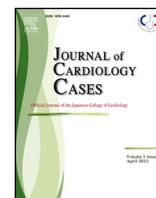




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Transcoronary mapping and chemical ablation of ventricular tachycardia in no-entry left ventricle

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ABSTRACT

It is challenging to perform ablation of ventricular tachycardia (VT) from the left ventricle (LV) in patients without catheter access to the LV. A 50-year-old man was referred to our hospital for VT. He underwent mechanical aortic and mitral valve replacement for infective endocarditis and embolic myocardial infarction in the left ventricular inferior wall during a surgery. Anti-arrhythmia drugs (AADs) such as sotalol and bisoprolol were initiated and implantable cardioverter defibrillator was implanted. However, 2 months after discharge, he was admitted again for cardiac implantable electronic device (CIED) infection and underwent complete CIED system removal. During hospitalization, VT easily occurred despite the use of AADs. We decided to perform transcoronary chemical ablation to treat this drug-refractory VT. A 0.014-inch guide-wire and a micro-catheter were advanced into coronary arteries. Pace map was conducted using a guide-wire and the micro artery branch feeding the VT exit area was detected. Ethanol infusion to this branch and the slightly basal side of the area eliminated the VT. We successfully treated VT in the no-entry LV by wire-guided mapping and ethanol ablation via coronary arteries. VT has not recurred during the follow-up period of 12 months.

< **Learning objective:** It is challenging to perform ventricular tachycardia (VT) ablation in patients with mechanical aortic and mitral valve replacement, because there is no catheter access to the left ventricle. Mapping via coronary arteries using guide-wires enables pace-mapping, finding VT exit sites, and identification of the appropriate branches for ethanol infusion. Therefore, transcoronary mapping and chemical ablation may be an alternative treatment for VT in a no entry left ventricle situation.>

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Introduction

Implantable cardioverter defibrillator (ICD) has been an established therapy for preventing sudden cardiac death in patients with ventricular arrhythmias [1]. Catheter ablation is also an effective treatment for ventricular tachycardia (VT), in addition to ICD implantation and medication such as amiodarone. VT ablation reduces ICD shocks, which is of great benefit for patients with implanted ICD for VT as repeated ICD shocks result in significant mortality [2,3]. VT ablation has shown the above-mentioned benefits, but it is challenging to perform VT ablation for patients with VT in the left ventricle (LV) after mechanical aortic and mitral valve replacement because both retrograde aortic approach and

atrial transseptal approach cannot be performed for these patients. We performed successful elimination of VT in a patient without catheter access to the LV. Here, we report transcoronary mapping and chemical ablation using a guide-wire.

Case report

A 50-year-old man was referred to our hospital for VT. He underwent mechanical aortic and mitral valve replacement for infective endocarditis and embolic myocardial infarction in the left ventricular inferior wall during a surgery when he was 26-years-old. Seven years after the first operation, mechanical double-valve replacement was performed again because of valve malfunction. ICD was implanted and anti-arrhythmia drugs (AADs) such as sotalol 80 mg/day and bisoprolol 5 mg/day were initiated. Two months after discharge, he was admitted again for cardiac implantable electronic device (CIED) infection and underwent complete CIED system removal. During hospitalization, VT easily

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occurred despite the use of AADs. We decided to perform transcatheter chemical ablation to treat this drug-refractory VT, as an endocardial approach was impossible because of mechanical aortic and mitral valves, and a sub-xiphoid epicardial approach seemed to be difficult for adhesions considering that an open-heart surgery was performed twice for this patient. The ethical committee of our hospital approved the study, and written informed consent was obtained from the patient. Multi-electrode catheters were positioned at the His recording lesion, right ventricular apex, and middle cardiac vein (MCV). Electroanatomical mapping was performed using a three-dimensional mapping system (CARTO 3 System, Biosense-Webster, Diamond Bar, CA, USA). A programmed stimulation induced hemodynamically stable VT (Fig. 1A). The pace map from the right ventricle or MCV did not match the clinical VT morphology; therefore, the VT exit site seemed to be in the LV. Coronary angiography was performed to approach the LV via coronary arteries, which showed two major posterior descending arteries (PDAs) from the left circumferential artery (Fig. 1B, C). A 0.014-inch angioplasty guide-wire (Run-through NS®, Terumo, Tokyo, JAPAN) and a micro-catheter (Caravel microcatheter, Asahi intec, Tokyo, JAPAN) were advanced into the PDAs. Micro-arteries from PDAs were contrasted by the tip injection technique using the micro-catheter (Fig. 1D, E), and pace-mapping was performed using the guide-wire. One electrode of the catheter positioned at the right ventricular apex was used for an indifferent electrode, and the guide-wire was connected to the cathode. Distal portion of the guide-wire <2 mm was uncovered with the micro-catheter. Therefore, the pacing portion of the guide-wire was less than 2 mm, which enabled selective myocardial capture by unipolar pacing. The pace-mapping from the middle portion of the PDAs was quite different from the clinical VT

morphology (Fig. 2A). Pace-mapping from the distal branch of PDAs was again conducted using a guide-wire and the pace-mapping at this site resembled the clinical VT morphology (Fig. 2B). Cold saline infusion to this branch terminated the VT with reproducibility. Ethanol infusion to this branch and the slightly proximal side of the branch eliminated the VT. Further programmed stimulation induced another VT of the left bundle branch block morphology. We started mapping by the ablation catheter in RV, but the earliest activation site in RV was later than the potential recorded by the electrode catheter positioned at the MCV. Radiofrequency application to the earliest activation site in RV did not terminate the second VT. Therefore, mapping via coronary arteries was re-attempted. The guide-wire was advanced to another branch of the PDA (Fig. 3A), which was feeding the area around the proximal side of the catheter at the MCV. Intracoronary electrocardiogram from this site preceded the onset of the VT by 41 ms (Fig. 3B). An over-the-wire balloon (Ryujin-plus OTW®, Terumo) was deployed, and 100% ethanol infusion to this branch directly terminated this VT (Fig. 3C, D). Creatine phosphokinase was elevated up to 745 IU/L on the day following the ablation. VT has not recurred during the follow-up period of 12 months.

Discussion

It is challenging to ablate VT in the LV in patients without catheter access to the LV. The usefulness of epicardial catheter ablation and ventricular transseptal approach in patients with mechanical double-valve replacement has been reported, but postoperative adhesion or complications are an often-encountered problem, and such procedures can be conducted at limited hospitals by experienced operators [4,5]. The efficacy of transcatheter

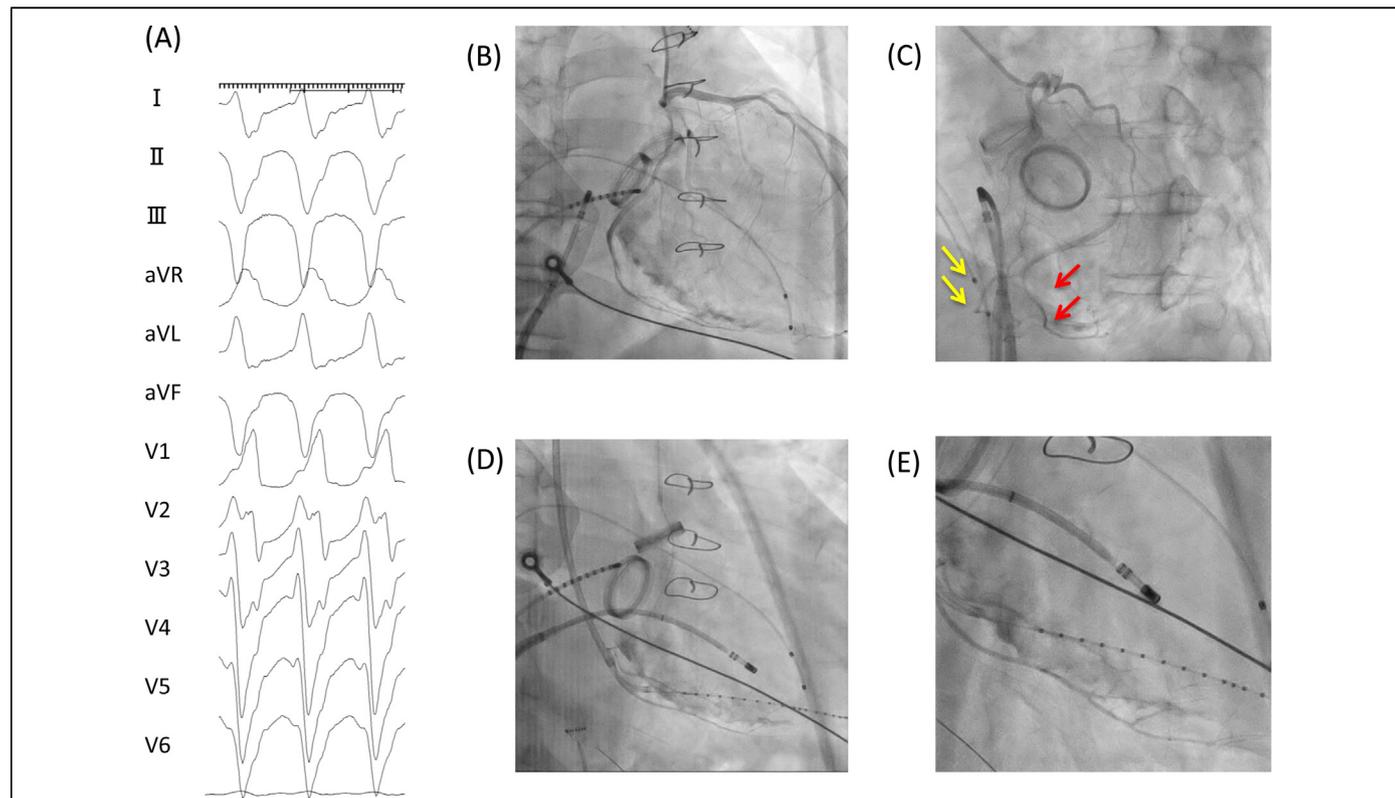
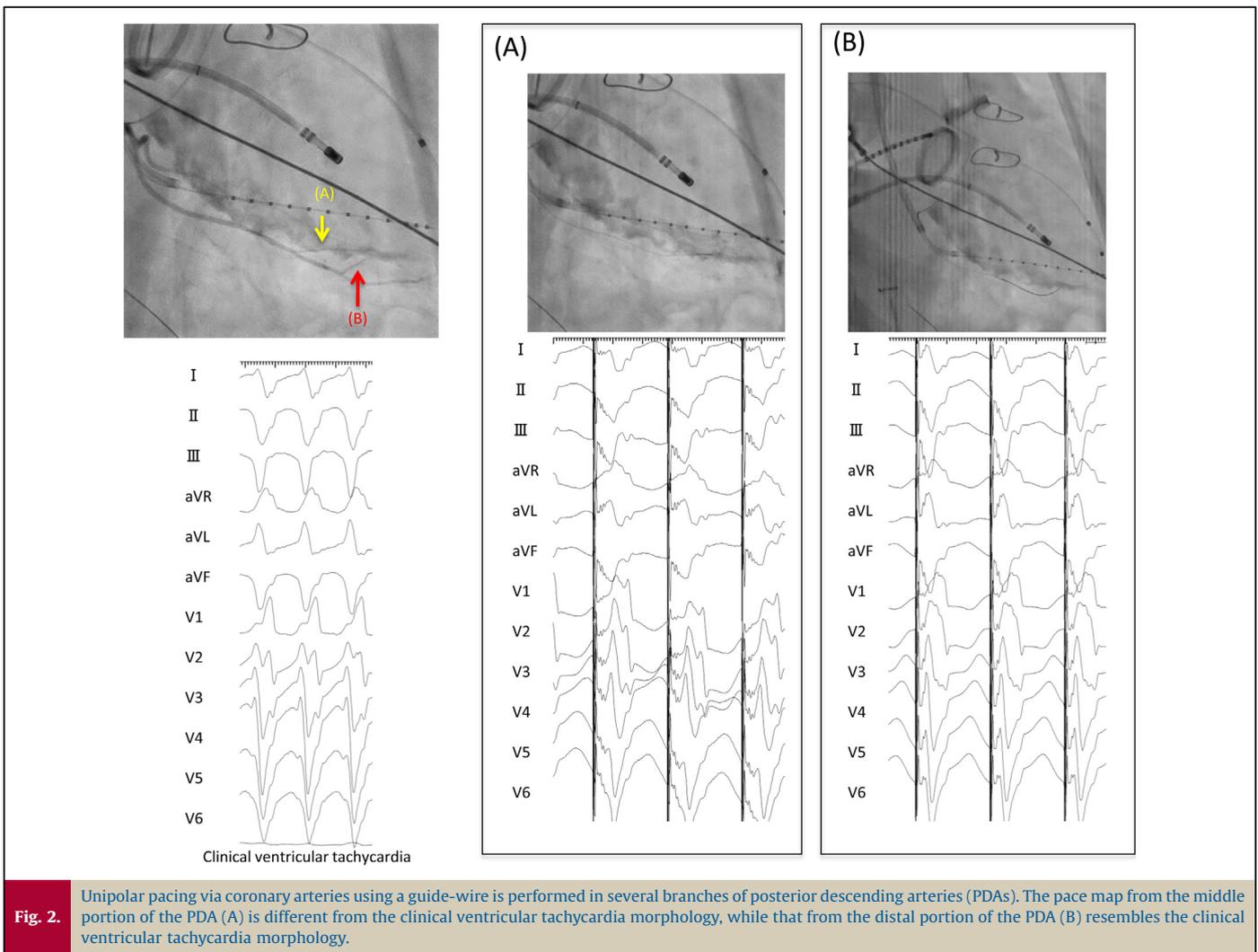


Fig. 1.

(A) Double stimuli from the right ventricular apex (600 ms-290 ms-260 ms) induces a ventricular tachycardia, which has a right bundle branch block morphology with a superior axis and deep S wave in V3-5. (B, C) Coronary angiography reveals two major posterior descending arteries (PDAs) from the left circumferential artery. (Red and yellow arrows). Panels B and C are the right anterior oblique and left anterior oblique views. (D, E) A 0.014-inch angioplasty guide-wire and micro-catheter are advanced to PDAs. Micro-arteries from PDAs are contrasted by the tip injection technique using the micro-catheter.



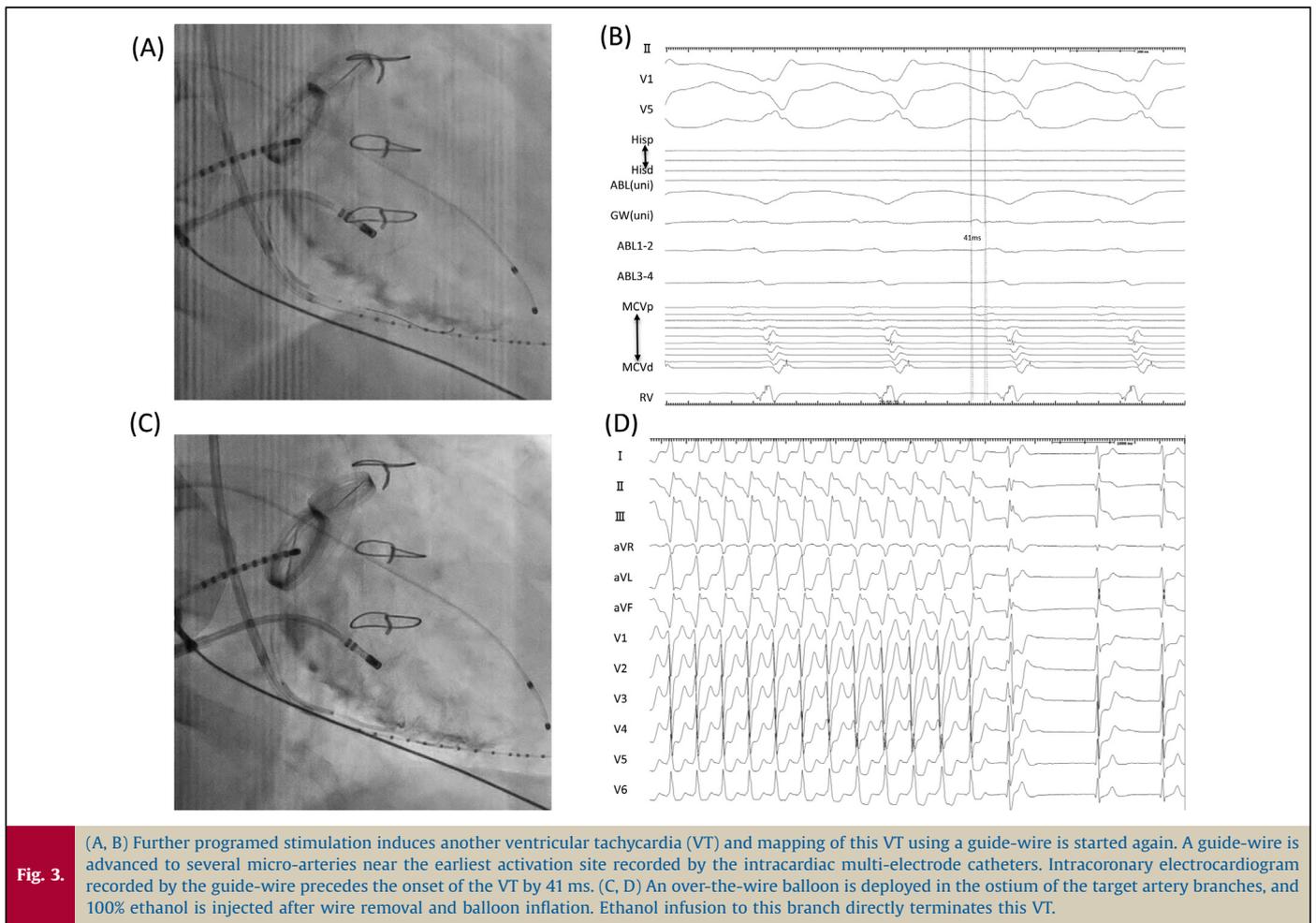
chemical ablation has also been reported before, but there were some problems concerning myocardial damage or atrioventricular block [6]. Transcoronary mapping using a guide-wire and chemical ablation via coronary arteries are not influenced by mechanical valves; therefore, this method can overcome problems of catheter access. The present method enables to perform ethanol infusion only to the vessels near the exit site and critical isthmus of the VT. Selective ethanol infusion may result in a smaller myocardium damaged and avoidance of injection to the vessels feeding atrioventricular node.

A high recurrence rate is associated with chemical ablation for VT, as reported before [7]. In the present case, the second VT was induced after the elimination of the first one. We fortunately terminated both the first and the second VTs because there were visible coronary branches feeding the target myocardium. In the present method for the second VT, whether ethanol infusion could eliminate the VT or not depended on the coronary artery anatomy. Retrograde coronary venous ethanol ablation can be an alternative method in cases with no visible arteries feeding the target myocardium [8].

We identified the artery near the exit site of first VT by pace-mapping using a coronary guide-wire, but the appropriate branch for ethanol infusion was not decided by pace-mapping alone. Cold saline injection and the termination of the VT were useful for detecting the culprit artery. Selective ethanol infusion only to the artery near the VT exit was not enough to treat the VT in this method, because VT exit site is like a delta area [9], and therefore

ablation to the central isthmus is needed to treat VTs in chemical ablation as well as catheter ablation. Entrainment pacing was a representative method to determine the central isthmus of VT circuit; however, in the present case, entrainment pacing was impossible because of high pacing threshold in the scar area. This was a limitation of this case report. Therefore, we identified the high pacing threshold area with endomyocardial calcification as a scar area (i.e. arrhythmogenic substrate). Ethanol infusion was added to the branch in the scar area near the exit site, which resulted in non-inducibility of the clinical VT. In no entry LV, voltage map of the endocardium cannot be obtained. Therefore, enhanced cardiac magnetic resonance (CMR) imaging may be useful if there is no contra-indication of enhanced CMR. If entrainment pacing was performed in the scar area by epicardial pacing using guide-wire and then central isthmus was detected, more selective ethanol infusion to the central isthmus could be possible and myocardial damage might be small.

Pace-mapping via coronary arteries using coronary guide-wires does not show a perfect pace map for several reasons as follows: (1) pace-mapping via coronary arteries is epicardial pacing, although VT exits in patients with old myocardial ischemia scar were endocardial sites. (2) Coronary guide wires may not be positioned at the exact site of VT exit, because the position of the guide-wire completely depends on the anatomy of coronary arteries. (3) Pacing artifact was prominent by unipolar pacing with a single coronary guide-wire.



There were advantages and disadvantages between the unipolar and bipolar pacing. Advantages of unipolar pacing in the present case were simplicity of the procedure and pacing threshold. Unipolar pacing was easily performed by the method described in the case report. Eventually, a good pace-map was obtained although the artifact was a disadvantage of unipolar pacing. Additional guide-wire insertion for an anode was needed for bipolar pacing. However, adding a guide-wire to the small branch of PDAs was a complex procedure. This was why we chose unipolar pacing. We advanced the guide-wire into the area of the old myocardial infarction for pace-mapping; therefore, the pacing threshold at the area was high. Previous reports showed that unipolar pacing threshold was lower compared with the bipolar pacing threshold [10], which was of great advantage for unipolar pacing in the present case.

In conclusion, transcatheter mapping and chemical ablation using coronary guide-wires may be an alternative, less invasive treatment for VT in the LV in patients with mechanical double-valve replacement. This method can be performed at hospitals other than experienced centers.

Disclosures

None.

Declarations of interest

None.

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