

## University of Groningen

### Ablation of atrial fibrillation

de Maat, Gijs Eduard

**IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.**

*Document Version*

Publisher's PDF, also known as Version of record

*Publication date:*

2018

[Link to publication in University of Groningen/UMCG research database](#)

*Citation for published version (APA):*

de Maat, G. E. (2018). *Ablation of atrial fibrillation: Moving to a heart team approach*. Rijksuniversiteit Groningen.

**Copyright**

Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

**Take-down policy**

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

*Downloaded from the University of Groningen/UMCG research database (Pure): <http://www.rug.nl/research/portal>. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.*

## Chapter 3

# **Relation of overweight and symptomatic atrial fibrillation: a case-report**

G.E. de Maat, H.Z.R. Gerds-Ploeger, M.A. Mariani, I.C. Van Gelder, J. Brügemann,  
M. Rienstra

*Heart Rhythm Case Rep 2015 1(5):342-344*

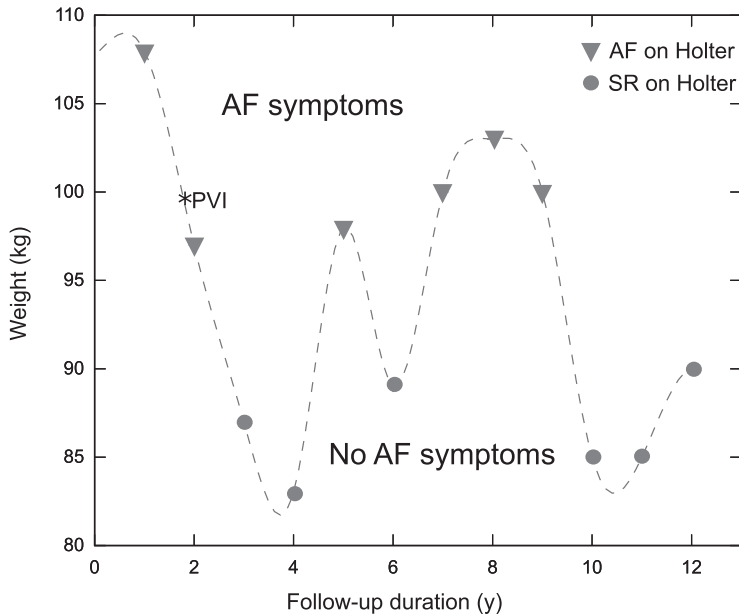


## INTRODUCTION

Over the past few decades, obesity has become a global epidemic and represents a major challenge for current and future health(1). Although the exact pathogenesis of AF is not completely understood, there is compelling evidence that obesity increases the risk for new-onset and recurrences of AF and increases progression to more persistent forms of AF(2). Weight reduction in patients with AF, reduces the burden and number of AF-episodes and cumulative AF duration(3). These findings support therapy directed at reduction of weight and controlling risk factors in the treatment of AF. We herein present the case of a 46-year-old male patient with symptomatic AF and a temporal relation between weight changes and recurrences of AF.

## CASE

A 46-year-old Caucasian male with a history of hypertension and 10 years paroxysmal AF was referred to our center. The patient had failed on a class IC and III anti-arrhythmic drugs, and suffered from a progressive frequency and severity of palpitations and fatigue (EHRA-score=3). The calculated CHA<sub>2</sub>DS<sub>2</sub>-VASc score was 1 (hypertension). The patient's body mass index was 28.3 kg/m<sup>2</sup> (97 kg, 185cm) at time of referral. In the work-up for PVI, transthoracic echocardiography showed normal cardiac function, and a left atrial volume indexed of 29.1mL/m<sup>2</sup>. The transcatheter PVI using radiofrequency energy was performed. After the PVI, the patient lost 10kg with the help of our institutional patient tailored 3-month cardiac rehabilitation program which constituted of low-intensity exercise guided by a physiotherapist, a balanced diet supported by our institutional nutritionist and nutritional psycho-education provided by the psychologist. Thereafter, the patient was free of atrial arrhythmias during the first 6 months. In the next 6 months the patient lost another 4kg, arriving at 83kg. The variance in patients weight over time is depicted in **Figure 1**. Two years after the first PVI, the patient regained weight up to 98kg and experienced a symptomatic recurrence of paroxysmal AF, which was confirmed by 24hour Holter monitoring. A second electrophysiological examination and PVI was scheduled, but with physical exercise the patient managed to lose weight up to 89kg. From that point he was asymptomatic and in sinus rhythm. The re-PVI was therefore not performed. The patient was in sinus rhythm for over 1 year, until he regained weight up to 100kg. This time, the recurrent AF episode was classified as persistent, which was confirmed by 24hour Holter monitoring. The patient underwent elective electrocardioversion and flecainide was restarted. Another year later, weighing 103 kg, the patient remained having severely symptomatic AF, with an AF-burden on 24 hour Holter monitoring of 39%. The patient was again referred to our cardiac rehabilitation facility, the patient lost 15 kg again and

**Figure 1.** Association between weight and heart rhythm

symptomatic AF disappeared. Since then, neither AF nor other atrial arrhythmias were seen on 24 hour Holter monitoring.

## DISCUSSION

Obesity is associated with multiple cardiovascular risk factors, e.g. hypertension, dyslipidemia, insulin resistance, obstructive sleep apnea syndrome, pericardial fat deposition, and a systemic inflammatory state(4). After adjustment for other risk factors, obesity and is associated with an increased risk of cardiovascular diseases, such as ischemic heart diseases, heart failure, and AF (5). There is abundant evidence for the involvement of obesity in the development of AF. Obese individuals have up to 2.4-fold increased risk for new-onset AF(6). The dynamic association of weight with AF prevalence was previously presented in the Women's Health Study(7). Mechanisms underlying the relation between obesity and new-onset AF, may relate to structural remodeling caused by elevated end-diastolic pressure, inflammation, and increased plasma volume(8,9). Also, obesity is known to be associated with sleep apnoe syndrome, an independent risk factor for AF(10).

Our case underscores the direct (12-year) temporal relation between weight and symptomatic AF. In the particular case when weight was > 95kg, the patient suffered from symptomatic AF recurrences. All 24-hour ECG registrations performed, in total 360 hours,

are depicted in **Figure 1**. After testing, no obesity-associated comorbidities like diabetes, metabolic syndrome and sleep apnea syndrome appeared to be present in our case. The finding that obesity itself may also induce AF or increase AF burden has been reported in previous studies(11). It is unknown if this risk factor is attributable only to body composition or also the level of physical activity(12). Also, the role of epicardial fat remains to be thoroughly investigated as a risk factor(13). It is important to recognize that obesity is a modifiable risk factor, although its management can be very challenging in the clinical practice. A recent randomized trial compared intervention with active weight management to general lifestyle advice. Results of this pivotal paper show that weight reduction with intensive risk factor management causes a significant reduction in AF symptom burden and severity(3). The recent ARREST-AF trial showed that aggressive risk factor improves long-term outcomes of AF ablation(14). Furthermore, if this weight loss is sustained at long-term follow-up, reduction of AF burden and maintenance of sinus rhythm are significantly higher compared to patients with weight fluctuation(15). In fact, our case nicely illustrates the relation of overweight and recurrence of symptomatic AF, and stresses the importance of weight counseling in patients referred for symptomatic AF, especially before considering invasive treatment modalities such as transcatheter or surgical pulmonary vein isolation (PVI). A cardiac rehabilitation program is an option for such patients.

## CONCLUSIONS

Weight reduction and life style management is important in the treatment of symptomatic AF and warrants more attention.

## REFERENCES

1. Finucane MM, Stevens GA, Cowan MJ, et al. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 91 million participants. *Lancet*. 2011;377:557–67.
2. Tsang, TS, Barnes ME, Miyasaka Y, Cha SS, Bailey KR, Verzosa GC, Seward JB, Gersh BJ. Obesity as a risk factor for the progression of paroxysmal to permanent atrial fibrillation: a longitudinal cohort study of 21 years. *Eur. Heart J*. 2008;29:2227-2233.
3. Abed, HS, Wittert GA, Leong DP, et al. Effect of weight reduction and cardiometabolic risk factor management on symptom burden and severity in patients with atrial fibrillation: a randomized clinical trial. *JAMA*. 2013;310: 2050-2060.
4. Asghar O, Alam U, Hayat SA, Aghamohammadzadeh R, Heagerty AM, Malik RA. Obesity, diabetes and atrial fibrillation; epidemiology, mechanisms and interventions. *Curr Cardiol Rev*. 2012;8: 253–64.
5. Rimm EB, Stampfer MJ, Giovannucci E, Ascherio A, Spiegelman D, Colditz GA, Willett WC. Body size and fat distribution as predictors of coronary heart disease among middle-aged and older US men. *Am. J. Epidemiol*. 1995;141, 1117-1127.
6. Frost L, Hune LJ, Vestergaard P. Overweight and obesity as risk factors for atrial fibrillation or flutter: the Danish Diet, Cancer, and Health Study. *Am. J. Med*. 2005; 118, 489-495.
7. Tedrow UB, Conen D, Ridker PM, Cook NK, Koplan BA, Mason JE, Buring JE, Albert CM. The long- and short-term impact of elevated body mass index on the risk of new atrial fibrillation, the WHS (women’s health study). *J Am Coll Cardiol* 2010;25;55(21):2319-27
8. Schoonderwoerd BA, Smit MD, Pen L, Van Gelder IC. New risk factors for atrial fibrillation: Causes of ‘not-so-lone atrial fibrillation’. *Europace*. 2008;10:668-73.
9. Lin YK, Chen YJ, Chen SA. Potential atrial arrhythmogenicity of adipocytes: Implications for the genesis of atrial fibrillation. *Med Hypotheses*. 2010;74:1026-9.
10. Steveson IH, Teichtahl H, Cunningham D, Ciavarella S, Gordon I, Kalman JM. Prevalence of sleep disordered breathing in paroxysmal and persistent atrial fibrillation patients with normal left ventricular function. *Eur Heart J* 2008;13:1662-9
11. Hatem SN, Sanders P. Epicardial adipose tissue and atrial fibrillation. *Cardiovasc Res*. 2014;102: 205-213.
12. Wyse DG, Van Gelder IC, Ellinor PT, Go AS, Kalman JM, Narayanan SM, Nattel S, Schotten U, Rienstra M. Lone atrial fibrillation: does it exist? *J Am Coll Cardiol* 2014;17:1715-23
13. Al Chekakie MO, Welles CC, Metoyer R, Ibrahim A, Shapira AR, Cytron J, Santucci P, Wilber DJ, Akar JG. Pericardial fat is independently associated with human atrial fibrillation. *J Am Coll Cardiol* 2010; 31:56(10):784-8
14. Pathak RK, Middeldorp ME, Lau DH, et al. Aggressive risk factor reduction study for atrial fibrillation and implications for the outcome of ablation: the ARREST-AF cohort study. *J Am Coll Cardiol* 2014; 64(21):2222-31
15. Pathak RK, Middeldorp ME, Meredith M, Mehta AB, Mahajan R, Wong CX, Twomey D, Elliot AD, Kalman JM, Abhayaratna WP, Lau P, Sanders P. Long-term effect of goal directed weight management in an atrial fibrillation cohort: a long-term follow-up study (LEGACY Study). *J Am Coll Cardiol* 2015; pii:S0735-1097(15):00761-5.