REVIEW ARTICLE

Obstructive sleep apnea, atrial fibrillation, and erectile dysfunction: are they only coexisting conditions or a new clinical syndrome? The concept of the OSAFED syndrome

Filip M. Szymański, Bartosz Puchalski, Krzysztof J. Filipiak

Department of Cardiology, Medical University of Warsaw, Warszawa, Poland

KEY WORDS

atrial fibrillation, erectile dysfunction, OSAFED, obstructive sleep apnea

ABSTRACT

Patients rarely suffer from only 1 disease. Most of them have several conditions with common risk factors and etiology, and which often increase the severity of each other. The phenotypes linked to 1 condition are often linked to many others. We describe 3 patients with obstructive sleep apnea (OSA), atrial fibrillation (AF), and erectile dysfunction (ED), all of which are highly prevalent in the general population. OSA is one of the most common sleep disorders, affecting approximately 24% of men and 9% of women between 30 and 60 years of age. AF is one of the most common arrhythmias, present in approximately 2% of the population, and erectile dysfunction can be found in 18% to 40% of the male population older than 20 years. The presence of these 3 conditions in the same patient may be not only a coincidence but rather a new clinical syndrome. We present data which allow one to consider OSA, AF, and ED as parts of a clinical syndrome: OSAFED (obstructive sleep apnea, atrial fibrillation, and erectile dysfunction), with a larger effect on the cardiovascular risk profile than those 3 conditions taken alone. Introducing the OSAFED acronym into everyday clinical practice would have the tremendous advantage of reminding health care workers to screen every patient with either OSA, AF, or ED for the remaining 2 diseases. This would result in an early diagnosis and break the vicious circle of mutual disease exacerbation.

Correspondence to:
Filip M. Szymanski, MD, PhD, I Katedra i Klinika Kardiologii, Warszawski Uniwersytet Medyczny, ul. Banacha 1A, 02-097 Warszawa, Poland, phone: +48-22-599-19-58, fax: +48-22-599-19-57, e-mail: filip.szymanski@wum.edu.pl
Received: August 26, 2013.
Revision accepted: September 25, 2013.
Published online: September 26, 2013.
Conflict of interest: none declared.
Pol Arch Med Wewn. 2013; 123 (12): 701-707

Clinical scenarios A 55-year-old man with diabetes and hypertension was admitted for the ablation of a source of AF. He had been diagnosed with paroxysmal AF 4 years before. His AF attacks caused palpitations, reduced exercise capacity, and breathlessness, and flared as often as once every 3 to 6 months. The symptoms were classified as being European Heart Rhythm Association (EHRA) class III. The patient said that, apart from arterial hypertension and diabetes, he was otherwise healthy. He was taking warfarin, metformin 500 mg bid, ramipril 5 mg od, with good glucose and blood pressure control. AF was recorded both on standard electrocardiogram (ECG) and during 24-hour ambulatory ECG monitoring. He was admitted 1 day prior to the procedure. During the night, other patients complained that he snored very loudly and had episodes of apnea. When he was interviewed, he reported that his wife often complained about his loud snoring and that he experienced daytime sleepiness. He also said that he had been having erectile problems for about 2 years. We decided to proceed with the ablation and scheduled polysomnography after the procedure, and he was referred for further consultation. After performing a transesophageal echocardiography (which did not reveal any thrombi in the left atrial appendage, but showed an enlargement of the left atrium, confirmed also later with transthoracic echocardiography with the following results: left atrium, 5.9×6.8 cm; area, 33.7 cm^2; volume 135 cm^3); the pulmonary vein was isolated. Polysomnography showed an apnea-hypopnea index (AHI) of 43.8/h and a mean night-time blood oxygen saturation of 91.3%. After the diagnosis of severe

FIGURE 1 Example of polysomnography results of a patient with severe obstructive sleep apnea Abbreviations: bpm – beats per minute, SpO_2 – oxygen saturation

OSA, the patient was introduced to continuous positive airway pressure (CPAP) therapy, which, after 4 months, he is still tolerating well. He also reports an improvement in sexual function and does not show any signs of AF, which was also undetected on follow-up 24-hour ambulatory ECG.

The second patient, a 61-year-old man, was admitted because of excessive fatigue and sleepiness, reduced exercise capacity, dyspnea, and loud, irregular snoring. He also reported feeling an irregular heartbeat and noticing elevated blood pressure values during home blood pressure monitoring, with nocturnal hypertension spikes and morning headaches. A physical examination revealed an arterial blood pressure of 150/100 mmHg, lower limb edema, an irregular heart rhythm, and a body mass index (BMI) of 35.7 kg/m^2. He underwent 24-hour ECG monitoring, which demonstrated permanent AF and pauses of up to 2.5 s. Polysomnography was performed, as the patient was classified as being in high risk for OSA (obesity, poor blood pressure control, loud snoring, and excessive sleepiness), and it revealed severe OSA with an AHI of 31.6/h and a mean night-time blood oxygen saturation of 90.9%. He was referred to CPAP therapy, and was prescribed perindopril 10 mg od, metoprolol 25 mg od, eplerenone 25 mg od, acenocoumarol and pantoprazole 20 mg od. After 3 months of CPAP therapy his AHI was reduced to 5.1/h, and we managed to obtain good blood pressure control. He was doing well and reported no complaints.[1] During one of the follow-up visits he mentioned erectile dysfunction (ED). Further investigation revealed that he had been having problems with erections for over 5 years but never spoke about them with his physician, as he linked them to his age and medications. Unfortunately, during his hospitalization he was not investigated for ED.

The next patient was an obese 57-year-old man referred to the cardiologist for erectile dysfunction. He complained of severe difficulties, for the past 6 months, in achieving and maintaining erections. Further examination revealed that he was also snoring loudly and irregularly. On a physical examination, we observed obesity (BMI, 40.2 kg/m^2), a short neck with a large circumference (47 cm), an arterial blood pressure of 148/95 mmHg, and, upon auscultation, an irregular heart rhythm. He never experienced heart palpitations or any other signs of AF. We observed AF during the ECG, which was further confirmed with 24-hour ambulatory ECG monitoring (5 h/day of AF). During polysomnography, we observed an AHI of 11.4/h and a mean night-time blood oxygen saturation of 94.6%. He was diagnosed with mild OSA, instructed to reduce his body mass and refrain from sleeping on his back, and given advice on the use of sleep medication and to avoid drinking alcohol before sleeping. ED drugs were also recommended.

Currently, as cardiologists in an aging society, we scarcely meet patients with only 1 disease. Most of our patients suffer from several conditions with shared risk factors, etiology, and which often increase the severity of each other. Phenotypes that are linked to 1 condition are often linked to many others.

We describe here 3 clinical conditions: obstructive sleep apnea (OSA), atrial fibrillation (AF), and erectile dysfunction, which are often not coincidentally overlapping. Perhaps these findings describe a new clinical syndrome. We present

data which allow one to consider OSA, AF, and ED as one clinical syndrome: OSAFED (obstructive sleep apnea, atrial fibrillation, and erectile dysfunction), which may have a bigger impact on the cardiovascular risk profile than those 3 conditions taken separately.

Obstructive sleep apnea OSA is one of the most common types of sleep disorder. Breathing disorders during sleep affect approximately 24% of men and 9% of women between the age of 30 and 60 years.[2] OSA is defined as a repetitive interruption of ventilation during sleep caused by the collapse of the pharyngeal part of the airway. Pharyngeal airway collapse may be due to muscle weakness, bone and soft tissue abnormalities such as enlarged tonsils or adenoids, or fat tissue deposits around the neck, which narrow the pharyngeal lumen. The most typical patient presenting with OSA is an obese, middle-aged man with daytime somnolence. Further investigation often reveals hypertension, sometimes in its resistant form, cardiac arrhythmias, nocturnal angina, and loud snoring. Unfortunately, OSA is a condition often forgotten in everyday clinical practice and deserves special consideration.[3]

Because of the anatomical and physiological mechanisms of OSA, its prevalence rises with age and body mass. OSA is definitely associated with weight. The prevalence of OSA has been proven to rise 4-fold with each standard deviation of the body mass index (BMI).[2] In morbidly obese patients, with a BMI of ≥60 kg/m^2, the prevalence of OSA is over 90%.[4] The risk of developing OSA is increased by weight gain. A 10% weight gain is associated with a 32% increase in the AHI and causes a 6-fold increase in the risk for developing moderate-to-severe OSA.[5] Furthermore, not only is obesity a risk factor for OSA, but also OSA represents a risk factor for obesity. Patients with OSA have reduced physical activity, exercise performance, energy metabolism, and motivation along with excessive daytime sleepiness.[6] Reduced physical activity is associated with a reduction in inflammation marker levels, and this reduction, without an alteration in caloric intake, promotes weight gain.[7] Conversely, weight loss alleviates the severity of OSA. Each 10% reduction in body mass reduces the AHI by 26%.[5] However, even successful weight loss after bariatric surgery does not fully cure OSA, which suggests that other mechanisms, besides fat tissue accumulation, are responsible for both conditions.[8] In patients with OSA, alterations in the vascular system occur more often than in the general population. Ongoing inflammation, sympathetic activation, and oxidative stress all contribute to the development of endothelial dysfunction, which is responsible, for instance, for atherosclerosis.[9] Endothelial function is impaired in OSA patients, but can be improved by CPAP therapy.[10] OSA is also associated with an increased inflammatory response and platelet activation and aggregation and, therefore, a more active coagulation.[11,12] The close association between OSA, obesity, and endothelial dysfunction obscures the specific effects on cardiovascular risk of each condition or their synergistic effect.

OSA is important in initiating the development of cardiac and vascular disease, and in patients with established cardiovascular disease, it accelerates disease progression. Its treatment can result in clinical improvement and a reduction in cardiovascular event rate and mortality. The conditions most often associated with OSA include: hypertension, heart failure, coronary artery disease, myocardial infarction, stroke, arrhythmias, and pulmonary arterial hypertension.[9,13]

Atrial fibrillation AF is one of the most common arrhythmias. AF affects approximately 2% of the population, and its prevalence increases with age. This arrhythmia affects 0.5% of people aged from 40 to 50 years, and 5% to 15% of those aged 80 years.[14,15] Because of a better life expectancy, the number of patients presenting with AF is greater every year.[16] Body mass correlates with AF. About one fourth of AF patients is obese, and the mean BMI of this population is 27.5 kg/m^2, which is classified as overweight.[17]

This arrhythmia is very important in clinical practice. Not only does it decrease the quality of life, but also it has a large financial burden on society. Hospital stays, procedure and medication costs, together with the costs of absences from work represent significant expenditure.[18] It is also important to note that AF markedly affects general health and increases cardiovascular risk, doubling the risk of death.[19] This arrhythmia also affects the risk for thromboembolic events. It is estimated that 1 in 5 strokes is caused by AF.[19] This includes ischemic and hemorrhagic strokes as well as intracerebral bleeding, regardless of their severity – from those that cause no persistent impairment to those that result in long-term disability or death. AF also is responsible for a large fraction of hospitalizations. The increased activation of the coagulation process contributes to systemic embolic events. Low-grade inflammation and endothelial dysfunction, both present in AF patients, have the same effect.[20] It is estimated that AF accounts for one-third of hospital admissions for cardiac arrhythmias.[16] AF aggravates the course of acute coronary syndromes, heart failure, and ventricular dysfunction.

Cognitive dysfunction and dementia often affect AF patients. It is suggested that this is caused by asymptomatic cerebral embolic events in the absence of an overt stroke.[21] AF-related symptoms, risk of death, frequent hospitalizations, and poor general health quality contribute negatively to the quality of life. Moreover, patients with AF have reduced exercise capacity and are more likely to suffer from depression and impaired sleep quality, all of which are risk factors for cardiovascular disease.[22]

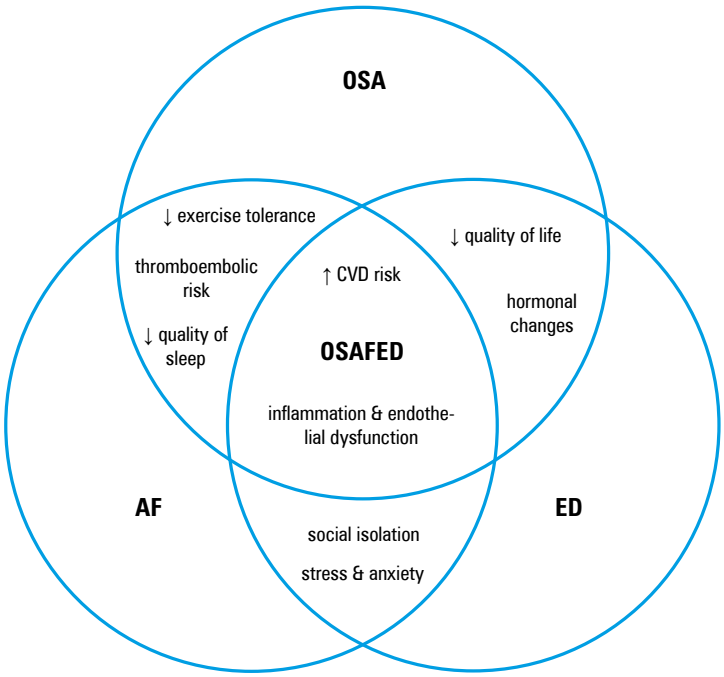

FIGURE 2 Clinical findings in obstructive sleep apnea, atrial fibrillation, and erectile dysfunction (OSAFED) Abbreviations: AF – atrial fibrillation, CVD – cardiovascular disease, ED – erectile dysfunction, OSA – obstructive sleep apnea

Erectile dysfunction ED is the inability to achieve or maintain an erection long enough to engage in sexual intercourse. This affects approximately from 18% to 40% of men older than 20 years.[23,24] In Poland, ED affects about 11% of men between the ages of 50 and 59 years, 4% between the ages of 30 and 49 years, and 3% between the ages of 18 and 24 years.[25] The prevalence of the condition increases with age and depends on many health-linked and psychosocial factors. Conditions that are associated with ED are diabetes mellitus, hypertension, coronary artery disease, obesity, difficult micturition, low socioeconomic status, a sedentary life style, smoking, depression, subjectively reported premature ejaculation, low libido, and irregular coitus.[24,26] The lowest prevalence of ED is noted in men who have no chronic medical conditions and follow healthy lifestyles. ED can be perceived as both a risk factor and a clinical manifestation of progressive atherosclerosis.[27]

ED is common in obese patients, even if they are young. It was shown that, among of men aged 20 to 45 years, obesity results in an odds ratio of 2.74 (95% confidence interval [CI], 1.1–6.8) for ED.[28] Obese men, independently of age, report less sexual desire (OR 1.48; CI 1.14–1.90).[29] However, approximately one-third of obese men with ED can regain sexual functions after 2 years of a healthy lifestyle, for instance, the Mediterranean diet and regular exercise.[30] In hypertensive patients with poor arterial blood pressure control ED occurs more often than in patients with well-controlled arterial blood pressure. Diabetes – alone or in the presence of hypertension – increases the odds of ED.[31]

ED is not only important in terms of sexual life, it is also a significant marker of future adverse cardiac events. Cardiovascular disease and ED share risk factors, including age, hypertension, dyslipidemia, smoking, obesity, and diabetes.[32] Patients with ED have a higher risk of cardiovascular disease (RR 1.48; 95% CI 1.25–1.74), coronary artery disease (RR 1.46; 95% CI 1.31–1.63), stroke (RR 1.35; 95% CI 1.19–1.54) and all-cause mortality (RR 1.19; 95% CI 1.05–1.34).[30] ED usually precedes the onset of coronary artery disease symptoms by 2 to 3 years and the occurrence of adverse cardiovascular events by 3 to 5 years.[33] ED is also a very important problem in patients with developed cardiovascular disease. In men, after myocardial infarction, both ED and anxiety associated with sexual activity are highly prevalent.[34-36]

The association between ED and cardiovascular disease, especially coronary artery disease, is thought to be found in their common risk factors and in the state of the systemic blood vessels and in ongoing low grade inflammation. Patients with ED and cardiovascular disease tend to have endothelial dysfunction expressed by elevated levels of biomarkers and shown by the results of the physical examination.[37] Increased circulating levels of inflammatory markers are noted in ED patients, as they are in cardiovascular disease.[38] These factors can be responsible for the improper function of the penile corpora cavernosa and the onset of atherosclerosis – including the coronary arteries).[39] The time interval between ED and cardiovascular disease symptoms may be partially explained by the differences in the lumen of the vessels.

Common ground There are several links between the 3 conditions, summarized in FIGURE 2. These 3 health problems are frequent and likely to coexist, as in the 3 patients described in the beginning of the article. As noted above, obesity plays a major role in the development and in the course of OSA, AF, and ED.[4,5,17,29] These are also associated with age.[2,14,15,23-25] The connection between OSA and AF is well-described in numerous publications. The prevalence of OSA in consecutive AF patients is estimated to be over 40%, much more than in the general population.[40] Complex pathomechanisms link both conditions. AF is partially a consequence of autonomic dysregulation, elevated sympathetic tone, oxidative stress, endothelial dysfunction, and left atrial stretch caused by arterial hypertension secondary to OSA. Atrial stretch causes aberrant conduction and, as a consequence, AF.[41] OSA not only plays a part in the onset of AF, but also decreases the effectiveness of invasive AF treatment. Patients with OSA are overall more likely to experience the recurrence of AF after pulmonary vein isolation, as well as after pharmacological and electric cardioversion.[42] As for OSA and ED, their concurrence is even more frequent. Almost 70% of male patients with OSA suffer from ED.[43] The odds for ED, in patients who are at high risk of OSA according to the Berlin Questionnaire and the Epworth Sleepiness Scale are 55.71 (95% CI, 3.36–923.81; P = 0.005).[44] AF, besides having a negative impact

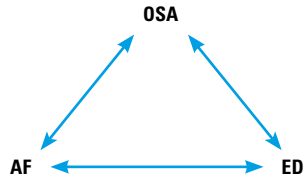

FIGURE 3 Mutual impact of obstructive sleep apnea (OSA), atrial fibrillation (AF), and erectile dysfunction (ED) syndrome components

on the overall quality of life, also hampers sexual activity in men.[45]

An important link between the single components of OSAFED is endothelial dysfunction. Patients with OSA tend to have lower levels of nitric oxide, which is responsible for vasodilatation and erection. Furthermore, the hypoxia which exists in OSA promotes the production of endothelin, a potent vasoconstrictor, which may act also on penile arteries.[46] In fact, endothelial dysfunction is one of the most important mechanisms responsible for erectile dysfunction, which in many cases is one of its first manifestations – earlier, for instance, than coronary artery disease or cerebral artery disease.[47] AF is also a risk factor for endothelial dysfunction. It is associated with impaired acetylcholine-mediated blood flow increase and reduced plasma nitrite/nitrate levels, and therefore associated with impaired blood flow and ED.[20] Similarly, oxidative stress – one of the promotors of endothelial dysfunction – occurs in AF and OSA, promoting ED.

Of course, the most important issue in case of OSA, AF, and ED, is the cardiovascular risk associated with each of the diseases. It is unclear whether the increased cardiovascular risk found in patients with OSA, AF, and ED, is secondary to the sleep apnea, the arrhythmia, the sexual dysfunction or to all of them. OSA, AF, and ED are not only risk factors, but diagnoses associated with certain prognoses, possible complications and established treatment methods which can affect the course of the disease. Every syndrome being a cluster of findings rather than an individual disease, it describes, however, ongoing processes in a manner that can be clinically useful. Naming the combination of the 3 diseases as OSAFED syndrome emphasizes their common pathogenesis and their effect on the cardiovascular system. We can assume, for instance, considering that those 3 conditions imply on their own endothelial dysfunction, inflammation or increased sympathetic tone, that the abnormality will be at least 3 times more severe if OSA, AF, and ED coexist. Furthermore, patients with OSAFED syndrome immediately will be at a high risk for cardiovascular disease.

Introducing OSAFED into everyday clinical practice would be highly advantageous. Cardiovascular disease is very common and still relatively unknown.[48] The promotion of healthy lifestyles is unsatisfactory.[49] Patients often do not know that their symptoms should be reported. OSAFED syndrome would remind health care workers to screen every patient with any of OSA, AF, or ED for the other 2 conditions, which would result in an early diagnosis and break the vicious circle of mutual disease exacerbation (FIGURE 3).

Conclusions The presence of untreated OSA, AF, or ED seems to decrease the efficacy of treatment of the other diseases. Given their prevalence and their effect on cardiovascular risk and long-term outcomes, the introduction of the OSAFED syndrome seems justified and necessary. Clinicians should assess individuals with any component of the OSAFED syndrome for the remaining ones. OSAFED can be a simple acronym, which will help remember an important connection and improve on the cardiovascular risk profile of many patients.

REFERENCES

1 Szymanski FM, Karpinski G, Platek AE, et al. Should cardiologist routinely screen and evaluate patients for sleep disordered breathing? Kardiol Pol. 2013; 71: 845-847.

2 Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med. 1993; 328: 1230-1235.

3 Szymański FM. [OSA patient – things to remember]. Kardiol Pol. 2012; 70: 30. Polish.

4 Lopez PP, Stefan B, Schulman CI, et al. Prevalence of sleep apnea in morbidly obese patients who Obesity and Obstructive Sleep Apnea 463 presented for weight loss surgery evaluation: more evidence for routine screening for obstructive sleep apnea before weight loss surgery. Am Surg. 2008; 74: 834-838.

5 Peppard PE, Young T, Palta M, et al. Longitudinal study of moderate weight change and sleep-disordered breathing. JAMA. 2000; 284: 3015-3021.

6 Shah N, Roux F. The relationship of obesity and obstructive sleep apnea. Clin Chest Med, 2009; 30: 455-465.

7 Karch I, Olszowska M, Tomkiewicz-Pająk L, et al. The effect of physical activity on serum levels of selected biomarkers of atherosclerosis. Kardiol Pol. 2013; 71: 55-60.

8 Lettieri CJ, Eliasson AH, Greenburg DL. Persistence of obstructive sleep apnea after surgical weight loss. J Clin Sleep Med, 2008; 4: 333-338.

9 Jóźwik-Plebanek K, Prejbisz A, Janaszek-Sitkowska H, et al. Obstructive sleep apnea and cardio-vascular damage. Kardiol Pol. 2012; 70: 735-740.

10 Ip MS, Tse HF, Lam B, et al. Endothelial function in obstructive sleep apnea and response to treatment. Am J Respir Crit Care Med. 2004; 169: 348-353.

11 Hrynkiewicz-Szymanska A, Szymanski FM, Filipiak KJ, et al. Can obstructive sleep apnea be a cause of in-stent thrombosis? Sleep Breath. 2011; 15: 607-609.

12 Shamsuzzaman AS, Winnicki M, Lanfranchi P, et al. Elevated C-reactive protein in patients with obstructive sleep apnea. Circulation. 2002; 105: 2462-2464.

13 Szymanski FM, Karpinski G, Hrynkiewicz-Szymanska A, et al. Resistant hypertension in an obese patient with obvious obstructive sleep apnea and occult pheochromocytoma. Can J Cardiol. 2012; 28: 397.e5-7.

14 Stewart S, Hart CL, Hole DJ, et al. Population prevalence, incidence, and predictors of atrial fibrillation in the Renfrew/Paisley study. Heart. 2001; 86: 516-521.

15 Go AS, Hylek EM, Phillips KA, et al. Prevalence of diagnosed atrial fibrillation in adults: national implications for rhythm management and stroke prevention: the AnTicoagulation and Risk Factors in Atrial Fibrillation (ATRIA) Study. JAMA. 2001; 285: 2370-2375.

16 European Heart Rhythm Association; European Association for Cardio-Thoracic Surgery, Camm AJ, Kirchhof P, Lip GY, et al. Guidelines for the management of atrial fibrillation: the Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC). Eur Heart J. 2010; 31: 2369-2429.

17 Nabauer M, Gerth A, Limbourg T, et al. The Registry of the German Competence NETwork on Atrial Fibrillation: patient characteristics and initial management. Europace. 2009; 11: 423-434.

18 Wolowacz SE, Samuel M, Brennan VK, et al. The cost of illness of atrial fibrillation: a systematic review of the recent literature. Europace. 2011; 13: 1375-1385.

19 Kirchhof P, Auricchio A, Bax J, et al. Outcome parameters for trials in atrial fibrillation: executive summary. Recommendations from a consensus

conference organized by the German Atrial Fibrillation Competence NETwork (AFNET) and the European Heart Rhythm Association (EHRA). Eur Heart J. 2007; 28: 2803-2817.

20 Li J, Solus J, Chen Q, et al. The role of inflammation and oxidative stress in atrial fibrillation. Heart Rhythm. 2010; 7: 438-444.

21 Knecht S, Oelschlager C, Duning T, et al. Atrial fibrillation in stroke-free patients is associated with memory impairment and hippocampal atrophy. Eur Heart J. 2008; 29: 2125-2132.

22 Thrall G, Lane D, Carroll D, et al. Quality of life in patients with atrial fibrillation: a systematic review. Am J Med. 2006; 119: 448 e1-e19.

23 Selvin E, Burnett AL, Platz EA. Prevalence and risk factors for erectile dysfunction in the US. Am J Med. 2007; 120: 151-157.

24 Shaeer O, Shaeer K. The Global Online Sexuality Survey (GOSS): the United States of America in 2011. Chapter I: erectile dysfunction among English-speakers. J Sex Med. 2012; 9: 3018-3027.

25 Izdebski Z. [Report on sexuality of Poles. 2011]. TNS OBOP. Polish.

26 Weber MF, Smith DP, O'Connell DL, et al. Risk factors for erectile dysfunction in a cohort of 108 477 Australian men. Med J Aust. 2013; 199: 107-111.

27 Wełnicki M, Mamcarz A. Is erectile dysfunction an independent risk factor of coronary heart disease or another clinical manifestation of progressive atherosclerosis? Kardiol Pol. 2012; 70: 953-957.

28 Andersen I, Heitmann BL, Wagner G. Obesity and sexual dysfunction in younger Danish men. J Sex Med. 2008; 5: 2053-2060.

29 Han TS, Tajar A, O'Neill TW, et al.; EMAS group. Impaired quality of life and sexual function in overweight and obese men: the European Male Ageing Study. Eur J Endocrinol. 2011; 164: 1003-1011.

30 Giugliano D, Giugliano F, Esposito K. Sexual dysfunction and the Mediterranean diet. Public Health Nutr. 2006; 9: 1118-1120.

31 Giuliano FA, Leriche A, Jaudinot EO, et al. Prevalence of erectile dysfunction among 7689 patients with diabetes or hypertension, or both. Urology. 2004; 64: 1196-1201.

32 Dong JY, Zhang YH, Qin LQ. Erectile dysfunction and risk of cardiovascular disease: meta-analysis of prospective cohort studies. J Am Coll Cardiol. 2011; 58: 1378-1385.

33 Jackson G, Boon N, Eardley I, et al. Erectile dysfunction and coronary artery disease prediction: evidence-based guidance and consensus. Int J Clin Pract. 2010; 64: 848-857.

34 Filipiak KJ, Głuchowski W, Stolarz P, et al. The sexual activity of young men six months after myocardial infarction. Kardiol Pol. 2002; 56: 40-47.

35 Puchalski B, Szymański FM, Kowalik R, et al. [Sexual dysfunction before heart failure in patients with cardiovascular risk factors – a retrospective pilot study]. Kardiol Pol. 2013; 71: 1168-1173.

36 Puchalski B, Szymański FM, Kowalik R, et al. [Sexual dysfunction in the first 9 months in men after heart failure]. Psychiatr Pol. 2013; 47: 811-826.

37 Rosato E, Barbano B, Gigante A, et al. Erectile dysfunction, endothelium dysfunction, and microvascular damage in patients with systemic sclerosis. J Sex Med. 2013; 10: 1380-1388.

38 Vlachopoulos C, Rokkas K, Ioakeimidis N, et al. Inflammation, metabolic syndrome, erectile dysfunction, and coronary artery disease: common links. Eur Urol. 2007; 52: 1590-1600.

39 Yao F, Huang Y, Zhang Y, et al. Subclinical endothelial dysfunction and low-grade inflammation play roles in the development of erectile dysfunction in young men with low risk of coronary heart disease. Int J Androl. 2012; 35: 653-659.

40 Gami AS, Pressman G, Caples SM, et al. Association of atrial fibrillation and obstructive sleep apnea. Circulation. 2004; 110: 364-367.

41 Goyal SK, Sharma A. Atrial fibrillation in obstructive sleep apnea. World J Cardiol. 2013; 5: 157-163.

42 Ng CY, Liu T, Shehata M, Stevens S, et al. Meta-analysis of obstructive sleep apnea as predictor of atrial fibrillation recurrence after catheter ablation. Am J Cardiol. 2011; 108: 47-51.

43 Budweiser S, Enderlein S, Jörres RA, et al. Sleep apnea is an independent correlate of erectile and sexual dysfunction. J Sex Med. 2009; 6: 3147-3157.

44 Szymanski FM, Filipiak KJ, Hrynkiewicz-Szymanska A, et al. The high risk of obstructive sleep apnea-an independent risk factor of erectile dysfunction in ST-segment elevation myocardial infarction patients. J Sex Med. 2011; 8: 1434-1438.

45 Dąbrowski R, Smolis-Bąk E, Kowalik I, et al. Quality of life and depression in patients with different patterns of atrial fibrillation. Kardiol Pol. 2010; 68: 1133-1139.

46 Arruda-Olson AM, Olson LJ, Nehra A, Somers VK. Sleep apnea and cardiovascular disease. Implications for understanding erectile dysfunction. Herz. 2003; 28: 298-303.

47 Elesber AA, Solomon H, Lennon RJ, et al. Coronary endothelial dysfunction is associated with erectile dysfunction and elevated asymmetric dimethylarginine in patients with early atherosclerosis. Eur Heart J. 2006; 27: 824-831.

48 Piwońska A, Piotrowski W, Broda G. Knowledge about arterial hypertension in the Polish population: the WOBASZ study. Kardiol Pol. 2012; 70: 140-146.

49 Borowiec A, Lignowska I, Drygas W. Attitudes towards healthy lifestyle promotion in mass media in the Polish adult population. Kardiol Pol. 2012; 70: 1030-1037.

ARTYKUŁ POGLĄDOWY

Obturacyjny bezdech senny, migotanie przedsionków i zaburzenia erekcji – tylko współistniejące schorzenia czy nowa jednostka chorobowa? Przedstawienie koncepcji zespołu OSAFED

Filip M. Szymański, Bartosz Puchalski, Krzysztof J. Filipiak

I Katedra i Klinika Kardiologii, Warszawski Uniwersytet Medyczny, Warszawa

SŁOWA KLUCZOWE

obturacyjny bezdech senny, migotanie przedsionków, OSAFED, zaburzenia erekcji

STRESZCZENIE

Większość pacjentów z chorobami układu krążenia zmaga się jednocześnie z kilkoma schorzeniami, które często mają wspólne czynniki ryzyka oraz etiologię, a w większości wypadków zaostrzają nawzajem swój przebieg. Fenotyp właściwy dla jednej choroby często występuje również w przypadku kilku innych jednostek. Poniżej prezentujemy opisy kliniczne przypadków pacjentów z obturacyjnym bezdechem sennym (*obstructive sleep apnea* – OSA), migotaniem przedsionków (*atrial fibrillation* – AF) i zaburzeniami erekcji (*erectile dysfunction* – ED). Te trzy schorzenia często występują w populacji ogólnej. OSA jest najczęściej występującym rodzajem zaburzeń oddychania w czasie snu, dotykającym około 24% mężczyzn i 9% kobiet w wieku od 30 do 60 lat. AF, jedno z najczęstszych zaburzeń rytmu serca obecne jest u około 2% populacji, a ED dotykają pomiędzy 18% a 40% mężczyzn powyżej 20. rż. Współwystępowanie tych trzech schorzeń u jednego pacjenta można uznać nie za przypadek, ale raczej za nowy zespół chorobowy. Poniżej prezentujemy dane, które pozwalają przypuszczać, że zespół (*obstructive sleep apnea*, *atrial fibrillation*, *erectile dysfunction* – OSAFED) może mieć istotniejszy wpływ na profil ryzyka sercowo-naczyniowego niż wszystkie trzy schorzenia z osobna. Wprowadzenie do praktyki klinicznej terminu OSAFED może przynieść jedną istotną zaletę – przypomnieć lekarzom praktykom o konieczności wykonywania u pacjentów z jedną ze składowych zespołu OSAFED badań mających na celu wykrycie pozostałych dwóch schorzeń, co wiąże się z wcześniejszą diagnozą i przerwaniem błędnego koła wzajemnego zaostrzania przebiegu opisanych schorzeń.

Adres do korespondencji:
dr n. med. Filip M. Szymański,
I Katedra i Klinika Kardiologii,
Warszawski Uniwersytet Medyczny,
ul. Banacha 1A, 02-097 Warszawa,
tel.: 22-599-19-58
fax: 22-599-19-57,
e-mail: filip.szymanski@wum.edu.pl
Praca wpłynęła: 26.08.2013.
Przyjęta do druku: 25.09.2013.
Publikacja *online*: 26.09.2013.
Nie zgłoszono sprzeczności interesów.
Pol Arch Med Wewn. 2013;
123 (12): 701-707