

University of Groningen

## Relation of overweight and symptomatic atrial fibrillation

de Maat, Gijs E.; Gerds-Ploeger, Hendyke Z.R.; Mariani, Massimo A.; Van Gelder, Isabelle C.; Brügemann, Johan; Rienstra, Michiel

*Published in:*  
HeartRhythm case reports

*DOI:*  
[10.1016/j.hrcr.2015.06.002](https://doi.org/10.1016/j.hrcr.2015.06.002)

**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:*  
2015

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

*Citation for published version (APA):*

de Maat, G. E., Gerds-Ploeger, H. Z. R., Mariani, M. A., Van Gelder, I. C., Brügemann, J., & Rienstra, M. (2015). Relation of overweight and symptomatic atrial fibrillation: A case report. *HeartRhythm case reports*, 1(5), 342-344. <https://doi.org/10.1016/j.hrcr.2015.06.002>

### 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).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: <https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment>.

### 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.*

# Relation of overweight and symptomatic atrial fibrillation: A case report



Gijs E. de Maat, MD, Hendyke Z.R. Gerds-Ploeger, NP, Massimo A. Mariani, MD, PhD, Isabelle C. Van Gelder, MD, PhD, Johan Brügemann, MD, PhD, Michiel Rienstra, MD, PhD

*From the Department of Cardiology and Cardiothoracic Surgery, University Medical Centre Groningen, Groningen, The Netherlands.*

## Introduction

Over the past few decades, obesity has become a global epidemic and represents a major challenge for current and future health.<sup>1</sup> Although the pathogenesis of atrial fibrillation (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.<sup>2</sup> Weight reduction in patients with AF reduces the burden and number of AF-episodes and cumulative AF duration.<sup>3</sup> These findings support therapy directed at reducing weight and controlling risk factors in the treatment of AF. We 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 report

A 46-year-old Caucasian male patient with a history of hypertension and 10 years paroxysmal AF was referred to our center. Treatment with class IC and III antiarrhythmic drugs had failed, and the patient experienced progressive frequency and severity of palpitations and fatigue (European Heart Rhythm Association score = 3). The calculated CHA<sub>2</sub>DS<sub>2</sub>-VASc (acronym for congestive heart failure, hypertension, age  $\geq 75$  y [double points], diabetes mellitus, prior stroke or thromboembolism [double points], vascular disease, age 65–74 years, sex category) score was 1 (hypertension). The patient's body mass index was 28.3 kg/m<sup>2</sup> (97 kg, 185 cm) at time of referral. In the workup for pulmonary vein isolation (PVI), transthoracic echocardiography results showed normal cardiac function, and a left atrial volume indexed of 29.1 mL/m<sup>2</sup>. The transcatheter PVI using radio-frequency energy was performed. After the PVI, the patient

lost 10 kg with the help of our institutional patient-tailored 3-month cardiac rehabilitation program, which constituted low-intensity exercise guided by a physiotherapist, a balanced diet supported by our institutional nutritionist, and nutritional psychoeducation provided by a psychologist. Thereafter, the patient was free of atrial arrhythmias during the first 6 months. In the next 6 months, the patient lost another 4 kg, arriving at 83 kg. The variance in the patient's weight over time is depicted in Figure 1. Two years after the first PVI, the patient regained weight up to 98 kg and experienced a symptomatic recurrence of paroxysmal AF, which was confirmed by 24-hour Holter monitoring. A second electrophysiological examination and PVI were scheduled, but with physical exercise the patient managed to lose weight to 89 kg. 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 a total of 100 kg. This time, the recurrent AF episode was classified as persistent, which was confirmed by 24-hour Holter monitoring. The patient underwent elective electrocardioversion, and flecainide was restarted. Another year later, weighing 103 kg, the patient continued to have 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 symptomatic AF disappeared. Since then, neither AF nor other atrial arrhythmias have been seen on 24-hour Holter monitoring.

## Discussion

Obesity is associated with multiple cardiovascular risk factors, for example, hypertension, dyslipidemia, insulin resistance, obstructive sleep apnea syndrome, pericardial fat deposition, and a systemic inflammatory state.<sup>4</sup> 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.<sup>5</sup> 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.<sup>6</sup> The dynamic association

**KEYWORDS** Atrial fibrillation; Obesity; Risk factors; Pulmonary vein isolation  
**ABBREVIATIONS** AF = atrial fibrillation; PVI = pulmonary vein isolation; SR = sinus rhythm (Heart Rhythm Case Reports 2015;1:342–344)

Conflict of interest: All authors state that there are no conflicts of interest.  
**Address reprint requests and correspondence:** Dr Isabelle C Van Gelder, Department of Cardiology, Thoraxcenter, University of Groningen, University Medical Center Groningen, P.O. Box 30.001, 9700 RB Groningen, The Netherlands. E-mail address: i.c.van.gelder@umcg.nl.

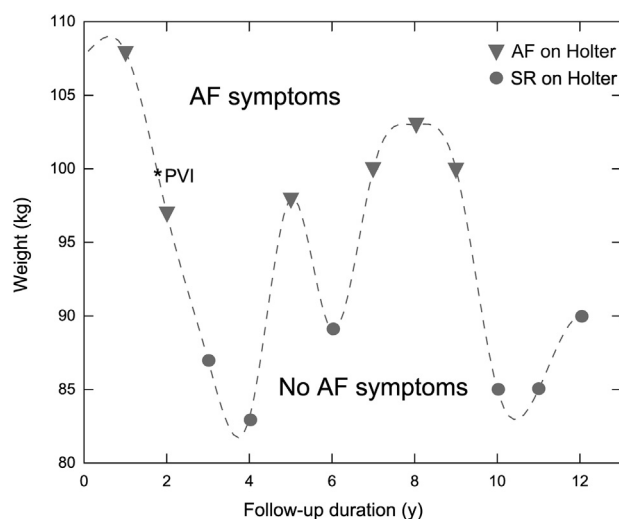
## KEY TEACHING POINTS

- Weight reduction and lifestyle management are important in the treatment of symptomatic atrial fibrillation.
- Strict weight management using a cardiac rehabilitation program is a treatment option for obese patients before invasive treatment modalities are deployed.

of weight with AF prevalence was previously presented in the Women's Health Study.<sup>7</sup> 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.<sup>8,9</sup> Also, obesity is known to be associated with sleep apnea syndrome, an independent risk factor for AF.<sup>10</sup>

Our case underscores the direct (12-year) temporal relation between weight and symptomatic AF. In this particular case, when weight was >95 kg, the patient experienced symptomatic AF recurrences. All 24-hour electrocardiogram registrations performed, in total 360 hours' worth, are depicted in Figure 1. After testing, no obesity-associated comorbidities, such as 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.<sup>11</sup> It is unknown whether this risk factor is attributable only to body composition or also to the level of physical activity.<sup>12</sup> Also, the role of epicardial fat remains to be thoroughly investigated as a risk factor.<sup>13</sup> Obesity is a modifiable risk factor, although its management can be very challenging in clinical practice. A recent randomized trial compared intervention with active weight management to modification with general lifestyle advice. Results of this pivotal paper show



**Figure 1** Association between weight and heart rhythm. AF = atrial fibrillation; PVI = pulmonary vein isolation; SR = sinus rhythm.

that weight reduction with intensive risk-factor management causes a significant reduction in AF symptom burden and severity.<sup>3</sup> The recent ARREST-AF trial showed that aggressive risk-factor management improves long-term outcomes of AF ablation.<sup>14</sup> 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 those in patients with weight fluctuation.<sup>15</sup> In fact, our case nicely illustrates the relation of overweight and recurrence of symptomatic AF, and it stresses the importance of weight counseling in patients referred for symptomatic AF, especially before considering invasive treatment modalities such as transcatheter or surgical PVI. A cardiac rehabilitation program is an option for such patients.

## Conclusions

Weight reduction and lifestyle management are important in the treatment of symptomatic AF and warrant more attention.

## Acknowledgments

We thank Dr. Bastiaan Geelhoed for his contribution to the figure.

## References

1. Finucane MM, Stevens GA, Cowan MJ, et al; Global Burden of Metabolic Risk Factors of Chronic Diseases Collaborating Group. (Body Mass Index). National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiologic studies with 960 country-years and 91 million participants. *Lancet* 2011;377:557–567.
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;8:253–264.
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):2319–2327.
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;10:668–673.
9. Lin YK, Chen YJ, Chen SA. Potential atrial arrhythmogenicity of adipocytes: Implications for the genesis of atrial fibrillation. *Med Hypotheses* 2010;74:1026–1029.
10. Steveson IH, Teichtahl H, Cunnington 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;13:1662–1669.
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;17:1715–1723.
13. Al Chekatie 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):784–788.
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;64:2222–2231.
  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;65:2159–2169.