

Cite this article as: Schwann TA, Al-Shaar L, Engoren MC, Bonnell MR, Goodwin M, Schwann AN *et al.* Effect of new-onset atrial fibrillation on cause-specific late mortality after coronary artery bypass grafting surgery. *Eur J Cardiothorac Surg* 2018;54:294–301.

Effect of new-onset atrial fibrillation on cause-specific late mortality after coronary artery bypass grafting surgery†

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Received 16 October 2017; received in revised form 9 January 2018; accepted 11 January 2018

Abstract

OBJECTIVES: Postoperative atrial fibrillation (POAF) is a common complication after coronary artery bypass grafting. Although transient, POAF is linked to increased late mortality. We hypothesized that POAF increases late cerebrovascular (CeV) and composite cerebrovascular/cardiovascular/vascular (CV* = CeV + CV + Other-V) but not non-cardiovascular (Non-CV) mortality.

METHODS: We analysed 8807 non-salvage coronary artery bypass grafting patients (1994–2011). Fifteen-year and time-segmented (early, 0–1 year; intermediate, 1–6 years and late, 6–15 years) all-cause and cause-specific mortality were compared for POAF versus No-POAF patients. Corresponding POAF versus No-POAF adjusted hazard ratios [AHRs (95% confidence interval, CI)] were derived using the competing risk Cox regression.

RESULTS: POAF occurred in 1992 (23%) patients. Complications other than POAF occurred in 1875 (21%) patients but were more frequent among POAF patients (31% vs 18%; $P < 0.001$). Overall mean follow-up was 9 ± 4 years. POAF patients had a higher 15-year unadjusted mortality (53% vs 39%; $P < 0.001$) and were consequently associated with higher adjusted all-cause [AHR (95% CI) = 1.23 (1.14–1.33)] and composite cardiovascular [CV*: AHR (95% CI) = 1.15 (1.02–1.30)] mortality. The trends towards a higher 15-year CeV [AHR (95% CI) = 1.34 (0.94–1.91)] and Non-CV [AHR (95% CI) = 1.12 (0.99–1.26)] mortality were not significant. Time-segmented analyses showed that (i) POAF increased all-cause mortality early, and this persisted in the intermediate and late periods and (ii) CeV [AHR (95% CI) = 2.14 (1.14–4.04)] and CV* [AHR (95% CI) = 1.31 (1.06–1.62)] mortality rates were increased in the intermediate but not in the early or late periods. Non-CV mortality was similar in POAF and No-POAF for all time intervals. These findings were corroborated in propensity-matched sub-cohorts and in sensitivity analyses in patients free of any other complication.

CONCLUSIONS: POAF is associated with worse long-term survival principally driven by increased intermediate-term cerebrovascular and cardiovascular mortality, while Non-CV mortality was similar.

Keywords: New-onset postoperative atrial fibrillation • Coronary artery bypass grafting • Mortality • Cause specific mortality

INTRODUCTION

New-onset postoperative atrial fibrillation (POAF) is a frequent postoperative complication affecting 15–40% of patients undergoing coronary artery bypass grafting (CABG) [1–10]. Although frequently limited in duration and traditionally viewed as a temporary and benign perioperative ‘nuisance’ with 98% of patients regaining normal sinus rhythm within 2 months of surgery [11], more recent data suggest that POAF is associated with increased

acute perioperative mortality, morbidity and resource utilization [1, 2, 5, 6, 9]. Beyond this acute risk, we and others have reported that POAF is also associated with increased late all-cause mortality [1–5, 7–9]. The mechanism behind this increased mortality is unclear [8, 12–15], and there is a paucity of data on cause-specific mortality in cardiac surgical patients who develop POAF. This is in contradistinction to the general non-surgery population, where atrial fibrillation increases subsequent mortality due to cardiovascular causes including sudden cardiac death, heart failure and stroke [13, 16–20].

We hypothesize that if the increase in late death after CABG is associated with POAF, it should manifest as an increase in

†Presented at the 31st Annual Meeting of the European Association for Cardio-Thoracic Surgery, Vienna, Austria, 7–11 October 2017.

cerebrovascular, cardiovascular or vascular events causing late mortality. This study explores the association between POAF and cause-specific long-term mortality and the chronology of this risk in CABG patients.

METHODS

We retrospectively analysed adult cardiac surgery data from 3 Ohio centres, which were prospectively collected in accordance with the Society of Thoracic Surgeons (STS) database definitions and criteria. This study was approved by the respective institutional review boards. Data collection did not require additional review of hospital records or interview of patients, and informed consent requirement was waived.

Study population

Patients undergoing isolated CABG or CABG with/without concurrent carotid reconstruction between 1994 and 2012 were studied. Patients were excluded in case of concomitant valvular, congenital cardiac or aortic surgery, history of preoperative atrial fibrillation/flutter or salvage status operations.

Perioperative management

POAF was defined, according to the STS database, as the occurrence of POAF or atrial flutter requiring treatment (either pharmacological or electrical) throughout the index hospitalization or within 30 days postoperatively. Telemetry was used in all patients during hospitalization. The operative technique [21] and perioperative management of POAF were previously described [3].

Primary end-points and follow-up

All-cause mortality was secured from searches of the US Social Security Death Index (<http://ssdi.genealogy.rootsweb.com>; last accessed November 2011). This was complemented by searching the Ohio State Death Registry for all study years (1994–2013) to confirm death as well as the primary cause of death using International Classification of Diseases codes [ICD9 (1996–1998) and ICD10 (1999–2013)]. The causes of death were categorized as follows: cerebrovascular [CeV; ICD10 (I60–I69), ICD9 (430–434, 436–438)]; cardiovascular [CV; ICD10 (I00–I09, I11, I13, I20–I51), ICD9 (390–398, 402, 404, 410–429)]; other vascular [Other-V; ICD10 (I10, I12, I15, I70, I71–78, I80–I99), ICD9 (401, 403, 40, 441–448, 451–459)] and non-cardiovascular (Non-CV; all other ICD9/ICD10 codes). A composite cerebrovascular/cardiovascular/vascular (CV*) death category was also used (CV* = CeV + CV + Other-V).

Postoperative complications were classified as follows: (i) severe [reoperation for valvular dysfunction or graft occlusion, myocardial infarction, deep sternum infection, septicaemia, stroke, coma, pulmonary embolism, renal failure, dialysis (newly required), cardiac arrest, multisystem failure and aortic dissection], (ii) moderate (reoperation for bleeding/tamponade or other cardiac reasons, thoracotomy, pneumonia, reintubation, tamponade, iliac/femoral dissection, anticoagulant event, heart block or acute limb ischaemia) or (iii) mild (any other complication). Transfusions were not considered as a complication.

Statistical methods

Continuous and categorical data were expressed as mean \pm standard deviation and counts (%), respectively. Bivariate comparisons were conducted with the independent *t*-test or the χ^2 test as applicable. Time-to-event analyses were truncated after 15 years because of the small number of patients with follow-up beyond 15 years. Time 0 for longitudinal follow-up was defined as the time of surgery. Covariate-adjusted hazard ratios with 95% confidence intervals [AHRs (95% CIs)] were calculated using competing risk regression models to quantify the POAF versus No-POAF effect on overall (0–15 years) post-discharge mortality. These models adjusted for possible confounding due to differences in patient demographics, preoperative risk factors and comorbidity, operative data including completeness of revascularization (difference between the number of coronary grafts and the number of diseased coronary systems) and choice of grafting conduits, the presence and severity of postoperative complications using a 4-category variable (none, mild, moderate and severe) and the year of surgery [continuous variable 1994 = 1, 2012 = 19] to account for potential variance in reporting POAF or its treatment (Table 1). AHRs were calculated for all-cause mortality, CeV, CV* and Non-CV. To relax the proportional hazard assumption, post-discharge mortality were then analysed in a time-segmented fashion as follows: early mortality, 0–1 year; intermediate mortality, 1–6 years and late mortality, 6–15 years. As a sensitivity analysis, the 15-year and time-segmented calculations based on the entire patient series ('all patients') were repeated after excluding patients who experienced any complication due to any other severity other than POAF ('no complication'). Finally, to validate our results, the entire analysis was repeated in propensity-matched POAF and No-POAF sub-cohorts. Herein, propensity scores or the probabilities of developing POAF were derived from non-parsimonious logistic regression models based on all variables included in covariate adjustment, irrespective of their statistical significance (Table 1). Two different propensity scores were calculated for 'all patients' and 'no complications patients', with both exhibiting moderately good discrimination between POAF versus No-POAF patients. Standardized differences were reduced substantially for the matched sub-cohorts to well below 10% in all cases (0.1–5.6%).

A *P*-value <0.05 was used to indicate significance. Analyses were done using SPSS version 23.0 software (IBM, Armonk, NY, USA) and Stata statistical (StataCorp 2013) software.

RESULTS

The 8807 CABG study population included 3239 (37%) patients with one or more complications, while 5568 (63%) patients had no postoperative complications. New onset of POAF was the most frequent complication ($n=1992$; 23% overall and 61% of complicated patients). Other types of complications were more frequent in case of POAF [628 of 1992 (31%) vs No-POAF, 1247 of 6815 (18%); $P < 0.001$] and were serious more often (moderate/severe: 72% vs 59%; $P < 0.001$). A detailed breakdown of complications in POAF and No-POAF cohorts is provided in [Supplementary Material, Table S1](#).

Overall baseline characteristics and operative data for POAF versus No-POAF groups were compared for the overall

Table 1: Patient characteristics and operative data for CABG cohorts with and without POAF

All patients (n = 8807)	All patients		No other complications	
	No-POAF (n = 6815)	POAF (n = 1992)	No-POAF (n = 5568)	POAF (n = 1364)
Continuous, mean ± SD				
Age (years)	63 ± 11	68 ± 10	62 ± 11	68 ± 10
BSA (m ²)	2 ± 0.2	2 ± 0.3	2.03 ± 0.25	2.0 ± 0.2
BMI (kg/m ²)	30 ± 5.6	30 ± 6	30 ± 5	30 ± 6
Ejection fraction (%)	49 ± 11	48 ± 12	50 ± 11	49 ± 11
Perfusion time (min)	79 ± 40	81 ± 41	77 ± 38	77 ± 37
Number of grafts	3 ± 1	3 ± 1	3 ± 1	3 ± 1
Revascularization index	0.56 ± 0.85	0.57 ± 0.85	0.57 ± 0.85	0.59 ± 0.83
Categorical (%)				
Male	68.5	70.5	69.2	72.4
Mild obesity (BMI 30–34.99 kg/m ²)	26.0	24.8	26.4	24.5
Moderate obesity (BMI 35–39.99 kg/m ²)	10.3	10.3	10.0	9.5
Severe obesity (BMI ≥40 kg/m ²)	5.0	7.1	4.5	6.3
Current smoker	22.4	17.7	22.6	17.2
Non-insulin-dependent diabetes	22.6	25.7	22.2	24.6
Insulin-dependent diabetes	13.2	11.8	12.2	11.0
Renal failure	3.1	4.1	2.6	3.4
Hypercholesterolaemia	70.9	69.8	71.9	71.3
Hypertension	80.1	83.7	79.6	83.1
Chronic lung disease	19.5	23.6	18.7	21.2
Peripheral vascular disease	16.0	20.9	14.8	18.7
Cerebrovascular disease	22.8	29.2	21.4	26.8
Myocardial infarction	54.4	57.1	53.1	53.2
Congestive heart failure	10.8	15.0	9.4	12.2
Arrhythmia (other than AF)	8.8	8.5	8.1	8.7
Multivessel disease	93.7	95.5	93.4	94.0
Three-vessel disease	71.6	76.4	70.7	73.2
Cardiogenic shock	1.1	1.3	0.6	0.9
PCI				
Balloon angioplasty	8.7	7.0	8.8	7.2
Stent	11.1	10.9	10.8	12.2
Medications				
Aspirin	71.9	70.3	71.9	69.8
Digitalis	4.3	5.8	3.7	5.4
Beta-blockers	66.0	69.6	65.9	70.2
Steroids	2.3	3.1	2.2	2.5
Inotropic	1.8	1.8	1.2	1.3
Operative data				
Reoperation	4.6	6.1	4.0	5.2
Isolated CABG	91.2	87.9	92.3	89.7
Urgent case status	58.7	57.7	58.4	56.9
Emergent case status	6.2	7.2	5.2	6.0
All arterial grafting	12.9	11.3	13.3	11.9
Bilateral ITA grafts	3.1	2.8	3.1	2.1
Radial artery grafts	37.2	40.5	38.0	42.5
Transfusion (any)	33.7	47.1	28.5	36.7

AF: atrial fibrillation; BMI: body mass index; BSA: body surface area; CABG: coronary artery bypass graft surgery; ITA: internal thoracic artery; POAF: new-onset postoperative atrial fibrillation; PCI: percutaneous coronary intervention.

population ('all patients') and for patients who did not experience other types of complications ('no complications') in Table 1. POAF patients were generally older, past smokers, more obese, had lower ejection fraction and had more preoperative comorbidities such as renal failure, hypercholesterolaemia, chronic lung disease, peripheral vascular disease, cerebrovascular disease and 3-vessel disease. They also had more preoperative use of medications such as digitalis, beta-blockers and steroids. POAF patients were also more frequent in cases of urgent and emergent operations, reoperation, non-isolated CABG, longer cross-clamp and perfusion times and when perioperative transfusions were administered.

Unadjusted survival

Patients were followed up for an average of 9 ± 4 years. Unadjusted 15-year mortality was substantially worse with POAF in the entire study population and also in the sub-cohort of patients not experiencing additional complications (Fig. 1; both $P < 0.001$). Total deaths over 15 years for the entire patient population were 1053 (53%) and 2641 (39%) for the POAF and No-POAF cohorts, respectively ($P < 0.001$). The corresponding overall and cause-specific (CeV, CV* and Non-CV) event counts at 1, 3, 6, 9, 12 and 15 years after CABG for POAF and No-POAF cohorts are detailed in Fig. 1, which showed that the relative incidence of

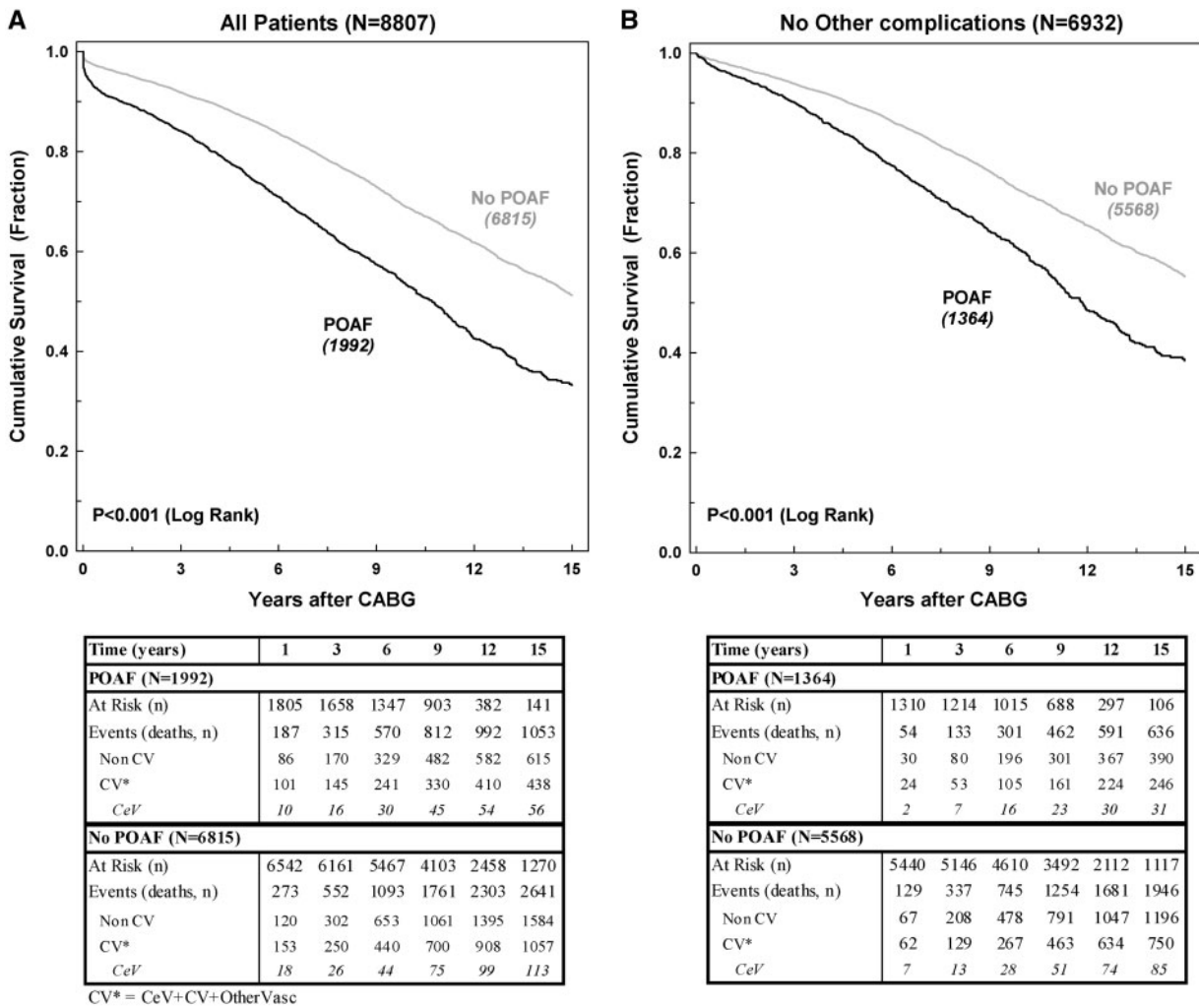


Figure 1: Comparison of unadjusted 15-year Kaplan-Meier survival estimates in CABG patients experiencing POAF versus No-POAF: (A) All patients irrespective of other complications and (B) patients with no other complications ('no complications'). Tables below each plot provide comparative breakdowns of patients at risk in addition to overall death events and cause-specific event counts. CABG: coronary artery bypass grafting; Non-CV: non-cardiovascular; CeV: cerebrovascular; CV*: composite cerebrovascular/cardiovascular/vascular; POAF: postoperative atrial fibrillation.

cause-specific death varied as a function of time after CABG, and these trends also differed for the POAF and No-POAF sub-cohorts. For example, in POAF versus No-POAF patients, CeV deaths (6% vs 4%), in the intermediate period (1-6 years), contributed an appreciably greater fraction of all known deaths, while this difference was relatively less for CV* deaths (44% vs 42%) and was actually reversed for Non-CV deaths (56% vs 58%).

Risk-adjusted survival

For the overall patient population, comprehensive covariate adjustment (including incidence and severity of other complications) and accounting for competing risks showed that POAF is a significant independent predictor of worse 15-year all-cause [AHR (95% CI)=1.23 (1.14-1.33)] and CV* [AHR (95% CI)=1.15 (1.02-1.30)] mortality but not for CeV [AHR (95% CI)=1.34 (0.94-1.91)] and Non-CV [AHR (95% CI)=1.12 (0.99-1.26)] mortality (Fig. 2A). The corresponding time-segmented regression analysis showed that POAF increased all-cause mortality relative to No-POAF in the early, intermediate and late term, but with decreasing effect magnitude (Fig. 2A). Importantly, however, CeV and

CV* mortality risk increased significantly only for the intermediate 1-6-year follow-up period with AHRs (95% CIs) of 2.14 (1.14-4.04) and 1.31 (1.06-1.62), respectively. These same regression analyses, restricted to patients not experiencing any other complications as a sensitivity analysis, showed largely similar and comparable magnitude POAF effects for both overall 15-year mortality and time-segmented analyses (Fig. 2B). The other confirmatory sensitivity analysis in propensity-matched sub-cohorts also showed consistent findings, and these results are summarized in Fig. 3.

DISCUSSION

Our findings add to an appreciable body of evidence that new onset of POAF following CABG is associated with increased long-term mortality. Our analysis uniquely informs the surgical community that the long-term mortality risk associated with POAF is (i) principally cerebrovascular, cardiovascular and vascular in nature (composite CV*) and (ii) the composite CV* and cerebrovascular mortality risks are predominantly evident in the intermediate postoperative term. These results were shown to be



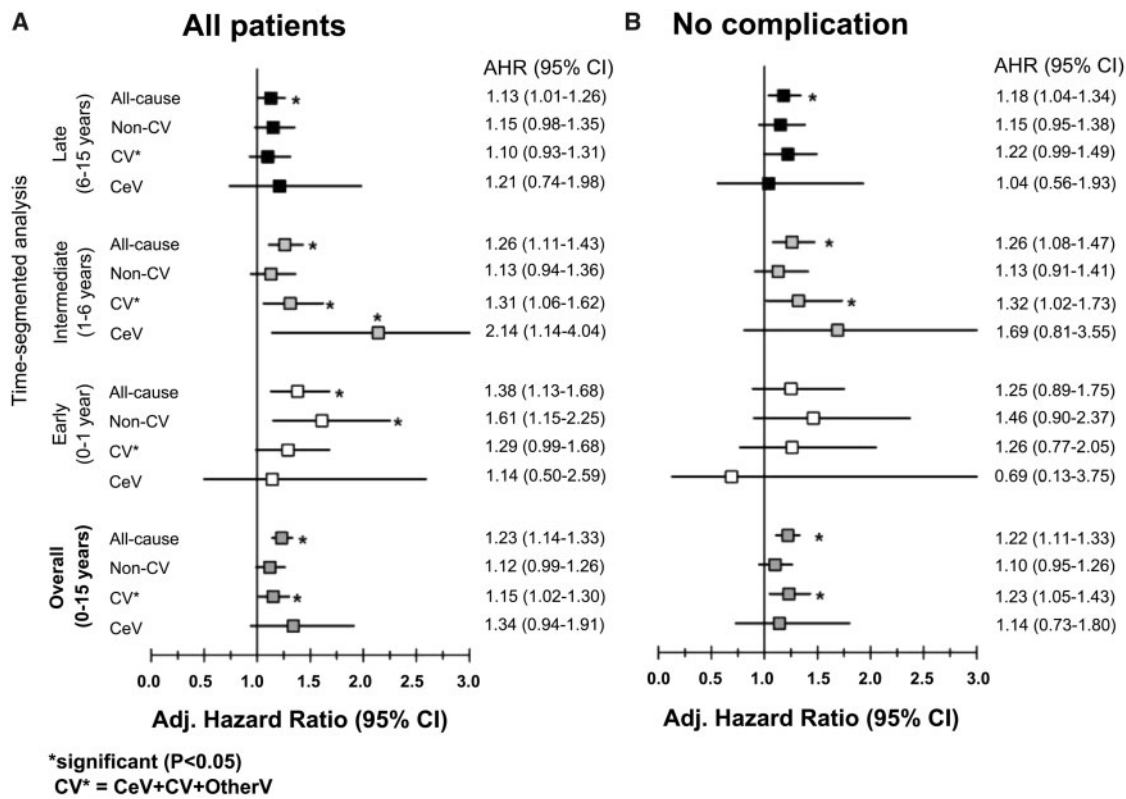


Figure 2: Forest plots showing the 15-year (overall) and time-segmented (early, intermediate and late) POAF versus No-POAF hazard ratios [AHR (95% CI)] for the overall population adjusted via comprehensive covariate risk adjustment: (A) all patients and (B) no complications patients. AHRs were shown for all cause and cause-specific mortality (CeV, CV* and Non-CV). AHR: adjusted hazard ratio; Non-CV: non-cardiovascular; CeV: cerebrovascular; CI: confidence interval; CV*: composite cerebrovascular/cardiovascular/vascular; POAF: postoperative atrial fibrillation.

highly robust in a series of complementary sensitivity analyses including restricting the analysis to patients experiencing no other complications and in propensity-matched POAF and No-POAF sub-cohorts. Also, noteworthy was the fact that the risk adjustment used in this study to ascertain long-term effects included operative factors [revascularization index, number and type (arterial or venous) of coronary grafts, cardiopulmonary bypass duration and transfusions] as well as postoperative factors (transfusion and incidence of severity of complications). Such a comprehensive approach to risk adjustment is needed, given that these factors have been shown to independently predict long-term outcomes after CABG [22], and most or all of these factors are highly dependent on patient age, which itself is a powerful predictor of new onset of POAF [23]. The adverse effects of POAF on patient outcomes and resource utilization are well recognized, yet the extensive efforts to decrease the rate of POAF have been constrained by the incomplete understanding of the specific causes of POAF, although inflammatory mechanisms and enhanced oxidative stress have been implicated [24, 25].

Our finding of increased long-term mortality associated with POAF corroborates the results of others [1, 2, 4, 7-9]. The increased risk of death in our POAF study group [covariate AHR (95% CI)=1.23 (1.14-1.33) and propensity score AHR (95% CI)=1.15 (1.05-1.26)] was similar in magnitude to the POAF mortality HR reported by Villareal *et al.* [9] [HR (95% CI)=1.50 (1.26-1.77)] and Ahlsson *et al.* [1] [HR (95% CI)=1.56 (1.23-1.98)] but lower than the 4-year mortality in a meta-analysis by Kaw *et al.* [5] [HR (95% CI)=2.19 (1.97-2.45)]. We have previously shown a similar long-term risk of POAF in a sub-cohort of CABG patients

who did not experience any other perioperative complication [3], suggesting that the increased mortality was attributable to POAF rather than to its interaction with other perioperative complications that may independently adversely impact survival. The noted increased all-cause mortality risk of POAF is highest in the early (0-1 years) postoperative period [AHR (95% CI)=1.38 (1.13-1.68)] and gradually declines thereafter to AHR (95% CI)=1.26 (1.11-1.43) in 1-6 years postoperatively and to AHR (95% CI)=1.13 (1.01-1.26) between 6 and 15 years. Thus, closer monitoring of POAF patients should be considered to possibly mitigate this increased mortality risk, especially in the light of recent reports indicating that even short-lived and subclinical atrial fibrillation detected incidentally by various implanted electrophysiology devices are associated with increased risk of adverse outcomes including stroke and embolic events [26].

In a previous report, we noted that the unadjusted survival of CABG patients with POAF who developed additional perioperative complications was worse compared with patients without such additional complications [3]. It is unclear whether POAF and these other complications comprised a self-perpetuating forward feedback loop resulting in the noted increased mortality or alternatively, whether the noted perioperative complications were simply a marker of sicker patients undergoing CABG, and it was, in fact, their concurrent comorbidities that largely drove the observed higher mortality risk. To clarify this, in this study, we included concurrent complications and their severity in our risk adjustment models, and in this analysis, POAF was still associated with increased all-cause mortality. Fauchier *et al.* [19] observed an increased risk of atrial fibrillation-associated mortality in the

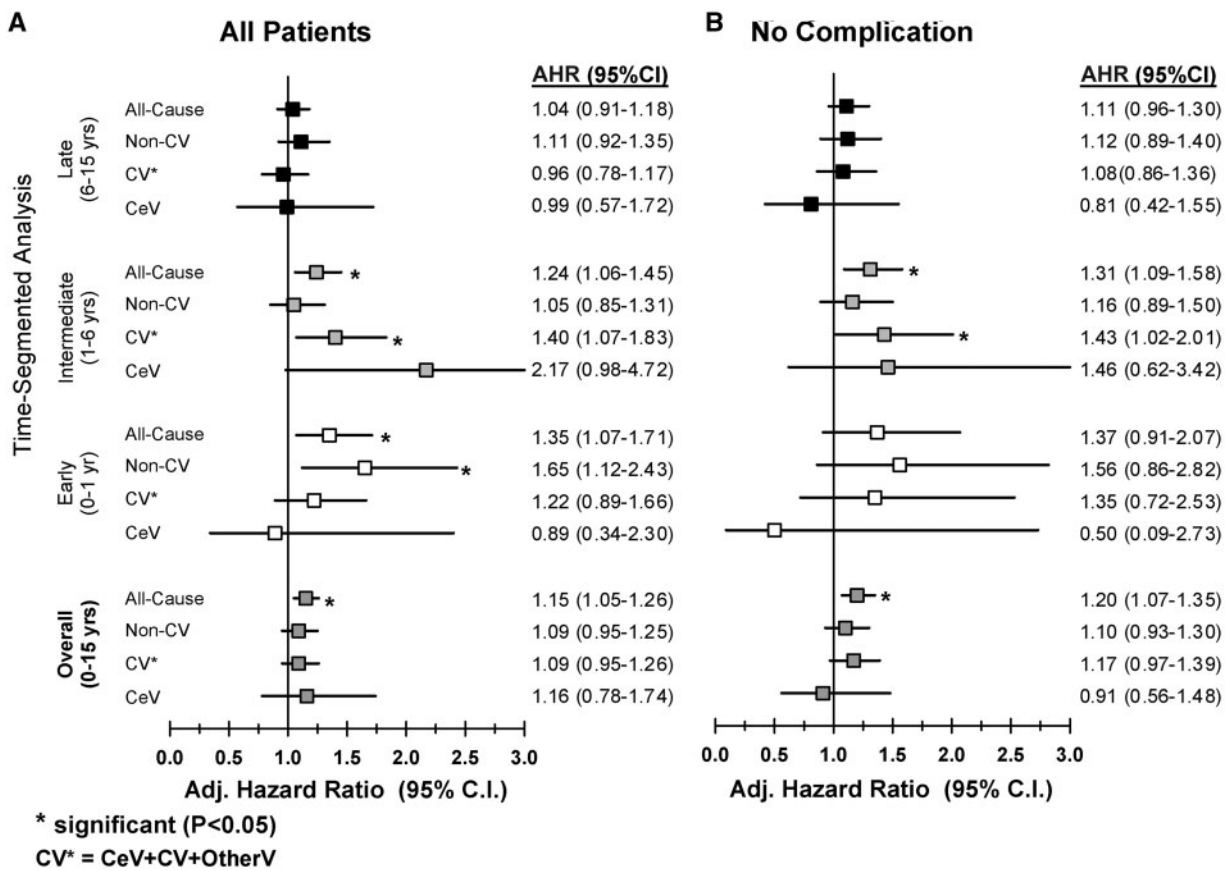


Figure 3: Forest plots showing the 15-year (overall) and time-segmented (early, intermediate and late) POAF versus No-POAF hazard ratios [AHR (95% CI)] in propensity-matched patient sub-cohorts: (A) all matched patients and (B) matched no complications patients. AHRs were shown for all-cause and cause-specific mortality (CeV, CV* and Non-CV). AHR: adjusted hazard ratio; Non-CV: non-cardiovascular; CeV: cerebrovascular; CI: confidence interval; CV*: composite cerebrovascular/cardiovascular/vascular; POAF: postoperative atrial fibrillation.

general population who had concurrent baseline comorbidities compared with the risk in patients without such comorbidities. Eisen *et al.* [18] also noted increased risk of sudden cardiac death in patients with atrial fibrillation who also had a history of prior myocardial infarction or heart failure.

Patients who developed POAF in our cohort were generally older and sicker than those who did not develop POAF (Table 1). LaPar *et al.* [6], in a statewide registry, also reported that POAF tended to develop in a higher risk patient population with an STS perioperative mortality risk of 2% and 1% among POAF and No-POAF patients, respectively. Our POAF patients had longer cardiopulmonary bypass and aortic cross-clamp times, despite receiving a similar number of coronary grafts as did No-POAF patients. Others also found increased operative times with POAF [4, 6, 9]. Although there is significant overlap between the risk factors for the development of POAF and factors associated with long-term mortality, most evidence suggests that POAF is an independent risk for long-term mortality rather than just a surrogate marker for sicker patients whose comorbidities, rather than the pathophysiology of POAF, impacts survival negatively [1, 2, 4, 5, 7, 8].

While our data link the POAF-associated increased late mortality to cerebrovascular, cardiac and other vascular causes, the direct pathophysiological mechanisms behind the noted increased mortality in patients developing POAF remain undefined and are beyond the scope of this study. The general atrial fibrillation population has been shown to have increased cardiovascular

mortality due to ischaemic stroke, sudden cardiac death, heart failure (both with preserved and diminished left ventricular function), myocardial infarction and peripheral vascular disease [13, 16, 18, 19]. The physiological factors thought to tie these clinical outcomes to atrial fibrillation include a diminished cardiac output associated with irregular ventricular contractions [27], loss of atrial-ventricular synchrony [28], systemic thromboembolism due to a prothrombotic state characteristic of atrial fibrillation and left atrial blood stasis [28] and diminished cerebral perfusion [14]. Although there are obvious differences between patients with atrial fibrillation in the general population and CABG patients with POAF, our data, nevertheless, are in line with these observations with a notable increase in the composite CV* (CeV + CV + Other-V) mortality within the overall study period and an increase in the CeV mortality and in the composite of CV* within the intermediate postoperative period among POAF patients. Importantly, Non-CV mortality was not appreciably increased over the entire study period neither in the covariate adjusted [AHR (95% CI) = 1.12 (0.99-1.26)] nor in the propensity-matched [AHR (95% CI) = 1.09 (0.95-1.25)] analysis (Figs 2 and 3), although this risk was significant in the early postoperative time period.

Given the well-established association of atrial fibrillation with thromboembolic events [28], our finding that CeV-attributed mortality was relatively low, accounting for only 5% of all deaths in POAF patients, was surprising, but similar to other reports. Ahlsson *et al.* [2] noted that POAF increases the risk of death due

to cerebral ischaemia from 0.2% of deaths in patients without POAF to 4% of deaths in patients with POAF after a median follow-up of 7 years and is consistent with other series reporting that stroke accounts for <10% of all deaths in patients developing POAF [19, 20]. It is thought, but not proven, that the increased mortality in POAF patients due to stroke stems from late recurrent atrial fibrillation, which predisposes to cerebral embolization. We are unable to assess the rate of recurrent atrial fibrillation that may have developed during the follow-up period outside of the 30-day postoperative time frame that informs the STS database. Recurrent late atrial fibrillation was reported in up to 25% of patients with POAF, representing an 8-fold higher risk compared with patients without POAF [2]. This may be a fruitful area of additional inquiry, given the finding that POAF has been found to be the strongest predictor of recurrent atrial fibrillation [2]. We are also limited by the lack of data on long-term anticoagulant use in our study population, and, hence, it is unclear whether the documented low rate of CeV deaths herein reflects the near-universal commitment to anticoagulation therapy in recent years, given its proven efficacy in decreasing stroke risk [28]. Overall, only 14–16% of POAF patients are currently discharged on anticoagulants [8].

Limitations

The limitations of our study relate to its retrospective nature with the well-known drawbacks of observational studies. As we relied on institutional STS Adult Cardiac Surgery Databases, which are designed to focus on acute perioperative outcomes, a number of important elements that would have increased the value of our study were unavailable to us. We were unable to determine the long-term rate of beta-blockers, statins and angiotensin-converting enzyme inhibitors use in our study cohort, which are important factors given the protective impact of these agents on the development of atrial fibrillation. In addition, we were unable to determine the recurrent rate of atrial fibrillation over the study period. Finally, we were unable to determine the rate of antiarrhythmic agents or anticoagulant use in our study cohort and thus ascertain their impact on our results. The exact accuracy of our cause-specific mortality rates, obtained from a governmental agency, was difficult to assess or corroborate. However, there is no reason to suspect these would disproportionately affect the comparison cohorts. Finally, because we used the STS definition of POAF that requires both the presence and the treatment of atrial fibrillation and effectively excludes self-limited bursts of atrial fibrillation that require no treatment, the true rate of POAF was likely underestimated as in other study cohorts [29].

CONCLUSION

In conclusion, our study confirmed multiple other investigations that POAF is associated with increased long-term all-cause mortality after CABG. Importantly, our analysis elucidated POAF and late mortality associations uniquely by showing that the increased mortality is principally cerebrovascular, cardiac and vascular in nature, and these deaths are particularly increased in the intermediate term (1–6 years). These findings may be related to a greater propensity of POAF patients for recurrent atrial fibrillation beyond the early term during which antiarrhythmic and anticoagulant treatments are more likely to be optimized.

The finding that Non-CV risk of mortality in POAF patients is not elevated is in line with our current understanding of the pathophysiology of atrial fibrillation. In the light of our results, closer monitoring of patients with POAF through at least the intermediate postoperative time period should be strongly considered to possibly mitigate the noted increased mortality risk, pending further confirmatory studies of such an approach.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *EJCTS* online.

Funding

This work was supported by institutional and departmental funds.

Conflict of interest: none declared.

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