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Case Report

Successful Management of Syncope and Sustained Ventricular Tachycardia/Fibrillation in a Nonagenarian with Do-Not-Resuscitation Status: A Case Report

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Abbreviations: AVN: Atrioventricular Node; LBBA: Left Bundle Branch Area; LBBB: Left Bundle Branch Block; RBBB: Right Bundle Branch Block.

Introduction

Sustained ventricular tachycardia/fibrillation typically leads to syncope, cardiac arrest, and sudden death. External defibrillation and cardiac resuscitation including chest compressions are cornerstones of successful treatment in such events [1]. These treatments, however, are typically contraindicated in patients who are on the do-not-resuscitate status. We here report a case where a rare form of sustained ventricular tachycardia/fibrillation triggered by extreme bradycardia and was completely reversed by using the left bundle branch area (LBBA) pacing technique.

Case Presentation

The patient is an elderly male in his 90's with permanent atrial fibrillation and slow ventricular response, chronic right bundle branch block (RBBB), mechanical aortic valve prothesis, pulmonary hypertension, severe tricuspid valve regurgitation and heart failure with a recovered left ventricular (LV) systolic function (EF 57%). He was initially seen in the outpatient clinic for bradycardia and moderate fatigue. A Holter monitor was placed and showed an average heart rate of 42bpm. Review of previous records showed electrocardiograms (ECG) with left bundle branch block (LBBB, Figure 1A and 1B). Permanent pacemaker (PPM) implantation was discussed but the patient declined the procedure due to lack of severe symptoms, advanced age and his do-not-resuscitate status. A few weeks later, he fell in the shower and suffered from superficial head injury after a spell of frank loss of consciousness. He presented to the ER with a ventricular escape rhythm of 33 bpm (Figure 1C). While hospitalized, he had recurrent syncopal events associated with increasingly frequent runs of non-sustained and sustained polymorphic ventricular tachycardia/fibrillation that fortunately all self-terminated. The uncorrected QT interval preceding these events exceeded 800ms (Figure 1D). Routine blood testing was unremarkable except for an INR of 1.9 and creatinine of 1.68mg/dL. His electrolytes were normal (potassium of 4.0 mmol/L and magnesium of 2.4mg/dL). The diagnosis of bradycardia dependent Torsades de Pointes was made. As the condition is reversible by ventricular pacing, the patient and family agreed to proceed with PPM implantation.

Management

The patient was urgently taken to the electrophysiology laboratory for pacemaker implantation. Because of his history of cardiomyopathy and anticipated frequent ventricular pacing, he was considered to be at increased risk of recurrent cardiomyopathy induced by right ventricular (RV) apical pacing. Additionally, because previous studies suggest that RV apical pacing or LV epicardial pacing may promote arrhythmogenicity, we elected to proceed with a trial of LBBA pacing. Through an axillary vein access, a Medtronic C315 delivery sheath was advanced to the RV septum in the vicinity of the LBB area (1.5 to 2 cm distal to the tricuspid annulus towards the ventricular apex). A Medtronic 3830 lead was advanced over the delivery sheath and penetrated the ventricular septum to engage the left bundle. At the final location, pacing demonstrated abrupt conversion of the paced QRS from a LBBB morphology to a much narrower RBBB morphology. An LV activation time (measured from pacing stimulus to R wave peak or S-RWPT) in V6 of 90 ms and a paced QRS duration of 120 ms suggested likely recruitment of the left bundle-Purkinje system. Pacing at high and low output demonstrated fixed S-RWPT with excellent sensing and pacing thresholds. The procedure was completed successfully without complications. The procedure time from lidocaine injection to pocket closure was 16 minutes with a fluoroscopy time of 1.6 minutes. A post-operative chest x-ray showed satisfactory lead position and an echocardiogram showing LBBA pacing lead seated appropriately in the deep septum (Figure 2).

Following the PPM implantation using the LBBA pacing technique, the patient's QT interval shortened markedly, and his ventricular tachycardia immediately resolved with no further arrhythmia episodes until hospital dismissal (Figure 3).

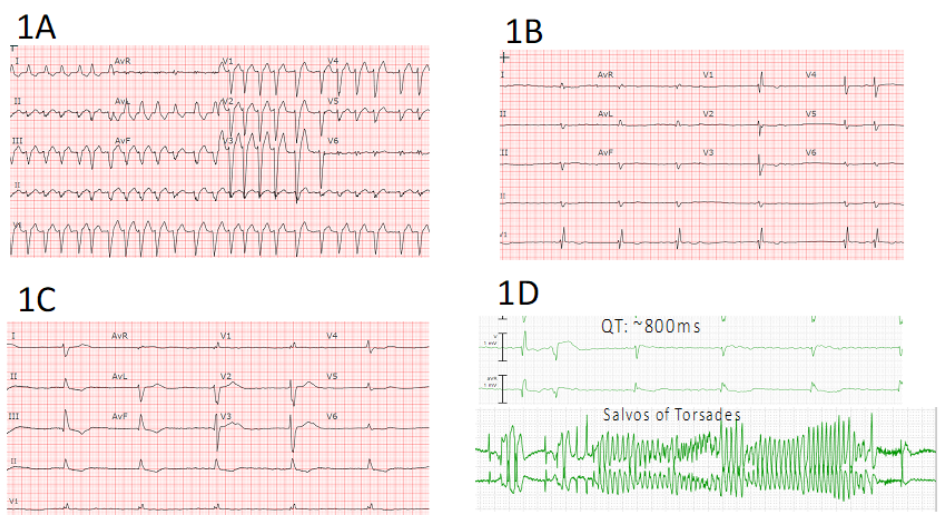

Figure 1: Tracings demonstrating alternating bundle branch block, followed by advanced heart block with a slow escape rhythm and ultimately bradycardia dependent QT prolongation and Torsades de Pointes. 1A: Remote baseline ECG showing atrial fibrillation with underlying LBBB. 1B: Recent ECG showing RBBB. 1C: Presenting ECG in the ER showing complete heart block with underlying ventricular escape rhythm. 1D: A representative tracing showing marked QT prolongation preceding an episode of Torades de Pointes resembling aborted ventricular fibrillation.

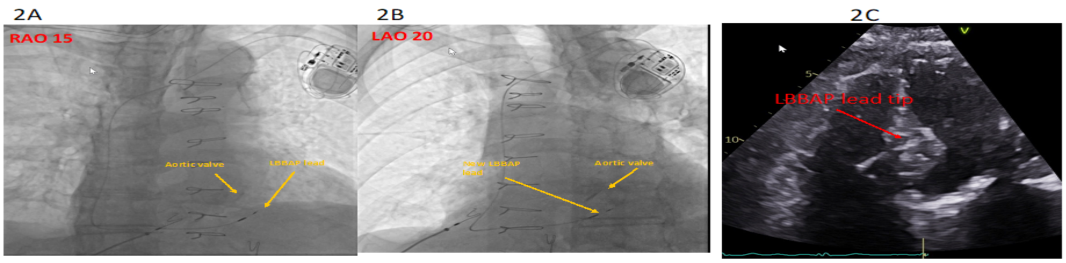

Figure 2: Demonstrating the left bundle branch area pacing lead position shown in RAO and LAO views on chest fluoroscopy (2A and 2B) and post-operative ECHO (2C).

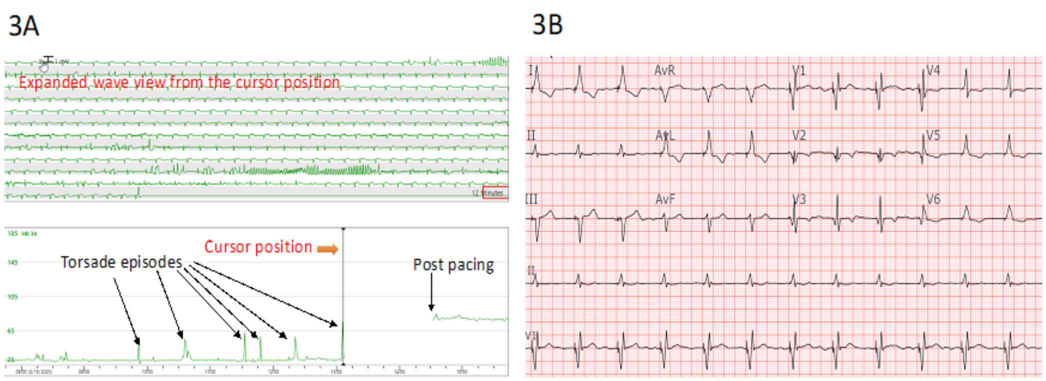

Figure 3: 3A: Monitor strips showing frequent runs of Torsades and immediate resolution after ventricular pacing using LBBA pacing technique. 3B: 12 lead ECG after LBBA pacing.

Discussion

The case described here highlights the critical importance of making the accurate clinical diagnosis on the outcomes. While most sustained ventricular tachycardia/fibrillation episodes carry poor prognosis and are typically not aggressively treated in elderly patients on do-not-resuscitation status, bradycardia dependent Torades are completely reversible by ventricular pacing.

Bradycardia induced by heart block is a well-recognized risk factor for acquired torsades de pointes [2]. Ventricular pacing has been shown to be a very effective treatment for this condition, although it is still unclear which ventricular pacing modality is best suited for this indication. Certain pacing modalities such as epicardial pacing or ventricular apical pacing have been shown to be proarrhythmic due to an abnormal ventricular depolarization sequence and increased dispersion of ventricular repolarization [3-5]. More recently, conduction system pacing, especially LBBA pacing, has rapidly emerged as a form of physiologic pacing that may be superior to conventional endocardial RV or epicardial LV pacing. However, it has recently been reported that conduction system pacing using His-bundle pacing may be associated with QT prolongation, raising the question of whether LBBA pacing can be safe and effective in managing patients with bradycardia induced QT prolongation and torsades de pointes [6].It is also unknown whether LBBA pacing can be achieved in patients who have diffuse conduction system disease involving both bundle branches. We postulate that the His-Purkinje system's unique, fast conduction properties can lead to a coordinated myocardial activation that not only maximizes myocardial contraction efficiency, but also minimizes risk of re-entry, making conduction system pacing using LBBA pacing the preferred modality to prevent pacing-induced ventricular pro-arrhythmia.

The case here suggests that conduction system pacing through LBBA pacing can be very effective in managing bradycardia induced torsades des pointes. In addition, we also demonstrated that even in patients with diffuse conduction system disease including bilateral bundle branch block requiring urgent pacemaker implant, LBBA pacing can be successfully and efficiently performed. To our knowledge, this is the first case report of successful LBBA pacing for management of torsades in a nonagenarian patient with bilateral bundle branch block leading to complete heart block.

There has been an explosion of studies and research in conduction system pacing especially LBBA pacing in recent years. Multiple emerging studies have shown that this pacing modality to be superior to RV apical pacing and at least as effective as LV epicardial pacing to promote cardiac resynchronization and prevent and/or reverse ventricular remodelling [7]. Conduction system pacing, particularly LBBAP, can achieve physiologic pacing, stable lead position with excellent threshold and sensing, as well as easy and efficient implantation after the initial learning phase. It has not been irrefutably demonstrated whether physiologic pacing has advantages over RV apical pacing in patients who have bradycardia-induced torsades. However, there are several reasons why conduction system pacing may be the preferred pacing modality. The His-Purkinje system's fast conduction properties couples with myocardial fibers and ensures an efficient and coordinated myocardial contraction reducing ventricular depolarization and minimizes the risk of re-entry [8]. On the other hand, studies have suggested that non-physiology pacing from epicardial LV and RV apical pacing may increase depolarization duration time, increase dispersion of ventricular repolarization [9] and be potentially arrhythmogenic [3,5,10-12]. Therefore, physiologic pacing may be advantageous in this setting [13,14]. Moreover, conduction system or physiologic pacing is less likely to promote ventricular dyssynchrony and less likely to be associated with pacing induced cardiomyopathy which in itself could lead to QT prolongation and be arrhythmogenic in the long term. In our case presented here, the QT interval was markedly reduced, although some of the shortened QT is due to QRS shortening duration. Large-scale studies are required to determine whether LBBA pacing is less pro-arrhythmogenic and superior to RV apical or LV epicardial pacing in treating bradycardia dependent ventricular arrhythmias.

Declarations

Ethics approval and consent to participate: Not applicable.

Consent for publication: The patient has consented for publication.

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Competing interests: None.

Availability of data and materials: Available upon reasonable request.

Ethics approval and consent to participate: A written consent was obtained from the patient to publish the case.

Disclosures: None.

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