Impact of atrial fibrillation on the risk of major adverse cardiac events following coronary revascularisation

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ABSTRACT

Objective Atrial fibrillation (AF) remains a highly prevalent arrhythmia with significant burden on morbidity and mortality. The impact of AF in the revascularised population remains incompletely described. Given the high prevalence of AF in the revascularised population, we sought to evaluate the incidence and prognosis in patients with pre-existing and new-onset AF following revascularisation.

Methods We used the University of Ottawa Heart Institute Revascularisation Registry to identify patients who underwent revascularisation between August 2015 and March 2020, who were prospectively followed for an average of one year. We conducted a retrospective cohort study analysing the association between AF and clinical outcomes. The primary outcome of interest was 1-year major adverse cardiac events (MACE) defined as a composite of death, myocardial infarction, unplanned revascularisation and cerebrovascular accidents. Moreover, secondary outcomes include the individual components of MACE and bleeding.

Results A total of 6704 patients underwent revascularisation and completed 1-year clinical follow-up. Median time to follow-up was 12.8 (IQR 11.2–15.9) months. One-year MACE occurred in 166 (21.8%) and 683 (11.5%) patients in AF and non-AF groups, respectively (adjusted HR, 1.61; 95% CI 1.29 to 2.01; p<0.0001). AF was independently predictive of 1-year mortality, myocardial infarction, unplanned revascularisation, cerebrovascular accident and bleeding. Within 1 year, 299 (4.5%) episodes of new-onset AF were observed. New-onset AF following revascularisation was also associated with 1-year MACE, mortality, myocardial infarction, cerebrovascular accident and unplanned revascularisation.

Conclusions Preprocedural and new-onset AF following revascularisation remains highly predictive 1-year MACE. AF should be considered in addition to traditional risk factors for adverse outcomes following revascularisation.

INTRODUCTION

Atrial fibrillation (AF) is a highly prevalent arrhythmia with a significant morbidity and mortality.1 The correlation between AF and outcomes following coronary revascularisation remains incompletely described. Many risk factors for AF and atherosclerotic disease are shared including diabetes, hypertension, advanced age and obesity.2 3 Furthermore, AF with rapid ventricular response can be a trigger for coronary assessment including angiography and subsequent revascularisation. In general, 5%–10% of patients referred for coronary revascularisation present with pre-existing AF, which may be associated with increased risk of thromboembolic and bleeding complications.4 5 Management of
patients with AF following percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) adds complexity as practitioners balance the risks of bleeding and systemic embolisation. With indications for oral anticoagulation (OAC) and/or dual-antiplatelet therapy, these patients are often treated for periods with ‘dual’ or ‘triple’ therapy increasing the risk of both minor and major bleeding.6,7

Of note, AF is a risk factor for adverse outcomes in a multitude of cardiac conditions. In the acute coronary syndrome (ACS) population, AF has been independently linked to higher mortality rates.1,2 Among patients undergoing CABG, AF is associated with an increased risk of stroke.3 Similarly, in patients undergoing revascularisation of left main disease, new-onset AF was an independent predictor of 3-year major adverse cardiac event (MACE).7 Finally, among patients with heart failure, the development of AF is associated with increased mortality.5 Thus, while the association of AF with adverse outcomes in these populations is established, its impact on postrevascularisation outcomes and comparative analysis to baseline risk factors remain poorly established.

Given the high prevalence of AF and the uncertain correlation to adverse events in all-comers undergoing revascularisation, we sought to evaluate the incidence and prognosis in patients with pre-existing and new-onset AF following revascularisation.

MATERIALS AND METHODS

Study population, data collection and clinical follow-up

The University of Ottawa of Heart Institute is a large tertiary-care serving 1.2 million people in the capital region of Canada.8,9 All revascularisation patients have their data collected in the Cardiovascular And Percutaneous clinical TriAIs revascularisation registry, a prospective registry of all patients undergoing coronary catheterisation. The registry captures over 1200 clinical data points with regard to both procedural and patient characteristics.10 Specific comorbidities are documented at the time of preprocedural assessment. Patients or the public were not involved in the design, conduct, reporting or dissemination plans of our research. All patients undergo standardised 1-year follow-up for evaluation of risk factor modification and to screen for adverse clinical events which are indexed in the registry. All patients with AF were diagnosed on the basis of electrocardiographic or telemetry data documenting AF lasting longer than 30 seconds. Atrial tachycardia and atrial flutter were not categorised separately and are not included in this analysis. Data were collected retrospectively and analysed to describe the association between AF and major adverse cardiovascular events.

Clinical outcomes

The primary endpoint of interest was MACE at 1 year, defined as a composite of death, myocardial infarction (MI), unplanned revascularisation and cerebrovascular accident. Secondary outcomes were individual components of MACE and bleeding at 1-year follow-up. MI was defined according to the fourth universal definition of MI (requiring a rise and fall of Troponin to higher than the 99th percentile upper reference limit and with one of: ischaemic symptoms, ECG changes, wall motion abnormality or intracoronary thrombus).11 Bleeding was defined according to the Thrombolysis in MI criteria, including CABG-related bleeding (defined as perioperative intracranial bleed within 48 hours, chest tube output greater than 2000 mL over 24 hours, transfusion of five or more units of packed red blood cells or reoperation due to bleeding) and non-CABG bleeding (defined as any intracranial bleeding, fatal bleeding and bleeding requiring intervention or hospitalisation).12 Moreover, we evaluated differences in outcomes by non-AF and new-onset AF.

Statistical analysis

Continuous variables were reported as mean ±SD or median (Q1–Q3), and categorical variables were reported as proportions (%). Continuous variables were compared by standard t-test or Mann-Whitney U test after testing for normality. Additionally, categorical variables were compared by χ² test or Fisher’s exact test.

AF and rates of primary and secondary endpoints were analysed by Kaplan-Meier curves generated to evaluate time-to-event data for clinical outcomes. Patients were censored following the first occurrence of MACE. Furthermore, HRs and two-sided 95% CIs with and without adjusting for age, sex, type 2 diabetes, hypertension, dyslipidaemia, ACS, prior stroke, vascular disease and anticoagulation use were generated using a Cox proportional hazards model.

All statistical analyses were performed using SAS V.9.4 (SAS Institute). All figures were created using GraphPad Prism V.8.4 (GraphPad Software, La Jolla, California, USA). A p<0.05 was considered statistically significant.
|                                | Overall (n=6704) | Non-AF (n=5943) | AF (n=761) |
|--------------------------------|------------------|-----------------|------------|
|                                | N                | %               | N          | %           | N          | %           | P value    |
| Age (mean±SD)                  | 66.2             | 11.7            | 65.2       | 11.6        | 74.2       | 9.8         | <0.0001    |
| Sex (male)—no (%)             | 5009             | 74.7            | 4434       | 74.6        | 575        | 75.6        | 0.57       |
| Hypertension—no (%)            | 4148             | 61.9            | 3597       | 60.5        | 551        | 72.4        | <0.0001    |
| Dyslipidaemia—no (%)           | 3987             | 59.5            | 3493       | 58.8        | 494        | 64.9        | 0.001      |
| Diabetes—no (%)                |                  |                 |            |             |            |             | <0.0001    |
| Type I                         | 51               | 0.8             | 49         | 0.8         | 2          | 0.3         |            |
| Type II                        | 1852             | 27.6            | 1583       | 26.6        | 269        | 35.3        |            |
| Smoking—no (%)                 |                  |                 |            |             |            |             | <0.0001    |
| Never                          | 4005             | 59.7            | 3498       | 58.9        | 507        | 66.6        |            |
| Remote (quit >1 month ago)     | 1505             | 22.4            | 1325       | 22.3        | 180        | 23.7        |            |
| Active                         | 1193             | 17.8            | 1119       | 18.8        | 74         | 9.7         |            |
| Family history of CAD—no (%)  | 875              | 13.1            | 804        | 13.5        | 71         | 9.3         | 0.001      |
| Atrial fibrillation—no (%)    |                  |                 |            |             |            |             |            |
| Valvular AF                    | 134              | 2.0             |            |             | 134        | 17.6        |            |
| Non-valvular AF                | 627              | 9.4             |            |             | 627        | 82.4        |            |
| Rate control                   |                  |                 |            |             |            |             |            |
| Beta-blocker                   | 414              | 6.2             |            |             | 414        | 54.4        |            |
| Calcium channel blocker        | 115              | 1.7             |            |             | 115        | 15.1        |            |
| Digoxin                        | 52               | 0.8             |            |             | 52         | 6.8         |            |
| Rhythm control                 |                  |                 |            |             |            |             |            |
| Amiodarone                     |                  |                 |            |             | 59         | 10.1        |            |
| Flecaïnide                     |                  |                 |            |             | 1          | 0.2         |            |
| TTE (n=2541)                   |                  |                 |            |             |            |             |            |
| LVEF—no (%)                    |                  |                 |            |             |            |             | <0.0001    |
| Normal                         | 1562             | 61.5            | 1377       | 69.1        | 185        | 33.8        |            |
| >45%                           | 442              | 17.4            | 260        | 13.1        | 182        | 33.2        |            |
| <45%                           | 385              | 15.2            | 262        | 13.2        | 123        | 22.5        |            |
| LA size (cm)—mean±SD          |                  |                 |            |             |            |             | <0.0001    |
| Mitral regurgitation—no (%)    | 271              | 10.7            | 55         | 2.8         | 216        | 39.4        | <0.0001    |
| CHADS2 (≥1)—no (%)            | 4916             | 73.3            | 4237       | 71.3        | 679        | 89.2        | <0.0001    |
| CHA2DS2-VASc (≥2)—no (%)      | 4509             | 67.3            | 3848       | 64.7        | 661        | 86.9        | <0.0001    |
| Oral anticoagulation           |                  |                 |            |             |            |             |            |
| Rivaroxaban                    | 167              | 2.5             | 10         | 0.2         | 157        | 20.6        |            |
| Apixaban                       | 213              | 3.2             | 11         | 0.2         | 202        | 26.5        |            |
| Dabigatran                     | 40               | 0.6             | 0          | 0.0         | 40         | 5.3         |            |
| Warfarin                       | 106              | 1.6             | 29         | 0.5         | 77         | 10.1        |            |
| Chronic alcohol use (>8 drinks/day) | 52               | 0.8             | 38         | 0.6         | 14         | 1.8         | 0.09       |
| Indications for angiography—no (%) |                  |                 |            |             |            |             |            |
| Acute coronary syndrome        | 3645             | 54.4            | 3239       | 54.5        | 406        | 53.4        | 0.55       |
| Staged PCI                     | 1042             | 15.5            | 932        | 15.7        | 110        | 14.5        | 0.38       |
| Stable CAD                     | 1832             | 27.3            | 1648       | 27.7        | 184        | 24.2        | 0.04       |
| Cardiogenic shock              | 100              | 1.5             | 79         | 1.3         | 21         | 2.8         | 0.002      |

Continued
RESULTS

Between August 2015 to March 2020, a total of 9315 patients underwent coronary angiography with consideration of revascularisation. After excluding those patients with incomplete 1-year follow-up and those who were not revascularised, a total of 6704 patients were identified. In this cohort, a total of 761 (11.4%) patients had AF (figure 1).

The baseline characteristics of patients are summarised in table 1. The mean age was 66.2 ±11.7 years and 5009 (74.7%) patients were male. Risk factor data that were collected, included type 2 diabetes (27.6%), hypertension (61.9%), dyslipidaemia (59.5%), active smoking (17.8%) and family history of CAD (13.1%). The above risk factors were chosen as they are established risk factors for the development of MACE following revascularisation. Comparison of the above risk factors with AF was performed to illustrate the magnitude of the impact of AF on outcomes. Indication for revascularisation varied, with 3645 (54.4%) patients presenting with ACS, 1042 (15.5%) patients with staged PCI, 1832 (27.3%) patients with stable CAD. Baseline medical therapy included 6144 (91.6%) patients with ASA, 6144 (91.6%) patients with P2Y12 inhibitors, 3164 (47.2%) patients with ACE inhibitor or angiotensin receptor blocker (ACEi/ARB), 3478 (51.9%) patients with beta-blockers and 5001 (74.6%) patients with statins.

Of the 761 (11.4%) patients with AF, 627 (82.4%) patients had non-valvular AF and 134 (17.6%) patients had valvular AF. Rate control strategy was observed in 581 (78.4%) patients, with 414 (54.4%) patients on beta-blockers, 115 (15.1%) patients on calcium channel blockers and 52 (6.8%) patients on digoxin. CHA2DS2-VASC score was ≥2 in 661 (86.9%) patients with AF, with 476 (64.2%) patients on OAC.

Clinical outcomes

Follow-up was completed in all patients, with a median time of follow-up of 12.8 (IQR 11.2–15.9) months. One-year MACE occurred in 166 (21.8%) and 683 (11.5%) patients in AF and non-AF groups, respectively (unadjusted HR 1.96; 95% CI 1.66 to 2.33; p<0.0001; figure 2A). Similar results were observed when new-onset AF was grouped with non-AF at baseline compared with pre-existing AF (unadjusted HR 1.89; 95% CI 1.54 to 2.33; p<0.0001; Online supplemental figure 1). Mortality in the cohort was low, with a 1-year death rate of 10.5% and 4.9% in the AF and non-AF group, respectively (unadjusted HR 2.17; 95% CI 1.69 to 2.78; p<0.0001; figure 2B). Repeat MI was infrequent occurring in 26 (3.4%) and 97
Figure 2  Kaplan-Meier curves following revascularisation stratified by non-atrial fibrillation (AF) and AF. (A) Patients with cumulative incidence of 1-year major adverse cardiac events (MACE). AF was associated with 1-year MACE (HR, 1.97; 95% CI 1.66 to 2.33; p<0.0001). (B) Patients with cumulative incidence of 1-year mortality. AF was associated with 1-year mortality (HR 2.17; 95% CI 1.69 to 2.78; p<0.0001). (C) Patients with cumulative incidence of 1-year myocardial infarction (MI). AF was associated with 1-year MI (HR 2.13; 95% CI 1.38 to 3.28; p=0.0005). (D) Patients with cumulative incidence of 1-year cerebrovascular accident. AF was associated with 1-year cerebrovascular accident (HR 2.55; 95% CI 1.50 to 4.33; p=0.0003). (E) Patients with cumulative incidence of 1-year unplanned revascularisation. AF was associated with 1-year unplanned revascularisation (HR 1.48; 95% CI 1.13 to 1.93; p=0.004). (F) Patients with cumulative incidence of 1-year bleeding. AF was associated with 1-year bleeding (HR, 1.65; 95% CI 1.04 to 2.61; p=0.03). All HRs are unadjusted values. A p<0.05 was considered statistically significant. CVA, cerebrovascular accident.
(1.6%) patients in the AF and non-AF group, respectively (unadjusted HR 2.13; 95% CI 1.38 to 3.28; p=0.0005; figure 2C). Comparatively, stroke, unplanned revascularisation and bleeding also remained higher in the AF group (figure 2D–F). Following adjustment for age, sex, type 2 diabetes, hypertension, dyslipidaemia, ACS, prior stroke, vascular disease and anticoagulation, AF remained independently associated with the outcomes (table 2).

New-onset versus non-AF

In the first 12 months, 299 episodes of new-onset AF occurred (4.5%). We evaluated differences between non-AF (n=6704) and new-onset AF (n=299) following revascularisation. Differences in 1-year MACE were observed between non-AF and new-onset AF (HR 1.96; 95% CI 1.52 to 2.53; p<0.0001; figure 3A). Similar to patients with established AF, mortality was higher in the new-onset AF group (4.9% vs 8.0%, HR 1.63; 95% CI 1.07 to 2.47; p=0.02; figure 3B). One-year bleeding, cerebrovascular accident (CVA) and myocardial infarction (MI) rates similarly remained higher in the new-onset AF group (figure 3C–E). The association between 1-year MACE and new-onset AF remained following adjustment for the prespecified variables (table 3).

AF and other cardiovascular risk factors

To enable comparison of risk between AF and traditional risk factors, we evaluated the relative contribution of AF to MACE risk following revascularisation in a multivariable model. Advanced age remained the greatest risk factor for MACE but was closely followed by AF and DM (age ≥75 HR 1.68, 95% CI 1.45 to 1.96; AF HR 1.55, 95% CI 1.30 to 1.86; diabetes HR 1.50, 95% CI 1.30 to 1.74; left ventricular (LV) dysfunction HR 1.29, 95% CI 1.01 to 1.65; and female sex HR 1.25, 95% CI 1.08 to 1.45; figure 4).

DISCUSSION

AF remains a highly prevalent condition strongly associated with advancing age and accumulation of cardiovascular risk factors. In our analysis, we make several observations with implications for prognosis of patients undergoing revascularisation. First, patients without established AF have an annualised incidence of new-onset AF of 4.5%, or 1 in 20 patients developing the rhythm in follow-up. Moreover, both baseline AF as well as new-onset AF remain independent predictors of MACE, an association that remained following adjustment for traditional cardiovascular risk factors. In fact, in 1 in 5 patients with AF experienced an adverse event with 1 in 10 patients dying in the first 12 months. While not directly causal, the markedly higher event rates seen in this cohort of patients highlight the need for focused studies of this high-risk cohort to evaluate whether disease specific interventions could modify risk.

Overall, AF was prevalent in patients with CAD undergoing revascularisation with 11.4% of patients with pre-existing or new-onset AF. Similar to previous studies, patients with AF had greater comorbid burden and advanced age compared with the non-AF cohort. Not surprisingly, differences in anticoagulation use were observed between AF and non-AF, along with differences in postprocedural antiplatelet, ACEi/ARB and beta-blocker medications. However, when adjusting for major confounders, AF remained independently and strongly predictive of MACE. While the implications for prognosis and monitoring are clear, it remains uncertain if AF is simply a marker of more advanced disease or a bona fide target for intervention in this cohort of patients.

Indeed, AF remains associated with a proinflammatory and prothrombotic state that may promote atherosclerosis and thrombosis leading to MACE. AF also increases thromboembolic risk and is more commonly treated with direct OAC or vitamin K antagonists to prevent the risk of ischaemic stroke, which increases the risk of bleeding. Patients with AF have a higher comorbid burden of metabolic syndrome, dyslipidaemia and peripheral artery disease, along with a prothrombotic state characterised by increased platelet activation. Finally, loss of atrioventricular synchrony can decrease effective cardiac output in patients with reduced LV function. Any and each of these may in part contribute

### Table 2: Clinical outcomes between non-AF and AF

|                     | Total (n=6704) | AF (n=761) | Non-AF (n=5943) | Unadjusted HR (95% CI) | Adjusted HR (95% CI)* |
|---------------------|---------------|------------|-----------------|------------------------|------------------------|
| 1-year outcomes     |               |            |                 |                        |                        |
| MACE                | 849 (12.7%)   | 166 (21.8%)| 683 (11.5%)     | 1.97 (1.66 to 2.33)    | 1.61 (1.29 to 2.01)    |
| Mortality           | 373 (5.6%)    | 80 (10.5%) | 293 (4.9%)      | 2.17 (1.69 to 2.78)    | 1.31 (0.95 to 1.80)    |
| Myocardial infarction| 123 (1.8%)   | 26 (3.4%)  | 97 (1.6%)       | 2.13 (1.38 to 3.28)    | 1.96 (1.11 to 3.46)    |
| Cerebrovascular accident | 74 (1.1%) | 18 (2.4%)  | 56 (0.9%)       | 2.55 (1.50 to 4.33)    | 1.45 (0.71 to 2.99)    |
| Unplanned revascularisation | 410 (6.1%) | 64 (8.4%)  | 346 (5.8%)      | 1.48 (1.13 to 1.93)    | 1.52 (1.08 to 2.15)    |
| Bleeding            | 129 (1.9%)    | 22 (2.9%)  | 107 (1.8%)      | 1.65 (1.04 to 2.61)    | 1.35 (0.73 to 2.50)    |

*Adjusted for age, sex, type 2 diabetes, hypertension, dyslipidaemia, acute coronary syndrome, prior CVA, vascular disease and anticoagulation.

AF: atrial fibrillation; CVA: cerebrovascular accident; MACE: major adverse cardiac event.
Coronary artery disease

Figure 3  Kaplan-Meier curves following revascularisation stratified by non-atrial fibrillation (AF) and new-onset AF. (A) Patients with cumulative incidence of 1-year MACE. New-onset AF was associated with 1-year MACEs (HR 1.96; 95% CI 1.52 to 2.53; p<0.0001). (B) Patients with cumulative incidence of 1-year mortality. New-onset AF was associated with 1-year mortality (HR 1.63; 95% CI 1.07 to 2.47; p=0.02). (C) Patients with cumulative incidence of 1-year myocardial infarction (MI). New-onset AF was associated with 1-year MI (HR 2.27; 95% CI 1.22 to 4.24; p=0.008). (D) Patients with cumulative incidence of 1-year cerebrovascular accident. New-onset AF was associated with 1-year cerebrovascular accident (HR 3.57; 95% CI 1.82 to 6.99; p<0.0001). (E) Patients with cumulative incidence of 1-year unplanned revascularisation. New-onset AF was associated with 1-year unplanned revascularisation (HR 1.59; 95% CI 1.07 to 2.35; p=0.02). (F) Patients with cumulative incidence of 1-year bleeding. New-onset AF was associated with 1-year bleeding (HR 2.06; 95% CI 1.11 to 3.83; p=0.02). All HRs are unadjusted values. A p<0.05 was considered statistically significant. CVA, cerebrovascular accident, MACE, major adverse cardiac event.
Chapter 4

Figure 4 Predictors of major adverse cardiac events following revascularisation. Variables include traditional cardiovascular risk factors along with atrial fibrillation and reduced LVEF. Data were presented as HRs with corresponding 95% CIs. A p < 0.05 was considered statistically significant. CAD, coronary artery disease; LVEF, left ventricular ejection fraction.

Table 3 Clinical outcomes between new-onset and no atrial fibrillation (AF)

| Variable                              | No AF (n=5943) | New-onset AF (n=299) | Unadjusted HR (95% CI) | Adjusted HR (95% CI)* |
|---------------------------------------|---------------|----------------------|------------------------|-----------------------|
| MACE                                  | 683           | 11.5                 | 65                     | 21.7                  | 1.96 (1.52 to 2.53) | 1.27 (1.09 to 1.47) |
| Mortality                             | 293           | 4.9                  | 24                     | 8.0                   | 1.63 (1.07 to 2.47) | 0.97 (0.76 to 1.25) |
| Myocardial infarction                 | 97            | 1.6                  | 11                     | 3.7                   | 2.27 (1.22 to 4.24) | 1.56 (1.08 to 2.25) |
| Cerebrovascular accident              | 56            | 0.9                  | 10                     | 3.3                   | 3.57 (1.82 to 6.99) | 1.55 (1.03 to 2.33) |
| Unplanned revascularisation           | 346           | 5.8                  | 27                     | 9.0                   | 1.59 (1.07 to 2.35) | 1.31 (1.05 to 1.65) |
| Bleeding                              | 107           | 1.8                  | 11                     | 3.7                   | 2.06 (1.11 to 3.83) | 1.27 (0.86 to 1.86) |

*adjusted for age, sex, type 2 diabetes, hypertension, dyslipidemia, acute coronary syndrome, prior CVA, vascular disease, and anticoagulation.

MACE, major adverse cardiac event.

Conclusions:

Addressing AF in tandem with the treatment of underlying disease is an effective therapeutic strategy in patients with a multitude of clinical syndromes. In patients with heart failure, for example, catheter ablation for AF is associated with decreased mortality. In the post-CABG setting, management of AF is recommended by many governing bodies, with no difference between rate and rhythm control strategies in these patients. However, prior descriptions have not assessed the risk in all patients undergoing revascularisation, specifically a cohort including patients with stable CAD. Our study demonstrates that AF is a significant contributor of MACE and merits strong consideration as part of secondary prevention along with management of underlying risk factors (figure 4). Current guidelines addressing AF in this population recommend the use of anticoagulant therapy along with P2Y12 inhibitors for prevention of thromboembolic events among patients undergoing PCI. Recent studies have demonstrated the effectiveness of maintaining sinus rhythm in lowering the risk of MACE compared with usual care. Maintenance of sinus rhythm represents a potential target for therapy; however, prospective studies elucidating the effect in patients undergoing revascularisation will be useful as a potential role in secondary prevention.

Certainly, our study is not without limitations. First, while the data and outcomes are prospectively indexed, the analysis remains retrospective and the sample size limited by available patients in follow-up. Second, while the registry provides high fidelity data owing to the physician assessments and patient-level data, the evaluation of the timing and interventions for AF are not routinely collected. Thus, strategies such as rhythm control vs rate control were not available for comparative analysis. Finally, the database used for this analysis did not include any data regarding patient-reported outcome measures (PROM). Given the link between PROM and hard outcomes in other disease states, reporting on PROMs in this patient population represents an area for future studies.

CONCLUSION

Preprocedural and new-onset AF following revascularisation are strongly predictive of 1-year MACE. AF should be considered as an isolated risk factor when risk stratifying patients prior to revascularisation. Dedicated studies of patients with AF undergoing revascularisation are warranted to identify therapeutic targets to reduce cardiovascular events.

Contributors

RGJ: data collection, study design, data analysis, writing of the manuscript. OA-R: data collection, study design, data analysis, writing of the manuscript. DM: data collection, writing of the manuscript. JS: data collection, writing of the manuscript. CS: data collection, writing of the manuscript. PDS: data collection, study design, writing of the manuscript. TC: data collection, writing of the manuscript. SP: data collection, writing of the manuscript. CYG: data collection, writing of the manuscript. OA-H: data collection, writing of the manuscript. BH: data collection, study design, data analysis, funding, writing of the manuscript.
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