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Atrial fibrillation and gastroesophageal reflux disease: From the cardiologist perspective

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Abstract

We have read with interest the paper by Roman C. and colleagues discussing the relationship between gastroesophageal reflux disease and atrial fibrillation. The review is presenting the available evidence for the common pathogenic mechanisms. However, from a cardiologist perspective, some available data were not highlighted in the review, cardiovascular involvement in gastroesophageal reflux is less assessed. Hypertension, obesity or diabetes mellitus are substrate for left atrial remodeling that initiate and sustained atrial fibrillation development. One of the pathophysiologic mechanisms in atrial fibrillation is the presence of a trigger. Gastroesophageal reflux could be only a trigger for this arrhythmia. We believe that atrial fibrillation should be considered as possible extraesophageal syndrome in the gastroesophageal reflux classification.

Key words: Atrial fibrillation; Gastroesophageal reflux disease; Trigger; Substrate; Vagal stimulation

Core tip: The cardiologist point of view about the relationship between atrial fibrillation and gastroesophageal reflux disease is less expressed in the literature. However cardiovascular involvement in gastroesophageal reflux is less assessed. Hypertension, obesity or diabetes mellitus are substrate for left atrial remodeling that initiate and sustained atrial fibrillation development. One of the pathophysiologic mechanisms in atrial fibrillation is the presence of a trigger. Gastroesophageal reflux could be only a trigger for this arrhythmia. We believe that atrial fibrillation should be considered as possible extraesophageal syndrome in the gastroesophageal reflux classification.

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TO THE EDITOR

We have read with interest the paper by Roman C. and colleagues discussing the relationship between gastroesophageal reflux disease (GERD) and atrial fibrillation (AF)[1]. The review is presenting the available evidence for the common pathogenic mechanisms. However, from a cardiologist perspective, some available data were not highlighted in the review.

Both GERD and AF are common in general population. The available data suggest that AF seems to be more frequent that reported in the review. The prevalence of AF increases with age and reaches the highest values (about 20%) in a population older than 60 years. Also, we are assisting in the last two decades to a 13% increase in incidence[2]. Over 6 million Europeans suffer from this arrhythmia, and its prevalence is estimated to at least double in the next 50 years as the population ages[2].

Atrial fibrillation occurs when structural and/or electrophysiological abnormalities (electrical, mechanical and structural remodeling of left atrium) alter atrial tissue to promote abnormal impulse formation and/or propagation. Beginning with this step AF begets AF, through remodeled atrial myocardium. Left atrial remodeling may be caused by diverse pathophysiologic mechanisms and AF may represent a final common phenotype for multiple disease pathways.

In a large majority of patients with AF we can find a pathology like hypertension, obesity or diabetes mellitus as substrate for left atrial remodeling. Even patients diagnosed with idiopathic AF have more often suffered from insidious coronary artery disease than healthy controls in sinus rhythm[3]. Most common substrates for both AF and GERD are obesity and aging. In addition both AF and GERD are associated with other pathologies like sleep apnea or diabetes mellitus.

In AF, like in other cardiac arrhythmias, three elementary electrophysiological mechanisms, alone or in combination, may be involved in the initiation and perpetuation of arrhythmia. These are: enhanced or abnormal automaticity, triggered activity and reentry. Enhanced or abnormal automaticity and triggered activity are subcellular mechanisms, whereas reentry is depending on intercellular and tissue properties.

The trigger and the substrate are at the origin of AF. The substrat reflects the concept according to which the triggered electrical activation perpetuates in an autonomous way. In addition, the trigger itself can modify the substrate, especially if active for a prolonged period.

Sympatho-vagal imbalance is one of the principal mechanisms of AF associated with GERD[4]. Although both sympathetic and parasympathetic components play a role in AF, the cholinergic component appears to be important for spontaneous initiation of AF. Electrical stimulation of the left atrial ganglionic plexi (situated on left atrial posterior wall, close to the esophagus) or the autonomic nerve endings with retrograde activation of the ganglia induces spontaneous firing from pulmonary veins followed by AF[4].

Majority of AF patients with GERD have triggered AF[5]. During radiofrequency ablation, these patients may have positive vagal response[5]. Gastroesophageal reflux could be only a trigger for AF in paroxysmal AF.

Less known, not only GERD may trigger AF, but also AF may determine the occurrence of GERD. It was speculated that an enlarged and fibrillating left atrium may compress or irritate the neighboring lower esophagus[6].

Indeed, cardiovascular involvement in GERD is less assessed. It is mandatory to extend the research in this field for better understanding the relationship of AF with GERD.

Recognizing that morbidity and mortality associated with AF had remained unacceptably high despite all efforts aimed at improving its management, the aetiology of AF was placed in the foreground for the first time by the Third Consensus Conference of the Atrial Fibrillation Competence Network/European Heart Rhythm Association[7].

It seems that among traditional cardiovascular risk factors, GERD could be an independent risk factor for AF. Also, we think that AF should be considered as possible extraesophageal syndrome in the GERD classification[8].

REFERENCES

1 **Roman C**, Bruley des Varannes S, Muresan L, Picos A, Dumitrascu DL. Atrial fibrillation in patients with gastroesophageal reflux disease: a comprehensive review. *World J Gastroenterol* 2014; **20**: 9592-9599 [PMID: 25071357 DOI: 10.3748/wjg.v20.i28.9592]

2 **Camm AJ**, Kirchhof P, Lip GY, Schotten U, Savelieva I, Ernst S, Van Gelder IC, Al-Attar N, Hindricks G, Prendergast B, Heidbuchel H, Alfieri O, Angelini A, Atar D, Colonna P, De Caterina R, De Sutter J, Goette A, Gorenek B, Heldal M, Hohloser SH, Kolh P, Le Heuzey JY, Ponikowski P, Rutten FH. 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 [PMID: 20802247 DOI: 10.1093/eurheartj/ehq278]

3 **Calò L**, Martino A. Insidious coronary artery disease: cause, consequence, or innocent bystander for idiopathic atrial fibrillation? *Heart Rhythm* 2012; **9**: 1930-1931 [PMID: 23063866 DOI: 10.1016/j.hrthm.2012.08.036]

4 **Reddy YM**, Singh D, Nagarajan D, Pillarisetti J, Biria M, Boolani H, Emert M, Chikkam V, Ryschon K, Vacek J, Bommana S, Atkins D, Verma A, Olyaee M, Dawn B, Lakkireddy D. Atrial fibrillation ablation in patients with gastroesophageal reflux disease or irritable bowel syndrome-the heart to gut connection! *J Interv Card Electrophysiol* 2013; **37**: 259-265 [PMID: 23736874 DOI: 10.1007/s10840-013-9807-5]

5 **Velagapudi P**, Turagam MK, Leal MA, Kocheril AG. Atrial fibrillation and acid reflux disease. *Clin Cardiol* 2012; **35**: 180-186 [PMID: 22318757 DOI: 10.1002/clc.21969]

6 **Tibbling L**, Wranne B. Oesophageal symptoms and manometry in valvular heart disease. *Acta Med Scand* 1979; **206**: 381-383 [PMID: 525438]

7 **Kirchhof P**, Lip GY, Van Gelder IC, Bax J, Hylek E, Kaab S, Schotten U, Wegscheider K, Boriani G, Brandes A, Ezekowitz M, Diener H, Haegeli L, Heidbuchel H, Lane D, Mont L, Willems S,

Dorian P, Aunes-Jansson M, Blomstrom-Lundqvist C, Borentain M, Breitenstein S, Brueckmann M, Cater N, Clemens A, Dobrev D, Dubner S, Edvardsson NG, Friberg L, Goette A, Gulizia M, Hatala R, Horwood J, Szumowski L, Kappenberger L, Kautzner J, Leute A, Lobban T, Meyer R, Millerhagen J, Morgan J, Muenzel F, Nabauer M, Baertels C, Oeff M, Paar D, Polifka J, Ravens U, Rosin L, Stegink W, Steinbeck G, Vardas P, Vincent A, Walter M, Breithardt G, Camm AJ. Comprehensive risk reduction in patients with atrial fibrillation: emerging diagnostic and therapeutic options--a report from the 3rd Atrial Fibrillation Competence NETwork/European Heart Rhythm Association consensus conference. *Europace* 2012; **14**: 8-27 [PMID: 21791573 DOI: 10.1093/europace/eur241]

8 **Vakil N**, van Zanten SV, Kahrilas P, Dent J, Jones R. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. *Am J Gastroenterol* 2006; **101**: 1900-1920; quiz 1943 [PMID: 16928254]

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