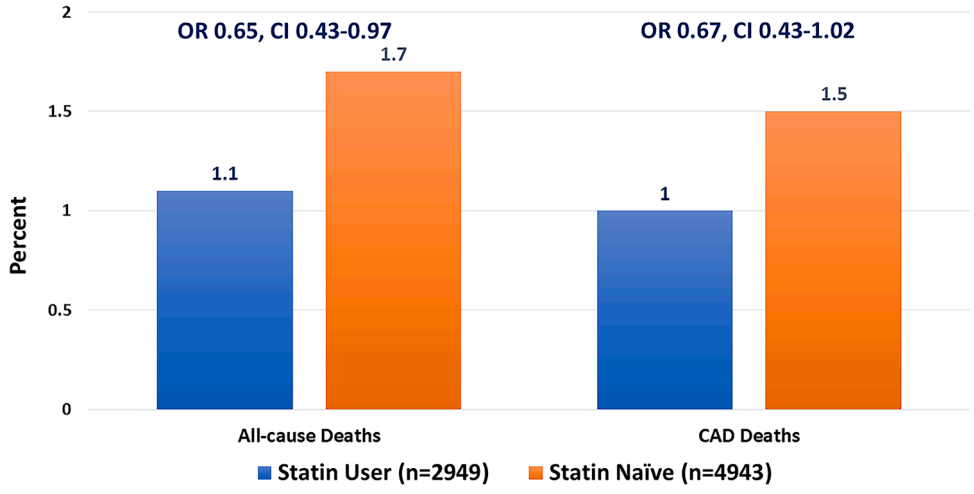

Fig. 1. In-hospital outcomes in statin-user vs statin-naïve acute coronary syndrome patients.

Table 3
In-hospital major cardiovascular events.

	Overall cohort ($n = 7892$)	Statin user group ($n = 2949$)	Statin naïve ($n = 4943$)	P value	Univariate logistic regression OR (95 %CI)	*Multivariate adjusted logistic regression OR (95 % CI)	**Multivariate adjusted logistic regression OR (95 %CI)
Primary:							
All-cause deaths	118(1.5)	33(1.1)	85(1.7)	0.033	0.65(0.43–0.97)	0.82(0.54–1.24)	0.91(0.46–1.81)
CAD/CVD deaths	102(1.3)	29(1.0)	73(1.5)	0.060	0.67(0.43–1.02)	0.87(0.56–1.36)	1.09(0.51–2.32)
Secondary:							
Recurrent ACS	81(1.0)	23(0.8)	58(1.2)	0.093	0.66(0.41–1.08)	0.85(0.52–1.41)	0.88(0.39–2.00)
Acute kidney injury deaths	10(0.1)	02(0.1)	08(0.2)	0.256	0.42(0.09–1.97)	0.37(0.07–1.80)	0.22(0.03–1.52)
Infection-related deaths	27(0.3)	08(0.3)	19(0.4)	0.405	0.71(0.31–1.61)	0.86(0.37–2.02)	2.25(0.54–9.27)

* Multivariate adjustment for age, sex, hypertension, diabetes, STEMI, previous PCI, previous CABG, LVEF, TVD, stents.
ACS acute coronary syndrome; CABG coronary artery bypass graft; CAD coronary artery disease; CVD cardiovascular disease; LVEF left ventricular ejection fraction; **Multivariate adjustments for previous medication use along with other variables; OR odds ratio; PCI percutaneous coronary intervention; TVD triple vessel disease.

therapy [42]. There are isolated studies from lower-income countries in Asia and Africa and none from India [9,10,14]. The benefit of onboard statin therapy along with other cardioprotective drugs, in our patients shows a cardioprotective effect irrespective of baseline risk factors, presentation, ejection fraction, coronary angiographic morphology, number of stents deployed, and other cardio-protective therapies. Long-term statin use can be protective due to the pleiotropic effects; however, in the present study, we have not recorded the duration and doses of statins before ACS and PCI, which is a study limitation. We also evaluated the influence of other cardioprotective therapies (beta--blockers and anti-platelet drugs) before the hospital admission. Prior intake of both medicines was associated with reduced in-hospital all--cause and CVD deaths on univariate analysis (Supplementary Table 1). The magnitude of the benefit was lower than statins, and the odds ratios attenuated following multivariate adjustment. This suggests that not only statins but previous intake of other cardioprotective medicines is associated with reduced in-hospital adverse events.

Other limitations of the study are, a tertiary care location of the registry and our data may not be nationally representative. Many patients enrolled are privately insured (40 %), suggesting a skewed sample that is not representative of India, where only 10–15 % have such insurance [18]. A low representation of women is also a significant limitation, but the data are similar to other CAD registries from India [44]. Conversely, lower total and LDL cholesterol levels in the statin-user group support internal validity. Secondly, we did not include all the ACS patients presenting to the hospital as patients who do not undergo PCI were not enrolled in the ACC—NCDR CathPCI registry [19]; or more sick patients or those with severe CAD and not suitable for PCI have been excluded. Thirdly, although we do not have data on the status of cardiovascular risk factor control or dose of statins at admission, lower total-, LDL- and non-HDL-cholesterol levels among statin users (Table 1) show compliance with statin therapy in this group. Chronic kidney disease has been diagnosed using serum creatinine levels and not the calculated glomerular filtration rate, and this is also a limitation. Fourthly, whether statin use was associated with a reduced incidence of in-hospital arrhythmia, including atrial fibrillation, atrial flutter, ventricular tachycardia, and ventricular fibrillation that contributed to the benefit in all-cause mortality, has not been studied in the present registry. The combination of prior statins use with beta-blockers is reported to be protective against ventricular arrhythmias, pone of the major causes of in-hospital deaths in ACS. Finally, the study cannot elucidate the mechanistic benefits, although previous studies have reported vasculoprotective and cardioprotective benefits of statin onboarding [3,28,29]. Chronic statin use leads to beneficial preconditioning and plaque-stabilising effects, as shown by lower STEMI incidence in the statin-user group (Table 1). The median duration of hospitalization was lower in the statin-user group, and significantly more stents were deployed in statin-users than statin-naïve patients. On the other hand, this is one of the larger prospective CAD intervention registries from lower-middle countries, specifically India, and standardized data have been obtained as part of the ACC—NCDR CathPCI registry.

In conclusion, this prospective PCI registry shows that patients with acute coronary syndrome on pre-admission statins have lower in-hospital all-cause deaths and a trend towards lower cardiovascular deaths. The data support the prescription of statins and other cardioprotective drugs in high-risk primary and secondary prevention patients [43]. Reduced cardiovascular events and mortality in large primary prevention statin trials among high-risk and intermediate-risk patients support our findings. The Prospective Urban Rural Epidemiology (PURE) study reported low use of secondary prevention therapies in lower-income countries [44]. Long-term randomized clinical trials are required in low to intermediate-cardiovascular-risk patients to demonstrate the benefits of statin use to prevent cardiovascular events and deaths, especially in lower-middle-income countries of South Asia [45].

CRediT authorship contribution statement

Rajeev Gupta: Writing – original draft, Supervision, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Prashant Dwivedi:** Writing – review & editing, Methodology, Investigation, Formal analysis. **Krishna K Sharma:** Writing – review & editing, Supervision, Project administration, Methodology, Formal analysis, Data curation. **Sanjeev K Sharma:** Writing – review & editing, Project administration, Methodology, Investigation, Data curation. **Jitender S Makkar:** Writing – review & editing, Methodology, Investigation, Data curation. **Atul Kasliwal:** Writing – review & editing, Methodology, Investigation. **Vishnu Natani:** Supervision, Project administration, Methodology, Data curation. **Raghubir S Khedar:** Writing – review & editing. **Samin K Sharma:** Writing – review & editing, Supervision, Methodology, Investigation, Funding acquisition. **Soneil Guptha:** Writing – review & editing, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ajpc.2025.100999.

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