

Short Communication

Atrial Fibrillation after Cardiac Surgery: Where are we Now?

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Abstract

Post-operative Atrial fibrillation (POAF) is the most common arrhythmia occurring after cardiac surgery, affecting between 30% and 50% of the patients, and it is generally associated with a higher risk of morbidity and mortality, prolonged in-hospital stay and a concomitant increased cost in hospitalisation. The development of POAF is a multifactorial process connected to pre-existent etiological factors, including genetic predisposition, and peri-operative aspects such as the use of cardio-pulmonary bypass, the inflammatory response to the operation, cardiac manipulations, type of heart surgery and electrolytes imbalance. Several pathophysiological pathways have been hypothesized, but there is no comprehensive knowledge of the factors leading to this complication. Even in terms of prevention and treatment there is no univocal approach. Beta-blocker are used in most of the prevention protocols, but others pharmacological strategies have been considered (i.e. amiodarone, anti-inflammatory drugs and statins). Furthermore, electrolytes correction (mainly potassium and magnesium) seems to have a very important role in POAF. The initial treatment of POAF is aimed to achieve pharmacological ventricular rate control. However, if atrial fibrillation persists for more than 24-48 hours or the patient is haemodynamically compromised despite the attempt of rate control, a rhythm control strategy should be considered, together with an anticoagulation therapy either with warfarin or the new oral anticoagulants which are showing encouraging results both in terms of safety and efficiency. Here, we report a contemporary overview of the incidence, mechanism, predisposing factors, prevention and treatment of POAF after cardiac surgery.

INTRODUCTION

Atrial fibrillation (AF) is the most common arrhythmia that occurs after cardiac surgery [1]. Symptoms include shortness of breath, palpitations, chest pain, tiredness and lightheadedness. Although it is often considered as a benign transitory complication, it is worth noting that it is associated to other complications such as cardiac failure, acute kidney injury, deep and superficial wound infections [2-4]. Post-operative AF (POAF) increases the risk of stroke by threefold after cardiac surgery [5]. Other direct consequences include increased length of staying in intensive care unit and/or hospital, hypotension, pulmonary oedema, and pacemaker placement. POAF has been also linked with an increased risk of death in both the short and long term [6], increases in hospital readmission, and a cost burden to both patients and health-care facilities.

It is still debated which the pathophysiological pathways are leading to POAF after cardiac surgery. The most recent evidence suggests that its development is multifactorial and influenced by several elements related to the non-physiologic nature of the cardiopulmonary bypass (CPB) and cardioplegic arrest, cardiac manipulations, inflammatory response and oxidative stress following myocardial ischaemia-reperfusion injury, pharmacologic triggers, and others [5, 7-9].

Furthermore, there is evidence suggesting that the atria remain electrically active during cardiopulmonary bypass surgery

despite adequate cardioplegic myocardial arrest, predisposing to ischemia and therefore susceptibility to arrhythmias [10].

Incidence and predisposing factors

It has been shown POAF occurs in approximately 20% to 40% of the cardiac surgery patients, depending on the type of procedure, with generally higher rates after valvular surgery (30–50%) and with the highest incidence in combined coronary artery bypass graft surgery (CABG) and valvular surgery (60–80%). Differences in incidence rates are likely related to patient populations, pre-existing comorbidities, and distinct surgical stressors and insults on the myocardium. Furthermore, various methods of detection, surveillance, and study methodologies also contribute to the reported variability. POAF typically occurs on the second day post operatively, with 70% of cases occurring within the first four days after surgery [2]. There is a large body of evidence suggesting that several elements can predict the development of POAF [11-20]. Advanced age at time of surgery is a significant predisposing factor across all types of CABG, valvular, and combined cardiac surgeries. Other variables associated with the development of POAF are: male gender, obesity and metabolic syndrome, chronic obstructive pulmonary disease, congestive heart failure, a high Euro SCORE, preoperative digoxin use, mitral valve disease, left atrial enlargement, withdrawal of beta-blocker or angiotensin-converting enzyme inhibitor (ACEi) postoperatively and past medical history of AF.

Other intraoperative factors such as cardiac venting through right superior pulmonary vein and inotropic use for > 30 min after cardiopulmonary bypass have also been identified as potential risk factors [21]. Additionally, blood measurements of electrolytes such as hypokalaemia and hypomagnesemia, as well as postoperative rising of white cell count, are associated with the development of POAF [22, 23].

Molecular alterations

It has been demonstrated that AF could be a heritable condition and dedicated studies led to the identification of genetic variants associated with common forms of AF, mainly related to a series of ion channels dysfunction [24]. Several studies also tried to identify the relationship between AF and genes encoding gap junction proteins, interleukins, signalling molecules and mediators [5, 24].

Moreover, some of the mechanisms predisposing a pre-existent molecular substrate leading to AF are related to genetic mutation that express in ion channels alterations and their key role in regulation atrial refractoriness. Some authors advocate that the concomitant alteration of L-type Ca^{2+} channels is represented in patients experiencing POAF. On the other hand, potassium (K^+) channels modifications have been identified in persistent AF even if their role in the occurrence of POAF is not been demonstrated [24].

Furthermore, disorder in cardiac electrical conduction can be related to atrial interstitial accumulation of collagen fibres in the extracellular matrix, inflammatory infiltration and/or amyloidosis. Relationship between age dependent atrial remodelling leading to structural alterations and the occurrence of POAF is also supported by several studies [5,24]. Particularly, POAF has been linked to the amount of fibrosis in right atrial appendages of patients undergoing cardiac surgery, although no histological variations have been related to the use of CPB [25].

Prevention

Interventions for the prevention or reduction of POAF can be essentially divided into pharmacologic and non-pharmacologic (or procedural) strategies. Among the pharmacologic strategies, beta-blockers are the most common medications used for the prevention of POAF. The attributed beneficial effect of these class II antiarrhythmic drugs is related to attenuation of increased sympathetic tone related to cardiac operations [13]. There are convincing evidences reporting that the use of prophylactic oral beta-blockers prior to elective cardiac surgery is safe and reduces the incidence of POAF [26]. Furthermore, current guidelines suggest that, unless contraindicated (such as in patients with poorly controlled asthma or heart failure), the use of beta-blocker is recommended in patient undergoing cardiac surgery (Class I) [27]. It has also been shown that discontinuation or withdrawal of beta-blockers in the perioperative period could predispose to POAF and for this reason it is recommended that the administration of beta-blockers should be restarted as soon as possible after the operation in patients under chronic therapy. In the last few years it seems to be a new increased use of sotalol for preventing POAF after cardiac surgery. Sotalol is a beta-blocker medication that also has some class III antiarrhythmic drug effects and has been evaluated for the reduction of POAF [13]. Its action is related not only to the inhibition of sympathetic system but also to the prolongation of the duration of the refractory

period in cardiac conductive tissue, through a certain degree of effects on sodium, potassium, and calcium channels [13, 28]. The current guidelines suggest the use of sotalol for the prevention of POAF after cardiac surgery in Class IIb [27]. However, some authors advocate that the prophylactic administration of sotalol is related to adverse side effects such as bradycardia and ventricular arrhythmias [20,28]. Among the pharmacologic strategies, amiodarone appears to play a potential role in the prevention of POAF. Amiodarone is a Class III antiarrhythmic drug that works by prolonging the action potential duration and hence refractory period of atrial, nodal, and ventricular tissues. Amiodarone has been extensively studied in the literature in its role in AF prophylaxis after cardiac surgery. Studies demonstrated a significant effect of amiodarone in reducing the rates of POAF and shortening hospital stays. However, given the significant variation among the trials in the timing of amiodarone initiation, the value of preoperative versus postoperative initiation of prophylaxis remains unclear [28,29]. Electrolytes depletion is very common during the cardiac surgery postoperative period as a result of several concomitant factors such as fluid o diuretic administration or consequences of using cardiopulmonary bypass [22,28]. Serum electrolyte concentrations directly affect the atrial automaticity, and conductive properties. Current clinical practice often includes intraoperative and postoperative repletion of magnesium and potassium, although its effects remain controversial [20]. A multicentre study demonstrated that potassium level inferior than 3.5 mmol/L was a predictive of serious perioperative arrhythmias [30]. Other authors demonstrated that the rate of POAF in patients with serum potassium levels of 3.9 mmol/l or less, compared with those with serum potassium levels of 4.4 mmol/l or greater were significantly different being 50.7% and 32.9%, respectively [31]. Magnesium depletion also may increase the risk of POAF and has been linked even with post-operative atrial tachyarrhythmia. In addition, hypomagnesemia may result in a suboptimal clinical response to potassium supplementation [28]. A metaanalysis concluded that magnesium administration was effective for reducing POAF with a similar efficacy to common anti arrhythmic drugs [32]. Inflammatory response and oxidative stress often associate with CPB and reperfusion injury contribute to the pathogenesis of the postoperative arrhythmia. For this reason, several anti-inflammatory agents and anti-oxidants including statins, steroids, Colchicine, Vitamins C and E, and acetylcysteine, have been used for the prevention of the POAF [33]. As already discussed, the use of cardiopulmonary bypass and the associated ischemia-reperfusion lead to a systemic inflammatory response which contributes to the development of POAF. Among other pleiotropic effect, statins also reduce inflammation and oxidative stress following cardiac surgery, and early studies showed that preoperative statins significantly reduced POAF incidence [2, 13, 16, 20]. Although proposed in the past for this purpose, based on the evidence up to date, current guidelines do not recommend statins for prophylaxis of AF after coronary artery bypass graft surgery [27]. Many authors have focused on the use of colchicine, a potent anti-inflammatory agent used in the treatment of acute gout flares which inhibits microtubule formation of neutrophils, therefore inhibiting inflammation caused by migration, activation, and degranulation of neutrophils [26]. A recent randomized controlled trial suggested that there is no significant benefit of colchicine in the prevention of postoperative AF [34]. The prophylactic use of colchicine may be limited by toxic side-effect

including gastrointestinal irritation and myelosuppression [28]. In spite of that, according to the recent guidelines administration of colchicine may be considered for patients postoperatively to reduce AF after cardiac surgery (class IIb) [27]. There is increasing evidence that specific intraoperative procedures can reduce the incidence of POAF or, at least, reduce the risk of thrombo-embolic complications. Intraoperative interventions aimed at preventing POAF include posterior pericardiotomy (in which an incision is made in the posterior pericardium from the left inferior pulmonary vein to the diaphragm, parallel to the left phrenic nerve), anterior fat pad (AFP) preservation (in which the AFP, which contains parasympathetic ganglia, is not excised), and left atrial appendage (LAA) exclusion from circulation [20]. LAA exclusion has emerged as a target for prophylactic stroke prevention and an alternative to long-term anticoagulation therapy. However, due to contrasting results, it is still unclear whether cardiac surgery patients should undergo concomitant LAA exclusion, or whether anticoagulants may be safely discontinued in patients who do. Large studies are ongoing to evaluate whether LAA exclusion is protective against stroke in the long-term, but recommendation of this technique for a possible short-term advantage cannot be made at present [20].

Treatment

Once occurred in the postoperative period, the treatment of AF after cardiac surgery is driven by clinical symptoms and hemodynamic stability. Although in many cases episodes of POAF are self-limiting without intervention within minutes or hours, persistent episodes of AF and those arising in haemodynamically unstable patients require prompt clinical intervention. POAF can be treated by rhythm control, ventricular rate control, and antithrombotic [35]. Electrical cardioversion should be performed immediately if patient is unstable and haemodynamically compromised by the new onset of POAF [27] even though if patients are not haemodynamically compromised is preferable the pharmacologic approach. For those patients, ventricular rate control can be performed using beta-blockers, non-dihydropyridine calcium antagonist, or amiodarone, whether pharmacological cardioversion can be achieved effectively using propafenone, flecainide or ibutilide [13].

Whenever those medications are used, the risk of drug-induced pro-arrhythmic effects must be considered, particularly in elderly patients and those with reduced ejection fraction or previous myocardial infarction [35].

The major concomitant adverse event related to cardioversion is thromboembolism, especially when POAF has been sustained for more than 48 h [13, 20, 25, 33]. A stunning atrium can facilitate the formation of thrombus and consequently the risk of embolic stroke that could be reduced by the use of anticoagulants medications [13], considering that the use of these drugs during the postoperative course can increase the risk of bleeding, pericardial effusion or tamponade (35). For this reason, after surgery, administration of anticoagulation must be pondered in the context of risk of stroke with the risk of perioperative bleeding [35, 37]. There are substantive evidences suggesting that CHADS2 and CHA2DS2-VASc scoring (two scoring systems combining cardiovascular and non-cardiovascular characteristics to determine a patient's stroke risk) are very useful risk assessment classification to adopt in this scenario [38]. Within the first 48 hours of POAF, anticoagulation to prevent thromboembolism

is strongly recommended when the patient's CHA2DS2-VASc score is higher than zero. However, patients with significant risk factors such as advanced age, uncontrolled hypertension, or a history of bleeding, could benefit of pharmacological or electric cardioversion without anticoagulation [13, 20, 27].

New Oral Anticoagulants (NOAC), generally used when there is a contraindication of warfarin, are showing better effectiveness compared to traditional warfarin at preventing stroke and major postoperative bleeding events also having reduced pharmacological interaction [15,25]. The use of NOAC should be avoided in patients with concomitant mechanical heart valve and in patient with poor renal function [15]. Anticoagulants should be continued for a minimum of 4–6 weeks after return to sinus rhythm and considering extending the therapy duration depending on the patient's stroke risk [27, 37].

CONCLUSIONS

POAF still represents the main arrhythmic complication after cardiac surgery and despite several studies we still do not entirely know its pathophysiological aspects. Several preventive and therapeutic measures have been introduced with contrasting results. Long term outcome after cardiac surgery can be negatively impacted by POAF and its thromboembolic sequelae.

REFERENCES

1. Asher CR, Miller DP, Grimm RA, Cosgrove DM 3rd, Chung MK. Analysis of risk factors for development of atrial fibrillation early after cardiac valvular surgery. *Am J Cardiol* 1998; 82:892–5.
2. Bessissow A, Khan J, Devereaux PJ, Alvarez-Garcia J, Alonso-Coello P. Postoperative atrial fibrillation in non-cardiac and cardiac surgery: an overview. *J ThrombHaemost*. 2015 Jun;13 Suppl1:S304-12.
3. Akintoye E, Sellke F, Marchioli R, Tavazzi L, Mozaffarian D. Factors associated with postoperative atrial fibrillation and other adverse events after cardiac surgery. *J Thorac Cardiovasc Surg*. 2018;155: 242-51.e10.
4. Bruno VD, Ascione R. Postoperative atrial fibrillation: Still in search of truth or a neglected complication? *J Thorac Cardiovasc Surg*. 2018 Jan;155(1):236-237.
5. Maesen B, Nijs J, Maessen J, Alessie M, Schotten U. Post-operative atrial fibrillation: a maze of mechanisms. *Europace*. 2012; 14: 159–74.
6. Mariscalco G, Klersy C, Zanobini M, Banach M, Ferrarese S, Borsani P, Cantore C, Biglioli P, Sala A. Atrial fibrillation after isolated coronary surgery affects late survival. *Circulation* 2008; 118: 1612–8.
7. Mathew JP. Atrial fibrillation following coronary artery bypass graft surgery. *JAMA*. 1996;276:300.
8. Ishii Y, Schuessler RB, Gaynor SL, et al. Inflammation of atrium after cardiac surgery is associated with inhomogeneity of atrial conduction and atrial fibrillation. *Circulation* 2005;111:2881– 8.
9. Gaudino M, Andreotti F, Zamparelli R, Di Castelnuovo A, Nasso G, Burzotta F, Iacoviello L, Donati MB, Schiavello R, Maseri A, Possati G. The -174G/C interleukin-6 polymorphism influences postoperative interleukin-6 levels and postoperative atrial fibrillation. Is atrial fibrillation an inflammatory complication? *Circulation* 2003; 108(Suppl. 1): II195–9.
10. Tchervenkov CI, Wynands JE, Symes JF, Malcolm ID, Dobell AR, Morin JE. Electrical behavior of the heart following highpotassium cardioplegia. *Ann ThoracSurg* 1983; 36: 314–9.
11. Filardo G, Pollock BD, da Graca B, Phan TK, Sass DM, Ailawadi G, et al.

- Underestimation of the incidence of new-onset post-coronary artery bypass grafting atrial fibrillation and its impact on 30-day mortality. *J Thorac Cardiovasc Surg.* 2017;154:1260-6.
12. Ascione R, Caputo M, Calori G, Lloyd CT, Underwood MJ, Angelini GD. Predictors of atrial fibrillation after conventional and beating heart coronary surgery. A prospective, randomized study. *Circulation.* 2000;102:1530-5.
 13. Echahidi N, Pibarot P, O'Hara G, Mathieu P. Mechanisms, prevention, and treatment of atrial fibrillation after cardiac surgery. *J Am Coll Cardiol.* 2008 Feb 26; 51(8): 793-801. doi: 10.1016/j.jacc.2007.10.043. Review.
 14. Mathew JP, Fontes ML, Tudor IC, et al. A multicenter risk index for atrial fibrillation after cardiac surgery. *JAMA.* 2004 Apr 14;291(14):1720-9.
 15. Philip I, Berroeta C, Leblanc I. Perioperative challenges of atrial fibrillation. *Curr Opin Anaesthesiol* 2014;27:344-52.
 16. Kievišas M, Keturakis V, Vaitiekūnas E, Dambrauskas L, Jankauskienė L, Kinduris Š. Prognostic factors of atrial fibrillation following coronary artery bypass graft surgery. *Gen Thorac Cardiovasc Surg.* 2017 Oct;65(10):566-574.
 17. Luo W, Huaibin W, Wenjun Z, Jie T1, Xiaokang O, Zi W, Yujian M. Predictors of Postoperative Atrial Fibrillation after Isolated On-Pump Coronary Artery Bypass Grafting in Patients ≥60 Years Old. *Heart Surg Forum.* 2017 Feb 28;20(1):E038-E042.
 18. Hernandez AV1, Kaw R, Pasupuleti V, Bina P, Ioannidis JP, Bueno H, Boersma E, Gillinov M; Cardiovascular Meta-Analyses Research Group. Association between obesity and postoperative atrial fibrillation in patients undergoing cardiac operations: a systematic review and meta-analysis. *Ann Thorac Surg.* 2013 Sep;96(3):1104-16.
 19. Bramer S1, van Straten AH, Soliman Hamad MA, Berreklouw E, van den Broek KC, Maessen JG. Body mass index predicts new-onset atrial fibrillation after cardiac surgery. *Eur J Cardiothorac Surg.* 2011 Nov;40(5):1185-90.
 20. Greenberg JW, Lancaster TS, Schuessler RB, Melby SJ. Postoperative atrial fibrillation following cardiac surgery: a persistent complication. *Eur J Cardiothorac Surg.* 2017 Oct 1;52(4):665-672. doi: 10.1093/ejcts/ezx039.
 21. Almassi GH, Schowalter T, Nicolosi AC, Aggarwal A, Moritz TE, Henderson WG, Tarazi R, Shroyer AL, Sethi GK, Grover FL, Hammermeister KE. Atrial fibrillation after cardiac surgery: a major morbid event? *Ann Surg* 1997; 226: 501-11 discussion 11-3.
 22. Hernandez-Leiva E, Dennis R, Isaza D, Umana JP. Hemoglobin and B-type natriuretic peptide preoperative values but not inflammatory markers, are associated with postoperative morbidity in cardiac surgery: a prospective cohort analytic study. *J Cardiothorac Surg* 2013; 8: 170.
 23. Abdelhadi RH, Gurm HS, van Wagoner DR, Chung MK. Relation of an exaggerated rise in white blood cells after coronary bypass or cardiac valve surgery to development of atrial fibrillation postoperatively. *Am J Cardiol* 2004; 93: 1176-8.
 24. Lubitz S.A. Yi B. A. Ellinor P.T. Genetics of Atrial Fibrillation *Heart Fail Clin.* 2010 April ; 6(2): 239-247.
 - 25.25. Mariscalco G, Engström KG, Ferrarese S, Cozzi G, Bruno VD, Sessa F, et al. Relationship between atrial histopathology and atrial fibrillation after coronary bypass surgery. *J Thorac Cardiovasc Surg.* 2006;131:1364-72
 26. Thein PM, White K, Banker K, Lunny C, Mirzaee S, Nasir A. Preoperative Use of Oral Beta-Adrenergic Blocking Agents and the Incidence of New-Onset Atrial Fibrillation After Cardiac Surgery. A Systematic Review and Meta-Analysis. *Heart Lung Circ.* 2018 Mar;27(3):310-321.
 27. January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, Cleveland JC Jr et al. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol* 2014;64:e1-76.
 28. Jesse M. Raiten, MD, KamrouzGhadimi, MD, John G.T. Augoustides, MD, FASE, FAHA, ‡ Harish Ramakrishna, MD, § Prakash A. Patel, MD, ‡ Stuart J. Weiss, MD, PhD, ‡ and Jacob T. Gutsche, MD ‡ Atrial Fibrillation After Cardiac Surgery: Clinical Update on Mechanisms and Prophylactic Strategies. *J CardiothoracVascAnesth.* 2015;29(3):806-16. doi: 10.1053/j.jvca.2015.01.001.
 29. Patel AA, White CM, Gillespie EL, Kluger J, Coleman CI. Safety of amiodarone in the prevention of post-operative atrial fibrillation: a meta-analysis. *Am J Health Syst Pharm* 2006;63:829-37
 30. Wahr JA, Parks R, Boisvert D, et al: Preoperative serum potassium levels and perioperative outcomes in cardiac surgery patients. Multicenter Study of Perioperative Ischemia Research Group. *JAMA* 281:2203-2210, 1999
 31. Auer J, Weber T, Berent R, Lamm G, Eber B. Serum potassium level and risk of postoperative atrial fibrillation in patients undergoing cardiac surgery. *J Am Coll Cardiol* 44:938-939, 2004.
 32. Miller S, Crystal E, Garfinkle M, Lau C, Lashevsky I, Connolly SJ. Effects of magnesium on atrial fibrillation after cardiac surgery: a meta-analysis. *Heart* 2005;91:618-23
 33. M.Zakkar R, Ascione A.F, James G.D, Angelini M.S, Suleiman. Inflammation, oxidative stress and postoperative atrial fibrillation in cardiac surgery. *Pharmacol Ther.* 2015 Oct;154:13-20. doi: 10.1016/j.pharmthera.2015.06.009. Epub 2015 Jun 24.
 - 34.34 Imazio M, Brucato A, Ferrazzi P, Pullara A, Adler Y, Barosi A, et al. COPPS-2 Investigators. Colchicine for prevention of postpericardiotomy syndrome and postoperative atrial fibrillation: the COPPS-2 randomized clinical trial. *JAMA* 2014;312:1016-23.
 - 35.35 Takeshi Omae, Yuichi Kanmura. Management of postoperative atrial fibrillation. *J Anesth* (2012) 26:429-437.
 - 36.36. LaPar DJ, Speir AM, Crosby IK, Fonner E Jr, Brown M, Rich JB, Quader M, Kern JA, Kron IL, Ailawadi G. Postoperative atrial fibrillation significantly increases mortality, hospital readmission, and hospital costs. *Ann Thorac Surg.* 2014 Aug;98(2):527-33;
 - 37.37. Epstein AE, Alexander JC, Gutterman DD, Maisel W, Wharton JM. Anticoagulation: American College of Chest Physicians guidelines for the prevention and management of post-operative atrial fibrillation after cardiac surgery. *Chest* 2005;128:245-7S.
 - 38.38 Chua SK1, Shyu KG, Lu MJ, Lien LM, Lin CH, Chao HH, Lo HM. Clinical utility of CHADS2 and CHA2DS2-VASc scoring systems for predicting postoperative atrial fibrillation after cardiac surgery. *J Thorac Cardiovasc Surg.* 2013 Oct;146(4):919-926.e1.

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