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Case Report

Catheter Ablation of Accelerated Idioventricular Rhythm Originating from Left Sinus of Valsalva after Cardiac Surgery

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Abstract

Accelerated idioventricular rhythm (AIVR) is a ventricular rhythm that is frequently observed in the reperfusion phase of an acute myocardial infarction or because of drug toxicity with digoxin and cocaine. This is also seen in patients with cardiomyopathy, congenital heart disease and myocarditis. Nonetheless, it is thought that AIVR has a good prognosis. In this case report, we described a patient with a history of repaired perimembranous subvalvular aortic interventricular defect who presented with high burden AIVR. We described the clinical manifestations, differential electrocardiographic diagnosis with parasystolic ventricular tachycardia, and management. This patient ultimately underwent successful catheter ablation of ectopic focus located in the proximity of left sinus of Valsalva.

Keywords: Accelerated idioventricular rhythm; Catheter ablation; Left sinus of Valsalva

Introduction

Accelerated idioventricular rhythm (AIVR) is due to enhanced automaticity of a ventricular ectopic focus that is not protected and independent of the dominant sinus rhythm. Typical electrocardiographic signs of AIVR are heart rate < 120 bpm, regular interectopic intervals, often with gradual onset and termination, atrioventricular dissociation, late variable coupling of the first beat of tachycardia often with fusion beats. In adults, AIVR is frequently observed during the reperfusion phase following a myocardial infarction [1], in patients with structural heart disease [2-5], and due to several drug toxicities such as with halothane, cocaine or digitalis [6-11]. AIVT can sporadically manifest in patients with normal hearts [12] and usually is a well-tolerated arrhythmia with a favorable prognosis. Sometimes AIVR must be differentiated from parasystolic ventricular tachycardia (PSVT) [13,14], in which the focus is protected and characterized by a unidirectional entry block since sinus impulses cannot penetrate it. Additionally,

PSVT can present electrocardiographic characteristics similar to those of AIVR, especially in cases of irregular PSVT due to the intrinsic properties of the focus itself [15,16], or disorders of impulse formation and conduction [17-20]. Therefore, differential diagnosis can be difficult. Hence, we are describing for the first time an unusual type of ventricular arrhythmia in a 21-year-old man who underwent surgical repair of a perimembranous subvalvular aortic interventricular defect and presented with AIVR arising from left SoV, refractory to antiarrhythmic drug. Although patients undergoing repair of isolated ventricular septal defect (VSD) frequently have good overall survival, some of them may experience diverse complications during follow-up including the development of atrial arrhythmias, pacemaker implantation for high-degree AV block and the development of some degree of left ventricular outflow tract obstruction (LVOTO) [21-23]. Furthermore, an analysis of outcomes performed from 1953 to 2009 in patients surviving the initial procedure reported an overall late mortality at 40 years of 8.8%, with 61% of these related to their underlying cardiac condition [24]. However, the presence of ventricular arrhythmias in these studies has not been reported.

Case Presentation

A 21-year-old patient was admitted to the Cardiology Department of Policlinico Riuniti of Foggia for assessment of ventricular extrasystoles in November 2018. At the age of 3 years, the patient underwent open heart surgery to get repaired a perimembranous subvalvular aortic interventricular defect with significant left-to-right shunt (gradient of 80 mmHg) and a dilated left atrium and ventricle. In the following years the patient was always asymptomatic and underwent regular cardiological examinations. At age 11, a 24-hour Holter monitoring showed sinus rhythm with 814 premature ventricular contractions (PVCs). Subsequently in 2016, he started having palpitations at rest and two episodes of syncope during effort. In March 2018, the patient underwent a second 24-hour Holter monitoring that showed "sinus rhythm with 48717 PVCs (burden: 52.6%) with periods of non-sustained monomorphic ventricular tachycardia (25 beats). The patient was unsuccessfully started on antiarrhythmic therapy with Flecainide 200 mg/day in association with a low dose of beta blocker for 6 months. The 12-lead ECG showed sinus rhythm alternating with ectopic ventricular rhythm at ventricular rate of 80 bpm with the following morphology: inferior axis, left bundle branch block morphology with precordial R/S transition zone in lead V3 (rS pattern in the V1-V2 leads). Corrected Q-T intervals were normal [Figure 1]. The echocardiogram showed normal left and right ventricular function with normal right and left ventricular outflow tract diameters. Cardiac MRI was not performed due to the presence of metallic stitches resulting from previous surgery.

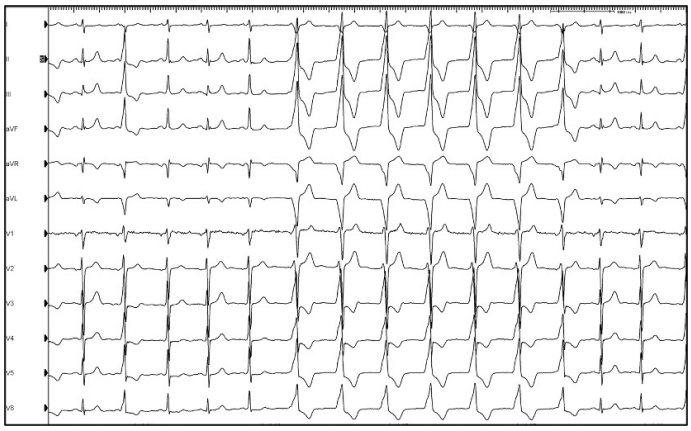

Figure 1: 12-lead electrocardiogram showing sinus rhythm alternating with accelerated ectopic ventricular rhythm at ventricular rate of 80 bpm. Ectopic beats had the following morphology: inferior axis, left bundle branch block morphology and rS pattern in the V1-V2 leads with precordial R/S transition zone in V3.

12-lead ECG differential diagnosis

Prolonged 12-lead ECG monitoring and intracardiac electrogram signals preceding transcatheter ablation showed respiratory sinus arrhythmia normally conducted to the ventricles alternating with successive PVCs at average ventricular rate between 75 and 108 bpm, fusion beats and variable coupling interval, always late, of the first beat of tachycardia. Intracardiac electrograms signals in Figure 2 shows sinus arrhythmia (empty circles) with different conduction of the sinus beats to the ventricles (black circles): the first is conducted with fusion; the second is blocked in the AV junction; the third is conducted to the ventricle with delay of AV interval due to concealed conduction of the ventricular beat to the atrio-ventricular junction. The minimum interval between ectopic and early sinus conducted beats was 550 ms, sometimes with prolongation of AV interval as already mentioned, and minor aberration of the QRS (fifth, seventh and twelfth beat in Figure 3A). Some electrocardiographic features suggest the diagnosis of AIVR rather than PSVT. First, ventricular rhythm had irregular interectopic intervals. Indeed, R-R intervals varied between 550 to 800 ms and are in relation to sinusal rhythm (isorhythmic). This irregularity of ectopic rhythm may be due to intrinsic properties of the focus influenced by neurovegetative and humoral factors [25,26]. Figures 3B, 3C and 3D show sinus rhythm with different frequencies of ventricular beats with gradually widening and narrowing of the QRS (concertina-like effect). This is due to the variable contribution to ventricular activation of the impulses conducted through the A-V node and the ectopic

beats, which creates isorhythmic dissociation and fusion beats with different widths. A prominent acceleration of ectopic focus due to autonomic influence is frequently seen in AIVR, as described in patient without demonstrable heart disease [12] and described for pregnant women in the antenatal period [26].

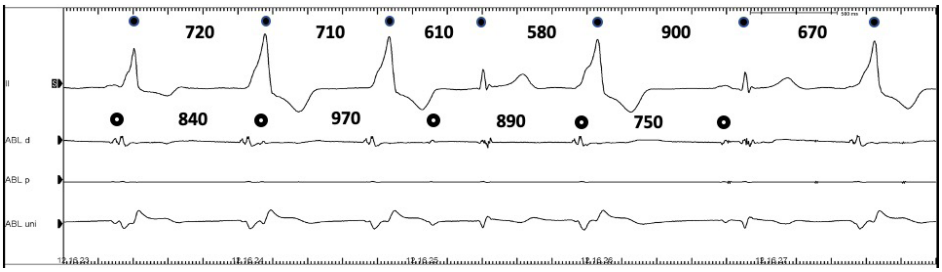

Figure 2: Intracardiac electrograms signals shows sinus arrhythmia (empty circles) with different conduction of the sinus beats to the ventricles (black circles): the first is conducted with fusion; the second is blocked in the AV junction; the third sinus beat is conducted to the ventricle with delay of AV interval due to concealed conduction of the ventricular beat to the atrio-ventricular junction.

Second, coupling intervals (CI) of the first ectopic beat is highly variable, ranging from 550 to 730 ms, and always late after the sinus impulse, often with fusion, as we can see in all the ECG traces of Fig. 3. This is a feature of AIVR, but may be present also in uncommon cases of PSVT with ectopic-ventricular (E-V) exit block due to concealed conduction of the sinus impulses [27,28]. In this case of PSVT, sinus impulses enter the junction between the focus and the surrounding myocardium and made the E-V junction refractory, creating an additional refractory period stopping relatively late ectopic impulses, despite they fall outside the refractory period of the ventricles. Only very late impulses are able to reach the surrounding myocardium, thus explaining why there are no parasystolic beats with short CI. However, in PSVT, long interectopic intervals are always mathematically related and multiple of the basic parasystolic cycle. Consequently, this is never observed in our patient because the ectopic focus is always evident only when sinus rhythm decreases, confirming the diagnosis of AIVR rather than PSVT. Third, interectopic intervals containing sinus complexes inside are always not multiples of the ectopic cycle, as we can see in Fig 3F and 3G. An interectopic interval including a single or double sinus beat is shorter than twice or three times the cycle of a couple. Nevertheless, the analysis of the dynamic interactions between the sinus rhythm and the ectopic rhythm does not follow the rules of intermittent or modulated parasystolic rhythm, as it was postulated by Schamroth in 1961 [29,30] and demonstrated by Jalife and Moe later with laboratory experiments [17]. Finally, as we can see in fig 3H and 3I, after one or more ventricular ectopic beats, ectopic ventricular rhythm disappears for one or two cycles. This may be due to temporary depletion of the automatic focus activity or to an exit block between the ectopic focus and ventricular myocardium. However, this phenomenon can be present in both arrhythmias.

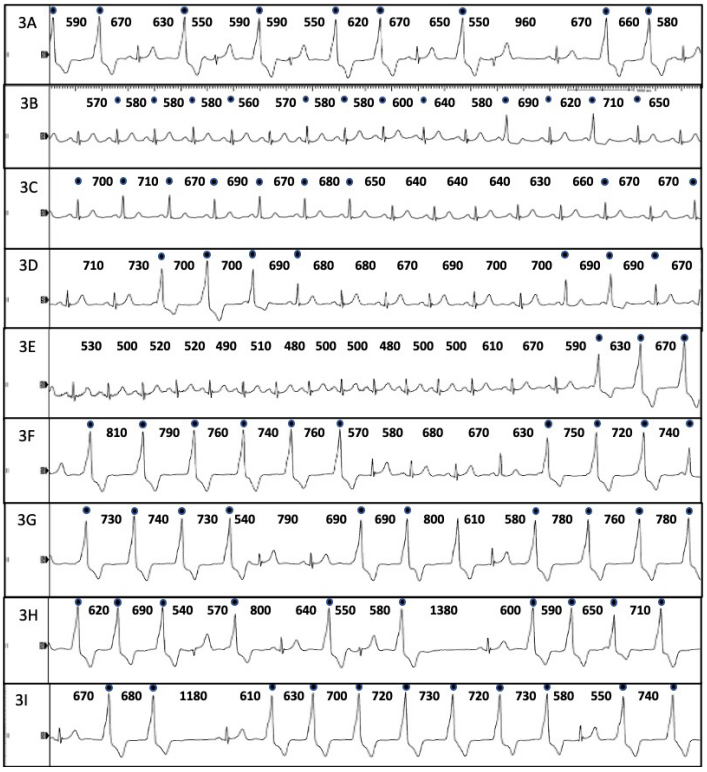

Figure 3: Different electrocardiographic traces during continuous monitoring performed before ablation. Detailed explanation in the text.

Transcatheter mapping and ablation

The patient underwent transcatheter mapping and ablation of the ventricular ectopic focus. The procedure was performed using a 3D mapping system (CARTO3, Biosense Webster,

Diamond Bar, CA). Mapping with an irrigated tip ablator catheter with contact force sensing (Thermocool SmartTouch, Biosense Webster, Diamond Bar, Inc) was inititally performed into the right ventricular outflow tract, according to the 12-lead ECG morphology of the ectopic focus. Activation mapping and pace-mapping were combined to identify the origin of ectopic focus. Activation times were assigned based on the earliest rapid downstroke of the unipolar signal (fastest dV/dt) correlating with the first sharp peak of the bipolar electrogram. The reference line was a sharp peak of QRS in precordial leads. Pace-mapping was performed using PaSoTM module (Biosense-Webster, CA, US). As a result, activation mapping in RVOT revealed that the earliest local activation was 14 ms before the onset of the QRS in the posterior septal region with a significant PaSoTM coefficient correlation (93%). However, 2 short radiofrequency deliveries (Power 30 W; max 15 gr) failed to eliminate the arrhythmia. Therefore, activation mapping and pace-mapping of the left ventricular outflow tract (LVOT) was performed via the transaortic route. In proximity of the left coronary cusp (LCC), the earliest local activation was 46 ms before the onset of the QRS with low voltage, fragmented signals and a QS pattern on unipolar recording. Pace-mapping showed a higher grade of similarity between the morphology of the PVC with the 12 lead ECG pacing morphology than that in RVOT evaluated with PasoTM module (96.3% vs. 93%) (Figures 4 and 5). We found the distance between this point and the ostium of the left coronary artery was 10 mm by angiographical evaluation. Subsequently, a low power radiofrequency ablation lesion (20 W for 30 seconds; max 20 g) was delivered with immediate and persistent disappearance of PVCs even under the administration of Isoproterenol. The patient was discharged from hospital after 2 days without arrhythmia. At 12 months follow-up, no recurrence of AIVR has been observed.

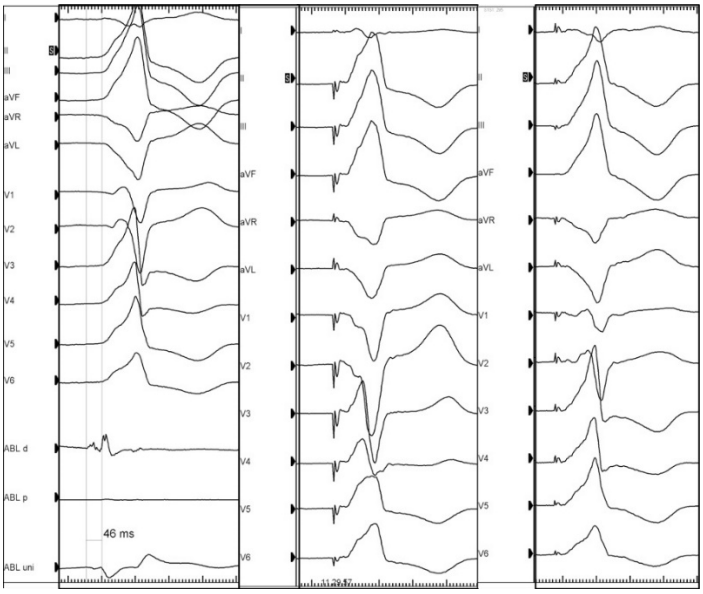

Figure 4: Left panel: 12-lead electrocardiogram of the extrasystole and corresponding intracardiac electrograms showing, on the ablation catheter (Abl d) located at the successful ablation site, an earliest local activation of 46 msec with low voltage and fragmented signals and a QS pattern on unipolar recording. Central panel: pace-mapping from RVOT yields a nonmatching pace map: when compared to the clinical PVC, the pacing morphology has QS in V2 and a later transition in V3. Right panel: pace-mapping from LVOT at the successful ablation site shows a perfect matching pace map.

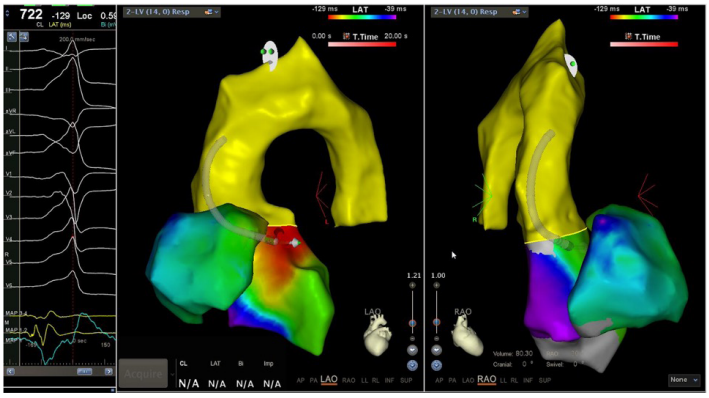

Figure 5: CARTO 3 mapping showing an electroanatomic rendering of the RVOT and LVOT with red areas defining the site of earliest activation near LCC.

Conclusions

We described a clinical case of AIVR in a patient who underwent previous closure of perimembranous subvalvular aortic interventricular defect. Adults with repaired or palliated congenital heart disease are more predisposed to ventricular arrhythmias and the presence of surgical scars coupled with viable but slowly conducting myocardial tissue lead to the creation of the substrate for reentrant VT [31]. In many cases, furthermore, ventricular arrhythmias substrates may be observed in relation to focal stretch-damaged Purkinje tissue. A recent review aimed the history of the main discoveries that lead to the identification and comprehension of AIVR [32]. However, transcatheter ablation of this type of arrhythmia has never been reported in a young patient with previous interventricular septal repair. AIVR is due to enhanced automaticity of an ectopic focus of ventricular origin, but the exact mechanism in a given pathological state or patient remain unclear. Another problem, as showed in this case report, was the differential diagnosis of AIVR and irregular PSVT in which intrinsic properties of the focus itself, or different and not well explained mechanisms of incomplete protection are present. Irregular intrinsic discharge of the ectopic focus is present both in the AIVR [25,26] and in the PSVT [33,34], but more relevant in the former. In PSVT, moreover, irregular interectopic intervals can be due to either incomplete protection of the parasystolic pacemaker during the entire cycle, due to reset [35] or modulation [17], or ectopic-ventricular (E-V) exit block due to occult conduction of the sinus impulses [27,28], as explained above. In this case, yet, the electrocardiographic characteristics based on irregular interectopic intervals in relation to variable frequencies, the always late coupling interval of the first tachycardia beat and long interectopic intervals never mathematically related and multiple of the basic interectopic cycle, made the AIVR diagnosis rather than PSVT. Moreover, CI from the prior QRS complex were highly variable ranging from 550 to 730 msec. Bradfield et al. [36] observed high variability of PVC CI in some patients with PVCs originating in SoV or great cardiac vein (GCV) compared with other regions that occurs at relatively fixed CI. They hypothesized that PVCs from these unusual areas, with narrow and relatively isolated muscle fibers, could occur seemingly randomly because of the lack of electrotonic inhibition due to dense surrounding myocardium. Indeed, this relative isolation may decrease the modulation of the PVC focus by the sinus rhythm focus as described by Takayagi et al [37]. Finally, we performed a low power successful ablation of the LCC ectopic focus. A recent study evaluating 10 patients with VT or PVCs mapped to the LCC, found that an ablation approach using low power was associated with a high immediate success without recurrences while avoiding complications [38].

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