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Impact of early statin therapy on development of atrial fibrillation at the acute stage of myocardial infarction: data from the FAST-MI register

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ABSTRACT

Background Atrial fibrillation developing at the acute stage of myocardial infarction is associated with untoward clinical outcomes. The aim of this study was to determine correlations between early statin therapy and atrial fibrillation in acute myocardial infarction.

Methods Patients (3396) with sinus rhythm developing acute myocardial infarction were enrolled in the French registry of Acute ST-elevation and non-ST-elevation Myocardial Infarction (FAST-MI).

Results Atrial fibrillation developed in 7.0% of patients without and 3.9% of patients with early (≤ 48 h of admission) statin therapy ($p < 0.001$). Multivariable analysis, including the propensity score for early statin treatment, showed that statin therapy was associated with reduced risk of atrial fibrillation (OR 0.64; 95% CI 0.45 to 0.92, $p = 0.017$). Compared to patients without early statin therapy, the OR for atrial fibrillation were 0.72 (0.49 to 1.04, $p = 0.080$), 0.52 (0.28 to 0.95, $p = 0.034$) and 0.40 (0.18 to 0.92, $p = 0.030$) in patients on conventional, intermediate and high doses respectively.

Conclusions This study is the first to document a correlation between early statin therapy and atrial fibrillation at the early stage of acute myocardial infarction.

INTRODUCTION

In patients with acute myocardial infarction (AMI), atrial fibrillation increases mortality and prolongs hospital stay.^{1 2} Statins may be effective in preventing the development of atrial fibrillation because they have potent anti-inflammatory and antioxidant properties,³⁻⁵ and a relationship between inflammation and occurrence of atrial fibrillation has been described.⁵⁻⁸ In a recent meta-analysis of studies assessing the role of preoperative statin therapy in patients undergoing cardiac surgery, a 33% reduction in the risk of developing atrial fibrillation was observed.⁹ In addition, atorvastatin prevents significant atrial fibrillation (≥ 10 min) in patients with bradyarrhythmias and an implanted pacemaker.¹⁰ A dose-related effect of statins on atrial fibrillation has been observed after cardiac surgery, with higher dose statins having the greatest preventive effect.¹¹

To the authors' knowledge, although an association between early statin use and mortality has

been reported in cohorts of patients with acute coronary syndromes,^{12 13} the impact on the occurrence of atrial fibrillation in patients with AMI has never been studied. The French registry of Acute ST-elevation and non-ST-elevation Myocardial Infarction (FAST-MI) recorded patterns of statin prescription during the acute phase of MI in 2005.¹⁴ The aim of the present analysis was to use data from the FAST-MI registry to analyse the correlation between early statin prescription and the development of acute atrial fibrillation.

METHODS

The methods for the prospective, multicentre FAST-MI registry have been described in detail elsewhere.¹⁵ The objective of the study was to collect comprehensive data on the management and outcome of patients admitted to intensive care units (ICU) in France with definite AMI. All institutions, academic hospitals, general and regional hospitals and private clinics with ICU authorised to receive AMI patients were given the opportunity to participate. Patients were recruited consecutively from the ICU over a period of 1 month (from October 2005) for non-diabetic patients, and 2 months for patients with known diabetes mellitus. In each centre, one physician responsible for the study provided a complete list of all patients who met the inclusion criteria and were admitted to the ICU during the study recruitment period. Patient care at each centre was performed according to usual practice, independent of the study.

Patient selection

Briefly, men and women aged > 18 years were eligible for inclusion in the registry if they had been admitted to an ICU with an AMI within 48 h of symptom onset. The AMI had to be characterised by elevation of troponin, creatine kinase or CK-MB, and be associated with at least one of the following elements: symptoms compatible with AMI; ECG changes on two or more contiguous leads with appearance of pathological Q waves (≥ 0.04 s); or persistent ST-segment elevation or depression. Patients were excluded if they had an iatrogenic MI. Written informed consent was provided by each patient, and the protocol was reviewed by the Committee for the Protection of Human Subjects in Biomedical Research of Saint Antoine University Hospital.

Acute coronary syndromes

For the present analysis, patients with ST-segment elevation and non-ST-segment elevation myocardial infarction were included when sinus rhythm was noted on the first ECG (the admission ECG).

Data collection

A computerised case-record form was completed for each eligible patient by research technicians. An audit was performed in three of the 21 administrative regions. Data on baseline demographic and clinical characteristics, in-hospital clinical course and therapeutic management (including maximum Killip class, diagnostic procedures, reperfusion therapies and medication (type and dose)) on admission, during the first 48 h of hospitalisation and at discharge, and in-hospital clinical events and survival, were recorded. In-hospital follow-up was 100% complete.

In this study, the correlation between early statin therapy (prescription within 48 h of admission) and development of atrial fibrillation during the hospital stay (ie, until the patients died, were discharged home or were discharged to a rehabilitation centre) was analysed. To enable measurement of a potential dose–effect relationship, high-dose statin therapy was defined as prescription of atorvastatin 80 mg or rosuvastatin 40 mg, and intermediate-dose statin therapy was defined as prescription of atorvastatin 40 mg or rosuvastatin 20 mg. All other statin prescriptions were considered to be conventional doses. Occurrence of new onset atrial fibrillation (or lack thereof) was one of the complications specifically mentioned in the ‘Complications’ section of the electronic case-record form; when appropriate, the date of onset of the first episode of atrial fibrillation also had to be recorded. Atrial fibrillation was investigator-reported at each of the centres. The presence of atrial fibrillation was documented by continuous monitoring while the patients were in the ICU, confirmed by 12-lead ECG recordings; once the patients had left the ICU, the presence of atrial fibrillation was documented by 12-lead ECG recordings, which were driven by the presence of symptoms or were performed in a systematic way in asymptomatic patients. Of the 159 patients who developed atrial fibrillation, the date of the first episode was recorded in 144 (91%).

Statistical analysis

Continuous variables are described as mean and SD or median and IQR. Categorical variables are described with absolute and relative frequency distributions. Comparisons between groups were made using one-way analysis of variance (ANOVA) and unpaired Student *t* tests or Mann–Whitney *U* tests for continuous variables and χ^2 tests for discrete variables. Binary logistic regression analysis (backward stepwise model) was used to assess independent predictors of atrial fibrillation. Because the occurrence of atrial fibrillation could be dated in over 90% of the patients, a backward stepwise Cox model was also used, with and without patients who developed atrial fibrillation on day 1 (ie, at a time when it was uncertain that statins had already been administered); patients who died without a prior episode of atrial fibrillation were censored at the time of death. All variables listed in table 1 were included in the different backward multivariable models. A propensity analysis was also performed for the early use of statins, using a multivariable logistic regression model including all variables listed in table 1, and the propensity score was used as a continuous covariable in the multivariable regression analyses. Adequacy of the propensity score was tested using the Hosmer–Lemeshow test, which yielded a *p* value of 0.84. Finally, for the 144 patients in whom the date of the first atrial fibrillation episode was recorded, Kaplan–Meier survival curves for the occurrence of atrial fibrillation were computed

with censoring of the patients who died without a prior episode of atrial fibrillation at the time of death; atrial-fibrillation-free survival curves were also generated. For all tests, *p*<0.05 was considered to be significant.

RESULTS

The study was conducted at 223 centres and included 3670 patients with AMI. A total of 3396 patients (those with sinus

Table 1 Patient baseline characteristics according to use of statin therapy within 48 h of admission

Variable	No statin (n=845)	Statin (n=2551)	<i>p</i> Value
Age in years, mean (SD)	71 (14)	65 (14)	<0.001
Women, n (%)	319 (38)	737 (29)	<0.001
BMI in kg/m ² , mean (SD)	27 (5)	27 (4.7)	0.37
Medical history, n (%)			
Hypertension	562 (67)	1419 (56)	<0.001
Diabetes mellitus	319 (38)	886 (35)	0.11
Hyperlipidaemia	294 (35)	1349 (53)	<0.001
Family history of coronary artery disease	151 (18)	644 (25)	<0.001
Current smoker	208 (25)	821 (32)	<0.001
Stroke	55 (7)	110 (4)	0.009
Transient ischaemic attack	26 (3)	85 (3)	0.73
Peripheral vascular disease	94 (11)	233 (9)	0.08
Congestive heart failure	59 (7)	111 (4)	0.002
Previous myocardial infarction	166 (20)	440 (17)	0.12
Percutaneous coronary intervention	117 (14)	345 (14)	0.81
Coronary artery bypass graft	49 (6)	141 (6)	0.77
Chronic renal failure	57 (7)	129 (5)	0.06
Chronic obstructive pulmonary disease	46 (6)	112 (4)	0.20
Type of AMI, n (%)			
STEMI with reperfusion	196 (23)	931 (37)	<0.001
STEMI without reperfusion	179 (21)	455 (18)	
NSTEMI	470 (56)	1165 (46)	
Anterior myocardial infarction, n (%)	172 (20)	536 (21)	0.68
Admission Killip class \geq II, n (%)	257 (31)	500 (20)	<0.001
Time to first call \geq 3 h, n (%)	331 (40)	1028 (41)	0.61
GRACE risk score, mean (SD)	157 (40)	144 (33)	<0.001
Medications used before AMI, n (%)			
Antiplatelet agents	293 (35)	796 (31)	0.06
β blockers	216 (26)	618 (24)	0.43
Statins	128 (15)	810 (32)	<0.001
ACE inhibitors	185 (22)	480 (19)	0.051
Angiotensin receptor blockers	153 (18)	357 (14)	0.004
Any diuretic	274 (32)	586 (23)	<0.001
Any calcium channel blocker	186 (22)	472 (19)	0.03
Amiodarone	43 (5)	57 (2)	<0.001
Digoxin	29 (3)	21 (1)	<0.001
Nitrates	120 (14)	113 (8)	<0.001
Medications within 48 h, n (%)			
Antiplatelet agents	749 (89)	2525 (99)	<0.001
Clopidogrel	585 (69)	2350 (92)	<0.001
Glycoprotein IIb/IIIa inhibitors	236 (28)	1039 (41)	<0.001
β blockers	422 (50)	1967 (77)	<0.001
Amiodarone	104 (12)	151 (6)	<0.001
Digoxin	18 (2)	22 (1)	0.003
ACE inhibitors	254 (30)	1327 (52)	<0.001
Angiotensin II receptor blocker	66 (8)	182 (7)	0.51
Any diuretic	343 (41)	774 (30)	<0.001
Any calcium channel blocker	161 (19)	476 (19)	0.80

AMI, acute myocardial infarction; GRACE, Global Registry of Acute Coronary Events; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction.

rhythm on the admission ECG) were included in this study, 2551 (75%) of whom received early statin therapy (figure 1).

Patient characteristics at baseline according to use of statin therapy within 48 h of admission are shown in table 1. Patients treated with statins were younger (65 vs 71 years) than those not given statins and a smaller proportion were women (29% vs 38%), had a higher rate of hyperlipidaemia and family history of coronary artery disease, and were more likely to be current smokers or to be diagnosed with ST-segment elevation myocardial infarction. They had a lower GRACE (Global Registry of Acute Coronary Events) risk score.¹⁶ They also were more likely to be on chronic statin therapy before the index event, but less likely to be on nitrates, calcium antagonists, diuretics, amiodarone or digoxin (all $p < 0.001$) (table 1). During the first 48 h, they were more likely to receive evidence-based medications (β blockers, antiplatelet agents and ACE inhibitors).

Development of atrial fibrillation

Atrial fibrillation developed in 4.7% of the whole population: in 7.0% (59/845) of patients who did not receive early statin therapy and in 3.9% (100/2551) of patients who did receive early statin therapy ($p < 0.001$). The date of the first episode of atrial fibrillation was known in 144 (91%) patients. Event curves for the occurrence of atrial fibrillation and atrial fibrillation-free survival in these patients are shown in figure 2. Other univariate correlates of development of atrial fibrillation were age (75 ± 11 vs 66 ± 14 years, $p < 0.001$), sex (41% vs 31% women, $p = 0.006$), hypertension (70% vs 58%, $p = 0.002$), higher admission Killip class (mean 1.71 ± 0.98 vs 1.32 ± 0.68 , $p < 0.001$), peripheral arterial disease (15% vs 9%, $p = 0.016$), less frequent family history of coronary artery disease (16% vs 24%, $p = 0.031$), less current smoking (21% vs 31%, $p = 0.012$), less frequent history of coronary artery bypass surgery (2% vs 6%, $p = 0.037$), higher GRACE risk score (174 ± 38 vs 146 ± 35 , $p < 0.001$) and atypical chest pain (31% vs 21%, $p = 0.009$).

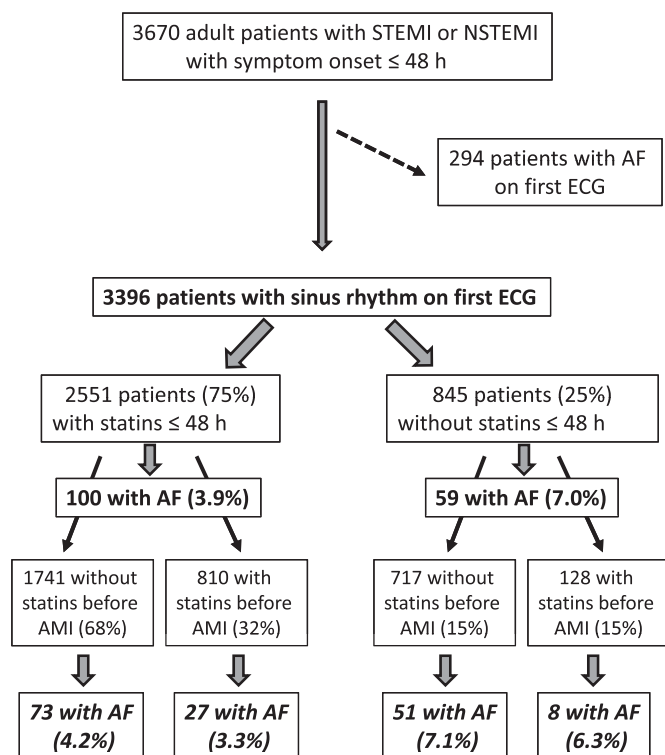


Figure 1 Flow chart of the population included in the current analysis.

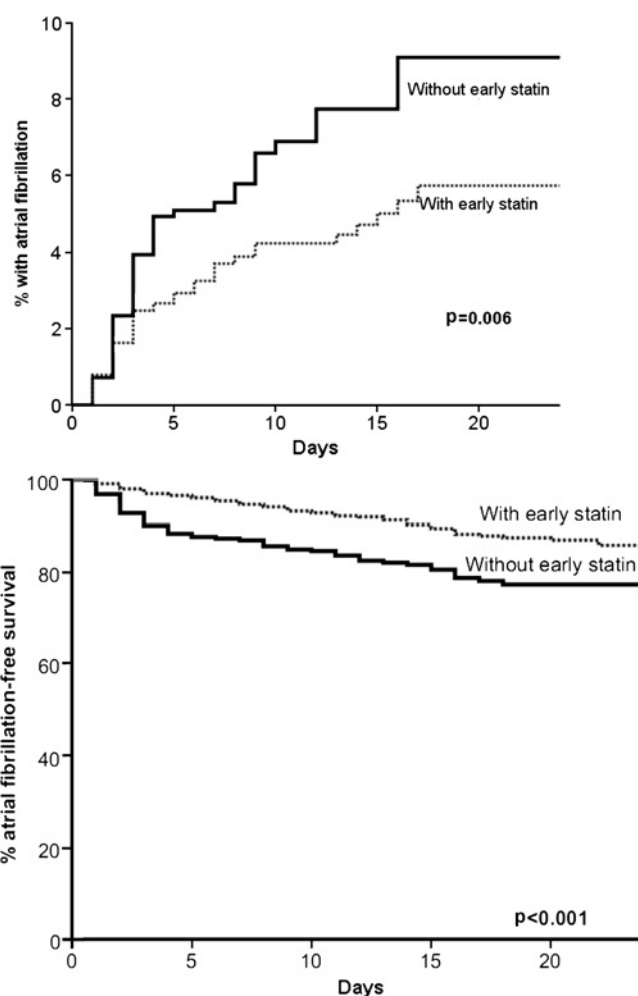


Figure 2 Kaplan–Meier event curves. Upper panel: occurrence of atrial fibrillation. Lower panel: atrial fibrillation-free survival.

Sensitivity analyses

In the 1635 patients with non ST-elevation MI, the occurrence of atrial fibrillation was 3.4% in those with vs 7.4% in those without early statin therapy ($p < 0.001$); the respective figures were 3.7% vs 7.3% for the 634 patients with ST-elevation MI without reperfusion therapy ($p = 0.06$) and 4.6% vs 5.6% in the 1127 patients with reperfused ST-elevation MI ($p = 0.55$).

The impact of statin therapy on acute atrial fibrillation was of the same magnitude in patients not treated with statins before the acute event (4.2% when early statin treatment was used vs 7.1% when it was not) and in those previously treated (3.3% in patients on continued treatment vs 6.3% when statin treatment was discontinued) (p for heterogeneity = 0.82) (table 2). In contrast, there was no impact of statin therapy in patients previously receiving amiodarone or digoxin (development of atrial fibrillation in 4.2% vs 4.5% in those not receiving statins), whereas the impact of statin therapy was marked in patients without previous treatment with amiodarone or digoxin (3.9% vs 7.2%, $p < 0.001$). Further sensitivity analyses were made and yielded consistent results. Occurrence of atrial fibrillation during the ICU stay (ie, during the period when ECG was constantly monitored) confirmed a lower rate of atrial fibrillation associated with early statin therapy (2.9% vs 4.4%, $p = 0.035$). Likewise, a specific analysis of atrial fibrillation occurring within 5 days of admission showed that it was less frequent in those who had received early statin treatment (2.8% vs 4.6%, $p = 0.007$). Finally,

Acute coronary syndromes

Table 2 Baseline characteristics and in-hospital atrial fibrillation occurrence, according to pre-event statin use and statin treatment during the first 48 h

	Statins before			No statin before		
	Statins at 48 h (n=810)	Statins stopped at 48 h (n=128)	p Value	Statins at 48 h (n=1741)	No statins at 48 h (n=717)	p Value
Age (mean±SD)	68±12	70±11	0.03	64±14	71±15	<0.001
GRACE score	147±33	157±39	<0.001	143±33	157±40	<0.001
Sex (W) (%)	239 (29.5)	47 (37)	0.10	498 (29)	272 (38)	<0.001
Hypertension (%)	229 (28)	25 (19.5)	0.04	838 (48)	459 (62)	<0.001
Diabetes (%)	380 (47)	66(52)	0.33	506 (29)	253 (35)	0.002
Current smoking (%)	195 (24)	20 (16)	0.035	626 (36)	1888 (26)	<0.001
Prior MI (%)	296 (36.5)	50 (39)	0.58	144 (8)	116 (16)	<0.001
Prior stroke (%)	60 (7)	6 (5)	0.27	50 (3)	49 (7)	<0.001
History of heart failure (%)	58 (7)	10 (8)	0.79	53 (3)	49 (7)	<0.001
Chronic renal failure (%)	68 (8)	9 (7)	0.60	61 (3.5)	48 (7)	<0.001
COPD (%)	38 (5)	7 (5.5)	0.71	74 (3)	39 (5.5)	0.19
STEMI (%)	333 (41)	51 (40)	0.79	1053 (60.5)	324 (45)	<0.001
Atrial fibrillation (%)	27 (3.3)	8 (6.3)	0.11	73 (4.2)	51 (7.1)	0.003
Adjusted OR for atrial fibrillation (95% CI)	0.42 (0.15 to 1.18)		0.10	0.73 (0.46 to 1.16)		0.19

Multivariable adjustment is made using logistic regression analysis, entering all variables listed in table 1.

COPD, chronic obstructive pulmonary disease; GRACE, Global Registry of Acute Coronary Events; MI, myocardial infarction; STEMI, ST-elevation myocardial infarction.

similar findings were observed in the patients who got statin late (ie, after 48 h) versus those who got statins within 48 h of admission (6.3% vs 3.9%, $p=0.031$), and in the group who got no statins throughout the hospital stay versus those with statins within 48 h of admission (7.6% vs 3.9%, $p<0.001$).

Multivariable analyses

Using logistic regression analysis, early statin therapy was associated with a significantly reduced risk of atrial fibrillation (OR 0.64, 95% CI 0.45 to 0.92, $p=0.017$). Other predictors were older age, higher GRACE risk score, nitrate use before the acute event, and use of loop diuretics during the first 48 h, whereas type of MI, history of coronary bypass surgery, and prior use of digoxin or amiodarone were associated with a reduced risk of developing atrial fibrillation (table 3). When Cox multivariable analysis was used in the 144 patients in whom the date of the first atrial fibrillation episode was known, the OR was 0.59 (95% CI 0.41 to 0.85, $p=0.005$). When the analysis was repeated after excluding patients who developed atrial fibrillation on day 1 or 2 (ie, who might have developed atrial fibrillation before

administration of statins), the OR was 0.60 (95% CI 0.37 to 0.95, $p=0.030$).

Dose effect

A conventional dose of statin was used in 1635 patients, 510 received an intermediate dose (atorvastatin 40 mg, $n=418$; rosuvastatin 20 mg, $n=3$) and 406 received a high dose (all atorvastatin 80 mg). There was a linear relationship between use and dose of statins and development of atrial fibrillation (figure 3; $p<0.001$; p for trend <0.001), which was confirmed by multivariable logistic regression: compared with patients who did not receive early statin therapy, the adjusted OR for atrial fibrillation were 0.72 (95% CI 0.49 to 1.04, $p=0.080$) with a conventional statin dose, 0.52 (95% CI 0.28 to 0.95, $p=0.034$) with an intermediate dose and 0.40 (95% CI 0.18 to 0.92, $p=0.030$) with a high dose.

DISCUSSION

In this nationwide observational study of patients hospitalised for AMI, early statin therapy was associated with a reduced risk of developing acute atrial fibrillation. The overall risk was reduced by 40%, whatever the type and dose of statin prescribed, increasing to 60% at the highest dose of statins. The present

Table 3 Independent variables associated with risk of developing atrial fibrillation

Variable	OR	95% CI	p Value
Statin treatment within 48 h	0.64	0.45 to 0.92	0.017
Age (vs <60 years)			
60–74 years	2.18	1.19 to 3.98	0.008
≥75 years	2.62	1.39 to 4.94	0.002
Type of myocardial infarction (reference: STEMI with reperfusion therapy)			
STEMI without reperfusion therapy	0.59	0.36 to 0.99	0.034
NSTEMI	0.64	0.42 to 0.97	0.044
Upper tertile of GRACE risk score	2.09	1.37 to 3.19	0.001
Prior coronary bypass surgery	0.25	0.08 to 0.79	0.019
Nitrates before admission	1.84	1.19 to 2.86	0.006
Digoxin or amiodarone before admission	0.42	0.18 to 0.99	0.048
Loop diuretics during first 48 h	2.83	1.94 to 4.12	<0.001

Multiple logistic regression analysis using all variables from table 1 and propensity score for getting statin therapy.

GRACE, Global Registry of Acute Coronary Events; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction.

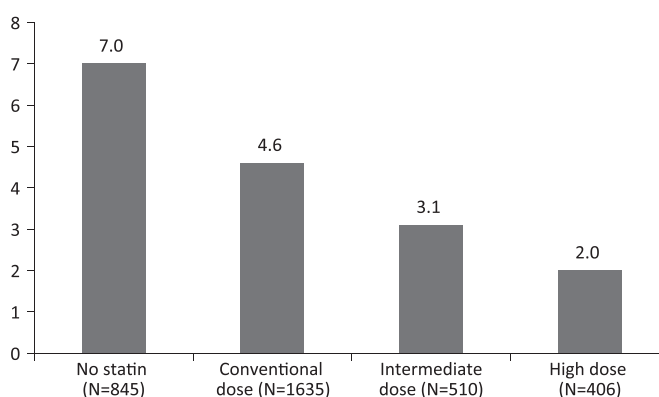


Figure 3 Relationship between statin dose and development of atrial fibrillation (p for trend <0.001). See text for definition of doses.

data show that patients given early statin therapy were also more likely to receive early treatment with other evidence-based therapies for acute coronary syndromes,^{17 18} including clopidogrel, glycoprotein IIb/IIIa inhibitors, β blockers and ACE inhibitors. The association between early prescription of statins and prevention of atrial fibrillation at the acute stage, however, was shown to be independent of other factors by multivariable analyses including the propensity score for receiving early statin therapy, be it logistic regression or Cox proportional hazard analyses, and confirmed by sensitivity analyses. In addition, a graded relationship between statin dose and prevention of atrial fibrillation was observed, although it must be noted that in the multivariable analysis, the CI between doses largely overlapped.

The findings from the present study concur with several other observational studies in different populations^{19–23} that reported a reduction in the risk of atrial fibrillation with statin therapy. Recently, an ancillary study from the GISSI-HF trial showed a strong trend to reduction in atrial fibrillation occurrence with rosuvastatin in a population of patients with chronic heart failure.²⁴ This also agrees with data from two small randomised trials in patients undergoing cardiac surgery or external cardioversion^{25 26} and a recent meta-analysis involving over 30 000 patients undergoing cardiac surgery.⁹ Likewise, in another meta-analysis of six trials involving statins in different clinical settings and in which the occurrence of atrial fibrillation was reported, the use of statins was associated with a decreased risk of atrial fibrillation (OR 0.39; 95% CI 0.18 to 0.85, $p=0.02$).²⁷ In the Myocardial Ischaemia Reduction with Aggressive Cholesterol Lowering (MIRACL) trial, however (which did not include patients with ST-segment elevation myocardial infarction and the results of which were reported in abstract form only), the use of atorvastatin was not associated with significant reduction in the risk of developing atrial fibrillation during the 4-month trial duration (OR 0.97).^{27 28} In the MIRACL trial, the average time from hospital admission to randomisation was 63 h,²⁸ whereas the present study examined the effect of statin treatment prescribed during the first 48 h from admission. Two large randomised clinical trials comparing different doses of statins following non-ST-elevation acute coronary syndromes showed no association between the use of higher doses and prevention of atrial fibrillation during long-term follow-up.²⁹ These findings do not preclude an effect of statin therapy on the occurrence of atrial fibrillation, as all patients were treated with statins. More importantly, the impact of statins may differ largely between the acute stage (present study), where inflammation is high, and the subacute or chronic stage, such as in the above-mentioned trials.

Underlying mechanisms

Statins have multiple pleiotropic effects, including preservation of endothelial function, anti-inflammatory and antioxidant properties that may be independent of their lipid-lowering properties.^{30–36} These effects underlie the hypothesis that statins may decrease the risk of atrial fibrillation.³⁶ Indeed, inflammation is one of the mechanisms involved in the development of atrial fibrillation.^{37 38} In this regard, a recent randomised trial in patients undergoing cardiac surgery has shown that corticosteroids reduced the incidence of atrial fibrillation (OR 0.54, 95% CI 0.38 to 0.83, $p=0.004$).³⁹ Finally, in the perioperative setting, it has also been suggested that the anti-fibrillatory effect of statins might be mediated by autonomic modulation, a factor that also might be of importance in the setting of AMI.⁴⁰ Whether these effects are limited to specific

statins or dosages, or are consistent for all drugs within the class, remains to be elucidated. In the present study, there was no evidence of a difference in effect according to the statin that was used, although the dose seemed to matter.

Strengths and limitations

As a non-randomised observational study, FAST-MI provides data on 'real-world' practice patterns and includes populations that are more representative of those treated in clinical practice than populations enrolled in randomised controlled trials. Observational studies also provide information on the therapeutic effect of medications used to treat patients in everyday practice. Observational data are, however, limited by potential biases inherent in such studies, including collection of non-randomised data, missing or incomplete information and potential confounding by drug indication or other unmeasured covariates, and do not give causal relationships. However, the multivariable analyses, including the use of a propensity score for receiving early treatment with statins, limit, although they do not preclude, the risk of bias. One of the obvious limitations of the present study was that there was no centralised reading of the ECG and no continuous recording of the ECG throughout the hospital stay, and brief periods of arrhythmias may not have been reported. This limitation (outcome measurement of atrial fibrillation) is inherent to almost all clinical trials in atrial fibrillation. Patients, however, were monitored continuously in the acute phase in the intensive care units. Although it is possible that some asymptomatic or short episodes of atrial fibrillation were missed, there is no obvious reason to think that these asymptomatic episodes were less likely to occur in patients treated earlier or with a higher dose of statin.

Clinical implications

Atrial fibrillation at the acute stage of MI poses real difficulties in its management, in particular with regard to the use of anticoagulants in patients who otherwise often receive potent combined antiplatelet therapy. In addition, patients with AMI developing atrial fibrillation have a definitely poorer long-term outcome, even in the current era of primary percutaneous intervention for ST-segment elevation myocardial infarction.^{41 42} Therefore, the fact that statin use was associated with a lower risk of developing this complication is important and provides an additional incentive for the early prescription of these medications in patients with acute coronary syndromes.

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Competing interests Dr Danchin has received fees for participating in symposia organised by, or served as a temporary consultant for Astra-Zeneca, Bristol-Myers Squibb, Merck Sharp & Dohme, Novartis, Pfizer, Sanofi-Aventis, all of whom manufacture statins, and has been involved in a number of studies sponsored by these companies.

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sponsored by these companies.

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