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Case Report

Radiofrequency Catheter Ablations of Left Ventricular Arrhythmias Originating from Posteromedial Followed by Anterolateral Papillary Muscles

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Abstract

A 61-year-old male presented fatigue, and ambulatory monitoring demonstrated frequent isolated premature ventricular beats (PVBs) with superior axis and right-bundle-branch-block (RBBB) configuration. Echocardiogram showed diffuse hypokinetic left ventricular (LV) wall motion and subnormal LV ejection fraction (LVEF). In electrophysiological study (EPS), pace map was optimal at the posteromedial papillary muscle (PM). Low-frequency mid-diastolic Purkinje potential (P1) preceded surface QRS complex of PVBs by 20 msec, and high-frequency Purkinje potential (P2) was observed immediately before QRS complex of sinus beats and PVBs. After successful radiofrequency ablation for PVBs arising from posteromedial PM, LVEF restored. However, one and half year later, PVBs showing inferior axis and RBBB appeared and LVEF declined. EPS and ablation were performed again and PVBs originating from anterolateral PM were eliminated. Considering that LV arrhythmia arising from PMs is refractory to ablation due to anatomical and technical reasons, careful follow-up is required.

Key Words : catheter ablation, papillary muscle, Purkinje potential, ventricular arrhythmia

Introduction

Ventricular arrhythmias originating from left ventricular (LV) papillary muscles (PMs) are emerging as a distinct form of idiopathic ventricular arrhythmias. This characteristic form of LV arrhythmias shows variety in electrocardiographic (ECG) manifestation and arises from posteromedial or anterolateral PMs. This type of LV arrhythmia is reported to occur originally by non-reentrant focal mechanisms, because PM arrhythmia is inducible by exercise or isoproterenol

infusion, whereas it is not entrained and not evoked by programmed stimulation¹⁾. However, reentrant mechanisms involving sub-endocardial Purkinje network is recently suspected²⁾. PM arrhythmia is a good candidate of radiofrequency (RF) catheter ablation (RFCA)^{1)~3)}, although recurrence rate is relatively high. This is attributed to individual variety and complex anatomy of PMs and technical problem of ablation catheter stability during RF energy application. In this sense, RFCA of PM arrhythmia is still challenging⁴⁾⁵⁾. Here, we report a case of successful RFCA

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of frequent isolated premature ventricular beats (PVBs) originating from posteromedial and followed by anterolateral PM and causing reversible impairment of LV function.

Case History

In November 2014, a 61-year-old male complained of general fatigue. This patient was referred to our hospital for the treatment of documented frequent isolated PVBs, which had been refractory to mexiletine, verapamil and bisoprolol titration. After admission, 12-lead ECG demonstrated sinus rhythm associated with isolated PVBs showing left-superior axis and right-bundle-branch-block (RBBB) configuration (Fig. 1A). Ambulatory monitoring showed frequent monomorphic PVBs (about 33,000 beats per day) without any runs of ventricular tachycardia (VT). The frequency of PVBs in the daytime was equivalent to that in the nighttime. Treadmill exercise test did not increase the frequency of PVBs. Transthoracic echocardiogram showed diffuse hypokinetic LV wall motion and subnormal LV ejection fraction (LVEF) of 45% without any structural heart diseases. Electrophysiological study (EPS) and RFCA were performed after obtaining written informed consent and discontinuing antiarrhythmic drugs. Multipolar electrode catheters were inserted from jugular vein into the coronary sinus (CS) and from right femoral vein to place at the His bundle region and right ventricular apex. During EPS, spontaneous isolated PVBs were recorded but VT was not induced by rapid or programmed stimulation. THERMOCOOL (Biosense Webster, Inc., Diamond Bar, CA, USA) irrigation catheter (7.5 Fr) was advanced into LV through aorta in a retrograde direction. Entire LV geometry was obtained by inserting SOUNDSTAR (Johnson & Johnson, Co. Ltd., New Brunswick, NJ, USA) catheter into LV. When ablation catheter was attached to the surface of posteromedial PM, low-frequency mid-diastolic Purkinje potential (P1) preceded QRS onset of PVBs by 20 msec, and

high-frequency presystolic Purkinje potential (P2) was observed immediately before the ventricular activation of sinus beats and PVBs (Fig. 1B). Although imperfect, pace mapping was optimal by ablation catheter at this position (Fig. 1C). Entrainment pacing was not performed because PVBs were isolated. During RF application to this site (the maximum power of 40W and the maximum temperature of 41°C), repetitive ventricular responses were observed. Focal ablation was added along with the longitudinal axis of posteromedial PM. Because PVBs with morphologies different slightly from those of initial PVBs remained, additive circumferential RFCA was performed around the base of posteromedial PM under the guidance of repetitive pace mappings⁴. After the provocation test using isoproterenol, PVBs arising from posteromedial PM were confirmed to be eliminated. This patient became asymptomatic, PVBs were reduced dramatically (<30 beats per day), and LVEF was restored to 63%.

One and half year after the initial RFCA, this patient presented with the same symptoms again. ECG documented PVBs showing inferior axis and RBBB morphology (Fig. 2A) which were frequent in ambulatory monitoring (>30,000 beats per day), and LVEF declined to 41% in August 2016. Because of his clinical course and PVBs morphology, PVBs were suspected to be arising from anterolateral PM³, and secondary EPS and RFCA were performed. Similarly to the first session, ablation catheter was advanced *via* aorta into LV in a retrograde approach under the guidance of LV geometry reconstructed by 3-D mapping system with image integration (Biosense Webster). High-amplitude and high-frequency potential reflecting exit pathway activation preceded QRS onset of PVBs by 32 msec (Fig. 2B) and pace map was optimal when ablation catheter was attached to the surface of anterolateral PM (Fig. 2C). PVBs disappeared by repetitive RFCA at the mid-portion and around the base of anterolateral PM under the same ablative condi-

Fig. 1A

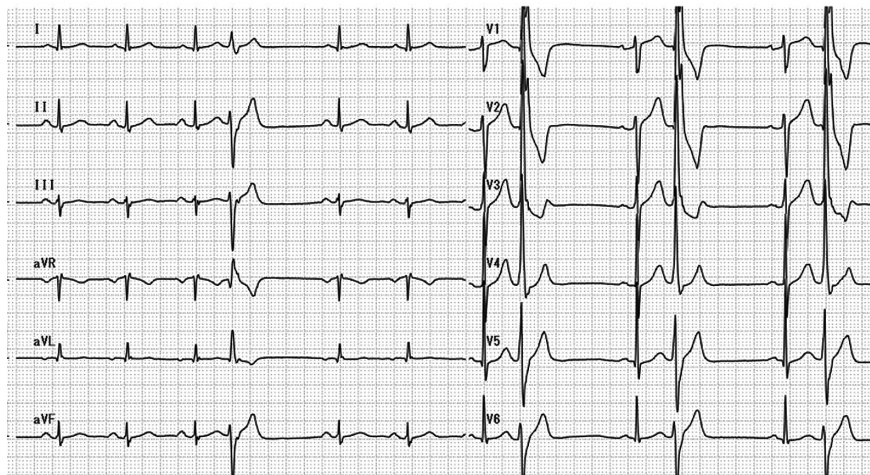


Fig. 1B

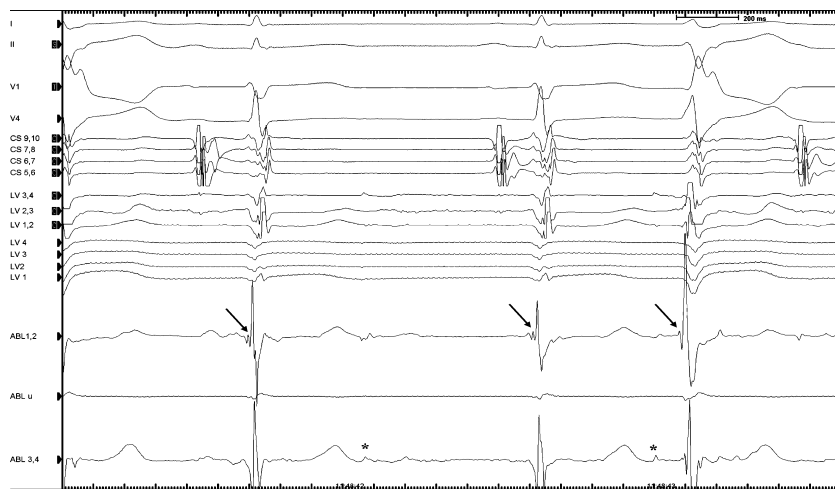


Fig. 1C

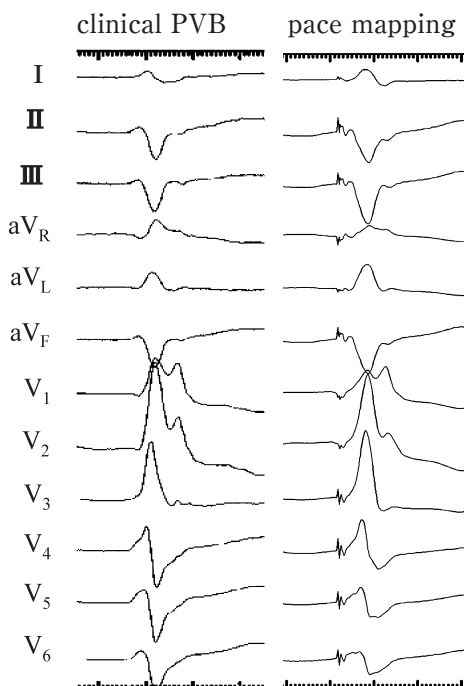


Fig. 1 (A) ECG at the first admission demonstrated sinus rhythm associated with frequent premature ventricular beats (PVBs) showing right-bundle-branch-block (RBBB) pattern, left-superior axis and a transitional zone of V₅. (B) Mid-diastolic Purkinje potential (P1) was recorded at the successful ablation site in sinus beats (stars), i.e., the first P1 (left star) was blocked and the second P1 (right star) was recorded 20 msec prior to the QRS onset of PVBs. High-frequency presystolic Purkinje potential (P2) was observed immediately before the onset of ventricular activation of both sinus beats and PVBs (arrows). (C) Pace mapping was optimal, although imperfect, when ablation catheter was attached to the surface of posteromedial papillary muscle (PM). ABL, electrograms recorded by ablation catheter ; CS, coronary sinus electrograms.

Fig. 2A

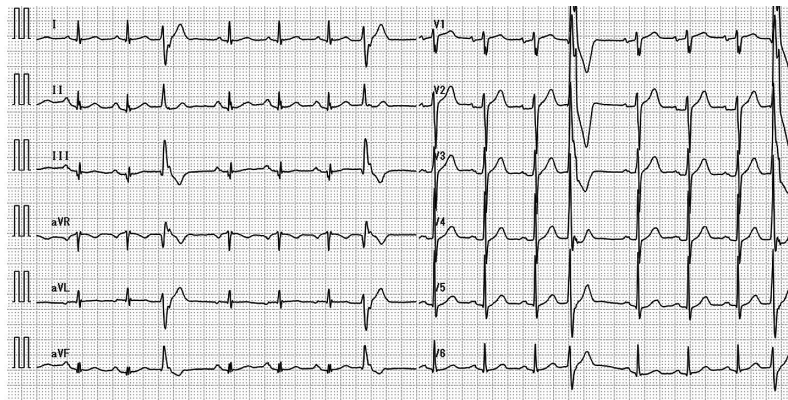


Fig. 2B

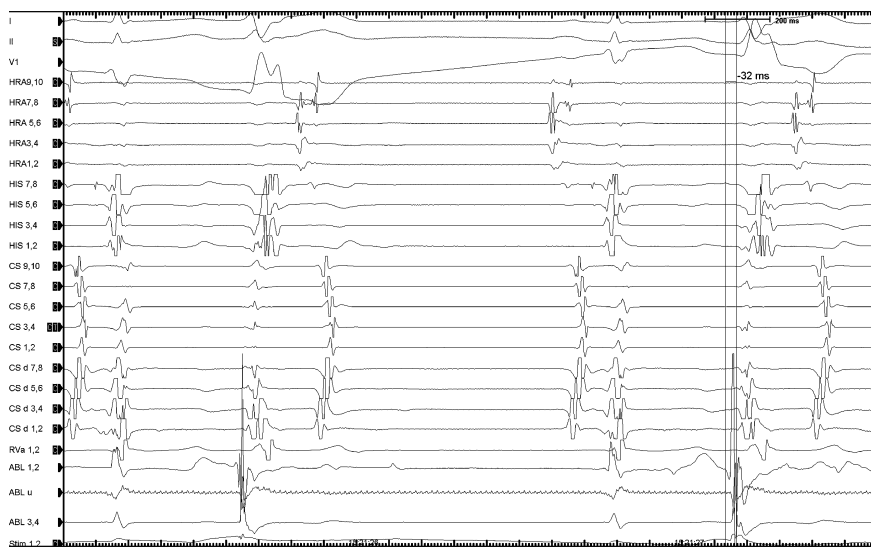


Fig. 2C

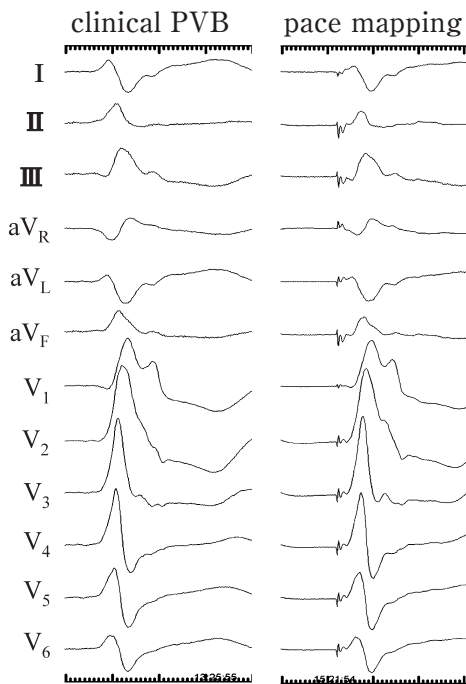


Fig. 2 (A) ECG at the second admission demonstrated sinus rhythm associated with frequent PVBs showing RBBB morphology, inferior axis and a transitional zone of V₆. Note that QRS morphologies of III and aV_F leads in sinus beats differ from those in Fig. 1 (A), implying potential ablative lesions on the posteromedial PM. (B) High-amplitude, high-frequency potential was recorded 32 msec prior to the surface QRS complex of PVBs at the successful ablation site. (C) Perfect pace map was obtained by ablation catheter attached to the mid-portion of anterolateral PM.

tion as in the first session. This patient became asymptomatic again, ambulatory monitoring recorded about 30 PVBs per day, and LVEF was restored to 68%. Fluoroscopic and 3-D mapping images in respective procedure were demonstrated in Fig. 3 (A, B, D, E). Intracardiac echocardiogram was performed to facilitate guidance of ablation catheter by inserting intracardiac echo probe into the right ventricle (Fig. 3C). Postablative mitral regurgitation did not occur. Considering recurrence of PM-arising arrhythmias is not rare^{1)–3)}, careful follow-up is still ongoing.

Discussion

We presented a case of idiopathic frequent PVBs originating from posteromedial followed by anterolateral PM with an interval of one and half year between the first and second session of RFCA. PMs are known as a distinctive source of LV arrhythmia in structurally normal and abnormal hearts. PM arrhythmia accounts for 4 to 12% of the whole idiopathic LV arrhythmias and is generally not fatal but drug-refractory and often impairs LV function and quality of life⁵⁾.

ECG of PM Arrhythmia

PM arrhythmia includes wide variety such as isolated PVBs, sustained or non-sustained VT and ventricular fibrillation triggered by PM-arising PVBs⁵⁾⁶⁾. In literature, posteromedial PM dominates over anterolateral PM¹⁾, whereas the former is equivalent to the latter as an arrhythmogenic focus⁴⁾. In our case, one and half year after the initial RFCA targeting PVBs arising from posteromedial PM, PVBs originating from anterolateral PM were ablated. PVBs from posteromedial PM demonstrated superior axis, whereas PVBs from anterolateral PM showed inferior axis, although both types of PVBs had QRS morphologies of RBBB and similar transitional zone (Fig. 1A, 2A), indicating that different foci among the two PMs are reflected more in the limb-leads than in the precordial leads. The anterolateral PM

originates from the anterolateral LV free wall, and posteromedial PM protrudes from the inferoseptal wall. The level of attachment in the former is always higher than that in the latter, and this is the main reason for the different axes between the two types of PVBs¹⁾²⁾, as shown in the fluoroscopic and 3-D mapping images (Fig. 3).

Mechanisms of PM Arrhythmia

Although PM arrhythmia has been considered to occur by focal mechanisms¹⁾, this type of arrhythmia is currently reported to be based on the fascicular reentrant mechanism²⁾. It is important but difficult to distinguish myocardial from fascicular origin due to their close anatomic relationship⁵⁾. Our case showed mid-diastolic (P1) and presystolic (P2) potential in the first session (Fig. 1B) at the optimal ablation sites. Whether P1 reflecting slow conduction within the circuit is blocked (Fig. 1B, left star) or not (Fig. 1B, right star) depends on the ventricular diastolic excitability that is variable in PMs suffering periodic stretching⁷⁾⁸⁾. High-frequency P2 observed immediately before the ventricular activation during sinus rhythm and PVBs (Fig. 1B, arrows) implies fascicular involvement, where endocardial layer of PMs is interconnected by sub-endocardial Purkinje fibers²⁾. However, it is not certain in our case that the mechanism of PM arrhythmia is fascicular reentry rather than focal activity²⁾. Because VT including Purkinje potential was not documented.

RFCA of PM Arrhythmia

RFCA is a first line therapy of drug-refractory PM-arising PVBs. So far success rate of RFCA is fair, and recurrence rate is relatively high for anatomic and technical reasons, i.e., complex anatomy, thickness and independent contraction of PMs, and difficulty to keep catheter tip stability during RF energy application⁶⁾. However, success rate is improving by using irrigated or non-irrigated 8-mm-tip ablation catheter¹⁾³⁾⁹⁾ and several real-time imaging modalities⁵⁾. The two

Fig. 3A

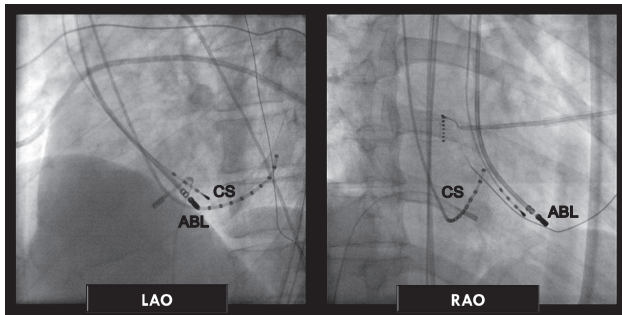


Fig. 3B

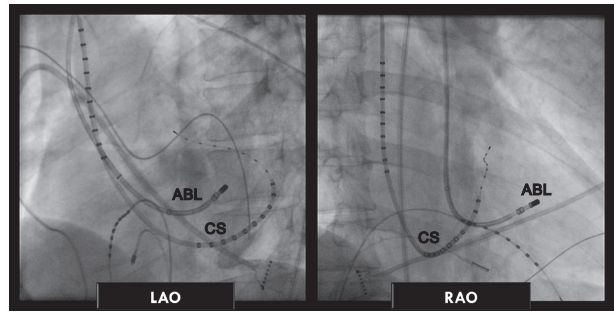


Fig. 3C

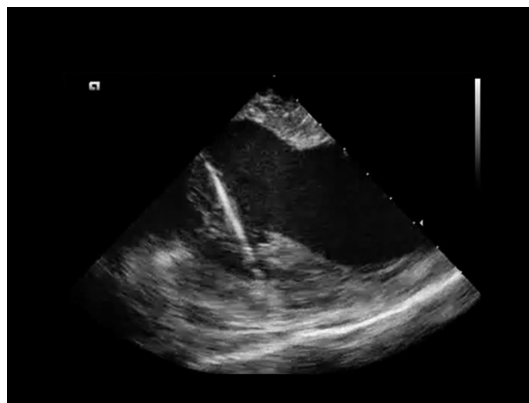


Fig. 3D

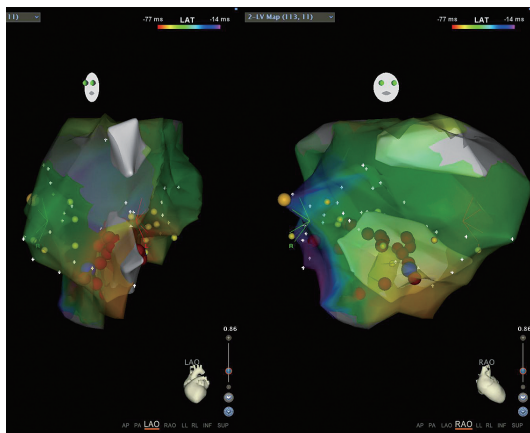


Fig. 3E

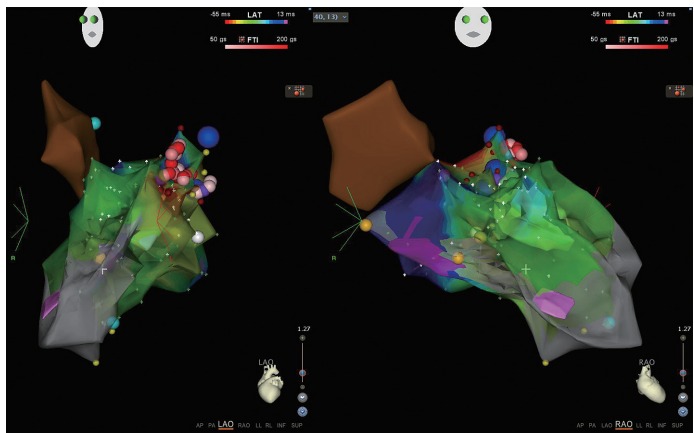


Fig. 3 Biplane fluoroscopic images of the first (**A**) and the second (**B**) session. In each panel, left picture is left anterior oblique view (LAO), and right picture is right anterior oblique view (RAO). In the retrograde trans-aortic approach, the ablation catheter tip was positioned at the optimal ablation site, i.e., the ablation catheter shape within the left ventricle (LV) is straight in **A**, whereas it is curved with a catheter tip-end directed upward in **B**. The ablation catheter and anterolateral papillary muscle were evident in the intracardiac echographic image obtained in the second session (**C**). Intracardiac echo probe was positioned within the right ventricle. Three-dimensional LV integrated mapping images in the first (**D**) and the second (**E**) session are demonstrated, and focal ablation points are indicated. In each panel, left picture is LAO, whereas right picture is RAO view. Posteromedial PM foci (**D**) are always lower relative to anterolateral PM foci (**E**). ABL, ablation catheter ; CS, coronary sinus.

sessions of RFCA in our case were successful considering symptom, ambulatory monitoring and echocardiogram. Many PM arrhythmias show constant pre-ablative QRS morphology. However, distinguishable changes in the QRS morphology are frequently observed during or long after the RFCA²⁾⁹⁾. In addition, circumferential ablation strategy in the base of PMs is proposed to abolish PM arrhythmia⁴⁾. This case also showed residual PVBs with gradually changing QRS morphologies during RFCA and required circumferential RFCA around the base of PMs to eliminate PVBs in both sessions, implying multiple exits of Purkinje network⁴⁾. Purkinje network is abundant in the attachment area of PMs²⁾. Therefore, considering future development of new circuit or exit in the Purkinje network modulated by repetitive RFCA, careful post-ablative follow-up is required.

Conclusion

We presented a case of isolated frequent PVBs originating from posteromedial followed by anterolateral PM. PVBs were treated successfully by sequential RFCA. Although focal activity cannot be excluded, fascicular reentrant mechanisms involving Purkinje network at the base of PMs are suspected and circumferential ablation to the bases of both PMs was added in two sessions of RFCA. This type of arrhythmia is rare in sustained form and hence not life-threatening but often impairs LV function and quality of life. Therefore, RFCA is recommended, and careful postablative follow-up is required.

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Conflict of interest

Funding Sources : We have no funding sources.

COI : We declare no conflict of interest.

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(和文抄録)

後内側乳頭筋ついで前外側乳頭筋に起源をもつ特発性心室期外収縮に 対して高周波心筋焼灼術を施行した 1 例

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症例は 61 歳の男性で全身倦怠感を主訴に来院した。ホルター心電図では右脚ブロック型で上方軸の心室期外収縮が頻発しており、心臓超音波検査では左室壁運動がびまん性に低下し左室駆出率が 45%であった。薬物治療に抵抗性の心室期外収縮に対して心筋焼灼術を行った。臨床電気生理学検査では後内側乳頭筋でのペースマップが最良で、同部にプルキンエ電位も認めたため通電を行ったところ心室期外収縮は消失し左室駆出率も 63%に回復した。しかし 1 年半後に右脚ブロック型で下方軸の心室期外収縮が頻発し始めたため前回と同様に心筋焼灼術を行った。臨床電気生理学検査では心室期外収縮は前外側乳頭筋に起源があり同部への通電で心室期外収縮は消失した。乳頭筋起源の不整脈は巣状興奮による場合が多いとされるが今回プルキンエ線維網を介する興奮旋回による機序が強く疑われた。また乳頭筋起源の心室期外収縮は致命的ではないものの心機能を低下させたり、心筋焼灼術に難渋するため注意深い経過観察が必要である。

キーワード：カテーテル心筋焼灼術，乳頭筋，プルキンエ電位，心室不整脈