Ventricular Tachycardia in A Patient with A Previous History of Endocarditis and Ankylosan Spondylitis: A Challenging Case

ABSTRACT

Cardiac conduction defects are commonly observed in patients with ankylosing spondylitis, infective endocarditis, and aortic valve replacement. Each of these clinical situations can also present with ventricular tachycardia by different mechanisms. Here we report the case of a 53-year-old man with a medical history of untreated ankylosing spondylitis and aortic valve replacement who presented with ventricular tachycardia and underwent successful catheter ablation. Most ventricular tachycardia episodes were intermittent and drug resistant, which could have been caused by abnormal automaticity rather than re-entry.

Keywords: Ankylosan spondylitis, ventricular tachycardia, catheter ablation

Case Report

A 53-year-old man with a 30-year history of untreated AS was admitted to our hospital with the complaints of fever and intermittent palpitations. He had intermittent palpitations since 25 years of age. He underwent AVR (St Jude mechanical bi-leaflet valve 29 mm) 24 years previously because of aortic valve endocarditis. His medications included ramipril, metoprolol, and warfarin. Physical examination revealed that he had a fever of 38°C, and cardiac examination revealed a 2/6 systolic murmur in the aortic area with a loud mechanical component of aortic second heart sound. Peripheral stigmata of endocarditis were not found. Electrocardiography (ECG) demonstrated a normal sinus rhythm with bigeminated ventricular extrasystoles and left anterior fascicular block. Transthoracic echocardiography revealed a functioning prosthetic aortic valve and mild paravalvar regurgitation and grade 2 diastolic dysfunction. Left ventricular ejection fraction was measured as 50%. Transesophageal echocardiography revealed a functioning prosthetic aortic valve and no vegetation or thrombus.

His blood chemistry on admission was within normal limits, except for an elevated C-reactive protein (CRP) level and erythrocyte sedimentation rate (ESR) (CRP, 81.8 mg/L; ESR, 26 mm/h).
The leukocyte counts on admission and throughout the hospital course were within the normal range. On the fifth day of admission, his fever subsided and CRP level and ESR decreased to 16.1 mg/L and 10 mm/h, respectively.

During the hospital course, he developed frequent nonsustained and sustained runs of monomorphic VT episodes that terminated with amiodarone (Figure 1). In one of these episodes, he experienced a cardiac arrest secondary to ventricular fibrillation, needing external electrical defibrillation. Despite continuous infusion of amiodarone, sustained and nonsustained VT episodes developed at frequent intervals with the same ECG morphology. Bigeminated ventricular extrasystoles were still present. QT and QTc intervals were in normal limits. Coronary angiography showed no coronary lesions.

After informed consent was obtained, the patient underwent an ICD-DR implantation. Catheter mapping and ablation were planned. In the electrophysiological study, no clinical VT was induced with programmed ventricular stimulation from the right ventricular apex. Therefore, left ventricle (LV) mapping using a transseptal approach was performed with a Brockenbrough needle and an 8.5 Fr Mobicath sheath under fluoroscopic guidance. The ablation catheter (THERMOCOOL™, Biosense Webster; Diamond Bar, CA, USA) was then advanced to LV via the transseptal sheath. Activation mapping was initiated in LV using a CARTO-3® mapping system (Biosense Webster, Diamond Bar, CA, USA), and fragmented, late, and Purkinje potentials were observed in the anterobasal region of LV. Substrate mapping revealed a very small scar area in this region. While an RF application was delivered at these sites, 2 different morphologies of VT episodes occurred. One of these episodes was likely compatible with epicardial VT (e.g., positive concordans and pseudo-delta wave ≥34 ms, intrinsicoid deflection time ≥85 ms, maximum deflection index ≥55 ms) that terminated with ICD shocks. RF ablation at the anterobasal site terminated the other monomorphic VT episode and rendered it noninducible. Sulfasalazine and diclofenac treatment for AS was added to his current medications, and intervention for the epicardial VT was scheduled at another time if recurrence occurred. At 1-month control, the frequency of ventricular extrasystoles reduced and no VT episodes were observed in ICD tracings (Figure 2).

**Discussion**

In patients with AS, IE, and AVR, there is an increased risk of cardiovascular complications such as conduction defects, VT, and cardiomyopathy. Many aspects of arrhythmogenesis and optimal arrhythmia control are not well established in patients with cardiac involvement of AS. In AS, the underlying chronic inflammation, particularly that in untreated patients, is likely responsible for cardiovascular involvement [1-3].

Conduction defects after AVR are commonly observed, affecting 1 in 3 patients. Sustained monomorphic VT after valvular surgery can be observed in 2 forms: myocardial VT, which is usually associated coronary artery disease, leading to scar tissue and/or significant left ventricular dysfunction, and bundle-branch reentry (BBR), which is caused by conduction defect in the His–Purkinje system that occurs in the early postoperative period. Because of the curative nature of therapy, BBR should be considered as the mechanism of VT in operated valvular heart disease patients, particularly when the arrhythmia occurs soon after valve surgery.
Unlike patients with BBR-VT, patients with myocardial VT have a high recurrence rate of VT [3, 4]. In our case, the ECG morphology suggested that the tachycardia was originating from mitral annulus, which was not standard right or left bundle branch block morphology found in BBR-VT.

AS is a chronic, inflammatory, and rheumatic disorder that primarily affects the spine, sacroiliac, and peripheral joints. AS is associated with an increased risk of several cardiac complications, including conduction defects (atrioventricular, bundle branch, and intraventricular blocks), valvular regurgitation, cardiomyopathy, pericarditis, and aortitis. Of these, conduction defects are the most common [5]. The incidence of cardiovascular involvement in AS ranges between 10% and 30%, and cardiac involvement is more commonly observed in patients with a longer disease duration [6].

Inflammation and fibrosis of the membranous portion of the interventricular septum and/or proliferation of smooth muscle cells or fibroblasts occluding the atrioventricular nodal artery are the most probable mechanisms that have been considered in the pathogenesis of conduction defects and cardiomyopathy, but they have not been completely proved to date [7, 8]. On the other hand, Brewerton et al. [9] studied myocardial tissue biopsies in 28 patients with AS who had no ischemic heart disease, hypertension, or valvular disease and provided strong evidence that cardiomypathy in AS is a clinical reality that is often overlooked or ignored by clinicians. Clinical signs of cardiomyopathy are typically associated with impaired diastolic functions [9].

As in this case, high CRP levels may be present in patients with active AS. Indeed, a 3-fold increased risk of ventricular tachycardia was demonstrated with high CRP levels [10].

In conclusion, conduction abnormalities, primarily atrioventricular block, are more commonly encountered in AS. Ventricular arrhythmia may rarely occur due to ventricular inflammation and fibrosis. To the best of our knowledge, AS-associated VT that could be cured by ablation has not yet been reported. This report also emphasizes on the importance of the suppression of inflammation in AS, which may prevent potentially fatal events due to myocardial involvement.

Informed Consent: Written informed consent was obtained from the patient whose case is reported here.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this case has received no financial support.

References
1. Kitkungvan D, Denktaş AE. Cardiac arrest and ventricular tachycardia from coronary embolism: an unusual presentation of infective endocarditis. Anadolu Kardiyol Derg 2014; 14: 204-5. [CrossRef]