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Efficacy of prescribing Inderal for polymorphic ventricular tachycardia in a young patient with the normal QT interval: A case report study

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Abstract

Polymorphic ventricular tachycardia (PVT) is a group of life-threatening heart rhythm disorders. These arrhythmias share similar electrocardiographic characteristics but require different modes of therapy for effective treatment. It is important to note that the medications that are considered the first-line treatment for one type of PVT may not be appropriate for another type, and may worsen the condition. Therefore, it is crucial to accurately diagnose the type of PVT before initiating treatment to provide the most effective therapy for the patient. A 42-year-old man was admitted to the emergency department with dyspnea, Levine sign, and severe chest pain. His electrocardiogram showed ST elevation, and the QT interval was normal. The patient was sent to the cath lab based on the treatment protocols. According to the results of angiography, three coronary arteries were severely obstructed. His coronary arteries did not open during percutaneous coronary intervention; thus, the healthcare team decided on open heart surgery. He suffered from recurrent PVT following open heart surgery and did not respond to any of the drugs suitable for this type of tachycardia. Inderal prevented the recurrence of ventricular tachycardia (VT) in a patient with polymorphic VT without QT prolongation, contrary to the healthcare team's expectations. Inderal was used as the last line of treatment because this patient's arrhythmia was polymorphic VT without QT prolongation. Inderal is typically used for treating VT in patients with long QT syndromes and heart structural disorders. This case report aims to highlight the impact of Inderal on polymorphic tachycardia, specifically in cases where the QT interval is not elongated. In this particular case, the standard treatment approaches were ineffective in preventing reversibility, but Inderal proved to be successful. Therefore, we feel it is important to document and share this case.

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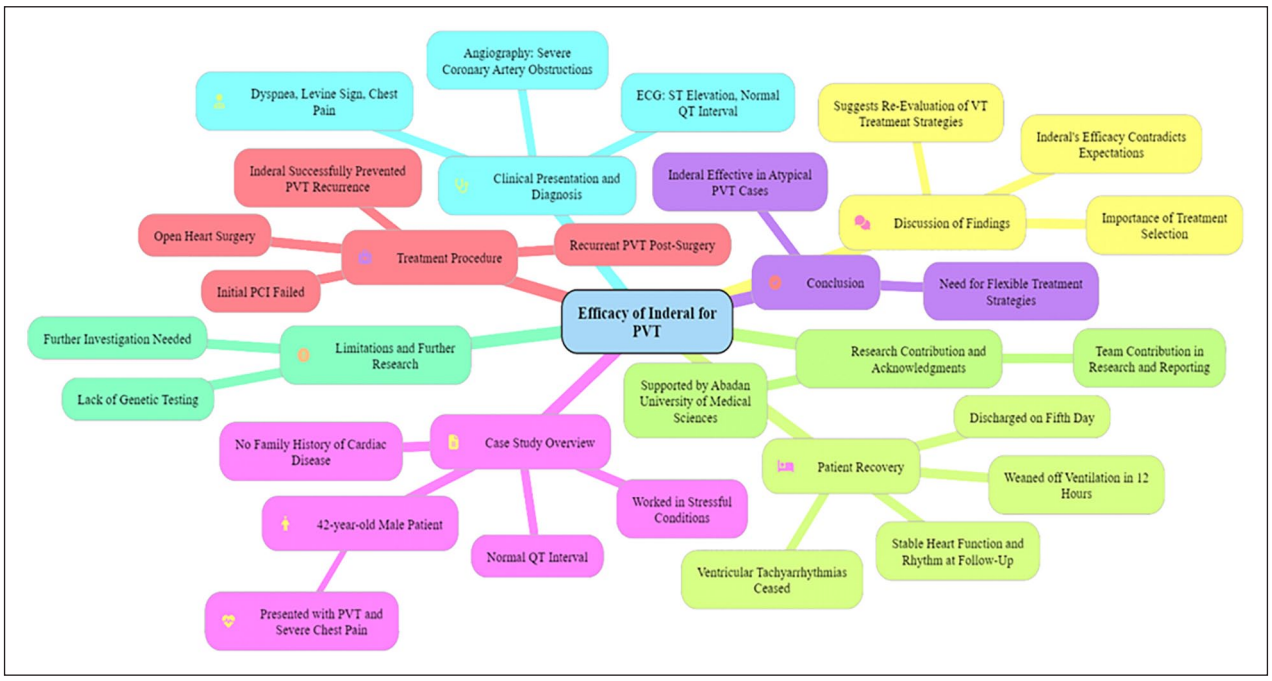
Graphical abstract



Keywords
Open heart surgery, ventricular tachycardia, antiarrhythmic drugs, Inderal, non-structural heart diseases

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Introduction

Ventricular tachycardia (VT) has a destructive effect on people's health and hearts and is a major cause of mortality among patients with cardiovascular diseases. VT leads to ventricular fibrillation (VF) and cardiac arrest.[1] The main causes of VT include cardiac ischemia, structural heart diseases, cardiomyopathy, digoxin toxicity, hypertension, and electrolyte imbalance.[2,3] Hypokalemia and hypomagnesemia are among the most common electrolyte disorders in heart surgery that may lead to VT.[3,4] The clinical manifestations of VT depend on the patient's age, structural heart disease, and the mechanism of VT. Its common symptoms consist of palpitations, dyspnea, chest pain, and syncope.[5] The heart rate in VT arrhythmia is more than 100 bpm; the QRS complex is wide, and the speed of each impulse in this arrhythmia is greater than 120 ms.[6] The classification of VT is based on multiple criteria, including the morphology of the electrocardiogram (ECG), duration, and hemodynamic status of the patient. These factors are crucial in determining the appropriate treatment for the condition. It is essential to consider all the parameters while evaluating VT to ensure an accurate diagnosis and develop an effective management plan.[7] VT can be classified into two types based on the morphological features of the QRS complex: monomorphic and polymorphic. Monomorphic VT (MVT) is characterized by a broad complex rhythm with a QRS duration of over 120 ms that remains uniform from beat to beat. It originates from a single focus and lacks any association between the P and QRS waveforms. Polymorphic ventricular tachycardia (PVT) is a type of heart rhythm disorder that presents with irregular beat-to-beat variations. Nonsustained VT occurs for less than 30 s, while sustained VT persists for more than 30 s.[7,8] PVT is a serious VT that causes a changing QRS pattern. Identifying the cause is crucial because different types of VT can show similar characteristics, and they need to require altered treatments. Figure 1 offers a classification and a step-by-step approach to differentiate these dangerous arrhythmias.[9] The treatment of VT is always selected based on the type of VT, the primary cause, and the injury.[10] Pharmacotherapy for VT is highly sensitive and is chosen according to the patient's condition.[11] Pharmacological treatment of VT is administered by using various drug classes, including beta-blockers, calcium blockers, potassium blockers, and other drug classes available in the guidelines.[12,13] According to studies, beta-blockers are one of the most important drug classes for treating and controlling VT.[4] Inderal (propranolol) is a beta-blocker used to treat ventricular arrhythmias, including VT.[14] By impacting the cardiac beta-adrenergic receptors, propranolol reduces heart rate,

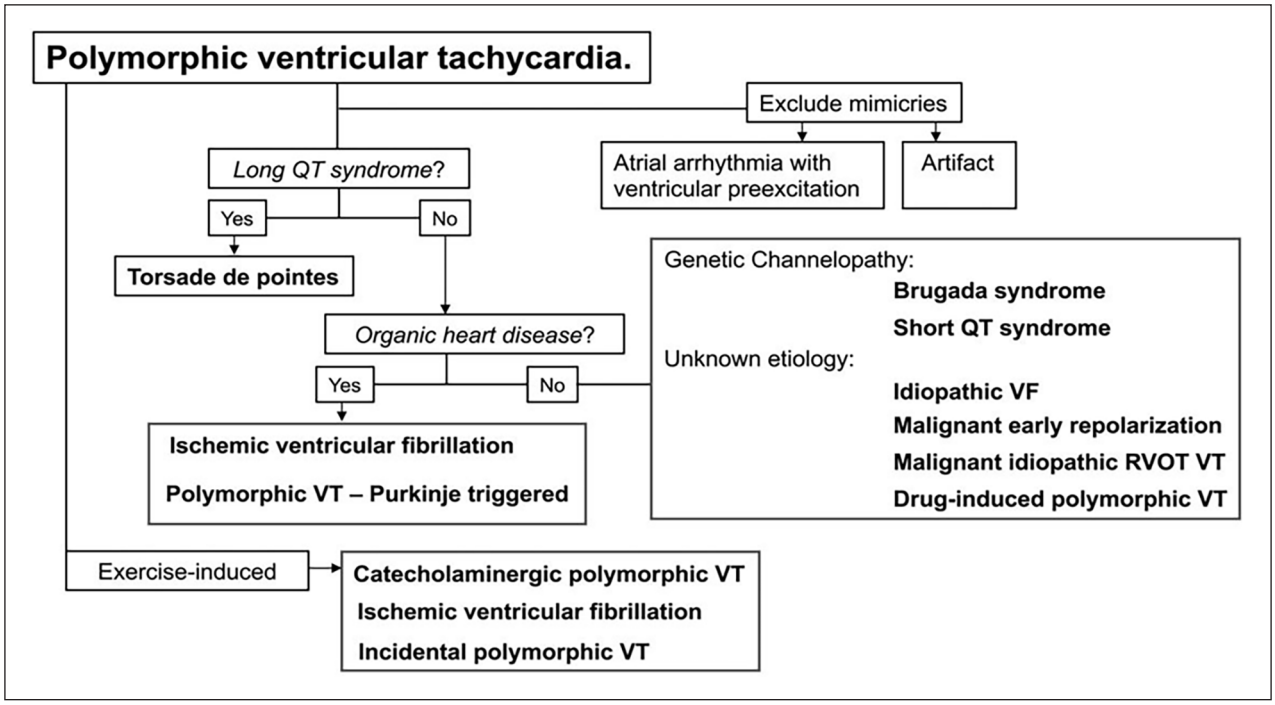

Figure 1. A step-by-step practical approach for distinguishing PVT and other malignant arrhythmias.
PVT: polymorphic ventricular tachycardia.

decreases bradycardia, and corrects tachycardia arrhythmias. It should be noted that Inderal has demonstrated fewer life-threatening arrhythmic events (LTAEs) compared to other beta-blockers.[15] This study presents a case of PVT in a young patient with no significant family history and without QT prolongation. The patient's arrhythmia was unresponsive to other treatments but successfully managed with Inderal.

Case presentation

A 42-year-old man was admitted to the emergency department with dyspnea, Levine sign, and severe chest pain. His vital signs at admission were as follows: body temperature (*T*): 36.7, respiratory rate (RR): 30, pulse rate (PR): 138, blood pressure (BP): 140/80, and oxygen saturation (SPO_2): 94%. A history was taken from the patient at the time of admission, and he did not report any underlying disease. In the history of medicine, working conditions were hard-hitting, and the job was considered one of the most challenging. After conducting a comprehensive analysis of the patient's medical and familial history, it has been concluded that there are no indications of any genetic ailments or dysfunctions in his history. Additionally, the QT interval falls within the standard limit. Based on these outcomes, it can be inferred that there are no hidden genetic elements that may be influencing the patient's current health status. His body mass index (weight 74 kg, height 178 cm) was normal, and he had a history of working in difficult and stressful conditions. An ECG was taken, which showed ST elevation, and the QT interval was normal. Therefore, code 247 was announced.[16,17] Physical examinations of the heart and lungs were performed, and the tests (complete blood count, prothrombin time, partial thromboplastin time, high-density lipoproteins, low-density lipoproteins, troponin, cholesterol, aspartate aminotransferase, alanine aminotransferase, triglyceride, arterial blood gas analysis, and international normalized ratio) were sent to the laboratory of the hospital to check the patient's clinical condition. Based on the results received from the laboratory, his troponin was positive, and the other test results were in the normal range. After we performed a physical examination and checked the test results, the patient underwent echocardiography. In the echocardiography, the heart valves functioned well, and prolapse was not observed. The pericardium and myocardium had normal thickness, and pericardial effusion was not seen. The center had angiography equipment; therefore, the patient was sent to the cath lab based on the treatment protocols. According to the results of angiography, three coronary arteries (right coronary artery, diagonal branch 1, Circumflex) were severely obstructed (respectively 84%, 94%, and 89%). His coronary arteries did not open during percutaneous coronary intervention; thus, the healthcare team decided on open heart surgery. The patient's pump time was 40 min; during the surgery, the amount of bleeding was normal, and the bleeding time was within the acceptable range. After the operation, the patient was admitted to the cardiac surgery intensive care unit (C-ICU). Following the heart surgery, the patient was monitored. CBC and electrolyte tests were also performed. The

patient underwent an echocardiogram after the heart surgery, and there were no changes in his ejection fraction (EF) status (EF = 55).[18] The patient's postoperative vital signs were as follows: PR: 85, *T*: 36.6, BP: 90/50, SPO_2: 100. After the operation, the patient was connected to a ventilator. The ventilator setup consisted of the mode: SIMV, FiO_2: 90%, PEEP: 5, and VT: 500. His electrolyte levels were monitored, and they were all within the normal range. He received a TNG infusion. After 1 h, he had R on *T* arrhythmia and, immediately afterward, polymorphic VT. Following the protocol, the treatment team started resuscitation and administered shocks (200 J).[19,20] With the start of resuscitation, the patient received the shock and cardiac massage, and his rhythm was normalized. After 2 min, R on *T* arrhythmia occurred again, and he suffered from polymorphic VT immediately. Therefore, drug treatments were started in addition to DC shock treatment. The first dose of amiodarone (300 mg bolus)[21] was injected; later in the resuscitation process, the amiodarone dose was reduced to 150 mg bolus. However, the R on *T* occurred again sometime later, followed by polymorphic VT. The healthcare team quickly started the shock (200 J) and lidocaine (1 mg/kg),[21] which was 74 mg bolus based on the patient's weight. As there was no result, during the mentioned process and the reoccurrence of VT, the healthcare team started the infusion of amiodarone (1 mg/min)[21] with the start of the shock. After several shocks, The recurrence process did not stop. In other words, the patient's arrhythmia could only be controlled by giving a shock, and common drug treatments could not prevent the polymorphic VT. Consequently, the cardiologist decided to administer Inderal (3 mg bolus)[21] to control continuous VTs after the other prescribed drugs failed. After receiving Inderal, the patient returned to normal sinus rhythm, the R on *T* arrhythmia, and frequent VTs stopped. After administering Inderal, echocardiography, and C-xray were taken from the patient again. His EF was 55, and there were no signs of cardiac tamponade or bleeding in the pleural area. The patient received post-CPR care due to receiving multiple shocks (21 times). He was weaned off the ventilator after 12 h. He was cared for in the cardiac surgery department for 48 h and in the post-ICU for 48 h. After completing the necessary training, which included guidance on resuming daily activities, monitoring daily activity levels, proper nutrition, self-care practices, and recognizing symptoms of relapse, the patient was discharged on the fifth day. then He visited the doctor at the outpatient department for an examination and follow-up. The doctor found that the patient's EF was 55%, indicating a favorable physical condition. Furthermore, as per the medical evaluation conducted during the patient's visit, the ECG revealed no abnormalities or notable findings. In addition, the patient's medication dosage was reassessed to ensure optimal efficacy and safety. The patient's condition remained stable, and he diligently followed the postoperative treatment process. Please refer to Figure 2 for an overview of the patient's therapeutic steps.

Discussion

According to the studies, there are two major types of VT: (1) MVT, which can cause VF and occurs when the structure of the heart is disturbed or ischemia has occurred, and (2) PVT occurs in heart ischemia, long QT interval, and hearts with healthy structures.[11] PVT is a concerning disturbance in heart rhythm that can result in fainting or cardiac arrest. It is critical to determine the root cause, as varying types of arrhythmias may necessitate diverse treatments. In this article study, we provide a contemporary classification of PVT and a helpful guide to assist in diagnosing and treating these hazardous heart rhythms. Arrhythmias can be categorized based on their mechanism, symptoms, and ECG changes. The PVT category includes VT with long QT syndromes, VT without QT prolongation, and it results from exercise. In a recent case study, a patient presented with PVT without QT prolongation. The recommended routine treatment in people who develop VT mainly includes sodium channel blockers, verapamil, and amiodarone. According to studies, beta-blockers are suitable drugs to affect VT with long QT syndromes. However, in this study, we observed that this class of drugs affects the VT without QT prolongation.[9,22–26] The short QT syndrome is typically genetic and may be observed in other family members. A noteworthy aspect is a history of sudden death in the person's family. In our study, there were no records of such occurrences. This type of arrhythmia usually appears at a young age, with atrial fibrillation being the most common symptom. At a younger age, it can present as VF. Based on the family history, this diagnosis was rejected.[27,28] The report highlights an unexpected reaction of the patient's heart rhythm to Inderal. The presented patient did not have a myocardial infarction, and no premature ventricular contractions arrhythmia was observed during his treatment process. This patient suffered from ischemia and heart injury, which could be the cause of VT, but the heart was structurally and functionally healthy. In addition to DC shock treatment, antiarrhythmic drugs were administered, which are one of the most important and oldest methods of preventing recurrence and treating VTs.[29] Before the development of ICD (implantable cardioverter-defibrillator), many studies revealed that beta-blockers, such as propranolol, play a vital role in preventing recurrent attacks. Note that this class of drugs is mainly used to correct arrhythmia in patients with a defective heart structure.[29–31] Due to the structurally and functionally healthy status of the patient's heart and the without QT prolongation ventricular tachycardia, the healthcare team chose Inderal as the last line of treatment. Contrary to the limitations of using beta-blockers in general arrhythmias, this drug is quite useful in this special condition.[29] After administering Inderal, the process of VT stopped and was prevented from recurring. In the case described herein, the healthcare team administered Inderal as the last line of treatment because there was no indication to use Inderal. Contrary to the expectation of the healthcare team, Inderal prevented the recurrence of VT in a patient

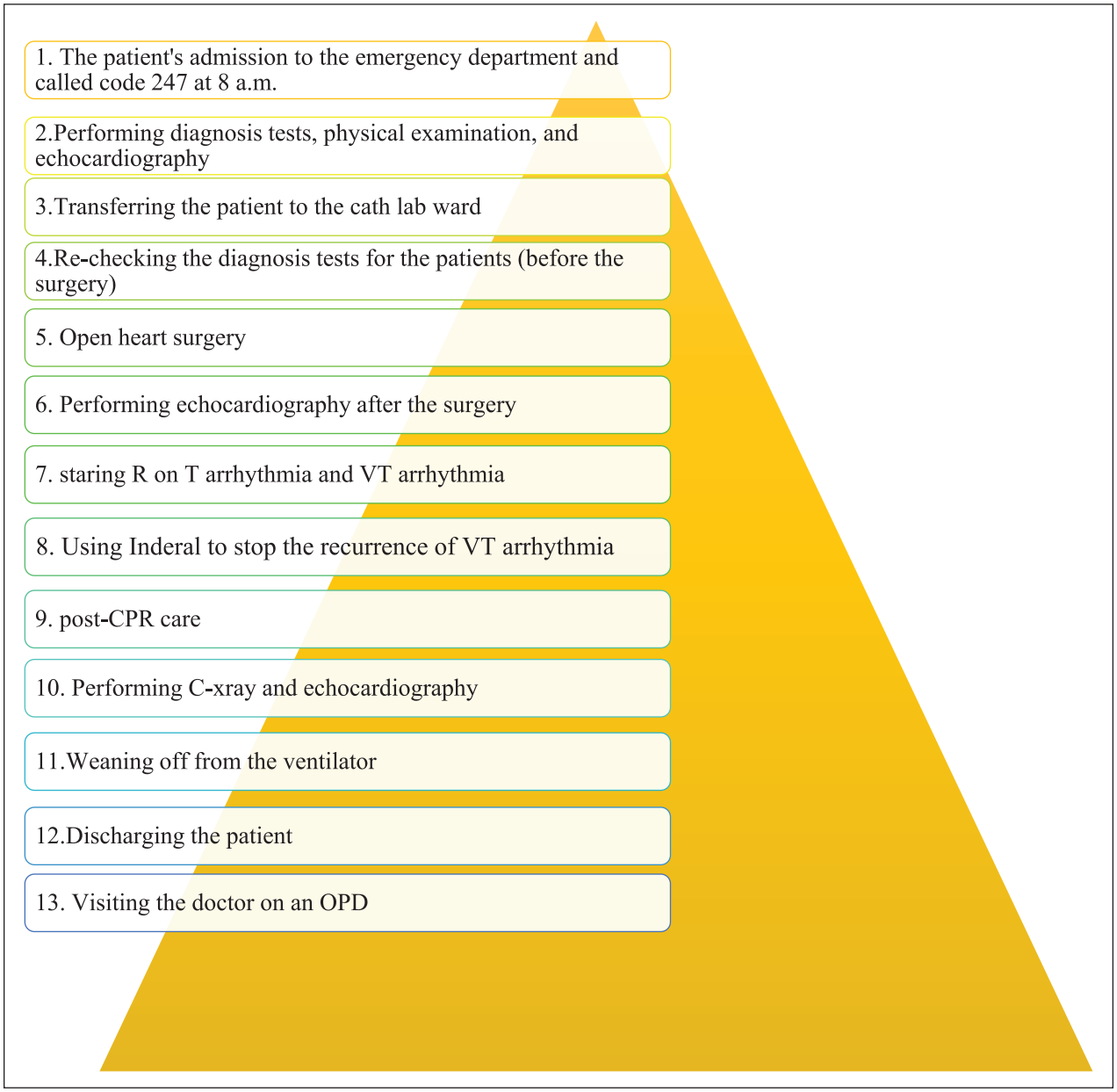

Figure 2. Summary figure.

with polymorphic VT. According to a study conducted by Mazzanti et al., beta-blockers can lead to various complications and outcomes, including LTAEs, which occur in about 15% of patients receiving this class of drugs. Propranolol, a beta-blocker, can have counterintuitive effects on the heart by blocking $Na_V1.5$ channels. It shortens the action potential duration in the endocardium while hyperpolarizing the membrane by activating the dominant transient outward current in the epicardium. Figure 3[,31] indicates the effect of Inderal on the cardiac action potential. It is noteworthy that emotional and exercise-induced syncope can impede beta-blocker therapy and heighten the risk of LTAEs. Hence, while using beta-blockers to manage PVT can be helpful, it is crucial to consider the potential side effects of a particular medication or treatment. These side effects can considerably affect a patient's health and well-being and must be thoroughly evaluated before making any decisions.[15,25,32] The main objective of this report is to raise sensitivity and awareness among hospital staff regarding the treatment of patients. It aims to minimize trial and error and facilitate correct and immediate diagnosis, particularly in cases where patients' lives are at risk.

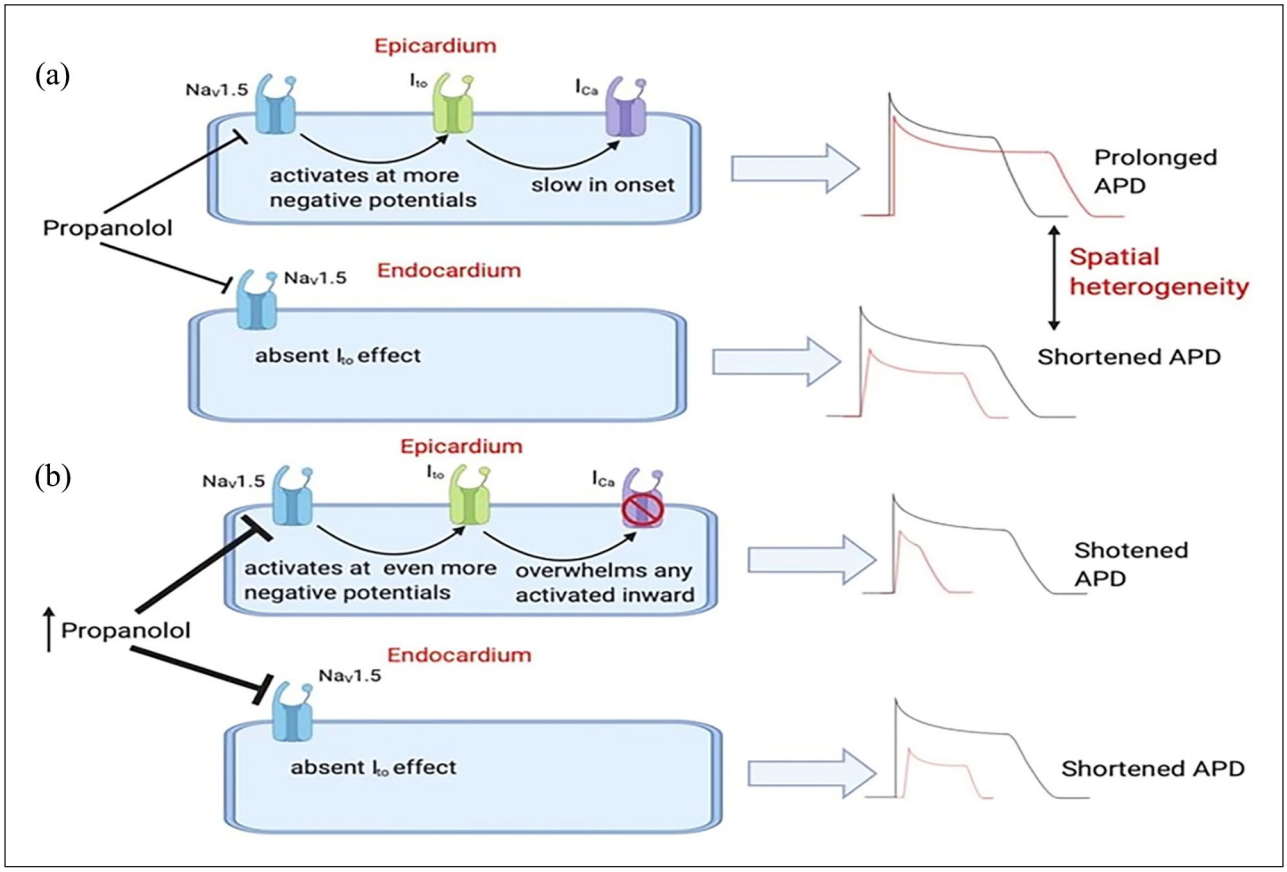

Figure 3. Propranolol's $Na_V1.5$ blocking effects can paradoxically affect the heart. (a) In the endocardium, it shortens the action potential duration (APD). (b) In the epicardium, it hyperpolarizes the membrane by opening the dominant transient outward.[32]

Conclusion

According to the mentioned references, Inderal is mainly used in MVT and patients with heart structure disorders. The result of the present study demonstrates the effect of the anti-arrhythmic drug Inderal on polymorphic VT.

Limitation

In this study, there was no evidence of genetic disease in the patient's medical history, and due to the limitations of the patient's treatment and follow-up, we could not investigate genomic tests. Therefore, further investigations are needed.

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Author contributions

M.S., E.M., J.S., R.T., Z.N., N.T., M.A.N., A.r.B., and A.v.B.: conceptualization; data curtain; formal analysis; investigation; project administration; resources; supervision; roles/writing – original draft. F.B.: cooperate in answering the reviewer's comments.

Availability of data and material

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethics approval

Ethical approval to report this case was obtained from "the Abadan University of Medical Science ethical board (IR.ABADANUMS.REC.1402.077)."

Informed consent

Written informed consent was obtained from the patient for his anonymized information to be published in this article. He was cognizant of the objectives of the study. Also, he was informed that his participation was voluntary, and he had the liberty to

withdraw from the study at any instance. We have assured the patient that his participation in this research is entirely voluntary. Choose or not participate with his. If you do not participate in all the services received in this center, you will continue and nothing will change.

Consent for publication

Not applicable.

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