Hybrid surgical-catheter epicardial ablation of recurrent ventricular tachycardia in an arrhythmogenic cardiomyopathy patient with pericardial adhesions following COVID-19 infection

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Introduction

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an inherited cardiomyopathy leading to myocardial fibrolipomatous infiltration associated with recurrent ventricular arrhythmia (VA). In addition to oral antiarrhythmics, endocardial catheter ablation reduces the burden of VA and implantable cardioverter-defibrillator (ICD) shocks.[1] Adjunctive epicardial ablation further improves the efficacy.[2,3] However, percutaneous access to the pericardial space can be challenging, particularly in patients with pre-existing pericardial disease, necessitating a different approach, which may include surgical access. Moreover, unexpectedly encountered pericardial adhesions without prior cardiac surgery or clinically evident pericarditis can be associated with lower short-term success and pose a greater difficulty to the unprepared operator.[4] We present a minimally invasive hybrid surgical-catheter technique for epicardial ablation in an ARVC patient with pericardial adhesions found unexpectedly following recent COVID-19 infection.

Case report

A 28-year-old male patient was first diagnosed with ARVC, with *PKP2* class IV frameshift variant, 7 years ago following an out-of-hospital ventricular fibrillation cardiac arrest. A single-chamber ICD was implanted. One year after diagnosis, the patient underwent endocardial ablation for symptomatic premature ventricular complex (PVC). Four PVC morphologies were inducible with isoproterenol, 3 of which were ablated in the right ventricle (RV): anteroseptal outflow tract and inferior and lateral tricuspid annulus. The left ventricle (LV) was scar-free on cardiac magnetic resonance imaging (MRI); therefore it was not mapped. No epicardial ablation was performed.

Metoprolol monotherapy maintained arrhythmia quiescence for the following 4 years before he developed episodes

KEY TEACHING POINTS

- Impacts of COVID-19 infection in arrhythmogenic cardiomyopathy are rarely reported. We present a case of unexpected encounter of pericardial adhesions in a patient with arrhythmogenic cardiomyopathy who developed recurrent ventricular tachycardia 1 month after COVID-19 infection.
- Pericardial abnormality and pericardial adhesions may occur in recently recovered COVID-19 infection, independent of the severity of the COVID-19 symptoms. This should be considered when planning for epicardial access and ablation.
- Hybrid surgical access for epicardial catheter ablation can be a safe option in select patients with difficult percutaneous access. The case demonstrates a minimally invasive technique to overcome pericardial adhesions that prevented standard subxiphoid percutaneous access for epicardial ablation. Close multidisciplinary planning, set-up of the electrophysiology suite, and modification of precordial electrocardiogram lead positions were important steps to ensure a successful patient outcome.

KEYWORDS Ventricular tachycardia; Arrhythmogenic cardiomyopathy; ARVC; COVID-19; Hybrid surgical ablation; Epicardial ablation; VT ablation; Pericardial adhesions

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of nonsustained and sustained ventricular tachycardia (VT) that were responsive to antitachycardia pacing (ATP). Sotalol was commenced, with modest improvement in rhythm control for another 1 year before the burden of VAs and frequency of ATP therapy gradually increased. He subsequently contracted COVID-19, confirmed with rapid antigen test, with symptoms of fatigue and high fevers, during which time he had 1 ICD shock. He had no clinical symptoms to suggest pericarditis, and medical attention was not sought.

One month after recovering from COVID-19 he presented multiple times to hospital with recurrent VT despite escalation of antiarrhythmics to oral amiodarone and propranolol. In total, he had 17 episodes of VT, 16 ATP attempts, 13 ICD shocks, and 3 external DC cardioversions in 4 months. The transthoracic echocardiogram demonstrated normal LV size and systolic function and moderately dilated and impaired RV. The patient was referred to our center for percutaneous endocardial and epicardial ablation. A repeat rapid antigen test was negative.

Endocardial ablation was performed via femoral venous access. Geometry and bipolar voltage map of the RV (305 mL) were created with CARTO® 3 Version 7 (Biosense Webster, Irvine, CA) using high-density mapping catheters PENTARAY® NAV (Biosense Webster) and DECANAV® (Biosense Webster), which demonstrated significant posterior RV scar extending from the tricuspid annulus to the apical regions. On unipolar voltage map, the scar was larger and extended to the anterior RV and RV outflow tract. Programmed stimulation induced 9 different VT morphologies, all of which had left bundle branch block morphology, and all but 1 were negative in leads II, III, and aVF. One morphology was particularly broad, suggesting an epicardial focus. Activation mapping was performed to 3 morphologies with stable hemodynamic profile demonstrating focal endocardial propagation. Pace maps (PASO® module) were created for each VT. Early activation areas and potential endocardial exit sites with good pace-match in the basolateral, midlateral, posterolateral, and anteroseptal aspect of the RV were ablated using an irrigated catheter (THERMOCOOL SMARTTOUCH® SF D-F Catheter, Biosense Webster).

Percutaneous epicardial access via standard subxiphoid approach was then attempted several times but without success. The Bentson guidewire met with resistance in the pericardial space. Contrast injection demonstrated loculated contrast pooling suggesting pericardial adhesions (Figure 1). Epicardial access was therefore abandoned and the patient was discharged on oral amiodarone.

In the ensuing 3 months, the patient had further episodes of recurrent VT and 8 ICD shocks. After discussion and planning with our cardiothoracic surgical service, he was brought back for a minimally invasive hybrid epicardial ablation in the electrophysiology suite.

The patient was positioned supine and anesthetized with a double-lumen endotracheal tube. Precordial electrocardiogram (ECG) lead placements were modified to allow surgical access (Figure 2A). The electroanatomic navigation magnet and reference patches for CARTO 3 were placed in standard

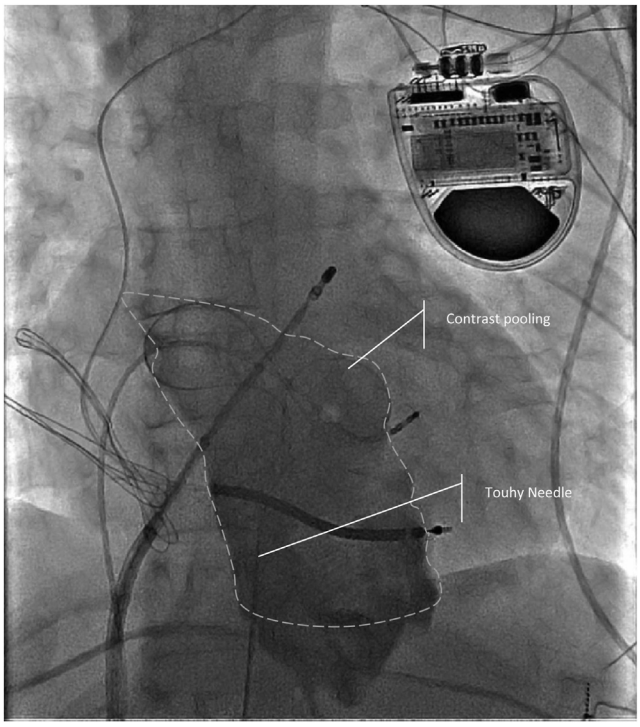

Figure 1 Anteroposterior fluoroscopy view of contrast pooling within pericardium demonstrating pericardial adhesion at initial subxiphoid percutaneous attempt.

positions. The left hemithorax was elevated using intravenous pressure bag behind the scapula. After the left lung was isolated, three 5 mm ports were placed in the third, fourth, and seventh intercostal spaces at the left anterior axillary line (Figure 2A). A thoracoscope was placed through the fourth intercostal port. Carbon dioxide insufflation was applied during the surgical components of the case.

A pericardiotomy in the mid RV, anterior to the left phrenic nerve, was created using electrocautery and enlarged superiorly with a LigaSure™ device (Medtronic, Minneapolis, MN). The pericardium appeared thickened, with evidence of prior inflammation (Figure 2B). A scalpel stab incision was made in the subcostal space at the left midclavicular line to accommodate a Touhy needle. A Bentson wire was guided through the pericardial window using video-assisted thoracoscopic surgical instruments. A small curl 8.5F Agilis™ NxT steerable introducer (Abbott, Chicago, IL) was advanced using a Seldinger technique. Lactated Ringer's solution was used for irrigation via the Agilis sheath to minimize distortions in tissue impedance. A 15F Blake drain was placed in the pericardial and left pleural space to monitor for bleeding.

Epicardial mapping was performed with a steerable DECANAV (Biosense Webster) mapping catheter. Adhesions were separated with careful catheter movements without difficulty. Three clinical VTs were easily induced during catheter manipulation and programmed stimulation. One common PVC morphology was also targeted. All the morphologies were left bundle branch block with the modified chest lead positions and negative in the inferior limb leads. Activation mapping was not possible owing to hemodynamic

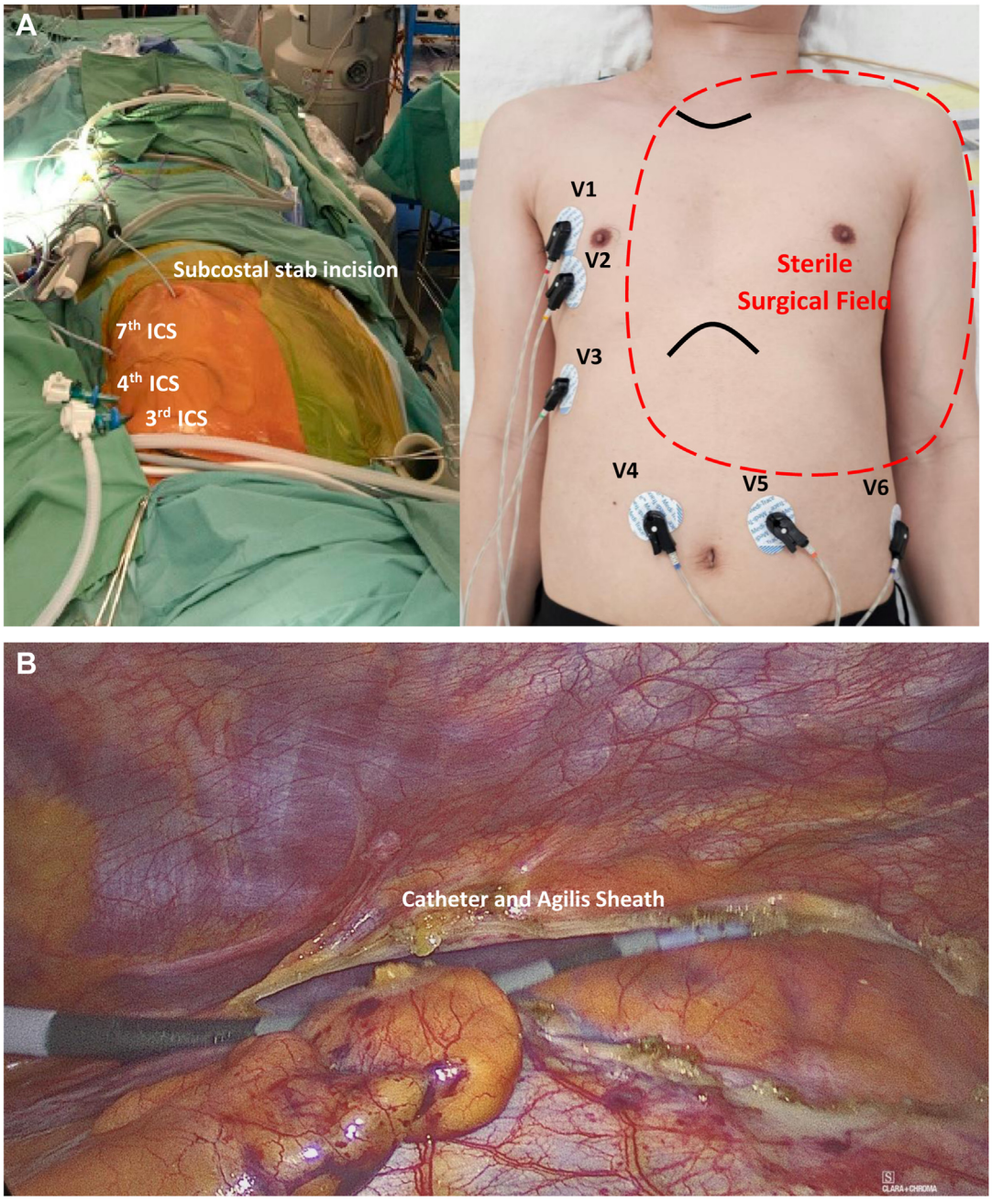

Figure 2 **A:** Thoracoscopic ports in intercostal space (ICS). Ablation catheter through subcostal incision. Modified precordial electrocardiography lead placement. **B:** Thoracoscopic image demonstrating thickened pericardium and steerable Agilis sheath within pericardial window.

instability in VT. Geometry, epicardial bipolar voltage map, and pace map of clinical VTs were created. The epicardial bipolar voltage map during sinus rhythm demonstrated a large epicardial RV scar extending from the RV base to the anterolateral and posterolateral aspect of the RV (Figure 3). During mapping, the Agilis sheath and mapping catheter came out of the pericardial space, which required repositioning through the fourth intercostal port.

Coronary angiography was performed to ensure ablations were away from coronary arteries. Epicardial scar border zones were homogenized with linear radiofrequency ablation lesion sets using a THERMOCOOL SMARTTOUCH SF D-F catheter. Fractionated, delayed signals and regions of good pace-match in the mid anterolateral, posterolateral, and apical RV, adjacent to scars, were targeted. At the end, the easily inducible clinical VT was noninducible with programmed stimulation to 3 extrastimuli from the RV apex with a 400 ms drive train. Nonclinical VT was still inducible (S1 400 ms, S2 280 ms, S3 240 ms, S4 300 ms) but was not targeted. The pericardial and left pleural drains were aspirated dry and left in situ. Triamcinolone 300 mg was injected into the pericardial space.

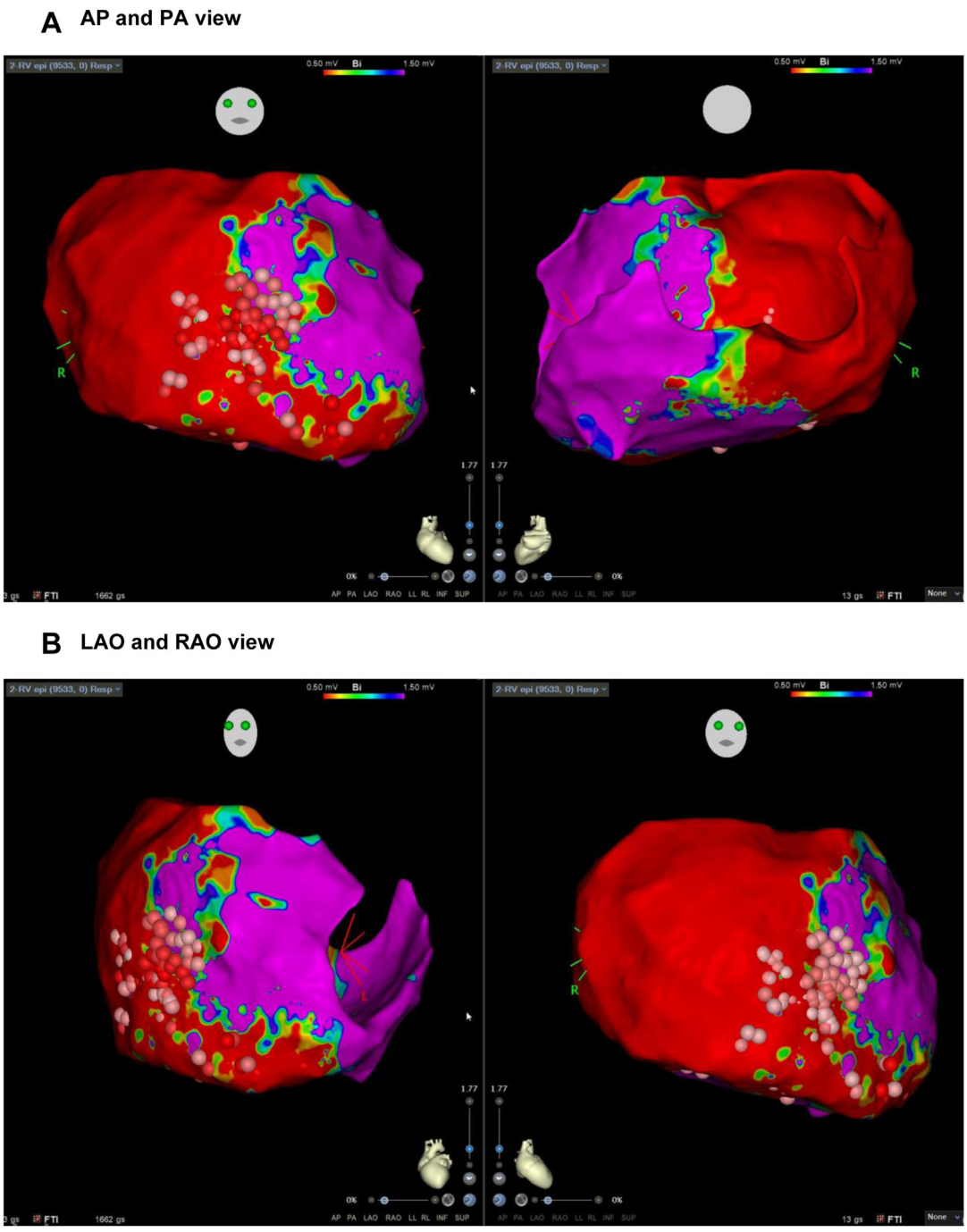

Figure 3 Epicardial bipolar voltage map (0.50 mV – 1.50 mV) on CARTO 3 with radiofrequency ablation dots. Significant epicardial scarring. Ablation performed to regions of best ventricular tachycardia pace-match sites, potential exits, fractionated delayed potentials, and heterogeneous border zones. **A:** Anteroposterior and posteroanterior view. **B:** Left anterior oblique and right anterior oblique view.

The patient was discharged the following day, after the drains were removed, with a 2-week course of oral colchicine. Device therapy has not been required in the 6-month period post ablation during follow-up.

Discussion

The fibrolipomatous infiltration of ARVC typically progresses from the subepicardial layer to the endocardium over time, and rarely from the RV to LV, resulting in extensive epicardial scarring and regional transmural substrates for VAs. Multiple observational studies have demonstrated improved ventricular arrhythmia-free periods with either up-front combined endo-epicardial VT ablation or sequential endocardial then epicardial ablation, particularly in patients with medication-refractory VT.[2,5–7]

Hybrid epicardial VT ablations are rarely reported. This is the first case performed in New Zealand. Case series have

reported on similar surgical techniques for other arrhythmogenic disease entities.[8–10] Li and colleagues[11] demonstrates an acceptable safety profile of hybrid surgical approach compared to percutaneous epicardial ablation in post–cardiac surgery VT patients, with a substantial proportion requiring pericardial adhesiolysis.

Standard epicardial access and ablation are usually performed percutaneously using a single subxiphoid puncture under fluoroscopy guidance. However, we were surprised to find pericardial adhesions that prevented advancement of the wire in the pericardial space without unduly increasing complication risks. The patient had no prior history of cardiac surgery. It is plausible that he had subclinical pericarditis from COVID-19, which is not uncommon. In a prospective study where 78% of recently recovered COVID-19 patients, mostly nonhospitalized with mild symptoms, had cardiac involvement assessed by cardiac MRI, a third of those had evidence of pericardial involvement and 60% had ongoing myocardial inflammation.[12] We did not perform a cardiac MRI prior to percutaneous VT ablation because pericardial disease was not clinically suspected at the time, especially with the urgency of VT storm. In hindsight, evidence of pericardial abnormality may have alerted us to the possibility of pericardial adhesions but might not have altered the sequence of procedural management. The hybrid approach with left thoracoscopy allowed direct visualization, demonstrating diseased and thickened pericardium, and video-assisted thoracoscopic surgical formation of a pericardial window for catheter access.

From the midclavicular line subcostal access port, manipulation of steerable sheath and mapping with the DECANAV catheter was a familiar position for cardiac electrophysiologists, comparable to the subxiphoid approach. Catheter manipulation was easy and areas closer to the pericardial window were accessible by looping the catheter. Firm catheter movements were sufficient to separate adhesions. A particular advantage of the hybrid technique was highlighted when the sheath and catheter were dislodged from the pericardial space and had to be repositioned to a craniolateral port. The new position provided the catheter a different angle of access with better stability and maneuverability within the pericardium. It also allows the potential to create an additional pericardial window in the event of multiple regions of adhesions. This minimally invasive surgical access did not impact on the length of stay of the patient.

ARVC is an uncommon cardiomyopathy. To the best of our knowledge, there is only 1 other case report that demonstrates the effect of COVID-19 infection on ARVC. Mukhopadhyay and colleagues[13] describe a patient who presented with VT storm after contracting COVID-19, which unmasked the eventual diagnosis of underlying ARVC.

In comparison, the temporal progression of our patient's arrhythmia highlights the potential impact of COVID-19 in those with ARVC. He had 4 years of relative arrhythmia quiescence after diagnosis on a combination of metoprolol monotherapy and endocardial PVC ablation. Consistent with the natural history of ARVC, he began to develop episodes of VT, albeit manageable with sotalol and ATP therapy. However, 1 month after recovering from symptomatically mild COVID-19 infection, the burden of VAs escalated to drug-refractory VT. There is increasing evidence that cardiac arrhythmias have emerged as well-documented post–COVID-19 infection sequelae, with an overall prevalence of 10%–20%, particularly in those with pre-existing cardiac structural disease and cardiovascular comorbidities.[14] Frequent PVCs have been reported as the most common ventricular arrhythmia, at 18%, and nonsustained VT at 5%.[14] The proposed mechanisms include varying combinations of ischemia; inflammatory cytokine surge (eg, interleukin-6 and tumor necrosis factor α), which can prolong ventricular action potential, leading to arrhythmogenicity; direct cardiomyocyte injury from myocarditis mediated by surface protein angiotensin-converting enzyme 2 receptor and viral spike protein interaction; and new scar formation.[15] It is thus conceivable that COVID-19 infection may dramatically lead to worsening arrhythmia when there is pro-arrhythmic substrate, as is the case for ARVC, independent of the severity of COVID-19 symptoms.

Conclusion

The case illustrates the impact of COVID-19 infection in an ARVC patient, leading to malignant ventricular arrhythmia and potentially the cause of pericardial adhesions, which complicated epicardial access for VT ablation. This was overcome by a minimally invasive hybrid surgical-catheter technique, which relied on close multidisciplinary planning and cooperation between the cardiothoracic and cardiology service. Set-up of the electrophysiology suite, procedural team briefs, and modification of precordial ECG lead positions to ensure unhindered electroanatomical mapping while allowing surgical access were important steps to ensure a successful patient outcome. Hybrid surgical access of the pericardial space can be a safe option in select patients with difficult percutaneous access.

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