Journal of Acute Medicine 14(1): 42-47, 2024
DOI:10.6705/j.jacme.202403_14(1).0006
Case Report

Overdrive Pacing for Persistent Torsades de Pointes and Pulseless Ventricular Tachycardia

Ewe Jin Koh[1,*], Xiao Qi Yee[1], Ming Lee Chin[2], Nisa Liyana bt Abdul Latib[3]

[1]Department of Internal Medicine, Taiping Hospital, Perak, Malaysia
[2]Department of Medicine and Paediatrics, Taiping Hospital, Perak, Malaysia
[3]Department of Internal Medicine, Bintulu Hospital, Sarawak, Malaysia

A 53-year-old presented to the emergency department following a fall and was found to have recurrent episodes of torsades de pointes and pulseless ventricular tachycardia on cardiac monitoring. He had been abusing nimetazepam for sleep issues over the preceding one month. Despite correction of electrolytes, the arrhythmias were persistent which necessitated temporary overdrive pacing. The patient made an uneventful recovery and the temporary pacing was successfully removed with no recurrence of the malignant arrhythmias prior to discharge.

Keywords: *benzodiazepineo, long QT syndrome, torsades de pointes, ventricular tachycardia, verdrive pacing*

Introduction

Torsades de pointes (TdP) is a life-threatening form of polymorphic ventricular tachycardia (VT) that can be caused by an acquired or congenitally prolonged QT interval.[1] Acquired cases are often caused by drugs and may be exacerbated by electrolyte abnormalities, most frequently hypokalaemia. While monomorphic tachycardias are most commonly related to acute coronary syndromes, TdP should not be overlooked as a potential cause of life threatening VTs. This case illustrates the role of temporary overdrive pacing in terminating the life threatening tachyarrhythmia. While sounding like a misnomer, the physiology of overdrive pacing and its relation to TdP is reviewed here. This case also highlights the potential life threatening side effects of sedatives and anxiolytics.

Case Presentation

A 53-year-old Malaysian Chinese gentleman with a known history hypertension and Type 2 diabetes mellitus was brought by his wife to the emergency department of a local district hospital following an episode of a fall at home. The fall happened as a result of sudden loss of consciousness while the patient was ambulating to the washroom. Approximately two days prior, his wife had already noticed that he had been having reduced oral intake and complained of nausea and vomiting. He has had trouble sleeping over the past one month and had been approximately abusing staggered tablets of oral nimetazepam (Erimin-5) over the preceding one month. Upon arrival to emergency department, he was bradycardic with a heart rate of 40 per minute on cardiac monitoring. His blood pressure was 136/70 mmHg, respiratory rate was 22 breaths/min, oxygen saturation was 99% on room air and was afebrile with a temperature of 36.5 degrees Celsius.

It was subsequently noted that he had developed multiple episodes of pulseless VT and TdP (Fig. 1). Each episode lasted for approximately 10 s, some of which terminated spontaneously, while some required defibrillation. During each of these episodes, he would lose and regain consciousness. He denied chest

Received: March 05, 2023; Revised: March 21, 2023; Accepted: April 10, 2023.
[*]Corresponding author: Ewe-Jin Koh, MD. Department of Internal Medicine, Taiping Hospital, 34000 Taiping, Perak, Malaysia.
Email: ewejinkoh@gmail.com

pain or diaphoresis, palpitations, a history of arrhythmias or previous similar episodes.

His serum potassium was only marginally low at 2.8 mmol/L. This was immediately corrected with intravenous 30 mmol/L of potassium. His other electrolytes were within normal limits. The thyroid function test and serum troponin were normal. His electrocardiogram upon reversion of the malignant arrhythmias revealed a grossly prolonged QT interval of 630 ms (Fig. 2).

There were otherwise ST or T segment changes to suggest ischemia. His chest radiograph was normal. Acute coronary syndrome was ruled out based on the lack of a suggestive history, normal physical examination, and lack of dynamic changes on his electrocardiogram (ECG) along with normal cardiac biomarkers. The patient continued to develop multiple episodes of pulseless VT while in the emergency department requiring defibrillation. Intravenous magnesium sulphate was given; however, the episodes continued to recur. Given the severity and frequency of the malignant tachyarrhythmia, it was determined that temporary overdrive pacing would be necessary. Emergent transvenous pacing via the femoral route was performed and the patient was overdrive paced at a rate of 100 bpm with successful resolution of the

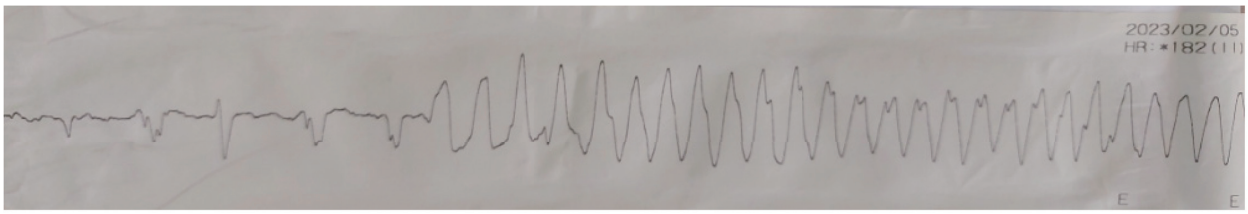

Fig. 1. ECG showing degeneration into pulseless ventricular tachycardia.

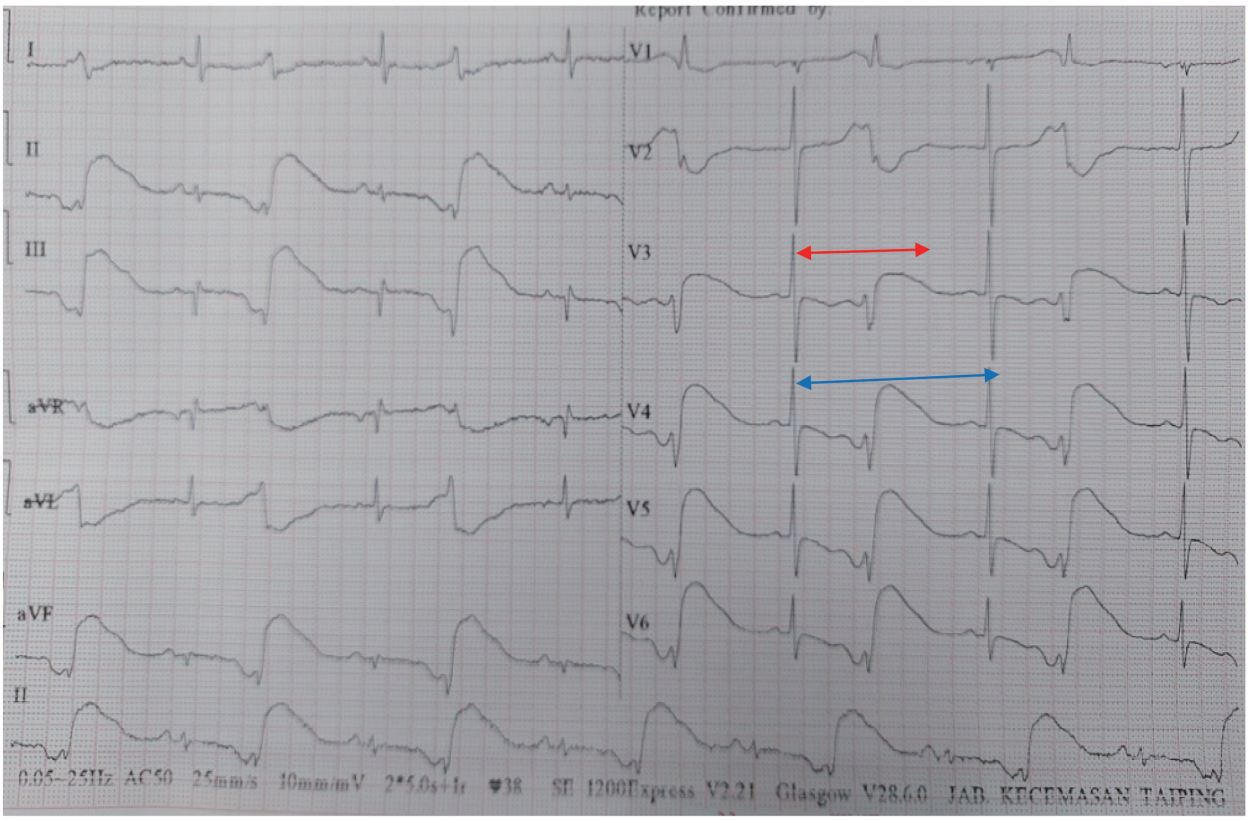

Fig. 2. ECG showing severe bradycardia with heart rate of 26 beats per minute and a prolonged QT interval of 630 ms. (red arrow depicting the actual QT interval; actual heart rate between R-R complexes depicted by blue arrow)

TdP and recurrent VT. The pacemaker was set at an output voltage of 5 V, and a sensitivity of 10 mA (Figs. 3 and 4).

In the ensuing days, there were no more episodes of pulseless VT. His echocardiogram revealed a left ventricular ejection fraction of 37% and global hypokinesia. A coronary angiogram revealed normal coronary anatomy. Therefore, the most likely explanation of the low ejection fraction is likely due to the arrhythmias. His serum electrolytes remained within the normal range over the next week and the transvenous pacer was successfully switched off with complete normalization of the QT interval (Fig. 5). The patient was discharged well.

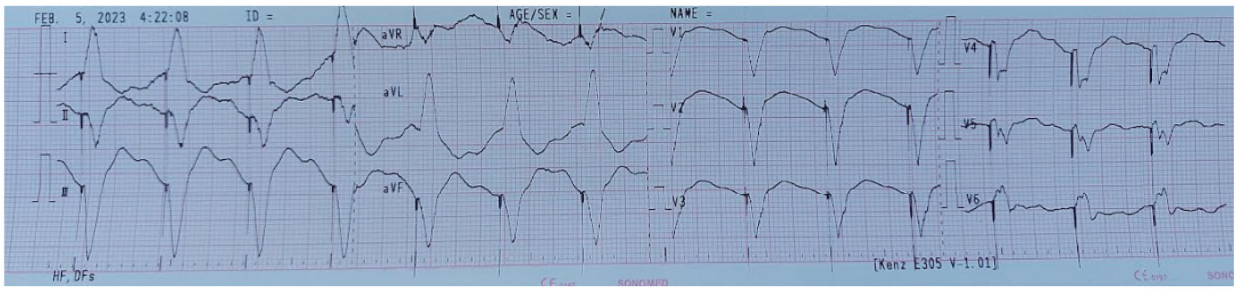

Fig. 3. ECG post transvenous pacing in the emergency department showing broad complex with left bundle branch pattern and pacing spikes seen, indicating the pacemaker is appropriately placed in the right ventricle.

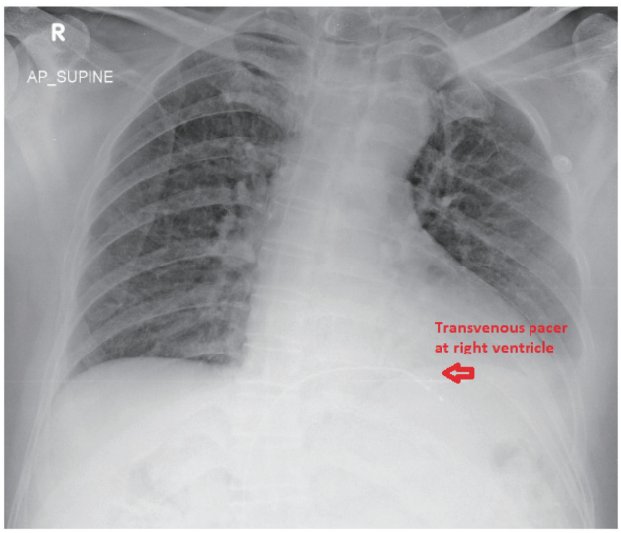

Fig. 4. Transvenous pacer in the right ventricle.

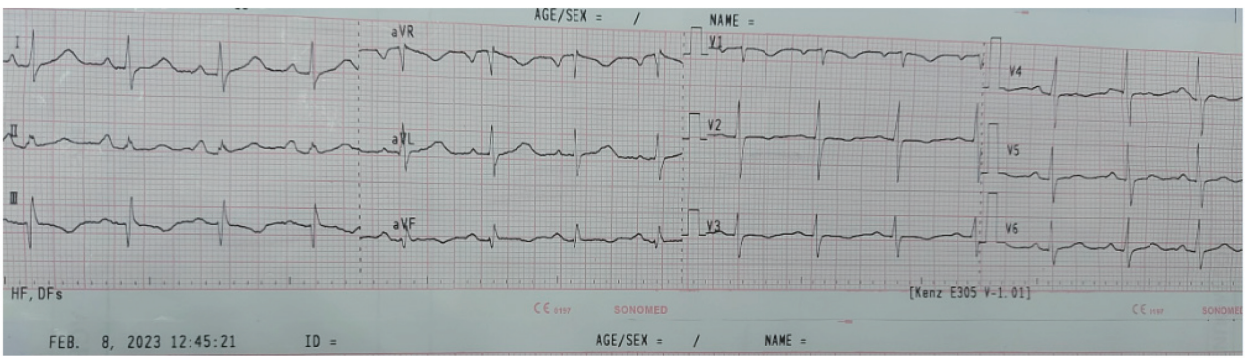

Fig. 5. ECG prior to discharge showing complete resolution of the prolonged QT interval and resolution of bradycardia.

A summary of the intervention process is depicted below. (A) Initial ECG in emergency department showing prolonged QT interval. (B) ECG post transvenous pacing showing shortening of the QT interval. (C) ECG prior to discharge in the ward after transvenous pacing was taken off showing complete resolution of the prolonged QT interval and reversion back to the patient's intrinsic rhythm (Fig. 6).

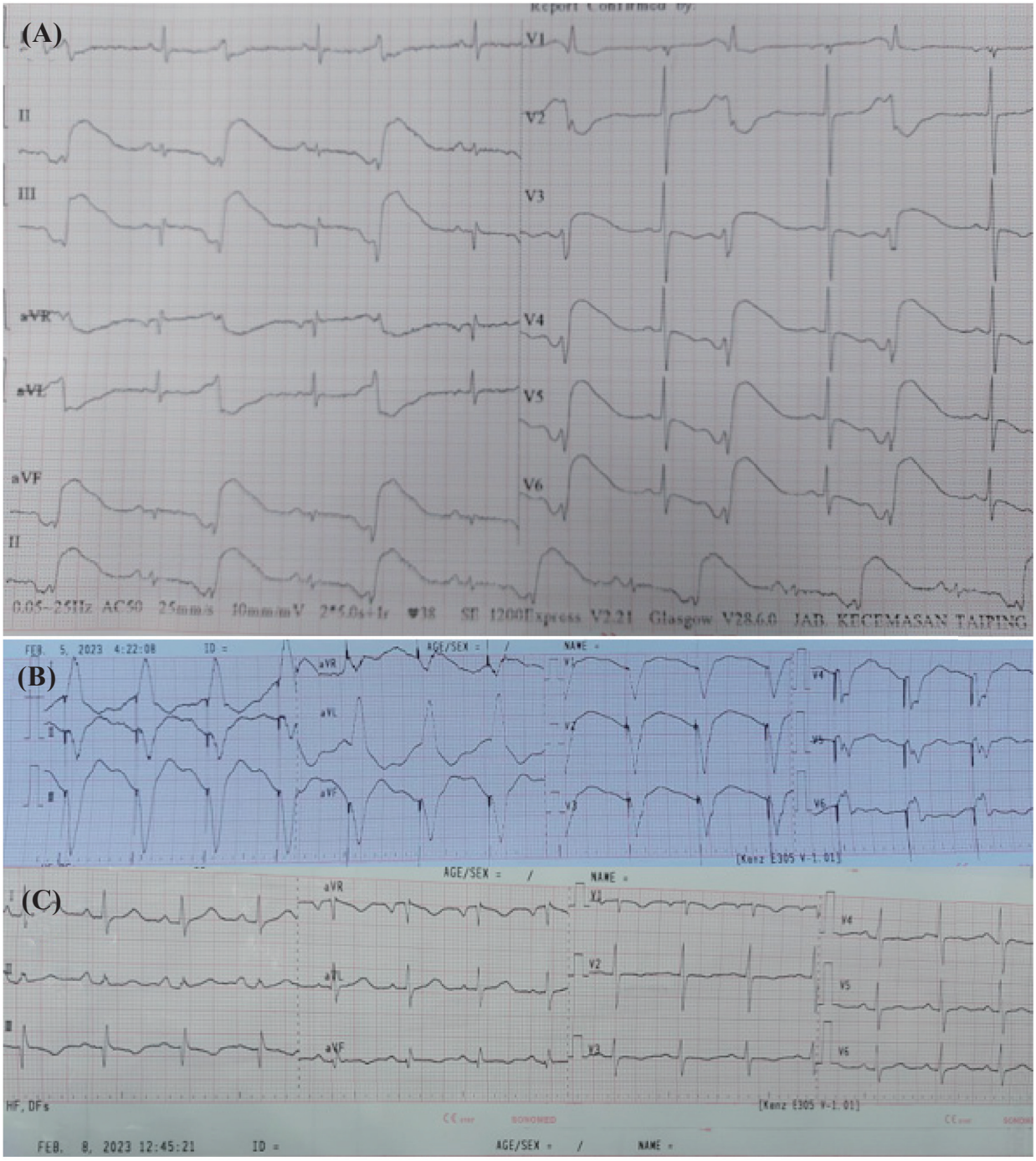

Fig. 6. ECG changes before and after transvenous pacing in a case of prolonged QT interval. (A) Initial ECG in emergency department showing prolonged QT interval. (B) ECG post transvenous pacing showing shortening of the QT interval. (C) ECG prior to discharge in the ward after transvenous pacing was taken off showing complete resolution of the prolonged QT interval and reversion back to the patient's intrinsic rhythm.

Discussion

TdP is a form of polymorphic VT. Two epidemiological studies done in the south of France and Belgium reported that the incidence of TdP is from 0.0032% per year to 0.16% per year, which is very low.[2,3] A nested case-control study conducted between 2005 and 2018, has shown that the in-hospital and 1-year mortality rates for TdP cases were 10.7% and 25.0% percent, respectively.[4] Considering the high mortality rate, much emphasis needs to be given to the recognition of this ECG finding.

Many drugs have been implicated to cause prolongation of the QT interval and increase the subsequent risk of TdP. These classically include antiarrhythmic agents, particularly amiodarone, dofetilide, quinidine, and sotalol, with quinidine possibly having the most torsadogenic potential. Antibiotics include macrolides and quinolones, while antipsychotics such as selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs) have been implicated.[5]

When TdP occurs, patients usually experience multiple episodes in rapid succession.[6] This was illustrated in our case. As these episodes may rapidly transform into a malignant VT, prompt delivery of therapy is imperative. Recommendations by the British Pharmacological Society recommends immediate treatment of TdP with intravenous magnesium sulphate and terminating prolonged episodes using electrical cardioversion. In refractory cases of recurrent TdP, the arrhythmia can be suppressed by increasing the underlying heart rate using isoproterenol (isoprenaline) or transvenous pacing.[7]

The suppression of the malignant ventricular arrhythmias lies in the fact that overdrive pacing paces the heart at a rate higher than the intrinsic heart rate, thereby shortening the QT interval and hence reducing the R on T phenomenon. Overdrive pacing for long QT syndromes has been described as early as the 1980s, a time when quinidine therapy use was more common.[8]

More recently, there have been case reports documenting the use of overdrive pacing in terminating TdP in patients treated with flunonazole and also the anti-cancer agent osimertinib.[9,10]

In our patient, he has been abusing nimetazepam. Nimetazepam (Erimin-5) is an intermediate-acting hypnotic drug which belongs to the benzodiazepine group. It was first synthesized in 1962 in Japan. Since early November 2015, it has ceased to be manufactured in Japan. However, this drug is most popular in Singapore, Malaysia, and Indonesia and is a street drug. It is also known on the street as "Happy 5".[11] We postulate that nimetazepam is the culprit drug that led to the prolonged QT interval in our patient as he was not taking any other medications and only had a marginally low serum potassium level at 2.8 mmol/L.

To date, there has only been one case report documenting benzodiazepines being implicated in prolonged QT intervals.[12] Therefore, it is important to recognize that many other medications/recreational drugs may also induce QT interval prolongation although not documented in literature.

Conclusion

Overdrive pacing has been shown to effectively terminate drug-induced prolonged QT intervals and consequent TdP by shortening the QT interval. Therefore, it is important to acknowledge the role that overdrive pacing has to offer. In the acute setting, physicians at the first point of care should be able to recognize the ECG findings suggestive of TdP and be adequately trained with temporary pacing modalities as TdP is potentially life threatening if not recognized and terminated promptly. The review also highlights the different drugs that may be implicated in causing QT interval prolongation.

Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Cohagan B, Brandis D. Torsade de Pointes. In: Salah A, Amal AG, Wendy BA, et al. eds. *StatPearls [Internet]*. Treasure Island, FL: StatPearls Publishing; 2023. Available at: https://www.ncbi.nlm.nih.gov/books/NBK459388/. Accessed August 8, 2023.
2. Molokhia M, Pathak A, Lapeyre-Mestre M, et al. Case ascertainment and estimated incidence of drug-induced long-QT syndrome: study in Southwest France. *Br J Clin Pharmacol* 2008;66:386-395. doi:10.1111/j.1365-2125.2008.03229.x
3. Vandael E, Vandenberk B, Vandenberghe J, Pincé H, Willems R, Foulon V. Incidence of Torsade de Pointes in a tertiary hospital population. *Int J Cardiol* 2017;243:511-515. doi:10.1016/j.ijcard.2017.05.072
4. Mantri N, Lu M, Zaroff JG, et al. Torsade de pointes: a nested case–control study in an integrated health-

care delivery system. *Ann Noninvasive Electrocardiol* 2022;27:e12888. doi:10.1111/anec.12888

5. Ayad RF, Assar MD, Simpson L, Garner JB, Schussler JM. Causes and management of drug-induced long QT syndrome. *Proc (Bayl Univ Med Cent)* 2010;23:250-255. doi:10.1080/08998280.2010.11928628
6. Khan IA. Long QT syndrome: diagnosis and management. *Am Heart J* 2002;143:7-14. doi:10.1067/mhj.2002.120295
7. Thomas SHL, Behr ER. Pharmacological treatment of acquired QT prolongation and torsades de pointes. *Br J Clin Pharmacol* 2016;81:420-427. doi:10.1111/bcp.12726
8. Moss AJ, Liu JE, Gottlieb S, Locati EH, Schwartz PJ, Robinson JL. Efficacy of permanent pacing in the management of high-risk patients with long QT syndrome. *Circulation* 1991;84:1524-1529. doi:10.1161/01.cir.84.4.1524
9. Overbey AN, Austin A, Seidensticker DF, Lin AH. Overdrive pacing in a patient with incessant torsades de pointes. *BMJ Case Rep* 2013;2013:bcr2013200146. doi:10.1136/bcr-2013-200146
10. Zhang Y, Wang X, Pan Y, Du B, Nanthakumar K, Yang P. Overdrive pacing in the acute management of osimertinib-induced ventricular arrhythmias: a case report and literature review. *Front Cardiovasc Med* 2022;9:934214. doi:10.3389/fcvm.2022.934214
11. Erimin 5. Solaceasia.org. Available at: https://www.solaceasia.org/substance-rehab/erimin-5. Accessed March 5, 2023.
12. Ziegenbein M, Kropp S. Lorazepam-induced prolongation of the QT interval in a patient with schizoaffective disorder and complete AV block. *Can J Psychiatry* 2004;49:414. doi:10.1177/070674370404900619