Post-cardiotomy pericardial effusion and postoperative atrial fibrillation risk

Yuta Kikuchi1 · Yasuaki Saijo2 · Masahiko Narita1 · Keisuke Shibagaki1 · Ryo Okubo1 · Shingo Kunioka1 · Tomonori Shirasaka1 · Hiroyuki Kamiya1

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Abstract
Postoperative atrial fibrillation (POAF) is a common complication after cardiovascular surgery [1–5]. Although a few reports have defined the pathogenesis of POAF [1, 3, 5–8], some clinical associations remain poorly characterized.

The risk factors for POAF have been previously reported as preoperative predictors (age, male sex, history of atrial fibrillation, congestive heart failure, arterial hypertension, obesity, white ethnicity, and chronic obstructive pulmonary disease), perioperative (intraoperative) predictors (mitral valve surgery, intra-aortic balloon pump, cross-clamp time, bicaval cannulation, and venting via a pulmonary vein), and postoperative predictors (pneumonia, respiratory events [ventilation for > 24 h and respiratory insufficiency], inotropic drug use, and atrial pacing) [1, 3, 5–8]. Dobrev et al. reported a conceptual model of POAF that included temporal development of the components of a vulnerable substrate to determine when and where these risk factors exert their effect [6]. Their report separated the risk factors into three groups: pre-existing atrial substrate, surgery-induced substrate, and transient postoperative factors. We believe that the transient postoperative factors listed in the report are likely important for preventing POAF because they are treatable components. Kaygin et al. reported that pericardial effusion (calculated with echocardiography) was a risk factor for atrial fibrillation after cardiac surgery [9]. Similarly, we observed POAF in numerous patients with significant pericardial effusion, suggesting that it belongs to the transient postoperative factors. They also demonstrated the effectiveness of posterior pericardiectomy in reducing the incidence of POAF.

Keywords Postoperative atrial fibrillation · computed tomography · Pericardial effusion · Intraoperative bleeding

Introduction

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The risk factors for POAF have been previously reported as preoperative predictors (age, male sex, history of atrial fibrillation, congestive heart failure, arterial hypertension, obesity, white ethnicity, and chronic obstructive pulmonary disease), perioperative (intraoperative) predictors (mitral valve surgery, intra-aortic balloon pump, cross-clamp time, bicaval cannulation, and venting via a pulmonary vein), and postoperative predictors (pneumonia, respiratory events [ventilation for > 24 h and respiratory insufficiency], inotropic drug use, and atrial pacing) [1, 3, 5–8]. Dobrev et al. reported a conceptual model of POAF that included temporal development of the components of a vulnerable substrate to determine when and where these risk factors exert their effect [6]. Their report separated the risk factors into three groups: pre-existing atrial substrate, surgery-induced substrate, and transient postoperative factors. We believe that the transient postoperative factors listed in the report are likely important for preventing POAF because they are treatable components. Kaygin et al. reported that pericardial effusion (calculated with echocardiography) was a risk factor for atrial fibrillation after cardiac surgery [9]. Similarly, we observed POAF in numerous patients with significant pericardial effusion, suggesting that it belongs to the transient postoperative factors. They also demonstrated the effectiveness of posterior pericardiectomy in reducing the incidence of POAF.
of atrial fibrillation [9]. Thus, early detection of the massive pericardial effusion and its drainage is important to prevent POAF.

We aimed to determine the extent of PE pericardial effusion in patients who experienced POAF compared to that of patients with uneventful postoperative courses, with the hypothesis that pericardial effusion would be more common and extensive in patients with POAF.

**Methods**

Data from 750 consecutive patients who underwent cardiovascular surgery from January 2016 to December 2019 in our department were retrospectively analyzed. Due to the study’s retrospective design, the requirement for informed patient consent was waived. The study was approved by the Institutional Review Board of Asahikawa Medical University (Date: 20. April. 2021, Approval Number: 20107).

Adult patients who underwent cardiac surgery via median sternotomy were included. Those with preoperative atrial fibrillation and pre-/postoperative placement of a pacemaker or implantable cardioverter-defibrillator, those who underwent minimally invasive surgery, and cases without median sternotomy were excluded.

We evaluated pericardial effusion using routine computed tomography (CT) at 7 ± 3 days postoperatively, and patients whose CT scan data were not available at that time were excluded. The thickest portion of the pericardial effusion was measured using an axial view of the CT (regardless of enhanced and plain without electrocardiogram gated) with ShadeQuest/ViewR (FUJIFILM, Tokyo, Japan) in our electronic medical record system (Fig. 1). The first author, who is certified by the Japanese Board of Cardiovascular Surgery, analyzed all cases.

Following exclusion, 294 patients were enrolled and classified into groups of those with (POAF + group; n = 127) and without (POAF− group; n = 167) POAF (Fig. 2).
We also analyzed the number of patients in each of the CHA2DS2-VASc score levels developed by Chua et al. as a potential predictor of POAF [10]. The CHA2DS2-VASc items include congestive heart failure, hypertension, age ≥ 75 years, diabetes mellitus, stroke/transient ischemic attack (TIA), vascular (coronary) disease, age between 65 and 74 years, and sex (female). Each item’s presence was scored as 1 point, except for age ≥ 75 years and stroke/TIA, which were scored as 2 points; the highest possible score was 9 (the total score of the CHA2DS2-VASc score in each group was analyzed using the Mann–Whitney U test).

The normal distribution of continuous variables was determined using the Kolmogorov–Smirnov test. Some variables failed the normality test and were therefore analyzed using the Mann–Whitney U test and are reported as median with interquartile range (IQR). Categorical variables were compared using Fisher’s exact test. Univariate analysis was performed to compare the preoperative characteristics, operative data, and postoperative outcomes/findings between the POAF+ and POAF− groups. For further evaluation, we conducted multivariate logistic regression analysis with explanatory variables of age, sex, hypertension, diabetes mellitus, chronic obstructive pulmonary disease, chronic kidney disease, congestive heart failure, vascular disease, stroke or TIA, operative procedure (coronary artery bypass grafting, valve surgery, aortic surgery), intraoperative bleeding, and postoperative pericardial effusion, which are previously reported POAF risk factors [3–6, 11, 12]. Missing data were managed using multiple imputation by chained equations using the above-mentioned outcomes and explanatory variables. Twenty-five datasets were imputed with 10 iterations each. We also evaluated the correlation between intraoperative bleeding volume and pericardial effusion using Spearman’s rank correlation coefficient.

Statistical analyses were conducted using EZR 2.7 (Saitama Medical Center, Jichi Medical University, Saitama, Japan) [13]. Multiple imputation and logistic regression analyses were performed with mice package in R software, version 4.0.3 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Preoperative patient characteristics are shown in Table 1. Patients in the POAF+ group were significantly older than those in the POAF− group (76 years, range: 68–80 years vs. 68 years, range: 56–75 years, p < 0.001). All other patient characteristics were similar between the groups.

Intraoperative data are shown in Table 2. The intraoperative bleeding volume was significantly higher in the POAF+ group than in the POAF− group (23.6 dL, range: 10.2–42.5 dL vs. 16.7 dL, range: 9–31.5 dL, p = 0.029). The number of aortic surgeries was significantly lower in the POAF+ group than in the POAF− group (40 [31.5%] vs. 76 [45.5%], p = 0.016).

Postoperative outcomes (Table 3) revealed that the average pericardial effusion thickness was 8.5 (range, 0–15.8) mm in the POAF+ group and 0 (0–9.3) mm in the POAF− group (p < 0.001) (Fig. 3). There were several outliers in the POAF− group; however, these did not appear to affect the statistical analyses.

Multivariate logistic regression analysis results after multiple imputation are shown in Table 4. There were significant differences in age (OR, 1.07; 95% CI 1.04–1.10; p < 0.001), intraoperative bleeding volume (OR, 1.02; 95% CI 1.00–1.03; p = 0.002), and postoperative pericardial effusion (OR, 1.04; 95% CI 1.01–1.08; p = 0.006), indicating that these are significantly associated with POAF. There was also a significant difference in the rates of aortic surgery (OR, 0.44; 95% CI 0.22–0.88; p = 0.019), suggesting that this is negatively associated with POAF. There was also a statistical correlation between intraoperative bleeding volume and pericardial effusion (Spearman’s rank correlation rho = 0.137, p = 0.0196), although the correlation was weak (Fig. 4).

Discussion

Dobrev et al. reported a conceptual model of POAF and indicated the influence of the pre-existing atrial substrate, surgery-induced substrate, and transient postoperative factors [6]. In this study, we focused on the post-cardiectomy pericardial effusion and hypothesized that the presence of pericardial effusion correlates with transient postoperative factors. One cause of pericardial effusion is postoperative bleeding or inflammation of the pericardium [14]. Moreover, pericardial inflammation is known to cause POAF. Although pericardial effusion and POAF are both consequences of inflammation, a correlation between pericardial effusion and POAF in this context has not been reported. Olesen et al. also reported a correlation between serum C-reactive protein level, which is an inflammatory marker, and the occurrence of POAF in patients who underwent coronary artery bypass grafting [8]. However, they did not specify the cause of inflammation.

Imazio et al. reported the effects of colchicine on POAF [15]. Although they concluded that colchicine did not prevent POAF or reduce postoperative pericardial effusion in postpericardiotomy syndrome, there was no consideration of the extent of pericardial effusion in these patients. The cause of POAF in that study was potentially a combination of inflammation and mechanical stress to the heart due to pericardial effusion. Mechanical stress caused by pericardial effusion may affect the autonomic nervous system (ANS).
### Table 1 Preoperative characteristics

| Preoperative characteristics                  | All patients | Missing | POAF+ | POAF- | p value |
|-----------------------------------------------|--------------|---------|-------|-------|---------|
|                                              | n = 294      | n (,%)  | n = 127 (%) | n = 167 (%) |        |
| Age, y median (IQR)                           | 71 (62–79)   | 0       | 76 (68–80) | 68 (56–75) | < 0.001 |
| Hypertension                                  | 176 (59.9)   | 0       | 73 (57.5) | 103 (61.7) | 0.474   |
| COPD                                          | 208 (70.8)   | 0       | 92 (72.4) | 116 (69.5) | 0.607   |
| Diabetes Mellitus                             | 80 (27.2)    | 0       | 39 (30.7) | 41 (24.6)  | 0.29    |
| Chronic kidney disease                        | 25 (8.5)     | 0       | 10 (7.9)  | 15 (9.0)   | 0.834   |
| Chronic heart failure                         | 75 (25.5)    | 0       | 33 (26.0) | 42 (25.1)  | 0.893   |
| Vascular disease                              | 115 (39.1)   | 23 (7.8) | 50 (39.4) | 65 (38.9)  | 1       |
| Stroke or TIA                                 | 42 (14.3)    | 23 (7.8) | 24 (18.6) | 18 (10.8)  | 0.061   |
| Preoperative ACS                              | 30 (10.2)    | 0       | 10 (7.9)  | 20 (12.0)  | 0.331   |
| Preoperative antiplatelet                     | 96 (32.7)    | 0       | 39 (30.7) | 57 (34.1)  | 0.616   |
| Preoperative anticoagulant                    | 18 (6.1)     | 0       | 76 (59.8) | 11 (6.6)   | 0.809   |
| Redo surgery                                  | 24 (8.2)     | 0       | 8 (6.3)   | 16 (9.6)   | 0.391   |
| CHA2DS2-VASc score                            | Total score, median (IQR) | 3 (2–5) | 23 (7.8) | 4 (2–5) | 3 (2–4) | 0.182 |
| 0                                             | 15 (5.1)     | 8 (4.8) | 7 (5.5) |
| 1                                             | 29 (9.9)     | 15 (9.0) | 14 (11.0) |
| 2                                             | 46 (15.7)    | 30 (18.0) | 16 (12.6) |
| 3                                             | 51 (17.4)    | 34 (20.4) | 17 (13.4) |
| 4                                             | 59 (20.1)    | 32 (19.2) | 27 (21.3) |
| 5                                             | 45 (15.3)    | 25 (15.0) | 20 (15.7) |
| 6                                             | 15 (5.1)     | 7 (4.2)  | 8 (6.3) |
| 7                                             | 10 (3.4)     | 2 (1.2)  | 8 (6.3) |
| 8                                             | 1 (0.3)      | 1 (0.6)  | (0) |

Bold indicates p < 0.05

POAF postoperative atrial fibrillation, IQR interquartile range, COPD chronic obstructive pulmonary disease, TIA transient ischemic attack, ACS acute coronary syndrome

### Table 2 Intraoperative data

| Intraoperative data                           | All patients | Missing | POAF+ | POAF- | p value |
|-----------------------------------------------|--------------|---------|-------|-------|---------|
|                                              | n = 294      | n (,%)  | n = 127 (%) | n = 167 (%) |        |
| Operative procedure                          |              |         |       |       |         |
| CABG                                          | 60 (20.4)    | 0       | 27 (21.3) | 33 (19.8) | 0.772   |
| Valve                                         | 115 (39.1)   | 0       | 44 (34.6) | 71 (42.5) | 0.186   |
| Aorta                                         | 116 (39.5)   | 0       | 40 (31.5) | 76 (45.5) | 0.016   |
| others                                       | 41 (14.0)    | 0       | 18 (14.2) | 23 (13.8) | 1       |
| Intraoperative bleeding, dL (IQR)             | 18.7 (9.3–35) | 5 (1.7) | 23.6 (10.2–42.5) | 16.7 (9–31.5) | 0.029   |
| Operation time, min (IQR)                    | 302 (255–375) | 48 (16.3) | 306 (262.8–358.5) | 298.5 (249.8–384.5) | 0.821   |
| CPB time, min (IQR)                          | 145 (117.8–172.3) | 34 (11.6) | 145 (117–171.8) | 144.5 (119.3–172.5) | 0.928   |
| Cross-clamp time, min (IQR)                  | 96 (76–120)  | 37 (12.6) | 96.5 (80–118.5) | 94 (74–120) | 0.39    |

Bold indicates p < 0.05

POAF postoperative atrial fibrillation, CABG coronary artery bypass grafting, IQR interquartile range, CPB cardiopulmonary bypass
which is also a transient postoperative factor associated with POAF. Congestive cardiac tamponade due to pericardial effusion activates the renin–angiotensin–aldosterone system and leads to ANS activation. ANS activation is essential to understanding POAF because several reports observed that β-blockers were effective for the prevention and treatment of POAF. If this hypothesis is correct, extending the time of tube drainage from the cardiac sac after surgery may be effective in reducing the prevalence of POAF. However, there is another hypothesis that pericardial effusion is secondary to POAF. Atrial fibrillation may precipitate congestive heart failure, which may lead to the accumulation of pericardial fluid [17, 18]. Thus, the findings of this study are insufficient for concluding the cause-and-effect relationship between pericardial effusion and POAF.

**Table 3** Postoperative outcomes

| Postoperative outcomes/findings | All patients | Missing | POAF+ | POAF- | p-value |
|---------------------------------|-------------|---------|-------|-------|---------|
|                                | n = 294     | n (%)   | n = 127 (%) | n = 167 (%) |         |
| Pericardial effusion, mm (IQR) | 5.9 (0–12.5) | 0       | 8.5 (0–15.8) | 0 (0–9.3) | <0.001  |
| Postoperative anticoagulant     | 226 (76.9)  | 0       | 102 (80.3) | 124 (74.3) | 0.264   |
| Postoperative antiplatelet      | 130 (44.2)  | 0       | 50 (39.4)  | 80 (47.9)  | 0.156   |
| Short term re-operation         | 14 (4.8)    | 0       | 6 (4.7)    | 8 (4.8)    | 1       |
| 30-day mortality                | 6 (2.0)     | 2 (0.7) | 5 (3.9)    | 1 (0.6)    | 0.088   |
| Stroke                          | 14 (4.8)    | 2 (0.7) | 8 (6.3)    | 6 (3.6)    | 0.408   |
| Renal failure                   | 7 (2.4)     | 2 (0.7) | 5 (3.9)    | 2 (1.2)    | 0.245   |
| Infection                       | 36 (12.2)   | 2 (0.7) | 18 (14.2)  | 18 (10.8)  | 0.377   |
| Hemorrhage or tamponade         | 8 (2.7)     | 2 (0.7) | 4 (3.1)    | 3 (1.8)    | 0.73    |
| Mediastinitis                   | 5 (1.7)     | 2 (0.7) | 0 (0)      | 5 (3.0)    | 0.072   |
| Prolonged ventilation > 21 days | 25 (8.5)    | 2 (0.7) | 11 (8.7)   | 14 (8.4)   | 1       |

Bold indicates p < 0.05

POAF postoperative atrial fibrillation, IQR interquartile range

![Figure 3](image-url) The difference in pericardial effusion between patients with and without postoperative atrial fibrillation

**Table 4** Multivariate analysis of previously published POAF risk factors with multiple imputations

|                      | Odds ratio | 95% CI     | p value |
|----------------------|------------|------------|---------|
| Age (per 1 year)     | 1.07       | 1.04–1.10  | <0.001  |
| Male (vs. female)    | 1.16       | 0.66–2.04  | 0.614   |
| HT                   | 0.70       | 0.36–1.35  | 0.286   |
| DM                   | 1.29       | 0.67–2.46  | 0.443   |
| COPD                 | 0.65       | 0.25–1.70  | 0.377   |
| CKD                  | 1.00       | 0.52–1.92  | 0.999   |
| CHF                  | 1.23       | 0.51–3.00  | 0.644   |
| Vascular disease     | 0.71       | 0.37–1.39  | 0.32    |
| Stroke or TIA        | 1.96       | 0.90–4.25  | 0.087   |
| CABG                 | 1.30       | 0.64–2.65  | 0.463   |
| Valve surgery        | 1.09       | 0.56–2.10  | 0.800   |
| Aortic surgery       | 0.44       | 0.22–0.88  | 0.019   |
| Intraoperative bleeding (per 1 dL) | 1.02       | 1.00–1.03  | 0.002   |
| Postoperative pericardial effusion (per 1 mm) | 1.04       | 1.01–1.08  | 0.006   |

Bold indicates p < 0.05

HT hypertension, DM diabetes mellitus, COPD chronic obstructive pulmonary disease, CKD chronic kidney disease, CHF chronic heart failure, TIA transient ischemic attack, CABG coronary artery bypass grafting
Our study also indicated that intraoperative bleeding volume was a positive predictor of POAF, despite the lack of a clinically significant relationship between intraoperative bleeding and pericardial effusion. This could suggest that patients who experienced significant intraoperative bleeding were transfused with blood derivatives, which can trigger a cytokine storm and cause POAF.

Conversely, aortic surgery was a significant negative predictor of POAF in our study, despite having higher intraoperative bleeding volumes. Eikelboom et al. reported that POAF occurred more frequently after CABG and valve surgery [7]. These conclusions also supported the induction of myocardial ischemia and structural remodeling of the atrium by mechanical stress associated with incisions, leading to POAF [6]. Additionally, a previous report observed that cardiopulmonary bypass time affected the occurrence of POAF [19], but we did not observe a statistical difference. The number of patients who underwent CABG in this study was low because they underwent postoperative CT scans within 2–3 days after surgery and were therefore excluded. Therefore, it is challenging to draw conclusions about the effects of CABG on POAF.

The occurrence of POAF despite the administration of β-blockers indicates that POAF can develop secondary to changes in ANS activity, inflammation, or myocardial ischemia and highlights the importance of risk reduction. Effective drainage of pericardial effusion has the potential to reduce inflammation in the cardiac sac and may subsequently decrease the occurrence of POAF. Further studies on this topic have the potential to reveal additional treatment options for POAF.

This study had several limitations. The single-center, retrospective cohort design using electronic charts could have introduced information and selection bias. Additionally, pericardial effusion thickness was calculated manually from CT images, making the measurements slightly subjective. However, these measurements were taken before we investigated POAF occurrence, which limited observer bias.

Conclusions

Age by itself has already been known as the risk factor for atrial fibrillation with or without cardiovascular operation. This study suggests that intraoperative bleeding volume, and postoperative pericardial effusion are positive...
predictive factors of POAF even if we adjusted for age using multivariate logistic regression analysis after multiple imputation. The results also suggest that aortic surgery is a negative predictive factor. However, the causal association between pericardial effusion and POAF remains unclear. Further studies are needed to confirm the role of pericardial effusion in POAF, such as biochemical testing of the pericardial fluid. This could help in understanding the mechanisms behind POAF.

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**Declarations**

**Conflict of interest** The authors have no conflicts of interest directly relevant to the content of this article.

**Ethical approval** The study was approved by the Institutional Review Board of Asahikawa Medical University. Date: 20 April, 2021. Approval Number: 20107.

**Informed consent** Owing to the retrospective design, the requirement for informed patient consent was waived.

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