Relationship between acute atrial fibrillation attack and intensive cigarette smoking

Yoğun sigara kullanımı ve akut atriyal fibrilasyon atağı arasındaki ilişki

Erdal Aktürk, İsa Sincer[1], Aytekin Güven[2], İrfan Barutçu[3]

Department of Cardiology, Faculty of Medicine, Adıyaman University, Adıyaman-*Turkey*
[1]Clinic of Cardiology, Avukat Cengiz Gökçek Hospital, Gaziantep-*Turkey*
[2]Department of Cardiology, Faculty of Medicine, Başkent University, Konya-*Turkey*
[3]Clinic of Cardiology, Avicenna Hospital, İstanbul-*Turkey*

Smoking is a firmly established cardiovascular risk factor. In general, it causes endothelial dysfunction, atherosclerosis and cardiac rhythm disorders through the combined effects of nicotine, carbon monoxide, and polycyclic aromatic hydrocarbons (1, 2). Smoking may thus change the myocardial substrate as well as action potentials. Both processes provoke and facilitate cardiac rhythm disorders.

Atrial fibrillation (AF) is the most common sustained cardiac rhythm disturbance. Several case reports are presented on the onset of AF after the ingestion of nicotine but the results of population-based studies on the association between smoking and atrial fibrillation are conflicting (1-3). We attempted to investigate the possible relationship between intensive cigarette smoking and acute atrial fibrillation (AAF) in patients admitted to emergency department with a diagnosis of AAF.

Forty-two patients admitted to İnönü University, Faculty of Medicine, emergency department with a diagnosis of AAF were included to study. All patients returned to normal sinus rhythm (NSR) by medical therapy. Detailed questioning and physical examination, echocardiographic examination, routine complete blood count, biochemical analysis and thyroid function tests were performed.

Cigarette smoking was defined as smoking ten or more cigarettes a day for at least 1 year without a quit attempt. Intensive cigarette smoking was defined as increased daily cigarette consumption by at least 50 percent immediately before the start of symptoms. The number of years of habitual smoking and the number of cigarettes smoked per day were also calculated. The number of cigarettes smoked (pack/day) was multiplied with the duration of smoking (years) and was expressed as the pack-years.

Patients who had etiologic factors for AAF other than smoking were carefully excluded, also obesity, vigorous exercise, hematological, pulmonary or any other systemic disorders, alcohol abuse or caffeine intake, and chronic cigarette smoking but had no history of intensive cigarette smoking within the last 1 day when the AAF was diagnosed.

All patients underwent 24-hour Holter electrocardiogram (ECG) monitoring two times during follow-up period. The first monitoring was performed 1 week after discharge from the hospital.

The second monitoring was performed after 1 month of treatment. Non-invasive stress tests or coronary angiography were also performed in those with suspected coronary artery disease. Atherosclerosis Risk In Communities (ARIC) (4) study's scoring system for developing of AF were done in all patients included in the study.

Baseline demographic characteristics of patients are summarized in Table 1. Sedative drugs (diazepam or midazolam) and low molecular weight heparin (enoxaparin) were started and continued until the sinus rhythm and effective atrial contractions could be obtained. Sinus rhythm was provided with i.v. amiodarone (in 23 cases) i.v. esmolol (in 12 cases) or i.v. propafenone (in 7 cases). Oral medications was also started and prescribed after discharge from the hospital.

First Holter records revealed that AAF repeated in 3 subjects one week later because they did not take drugs and sustained the cigarette. During follow-up period; 20 subjects quitted the smoking with (n=6) and without (n=14) medical support, the other persons is quite reduce cigarette smoking. In the second Holter monitoring, there was neither AF nor any other rhythm disturbances. Cigarette

Address for Correspondence/Yazışma Adresi: Dr. Erdal Aktürk, Adıyaman Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Adıyaman-*Türkiye*
Phone: +90 416 223 16 90 E-mail: erdalakturk@hotmail.com
Accepted Date/Kabul Tarihi: 26.03.2012 **Available Online Date/Çevrimiçi Yayın Tarihi:** 15.06.2012

doi:10.5152/akd.2012.160

Table 1. Baseline demographic characteristics of patients

Age, years	40±8
Female/male, n	15/27
Body mass index, kg/m^2	25±3
Systolic blood pressure, mmHg	106±13
Diastolic blood pressure, mmHg	66±5
LDL cholesterol level, mg/dL	104±20
HDL cholesterol level, mg/dL	37±8
Triglycerides level, mg/dL	130±33
Total cholesterol level, mg/dL	168±20
Thyroid stimulating hormone, IU /mL	1.19±0.3
Total triiodothyronine, ng/dL	4.8±3.1
Total thyroxine, ng/dL	1.31±0.24
Free triiodothyronine, pg/mL	1.89±1.05
Free thyroxine, ng/dL	2.26±0.6
Left ventricular ejection fraction, %	64±6
Left ventricular end-diastolic diameter, mm	48±2
Left ventricular end-systolic diameter, mm	34±2
Left atrial diameter, mm	32±3
Right atrial diameter, mm	31±4
Septal thickness, mm	9±2
Posterior wall thickness, mm	9±1
Mean pulmonary artery pressure, mmHg	20±4
ARIC AF risk scoring	4.6±1.3

Data are presented and numbers and mean±SD values
AF - atrial fibrillation, HDL - high-density lipoprotein, LDL - low-density lipoprotein

Table 2. Cigarette consumption and time results

Onset of smoking age, years	25±7
Duration of habitual smoking, years	12±7
Amount of smoking, pack-years	15±3.5
Intensive cigarette smoking, cigarette/day, %	23±7, ≥50
Duration from the onset of complaints to emergency room, hours	10.5±6
Returning time to NSR, hours	11±6
Follow-up time, months	7±3

Data are presented as mean±SD values and percentage
NSR - normal sinus rhythm

consumption and time results are shown in the Table 2. There was a positive correlation between the returning time to NSR and age, amount of cigarettes (pack-years) and right atrium (RA) diameter, returning time to NSR and pack-years (r=0.656 p=0.0001, r=0.523 p=0.0001, r=0.726 p<0.0001, respectively). There is a dose dependent relationship between cigarette smoking and returning time to sinus rhythm from AAF (r=0.726, p=0.0001).

Cigarette smoking is implicated in a wide spectrum of cardiac rhythm disorders, including transient sinus arrest and/or bradycardia, sinus tachycardia, atrial fibrillation, sinoatrial block, AV block, and ventricular tachyarrhythmias (2, 3, 5, 6).

A previous study has found that smoking has a triggering effect in the initiation of AF and atrial arrhythmias by changing the structure of the atrium and causing atrial fibrosis (7). Other study has found that the prolonged administration of nicotine is also associated with the loss of intracellular K+ and the emerging of cardiac necrosis (8). Another, evaluating the effects of smoking on left atrial (LA) and RA by electrophysiological study. It has been shown that smoking has a voltage reduction effect on RA and LA, also the total activation time of RA has been longer in the smoker patients, but that is not the case in LA (9). In our study we found that RA diameter was well correlated with amount of cigarettes consumption, but no correlation between amount of cigarettes and LA dimension.

Today the most commonly used scoring system for ten-year risk of AF development are based on ARIC Study. According to ARIC AF risk scoring system, our patient's ten-years total risk of developing AF in the smokers is 2% or less. Although the percentages of the risk for developing AF were very low in the subjects included in our study, chronic smokers have come to the emergency room with AAF after intensive cigarette consumption. These results, we can speculate that smoking might be an independent predictor for the development of AF. Therefore, this should be kept in mind as an etiological factor in those subjects who presented to emergency department with AAF. However, our overall findings should be confirmed by large scale and long-term follow-up studies.

Acknowledgement

We would like to thank Assist. Prof. Dr. Nusret Açıkgöz, Assoc. Prof. Dr. Necip Ermiş, Prof. Dr. Hasan Pekdemir and Prof. Dr. Ramazan Özdemir, from Inönü University, Faculty of Medicine, Department of Cardiology, for their contributions to the study.

Conflict of interest: None declared.

References

1. Lakier JB. Smoking and cardiovascular disease. Am J Med 1992; 93: 8-12. [CrossRef]
2. Benowitz N. Drug therapy: Pharmacologic aspects of cigarette smoking and nicotine addiction. N Eng J Med 1988; 319: 1318-30. [CrossRef]
3. Heeringa J, Kors JA, Hofman A, van Rooij FJ, Witteman JC. Cigarette smoking and risk of atrial fibrillation: the Rotterdam Study. Am Heart J 2008; 156: 1163-9. [CrossRef]
4. Chamberlain AM, Agarwal SK, Folsom AR, Soliman EZ, Chambless LE, Crow R, et al. A clinical risk score for atrial fibrillation in a biracial prospective cohort (from the Atherosclerosis Risk in Communities [ARIC] study). Am J Cardiol 2011; 107: 85-91. [CrossRef]
5. Benjamin EJ, Levy D, Vaziri SM, D'Agostino RB, Belanger AJ, Wolf PA. Independent risk factors for atrial fibrillation in a population-based cohort. The Framingham Heart Study. JAMA 1994; 271: 840-4. [CrossRef]
6. Escobedo LG, Zack MM. Comparison of sudden death and non sudden coronary deaths in the United States. Circulation 1996; 93: 2033-6. [CrossRef]
7. Li D, Fareh S, Leung TK, Nattel S. Promotion of atrial fibrillation by heart failure in dogs: electrical remodeling of a different sort. Circulation 1999; 100: 87–95. [CrossRef]
8. Wenzel DG, Stark LG. Effect of nicotine on cardiac necrosis induced by isoproterenol. Am Heart J 1966; 71: 368-370. [CrossRef]
9. Tuan TC, Chang SL, Tai CT, Lin YJ, Hu YF, Lo LW, et al. Impairment of the atrial substrates by chronic cigarette smoking in patients with atrial fibrillation. J Cardiovasc Electrophysiol 2008; 19: 259-65. [CrossRef]