

Incessant Malignant Ventricular Arrhythmia in a Patient with Advanced Heart Failure: A Case Report

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Introduction

The occurrence of refractory ventricular arrhythmia in patients with heart failure (HF) is a therapeutic dilemma.¹ These patients, especially in the presence of advanced disease, may experience multiple triggering factors for ventricular arrhythmia, such as myocardial fibrosis, ischemia, hydroelectrolytic disorders, ventricular distention, the arrhythmogenic potential of drugs, and the underlying disease itself.^{1–3} Conversely, sustained ventricular arrhythmias cause hemodynamic compromise, resulting in further ischemia and ventricular distension.¹ This can lead to a vicious cycle of hemodynamic worsening with an increasingly arrhythmogenic myocardium.¹ Usual treatment consists of direct current (DC) cardioversion, antiarrhythmic drugs, myocardial revascularization (when indicated), and catheter ablation. However, invasive procedures are always considered high risk in such cases. We report the case of a patient with incessant ventricular arrhythmia resistant to several types of treatment.

Case Report

A 53-year-old male patient was transferred to our hospital due to an episode of sustained ventricular tachycardia (VT) that had occurred 48 hours earlier and was initially treated in other hospital. The patient had been on the heart transplant waiting list for 2 years due to the presence of idiopathic dilated cardiomyopathy with a left ventricular ejection fraction (LVEF) of 26%. He underwent VT ablation 5 years ago and cardiac resynchronization therapy (CRT) device implantation 3 years ago. There was also a history of chronic atrial fibrillation (anticoagulated), hypertension, depression, and hypothyroidism. Before the event, the patient was in New York Heart Association (NYHA) functional class II. Immediately after admission, he had another episode of sustained VT with hemodynamic instability and underwent DC cardioversion. After the episode, the patient developed signs of low cardiac output

and was started on dobutamine, sodium nitroprusside, and intravenous amiodarone. Electronic evaluation of the CRT device showed several episodes of sustained VT in the last 24 hours. Hydroelectrolytic disorders were also recognized and immediately corrected. Increased free T4 (2.04 ng/dL, reference value: 0.89–1.76 ng/dL) and normal thyrotropin levels were also identified. Amiodarone-induced thyroiditis was suspected and empirical treatment with corticosteroids was started. However, a new episode of sustained VT requiring DC cardioversion occurred in the next morning. During the afternoon, the patient had an episode of cardiac arrest with polymorphic VT rhythm (*torsades de pointes*) and underwent electrical defibrillation. Electrocardiogram showed a prolonged QT interval, thus the infused dobutamine dose was reduced and amiodarone infusion was changed to lidocaine. The patient had another episode of sustained VT requiring DC cardioversion on the following day, and amiodarone infusion was subsequently reinstated. An episode of supraventricular tachycardia occurred on the same day and rapidly progressed to acute pulmonary edema. Due to refractory ventricular arrhythmias and acute decompensated HF, the patient underwent intra-aortic balloon pumping (IABP) implantation on the following day (day 4 of hospitalization). After IABP, the patient was weaned off dobutamine and there was no recurrence of ventricular arrhythmias. However, the patient developed nosocomial pneumonia and required antibiotic therapy for 14 days (piperacillin-tazobactam, meropenem, and teicoplanin). Because the patient was dependent on mechanical circulatory support and the possibility of further VT ablation was excluded, his status on the transplant waiting list was reactivated and updated to urgent. On day 63 after IABP without arrhythmia recurrence, the patient underwent a heart transplant.

Discussion

The case described here shows the multifactorial mechanism of ventricular arrhythmia genesis in a patient with advanced HF. In addition to the arrhythmogenic substrate (idiopathic dilated cardiomyopathy which needed previous ablation), hydroelectrolytic disorders and amiodarone-induced thyroidopathy may have contributed to the onset of the condition. This leads to the vicious cycle of hemodynamic deterioration with perpetuation of the arrhythmic condition. The difficulty in clinical management is also evident: inotropic agents have the potential for hemodynamic stabilization at the expense of a proarrhythmic effect. Conversely, the occurrence of *torsades de pointes* due to QT prolongation exemplifies a known fact that antiarrhythmic agents may also cause proarrhythmic effects, although by different mechanisms. This issue was

Keywords

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resolved by using mechanical circulatory support through IABP for hemodynamic stabilization and ventricular arrhythmia control. IABP also allowed for dobutamine weaning, resulting in the removal of a proarrhythmic factor.

Some case reports and series describing the use of IABP with the primary objective of controlling ventricular arrhythmias have been published.^{1,4-7} In one of the first cases described, IABP was able to control ventricular arrhythmias in the setting of post-infarction in a patient who received more than 120 electrical cardioversions.⁷ Other reports have also demonstrated the use of IABP for refractory ventricular arrhythmia control in patients without coronary artery disease.^{1,4,6} In a case series of 12 patients, IABP was effective in controlling ventricular arrhythmias in 18 cases.¹ Of these, 5 underwent a heart transplant, 12 became stable and were weaned off IAB support, and 3 were refractory to IABP.¹ One patient was diagnosed with cardiac amyloidosis and associated systemic involvement and was considered unsuitable for transplantation.¹ In this study, coronary artery disease (acute and chronic) was evident in 18 of 21 patients and all had ventricular dysfunction, with a mean LVEF of 29%.¹ Nineteen patients were discharged from hospital and followed up for 25.7 months, with a survival rate of 95%.¹

Several mechanisms have been proposed to explain how IABP helps to control ventricular arrhythmias.^{1,5} The primary mechanism is the increase in coronary perfusion by active insufflation during diastole, which may reduce ischemia as a precipitating factor in ventricular arrhythmia genesis.⁵ However, IABP may also be effective in patients with no evidence of coronary artery disease.^{4,6} In such cases, the support provided by IABP may allow sufficient time for anti-arrhythmic drugs to work and interrupt the vicious cycle of hemodynamic deterioration and arrhythmogenesis.¹ Finally, dilation and increased tension in the left ventricular wall

have been shown to cause electrophysiological changes in the myocardium, creating an arrhythmogenic substrate.^{1,3,8} This may be a particularly important mechanism in patients with advanced HF. In this case, IABP acts directly by reducing ventricular afterload, consequently decreasing tension in the left ventricular wall.^{1,5}

The case described here emphasizes the complexity of managing incessant ventricular arrhythmias in a patient with advanced HF. It also shows the role of IABP in hemodynamic stabilization, interruption of the cycle of progressive arrhythmogenesis, and as a bridge to heart transplantation.

Author Contributions

Writing of the manuscript and Critical revision of the manuscript for intellectual content: Murad CM e Campos IW.

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References

1. Fotopoulos GD, Mason MJ, Walker S, Jepson NS, Patel DJ, Mitchell AG, et al. Stabilisation of Medically Refractory Ventricular Arrhythmia by Intra-aortic Balloon Counterpulsation. *Heart*. 1999;82(1):96-100. doi: 10.1136/hrt.82.1.96.
2. Francis GS. Development of Arrhythmias in the Patient with Congestive Heart Failure: Pathophysiology, Prevalence and Prognosis. *Am J Cardiol*. 1986;57(3):3-7. doi: 10.1016/0002-9149(86)90991-4.
3. Koilpillai C, Quiñones MA, Greenberg B, Limacher MC, Shindler D, Pratt CM, et al. Relation of Ventricular Size and Function to Heart Failure Status and Ventricular Dysrhythmia in Patients with Severe Left Ventricular Dysfunction. *Am J Cardiol*. 1996;77(8):606-11. doi: 10.1016/s0002-9149(97)89315-0.
4. Goyal D, Nadar SK, Wrigley B, Koganti S, Banerjee P. Successful Use of Intra-aortic Counter Pulsation Therapy for Intractable Ventricular Arrhythmia in Patient with Severe Left Ventricular Dysfunction and Normal Coronary Arteries. *Cardiol J*. 2010;17(4):401-3.
5. Cowell RP, Paul VE, Ilsley CD. The Use of Intra-aortic Balloon Counterpulsation in Malignant Ventricular Arrhythmias. *Int J Cardiol*. 1993;39(3):219-21. doi: 10.1016/0167-5273(93)90043-g.
6. Lisi E, Guida V, Blengino S, Pedrazzi E, Ossoli D, Parati G. Intra-aortic Balloon Pump for Treatment of Refractory Ventricular Tachycardia in Tako-Tsubo Cardiomyopathy: A Case Report. *Int J Cardiol*. 2014;174(1):135-6. doi: 10.1016/j.ijcard.2014.03.102.
7. Culliford AT, Madden MR, Isom OW, Glassman E. Intra-aortic Balloon Counterpulsation. Refractory Ventricular Tachycardia. *JAMA*. 1978;239(5):431-2.
8. Dean JW, Lab MJ. Arrhythmia in Heart Failure: Role of Mechanically Induced Changes in Electrophysiology. *Lancet*. 1989;1(8650):1309-12. doi: 10.1016/s0140-6736(89)92697-4.



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