CASE REPORT

Catheter ablation targeting Purkinje potentials controlled ventricular fibrillation in a patient with a malignant lymphoma occurring in the ventricular septum

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SUMMARY

Malignant lymphoma is known to cause various types of arrhythmia, including ventricular fibrillation. However, radiofrequency catheter ablation of ventricular fibrillation associated with malignant lymphoma has never been reported. We describe the case of a 53-year-old man with refractory ventricular fibrillation that was associated with malignant lymphoma. Electrophysiological testing revealed that a Purkinje potential appeared before ventricular contraction at the tumour site. We successfully treated the ventricular fibrillation with radiofrequency catheter ablation, using the Purkinje potential as an indicator. Physicians should consider this treatment if ventricular fibrillation cannot be controlled using other strategies.

BACKGROUND

Malignant lymphoma is known to cause various types of arrhythmia, including ventricular fibrillation (VF). To treat this condition, drug therapy and implantable cardioverter defibrillators are considered the established treatments. However, radiofrequency catheter ablation (RFCA) has also recently been reported as a treatment for idiopathic or ischaemic VF. In this case, we performed electrophysiological testing for VF that was associated with cardiac invasion by a malignant lymphoma. A Purkinje potential was observed to precede the trigger for premature ventricular contraction (PVC), and its ablation successfully controlled the VF.

CASE PRESENTATION

A 53-year-old man with no remarkable medical history presented at our hospital with dyspnoea and oedema. The patient was diagnosed with congestive heart failure and was subsequently admitted. ECG recorded his sinus rhythm, with a negative T wave in the inferior leads and ST segment depression in the lateral leads, although his medical check-up findings from several years ago were normal. Transthoracic echocardiography revealed pronounced thickening of the septum and inferior wall, with reduced movement in all walls, except in the lateral walls (figure 1A). The patient's brain natriuretic peptide level was 187 pg/mL, while his ferritin and soluble interleukin-2 receptor antibody levels were within the normal ranges. Myocardial biopsy at the right ventricular septum only

recorded interstitial oedema. Cardiac CT revealed thickening of the septum and inferior wall, and MRI recorded poor contrast during the early phase and delayed contrast enhancement in the same area (figure 1B–D). Whole-body CT also recorded multiple enlarged lymph nodes in the abdominal cavity. After a gastric mucosal biopsy, we reached a definitive diagnosis of a large B-cell malignant lymphoma.

TREATMENT

Although the patient's heart failure was compensated without catecholamine, VF occurred immediately after the diagnosis and could not be controlled using amiodarone, deep sedation with tracheal intubation and artificial ventilation and overdrive pacing; thus, we performed repeated electrical defibrillation. The VF and sustained ventricular tachycardia (VT) occurred frequently and were consistently triggered by the PVC during the monitoring. As a right bundle block-type trigger

Figure 1 Early imaging results with the tumour lesions indicated. (A) Transthoracic echocardiography reveals septum hypertrophy. (B) Early-phase contrast MRI, in which the most severely hypertrophic portion of the septum exhibits poor contrast enhancement. (C) Contrast-enhanced CT reveals heterogeneous hypertrophy with poor contrast enhancement. (D) The same portion exhibits delayed contrast enhancement. The arrows indicate the various tumorous lesions.

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Figure 2 Twelve-lead ECG exhibiting ventricular fibrillation that was triggered by a premature ventricular contraction within the dotted box.

PVC was recorded, and electrophysiological testing and RFCA were conducted. An 8 F sheath was introduced into the right femoral artery, and a non-irrigated ablation catheter with a 4 mm tip (Mariner MC, Medtronic, Minnesota, USA) and an Atakr generator (Medtronic) were introduced in a retrograde fashion into the left ventricle. Three-dimensional mapping systems, such as the CARTO system, were not available. During

the testing, despite the VF occurring frequently after multiple different PVCs (figures 2 and 3), we could not confirm a reproducible pattern with pace mapping. However, repetitive Purkinje potentials preceding the QRS were recorded in the left ventricular septum (where the tumour was situated) during the VF and sinus rhythm (figure 3, upper panel), and these potentials were not recorded at any other sites. Thus, we targeted the Purkinje potentials using RFCA, as they might contribute to maintaining the VF, rather than targeting the PVCs.

RFCA was performed at the left ventricular septum where the Purkinje potentials were recorded (figure 3, lower column), under temperature control ($<60^{\circ}\text{C}$, 25–30W); 37 defibrillations were needed for VT and VF during the session. The first and second RFCAs were immediately stopped because VT and VF occurred, although the third and fourth RFCAs were performed for 30 s without VT and VF. Based on the potential, we added fifth and sixth RFCAs, which completely suppressed the VT and VF. We also performed seventh and eighth RFCAs as bonus ablations. No accelerations occurred during the RFCAs, although pace mapping was not available before the ablation, because pacing easily induced VT and VF. After the ablations, pacing at the ablation sites did not provide an excellent pace map.

After this procedure, the VF was controlled and did not recur during an entire day of monitoring, although the PVC persisted. Rituximab, cyclophosphamide, doxorubicin, vincristine and prednisolone (R-CHOP) therapy was initiated on day 2 after the procedure. The VF transiently recurred immediately after the chemotherapy was started, although it subsequently disappeared. Despite the reduced cardiac function persisting for 5 days after the chemotherapy, the cardiac hypertrophy of the septum markedly improved (figure 4).

Figure 3 Intracardiac tracings (upper panels) recorded at the ablation sites during sinus rhythm (panel A) and the initiation of ventricular fibrillation (panel B), with fluoroscopic images exhibiting the ablation sites (lower panels). (A) At the ablation sites, the Purkinje potentials (indicated by black arrows) were recorded before the QRS complexes during sinus rhythm, and the small spikes that were recorded at 1 s intervals were artefacts (indicated by white arrows). (B) The Purkinje potentials preceded the ventricular contraction at the ablation sites. Eight ablations (six effective ablations) were performed; the ablation sites are indicated by asterisks (selected according to the electrical potentials, rather than fluoroscopy).

Figure 4 Transthoracic echocardiography before and after chemotherapy. (A) The long-axis view and (B) short-axis view before chemotherapy. (C) The long-axis view and (D) short-axis view after chemotherapy. White arrows indicate the left ventricular septum where the tumour was situated.

OUTCOME AND FOLLOW-UP

VF did not subsequently recur, despite the patient not taking antiarrhythmic medication. However, he died 9 months after the R-CHOP therapy due to progression of the malignant lymphoma.

DISCUSSION

The novelty of the present case lies in the successful use of RFCA to treat VF that was associated with malignant lymphoma, using the Purkinje potential that preceded the VF as an indicator. Haïssaguerre *et al*¹ first discovered the role of the peripheral Purkinje fibres in idiopathic VF, and subsequently summarised various methods for their mapping and ablation.² Cardiac infiltration by malignant lymphoma is known to trigger a wide variety of arrhythmias, such as polymorphic VT.³ In this context, some arrhythmias can be controlled using chemotherapy.⁴ However, the VF in the present case could not be controlled using the initial treatments, and as the patient could not wait for chemotherapy, emergency RFCA was performed. Interestingly, a Purkinje potential was observed close to the site of the tumour, as recorded via MRI, and the VF was controlled by damaging the Purkinje network, rather than ablation of the trigger PVC. A previous study has described how re-entry of the Purkinje network near the right ventricular strip papillary muscles is important for the maintenance of VF in pigs,⁵ and this report is consistent with our findings. Furthermore, ventricular arrhythmia that is associated with a cardiac tumour is generally considered the result of an advanced atrioventricular block with His-bundle compression. However, we could not identify bradycardia during the hospitalisation in our case. Furthermore, several other cases have reported confirmed VF without bradycardia and these cases involved haemangioma, fibrosarcoma and fibrolipoma.^{6–8} The common factor in these cases might be the mechanical stretching, which we speculate may increase the electrical instability and induce VF.

The antitumour effect of R-CHOP chemotherapy may also have affected our patient's arrhythmia. However, there was no

recurrence of the VF during the 24 h prior to initiating chemotherapy, despite its frequent appearance prior to the RFCA, which suggests that the RFCA was successful in controlling the VF. In a similar reported case, a patient with malignant lymphoma experienced transient ventricular arrhythmia after chemotherapy.⁹ Although it is unclear whether tumour lysis or the relief of mechanical stress was involved, caution is necessary in cases where arrhythmia is triggered immediately after chemotherapy.

Learning points

- ▶ Malignant lymphoma of the heart can cause ventricular fibrillation.
- ▶ The Purkinje potential has a role in the maintenance of ventricular fibrillation.
- ▶ Radiofrequency catheter ablation targeting the Purkinje potential is a treatment option for ventricular fibrillation.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Haïssaguerre M, Shah DC, Jais P, *et al*. Role of Purkinje conducting system in triggering of idiopathic ventricular fibrillation. *Lancet* 2002;359:677–8.
- 2 Haïssaguerre M, Shoda M, Jais P, *et al*. Mapping and ablation of idiopathic ventricular fibrillation. *Circulation* 2002;106:962–7.
- 3 Kohl O, Hölschermann H, Pralle H, *et al*. Therapy-resistant ventricular tachycardia in a woman with primary cardiac non-Hodgkin lymphoma. *Med Klin (Munich)* 2000;95:517–22. (Article in German)
- 4 Manojkumar R, Sharma A, Grover A. Secondary lymphoma of the heart presenting as recurrent syncope. *Indian Heart J* 2001;53:221–3.
- 5 Kim YH, Xie F, Yashima M, *et al*. Role of papillary muscle in the generation and maintenance of re-entry during ventricular tachycardia and fibrillation in isolated swine right ventricle. *Circulation* 1999;100:1450–9.

- 6 Kassi M, Polsani V, Shen S, *et al.* A rare case of cardiac hemangioma causing ventricular fibrillation. *J Am Coll Cardiol* 2013;62:562.
- 7 Yudi M, Batra R. A case of aborted sudden cardiac death and consequent extreme electrical storm secondary to a metastatic cardiac tumor. *Pacing Clin Electrophysiol* 2013;36:e125–8.
- 8 Sajeev CG, Kalathingathodika S, Nair A, *et al.* Malignant arrhythmia with benign tumour: fibrolipoma of the left ventricle. *J Cardiovasc Ultrasound* 2014;22:151–4.
- 9 Tanoue K, Sanada J, Kayano T, *et al.* Malignant lymphoma with various cardiac manifestations: a case report. *J Cardiol* 2002;40:117–23. (Article in Japanese)

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