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Serum C - Reactive Protein Level as a Predictor of New-Onset Postoperative Atrial Fibrillation in Off-Pump Coronary Artery Bypass Grafting

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ABSTRACT

Background: Postoperative atrial fibrillation (POAF) complicates up to 30% of coronary artery bypass grafting procedures and is linked to prolonged hospitalization, increased stroke risk, and higher mortality. Although off-pump CABG (OPCAB) avoids cardiopulmonary bypass–induced inflammation, POAF remains common, suggesting that even minimal surgical trauma provokes an inflammatory environment sufficient to trigger arrhythmia. Serum C-reactive protein (CRP) is an inexpensive, readily available marker of systemic inflammation; if elevated early after surgery, it may identify patients at higher arrhythmic risk and thus guide targeted prophylaxis. **Methods:** In this analytical cross-sectional study, 100 consecutive OPCAB patients (age 35–70 years) were enrolled at the Armed Forces Institute of Cardiology/NIHD, Rawalpindi. Daily serum CRP was measured from postoperative day (POD) 0 through POD5. Patients were stratified into two groups CRP < 10 mg/L versus CRP ≥ 10 mg/L based on the operational definition of elevated inflammation. Continuous ECG monitoring and daily 12-lead ECGs identified new-onset POAF (absence of P waves, irregular RR intervals) within five days. Multivariate logistic regression adjusted for hypertension, diabetes, albumin level, and other confounders. **Results:** POAF incidence was significantly higher in the elevated-CRP group (36% vs. 14%, p=0.02). After adjustment, CRP ≥ 10 mg/L conferred a 1.29-fold increased odds of POAF (95% CI: 1.09–1.52; p=0.03), hypertension a 4.7-fold increase (p=0.02), and hypoalbuminemia a protective but clinically counterintuitive association (aOR 0.05; p=0.01), likely reflecting complex nutritional–inflammatory interactions. Receiver-operator characteristic analysis yielded an AUC of 0.68 for CRP (cut-off 10 mg/L), with 72% sensitivity and 57% specificity, underscoring moderate discrimination. **Conclusion and Implications:** Elevated early postoperative CRP independently predicts POAF in OPCAB patients, affirming the causal role of surgical inflammation in arrhythmogenesis. Although CRP alone offers moderate accuracy, its routine measurement could flag high-risk individuals for intensified monitoring, anti-inflammatory prophylaxis (e.g., colchicine, statins), or early antiarrhythmic therapy. Integrating CRP into multifactorial risk models may enhance personalized perioperative care and ultimately reduce the burden of POAF.

INTRODUCTION

Atrial Fibrillation (AF) is a chaotic irregular atrial rhythm at 300-600 beats per minute (bpm), to which the AV node responds intermittently and hence there is an irregular ventricular rate. It is diagnosed through ECG by lack of p-waves and irregular intervals between R-R waves. It is the most common rhythm abnormality after cardiac surgical procedure. It occurs in 30% of patients undergoing Coronary Artery Bypass Grafting (CABG) and much more frequently in valve replacement cases. In AF, clinically significant drop in the cardiac output often occurs as the ventricles aren't primed reliably by the atria. As a result, the ventricular contractions are ineffective leading to complications like decompensated heart failure (in patients with borderline cardiac reserves) and systemic embolism/ischemic stroke (due to stasis)[1]. In Coronary

Artery Disease (CAD), AF is an unfavorable prognostic indicator and thus needs rapid treatment. It is important to maintain sinus rhythm during and after grafting, in Off-Pump Coronary Artery Bypass grafting (OPCAB) operations. Many factors are known to contribute to new-onset Post-Operative Atrial Fibrillation (POAF) including blood and blood products transfusions, old age, long CPB and Cross-Clamp times (in On-Pump Cases), manual manipulation of the heart during operation, incomplete revascularization and electrolyte disturbances[2]. Several studies have demonstrated the role of inflammation and oxidative stress in the pathogenesis of POAF. Both are consequences of using cardiopulmonary bypass and reperfusion following ischemic cardioplegic arrest in on-pump coronary artery bypass grafting (CABG)[3]. Serum C - reactive protein (CRP) level is a well-known indicator of inflammation. In On-pump CABG inflammatory markers are raised in early post-operative period due to contact of blood with non-endothelial circuit of cardiopulmonary bypass pump. Off-pump CABG (OPCAB), as opposed to traditional on-pump CABG, avoids the use of cardiopulmonary bypass, which can reduce the inflammatory response and associated complications. However, despite this, new- onset atrial fibrillation remains a frequent complication after OPCAB. The underlying mechanisms for AF in off-pump CABG patients are not entirely understood, but it is believed that factors such as surgical trauma, ischemia-reperfusion injury, and inflammatory responses contribute to atrial electrical remodeling and the subsequent development of AF[4]. The purpose of the research is to study the role of high CRP level in early post-operative period, in predicting new-onset POAF in patients undergoing OPCAB.

Rationale of Study

Exploring the potential role of serum CRP levels in predicting POAF in OPCAB patients is of particular interest. If CRP is shown to be an effective predictor, it could provide an easily measurable and inexpensive biomarker for identifying patients at higher risk for POAF, allowing for targeted preventive strategies such as early anti-arrhythmic drug therapy, more intensive monitoring, or prophylactic interventions**.**

Objective

To determine early postoperative serum CRP level as a predictor of new onset postoperative atrial fibrillation (POAF) in patients undergoing off-pump coronary artery bypass grafting (OPCAB).

METHODOLOGY

Study Design and Setting

Analytical Cross-sectional study was conducted at the Department of Cardiac Surgery, Armed Forces Institute of Cardiology and National Institute of Heart Diseases, Rawalpindi, after approval from Institutional Ethical Review Board (IERB) and College of Physicians and Surgeons Pakistan (CPSP) on April 15, 2025, the study continued until the completion of sample size on June 30, 2025.

Inclusion Criteria

All patients aged 35-70 years, of either male or female gender, who underwent OPCAB were included in this study.

Exclusion Criteria

- Those who have congenital heart disease.
- Those with chronic kidney disease and hemodialysis dependence.
- Those who have valve pathology or preoperative rhythm abnormality.
- Pregnant women.
- Those who require coronary endarterectomy or patch-plasty.
- Those who require additional cardiac surgical procedures.
- Those having fever in early postoperative period (till POD#2).
- Conversion to on-pump.

Sampling Technique: Non-probability consecutive sampling technique was used.

Sample Size: Considering the proportions of 0.251 and 0.749 of POAF (+ve) and POAF (-ve) after off-pump CABG,[15] margin of error as 0.05 and power of study as 90% for two-sided test, a sample size of 40 patients was calculated by WHO sample size calculator. However, we'll collect data from 100 patients (n_1=50, n_2=50).

Study Administration and Ethical Issues

Study Plan: After approval of the study proposal from ethical committee and attaining the informed consent of the patients, all patients undergoing OPCAB and fulfilling the inclusion and exclusion criteria were recruited in the study. In all these patients, postoperative CRP levels were documented every morning till discharge, starting morning of POD 1. Then the patients were divided into two groups. Those with CRP level ≥ 10mg/L at any point and those with CRP level < 10mg/L. All these patients were then assessed on POD zero, 1, 2, 3, 4, 5 and at discharge for development of POAF. Presence or absence of POAF with ECG was documented.

Ethical Issues: Patient anonymity and confidentiality were maintained with utmost priority. No personal identifiers such as "patient name or contact details" were included in documentation.

Data Collection, Management and Analysis

Data analysis was performed using SPSS version 28.0 IF006 (IBM Corp. Armonk, NY, USA). The numeric variables were presented as mean and standard deviation (SD) while frequency and percentages were used for categorical variables. Data was stratified by age, use of blood and blood products and incomplete revascularization to deal with effect modifiers. The results were demonstrated in graphical and tabular forms. Post-stratification Cochran Q, followed by McNemar test for pairwise comparison were used at 5% significance level. A multivariate logistic regression model was constructed to assess the predictive capability of CRP levels for new-onset postoperative AF. Adjustments were made for potential confounding variables, such as age, use of blood and blood products, and incomplete revascularization. The results were presented as odds ratios (ORs) with 95% confidence intervals (CIs).

Data collection tool: (questionnaire)

RESULTS

Total n=100 patients were recruited in this study with equal distribution of males and females in both groups. Group II had a significantly higher percentage of diabetes mellitus (80% vs. 50%, p=0.04) and elevated markers of systemic inflammation and cardiac stress, including pro-BNP (p=0.001), HbA1c (p=0.006), CRP/albumin ratio (p=0.001), and INR (p=0.001). This group also showed lower LDL and triglyceride levels and higher HDL (p=0.001 for all. Intraoperatively, Group II had higher cases of incomplete revascularization (p=0.05), residual disease (p=0.05), and poorer conduit quality (p=0.001), although surgery duration was significantly shorter (p=0.001). Postoperatively, patients in Group II experienced higher CRP levels (p=0.001), longer intubation times (p=0.001), increased blood loss (p=0.001), and prolonged hospital stays (p=0.001). Despite a more complicated postoperative course, Group II required fewer RCC transfusions (p=0.001). All procedural parameters are mentioned in Table 1.

Table 1

Comparison of Demographics, Perioperative parameters and Complications between the study groups (n=100)

Variables		`Group-I (n=50) [CRP level <10 (mg/L)]	Group-II (n=50) [CRP level ≥10(mg/L)]	p-value
Demographics		**Mean± SD**		
Age(year)		65.96±6.73	66.74±7.51	0.58
BMI(kg/m²)		27.26±5.87	27.78±7.03	0.68
BSA(m²)		1.95±0.20	1.96±0.17	0.78
Gender		**Frequency (%)**		
	Male	30(60.0%)	30(60.0%)	1.00
	Female	20(40.0%)	20(40.0%)	
Comorbid		**Frequency (%)**		
Hypertension	Yes	35(70.0%)	40(80.0%)	0.35
	No	15(30.0%)	10(20.0%)	
DM	Yes	25(50.0%)	40(80.0%)	0.04
	No	25(50.0%)	10(20.0%)	
Dyslipidemia	Yes	35(70.0%)	35(70.0%)	1.00
	No	15(30.0%)	15(30.0%)	
COPD	Yes	10(20.0%)	10(20.0%)	1.00
	No	40(80.0%)	40(80.0%)	
Smoking	Yes	35(70.0%)	35(70.0%)	1.00
	No	15(30.0%)	15(30.0%)	
IHD	SIHD	20(40.0%)	15(30.0%)	0.63
	NSTEMI	10(20.0%)	10(20.0%)	
	STEMI	10(20.0%)	15(30.0%)	
	UA	10(20.0%)	10(20.0%)	
Preoperative Parameters		**Median(IQR)**		
Pro-BNP (pg/mL)		105.00(95.00-120.00)	176.50(104.00-190.00)	0.001
HbA1c (%)		6.45(5.40-7.20)	8.25(5.50-9.40)	0.006
Albumin(g/dL)		3.75(36.0-3.90)	3.80(3.50-4.20)	0.11
CRP/Albumin Ratio		2.10(1.70-2.40)	8.25(5.50-9.40)	0.001
Lactate (mmol/L)		2.30(1.80-3.00)	1.25(1.00-1.60)	0.001
INR		1.00(1.00-1.10)	1.15(1.00-1.20)	0.001
Electrolytes Concentration		**Mean±SD**		
Na^+ (mmol/L)		126.50±2.75	126.20±2.33	0.55
K^+ (mmol/L)		4.53±0.36	3.91±0.30	0.001
Ca^+ (mmol/L)		8.86±0.38	8.14±0.41	0.001
Liver Status		**Median(IQR)**		
LDL (mg/dL)		150.00(130.00-170.00)	111.50(100.00-130.00)	0.001
HDL (mg/dL)		37.00(32.00-42.00)	46.00(40.00-50.00)	0.001
TRG (mg/dL)		185.00(150.00-230.00)	131.50(120.00-150.00)	0.001
Cardiac Status		**Median(IQR)**		
EF (%)		45.50(43.00-47.00)	46.00(44.00-48.00)	0.08
STS score		3.30(2.90-4.00)	3.10(2.90-3.60)	0.07
CCS		**Frequency (%)**		
	Grade 2	40(80.0%)	40(80.0%)	1.00
	Grade 3	10(20.0%)	10(20.0%)	
NYHA	Class II	40(80.0%)	35(70.0%)	0.35
	Class III	10(20.0%)	15(30.0%)	
Medications		**Frequency (%)**		
Statins		Yes 50(100.0%)	45(90.0%)	0.05
		No -	5(10.0%)	
ACE inhibitors		Yes 35(70.0%)	40(80.0%)	0.35
		No 15(30.0%)	10(20.0%)	
Calcium Channel blockers		Yes 10(20.0%)	35(70.0%)	0.01

			No	40(80.0%)	15(30.0%)	
Anticoagulant			Yes	15(30.0%)	10(20.0%)	0.01
			No	35(70.0%)	40(80.0%)	
Anti-diabetic drugs			Yes	10(20.0%)	20(40.0%)	0.04
			No	40(80.0%)	30(60.0%)	
Diuretics			Yes	15(30.0%)	5(10.0%0	0.02
			No	35(70.0%)	45(90.0%)	
Intraoperative Parameters				**Frequency (%)**		
Incomplete Revascularization			Yes	50(100.0%)	45(90.0%)	0.05
			No	0	5	
Multi-Arterial			Yes	35(70.0%)	25(50.0%)	0.06
			No	10(20.0%)	25(50.0%)	
Rhythm Abnormality			Yes	5(10.0%)	5(10.0%)	1.00
			No	45(90.0%)	45(90.0%)	
Residual Disease			Yes	0	5(10.0%)	0.05
			No	50(100.0%)	456(90.0%)	
No. of Revascularized Vessels			2	10(20.0%)	5(10.0%)	0.36
			3	25(50.0%)	30(60.0%0	
			4	15(30.0%)	15(30.0%)	
Inotropic Support			Mild	35(70.0%)	30(60.0%)	0.08
			Moderate	15(30.0%)	15(30.0%)	
			High	0	5(10.0%)	
Quality of Conduits			Good	50(100.0%)	40(80.0%)	0.001
			Bad	-	10(20.0%)	
Quality of Targets			Good	35(70.0%)	35(70.0%)	
			Bad	15(30.0%)	15(30.0%)	
Rhythm Managed By			Not Applicable	45(90.0%)	45(90.0%)	1.00
			DCCV	5(10.0%)	5(10.0%)	
Duration of Surgery (minutes)				**Median(IQR)**		0.001
				134.00(124.00-142.00)	115.00(102.00-121.00)	
Post-operative Parameters				**Median(IQR)**		
CRP(mg/L)				7.60(6.30-8.90)	15.45(11.50-17.10)	0.001
Intubation (hours)				11.50(8.00-16.00)	22.50(21.00-27.00)	0.001
Blood Loss(ml)				357.50(220.00-540.00)	415.00(280.00-520.00)	0.001
Hospital stay(days)				6.00(5.00-7.00)	9.50(8.00-11.00)	0.001
Complications	Reintubation		Yes	-	5(10.0%)	0.05
			No	50(100.0%)	45(90.0%)	
	AKI		Yes	10(20.0%)	10(20.0%)	1.00
			No	40(80.0%)	40(80.0%)	
	Re-exploration		Yes	-	5(10.0%)	0.05
			No	50(100.0%)	45(90.0%)	
No. of RCC Transfused				1.50(1.00-2.00)	1.00(0.00-1.00)	0.001

Figure 1

Distribution of POAF among the Study Groups (n=100)

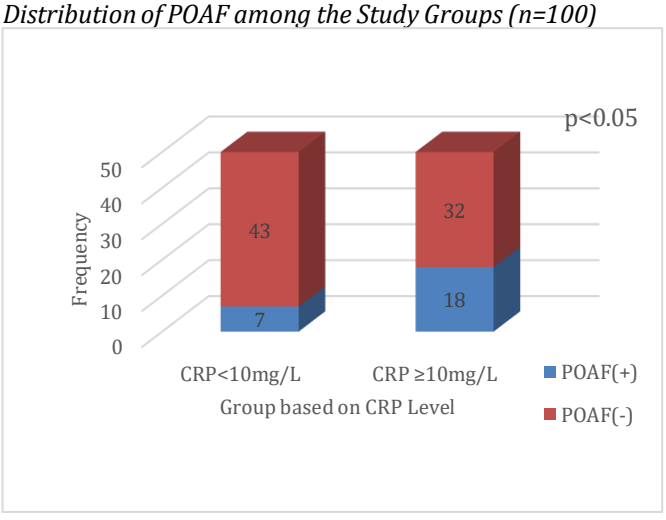

Figure 1 shows a slightly higher incidence of POAF among patients with CRP ≥10 mg/L (18 vs. 7), with the statistical significant difference (p = 0.02).

In Group I, hemoglobin (Hb) levels dropped from 13.20 ± 0.48 g/dL preoperatively to 9.87 ± 1.13 g/dL postoperatively, while in Group II (CRP ≥10 mg/L), Hb decreased from 12.99 ± 0.91 g/dL to 10.04 ± 0.96 g/dL (p=0.001). Platelet counts declined in both groups, from 223.70 ± 58.07 × 10^9/L to 173.88 ± 25.24 × 10^9/L in Group I, and from 231.60 ± 57.60 × 10^9/L to 171.97 ± 27.14 × 10^9/L in Group II as shown in Table 2.

Table 2

Preoperative and Post-Operative Hematological Parameters of Study Participants (n=100)

Variables	Group-I		Group-II		p-value
	Pre-op	Post-op	Pre-op	Post-op	
Hb (g/dL)	13.20 ± .48	9.87 ± 1.13	12.99 ± 0.91	10.04 ± 0.96	0.001
TLC (×10^9/L)	7.83 ± 1.07	13.22 ± 2.58	8.950 ± 1.11	12.28 ± 2.51	0.001
PLT(×10^9/L)	223.70 ± 58.07	173.88 ± 25.24	231.60 ± 57.60	171.97 ± 27.14	0.001
Lymphocyte Count (%)	1.15 ± 0.29	16.07 ± 5.21	1.55 ± 0.29	17.13 ± 5.09	0.001
Neutrophil Count (%)	6.93 ± 1.30	76.41 ± 7.24	5.04 ± 0.75	75.18 ± 6.95	0.001

In univariate analysis, elevated CRP levels (≥10 mg/L) and hypertension were significantly associated with increased

odds of POAF, while diabetes and low albumin levels were associated with lower odds. After adjustment, CRP (aOR: 1.29, p=0.03), HTN (aOR: 4.69, p=0.02), and albumin (aOR: 0.05, p=0.01) remained independent predictors of POAF. Age, EF, lipid profile parameters, and blood counts were not significantly associated with POAF. (Table 3).

Table 3

Predictors of POAF

Variable	POAF			
	uOR (95% CI)	p-value	aOR (95% CI)	p-value
CRP(≥10mg/L)	1.14 (1.02-1.27)	0.01	1.29 (1.09-1.52)	0.03
Age (years)	1.01 (0.95-1.08)	0.61		
HTN	3.42 (1.28-9.12)	0.01	4.69 (1.17-18.70)	0.02
Diabetes	0.49 (0.27-1..89)	0.02	0.08 (0.01-0.58)	0.01
EF(%)	1.02 (0.84-1.23)	0.81		
LDL (mg/dL)	1.00 (0.98-1.05)	0.61		
HDL (mg/dL)	0.96 (0.90-1.03)	0.32		
Triglycerides (mg/dL)	1.01 (0.99-1.03)	0.68		
Albumin (g/dL)	0.26 (0.06-1.16)	0.02	0.05 (0.006-0.52)	0.01
CRP/Albumin Ratio	1.48 (0.96-2.83)	0.97		
Hb (g/dL)	0.98 (0.64-1.52)	0.96		
TLC ($\times 10^9$/L)	0.90 (0.98-1.01)	0.29		
PLT($\times 10^9$/L)	1.01 (0.85-1.20)	0.77		
Lymphocyte Count (%)	1.00 (0.92-1.10)	0.76		
Neutrophil Count (%)	0.97 (0.97-1.06)	0.38		

Figure 2

ROC Curve Showing CRP Level's Ability to Predict POAF

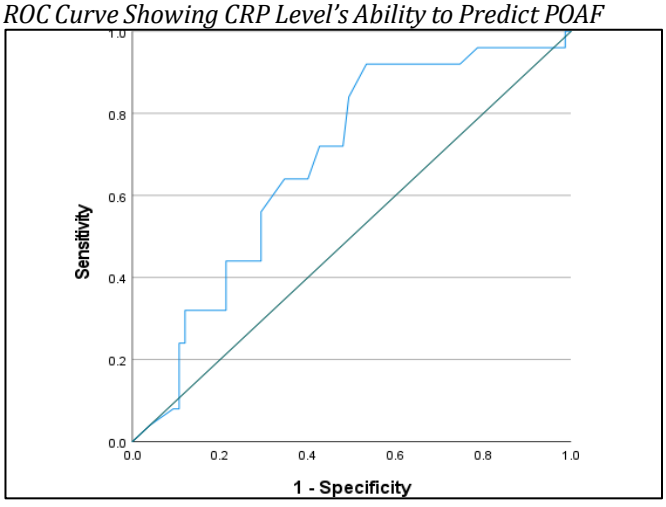

Figure 2 demonstrated limited accuracy in predicting POAF, with an AUC of 0.68 (95% CI: 0.56–0.79; p=0.001), indicating poor discriminative ability. At the cut-off value of 10.00, the test showed moderate sensitivity 72% but low specificity 57.3%.

DISCUSSION

In our study of OPCAB patients, Group II (CRP ≥10 mg/L) exhibited a markedly higher incidence of new-onset POAF as well as evidence of more pronounced systemic inflammation and a complicated perioperative course. Group II had significantly greater comorbidity burden (e.g. diabetes, hypertension) and higher biomarkers of inflammation than the Group I (CRP<10). Intraoperatively, this cohort suffered more technical challenges – incomplete revascularization, residual coronary disease, and poorer conduit quality – suggesting advanced disease and inflammatory vascular changes. Postoperatively, Group II required longer ventilation and ICU stays, with significantly longer overall hospitalization. These findings are consistent with the well-known sequelae of POAF: patients who develop atrial fibrillation after cardiac surgery suffer longer intensive care and hospital stays and higher rates of complications.[5] Importantly, our multivariable analysis confirmed that CRP elevation independently predicted POAF (adjusted OR≈1.3), underscoring the link between inflammation and arrhythmogenesis. In sum, patients with CRP≥10 mg/L formed a high-risk subgroup characterized by more inflammation and poorer outcomes, reinforcing the prognostic significance of perioperative inflammatory status for POAF risk.

The association of elevated CRP and POAF fits well with the current understanding that acute surgical inflammation is a key trigger for postoperative atrial arrhythmias. Cardiac surgery induces a marked systemic inflammatory response – even without cardiopulmonary bypass – and postoperative CRP typically peaks around 48 h, coinciding with the observed peak incidence of POAF.[6, 7] Mechanistically, pro-inflammatory cytokines (e.g. IL-6, IL-1β) released in this setting promote atrial electrical and structural remodeling. For example, inflamed peri-atrial adipose tissue secretes IL-1β and other mediators that slow atrial conduction velocity and increase heterogeneity, thereby facilitating re-entrant atrial fibrillation. Inflammation also amplifies other triggers of AF: surgical trauma and reperfusion injury lead to sympathetic overactivity, calcium overload, and oxidative stress, all of which further shorten atrial refractoriness.[8, 9] Consistent with this paradigm, anti-inflammatory interventions have proven to reduce POAF in trials: the COPPS trial[10] showed colchicine cut POAF incidence from 22% to 12% (≈45% relative risk reduction), highlighting the contributory role of inflammation.[11] Likewise, several studies suggest statin therapy (with its anti-inflammatory effects) lowers POAF risk.[12] In sum, our data reinforce the concept that heightened perioperative inflammation (for which CRP is a convenient biomarker) predisposes to POAF by creating an arrhythmogenic substrate in the post-surgical heart.

Our findings align with recent literature linking CRP and systemic inflammation to postoperative AF. For instance, Del Monaco and colleagues (2024)[13] reported that among a broad surgical population, patients who developed postoperative AF had significantly higher CRP levels, and CRP remained an independent predictor of POAF (HR ~1.8 per log-unit increase). Similarly, Mittal et al. (2024)[3] showed that patients developing new-onset AF after cardiac surgery had significantly higher preoperative CRP, with elevated CRP correlating with increased AF risk. These observations echo earlier reports in cardiac cohorts:

for example, in mixed CABG populations, patients with higher baseline or early postoperative CRP were far more likely to develop POAF than those with lower CRP (e.g. one study found 30% vs 8% AF incidence in off-pump patients with high vs low preop CRP).[14] At the same time, meta-analytic data remind us of CRP's moderate discriminative power. A recent meta-analysis[15] noted that while higher preoperative CRP was associated with greater odds of POAF, the effect size was modest (OR≈1.3) and predictive accuracy limited. Our own ROC analysis (AUC≈0.68) similarly indicates only fair discrimination by CRP alone. These observations suggest that, although CRP elevation is a marker of increased POAF propensity, it should be viewed as part of a multifactorial risk profile. Indeed, composite inflammatory indices (e.g. systemic immune–inflammation index, CRP/albumin ratio, neutrophil-to-lymphocyte ratio) have shown stronger associations with POAF than CRP alone. For example, pooled data show an elevated Systemic Immune-Inflammatory Index more than triples the odds of POAF (OR≈3.2), while CRP's independent predictive value is comparatively weaker. Thus, our results highlight CRP as a valuable but not definitive predictor; integrating CRP with other markers and clinical variables may yield better risk stratification. Clinically, these findings have important implications for perioperative management. An easily obtained CRP measurement may help identify OPCAB patients at especially high risk for AF. Such patients could benefit from intensified prophylaxis or monitoring. Notably, though CRP itself is not currently incorporated in standard risk algorithms, expert consensus increasingly acknowledges inflammation as a modifiable driver of POAF. For example, the observed relationship between elevated CRP and AF supports considering anti-inflammatory strategies in high-risk cases. Aside from colchicine (as noted above), trials have shown that preoperative beta-blockers or statins (which also dampen the inflammatory response) reduce POAF incidence. Moreover, guidelines on AF[16] and perioperative care[17] now emphasize aggressive management of risk factors – including optimizing treatment of comorbidities such as hypertension, heart failure, and diabetes (all of which were overrepresented in our high-CRP group). While no professional guideline yet recommends routine CRP screening for POAF risk, our data (in concert with recent literature) suggest that elevated perioperative CRP is a red flag warranting closer surveillance and possibly prophylactic therapy.

Finally, the concordance of our results with recent studies underlines the broader pathophysiologic principle that surgical inflammation and AF are tightly linked. It is noteworthy that even in off-pump surgery – which avoids cardiopulmonary bypass – significant systemic inflammation can still occur, as reflected by CRP. This implies that OPCAB patients with heightened inflammatory markers carry a similar arrhythmic risk as on-pump patients. Our study thus extends the inflammatory hypothesis of POAF into the OPCAB setting. Future work should investigate whether combining CRP with other biomarkers (e.g. natriuretic peptides, cellular inflammation indices) improves prediction, and whether targeted anti-inflammatory interventions in high-CRP patients can reduce POAF and its sequelae. In sum, our findings support that a robust perioperative inflammatory response – indicated by elevated CRP – identifies a subset of OPCAB patients who are more prone to AF and to prolonged, complication-prone recovery. Recognizing this link helps explain our observations and is corroborated by multiple recent analyses, underscoring the utility of inflammatory markers in understanding and potentially guiding prevention of POAF.

CONCLUSION

The study concluded that elevated early postoperative CRP independently predicts POAF in OPCAB patients, affirming the causal role of surgical inflammation in arrhythmogenesis. Although CRP alone offers moderate accuracy, its routine measurement could flag high-risk individuals for intensified monitoring, anti-inflammatory prophylaxis (e.g., colchicine, statins), or early antiarrhythmic therapy. Integrating CRP into multifactorial risk models may enhance personalized perioperative care and ultimately reduce the burden of POAF.

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