MATHEWS Open Access Journals

ISSN: 2572-6420

Short Communication | Mathews Journal of Cardiology

Augmented Atrial Vulnerability in Patients with Sinus Node Dysfunction: Is the Electrophysiologic Abnormality Limited Only to the Sinus Node?

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Received Date: 12 Nov 2018
Accepted Date: 14 Nov 2018
Published Date: 15 Nov 2018

Citation: Centurión OA, Alderete JF, Torales JM, García LB, et al. (2018). Augmented Atrial Vulnerability in Patients with Sinus Node Dysfunction: Is the Electrophysiologic Abnormality Limited Only to the Sinus Node?. M J Cardiol. 3(1): 019.

INTRODUCTION

Sinus node dysfunction (SND) refers to an entity with abnormalities in the functionality of the sinus node which may be due to an alteration in the generation of impulses within the sinus node itself, as well as, a disturbance of the conduction of impulses from the sinus node to the atrial muscle. Typical electrocardiographic features correlating with clinical findings are one or more episodes of extreme sinus bradycardia, or sinus pauses due to sinoatrial block or sinus arrest, or episodes of alternating bradycardia and atrial tachyarrhythmias. Bradycardia may be mainly due by two mechanisms in the SND. It could be produced by an alteration in the generation of impulses within the sinus node. In addition, it may be generated by a disturbance of the conduction of impulses from the sinus node to the surrounding atrial myocardium [1-4]. SND is usually secondary to aging structural changes of the sinus node and the surrounding atrial myocardium. Patients with this disorder are often elderly with symptoms of stunning, pre-syncope, syncope and palpitations and generally have other comorbidities. It may be difficult to establish a frank relationship between symptoms and the electrocardiogram (ECG). This is due to the fact that symptoms may be variable in nature, non-specific and frequently transient. Hence, the conventional ECG is not sufficient to make a clear diagnosis of the symptoms that present the patients. Therefore, additional diagnostic tests may be required.

The diagnosis of SND should not be definitely performed until other potentially reversible causes have been excluded. For example, the use of drugs, myocardial ischemia, hypothyroidism, and autonomic imbalance should be excluded as possible causes. Moreover, we have to consider that well-trained athletes often have bradycardia. The definite diagnosis of SND will be made by establishing a correlation between the patient's clinical symptoms and the ECG findings. If the ECG and repeated 24-hour Holter ECG monitoring fail to document the cause of a patient's symptoms, consideration should be given to utilize an implantable continuous loop-recorder device [5]. Stress tests can help identify the abnormal function of the sinus node, exclude myocardial ischemia, and can help guide device programming for patients who eventually receive a permanent pacemaker [2]. Electrophysiological studies allow determine certain parameters of SND and atrial vulnerability. Therefore, this invasive study could be considered especially in those patients who persist symptomatic and in those who have not documented episodes of the ECG alterations described above [6-9].

Electrophysiological investigations based on the recording of abnormally prolonged and fractionated atrial local electrograms by endocardial catheter mapping during sinus rhythm and their characteristic distribution in the right atrium of patients with SND have provided important knowledge about the electrophysiological properties of the diseased atrium [10-15]. It is well known that abnormal atrial electrogram results in an irregular atrial conduction characterized by a non-ho-

mogeneous local electrical activity, related to an anisotropic, non-uniform and delayed conduction through diseased atrial walls [16-21]. The detection of abnormal atrial electrograms in the SND identifies a group of patients with increased atrial vulnerability and a significantly higher incidence of spontaneous or induced episodes of atrial fibrillation. Regarding duration and fractionation of electrograms, it has been shown that the slowing of the conduction velocity causes a decrease in the amplitude and an increase in the duration of the extracellular electrogram of the canine atria and Purkinje system [22, 23]. In addition, it has been studied and demonstrated in a computerized model of electrogram generation that the decreased conduction velocity was responsible for the increase in electrogram duration, while intracellular resistance increased, was responsible for the fractional nature of the electrogram [24].

Patients with SND present histological alterations not only within the sinus node but also in the atrial myocardium suggesting that the tissue damage is not limited to sinus node itself. Detailed and quantitative pathological studies performed in patients with SND have demonstrated extensive atrial myocardial fibrosis in the vicinity of the sinus node and internodal tracts (25, 26). In addition, it has been demonstrated histologically that the tissues where the abnormally prolonged and fractionated electrograms originate present fibro-degenerative processes [25, 26]. When the atrial walls are markedly altered by fibrosis, the depolarization wave must frequently change direction with respect to the longitudinal orientation of the myocardial fiber. This would cause unidirectional block, slow conduction and dispersion of the refractory periods in certain places, generating the fundamental elements of the reentry mechanism [27-29]. This tissue damage induced electrophysiological changes generate episodes of PAF in SND patient. Centurión OA et al. designed a study to evaluate the relationship between certain electrophysiological parameters that indicate increased atrial vulnerability and abnormal atrial electrograms in patients with SND [27]. In this study, an attempt was made to clarify the importance and significance of the recording of abnormal atrial electrograms during sinus rhythm in patients with SND susceptible of developing episode of PAF. By programmed atrial stimulation with single extra-stimulus, we tried to induce the electrophysiological indicators of increased atrial vulnerability, namely, the fragmented atrial activity, atrial conduction delay, repetitive atrial firing and sustained atrial fibrillation. We demonstrated that patients who had abnormal atrial electrograms had a significantly increased atrial vulnerability, compared to those patients who had normal electrograms. Abnormal atrial electrograms showed a very good specificity and positive predictive value when evaluating the induction of sustained episode of PAF. The specificity demonstrated was 94% with a positive predictive value of 93% [27].

In conclusion, although there are several factors that influence to a greater or lesser degree the appearance of AF in these patients with SND, abnormal atrial electrograms recorded during sinus rhythm in patients with electrophysiological alterations of the atrial myocardium could be considered as indicators of an increased atrial vulnerability. The clinical implication demonstrated is that the detection of abnormal atrial electrograms during sinus rhythm in SND patients susceptible of developing AF can help to identify a group of patients with significantly increased atrial vulnerability and, a significantly higher incidence of spontaneous or induced episodes of PAF. Therefore, we can assume that patients with SND present histological alterations not only within the sinus node but also in the atrial myocardium suggesting that the tissue damage is not limited only to the sinus node itself.

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