

Seven Things We Need to Know about Postoperative Atrial Fibrillation: Editorial

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Introduction

Atrial fibrillation (AF) remains the most frequently occurring arrhythmia in the world and despite a greater understanding of the arrhythmia, rates of AF are rising sharply, increasing three-fold in the last 50 years [1]. Acute postoperative AF (POAF) occurs in the near postoperative period following both cardiac and noncardiac surgeries. The incidence ranges from 30 to 60% following cardiac surgery, and 5 to 10% following noncardiac surgery, which is not as well studied [2-8]. At times, POAF may be self-limiting and resolve spontaneously with no immediate complications, and, as a result, it is sometimes perceived as a clinically insignificant and transient condition. The proportion of POAF cases that resolve spontaneously has not been quantified, but with over 900,000 cardiac surgeries every year in the United States alone, [9] it is plausible that several hundreds of thousands of patients a year experience non-limiting POAF. Noncardiac surgery patients may not be continuously monitored for AF following their surgeries. The result is that the actual incidence of POAF may be underestimated, just as the impact of POAF on the healthcare system may be minimized [10]. POAF is associated with a significantly higher risk of stroke; in one study, stroke risk was 11.1% for POAF patients compared to 1.9% in patients who maintained sinus rhythm [11]. POAF is also associated with greater morbidity, longer hospital stays, and higher mortality [10, 12]. Yet despite its prevalence and associated risks, there is very little information to guide the clinician and some questions that demand answers.

Is there a Clinically Meaningful Difference between POAF following Cardiac Surgery and POAF Following Noncardiac Surgery? And how does this Compare to Nonvalvular AF?

The clinical course of POAF is not well studied in noncardiac surgery patients, [13] and it cannot be said if POAF following noncardiac surgery has different risks or outcomes than POAF after cardiac surgery. Noncardiac patients with POAF have a

greater risk of congestive heart failure, myocardial infarction, cardiac arrest, bacterial pneumonia, and longer hospital stays [12]. POAF is associated with a risk of stroke. POAF patients have a 37% higher risk of late stroke and a 37% higher risk of late mortality compared to those without POAF. Interestingly, the risk of late stroke was higher in noncardiac surgery than in cardiac surgery patients [14]. POAF following coronary artery bypass graft (CABG) procedures, the risk for thromboembolism was lower than in patients with nonvalvular AF [15]. In a study of 22,007 noncardiac surgery patients who developed POAF, 5% had thromboembolism within four years [16]. In a study of 675 cardiac surgery patients with POAF matched to 2,025 patients with nonvalvular AF, the long-term risk of thromboembolism was similar in both groups [17].

In a study of 744 cardiac surgery patients, five-year survival was 83% for those with POAF and 93% for those without ($p < 0.001$) [18]. POAF in both noncardiac and cardiac surgery patients increased the risk of early and long-term mortality [14].

Of interest is the fact that POAF following noncardiac surgery appears to be associated with more harmful effects than POAF after cardiac surgery [19]. POAF increases the risk of stroke in cardiac surgery patients by about 20%, but it doubles the risk of stroke in noncardiac surgery patients [14]. Lin and colleagues proposed an explanation of this by saying that manipulation of the cardiac tissue may precipitate POAF in cardiac surgery but as the heart heals, these factors no longer affect the heart. The mechanism of POAF following noncardiac surgery has not been adequately studied, but is likely somewhat different than the mechanisms of POAF following cardiac surgery and could involve an interplay of several factors [14, 20, 21].

How Many Patients Develop POAF Following Noncardiac Surgery?

The rate of POAF following noncardiac surgery is not well studied and reported incidences vary widely from 10% to 30% [5, 21].

However, with about 300 million noncardiac surgery patients per year around the world, patients likely to experience POAF following noncardiac surgery represent a considerable population [22]. The rate of POAF appears to vary with the type of surgery and it is higher following noncardiac thoracic than other surgeries [5, 21]. A study of 24,125 patients undergoing noncardiac surgery under general anesthesia found that hip fixation and laparotomy surgeries likewise produce high rates of POAF. POAF occurred at a rate of 10% in noncardiac cancer-surgery patients [23]. It should be noted that this rate may be underestimated because noncardiac patients are not routinely monitored for POAF and those who are monitored may not be continuously monitored. Because noncardiac surgery patients are often not monitored for AF or are monitored, at most, intermittently, POAF may easily go undetected [24]. POAF following noncardiac surgery typically occurs in the first four days after surgery [21].

Are there Long-term Risks with POAF?

Since many cases of POAF are self-limiting without complication or may occur sub-clinically in noncardiac surgery patients, there is a tendency to think of it as a transient or at least temporally sequestered phenomenon. However, POAF following surgery may recur as a more persistent late form of AF long after surgery. Recurrent AF has a significant and little-appreciated association with POAF following noncardiac surgery at one year (31% of POAF vs. 3% of those without POAF, $p < 0.001$). It is alarming that 92% of the patients who developed recurrent AF within a year after noncardiac surgery had asymptomatic AF [23].

The risk of POAF transitioning into late or so-called “recurrent” AF is clinically important. In a study of 322,688 patients who underwent major noncardiac surgery under general or local anesthesia, 0.6% developed POAF, but of that population, 16.8% developed recurrent AF [25]. Those with recurrent AF had a significantly greater rate of thromboembolic events and a greater risk of major bleeding events [25]. However, all-cause mortality was not significantly different in those with or without recurrent AF (45.3% vs. 30.5%, $p = 0.15$) [25].

A study of 23 CABG patients with POAF used injectable cardiac monitors and found 60.9% experienced recurrent AF ($n = 14$) and persisted for over three months in 10/14 of them. Of the 17 patients followed for a year or more, 8/17 had recurrent AF more than one year after the CABG surgery with the meantime from surgery to the first episode of recurrent AF 143 ± 22.5 days [26]. In a meta-analysis (8 studies, $n = 1,157$) noninvasive monitoring detected POAF in the first four weeks after hospital discharge in 28.3% of cardiac surgery patients and recurrent AF occurred at a rate of 61% to 100% over two years, and 40% to 93% of episodes were asymptomatic [27]. In a study of noncardiac surgery patients, those who developed POAF (0.8%) had a cumulative one-year risk of stroke of 1.5% compared to 0.4% of patients who did not

experience POAF. It should be noted that a higher CHA₂DS₂-VASc score in these patients was associated with an increased one-year cumulative stroke risk.

Is POAF After Noncardiac Surgery a Clinically Meaningful Event?

For noncardiac surgery patients, POAF is associated with a higher risk for stroke, myocardial infarction, and all-cause mortality [28]. Noncardiac surgery patients who develop POAF also have a greater risk of congestive heart failure, myocardial infarction, cardiac arrest, bacterial pneumonia [29] and longer hospital stays [12]. It has been speculated that POAF after noncardiac surgery has a different etiology than POAF after cardiac surgery, which may be the reason for these differences [8,21].

Nor is it known if noncardiac surgery patients who develop POAF have an elevated risk for thromboembolic events. A retrospective database study found noncardiac surgery patients developed POAF at a rate of 0.4% and of these patients 3830 were matched with 15,320 patients with nonsurgical nonvalvular AF [15]. Long-term thromboembolic events were similar in the POAF and the nonvalvular AF patients (31.7 vs. 29.9 events per 1000 person-years) and anticoagulation therapy in POAF patients was associated with a significantly lower risk of thromboembolism [15].

Noncardiac surgery patients with POAF have an increased 30-day and one-year risk of stroke than cardiac surgery patients with POAF [14]. Overall, POAF patients had a 62% greater chance of early stroke and 44% greater change of early death compared to those without POAF [14]. While POAF in noncardiac surgery patients may go undetected, it can be a clinically important event.

What are the Costs Associated with POAF?

POAF following CABG surgery was shown to significantly prolong hospital stay compared to patients who did not develop POAF (15.1 \pm 9.0 versus 10.0 \pm 4.6 days, respectively) [30], and such extended hospitalizations are associated with higher costs. In a retrospective database study of CABG patients with and without acute POAF ($n = 549$ and 1,547, respectively), those with POAF had discharge costs \$13,993 higher than those without POAF and 3.9 days longer length of stay in the hospital. At one year post-discharge, POAF patients were at a significantly higher risk for mortality ($p < 0.01$) and had a total of \$15,593 higher costs, most of which was due to the prolonged hospitalization for the surgery [31]. Extrapolated out to the national level and based on the AF prevalence by age and sex, the national incremental costs of AF in the United States range from \$6 to \$26 billion [32]. The direct medical costs in an AF patient are about 73% greater than the costs in a matched control subject without AF. The primary incremental cost associated with AF is hospitalization [32]. Since the global incidence of AF is rising [1], these costs may be expected to increase markedly.

What are the Risk Factors for POAF?

Risk factors for POAF may be specific to the patient or they may relate to the type of surgery. In general, the main risk factors for POAF after cardiac or noncardiac surgery include older age, male sex, a history of congestive heart failure, and hypertension. Among noncardiac surgery patients only, lung illness, and brain natriuretic peptide (BNP) are also predictive factors [15,21,33,34]. The type of surgery plays a role; cardiac surgery has a higher incidence of POAF than noncardiac surgery.

A POAF risk-stratification algorithm was developed awarding one point for the male sex, one point for heart rate ≥ 72 bpm, three points for those between the ages of 55 and 74, and four points for those 75 and older. In a study of 856 patients, 17.2% developed POAF but none of them had a score of 0 using this algorithm; of those who scores 6 on this metric, 35.3% developed POAF [35]. A somewhat different risk-stratification algorithm for postoperative complications in general following cardiac surgery assigns one point each for age between 60 and 69, history of the chronic obstructive pulmonary disorder (COPD), estimated glomerular filtration rate <15 mL/min/1.73 m² or dialysis, emergency surgery, preoperative need for an intra-aortic balloon pump, left-ventricular ejection fraction $<30\%$, and undergoing valvular surgery. Furthermore, one extra point is assigned for each decade above 70. Patients who scored 3 or higher on this metric had a higher rate of complications, greater mortality, higher rate of stroke, and more need for renal replacement therapy [36].

The CHA₂DS₂-VASc scoring tool was designed to assess AF patients for their risk for ischemic stroke. A study of 2385 patients used this metric to evaluate the risk of new-onset POAF in cardiac surgery patients [37]. In this cohort, 15.9% developed POAF and the mean CHA₂DS₂-VASc score for those with POAF was 3.6 ± 1.7 compared to 2.8 ± 1.7 for those without POAF ($p < 0.0001$). Multivariate analysis found that as the score increased from 0 to 9, the risk of POAF increased from 8.2 to 42.3%. Thus, every incremental CHA₂DS₂-VASc point increased the risk of POAF [37]. A study of 277 cardiac surgery patients found that a higher score on the CHA₂DS₂-VASc instrument was predictive of POAF [38]. A systematic review of 12 studies ($n=18,086$) found the CHA₂DS₂-VASc tool was an independent predictor of POAF after cardiac surgery (odds ratio 1.5, 95% CI, 1.3-1.7) with 70% specificity and 72% sensitivity [39]. Valve surgery appears to be associated with a high rate of POAF, which may develop into recurrent AF [21]. In a retrospective study of 10,461 cardiac valve surgery patients, 13% developed POAF within two weeks after and the rate of AF at seven years postoperatively was 9%. Risk factors for POAF in this population were preoperative tricuspid valve regurgitation, mitral valve replacement, and history of cardiac surgery [21].

POAF may also occur following pulmonary resection; in fact, AF is the most common postoperative arrhythmia following

lung cancer surgery [40]. In one study, POAF occurred in 5.2% of cancer-related lobectomies and conferred a poorer prognosis than patients who did not have POAF [41]. In this study ($n=947$ lung cancer patients), poor performance status, COPD, history of paroxysmal AF, and intraoperative blood transfusion emerged in multivariate analysis as POAF predictors [41]. Other risk factors for POAF following pulmonary surgery include male sex, resected lung volume, BNP, and left-ventricular early transmural velocity/mitral annular early diastolic velocity, but none of these independent predictors had a strong predictive value [40].

A meta-analysis of 24 studies reported standardized mean differences between POAF and non-POAF in cardiac surgery patients and found age, left-atrial diameter, left-ventricular ejection fraction, heart failure, chronic obstructive pulmonary disease, hypertension, and history of myocardial infarction to be significant independent predictors of POAF; diabetes was only slightly predictive (1.06, 1.00-1.13) [42]. In a database study of 370,447 surgical patients, 3.0% developed POAF during their stay in the hospital, of whom about two-thirds (67%) had a history of diagnosed AF. Black patients had a significantly lower risk of POAF than other patients (adjusted odds ratio 0.53, 95% CI, 0.48-0.59, $p < 0.001$) [33].

Why do we see Racial Differences with POAF?

Following CABG surgery, Black Americans are at significantly lower risk of POAF than Americans of European ancestry (29.3% vs. 18.5%, $p=0.001$) [43]. In a propensity-adjusted analysis, POAF occurred in 22% of Black patients, 29% in other non-Caucasian patients, and 35% of Caucasian patients ($p < 0.0001$ for Black versus Caucasian). Other studies of CABG patients have confirmed this [44]. Hispanic patients have a similar rate of POAF as Caucasians [45].

There are other racial differences as well. In a study of multiethnic Asian cardiac surgery patients conducted in Singapore ($n=2,168$), POAF occurred in 17.3% of patients, with most cases occurring in the first 72 hours after the operation. Among the patients, Chinese had a greater risk than Indians (odds ratio 2.09, 95% CI, 1.28-3.41, $p=0.003$) for developing POAF, and Malaysians were at greater risk than Indians (OR 2.43, 95% CI, 1.36-4.05, $p=0.002$) [46]. Among Asian patients, cardiac surgery patients with POAF had an increased risk for recurrent AF, heart failure, and increased mortality, but not an increased risk for ischemic stroke [47]. Racial and ethnic disparities in POAF suggest that there may be some heritability factors involved in the development of POAF, although more research is needed.

Discussion

POAF is prevalent following cardiac and noncardiac surgery, and POAF is associated with potentially life-threatening complications, yet there are still knowledge gaps in its etiology, epidemiology, and treatment (it goes beyond the scope of this article to describe

treatment strategies). To some extent, the incidence of POAF may not be known, particularly in the context of specific surgeries or specific patient populations. While cardiac surgery patients may be monitored continuously in the perioperative period for cardiac arrhythmias, such continuous monitoring is less frequent among noncardiac surgery patients. POAF, like paroxysmal AF, can be very challenging to get “on the record,” as the arrhythmia can start without warning, may not last very long, may be self-limiting, and is often asymptomatic. POAF has the potential to develop into recurrent AF. The advent of injectable cardiac monitors and, to a lesser extent, patient wearables, have expanded our understanding of chronic AF prevalence.

POAF is complicated, because there appears to be clinically important distinctions between POAF after cardiac versus noncardiac surgery. More research is needed. There are also suggestions that POAF in the immediate postsurgical period may be different than POAF that develops in the ensuing days; again, more study is needed.

Our growing appreciation of POAF reveals a few unmet medical needs. There needs to be better but cost-effective ways to monitor surgical patients (even noncardiac surgery patients) for POAF during their hospital recovery times. Furthermore, safe, effective, and cost-effective POAF treatments are needed.

Conclusion

Although POAF in some cases may be self-limiting and asymptomatic, it is a prevalent condition associated with increased costs, considerable morbidity, and mortality. In some cases, POAF may lead to recurrent AF. There are some mistaken notions about POAF, namely that it develops only after cardiac surgery (POAF develops after noncardiac surgeries as well and may have more severe consequences), that it is not a serious problem (it may lead to immediate effects such as myocardial infarction, stroke, or thromboembolic events or develop into recurrent AF), and that the condition is thoroughly elucidated. More research is needed to better understand safe, effective, and cost-effective ways to treat POAF.

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