



Transient ST Elevation in Vagally Mediated Atrial Fibrillation

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Abstract

We report a case of vagally mediated atrial fibrillation on a young otherwise healthy man, with straight type ST-segment elevation in inferolateral leads that resolved a few hours after restoration of sinus rhythm, a phenomenon that has never been previously reported. Even though no definite conclusion about the underlying mechanism of the ST-elevation can be made, this effect might probably be the result of intense parasympathetic tone and could be used to differentiate the causality.

Introduction

A 35-year old Caucasian man, with no significant past medical history, presented in the early hours in October, complaining of palpitations and dizziness. He reported initiation of the palpitations during his sleep, something that occurred 18 and 6 months before, again during relaxation time in the evening in the first case and during his sleep in the latter. He denied recent illness and drug ingestion. The patient also denied any shortness of breath and chest pain during his hospitalization.

His physical examination revealed an irregularly irregular heart rate at approximately 90 beats per minute and no murmurs or abnormal heart sounds on auscultation. His blood pressure was 91/67mmHg and he had normal body temperature and oxygen saturation on ambient air.

The electrocardiogram demonstrated atrial fibrillation with a mild straight-type ST elevation in inferolateral leads (figure 1). Serum chemistry suggested no systemic illness including electrolyte imbalance and thyroid dysfunction.

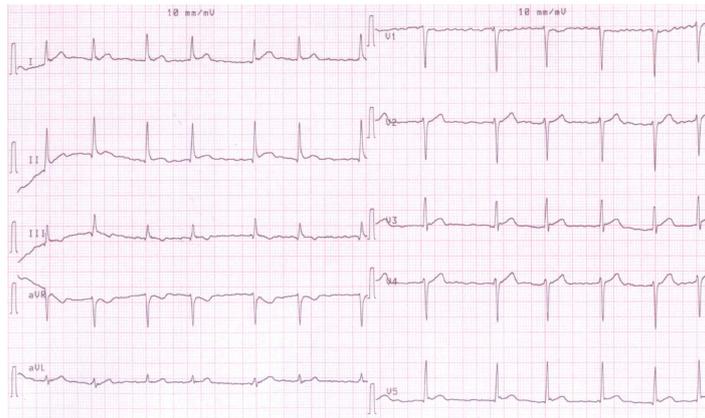
Cardiac biomarker levels (repeated at 9 and 24 hours) were unremarkable while the chest X-ray and transthoracic echocardiography demonstrated no structural abnormalities.

A single oral loading dose of propafenone (600mg) was given and sinus rhythm was restored 3 hours later. An ECG 12 hours following cardioversion (next morning), (figure 2), surprisingly demonstrated attenuation of the ST segment elevation recorded while being on atrial fibrillation and showed similar findings with an electrocardiogram performed 6 months before, during previous cardiological evaluation. His blood pressure was also restored to normal (126/81mmHg).

Discussion

Vagally mediated atrial fibrillation occurs more frequently in young healthy men than in women (ratio 4:1).¹ The age of onset is usually 30–50 years. It hardly ever occurs in a structurally diseased heart, probably because any cardiac disease tends to shift the vagosympathetic balance towards a sympathetic predominance.² Rather

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Figure 1: Non rapid atrial fibrillation with ST segment elevation in inferolateral leads

than being precipitated by emotional or physical stress, the arrhythmia more likely occurs at relaxation periods and most frequently in the evening as indicated by heart rate variability studies and by the presence of bradycardia in individuals with structurally normal hearts.³ Other inciting events (reflecting increased vagal tone) include cough, nausea, rest, post-prandial states and alcohol.⁴ Vagally mediated atrial fibrillation was also reported on a healthy 43-year-old woman after ingestion of frozen yogurt. The authors hypothesized that the episode was the result of intense vagal stimulation caused by the extremely cold temperature of the dessert.⁵

Holter monitoring may show sinus bradycardia before the onset of atrial fibrillation and a slow ventricular response during the episode.⁶ Vagal stimulation has been shown to shorten the atrial refractory period⁷, thereby decreasing the wavelength of atrial excitation wave fronts. The shorter the wavelength, the higher is the probability that multiple reentrant circuits can exist simultaneously in the atrial myocardium. Furthermore, vagal stimulation has also been shown to facilitate reentry,⁸ and this effect has been used to induce or maintain AF in experimental models.⁹ Recently, Schauerte and co-workers¹⁰ showed that transvascular atrial parasympathetic nerve system modification by radiofrequency catheter ablation could abolish vagally-mediated AF in dogs, thus proving the major role played by the parasympathetic tone on the induction and/or maintenance of AF.

The high level of vagus nerve tone maintained during slow-wave sleep, has the capacity to exacerbate atrial fibrillation in patients whose atria are particularly prone to the arrhythmogenic influ-

ence of acetylcholine.¹¹

Conversely, adrenergically mediated paroxysmal atrial fibrillation occurs more commonly in patients older than 50 years, during the daytime, and during periods of increased physical or emotional stress. The diseased myocardium is more sensitive to sympathetic stimulation evoked by these stressors.

ECG changes at night have been recorded and described. In addition to the occurrence of transient episodes of increased heart rate, the ST-segment is generally elevated throughout the night when compared to the day and is associated with a slower heart rate.¹² Such ST elevation, especially seen in young individuals, may be considered to be part of the normal variant, the early repolarization pattern.¹³

In addition, reports have described transient ST segment elevation predominantly in the inferior leads, when transeptal LA catheterization was used during AF ablation procedures.¹⁴⁻²¹ The authors have yielded no definite conclusion about the underlying mechanism of the ST elevation, but some have proposed that autonomic imbalance via enhanced parasympathetic stimulation could be the cause. Others have suggested that a probable explanation of ST segment elevation in the inferolateral leads, could be an embolic attack in a coronary artery (RCA or LCx) or air embolism during the procedure. Our patient neither had such a procedure performed nor experienced any chest pain, and his serum cardiac biomarker levels and repeat electrocardiogram were unremarkable. Thus we deemed unnecessary to

Figure 2: ECG 12 hours after restoration of sinus rhythm showing attenuation of the ST segment elevation



perform either a coronary angiography or a transesophageal echocardiography study.

The selective location of the ST-segment injury pattern may indicate a link between the atrial septum, the Left Atrium and the parasympathetic nervous system. An intricate intracardiac system of nerves has been identified in animal and human hearts.^{22,23} Atrial ganglionated plexuses were found on the superior surface of the left and right atria, the posterior medial surface of the LA, the inferior and lateral aspects of the posterior LA, and the interatrial septum. Hence, it is plausible that manipulation of the atrial septum and the LA causes selective activation of right cardiac parasympathetic nerve stimulation to the inferior or posterior myocardial region.

The transient ST elevation as well as medium ventricular response could also be the result of a Bezold-Jarisch-like reflex, as in 2 cases reported by Arita and colleagues.¹⁶ This could as well explain the asymptomatic hypotension experienced by our patient while on non-rapid atrial fibrillation. Experimental evidence suggests that this cardiac reflex results from the activation of inhibitory cardiac receptors with vagal afferents that are located predominantly in the inferoposterior wall of the left ventricle.²⁴

Published data on the treatment of vagally mediated AF is limited. Beta blockers and digoxin are not only ineffective but are contraindicated, as they

tend to precipitate the arrhythmia by shifting the balance toward vagal effects on the atria.²⁵ When episodes are more frequent or symptoms become intolerable, long-term use of antiarrhythmics has been shown to maintain sinus rhythm at 1 year 50% to 60% of the time, but these medications carry proarrhythmic risks.²⁶ Flecainide and propafenone can be used in the treatment of vagally mediated atrial fibrillation as both medications have also vagolytic actions, with flecainide's vagolytic effect being more significant.²⁶ Atrial pacing prevents vagally induced AF and can be used for patients with frequent recurrences.²⁷

Risk stratification for stroke and thromboembolism should also be determined using current risk scores and appropriate prophylaxis should be initiated if needed.^{28,29}

Conclusions

We report a case of vagally mediated atrial fibrillation on a young otherwise healthy individual, with straight type ST-segment elevation in inferolateral leads that resolved a few hours after cardioversion, a phenomenon that to our knowledge has never been reported. Even though no definite conclusion about the underlying mechanism of the ST-elevation can be made, this effect might probably be the result of intense parasympathetic tone which is the underlying pathophysiology of the nosological entity. Consequently, it could be

used to differentiate the causality. Further case studies are needed before a definite link between the reported observation and vagally mediated atrial fibrillation can be established.

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