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The mystery of the missing P waves: a case report

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Abstract

Background This case highlights the diagnostic challenges of atrial flutter with concealed atrial activity on surface electrocardiograms, emphasizing the necessity of invasive electrophysiological study to avoid unnecessary pacemaker implantation in patients with structural heart disease—a scenario rarely documented in current literature.

Case presentation A 60-year-old Chinese woman with rheumatic mitral stenosis and prior maze procedure presented with fatigue and bradycardia (heart rate, 47 beats per minute). Surface electrocardiograms (including modified Lewis leads) revealed no discernible P waves, while echocardiography demonstrated atrial mechanical silence. Electrophysiological study identified cavotricuspid isthmus-dependent atrial flutter with extensive right atrial low-voltage zones (voltage < 0.5 mV), explaining the absent surface atrial activity. Radiofrequency ablation achieved bidirectional isthmus block, restoring sinus rhythm (heart rate, 59 beats per minute) without pacemaker requirement.

Conclusion In patients with bradycardia and electromechanical atrial dissociation, electrophysiological study proves indispensable for detecting atrial flutter obscured by severe fibrosis. This approach prevents inappropriate pacemaker implantation while restoring physiological rhythm, establishing a paradigm for managing complex arrhythmias in structural heart disease.

Keywords Atrial flutter, Electrophysiological study (EPS), Radiofrequency ablation, Catheter ablation, Rheumatic heart disease, Case report

Introduction

Atrial flutter (AFL) is a common supraventricular arrhythmia characterized by rapid atrial activation, typically with an organized pattern of conduction. While classical AFL often presents with a sawtooth pattern on electrocardiography (ECG), atypical cases can exhibit irregular conduction, making differential diagnosis challenging. In some cases, atrial waves may be difficult to detect on ECG, further complicating the diagnosis [1–3].

In clinical practice, distinguishing AFL from other atrial tachyarrhythmias, such as atrial fibrillation (AF) or focal atrial tachycardia (AT), is crucial for appropriate management [4–7]. Misdiagnosis or uncertainty in classification may lead to suboptimal treatment strategies, as different atrial tachyarrhythmias require distinct therapeutic approaches, including rate control, antiarrhythmic drugs, catheter ablation, or anticoagulation therapy. Traditional diagnostic tools, including surface ECG and Holter monitoring, may not always provide definitive findings, particularly in cases with variable conduction patterns. In such instances,

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intracardiac electrophysiology study (EPS) plays a pivotal role in confirming the diagnosis and guiding appropriate treatment [8, 9].

We present a case of complex AFL in which standard ECG findings were inconclusive, leading to diagnostic uncertainty and challenges in treatment planning. The final diagnosis was established through EPS, which not only confirmed the arrhythmia mechanism but also provided guidance for targeted intervention. This report underscores the diagnostic value of EPS in challenging cases of atrial arrhythmia and provides insights into key electrophysiological findings for differentiating atypical AFL from other tachyarrhythmias.

Case report

A 60-year-old Chinese woman with rheumatic mitral stenosis and prior mitral valve replacement/maze procedure for atrial fibrillation presented with persistent fatigue while on therapeutic warfarin (international normalized ratio (INR) 2.1). Initial assessment demonstrated symptomatic bradycardia (47 beats per minute (bpm)) with complete absence of atrial activity on 12-lead ECG despite technical optimizations: increased calibration (20 mm/mV

Fig. 1 The surface electrocardiogram showed no visible atrial waves. **A** Initial 12-lead electrocardiogram demonstrating the absence of P waves, flutter waves (F waves), and fibrillation waves (f waves). **B** Calibration increased from 10 to 20 mm/mV and paper speed from 25 to 50 mm/second; no clearly visible P waves

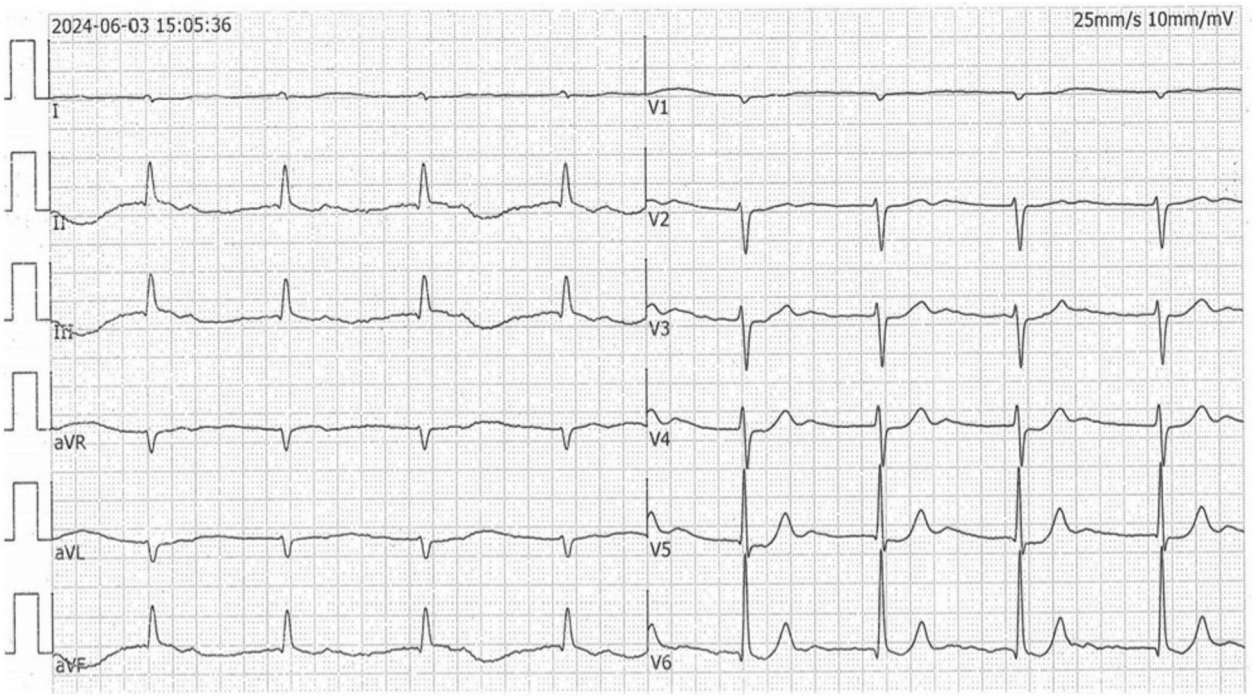

Fig. 2 The modified Lewis lead showed no visible P waves

versus standard 10 mm/mV) and accelerated paper speed (50 mm/second versus 25 mm/second; Fig. 1A, B). Modified Lewis lead configuration (right arm (RA) electrode at sternal manubrium, left arm (LA) at right fourth intercostal space, and left leg (LL) unchanged) likewise failed to detect P waves (Fig. 2). Subsequent 24-h Holter monitoring recorded ventricular pauses up to 7.7 seconds (Fig. 3), while transthoracic echocardiography revealed a monophasic mitral inflow pattern (isolated E wave without A wave; Fig. 4), suggesting atrial mechanical standstill.

Invasive electrophysiological evaluation resolved the diagnostic impasse. Intracardiac mapping demonstrated (1) cavotricuspid isthmus-dependent atrial flutter with counterclockwise activation (cycle length 220 milliseconds; Fig. 5A) and (2) extensive right atrial low-voltage zones (< 0.15 mV during sinus rhythm) indicative of severe fibrosis (Fig. 5B). Radiofrequency ablation targeting the cavotricuspid isthmus achieved bidirectional conduction block (Fig. 6), successfully terminating the arrhythmia despite a transient 6082 millisecond sinus arrest post-ablation. Post-procedural rhythm stabilized at 59 bpm with persistent absence of surface-detectable P waves on modified Lewis lead ECG (Fig. 7), correlating with ongoing atrial electromechanical dissociation. The patient reported marked symptomatic improvement and was discharged without requiring permanent pacemaker implantation.

Discussion

This case illustrates the complex interplay between advanced cardiac structural changes and the diagnostic challenges they pose, particularly in the detection and management of atrial arrhythmias. In patients with extensive atrial fibrosis, traditional diagnostic methods such as surface ECGs and even enhanced approaches such as the modified Lewis lead ECG [10]

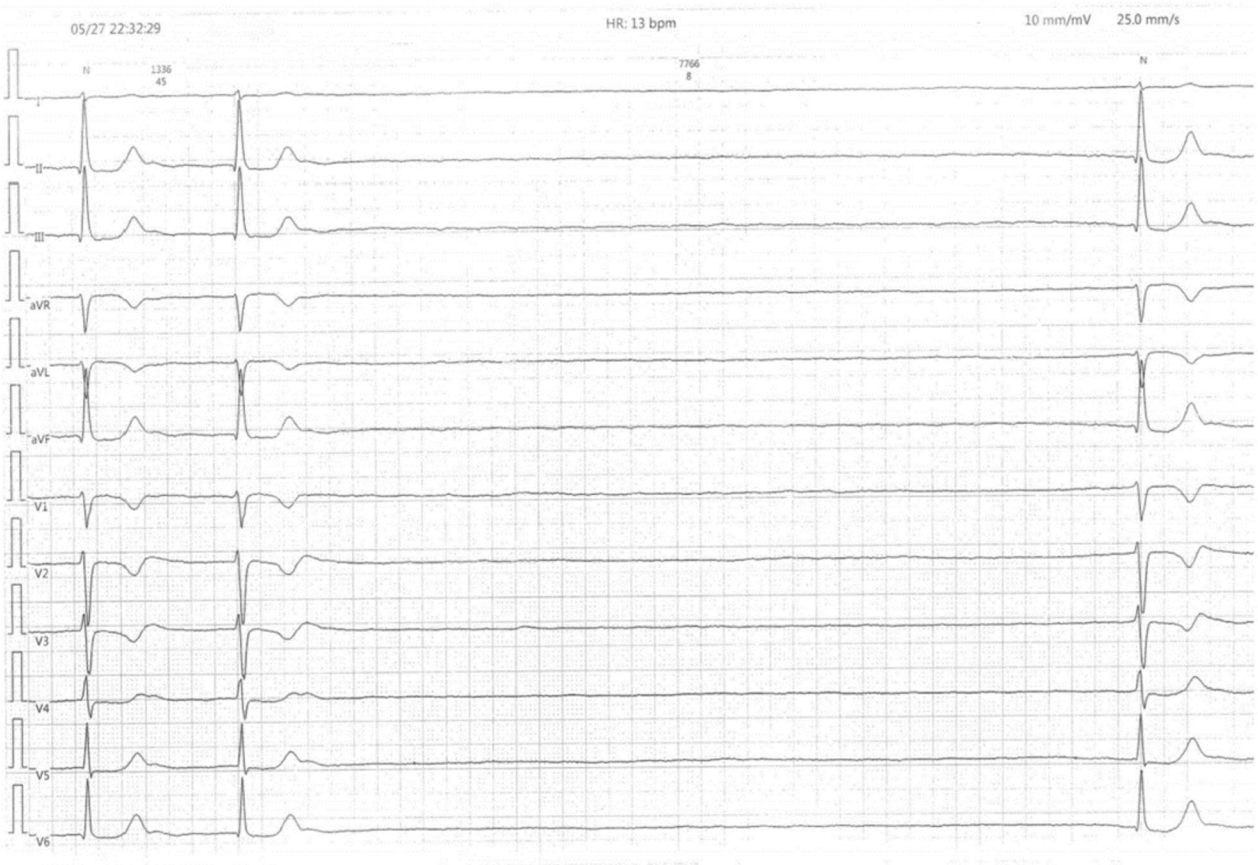

Fig. 3 Holter monitor results indicating the absence of P waves, a ventricular rate of approximately 47 beats per minute, and the longest cardiac pause of 7.7 seconds occurring at night

may fail to detect atrial activity. This failure is often due to the presence of significant scarring and low-voltage areas within the atrium [11], as confirmed by the electrophysiological study (EPS) in this patient. The EPS was pivotal, not only in identifying these structural abnormalities but also in uncovering the underlying atrial flutter that was not apparent on the initial ECG assessments [12].

On the basis of the ECG findings of absent P waves and a ventricular rate of 47 bpm, the differential diagnosis included junctional rhythm, atrial silence, atrial fibrillation with atrioventricular block (AF with AVB), and atrial flutter (AFL) or atrial tachycardia (AT) with invisible P waves. Given the regular rhythm and the successful termination of the arrhythmia with cavotricuspid isthmus ablation, the most likely diagnosis is cavotricuspid isthmus-dependent atrial flutter.

Given the patient's bradycardia and prolonged RR intervals, the initial clinical consideration might typically lean toward pacemaker implantation [13]. This conventional approach, however, could have led to inappropriate treatment and potential complications

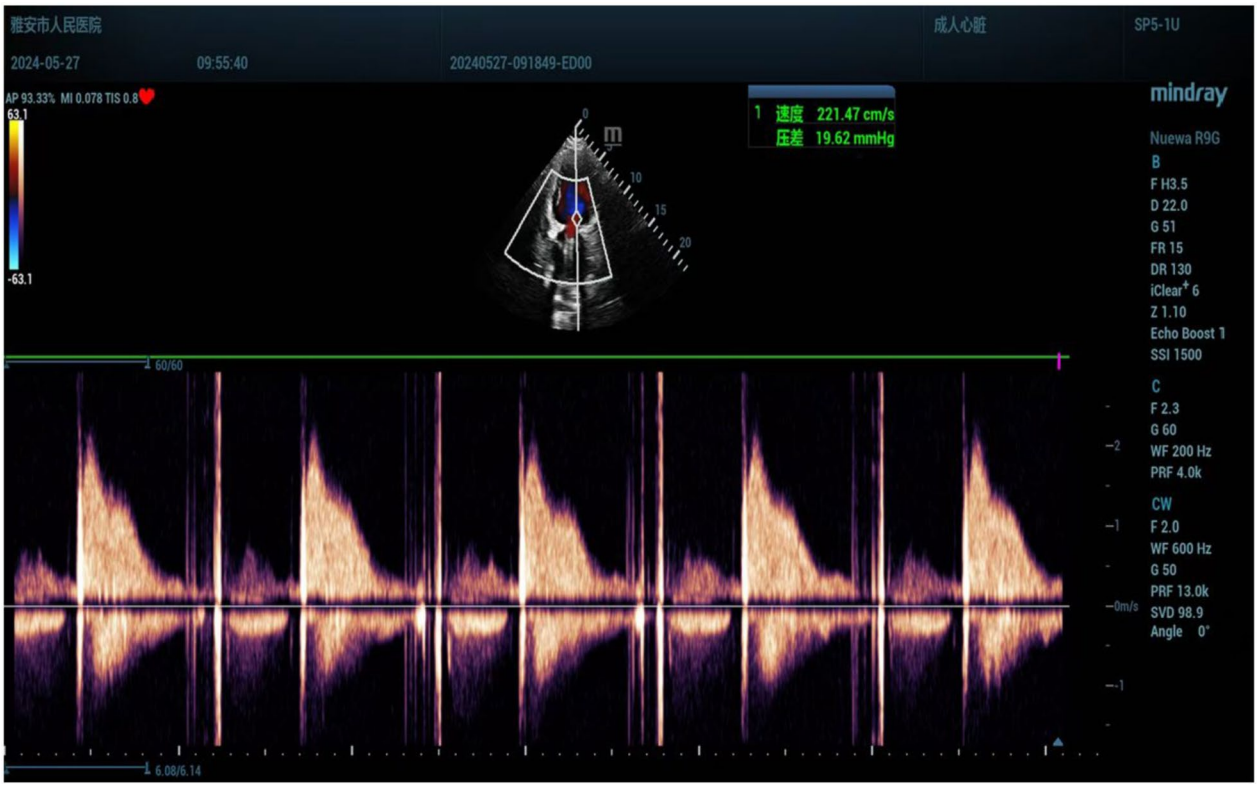

Fig. 4 The echocardiogram revealed a monophasic transmitral flow pattern

such as pacemaker syndrome [14], characterized by atrioventricular dyssynchrony. Instead, the EPS guided the therapeutic strategy toward a more precise intervention—radiofrequency ablation of the cavotricuspid isthmus. This procedure successfully restored sinus rhythm and normalized the heart rate to 59 beats per minute, illustrating the critical role of targeted intracardiac interventions in cases where extracardiac cues are misleading.

This case reinforces the essential role of thorough electrophysiological evaluations before proceeding with pacemaker implantation in patients with unexplained bradycardia and arrhythmias. By correctly identifying the atrial flutter and addressing it with appropriate ablation, the need for a pacemaker was obviated, thereby avoiding the risk of pacemaker syndrome and highlighting the importance of precision in cardiac arrhythmia management. Such insights are crucial for advancing our understanding and treatment of complex arrhythmias in the context of significant atrial fibrosis, ensuring that interventions are both appropriate and effective.

Conclusion

Electrophysiological studies provide crucial insights in cases where atrial activity is obscured on standard ECGs, guiding appropriate interventions such as radiofrequency

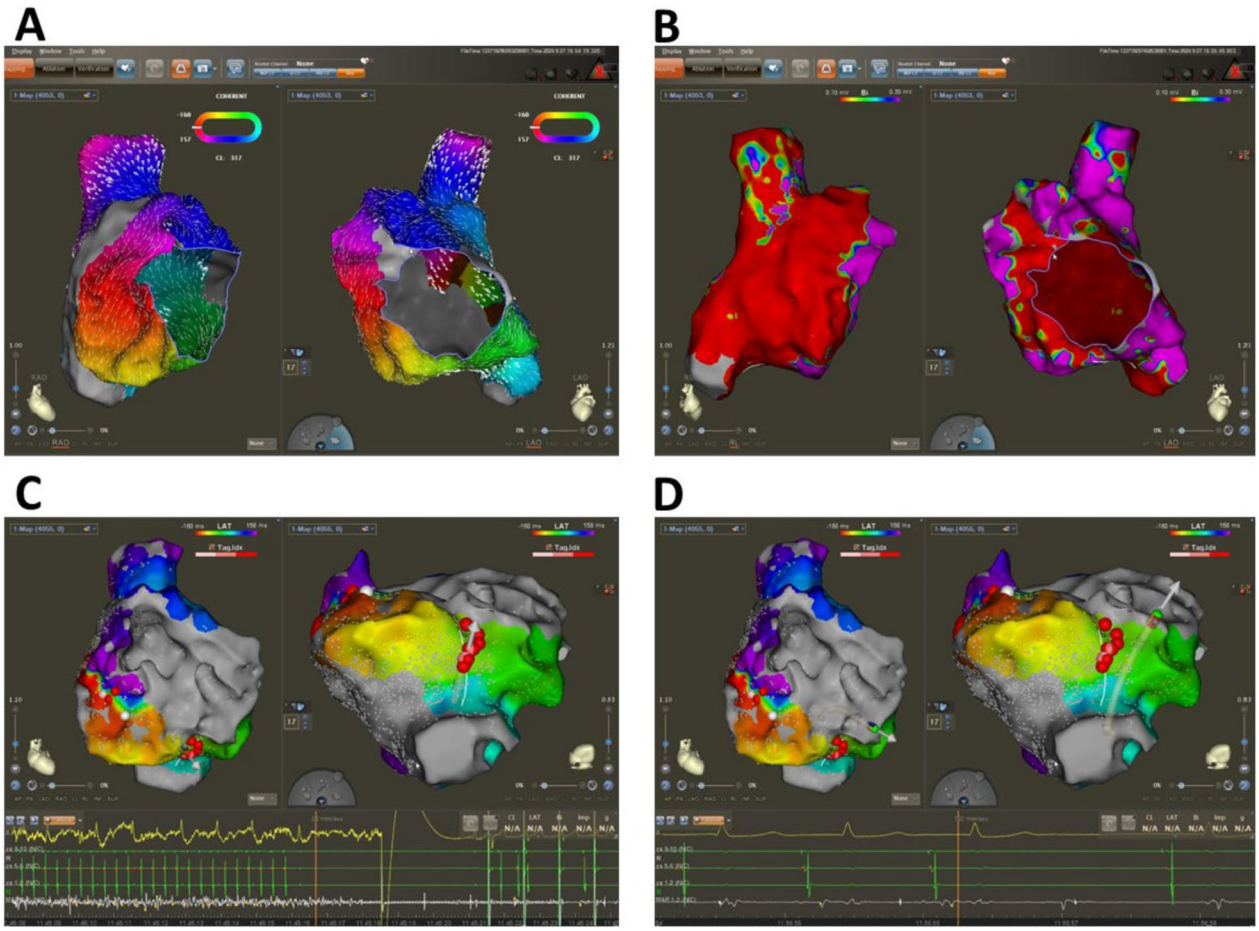

Fig. 5 Invasive electrophysiological study. **A** The order of atrial activation in the patient's right atrium, characterized by counterclockwise atrial flutter. **B** Atrial flutter accompanied by a large low-voltage area in the right atrium. **C**, **D** Successful restoration of sinus rhythm following ablation of atrial flutter. The arrows in **C** and **D** indicate the direction of contact force applied by the radiofrequency ablation catheter on the myocardium

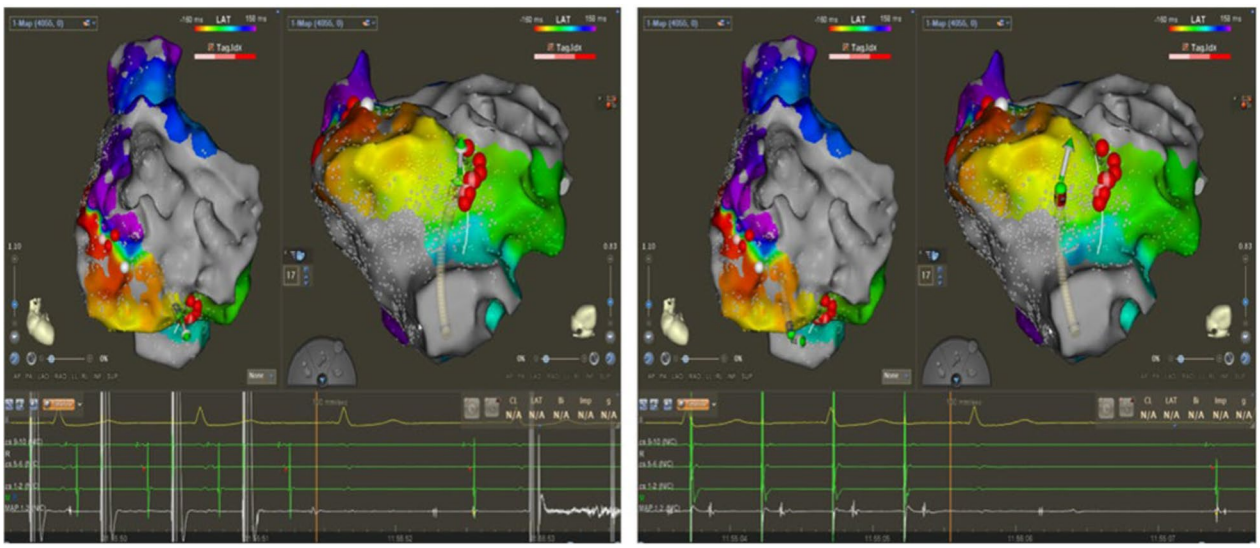

Fig. 6 Mapping indicates atrial flutter dependent on the tricuspid isthmus. The end of the ablation confirmed successful block of the tricuspid isthmus

Fig. 7 The patient's heart rate restored to 59 beats per minute with sinus rhythm after the procedure. **A** Intracardiac recording during the procedure showing restoration of sinus rhythm. **B** Postoperative modified Lewis lead electrocardiogram displaying a ventricular rate of 59 beats per minute, with no visible atrial waves

ablation over pacemaker implantation, thereby ensuring synchronized atrial and ventricular contractions and improving patient outcomes.

Abbreviations

ECG	Electrocardiogram
INR	International normalized ratio
EPS	Electrophysiological study
RR	RR interval

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Author contributions
YGC, HBZ, and CLH drafted and corrected the manuscript; and YGC and PB were involved in investigation and data collection. All authors read and approved the final manuscript for publication.

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Data availability
All relevant data supporting the conclusions of this article are included within the article.

Declarations

Ethics approval and consent to participate
This study was approved by the ethics committee, with the approval no. 2024010. Written informed consent was obtained from the patient for publication of this case report.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors declare no competing interests.

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