


Ventricular Tachycardia in Anteroseptal STEMI

Luzelia Marta Sequeira Saldanha
Medical Doctor at Leona Kefamenanu Hospital, Timor Tengah Utara,
East Nusa Tenggara, Indonesia

ABSTRACT

Introduction: Fatal ventricular tachyarrhythmias such as ventricular tachycardia (VT) or ventricular fibrillation (VF) are the most frequent cause of sudden cardiac death. In-hospital mortality of VT in STEMI was a 4- to 5-fold increase compared to STEMI without ventricular tachyarrhythmia; VT and VF were also initially thought to be the most common causes of out-of-hospital cardiac arrest. Late VT (occurs beyond 24-48 hours of STEMI onset) is a predictor for worse prognosis. **Case**: A 49-year-old female presented with continuous chest pain for 5 days and a rapid heartbeat (palpitation) for 4 days before hospital admission. Initial examination revealed VT and ST elevation on ECG, leading to a diagnosis of anteroseptal STEMI with VT. Treatment included aspirin, clopidogrel, heparin, isosorbide dinitrate, bisoprolol, and simvastatin. The VT resolved about 2 hours after the initial treatment, and the evolution of STEMI (on ECG) was observed. **Conclusion:** Early recognition and prompt treatment for VT patients can prevent hemodynamic instability and reduce mortality.

Keywords: STEMI, sudden cardiac death, ventricular tachycardia.

ABSTRAK

Pendahuluan: Takiaritmia ventrikel yang fatal di antaranya takikardi ventrikel (*ventricular tachycardia*/VT) atau fibrilasi ventrikel (*ventricular fibrillation*/VF) adalah penyebab tersering kematian jantung mendadak. Angka kematian di rumah sakit pada VT pada STEMI meningkat 4 hingga 5 kali lipat dibandingkan dengan STEMI tanpa aritmia ventrikular; VT dan VF juga awalnya dianggap sebagai penyebab paling umum dari henti jantung di luar rumah sakit. VT *onset* lambat (terjadi 24–48 jam setelah *onset* STEMI) adalah prediktor prognosis buruk. **Kasus**: Wanita berusia 49 tahun dengan nyeri dada terus-menerus selama 5 hari dan rasa berdebar-debar (palpitasi) selama 4 hari sebelum masuk rumah sakit. Pemeriksaan awal menemukan VT dan elevasi ST pada EKG, yang mengarah pada diagnosis STEMI anteroseptal dengan VT. Pengobatan meliputi *aspirin, clopidogrel, heparin, isosorbide dinitrate, bisoprolol,* dan *simvastatin*. VT teratasi sekitar 2 jam setelah pengobatan awal, dan evolusi STEMI (pada EKG) terlihat. **Simpulan**: Pengenalan dini dan pengobatan segera pada pasien VT dapat mencegah ketidakstabilan hemodinamik dan mengurangi mortalitas. **Luzelia Marta Sequeira Saldanha. Takikardia Ventrikel pada STEMI Anteroseptal.**

Kata Kunci: STEMI, kematian jantung mendadak, takikardi ventrikel.

INTRODUCTION

According to the National Health Survey of Indonesia, the prevalence of heart disease in 2018 was 1.5%, increased from 0.5% in 2013.[1] The American Heart Association (AHA) 2022 heart disease and stroke statistical update stated that globally in 2020, 244.1 million people were living with ischemic heart disease (IHD), and the mortality rate was 112.37 per 100,000.[2] Data on ST-elevation myocardial infarction (STEMI) prevalence in Indonesia are still limited.[3] The Jakarta Cardiovascular Care Unit Network System in 2014 found 2,090 acute coronary syndrome (ACS) patients from 2008 to 2011; 1,505 were STEMI and 585 were non-STEMI.[3] About 5% to 10% of acute coronary syndrome (ACS) patients have ventricular arrhythmias.[4] STEMI with late presentation (more than 12 hours) or STEMI patients without reperfusion therapy, either PCI or fibrinolytic therapy, was significantly associated with high mortality.[3]

About 50% of deaths in IHD are due to sudden cardiac death.[5] Fatal ventricular tachyarrhythmias such as ventricular tachycardia (VT) or ventricular fibrillation (VF) are the most frequent cause of sudden cardiac death.[4,6] The most common cause of fatal ventricular tachyarrhythmias is ischemic heart disease in an acute or chronic stage.[5] Compared to STEMI, sustained ventricular arrhythmias are less common in acute unstable angina or NSTEMI.[6] The incidence of VF or VT in the STEMI setting was 2.1%-0.8% VT, 1% VF, and 0.3% was a combination of VT and VF[6]—while 80%-90% of ventricular arrhythmias in STEMI occur in the first 48 hours.[6,7]

Ventricular tachycardia is a tachyarrhythmia characterized by a wide QRS complex ≥120 ms with ≥3 consecutive heart rates of >100 beats per minute.[4,8] Based on hemodynamic stability and duration, ventricular tachycardia is divided into sustained and non-sustained types. Sustained VT is ventricular tachycardia that lasts longer than 30 seconds in duration or causes hemodynamic instability in

Alamat Korespondensi *email: uze_saldanha_46@yahoo.com*

less than 30 seconds; non-sustained VT is ventricular tachycardia that lasts less than 30 seconds in duration.[4,6,8] Based on morphology, VT is divided into monomorphic and polymorphic, and based on mechanism, VT is divided into re-entry mechanism, triggered activity mechanism, and abnormal automaticity mechanism.[4,6,8] Besides coronary artery disease as the main cause of VT, other etiologies of VT are ischemic or non-ischemic dilated cardiomyopathy, inherited cardiac channelopathies, adult and congenital structural heart disease, infiltrative cardiomyopathy, digitalis toxicity, and electrolyte imbalance (hypocalcemia, hypokalemia, and hypomagnesemia).[4,6,9] In-hospital mortality of VT in STEMI was a 4- to 5-fold increase compared to STEMI without ventricular tachyarrhythmia; VT and VF were also initially thought to be the most common causes of out-of-hospital cardiac arrest.[10] Late VT (occurs beyond 24-48 hours) is a predictor for worse prognosis.[6]

CASE

A 49-year-old female presented to the emergency unit with continuous chest pain for the last 5 days. The pain was described as heaviness in the substernal chest spreading to the left arm accompanied by nausea and diaphoresis. Chest pain was experienced since 5 days before hospital admission and rapid heartbeat/palpitation on the morning of the day after. No past history of chest pain. Hypertension was diagnosed 2 years ago during routine clinic check-ups, but no medication was taken. The highest recorded blood pressure was 170/100. She had no diabetes or any other comorbidities. No family history of heart diseases or sudden cardiac death. No contraceptive medication used and no smoking history. No history of blackout. On admission the patient was compos mentis; the vital signs were blood pressure 130/110 mmHg, heart rate 196 beats per minute, respiratory rate 20 breaths per minute, oxygen saturation 98% on room air, and a pain score of 6 out of 10. Further examinations revealed regular cardiac rhythm, normal breath sounds, and the absence of jugular venous distension, murmurs, or peripheral edema. An immediate 12-lead electrocardiography (ECG) showed ventricular tachycardia (monomorphic) in all leads and ST elevation in leads V1-V4 **(Figure 1)**, leading to the diagnosis of anteroseptal STEMI with ventricular tachycardia. The results of the blood analysis were leukocytes 13,690/µL, random blood glucose 166 mg/dL, and no electrolyte abnormalities. The diagnosis was anteroseptal STEMI with ventricular tachycardia.

The patient was loaded with oral 300 mg aspirin and oral 300 mg clopidogrel, followed by oral bisoprolol 5 mg, oral simvastatin 20 mg, subcutaneous unfractionated heparin 5,000 IU, and sublingual isosorbide dinitrate 5 mg. Amiodarone was not given because the patient already had sinus rhythm, albeit still tachycardia **(Figure 2)**. The patient

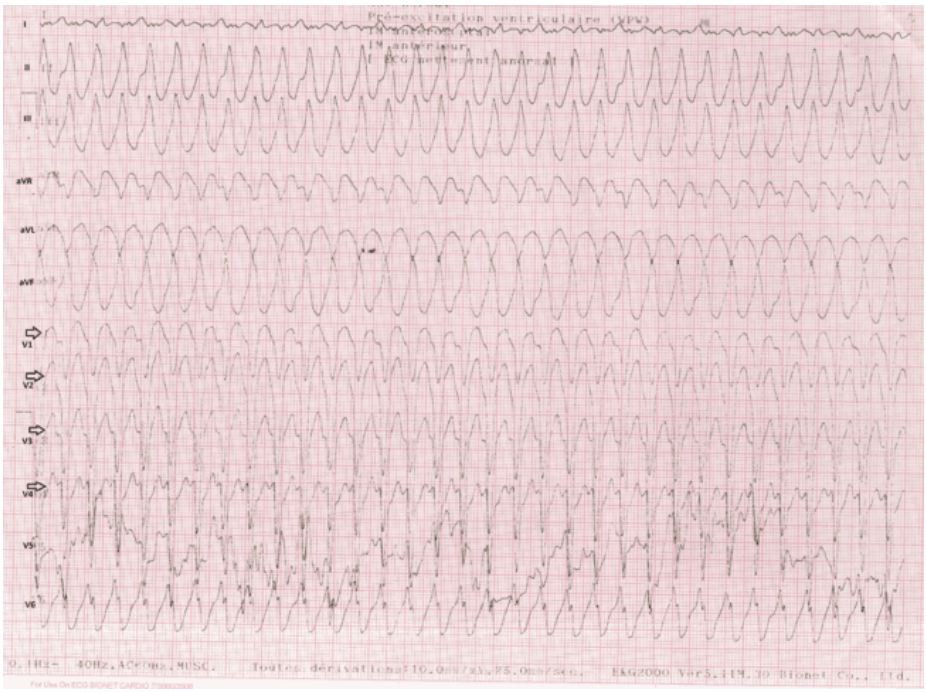

Figure 1. ECG on admission. VT monomorphic with 197 bpm, ST elevation (arrows) in leads V1-V4.

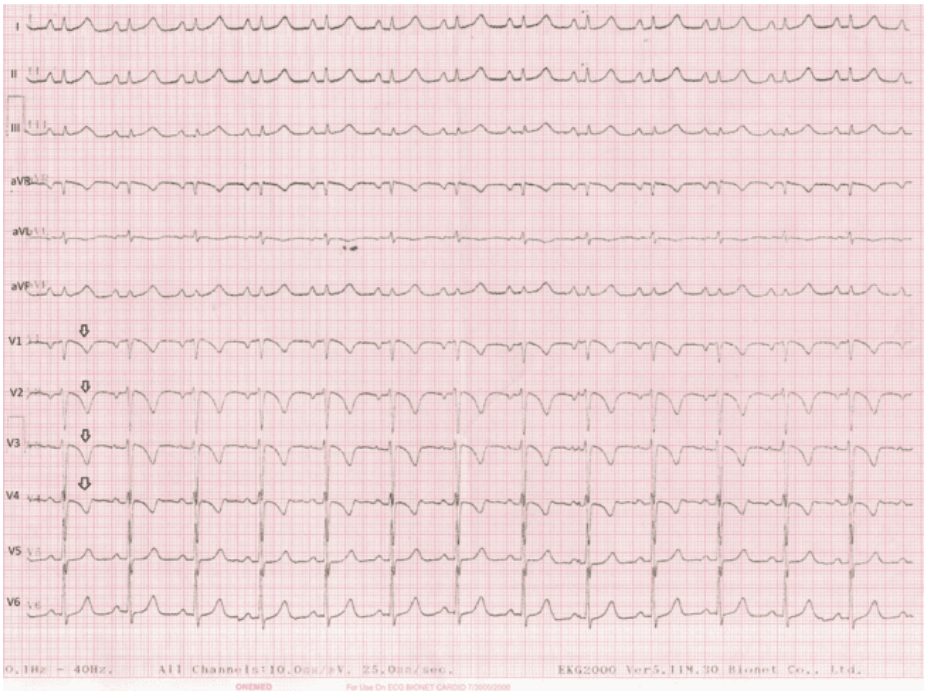

Figure 2. ECG about 2 hours after treatment without amiodarone. Sinus tachycardia 121 bpm, normo-axis, T inverted (arrows) in leads V1-V4 (STEMI evolution).


was transferred to ICU because ICCU is not available. About 2 hours after treatment initiation, the VT was resolved, and the ECG already showed sinus tachycardia with STEMI evolution (T inversion in leads V1-V4). Blood pressure was 121/87 mmHg, heart rate was 121 bpm, respiratory rate was 22 breaths per minute, and the pain score was 3 out of 10. The treatment was continued with an oral aspirin maintenance dose of 80 mg once a day, oral clopidogrel 75 mg once a day, oral bisoprolol 5 mg once a day, subcutaneous unfractionated heparin 5,000 IU twice a day, oral simvastatin 20 mg once a day, and oral isosorbide dinitrate 5 mg thrice a day.

After 12 hours, the ECG showed sinus rhythm with STEMI resolution **(Figure 3)**. The hemodynamics were stable: blood pressure 121/77 mmHg, heart rate 71 bpm, respiratory rate 19 breaths per minute, and no heart-racing feeling. The chest pain was still at a score of 3 out of 10. The patient was observed for 3 days in the ICU and 3 days in a regular room. On day 5 the patient had hypertension gr II after two measurements (blood pressure 140/100 mmHg, 160/90 mmHg), so lisinopril (ACE inhibitor) 5 mg oral once a day was added. The patient was discharged on day 7 with no chest pain, no sensation of rapid heartbeat (palpitation), blood pressure of 130/70 mmHg, heart rate of 68 bpm, respiratory rate of 18 per minute, and an ECG that showed sinus rhythm, 65 bpm, normo-axis, and T inversion in leads V1-V3 **(Figure 4)**. The treatment was once-daily aspirin 80 mg, clopidogrel 75 mg, bisoprolol 2.5 mg, lisinopril 5 mg, simvastatin 20 mg, and isosorbide dinitrate 5 mg.

DISCUSSION

Approximately 90% of VT occurs in patients with structural heart disease, and 10% occurs in patients with no structural heart disease.[8] VT during myocardial infarction (MI) is more likely in patients with previous MI.[11] VT in STEMI was mostly with the re-entry mechanism (75%), and 25% was triggered by the activity mechanism.[12] Re-entry mechanism is when electrical impulses repeatedly circulate through the heart tissue or electrical impulses continuously loop through a circuit within the heart tissue, causing rapid and abnormal heartbeats.[12] In the ischemic myocardium, the normal pathways are disrupted, leading to this re-entry of impulses.[12] Triggered activity mechanism is when abnormal impulses are generated in the heart cells due to afterdepolarizations, abnormal electrical activities following normal heartbeats.[12] The onset and size of ischemia during STEMI are related to the occurrence of ventricular arrhythmias.[12] A previous study found that 90% of ventricular arrhythmias occur during the first 24 hours after STEMI onset, 3.5% during 24 to 48 hours, and 7.5% after 48 hours.[11] If VT occurs during the first 24 hours after experiencing MI, it's classified as early VT. Early VT is divided into immediate, if it occurs 10 to 30 minutes after experiencing MI, and delayed, if it occurs 6 to 24 hours after experiencing MI. Late VT is described as VT occurring 24 to 48 hours after MI, and chronic VT occurs after 48 hours.[11] In this case, based on the patient's symptoms, VT occurred within

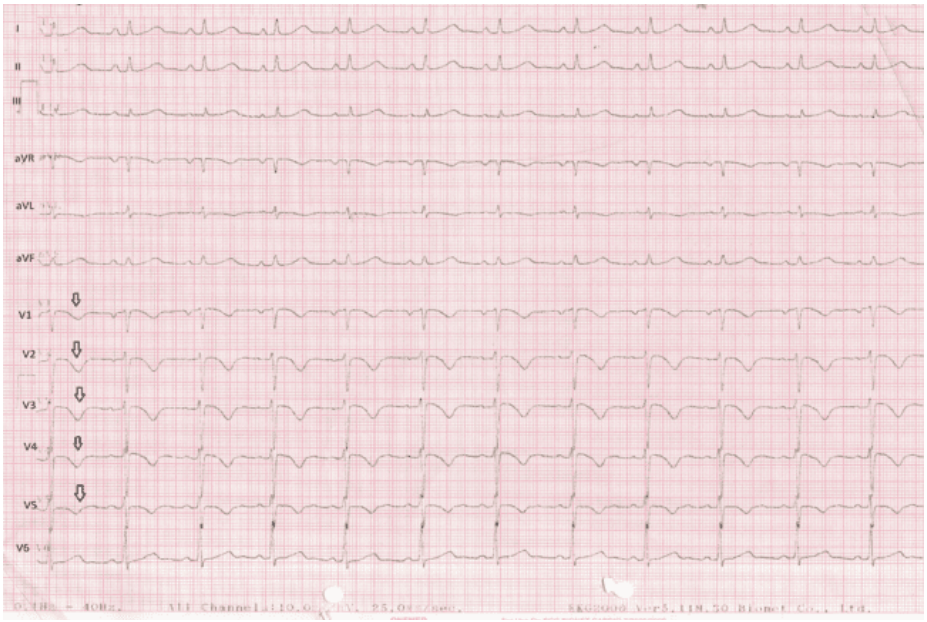

Figure 3. ECG 12 hours after admission. Sinus rhythm 71 bpm, normo-axis, T wave inversion (arrows) in leads V1-V5.

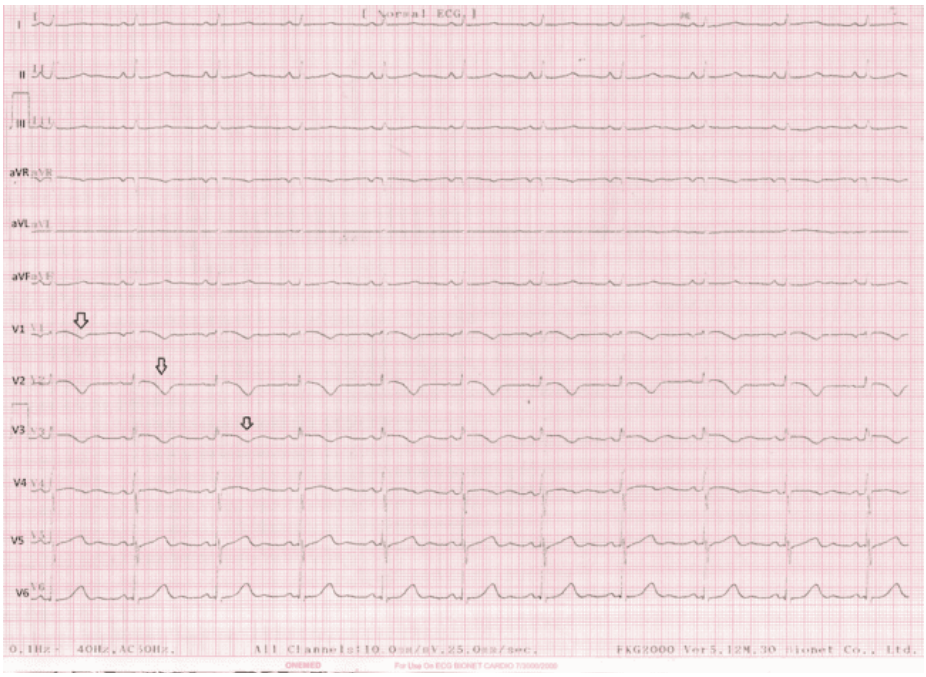

Figure 4. ECG on day 7 after admission, before hospital discharge. Sinus rhythm 65 bpm, normo-axis, and T wave inversion (arrows) in leads V1-V3.

the first 24 hours of STEMI, but the exact time of occurrence cannot be confirmed because it happened outside the hospital. Based on the timing of the arrhythmia, this patient was in the delayed phase (6 to 24 hours) after the onset of MI, as the patient reported feeling palpitations in the morning following continuous chest pain the previous evening (assuming evening to morning is less than 24 hours). In the myocardial infarction (MI) setting, early-phase arrhythmias are usually non-sustained ventricular tachycardia (VT).[6] However, if the arrhythmias occur in the chronic phase (more than 48 hours after the MI onset), they are typically sustained monomorphic VT (SMVT).[6]

Infarct scar tissue induced ventricular tachycardia by setting the stage for a re-entry circuit for the re-entry mechanism; the newly ischemic areas can create a stable re-entry circuit, producing VT.[13] Ventricular arrhythmias during the first 10 minutes of occlusion (in myocardial infarction) are due to the re-entry mechanism within the ischemic myocardium; the size and location of the re-entrant (in the re-entry mechanism) or the specific re-entry area can vary from beat to beat due to the dynamic nature of the ischemic damage, causing irregular and polymorphic VT that generates to ventricular fibrillation.[13,14] The mechanism of the delayed phase of spontaneous VA (4 hours to 3 days after MI onset) also included automaticity in Purkinje fibers that facilitated the rise of the re-entry mechanism by localized indirect conduction block and triggered activity due to delayed after-depolarization.[13] This case presented monomorphic VT at the time of admission, which was 4 days after chest pain (MI) onset. The assumption is that VT had already occurred for more than 4 days when the patient began to feel a continuous rapid heartbeat.

The management of STEMI aims to reduce mortality and to prevent permanent cardiac injury.[13] Delayed therapy for STEMI is less effective.[15] Unfractionated heparin (UFH) is one of the anticoagulant choices in STEMI treatment and a standard therapy for ACS patients.[16] UFH was found to significantly reduce recurrent events of ischemia in ACS.[16] A retrospective observational study found that prehospital UFH treatment in STEMI was associated with significant long-term survival improvement.[17] A randomized trial comparing bivalirudin and UFH found that the rate of major adverse cardiovascular events was not different in the two groups.[18] A case-control trial found that administering UFH in the emergency room before PCI in STEMI patients was associated with a higher thrombolysis rate in grade 2 myocardial infarction (TIMI) and required a smaller diameter and shorter stent length as compared to the control group.[19]

For patients presenting late or considered ineligible for reperfusion therapy, a systematic review of randomized trials found that in the absence of aspirin, UFH reduced mortality (11.4% vs 14.9% in the control group, RRR: 25 ± 8%, 95% CI: 10% to 38%, p=0.002) and that adding UFH to aspirin also reduced mortality (8.6% vs 9.1% in the aspirin-alone group, p=0.03) and re-infarction (3% vs 3.3% for aspirin alone, p=0.04).[20] A study of 584 STEMI patients that did not receive reperfusion therapy found that there is significant interaction between UFH and clopidogrel (p<0.001); UFH in clopidogrel users was associated with lower risk of in-hospital death (adjOR 0.038; 95% 0.038;95%CI 0.30-0.70, p=0.002), but the mortality rate was not statistically significantly different compared to enoxaparin in clopidogrel users (p=0.08) or enoxaparin in non-clopidogrel users (p=0.85) and UFH in non-clopidogrel users 4p=0.85).[21] Another study of 1063 STEMI patients who did not receive mechanical or pharmacological reperfusion found that UFH was significantly associated with lower risk of in-hospital mortality in clopidogrel users (multivariate adjusted regression OR 0.62, 95% CI 0.41-0.94) compared to non-clopidogrel users (OR 0.94, 95% CI 0.55-1.60).[22]

Beta blockers were known to decrease mortality both during MI and for long-term administration after MI.[23] A meta-analysis of randomized trials found that in-hospital mortality of STEMI patients who received IV beta blockers was lower compared to the control group (RR=0.92, CI 95%, 0.86-1.00, p=0.04) and also reduced the risk of ventricular tachyarrhythmias (RR=0.61, 95% CI 0.47-0.79, p=0.0003) and myocardial reinfarction (RR=0.73, 95% CI 0.59-0.91, p=0.0004).[24] A non-randomized cohort study found immediate (30 minutes to 24 hours after first ECG) bisoprolol treatment in STEMI patients has significantly lower cardiovascular mortality (p=0.0022) than the delayed treatment group, and also the result of multivariable Cox regression analysis found immediate bisoprolol therapy is independently protective against death of any cause (OR 0.55, p=0.033).[25] But the different result from a registry study of 34,661 STEMI and NSTEMI patients found that administering beta blockers immediately in the emergency room for STEMI and NSTEMI patients increases the risk of cardiogenic shock (NSTEMI: OR 1.23, 95% CI 1.08, 1.40, p=0.0016; STEMI: OR 1.30, 95% CI 1.03, 1.63, p=0.025); this risk increased in patients with two or more risk factors for shock (e.g., age >70 years, heart rate >110 bpm, systolic blood pressure <120 mmHg).[26] Beta blockers are effective in suppressing ventricular ectopic beats, also reducing sudden cardiac death (SCD) in a spectrum of cardiac disorders with or without heart failure.[27] Beta blockers are first-line therapy in the management of ventricular arrhythmias and prevention of sudden cardiac death in patients with ischemic heart disease.[23,27] A large retrospective registry study with subjects surviving at least one episode of ventricular tachyarrhythmia found that beta blockers and ACE inhibitors were associated with increased secondary survival (surviving from the secondary attack of ventricular tachyarrhythmia) rate, especially in patients with VF and LVEF <35%.[28] The present study recommends beta blockers as the first-line therapy in patients presenting with ventricular tachyarrhythmias.[28]

Amiodarone is the most studied and most commonly used medication in the treatment of secondary ventricular arrhythmias, and it remains the only guideline-directed medication in patients with either ischemic or non-ischemic cardiomyopathy; in the setting of secondary prevention, amiodarone has been a long-established therapy.[29] Amiodarone remains safe and effective in the short term as an anti-arrhythmic drug for VAs.[30] Amiodarone has an overlapping mechanism with all other classes of antiarrhythmics and also has vasodilatory and negative inotropic effects.[29] Amiodarone has the strongest evidence for routine use in clinical practice but is often avoided because of its long-term side effects (thyroid toxicity, pulmonary fibrosis, etc).[29,30] A cohort study of 1,354 ventricular tachyarrhythmia survivors from 2002 until 2016 at one institution found that beta blocker therapy was associated with


better long-term survival than beta blockers combined with amiodarone therapy (HR = 0.491; 95% CI 0.362-0.666; p=0.001) and after propensity score matching (HR = 0.590; 95% CI 0.378-0.921; p=0.020).[31] This case was about to receive amiodarone but was not administered because the patient already responded to bisoprolol; sinus rhythm was resumed after 12 hours of treatment and continued until day 7 of admission. No secondary attack or any other VT episode was recorded during follow-up, and the hemodynamics were stable.

CONCLUSION

This case report highlights the challenges of managing ST-Elevation Myocardial Infarction (STEMI) complicated by ventricular tachycardia (VT). The patient responded well to a combination of anticoagulant (UFH), DAPT, beta-blocker (bisoprolol), and other standard medications. Notably, VT was resolved without the need for amiodarone, demonstrating the potential of beta-blockers in acute settings. This case emphasizes the importance of rapid and comprehensive treatment to improve outcomes in similar scenarios.

ACKNOWLEDGMENTS

I thank the patient and the patient's family for consenting to publish the clinical information and also dr. I G M A as the internist of this patient, for consenting to let me follow up and publish clinical information of his patient.

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