ARCHIVES OF THE TURKISH SOCIETY OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Fingers on the Wrist and Taming the Funny Current, Rejuvenate the Wounded Heart: Ivabradine-Responsive Atrial Tachycardia

Parmaklar Bilekte iken Funny Akımının Ehlileştirilmesi Yaralı Kalbi Gençleştiriyor: Ivabradine Yanıt Veren Atriyal Taşikardi

ABSTRACT

A 19-year-old male presented with dyspnea on exertion (New York Heart Association [NYHA] class II) and occasional palpitations for six months. He had initially been evaluated at another facility and diagnosed with dilated cardiomyopathy. Despite treatment, there was no improvement in his symptoms. On evaluation at our centre, his previous electrocardiograms appeared normal. However, palpation of his radial pulse for one minute revealed runs of regular tachycardia, interspersed with a normal pulse rate. A 30-second rhythm strip electrocardiogram (ECG) showed multiple runs of ectopic tachycardia originating from the right atrial appendage, interspersed with ectopic atrial rhythms. Echocardiography showed severe left ventricle (LV) dysfunction with an ejection fraction of 20-25%. Radio-frequency ablation was recommended, but the patient declined. Instead, he was started on Ivabradine. After a month, his symptoms fully resolved. The ECG displayed a normal sinus rhythm with no tachycardia, and his left ventricular ejection function improved.

Keywords: Arrhythmia, heart failure, left ventricular dysfunction, supraventricular tachycardia

ÖZET

On dokuz yaşında erkek hasta, 6 aydır NYHA sınıf II eforla nefes darlığı ve ara sıra çarpıntı şikayetiyle başvurdu. İlk olarak başka bir yerde değerlendirilmiş ve dilate kardiyomiyopati tanısı konmuştu. Tedaviye rağmen belirtilerinde herhangi bir iyileşme yoktu. Merkezimizdeki değerlendirmede, önceki elektrokardiyogramları normal görünüyordu. Ancak, bir dakika boyunca radyal nabzının palpasyonu, normal nabız hızıyla aralıklı düzenli taşikardi ataklarını ortaya çıkardı. Otuz saniyelik ritim şeridi elektrokardiyogramında (EKG), aralıklı ektopik atriyal ritimlerle, çok sayıda sağ atriyal apendiksten kaynaklanan ektopik taşikardi görüldü. Ekokardiyografi, %20-25'lik ejeksiyon fraksiyonu ile ciddi LV disfonksiyonunu ortaya koydu. Hastaya radyofrekans ablasyonu önerildi. Ancak hasta ablasyon için istekli olmadığından, ivabradin başlandı. Bir ay sonra, belirtileri tamamen düzeldi. EKG, taşikardi serileri olmaksızın normal sinüs ritmini gösterdi ve sol ventrikül ejeksiyon fonksiyonu iyileşti.

Anahtar Kelimeler: Aritmi, kalp yetmezliği, sol ventrikül disfonksiyonu, supraventriküler taşikardi

Cardiomyopathy comprises a heterogeneous and diverse group of disorders affecting the myocardium, leading to ventricular dysfunction. In a significant proportion of cases, the etiology for reduced ejection fraction is completely reversible. With the advent of newer imaging techniques and biomarkers for the diagnosis and management of various ailments, the time-honored skill of clinical examination is gradually being overshadowed. We present a case of severe reduced ventricular dysfunction previously treated as dilated cardiomyopathy, which was eventually diagnosed as ectopic atrial tachycardia. The diagnosis was made through a simple clinical examination and systematic analysis of the electrocardiogram (ECG) and was successfully managed with Ivabradine. This case underscores the importance of clinical examination and the attention to detail in basic diagnostic tests like the ECG, even in this modern era. Additionally, it highlights the potential therapeutic use of Ivabradine in treating ectopic atrial tachycardia.

CASE REPORT
OLGU SUNUMU

A. Shaheer Ahmed
Gaurav Kumar Divani
Nitish Rai
Anwar Hussain Ansari

Department of Cardiology, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, India

Corresponding author:
A. Shaheer Ahmed
✉ ahmedshaheer53@gmail.com

Received: December 05, 2022
Accepted: March 27, 2023

Cite this article as: Ahmed AS, Divani GK, Rai, N, Ansari AH. Fingers on the wrist and taming the funny current, rejuvenate the wounded heart: ivabradine-responsive atrial tachycardia. *Turk Kardiyol Dern Ars.* 2023;51(8):580-583.

DOI:10.5543/tkda.2023.60296

Case Report

A 19-year-old male presented with complaints of dyspnea on exertion (New York Heart Association [NYHA] class II) and palpitations for six months. He had no history of angina, syncope, or pedal edema. Prior evaluations at two different centres diagnosed him with dilated cardiomyopathy, and he was treated with a beta-blocker, angiotensin-converting enzyme inhibitor (ACEI), diuretic, and spironolactone. Despite this treatment, his symptoms did not improve. A review of his previous medical records indicated several normal electrocardiograms and severe left ventricular dysfunction.

Upon examination of the radial pulse over a full minute (60 seconds), there were sudden-onset runs of increased pulse rate, which were regular, interspersed with periods of normal pulse rate. His blood pressure was within the normal range, and pulses were palpable equally in all limbs. Cardiac auscultation revealed no abnormalities. Even though previous electrocardiograms appeared normal, given his tachycardia on clinical examination, we conducted a 30-second rhythm strip electrocardiogram. This ECG showed incessant runs of regular narrow complex tachycardia. Detailed examination identified this as a long RP tachycardia, with negative P waves in lead V1, P wave transition in lead V6, and positive P waves in the inferior leads (Figure 1). The P wave morphology during non-tachycardia segment suggested an ectopic focus from the left atrial appendage/left pulmonary vein (Figure 2). The tachycardia might have been overlooked in previous ECGs because standard ECG recordings typically last around 2.5 seconds. The accompanying normal rhythm strip that comes with a lead ECG records for 10 seconds. Unfortunately, previous ECGs were taken during tachycardia-free intervals. Echocardiography showed severe left ventricular dysfunction, with the left ventricle dilated (end-systolic/end-diastolic dimensions: 48/57 mm) and a left ventricular ejection fraction of 20-25%. Serum electrolytes, thyroid profile, and vitamin D levels were all within normal limits. Given the patient's incessant ectopic atrial tachycardia and related cardiomyopathy, catheter ablation was recommended. However, the patient declined any invasive procedures. Consequently, he was prescribed Ivabradine 2.5 mg twice daily, in addition to his ongoing heart failure medication.

On a follow-up 10 days later, no tachycardia was observed, and the electrocardiogram showed a normal sinus rhythm and typical P wave origin (Figure 3). At this point, the patient agreed to catheter ablation. However, tachycardia could not be induced in the electrophysiology lab. Therefore, the patient continued with Ivabradine. After one month, his left ventricular ejection fraction improved to 40-45%, and the global longitudinal strain by speckle tracking echocardiography was -16.2%. There was a marked improvement in the left ventricle (LV) dimensions (end-systolic/end-diastolic: 34/50 mm), and the patient remained asymptomatic without any recurrence of tachycardia.

ABBREVIATIONS

EAT	Ectopic atrial tachycardia
ECH	Electrocardiogram
LV	Left ventricular
NYHA	New York Heart Association

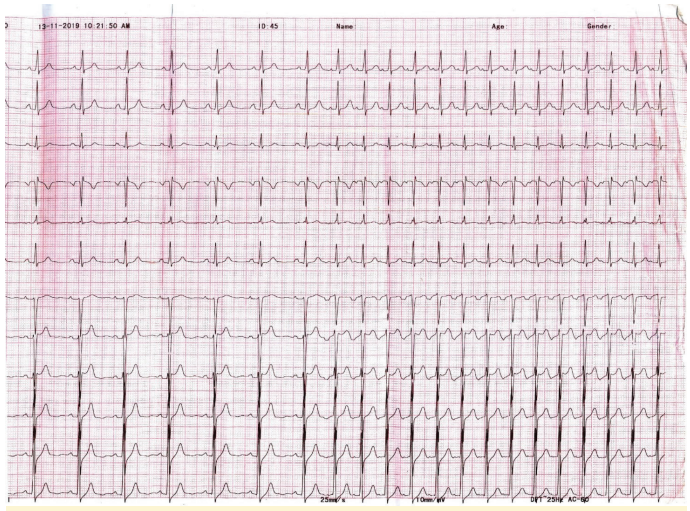

Figure 1. Electrocardiogram at presentation showing ectopic atrial rhythm and runs of atrial tachycardia.

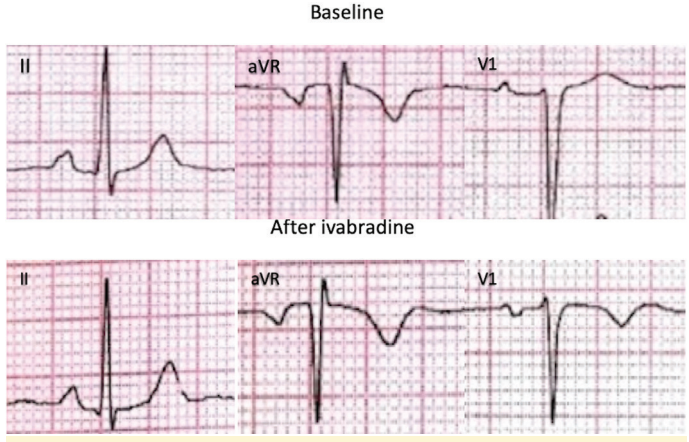

Figure 2. Electrocardiogram showing P wave morphology in leads II, aVR, and V1.

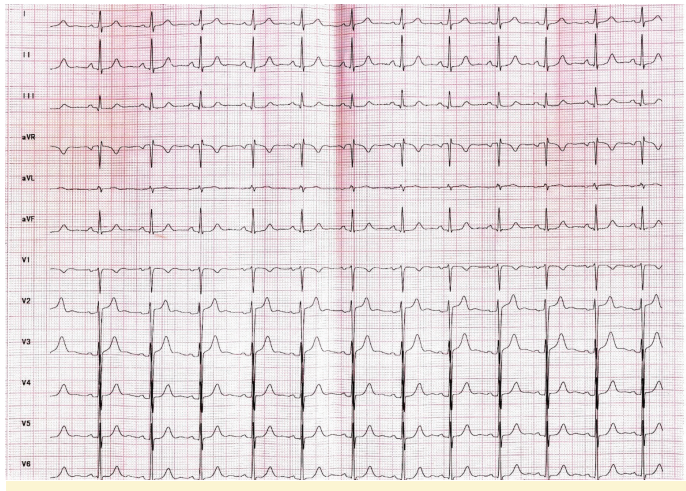

Figure 3. Electrocardiogram on follow-up showing normal sinus rhythm.

Discussion

Examination of the pulse provides vital information for diagnosing left ventricular dysfunction. Pulsus alternans, which is characterized by a weaker pulse alternating with a stronger one, is diagnostic of a dilated left ventricle and severe ventricular dysfunction. A slow-rising, low-volume pulse suggests aortic stenosis as the cause of ventricular dysfunction.[1] Palpating the pulse for a full minute helps identify arrhythmic causes of ventricular dysfunction, which might not be evident on a routine 12-lead ECG, such as ventricular premature beats and ectopic atrial tachycardia.

Attention to the P wave morphology provides valuable diagnostic information. The normal P wave morphology is positive in leads I, II, negative in aVR, and biphasic in lead V1. The normal P wave axis ranges from 0-70°. However, in our case, the baseline ECG during the tachycardia-free interval showed a monophasic P wave (positive in lead V1), suggesting an ectopic origin. During the tachycardia, the P wave was negative in leads V1-V4 and positive in the inferior leads, indicative of a right atrial appendage origin. In patients presenting with ventricular dysfunction and an ectopic atrial rhythm, an evaluation for atrial tachycardia should be conducted.[2]

Tachycardiomyopathy is defined as an impairment of left ventricular function secondary to long-standing tachycardia. This condition can be completely or partially reversed after normalization of the rate and rhythm abnormality.[3] Incessant tachycardias, like ectopic atrial tachycardia (EAT) and paroxysmal junction reciprocating tachycardia (PJRT), often present with ventricular dysfunction. Both these arrhythmias can be paroxysmal, making early diagnosis challenging at times. Focal atrial tachycardias originating from the right atrial appendage and pulmonary veins are more frequently associated with cardiomyopathy.[4] Atrial fibrillation and flutter can also lead to tachycardiomyopathy if the ventricular rate is not controlled. Frequent ventricular premature contractions and the presence of an accessory pathway, especially the right free wall pathway, can predispose individuals to ventricular dysfunction due to dyssynchrony.[5] Among ventricular arrhythmias, fascicular tachycardias and outflow tract tachycardias can lead to ventricular dysfunction. Several mechanisms are postulated in the pathogenesis of tachycardiomyopathy, including adenosine triphosphate (ATP) depletion, reduced responsiveness and number of beta-adrenergic receptors, abnormal calcium handling, and decreased coronary blood flow due to a shortened diastolic period.[6,7] The onset of tachycardiomyopathy can vary, ranging between three days and 120 days. Three stages in the development of ventricular dysfunction have been described: the compensatory phase (3-7 days), the LV dysfunction phase (7-21 days), and the LV failure phase (> 3 weeks). The first phase is characterized by neurohormonal activation and extracellular matrix changes. The second and third phases involve cellular remodeling with LV systolic dysfunction and LV dilatation, respectively.[8] Upon treating arrhythmias, the earliest signs of improvement in ventricular function can be observed within 48 hours, and in most cases, complete recovery is achieved by three months.

Ivabradine acts on the If funny current, which is a mixed sodium-potassium inward current responsible for spontaneous diastolic depolarization. Ivabradine is currently indicated for controlling heart rate in heart failure with reduced ejection and stable angina if the heart rate is more than 70/min despite taking the maximum tolerated dosage of beta blockers. It is also used in inappropriate sinus tachycardia. Lately, there are reports of its usage in junctional ectopic tachycardia.[9,10] However, there is limited data on its usage in EAT.[11-14] In a recent study, it was found that 64% of patients with ectopic atrial tachycardia respond to Ivabradine. Ivabradine responsiveness is maximal when the focus is located in the atrial appendages.[15] In our case, the foci of tachycardia and ectopic atrial rhythm were in the appendages. The mechanism of arrhythmogenesis in Ivabradine-responsive ectopic atrial tachycardia is probably enhanced automaticity, whereas those non-responsive might have micro re-entry. Ivabradine in the pediatric population has been shown to reverse ventricular dysfunction in those with junctional ectopic tachycardia within a month. Ivabradine in combination with beta blockers might be an effective option for ectopic atrial tachycardia in the pediatric population where catheter ablation is technically difficult, for those not willing for ablation, and as a bridge to catheter ablation in locations with resource-limited settings where facilities for catheter ablation are not readily available.

A detailed clinical examination still remains vital in the diagnostic workup of cardiovascular diseases, even in the 21st century where much weightage is given to technology-based diagnosis. We should develop the habit of palpating the pulse for at least a whole minute during the first visit, as it can provide us with a lot of information vital for diagnosis and management. Although, on the face of it, the previous ECGs appeared normal, had attention been given to the morphology of the P wave in lead V1, atrial tachycardia could have been suspected during the first medical contact. The finger on the radial pulse and Ivabradine (taming the funny current) helped improve the ventricular function in this case.

Conclusion

Evaluation for a possible reversible cause of reduced left ventricular ejection fraction should be done in all patients. Palpating the radial pulse for a whole minute is a vital component of the cardiovascular examination. Ivabradine might be an effective alternative for patients with ectopic atrial tachycardia in the pediatric population who are not willing for catheter ablation and those in areas where facilities for ablation are not available. A systematic analysis of P wave morphology in a 12-lead electrocardiogram might provide a vital clue for a possible diagnosis of atrial arrhythmias. This differential diagnosis is essential and should be kept in mind for every pediatric dilated cardiomyopathy referred for cardiac transplantation.

Informed Consent: Written informed consent was obtained from the patient.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – A.S.A., N.R., A.H.A.; Design – A.S.A.; Supervision – A.S.A.; Materials – A.S.A., G.D., N.R.; Data Collection and/or Processing – A.S.A., G.D., N.R.; Analysis and/or Interpretation – A.S.A., G.D.; Literature Search – A.S.A., G.D., N.R.; Writing – A.S.A., N.R.; Critical Review – A.S.A., A.H.A.

Conflict of Interest: No conflict of interest disclosure has been received from the authors.

Funding: The authors declared that this study received no financial support.

References

1. Leier CV, Chatterjee K. The physical examination in heart failure--Part I. *Congest Heart Fail.* 2007;13(1):41-47. [CrossRef]
2. Platonov PG. P-wave morphology: underlying mechanisms and clinical implications. *Ann Noninvasive Electrocardiol*. 2012;17(3):161-169. [CrossRef]
3. Gallagher JJ. Tachycardia and cardiomyopathy: the chicken-egg dilemma revisited. *J Am Coll Cardiol.* 1985;6(5):1172-1173. [CrossRef]
4. Kistler PM, Sanders P, Fynn SP, et al. Electrophysiological and electrocardiographic characteristics of focal atrial tachycardia originating from the pulmonary veins: acute and long-term outcomes of radiofrequency ablation. *Circulation.* 2003;108(16):1968-1975. [CrossRef]
5. Dai C, Guo B, et al. The effect of ventricular pre-excitation on ventricular wall motion and left ventricular systolic function. *Europace.* 2018;20(7):1175-1181. [CrossRef]
6. Khasnis A, Jongnarangsin K, Abela G, Veerareddy S, Reddy V, Thakur R. Tachycardia-induced cardiomyopathy: a review of literature. *Pacing Clin Electrophysiol.* 2005;28(7):710-721. [CrossRef]
7. Martin CA, Lambiase PD. Pathophysiology, diagnosis and treatment of tachycardiomyopathy. *Heart.* 2017;103(19):1543-1552. [CrossRef]
8. Ulus T, Okyay K, Kabul HK, et al. Turkish Society of Cardiology consensus paper on management of arrhythmia-induced cardiomyopathy. *Anatol J Cardiol.* 2019;21(2):98-106. [CrossRef]
9. Dieks JK, Klehs S, Müller MJ, Paul T, Krause U. Adjunctive ivabradine in combination with amiodarone: A novel therapy for pediatric congenital junctional ectopic tachycardia. *Heart Rhythm.* 2016;13(6):1297-1302. [CrossRef]
10. Janson CM, Tan RB, Iyer VR, Vogel RL, Vetter VL, Shah MJ. Ivabradine for treatment of tachyarrhythmias in children and young adults. *Heart Rhythm Case Rep.* 2019;5(6):333-337. [CrossRef]
11. Cohen MI, Cohen JA, Shope C, Stollar L, Collazo L. Ivabradine as a stabilising anti-arrhythmic agent for multifocal atrial tachycardia. *Cardiol Young.* 2020;30(6):899-902. [CrossRef]
12. Bohora S, Lokhandwala Y, Parekh P, Vasavda A. Reversal of tachycardiomyopathy due to left atrial tachycardia by ivabradine. *J Cardiovasc Electrophysiol.* 2011;22(3):340-342. [CrossRef]
13. Meles E, Carbone C, Maggiolini S, et al. A case of atrial tachycardia treated with ivabradine as bridge to ablation. *J Cardiovasc Electrophysiol.* 2015;26(5):565-568. [CrossRef]
14. Michel H, Heißenhuber F, Wellmann S, Melter M, Gerling S. Ectopic Atrial Tachycardia in a 12-Month-Old Girl Treated With Ivabradine and Beta-Blocker, a Case Report. *Front Pediatr.* 2020;8:313. [CrossRef]
15. Banavalikar B, Shenthar J, Padmanabhan D, et al. Clinical and electrophysiological correlates of incessant ivabradine-sensitive atrial tachycardia. *Circ Arrhythm Electrophysiol.* 2019;12(8):e007387. [CrossRef]