An unusual case of syncope

Shruti Bajad¹, Chetan Rijhwani²

ABSTRACT

Early repolarization syndrome (ERS) is a common electrocardiography (ECG) variant which may prove to be an indicator of increased long-term cardiac mortality in general population. Although the condition is usually considered to be benign, potential arrhythmogenicity of it has been suggested by experimental studies. ERS involving inferolateral leads in the ECG has been seen to be associated with syncopal attacks or sudden cardiac death in young healthy persons. Here, we report a rare case of young asymptomatic healthy male who presented with syncope while at rest and sinus bradycardia with incidental findings of ERS on ECG.

Key Words: Electrocardiography, sudden cardiac death, syncope

Introduction

Early repolarization syndrome (ERS) is known to have an estimated prevalence of about 3% [1]. Studies suggest a higher risk of ERS in athletes, cocaine drug abusers or ventricular cardiac defects [1]. Previously considered a benign condition with a normal electrocardiogram (EKG) variant demonstrating "J-point elevation," recent evidence has debated ERS to impact prognosis and quality of life, warranting vigilant monitoring. Considering the EKG finding, ERS has often been misdiagnosed as Brugada syndrome (BS) and ventricular arrhythmias leading to sudden cardiac death (SCD) in young patients. Here, we report a rare case of young asymptomatic healthy male (nonathelete) who presented with syncope while at rest and sinus bradycardia with incidental findings of ERS on ECG.

Case Report

A 23-year-old young male presented with a short episode of unconsciousness while praying at the church. During prehospital transport, the patient is alleged to have regained consciousness. On initial examination at the emergency room, his Glasgow Coma Score was 15, was bradycardic (pulse: 40/min) and his blood pressure (BP) not recordable. Examination was unremarkable otherwise. The patient denied any similar episode in the past with no previous syncopal attacks, chest pain, palpitations or any sudden death, or cardiac diseases within his family. He denied use of any drugs or addiction.

The patient was moved to the Intensive Care Unit for monitoring and further evaluation. EKG demonstrated sinus bradycardia with an elevation of J-point in the inferior and lateral leads,

¹Department of Rheumatology and Clinical Immunology, Medanta - The Medicity, Gurgaon, Haryana, India; ²Department of Medicine, Fortis Hospital, New Delhi, India.

Corresponding Author

Shruti Bajad, E-mail: shrutibajad@yahoo.in

peaked T-waves in V3–V6 along with ST-elevation with upward concavity in V2–V4 [Figure 1]. Blood investigations were unremarkable except for monocytosis (21%). Serum electrolytes and renal and liver functions were within normal limits. His thyroid profile was normal, table tilt test was negative, and magnetic resonance imaging of the brain was normal. Based on initial findings, a diagnosis of ERS was made.

Holter monitoring was conducted to closely monitor any variations to his vitals and cardiac rhythm. The patient developed two episodes of ventricular tachycardia (VT), each lasting <10 s. In contrary to patients with ERS, VT was unusual as these patients generally develop ventricular fibrillation. The patient received infusion of intravenous (iv) fluids, atropine, and symptomatic treatment following which his BP stabilized to 110/70 mm Hg and pulse 44/min. Postural hypotension was not noted. At this time, his EKG demonstrated sinus rhythm at rest with normal pulse rate. EKG elevation in J-point became less during exercise and aggravated on giving propranolol. At rest, he maintained a pulse of 40–44 beats per min. His condition being stable, the patient was discharged home on day 3 and was advised regular follow-up with options for pacemaker and implantable cardioverter defibrillator.

Discussion

ERS was considered a benign or normal variant of electrocardiographic pattern of ventricular repolarization until recently [2]. The presence of early repolarization was defined as an elevation of the QRS–ST junction (J-point) in at least two leads. The amplitude of J-point elevation is expected to be at least 1 mm (0.1 mV) above the baseline level [3,4]. Recently, this concept has observed a change in its diagnosis with many studies demonstrating the possibility of it not being a benign clinical feature. The prevalence of ERS is age-dependent, predominantly seen in young adults and African-American population. However, the genetic basis of ERS is still largely unknown although it has been reported

that 16% of patients with ERS usually have a positive family history [5].

Thus far, clinical interest in this syndrome has been confined to its differential diagnosis toward myocardial infraction (MI), acute pericarditis, BS, and SCD [6]. Although latter includes unexpected natural cause of death due to cardiac causes with unknown history of heart disease [7], the most common cause noted in SCD is from fatal causes of ventricular arrhythmia and those related to the electrophysiological disorders of BS. BS resembles closely to ERS in the prominence of J-wave with a defective right bundle branch block and an elevation of the ST-segment in leads (V1-V3) [8]. To differentiate, BS EKG is observed on the right precordial leads and when inducing a block to the sodium channel, it shows an increase in J-point compared to ERS EKG which diminishes the J-point [9]. Other differentials of ERS such as MI and acute pericarditis demonstrate contrasting clinical features compared to ERS. Although they may present with initial J-point elevation, acute pericarditis shows the deviation of the PR segment which is not present in ERS. While MI may have an initial J-point elevation associated with ST elevation MI.

The clinical feature of ERS is divided into two categories. The first type features the patients with syncopal attacks who may or may not be athletes and with maximum episodes occurring during exertion and patients surviving from a postcardiac arrest episode [10]. These patients are noted to have higher chance of recurrence of cardiac events which poses a higher risk [11]. The second type includes asymptomatic patients with incidental EKG features of ERS and have less risk from cardiac adverse effects [11-14]. Our case of a young male with no strenuous activity presented with clinical features of syncope associated with EKG findings pointing toward a diagnosis of ERS. Thus, this is an unusual fact for this syndrome as the patient got unprovoked syncope. Moreover, sinus bradycardia is always found in conjunction with ERS when the patient is athletic, but was present without it in our case. Thus, it proves that this combination of bradycardia and ERS can be present in patients who are not athletes and can be potentially dangerous for future cardiac arrest if not addressed.

Haïssaguerre et. al. reported that in nearly one-third of case subjects, EKG obtained before cardiac arrest showed early repolarization [11]. This indicate that this abnormality could not be the result of the trauma attained from sudden cardiac arrest, resuscitation efforts, or drugs used for resuscitation. The repolarization abnormality that was recorded by inferolateral leads may be a marker of underlying electrical vulnerability that increases the risk of fatal arrhythmias under conditions that need to be investigated. This patient presented with syncope and ECG pattern of early repolarization in inferolateral leads placing him in high risk as ERS involving inferolateral leads makes any individual vulnerable for arrhythmias [11]. Tikkanen et. al. stated that J-point elevation of more than 0.2



Figure 1 (a) J-point elevation in lead II. (b) J-point elevation in V4, V5, and V6. (c) ST elevation in inferior leads

mV in inferior leads was a stronger predictor of death from cardiac causes than other well-known electrocardiographic risk markers, such as the QTc interval and signs of left ventricular hypertrophy. Furthermore, the early-repolarization pattern remained an independent prognostic marker even after adjustment for several other risk factors [15]. Therefore, it is pertinent that physicians investigate ERS in patients presenting with syncope and gauge family history to screen for cardiac adverse effects. However, investigating every patient with syncope attack is not cost-effective, especially in developing countries.

Conclusion

Patients with ERS should be investigated to rule out risks of SCD along with regular follow-ups. Research is warranted to identify risk-factors associated with ERS and appropriate measures of prevention.

References

- Nam GB, Ko KH, Kim J, et. al. Mode of onset of ventricular fibrillation in patients with early repolarization pattern vs. Brugada syndrome. *Eur Heart J* 2010;31(3):330-9.
- Gussak I, Antzelevitch C. Early repolarization syndrome: clinical characteristics and possible cellular and ionic mechanisms. *J Electrocardiol* 2000;33(4):299-309.
- Klatsky AL, Oehm R, Cooper RA, et. al. The early repolarization normal variant electrocardiogram: correlates and consequences. Am J Med 2003;115(3):171-7.
- Mehta M, Jain AC, Mehta A. Early repolarization. Clin Cardiol 1999;22(2):59-65.
- Liu X, Shah A, Sacher F, et. al. Clinical frontiers in electrocardiographic early repolarization syndrome: does a good guy turn bad now? *Chin Med J (Engl)* 2010;123(11):1459-62.
- Di Grande A, Tabita V, Lizzio MM, et. al. Early repolarization syndrome and Brugada syndrome: is there any linkage? Eur J Intern Med 2008;19(4):236-40.
- Brugada J, Brugada R, Brugada P. Right bundle-branch block and ST-segment elevation in leads V1 through V3: a marker for sudden death in patients without demonstrable structural heart disease. *Circulation* 1998;97(5):457-60.

CASE REPORT

- Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and sudden cardiac death: a distinct clinical and electrocardiographic syndrome. A multicenter report. *J Am Coll Cardiol* 1992;20(6):1391-6.
- Kawata H, Noda T, Yamada Y, et. al. Effect of sodium-channel blockade on early repolarization in inferior/lateral leads in patients with idiopathic ventricular fibrillation and Brugada syndrome. *Heart Rhythm* 2012;9(1):77-83.
- Aizawa Y, Chinushi M, Hasegawa K, et. al. Electrical storm in idiopathic ventricular fibrillation is associated with early repolarization. *J Am Coll Cardiol* 2013;62(11):1015-9.
- Haïssaguerre M, Derval N, Sacher F, et. al. Sudden cardiac arrest associated with early repolarization. N Engl J Med 2008;358(19):2016-23.
- Rosso R, Halkin A, Viskin S. J waves and early repolarization: do not confuse me with the facts! *Heart Rhythm* 2012;9(10):1603-4.
- Viskin S, Rosso R, Halkin A. Making sense of early repolarization. Heart Rhythm 2012;9(4):566-8.
- Ali A, Butt N, Sheikh AS. Early repolarization syndrome: a cause of sudden cardiac death. World J Cardiol 2015;7(6):466-75.
- Tikkanen JT, Anttonen O, Junttila MJ, et. al. Long-term outcome associated with early repolarization on electrocardiography. N Engl J Med 2009;361(26):2529-37.

Authors' Contributions

Both authors participated in the clinical diagnosis, data acquisition, and drafting the manuscript. All authors have read and approved the final manuscript.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Competing Interests

The authors declare that they have no competing interests.

Funding

Sources of funding: None.

Please cite this paper as: Bajad S, Rijhwani C. An unusual case of syncope. *Int J Stud Res* 2017;7(1):11-3.



This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.