

Atrial fibrillation in patients with atrial septal aneurysm

Marianna Janion and Jacek Kurzawski

Faculty of Health Sciences, Świętokrzyskie Cardiology Centre,
Provincial Complex of Hospital, Kielce, Poland

Abstract

Background: *To assess the incidence of paroxysmal atrial fibrillation (AF) in patients with atrial septal aneurysm (ASA) and the relationship between ASA morphology and the incidence of AF.*

Methods: *Among 12,941 patients evaluated echocardiographically, 88 (0.68%) were diagnosed with ASA [with 35 (39.8%) males and 53 (60.2%) females; mean age, 54.3 ± 14.4 years]. The morphology of the aneurysm and the atria was evaluated by echocardiography, P wave dispersion was evaluated by 12-lead electrocardiography (ECG) and the presence of AF was confirmed by 24-hour ambulatory ECG monitoring. ASA was diagnosed when the base of the aneurysm on echocardiography exceeded 15 mm and its protrusion exceeded 7.5 mm.*

Results: *Paroxysmal AF was documented in 15 (17.0%) patients with ASA. We showed that the presence of AF depended on the area of the aneurysm, with the latter positively correlating with the area of the left atrium. We further found a significant relationship between the presence of atrial arrhythmia and the dispersion of P wave duration ($p < 0.005$). The remaining characteristics, such as gender and age, embolic events, interatrial shunt and comorbidities showed no correlation with the occurrence of AF.*

Conclusions: *The occurrence of paroxysmal AF in patients with ASA depends on the area of the aneurysm and the increased area of the left atrium. Patients with ASA and paroxysmal AF display a significantly higher dispersion of sinus P wave duration versus patients with ASA but without paroxysmal AF. The presence of ASA structure oscillation, the direction of aneurysmal protrusion and the presence of interatrial shunt do not significantly affect the incidence of AF. (Cardiol J 2007; 14: 580–584)*

Key words: atrial septal aneurysm, atrial fibrillation

Introduction

In 1934 Lang and Posselt [1] published the first report of atrial septal aneurysm (ASA). Currently, given the much better imaging techniques, the

incidence of ASA, depending on the imaging method, is 0.2–4.0% on transthoracic echocardiography (TTE) and 2.0–8.0% on transoesophageal echocardiography (TOE) [2–5]. The presence of an additional structure within the interatrial septum may affect the duration of atrial wave spread [6]. Furthermore, the diagnosis of such an abnormality within the interatrial septum is quite often associated with the presence of interatrial shunt, which may in turn contribute to the occurrence of crossed embolism [7]. These phenomena may frequently be associated

Address for correspondence: Marianna Janion
Grenadierów 44, 25–212 Kielce, Poland
Tel: +48 41 362 17 17; e-mail: mjanion@interia.pl
Received: 20.04.2007 Accepted: 20.09.2007

with the presence of atrial fibrillation (AF) and/or flutter [8]. It may therefore be interesting to evaluate the presence of atrial arrhythmias in patients with ASA.

The aim of the study was to evaluate the incidence of AF in patients with ASA and the relationship between the morphology of ASA and the incidence of AF.

Methods

Study group

Between 2002 and 2005 we examined 12,941 patients by TTE or TOE at the Świętokrzyskie Cardiology Centre in Kielce, Poland and found ASA in 88 (0.68%) patients. The study group comprised 35 (39.8%) men and 53 (60.2%) women. The mean age was 54.3 ± 14.4 years (men, 58.7 ± 12.6 years; women, 50.8 ± 14.5 years). The following co-morbidities were taken into consideration in the clinical characterisation of the patients: hypertension, diabetes mellitus, hyperlipidaemia, ischaemic heart disease.

Echocardiography

All the subjects were evaluated by echocardiography with the presentation of the interatrial septum in the longitudinal and transverse views. Atrial septal aneurysm was diagnosed when the base of the aneurysm exceeded 15 mm and the protrusion of its structure exceeded 7.5 mm [2, 9]. TTE was used to evaluate the presence of any interatrial shunt, left atrial size in the transverse dimension in the parasternal longitudinal view (M-mode) and the area of both atria calculated planimetrically in the apical four-chamber view (2D). All the subjects included in the study underwent TOE, which verified the measurements, and the direction of protrusion and the mobility of the aneurismal structure were evaluated.

Electrocardiography

The P wave duration and dispersion were measured using the method discussed in detail by Madias et al. [10]. For this purpose, on the day of inclusion in the study, two electrocardiograms were obtained at a 30-second interval with a simultaneous recording of 12 leads and an automatic measurement of the mean P wave duration (t_p [ms]), and its dispersion was determined as the absolute difference between the measured means. The diagnostic tests were performed in normokalaemia and without any other metabolic abnormalities (diabetic patients had a compensated glucose profile during

the study) which might affect the development of atrial arrhythmia.

Two 24-hour ambulatory ECGs were obtained in the study group at one month's interval to look for episodes of AF. Atrial fibrillation of more than 30 seconds' duration was considered to be an episode of AF.

Statistical analysis

Evaluation of the incidence of AF depending on such pre-existing qualitative traits as the gender, co-morbidities, embolic complications, direction of aneurismal wall protrusion, oscillation of the aneurismal wall or the interatrial shunt was performed using χ^2 test of independence.

The quantitative variables were presented as means \pm standard deviation. The occurrence of such qualitative variables as the age, area of the aneurysm, size of the left atrium, area of the left and right atria and the dispersion of P wave duration was evaluated using the t-Student test. In the case of a larger number of comparable traits, the F-Snedecor test was used. Correlation of quantitative traits was evaluated by the analysis of the correlation function and where significant correlation between the traits was found, a linear regression function was determined.

Results

During the 24-hour ambulatory ECG monitoring 15 (17.0%) patients were diagnosed with paroxysmal AF. Results of the comparison of patients with paroxysmal AF with patients without AF are summarised in Table 1.

In the group of patients with AF there were significantly more patients with a high ASA area, greater length of the left atrium in M-mode and a higher area of the left and right atria. A significant correlation was shown between the incidence of AF and aneurismal area ($p < 0.0001$; Snedecor test). The remaining traits (gender and age, embolic events, presence of the interatrial shunt, co-morbidities) did not correlate with the occurrence of AF. Due to the small number of patients with documented coronary artery disease we did not evaluate the relationship between the occurrence of AF and ischaemic heart disease.

We also found a significant relationship between the presence of AF and the dispersion of P wave duration ($p < 0.005$) (Table 1).

Because aneurismal area is significantly associated with the presence of AF and because there is a relationship between aneurismal area and left

Table 1. A comparison of the study patients with atrial septal aneurysm (ASA) and atrial fibrillation (AF) with those with ASA but without AF.

| Parameter | ASA and AF (n = 15) | ASA without AF (n = 73) | P value |
|---|---------------------|-------------------------|----------|
| Age (years) | 58.5 ± 14.4 | 53.4 ± 14.1 | NS |
| Female gender | 9 (60%) | 44 (60.3%) | NS |
| Co-morbidities | 2 (13.3%) | 7 (9.6) | NS |
| Embolic complications | 3 (20.0%) | 9 (12.3%) | NS |
| Direction of ASA protrusion into the right atrium | 9 (60%) | 43 (58.9%) | NS |
| Aneurismal oscillations | 8 (53.3%) | 37 (50.7%) | NS |
| Presence of interatrial shunt | 8 (53.3%) | 39 (53.4%) | NS |
| ASA size [cm ²] | 1.94 ± 0.35 | 1.44 ± 0.19 | < 0.0001 |
| Length of the left atrium (M-mode) [cm] | 4.51 ± 1.08 | 3.74 ± 0.57 | < 0.0001 |
| Area of the left atrium [cm ²] | 31.64 ± 11.37 | 21.47 ± 5.08 | < 0.0001 |
| Area of the right atrium [cm ²] | 27.21 ± 10.8 | 16.86 ± 4.69 | < 0.0001 |
| Dispersion of P wave duration [ms] | 13.6 ± 9.3 | 5.8 ± 5.2 | < 0.005 |

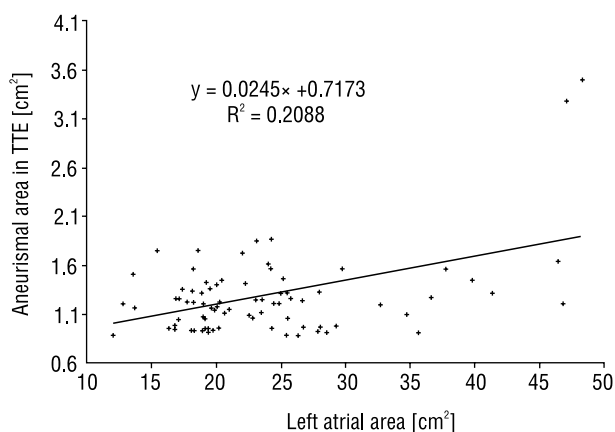


Figure 1. Correlation between the aneurismal area and the left atrial area.

atrial area (the size of the aneurysm positively correlates with the areas of the aneurism and the left atrium (the size of the aneurism positively correlates with the left atrial size, $p < 0.0001$, Fig. 1), it may be concluded that the presence of AF was significantly related to the left atrial area.

Discussion

Atrial fibrillation is one of the most common supraventricular arrhythmias. It usually starts from brief, often almost imperceptible paroxysms, which gradually develop into longer and eventually persistent atrial fibrillation. The paroxysmal pattern of this arrhythmia accounts for 30–70% of all AF cases depending on the population studied [11]. The exact incidence of paroxysmal AF is unknown. In the Framingham population, over 22 years of follow-up,

the incidence of this arrhythmia in the 55–65 age group (similar to the mean age of our patients) was found to be 3.79% [12, 13]. The presence of other cardiovascular diseases (valvular heart disease, ischaemic heart disease, hypertension, myocarditis etc.) may increase the incidence of both paroxysmal and persistent AF by 3–5 times, and the major factor responsible for this fact is left atrial enlargement. In each case, the presence of AF is associated with increased mortality, irrespective of pre-existing cardiovascular disease [13–15].

The electrophysiological mechanism responsible for AF is re-entry. As a result of the natural atrial non-homogeneity (pulmonary and systemic venous ostia) the stimulation wave spreading from the sinus node to the atrioventricular node undergoes fragmentation and the numerous factors (nervous factors, endocrine factors, atrial stretching etc.) increase the likelihood of unidirectional blocks and re-entrant stimulus. The development of AF is conditional upon the co-existence of at least 3 such re-entrant stimulation waves. The larger the number of such waves, the greater the propensity for the arrhythmia to become persistent. The presence of ASA increases the anatomical non-homogeneity of the atrium, which may affect the duration of dispersion of atrial stimulation wave and cause paroxysms of the arrhythmia [6].

Our results point to the presence of ASA in 0.68% of echocardiographically evaluated patients, which is consistent with estimates made by other authors (0.2–8.0%) [2–6]. As demonstrated, the presence of ASA may increase the likelihood of atrial arrhythmias. In our study, AF was present in 17% of ASA patients. In other studies, the incidence of

AF in ASA patients ranged from 0% to 23% [3, 8, 9, 16] and a considerable propensity to AF development was observed when artificial stimulation was used. In their experiment, Berthet et al. [17] invasively provoked AF and an AF episode of more than 60 seconds' duration was provoked in 66% of ASA patients and in 31% of patients the episode lasted from 5 minutes to 24 hours.

It is a well-known fact that large sizes of the atria contribute to the development of atrial arrhythmias. Our study confirmed the strongest relationship of the occurrence of AF principally in patients with considerably increased area of the left atrium.

According to the literature concerning the occurrence of AF, one of the predisposing factors is the dispersion of P wave duration appearing in patients with paroxysmal AF [18, 19]. The presence of dispersion may also be a predicting factor of recurrence of atrial fibrillation or flutter in patients undergoing conversion of the atrial arrhythmia [20, 21]. The reduced inflow into the left atrium in the course of atrial dysfunction may also affect the appearance of sinus P wave dispersion [19, 22]. The dispersion of sinus P wave duration that we measured in our study in the group with paroxysmal AF was significantly greater ($p < 0.005$) versus the group without the arrhythmia. Of note is the fact that the presence and magnitude of the dispersion were independent of the aneurismal and atrial sizes, which may indirectly indicate that the presence of ASA increases the risk of AF.

Some authors consider atrial fibrillation and flutter to be an independent risk factor for embolism. Schneider et al. [8] and Coskun et al. [6] found that patients with ASA and a history of embolism displayed a lower incidence of concomitant atrial arrhythmia, and that the arrhythmia was most commonly accompanied by another heart abnormality which might have been responsible for evoking the former. Because in our study the number of patients with ASA and AF as well as embolic events was small, we could not find the relationship between the presence of this arrhythmia and embolic complications.

When examining other morphological parameters of the aneurysm, such as the presence of interatrial shunt, septal oscillations and the direction of aneurismal protrusion, we found no relationship between their occurrence and the presence of atrial fibrillation.

Conclusions

1. The development of paroxysmal atrial fibrillation in patients with ASA depends on the area

of the aneurysm and the increased area of the left atrium.

2. In patients with ASA and paroxysmal atrial fibrillation there is a significantly higher dispersion of P wave duration versus patients with ASA but without AF.
3. The presence of oscillation of the ASA structure, direction of aneurismal protrusion and the possible presence of interatrial shunt do not significantly affect the incidence of atrial fibrillation.

References

1. Lang FJ, Posselt A. Aneurysmatische Vorwölbung der Fossa ovalis in den linken Vorhof. *Wien Med Wochenschr*, 1934; 84: 392–396.
2. Hanley PC, Tajik AJ, Hynes JK et al. Diagnosis and classification of atrial septal aneurysms by two-dimensional echocardiography: Report of 80 consecutive cases. *J Am Coll Cardiol*, 1985; 6: 1370–1383.
3. Marazanhof M, Roudaut R, Cohen A et al. Atrial septal aneurysm. Morphological characteristics in large population: pathological associations. A French multicenter study on 259 investigated by transesophageal echocardiography. *Int J Cardiol*, 1995; 52: 59–65.
4. Agmon Y, Khandheria BK, Meissner I et al. Frequency of atrial septal aneurysms in patients with cerebral ischaemic events. *Circulation*, 1999; 99: 1942–1944.
5. Ossemann M, Laloux P, Marchandise B, Jamart J. Association between stroke and atrial septal aneurysm assessed by transesophageal echocardiography in a cardiologic population. *Acta Neurol Bel*, 1995; 95: 170–177.
6. Coskun S, Sekuri C, Bayturan O. Diagnosis of asymptomatic atrial septal aneurysms using two dimensional color Doppler and contrast transthoracic echocardiography. *Chin Med J*, 2003; 116: 699–702.
7. Schneider B, Hanrath P, Vogel P, Meinertz T. Improved morphologic characterization of atrial septal aneurysm by transesophageal echocardiography: Relation to cerebrovascular events. *J Am Coll Cardiol*, 1990; 16: 1000–1009.
8. Schneider B, Hofmann T, Meinertz T. Is there an association of atrial septal aneurysm with arrhythmias? *Cardiology*, 1999; 91: 87–91.
9. Mügge A, Daniel GD, Angermann C et al. Atrial septal aneurysm in adult patients. *Circulation*, 1995; 91: 2785–2792.
10. Madias JE. P-wave duration and dispersion in patients with peripheral edema and its amelioration. *Ind Pacing Electrophysiol J*, 2007; 7: 7–18.

11. Davidson E, Weinberger J, Rotenberg Z et al. Atrial fibrillation. *Arch Intern Med*, 1989; 149: 457–459.
12. Wolf PA, Benjamin EJ, Belanger AJ et al. Secular trends in the prevalence of atrial fibrillation. The Framingham Study. *Am Heart J*, 1996; 131: 790–795.
13. Benjamin EJ, Wolf PA, Kannel WB, D'Agostino RB. Impact of atrial fibrillation on the risk of death — the Framingham Heart Study. *Circulation*, 1998; 98: 946–952.
14. PsatyBM, Manolio TA, Kuller LH. Incidence of and risk factors for atrial fibrillation in elder adults. *Circulation*, 1998; 96: 2455–2461.
15. Jouven X, Desnos M, Guerot C, Ducimetiere P. Idiopathic atrial fibrillation as a risk factor for mortality. *Eur Heart J*, 1999; 20: 896–899.
16. Olivares-Reyes A, Chan S, Lazar EJ et al. Atrial septal aneurysm: a new classification in two hundred five adults. *J Am Soc Echocardiogr*, 1997; 19: 644–656.
17. Berthet K, Lavergne T, Cohen A et al. Significant association of atrial vulnerability with atrial septal abnormalities in young patients with ischaemic stroke of unknown cause. *Stroke*, 2000; 31: 389–403.
18. Dogan A, Kahraman H, Ozturk M, Avsar A. P wave dispersion and left atrial appendage function for predicting recurrence after conversion of atrial fibrillation and relation of P wave dispersion to appendage function. *Echocardiography*, 2004; 21: 523–530.
19. Rosiak M, Bolinska H, Ruta J. P wave dispersion and P wave duration on SAECG in predicting atrial fibrillation in patients with acute myocardial infarction. *Ann Noninvasive Elctrocardiol*, 2002; 7: 363–368.
20. Yilmaz R, Demirbag R, Durmus I et al. Association of stage of left ventricular diastolic dysfunction with P wave dispersion and occurrence of atrial fibrillation after first acute anterior myocardial infarction. *Ann Noninvasive Electrocardiol*, 2004; 9: 330–338.
21. Soyulu M, Demir AD, Ozdemir O et al. Increased P wave dispersion after the radiofrequency catheter ablation in overt pre-excitation patients: the role of atrial vulnerability. *Int J Cardiol*, 2004; 95: 167–170.
22. Celik T, Iyisoy A, Kurasaklioglu H et al. Effects of primary percutaneous coronary intervention on P wave dispersion. *Ann Noninvasive Electrocardiol*, 2005; 10: 342–347.