Re-entrant ventricular tachycardia in a postoperative case of tetralogy of Fallot – Ablated successfully under the three-dimensional mapping system

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ABSTRACT

A 47-year-old female underwent cardiac repair for tetralogy of Fallot at the age of 12 years. Subsequently, she was asymptomatic on follow-up. Recently, she presented elsewhere with palpitations and presyncope with documented ventricular tachycardia (VT) having left bundle branch block morphology with inferior QRS axis and late precordial transition. She was reported to have cardioverted and referred to our center for electrophysiology study (EP). She underwent EP study which induced clinical VT which was hemodynamically stable and the mechanism of VT was confirmed as re-entry. With the help of three-dimensional mapping system, VT circuit was identified in the posterior right ventricular outflow tract region between the pulmonary valve and upper end of ventricular septal defect patch. Delivery of radiofrequency energy during VT terminated the tachycardia with no further inducible VT despite aggressive pacing protocols.

Keywords: Catheter ablation, tetralogy of Fallot, ventricular tachycardia

INTRODUCTION

Tetralogy of Fallot (ToF) is the most common cyanotic congenital heart disease irrespective of age, with an incidence of approximately 10% among all congenital heart diseases.[1,2] There is the increasing prevalence of repaired TOF among adults because of improved surgical interventions.[1,2] However, these people are at higher risk for sudden death on long-term follow-up which is related to progressive hemodynamic problems and/or surgical scarring leading to ventricular tachycardia [VT] or ventricular fibrillation which accounts for one third to half of late deaths.[3] VT, in these patients, is usually caused by a macro-re-entrant mechanism although other mechanisms (micro-re-entrant and nonre-entrant mechanisms) are also involved. Re-entry circuits are related to critical isthmuses surrounded by unexcitable structures such as surgical scars, valve annulus, and surgical patches.[4,5]

In 2007 landmark paper by Zeppenfeld *et al.* described several anatomical isthmuses in surgical repaired ToF patients. They were reported to be between (1) tricuspid annulus and right ventricular outflow tract (RVOT) patch/right ventricular incision (isthmus 1), (2) between right ventricular incision site and pulmonary valve (isthmus 2), (3) between pulmonary valve and ventricular septal defect (VSD) patch (isthmus 3), and (4) between VSD patch and tricuspid annulus (isthmus 4). These critical isthmuses are identified as the sources for macro-re-entry.[5] The most frequent isthmus was

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present between the pulmonary valve and the VSD patch (isthmus 3), the second was between the tricuspid valve and the RVOT patch (isthmus 1).

In 2017, Kapel *et al.* on the electrophysiological properties of these anatomical isthmuses suggest that an anatomical isthmus in the RVOT region of ToF will only support sustained VT when it is long, narrow, slowly conducting, and low voltage with low conduction velocity index appears to be the most critical variable.[6]

The feasibility of catheter ablation, applying conventional mapping techniques, has been reported.[7] However, complex anatomy, hypertrophied myocardium, broad isthmuses, hemodynamic instability, or non inducibility of the VTs may explain ablation failure in 50% of cases.[8,9]

In this report, we describe a successful VT ablation guided by three-dimensional (3D) mapping system in an adult patient who has undergone cardiac repair for ToF at the age of 12 years.

CASE REPORT

The patient was a 47-year-old female who had undergone cardiac repair for ToF at the age of 12 years. She presented elsewhere with palpitations and presyncope with documented left bundle branch block (LBBB) VT [Figure 1a]. She was reported to have electrically cardioverted to sinus rhythm [Figure 1b] in view of her borderline hemodynamic status. An echocardiogram performed later showed no residual VSD, moderate RVOT obstruction, moderate pulmonary regurgitation, and good RV function.

The patient was taken up for an EP study after informed consent. 6F quadripolar diagnostic EP catheter was introduced from the right femoral vein (RFV) and placed across the tricuspid valve to record the His bundle activation and in the RV apex for pacing. Simultaneous recording of the surface electrocardiogram and intracardiac electrograms were done using BARD EP system. 3D mapping of the RV, using the NavX (St. Jude Medical, St. Paul, MN,

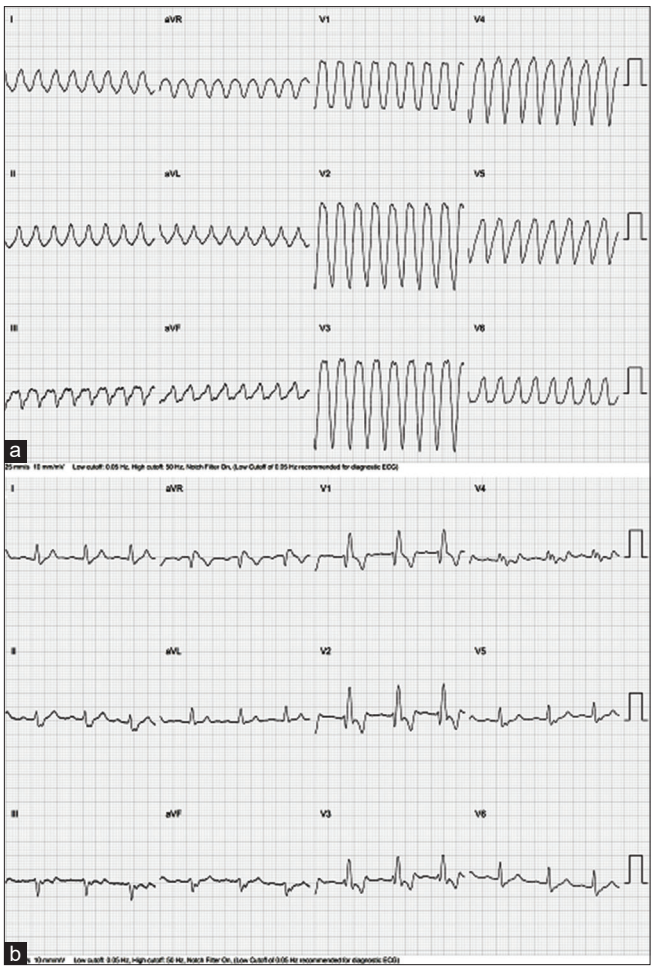

Figure 1: (a) Surface electrocardiogram showing ventricular tachycardia with left bundle branch block morphology. (b) Surface electrocardiogram during sinus rhythm

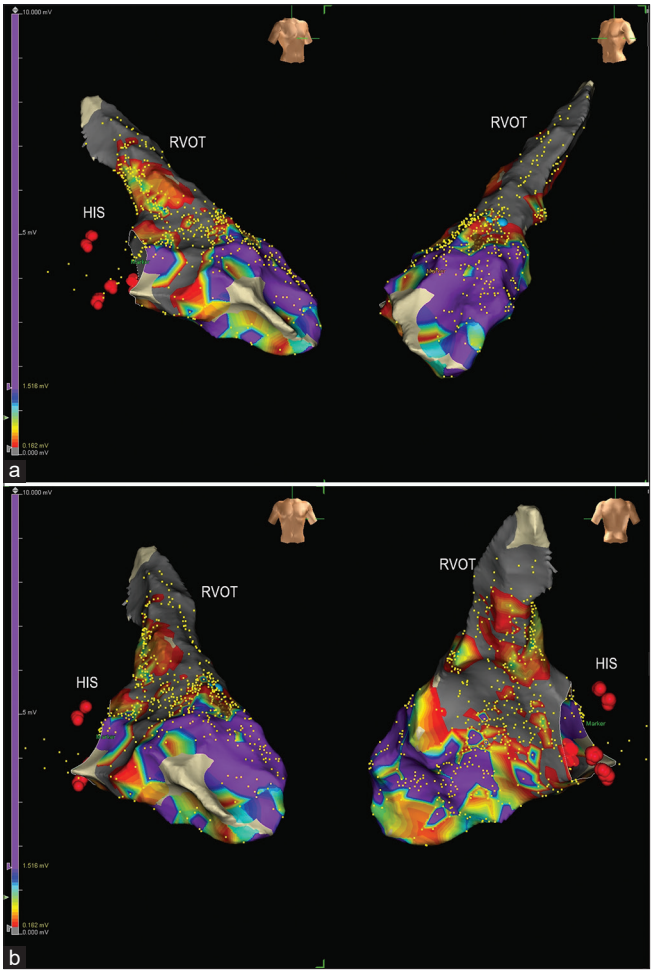

Figure 2: (a) Voltage map of the right ventricle in right anterior oblique (RAO) and leftanterior oblique (LAO) views. (b) Voltage map of the right ventricle in anteroposterior and posteroanterior views

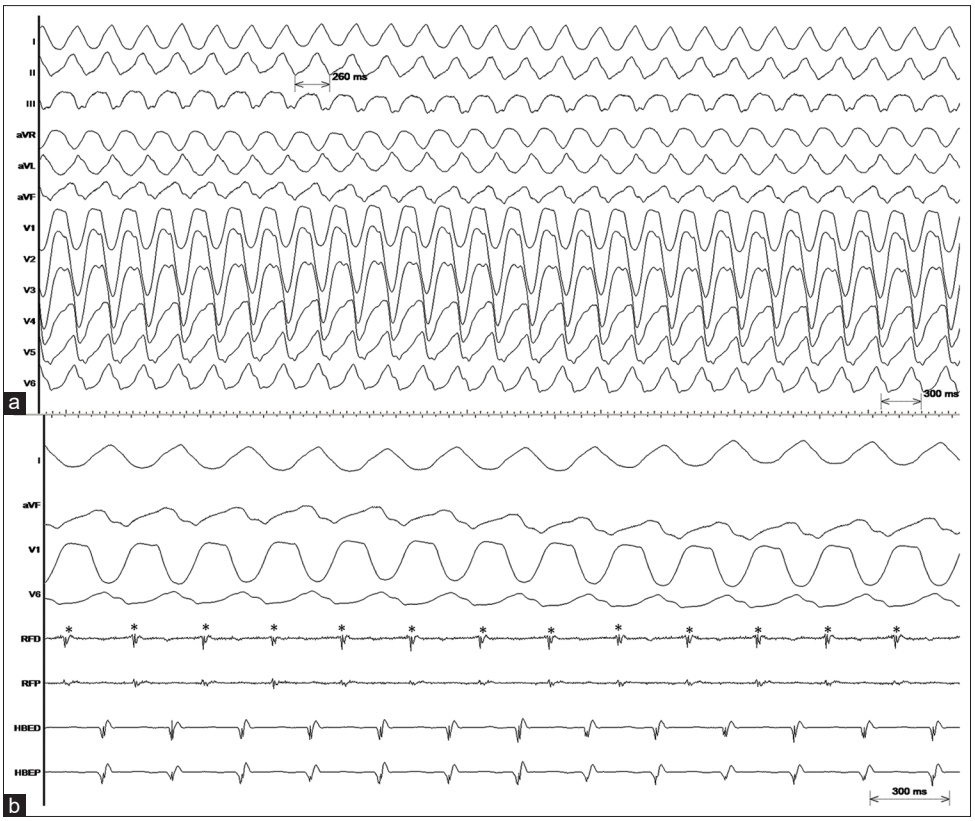

Figure 3: (a) 12-lead surface electrocardiogram of the ventricular tachycardia induced in EP lab. (b) Surface electrocardiogram-leads I, aVF, V1, V6 and intracardiac electrograms – RFD (mapping and ablation distal), RFP (mapping and ablation proximal), his bundle electrogram distal, his bundle electrogram proximal – showing mid diastolic potentials (*) during the ventricular tachycardia with mapping catheter positioned at the posterior wall of right ventricular outflow tract

USA) mapping system and FlexAbility™ mapping and ablation catheter, St. Jude Medical, F curve (4 mm saline irrigated catheter, with deflectable-tip), during sinus rhythm and VT was performed as previously reported.[10] Voltage map [Figure 2a and b] of RV was created in sinus rhythm which showed scar [as defined by local bipolar electrogram voltage of <0.15mV; scar identified as gray-colored areas] in the RVOT and basal ventricular septum. Gray-colored regions identified by voltage map in fact represented surgically created scar due to VSD patch and RVOT patch. Purple areas are healthy areas with local bipolar electrogram voltage of more than 1.5 mV. Border zones (defined by local electrogram voltage between 0.15mV and 1.5 mV) were identified in the posterior RVOT between the pulmonary valve and the basal septal scar, which represented the VSD patch. This in fact corresponded to the isthmus 3 where mid diastolic potentials were recorded during the tachycardia [Figure 3b].

VT induction was done with programmed ventricular stimulation. With single ventricular extra (S1:S2) of 500:240 ms, hemodynamically stable VT with tachycardia cycle length (TCL) of 260 ms, LBBB morphology, inferior QRS axis with late precordial transition [Figure 3a] which was similar to clinical VT got induced.

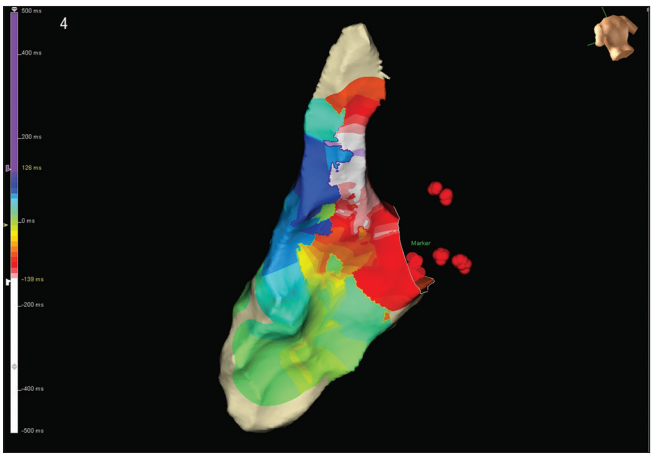

Figure 4: Activation mapping during ventricular tachycardia showing "early meets late" pattern suggestive of re-entry at posterior right ventricular outflow tract

As the VT was hemodynamically stable, activation map was created using the NavX (St. Jude Medical, St. Paul, MN, USA) 3D mapping system which showed "early meets late" pattern (white color representing early activated area meeting blue color representing late activated area) suggestive of re-entry in the basal posterior RVOT region [Figure 4]. Slowing of conduction during

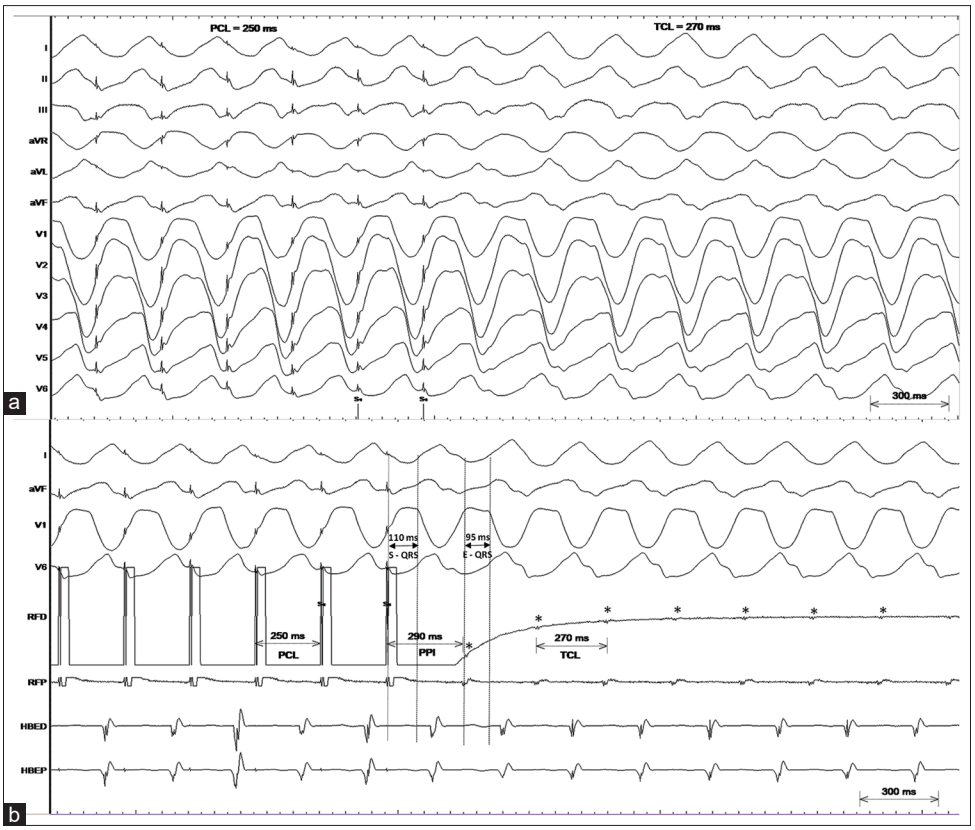

Figure 5: (a) Surface electrocardiogram during ventricular entrainment showing entrainment with concealed fusion. (b) Surface electrocardiogram-leads I, aVF, V1, V6 and intracardiac electrograms – RFD (mapping and ablation distal), RFP (mapping and ablation proximal), his bundle electrogram distal, his bundle electrogram proximal – showing post entrainment response

tachycardia (suggestive of isthmus) was appreciated in the propagation map [Video 1].

Entrainment maneuver [Figure 5a and b] was also performed as the VT was hemodynamically stable. Ventricular overdrive pacing was performed from posterior RVOT region during VT at a cycle length 20 ms less than the TCL. Entrainment with concealed fusion [Figure 5a] was noted with similar QRS morphology during ventricular pacing and during VT. Postentrainment responses (response after cessation of pacing with VT continuing) showing postpacing interval (PPI) minus TCL of <20 ms, stimulus to QRS/VT cycle length of 40%, and stimulus to QRS minus electrogram to QRS <20 ms are very suggestive that ventricular entrainment was being performed from the central part of critical isthmus of the tachycardia circuit which corresponded to the isthmus 3.

The VT circuit could be mapped to the posterior RVOT region with demonstration of critical isthmus in the region between pulmonary valve and basal septal scar representing VSD patch. Delivery of radiofrequency (RF) energy with FlexAbility™ mapping and ablation catheter, St. Jude Medical, F curve (4 mm saline irrigated catheter, with deflectable-tip) with following settings (power - 30W, temperature - 43°C, flow rate 17 ml/min) at this area resulted in termination of VT [Figure 6a]. Consolidation RF impulses were delivered around this area, and it was connected to upper end of the basal septal scar. Substrate modification was also done in the rest of the posterior RVOT region targeting late potentials and fractionated signals. There was no further inducible VT despite aggressive pacing protocols [Figure 6b].

DISCUSSION

We presented a case of successful VT ablation guided by 3D mapping at isthmus 3 in a surgically repaired ToF patient with documented episode of spontaneous VT. Mapping studies demonstrated in surgically repaired ToF patients with spontaneous VT substrates are usually located in anatomically defined isthmuses. Anatomical isthmuses are result of malformation and the type of repair. Out of the 4 isthmuses described by Zeppenfeld *et al.*, isthmus 3 is the most common substrate for VT with abnormally low conduction velocity index.[5] In the postmortem histological analysis, isthmus 3 had the highest degree of fibrosis, thereby providing the potential histological substrate for slow conduction. Isthmus 3 between the VSD patch and the pulmonary valve is the most common substrate for VT in patients who underwent ToF repair through whatever surgical

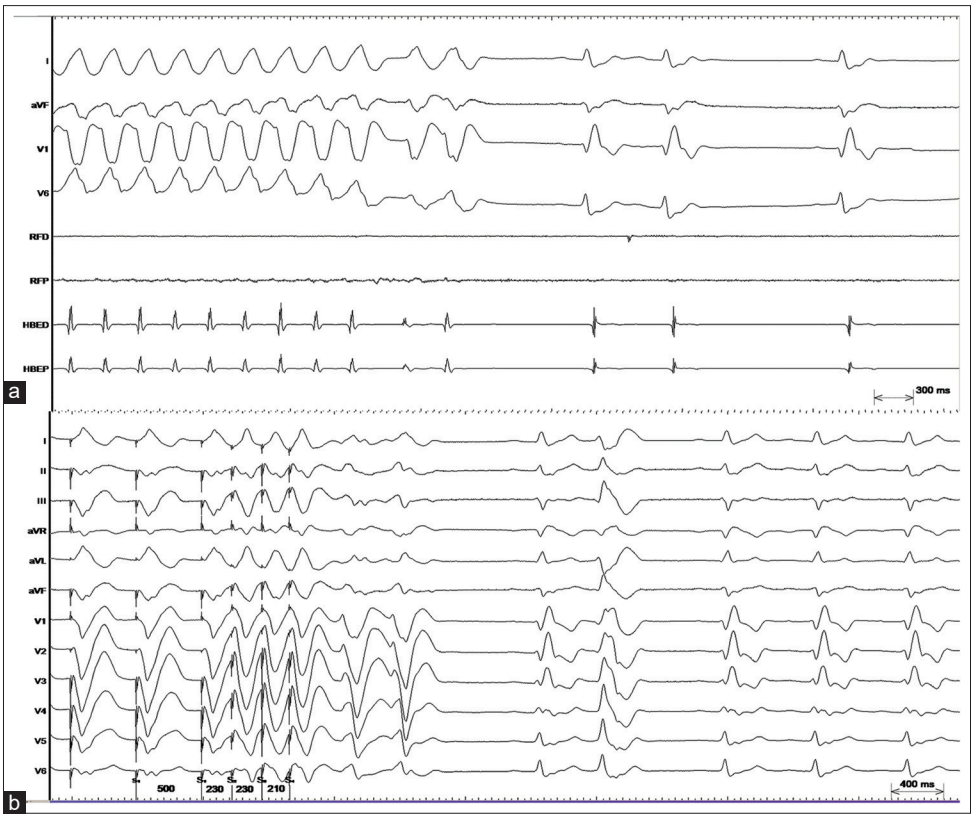

Figure 6: (a) Surface electrocardiogram-leads I, aVF, V1, V6 and intracardiac electrograms – RFD (mapping and ablation distal), RFP (mapping and ablation proximal), his bundle electrogram distal, his bundle electrogram proximal – showing termination of ventricular tachycardia on radiofrequency ablation. (b) Surface electrocardiogram-leads I, aVF, V1, V6, and intracardiac electrograms – RFD (mapping and ablation distal), RFP (mapping and ablation proximal), his bundle electrogram distal, his bundle electrogram proximal – showing no inducible ventricular tachycardia despite delivering triple extra stimuli from the right ventricular apex

technique either it is classical trans-ventricular or a trans-atrial: Trans-pulmonary approach.[11]

CONCLUSION

VT, in surgically repaired ToF patients, is usually caused by a macro-reentrant mechanism with substrate usually located in anatomically defined isthmuses and the re-entrant VT is amenable to RF ablation.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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