REVIEW

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Peri-postoperative Atrial Fibrillation in Non-cardiothoracic Surgeries: Approach of the Anesthesiologist

Kardiyotorasik Olmayan Cerrahilerde Peri-postoperatif Dönem Atriyal Fibrilasyon: Anesteziyolog Yaklaşımı

Hülya Yılmaz Ak[1], Kübra Taşkın[1], Kerem Erkalp[2]

[1]University of Health Sciences Turkey, Kartal Dr. Lütfi Kırdar City Hospital, Clinic of Anesthesiology and Reanimation, İstanbul, Turkey
[2]İstanbul University-Cerrahpaşa, Institute of Cardiology, Department of Anesthesiology and Reanimation, İstanbul, Turkey

Abstract

In this study, our aim was to summarize the current knowledge on the epidemiology, pathophysiology and management of new-onset perioperative and postoperative atrial fibrillation (POAF) in non-cardiothoracic surgery and to provide a practical approach for anesthesiologists and non-cardiologist clinicians. Various findings such as age, hypertension, diabetes mellitus, cardiac risk factor, premature beats on preoperative electrocardiogram, left anterior fascicular block or left ventricular hypertrophy pose an elevated risk for POAF. The first thing to do in patients with POAF is to determine the origin of the arrhythmia. In most cases, identifying and eliminating the triggering cause will suffice. On the other hand, hemodynamic data should be evaluated. The primary goal of treatment in patients with life-threatening symptoms is to maintain hemodynamic stability. Deterioration of hemodynamic stability and development of shock with AF with high ventricular rate is a condition that requires immediate cardioversion. Rate control therapy increasing dose with continuous cardiac monitoring to a heart rate <110 should be performed on hemodynamically stable patients with POAF. β-blockers and non-dihydropyridine calcium channel blockers (diltiazem and verapamil) are used for rate control in AF. If there is peripheral vascular disease, congestive heart failure, diabetes, hypertension or history of thromboembolic event, attention should be paid and postoperative bleeding risk should be calculated. There is a risk of bleeding in the postoperative period and POAF usually lasts less than 24 hours and improves spontaneously, and the use of heparin at a therapeutic dose is not required. As a general rule, therapeutic doses of anticoagulants are recommended for POAF lasting longer than 48 hours and for frequent recurrent AF attack.

Keywords: Anesthesia, atrial fibrillation, non-cardiothoracic surgery

Öz

Bu makalede, anesteziyologlar ve kardiyolog olmayan klinisyenler için kardiyotorasik olmayan cerrahilerde yeni başlayan perioperatif ve postoperatif atriyal fibrilasyonun (POAF) epidemiyolojisi, patofizyolojisi ve yönetimine ilişkin mevcut bilgileri özetlemeyi ve pratik bir yaklaşım sağlamayı amaçladık. Yaş, hipertansiyon, diabetes mellitus, kardiyak risk faktörü, preoperatif elektrokardiyogramda erken atımlar, sol anterior fasiküler blok veya sol ventrikül hipertrofisi gibi çeşitli bulgular POAF için daha yüksek risk oluşturur. POAF'li hastalarda yapılması gerekenlerin başında, bu aritminin altında yatan nedenin belirlenmesi gelmektedir. Çoğu durumda, tetikleyici nedenin tespit edilip ortadan kaldırılması yeterli olacaktır. Bununla birlikte hemodinamik veriler değerlendirilmelidir. Yaşamı tehdit eden semptomatik hastalarda tedavinin ilk amacı, hemodinamik stabiliteyi sağlamaktır. Yüksek ventrikül hızlı AF ile hemodinamik stabilitenin bozulması, şok tablosunun gelişimi acil kardiyoversiyon gerektiren bir durumdur. POAF'si olan hemodinamik olarak stabil hastalara, kalp hızı <110 olacak şekilde sürekli kardiyak monitörizasyon yapılarak artan dozda hız kontrol tedavisi yapılmalıdır. β-blokerler ve dihidropiridin grubu olmayan kalsiyum kanal blokerleri (diltiazem ve verapamil), AF'de hız kontrolünün sağlanmasında kullanılır. Konjestif kalp yetmezliği, hipertansiyon, diyabet, geçirilmiş tromboembolik olay, periferik damar hastalığı varsa dikkat edilmeli ve ameliyat sonrası kanama riski hesaplanmalıdır. Postoperatif süreçte kanama riskinin olması ve POAF'nin çoğunlukla 24 saatten daha kısa sürüp kendiliğinden geçmesi tedavi dozunda heparin kullanımını gerektirmez. Genel bir kural olarak 48 saatten uzun sürenlerde ve sık tekrar eden AF ataklarında tedavi edici dozda antikoagülan önerilmektedir.

Anahtar kelimeler: Anestezi, atriyal fibrilasyon, kardiyotorasik dışı cerrahi

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Address for Correspondence: Hülya Yılmaz Ak, University of Health Sciences Turkey, Kartal Dr. Lütfi Kırdar City Hospital, Clinic of Anesthesiology and Reanimation, İstanbul, Turkey
E-mail: hulyayilmazak@gmail.com **ORCID:** orcid.org/0000-0002-8682-6254 **Received:** 13.07.2022 **Accepted:** 24.08.2022

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Introduction

Atrial fibrillation (AF) is the condition caused by supraventricular tachyarrhythmia with uncoordinated atrial electrical activation and this leads to inefficient atrial contraction. Irregular R-R intervals despite usual atrioventricular conduction, missed obvious P wave repeats, and irregularities in atrial activation are the electrocardiographic characteristics of AF (1). New-onset atrial arrhythmias have been reported to develop in 16-46% of patients after cardiac surgery, in 3-30% of patients after thoracic surgery, and in up to 8% of non-cardiothoracic surgery patients (2). Following non-cardiothoracic surgery, AF usually occurs within the first four postoperative days. In this article, we aimed to summarize the up-to-date knowledge on the epidemiology, pathophysiology and management of new-onset perioperative and postoperative AF (POAF) in non-cardiothoracic surgery and to provide a practical approach for anesthesiologists and non-cardiologist clinicians.

Perioperative AF is common in cardiothoracic surgeries and is thought to occur secondary to direct mechanical myocardial or pericardial irritation (3). However, the pathophysiology of AF associated with non-cardiothoracic surgery remains elusive. Its cause is suggested to be a preoperative or postoperative inflammatory response triggering the dysregulation in the electrical activity in atrial myocytes (4). Moreover, increased sympathetic activity due to stress from surgery and anesthesia predisposes the patient to arrhythmias. Clinical conditions including intraoperative hypotension, hypovolemia, anemia, trauma and pain may have an impact on sympathetic activity. Several other mechanisms that may trigger arrhythmia includes electrophysiological disorders and metabolic dysregulations like hypoglycemia and electrolyte imbalance. Hypoxia may also cause arrhythmias via vasoconstriction of pulmonary vein and may lead to increase in the right ventricular pressure and right atrial distension. In addition, hypoxia may lead to ischemia of atrial myocardial cells by changing the conductive system in the cardiac tissue. Hypovolemia is also suggested to be another contributing mechanism that may lead to AF development. Increased intravascular volume caused by hypervolemia leads causes stretching of the right atrium and triggers the development of AF (5).

Prevention

Several studies have reported several predictors for POAF (2,6,7). Knowing these predictors and being prepared are important for the management of AF. Age is an important predictor of POAF when demographic data are considered. The incidence of AF increases with age. The incidence of AF is 2.3% in people older than 40 years, and this rate increases to 5.9% in people older than 65 years (8,9). Men have been reported to have a higher POAF incidence compared to women (10). On the other hand, the effect of body mass index (BMI) on AF is controversial. Although BMI has been defined as a risk factor for AF in some cardiac surgery studies, no significant effect of BMI on AF development has been demonstrated in some studies (11-13).

It is well established that hypertension is a risk factor for POAF, as indicated by both animal and human studies (6,14). Possible hemodynamic mechanisms and left atrial distension and pressure, as well as an increased thickness of left ventricular wall, predispose to AF.

Studies have reported that diabetes mellitus (DM)-related inflammation may contribute to the pathophysiology of AF (15,16). In addition, in a study by Iguchi et al. (7), DM was shown to be an independent factor associated with AF. It is very important whether the patients have a previous cardiac risk factor for the POAF development. In the study of Christians et al. (17) on non-cardiothoracic surgery patients, 67% of the patients exhibited at least one cardiac risk factor (18). Besides the clinical factors, various findings on the preoperative electrocardiogram including premature atrial complexes, left anterior fascicular block, or left ventricular hypertrophy constitute the higher risk factors of POAF.

Management

The first thing to do in patients with POAF is to determine the underlying cause of this arrhythmia. In most cases, identification and elimination of the triggering cause will suffice. On the other hand, hemodynamic data should be evaluated. The primary goal of treatment in patients with life-threatening symptoms is to maintain hemodynamic stability. Deterioration of hemodynamic stability and development of shock with AF with high ventricular rate is a condition that requires immediate cardioversion [Class I-European Society of Cardiology (ESC)] (Table 1). Direct transthoracic cardioversion is effective in converting the heart rhythm of the patient to sinus rhythm. Moreover, the need for anesthesia-analgesia should not be overlooked, as there will be pain and discomfort in cardioversion where high-energy electrical shocks are applied.

Rate control therapy including increasing dose with continuous cardiac monitoring to a heart rate <110

Table 1. Peri-postoperative atrial fibrillation in non-cardiothoracic surgeries: Practical approach

Management of peri-postoperative atrial fibrillation

Identify and treat the factors that trigger AF!
Hypoxia?-Infection?
Electrolyte abnormality?-Hypovolemia?
CAD or CHF?-Bleeding?

Stable hemodynamics
Rate control to aim for HR <110
If decompensate HF or EF <35%, administer amiodarone, NOT betablocker or CCB.
1. Apart from this, administer metoprolol 2 mg IV (for 1 minute), repeat every 5-10 minutes if a positive response is obtained and no side effects occur, the initial maximum IV dose should be 10 mg in total.
2. If there is no response or insufficient response to >4 mg metoprolol, administer diltiazem 5 mg (within 1 min), if positive response is obtained and no side effects occur, top up to a maximum of 20 mg at 10-minute intervals.

Unstable hemodynamics
Pale, shock, hypotension, decreased peripheral pulses, pulmonary edema, chest pain
Synchronized cardioversion at 200 joule monophase

Resistant ventricular HR or side effects are present
1: Digoxin 0.5 mg IV then 0.25 mg IV in 6 h. and if renal function is normal, 0.25 mg IV in 12 h (Not first-choice drug alone. It can be combined with beta-blocker and CCB in resistant AF).
2: Amiodarone 150 mg IV over 10 min followed by infusion for rate and rhythm control (decompensated heart failure, patient with EF <35)

Resistant AF lasting more than 48 hours

Patient group with risk
(Congestive heart failure, hypertension, diabetes, previous thromboembolic event, peripheral vascular disease, cerebrovascular disease etc.)
▪ Consult cardiology!
▪ What is the bleeding risk?
Low risk of bleeding: Start warfarin
High risk of bleeding: Electrical cardioversion and low molecular weight heparin (LMWH) are recommended after control with TEE.

Patient group without risk
▪ Daily oral acetylsalicylic acid treatment recommended

CAD: Coronary artery disease, HR: Heart rate, IV: Intravenous, EF: Ejection fraction, CHF: Cardiac heart failure, CCB: Calcium channel blocker, AF: Atrial fibrillation, TEE: Transesophageal echo

(class I-ESC) should be performed in hemodynamically stable patients with POAF (Table 1). β-blockers and non-dihydropyridine calcium channel blockers (CCB) (diltiazem and verapamil) are used for rate control in AF (19). After non-cardiac surgery, β-blockers accelerate the conversion of AF to sinus rhythm. Moreover, preoperative use of β-blockers has been associated with better control of arrhythmia in several studies. Digoxin alone itself is not the drug of first choice in acute AF, except for patients with congestive heart failure. Nevertheless, digoxin can be used in combination with beta-blockers and CCB in resistant AF (20). Amiodarone is generally preferred when other antiarrhythmic drugs are ineffective or cannot be used due to its serious side effects (heart block, bradycardia, hypotension, pulmonary fibrosis, and thyroid and hepatic dysfunction). In patients with decompensated heart failure or with ejection fraction <35%, amiodarone should be given instead of beta-blockers and CCBs because of their negative inotropic effects (20).

There is a risk of bleeding in the postoperative period and POAF usually lasts less than 24 hours and improves spontaneously, and the use of heparin at a therapeutic dose is not required. As a general rule, therapeutic doses of anticoagulants are recommended for POAF lasting longer than 48 hours and for frequent recurrent AF attack (21,22). Before starting anticoagulant therapy, it should be personalized for the patient. In the cases of hypertension, congestive heart failure, diabetes, history of thromboembolic event and peripheral vascular disease, attention should be paid and postoperative bleeding risk should be calculated (Table 1) (22).

In the postoperative period, AF usually occurs within the first four days, and the hearth rhythm of most patients who develop AF returns to spontaneous sinus rhythm. Therefore, many physicians question the necessity of diagnosing and treating this self-limiting arrhythmia that does not usually result in hemodynamic deterioration and mortality. Nevertheless, evidence obtained since the 1980s indicates poor prognosis with an increased risk of postoperative complications of POAF (19,22). POAF should not be underestimated as a temporary complication occurring only in the perioperative period because it can cause serious and fatal problems in non-cardiothoracic surgery. Previous studies indicated that POAF could recur in patients, leading to cardiac complications and embolic

events (23). In addition, higher mortality rates were observed in patients with POAF (16,24,25). Therefore, documentation of POAF is necessary even in asymptomatic patients.

To decrease the risk of POAF, prophylactic treatment with β-blocking agents, amiodarone, CCB, and magnesium may be acknowledged in the pre-surgical period. Notwithstanding, considering the side effects of these drugs, which will only benefit a few patients in prevention of POAF, it is pointless to use them in all patients. Prophylaxis can be considered to decrease the incidence and severity of POAF only in high-risk patients (uncontrolled DM, coronary artery disease, heart failure, cerebrovascular events, etc.); however, more studies regarding the safety and efficacy of this approach are required (16).

Although POAF is not as common as in cardiac surgery, it can also be encountered in non-cardiothoracic surgery. In particular, POAF, which affects hemodynamics, should be treated quickly. In addition, β-blockers and CCB have a high efficacy in the intervention of POAF in patients with stable hemodynamics. Amiodarone should be preferred in heart failure patients. Anticoagulant therapy should be considered in AF attacks lasting more than 48 hours or frequently recurring. Anesthesiologists must be prepared for POAF and also document it, even in asymptomatic patients.

Ethics

Peer-review: Internally peer-reviewed.

Authorship Contributions

Concept: H.Y.A., K.T., K.E., Design: H.Y.A., K.T., K.E., Literature Search: H.Y.A., K.T., K.E., Writing: H.Y.A.

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