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CASE REPORT
Coronary heart disease

Precipitation of *de novo* atrial fibrillation during Shockwave Intravascular Lithotripsy® after pacing capture during the treatment of proximal right coronary artery disease: a case report

Elizabeth Curtis *, Ali Khan, Seif El-Jack, and Tim Glenie

Cardiology Department, North Shore Hospital, Waitemata District Healthboard, 124 Shakespeare Rd, Takapuna, Auckland 0620, New Zealand

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Background Shockwave Intravascular Lithotripsy® has been recently translated to the treatment of coronary artery disease with a long history of use for ureteric stones where it has been observed to have caused cardiac arrhythmias. The risk of arrhythmia with the use of this method in coronary artery disease is currently unknown.

Case summary A 72-year-old man undergoing planned percutaneous intervention to a heavily calcified proximal right coronary artery (RCA) lesion using S-IVL developed pacing capture from the device and subsequently new atrial fibrillation (AF) during the procedure. The technique resulted in successful treatment of the coronary lesion and he spontaneously reverted within an hour of the procedure before discharge.

Discussion We postulate the pulsed energy delivered to break apart the calcium has the capacity to cause depolarization, and therefore, affect cardiac rhythm as seen in treatment of renal stones in the past before the introduction of routine electrocardiogram (ECG) gating. In this case, the proximity of the RCA to the right atrium caused short circuiting and development of AF in a susceptible patient. Both the pacing implications and the risk of arrhythmia needs to be investigated further and the potential for ECG gating of the pulsed energy to mitigate this effect needs to be explored to enhance the safety of this technique.

Keywords Shockwave Intravascular Lithotripsy® • Atrial fibrillation • Arrhythmia • Pacing • Case report

Learning points

- The possibility of pacing capture occurs during use of Shockwave Intravascular Lithotripsy® (S-IVL) in a proximal right coronary artery lesion.
- Atrial fibrillation caused by S-IVL by mechanism of short circuiting the atrium in a susceptible patient.
- Potential requirement for electrocardiogram gating to mitigate the risk of arrhythmia and pacing capture by the device.

* Corresponding author. Tel: +64 273 757 757, Email: libby.curtis@waitematadhb.govt.nz

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Introduction

We report a case of pacing capture caused by Shockwave Intravascular Lithotripsy® (S-IVL, Shockwave Medical, CA, USA) and resultant development of new atrial fibrillation (AF) in a patient undergoing percutaneous coronary intervention (PCI) of a heavily calcified lesion in the proximal right coronary artery (RCA).

Ultrasound has developed as a tool for the treatment of ureteric stones and lithotripsy had been in use since 1992 in order to facilitate their removal.[1] Recently the principle has been translated to use in coronary artery disease as a way of lesion preparation for optimizing stent expansion in the percutaneous treatment of calcified coronary lesions.[2] The lithotripsy device generates pulsatile mechanical energy that disrupts the calcium at the target site which allows optimal dilation of calcified coronary lesions. However, this energy theoretically can trigger cardiac depolarization and arrhythmia. This was demonstrated by the use of lithotripsy in patients with renal stones causing ventricular ectopics, electrocardiogram (ECG) capture with pacing and supraventricular tachycardia.[3,4] Arrhythmias have been documented in up to 20% of patients treated by lithotripsy for renal stones, generally benign and greatly reduced by the introduction of ECG gating techniques.[4] Experience with the novel use of S-IVL in coronary artery intervention is limited at this time and the risk of arrhythmias is currently unknown.

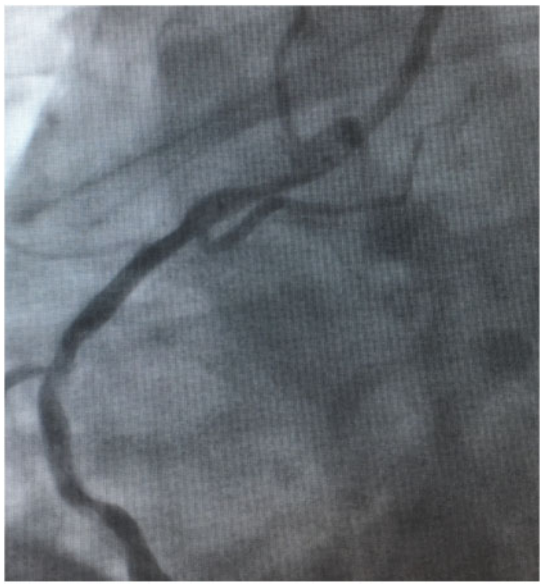

Figure 1 Proximal right coronary artery lesion before treatment with Shockwave Intravascular Lithotripsy® and percutaneous coronary intervention.

Timeline

6 months prior to presentation: exertional angina.
1 week prior to intervention: diagnostic angiogram showing severely calcified right proximal artery coronary lesion.
Percutaneous intervention: planned using Shockwave Intravascular Lithotripsy given significant calcification.
During intervention: electrocardiogram capture and pacing during delivery of Shockwave Intravascular Lithotripsy® seen on monitoring. Immediately after cessation of shockwave degeneration of rhythm into atrial fibrillation.
Post-intervention: during recovery, spontaneous reversion to sinus rhythm.

Case presentation

A 72-year-old man with known coronary disease and previous PCI to left anterior descending artery (LAD) was being investigated for exertional breathlessness and chest tightness which had progressed over 6 months' duration. He has a history of Type 2 diabetes mellitus, moderate aortic stenosis, and previous Wolf–Parkinson–White syndrome treated by radiofrequency ablation 20 years earlier. His baseline ECG showed sinus rhythm with no pre-excitation. He had neither history of palpitations nor documentation of AF prior to the procedure. His echocardiogram showed a normal left ventricular function with ejection fraction 60–65%, the left atrium was mildly dilated 43 mm diameter, 36 mL/m^2, his right atrium was of normal size. Medications prior to the angioplasty included aspirin 100 mg daily, candesartan 8 mg daily, metoprolol controlled release (CR) 95 mg mane, atorvastatin 80 mg daily, metformin 1 g twice daily, and pantoprazole 40 mg daily. His coronary angiography showed severe calcific disease of the proximal RCA as it coursed in the atrioventricular groove; the LAD stent was patent and circumflex artery had mild disease.

An elective PCI with planned S-IVL to achieve optimal result was undertaken in consideration of the severe calcific nature of the proximal RCA lesion (*Figure 1*). Pre-dilation with a 2.0 mm compliant balloon facilitated the S-IVL balloon positioning. With the first cycle of 10 pulses of shockwave lithotripsy, clear pacing capture with the pulses was observed on the electrocardiographic monitoring (*Figure 2*) with corresponding decline in intra-aortic blood pressure. His cardiac rhythm evolved into AF at the end immediately post-lithotripsy, at a rate of 90–110 b.p.m., the blood pressure remained stable, between 100 and 110 mmHg systolic and patient was asymptomatic so the case was continued (*Figure 3*). Full therapy with seven subsequent 10 pulse cycles was delivered and the stent was successfully deployed with no immediate vascular complications and a good angiographic result (Supplementary material online, *Video S1*). He reverted to sinus rhythm prior to discharge within an hour of the procedure and remains in sinus rhythm at 1 month post-discharge without symptoms suggestive of paroxysmal AF and so no anticoagulation therapy was commenced beyond dual antiplatelet therapy for the stent. His symptoms of chest tightness and breathlessness on exertion have also been relieved post-PCI.

Discussion

We have recently published our early first real-world experience of S-IVL in unselected lesions[5] and our experience in its use continues.

Figure 2 Sinus rhythm pre-Shockwave Intravascular Lithotripsy. Pacing of the electrocardiogram by Shockwave Intravascular Lithotripsy with correspondent fall in systemic blood pressure.

Figure 3 Degeneration into atrial fibrillation post-Shockwave Intravascular Lithotripsy.

This is the first case we encounter of S-IVL causing development of new AF. We postulate this may have been caused by the short circuiting of the atrium by the pulses of energy generated due to the proximity of the proximal RCA, and therefore, the S-IVL balloon to right atrium while treating this lesion, triggering a re-entrant pathway in a patient with susceptibility to developing AF. This patient had an enlarged atrium at baseline which does increase the risk of atrial arrhythmias, as well as the history of previous ablation for Wolff-Parkinson-White (WPW) contributing to the risk of our patient to developing AF. Patients who have a history of WPW are known to have a higher risk of atrial arrhythmias and in an observational series has shown higher rates of AF in patients who have had an ablation for WPW, compared to those without ablation.[6] From the use of lithotripsy in the treatment of ureteric stones, it has already been demonstrated that this technology has the potential to trigger cardiac depolarization, pacing, and initiate supraventricular arrhythmias.[3] The likelihood of causing cardiac arrhythmia during treatment of renal stones has been associated with right-sided stones more than left, suggesting positioning affects the propensity to arrhythmia and by extrapolation when the stimulus is in even closer proximity (as in the case of S-IVL) to the heart the probability may be higher.[7]

This phenomenon may not be observed with therapy delivered elsewhere in the coronary system or all patients and patients without other risk factors for AF, the pacing capture may not result in AF. Other potential explanation is related to relative ischaemia during balloon inflation, but given the clear evidence of cardiac depolarization in this case, without significant symptoms of ischaemia, this is less likely than the explanation of mechanical depolarization caused by the shockwave resulting in short circuiting and the development of a re-entrant atrial pathway in a susceptible patient.[8]

The pacing implication of S-IVL and its arrhythmia potential requires further study and future consideration for ECG gating of the S-IVL device to mitigate ECG capture and potential for subsequent arrhythmia.

Conclusion

We describe the development of *de novo* AF in a patient with some risk factors during percutaneous treatment of a calcified proximal RCA lesion with S-IVL in order to achieve optimal pre-dilation. The patient had clear pacing capture followed immediately by AF which was likely caused by the short circuiting caused by the pulses. Fortunately this patient self-reverted within an hour post-procedure and no further documentation of AF has been demonstrated. This case has implications of potential risks of arrhythmia with the technology being applied to the coronary tree and may require consideration if ECG gating is required, as used in the treatment of renal stones, to prevent arrhythmia.

Lead author biography

Elizabeth Curtis is a general medicine and cardiology trainee based in Auckland, New Zealand, having attained her medical degree (MBChB) at the University of Otago, Dunedin in 2012. She is interested in general cardiology, heart failure, and imaging and is currently working at North Shore Hospital in the Cardiology department as the cardiac catheterization registrar.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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