Abolishment of high-risk left lateral accessory pathway by myocardial infarction: a blessing in disguise? A case report

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Background
Antegrade conducting left lateral accessory pathways are a risk for supraventricular tachycardias and pre-excited atrial fibrillation. Rarely, an anomalous coronary sinus can cause difficulty in locating the pathway. The left circumflex coronary artery and obtuse marginal branches supply the posterolateral left ventricle. We describe a case report of a high-risk accessory pathway associated with an anomalous coronary sinus which, between successive electrophysiology studies, was obliterated by a felicitous acute coronary syndrome in the left circumflex territory.

Case summary
A 49-year-old male with palpitations and manifest pre-excitation was referred for electrophysiology study. Initial study revealed a high-risk left lateral accessory pathway with antegrade effective refractory period of 240 ms and rapidly conducting pre-excited atrial fibrillation. The coronary sinus could not be cannulated to localize the pathway. Coronary angiography and cardiac computed tomography showed an anomalous coronary sinus emptying into the right atrial free wall and patent coronaries. While awaiting repeat electrophysiology study, the patient suffered an acute coronary syndrome with immediate loss of previously visible pre-excitation on electrocardiogram, and underwent stenting of an occluded marginal branch of the circumflex. Repeat electrophysiology study demonstrated a now low-risk accessory pathway (effective refractory period 390 ms). Since infarction, the patient’s palpitations have fully settled with all subsequent electrocardiograms devoid of manifest pre-excitation.

Discussion
Left lateral accessory pathways, which can associate with an anomalous coronary sinus, derive from tissue similar to normal ventricular myocardium and are vulnerable to ischaemic insults in the area subtended by the circumflex artery.

Keywords
Left lateral • Accessory pathway • Pre-excitation • Anomalous • Coronary sinus • Circumflex • Myocardial infarction • Ablation • Case report

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### Learning points
- Left lateral accessory pathways are commonly implicated in supraventricular tachycardia and can be associated with pre-excited atrial fibrillation.
- Occasionally, locating a left lateral accessory pathway during electrophysiology study is difficult when there is an associated anomalous coronary sinus and may require more sophisticated electroanatomical mapping equipment.
- The left circumflex artery runs around the posterior mitral valve annulus to supply the posterolateral left ventricular surface, where accessory pathways often insert.
- Left lateral accessory pathways are vulnerable to ischaemic insults in the left circumflex territory, and may even be rendered defunct during acute coronary syndrome.

### Introduction
Accessory pathway (AP)-mediated supraventricular tachycardias are common, often warranting an electrophysiology study (EPS) and treatment by catheter ablation. A multipolar catheter placed within the coronary sinus (CS) during EPS provides a helpful means of localizing APs through assessment of timings of antegrade and retrograde atrioventricular (AV) conduction from the septal to lateral aspect of the mitral valve annulus (MVA). On rare occasions, an anomalous CS precluding catheter placement poses diagnostic challenges.

The left circumflex (LCx) coronary artery subtends the posterior and lateral MVA where many APs are located. We present an intriguing case of a high-risk left lateral AP (LLAP) characterized by an anomalous CS at initial EPS with the LLAP later rendered clinically inert through a serendipitous acute coronary syndrome (ACS) from occlusion of an obtuse marginal (OM) branch of the LCx.

### Case presentation
A 49-year-old male was referred for an EPS after presenting with a 15-year history of presyncopal palpitations. Background history was notable for smoking, hypertension (admission systolic blood pressure on antihypertensives 130/75 mmHg), hypercholesterolaemia (low-density lipoprotein level following statin therapy of 4.1 mmol/L), diabetes, and previous SCORE-calculated 10-year risk of 3%. Regular medications included simvastatin 40 mg o.d., metformin 500 mg b.i.d., ramipril 10 mg o.d., and bisoprolol 10 mg o.d. which was held for 5 days before EPS. Physical examination on admission was unremarkable. Resting 12-lead electrocardiogram (ECG) showed sinus rhythm with a short PR interval and overt pre-excitation. An Rs morphology in V1, as well as negative delta waves in II, III, and aVF (Figure 1A) describes typical ECG characteristics of a posteriorly located LLAP.

At initial EPS, quadripolar catheters were placed in the standard His and right ventricular apex (RVA) locations. Placement of a decapolar catheter within the CS was challenging, so basic EPS was conducted just using His and RVA catheters. This revealed an abnormally short His-ventricular (HV) interval of 16 ms, non-decremental AV nodal conduction from an abnormal pathway.

### Timeline

| Timeframe                  | Description                                                                                                                                 |
|----------------------------|---------------------------------------------------------------------------------------------------------------------------------------------|
| **Day 1:** First EPS       | Electrocardiogram (ECG): manifest pre-excitation suggesting left lateral accessory pathway (LLAP)                                           |
|                            | Accessory pathway effective refractory period (APERP) 240 ms, rapidly conducted pre-excited atrial fibrillation                                |
|                            | Coronary angiography: patent left circumflex (LCx) artery, poorly defined anomalous coronary sinus (CS) not amenable to decapolar catheter placement |
|                            | Listed for repeat EPS with additional electroanatomical mapping equipment                                                                    |
| **Day 20:** Cardiac CT     | Anomalous CS emptying into right atrial appendage                                                                                           |
|                            | Patent LCx                                                                                                                                  |
| **Day 35:** ACS            | ECG: lost manifest pre-excitation                                                                                                           |
|                            | Coronary angiography: occluded LCx, successful stenting                                                                                      |
|                            | Echocardiography: preserved left ventricular ejection fraction, posterolateral hypokinesis                                                    |
| **Day 60:** Repeat EPS     | ECG: manifest pre-excitation absent                                                                                                         |
|                            | EPS: APERP no longer high-risk (390 ms)                                                                                                     |
|                            | Prior palpitations fully settled                                                                                                           |
|                            | No ablation performed                                                                                                                       |

### Section/Category: Arrhythmias / Electrophysiology

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conduction, accentuated pre-excitation with single atrial extrastimuli (AES) indicative of a left posterolateral AP insertion, and an AP effective refractory period (APERP) of 240 ms (Figure 1B). The patient also developed recurrent pre-excited atrial fibrillation with a shortest RR interval of 306 ms during burst atrial pacing (Figure 1C) which spontaneously resolved. As the CS could not be found (Figure 2A), coronary angiography was undertaken. Relevantly, the left coronary artery had only mild atherosclerotic disease (Figure 2B). On late-phase filling cine images, there was an anomalous CS with a poorly defined origin near the right atrial free wall, a narrow elliptical os, a thin proximal section and a bulbous ectatic looking mid- to distal course (Figure 2CD, arrows).

Owing to the propensity for recurrent pre-excited AF requiring multiple DC cardioversions as well as the anomalous CS, ablation was not pursued in this instance, and a plan was made for repeat ablation under general anaesthesia guided by 3D mapping. In the interim, the patient underwent cardiac computed tomography (CT) (Figure 2E–J) which confirmed an anomalous CS emptying into the right atrial appendage on the right atrial free wall (Figure 2E–G, arrows) and a patent LCx artery (Figure 2H–J, arrows).

Four weeks later, the patient presented to hospital with central chest discomfort and an elevated high-sensitivity cardiac troponin T of 810 ng/L (normal <22 ng/L). ECG revealed absence of previously visible pre-excitation and a new rS morphology in lead V1 (Figure 3A). Echocardiography showed a preserved left ventricular (LV) ejection fraction with basal posterolateral hypokinesis (Figure 3B). Coronary angiography showed acute occlusion of an OM branch of the LCx which was successfully reconstituted with a drug-eluting stent (Figure 3C–E). Patient made an uneventful recovery following stenting and was discharged on aspirin 75 mg, clopidogrel 75 mg o.d. and simvastatin was switched to atorvastatin 80 mg o.d. in addition to his other usual medications.

At repeat EPS, after holding bisoprolol for 5 days, baseline HV interval was now 40 ms with decremental anterograde nodal conduction. Isoprenaline administration revealed predominantly nodal conduction with rare alternating pre-excitation and an APERP 390 ms (previously

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**Figure 1** Baseline 12-lead electrocardiogram and initial electrophysiology study. (A) electrocardiogram showing short PR interval and manifest pre-excitation with positive delta wave in V1, Rs QRS morphology and negative delta wave in II, III, and aVF. (B) atrial extrastimulus testing during electrophysiology study showing maximal pre-excitation at 400/240 ms with positive delta wave in V1 (green box), negative delta waves in II, III, aVF (red boxes), and V1/I QRS >1 (blue boxes) in keeping with posterolateral accessory pathway (C) atrial fibrillation during atrial burst pacing with a shortest pre-excited RR interval of 306 ms. I, II, V2, V3, V4: surface ECG leads.
Figure 2. Coronary angiography and cardiac computed tomography scan. (A) Left anterior oblique (LAO) view of unsuccessful coronary sinus catheter placement. (B) Right anterior oblique (RAO) caudal view of patent left circumflex artery and obtuse marginal branches. (C,D) Late-phase filling images during coronary angiography showing anomalous coronary sinus (yellow arrows) in right anterior oblique (C) and left anterior oblique (D) views. (E–G) Cardiac computed tomography scan showing anomalous coronary sinus emptying into right atrial free wall on axial (E) and reconstructed (F, G) views (blue arrows). (H–J) Cardiac computed tomography scan arterial phase showing axial images of patent left circumflex (red arrows).
Adenosine was administered and failed to unmask pre-excitation even in the presence of complete AV and VA block (Figure 4B). Given that the AP was now no longer clinically significant, ablation was not pursued. Since the ACS, the patient reports no further palpitations and there has been a complete absence of any overt pre-excitation on ECG.

**Discussion**

To our knowledge, this is the first case report describing ischaemic ‘ablation’ of a high-risk LLAP confirmed on serial electrocardiographic and electrophysiologic evaluations, and supported by appropriate imaging of both coronary venous and arterial systems. The presence of an anomalous CS was particularly felicitous in this instance, resulting in the first EPS being aborted and thereby allowing for an imaging study detailing not only the course of the anomalous CS but, relevantly in this case, the course of the LCx subtending the posterolateral expanse around the MVA, where the AP was by surface ECG localization.

Anomalous CS variants are very rare (<1%) and can pose a significant challenge to electrophysiologists attempting to localize APs using traditional electrogram-mapping techniques. Embryologically, the CS and APs both develop through epithelially derived cells at around 7–8 weeks gestation, and higher incidences of APs are reported in patients with an anomalous CS. Successful LLAP ablation in patients with anomalous CS variants is possible, but often requires more sophisticated set-ups involving high-density mapping or multi-modality mapping.

The LCx supplies the posterolateral LV, running a course in the posterior AV groove along the MVA between the left atrium and LV in the coronary sulcus. OM branches emerge perpendicularly from the LCx vessel and supply the majority of the posterior and lateral LV. The proximity of the LCx to the MVA is well described, with previously reported cases of LCx injury during mitral isthmus ablation. There have also been previous cases of pre-excitation mimicking ACS or being unmasked by ACS, but never being conclusively ‘ablated’ by ACS.

As AP tissue is thought to arise as a result of improper atrial and ventricular folding during embyogenesis, and is histologically similar
to normal cardiac myocardium, it is equally vulnerable to ischaemic injury. In our case, the complete abolition of pre-excitation on 12-lead ECG following ACS in an anatomically correct coronary watershed, with repeat EPS later confirming poorly evoked pre-excitation with a now much longer APERP (240 ms to 390 ms) support the ACS as being causative for loss of AP. It is conceivable that AP loss could have occurred as a consequence of direct barotrauma during balloon and stent delivery (Figure 4C). However, the fact that pre-excitation was already absent at the event of pre-treated ACS lends strength to our hypothesis.

**Conclusion**

We describe a case of an acute coronary syndrome due to left circumflex artery occlusion fortuitously abolishing an adjacent high-risk left lateral accessory pathway, the ablation of which was initially delayed due to an anomalous coronary sinus.

**Lead author biography**

Dr Jim O’Brien is a Fellow in Cardiac Electrophysiology in Liverpool Heart and Chest Hospital, UK.

**Supplementary material**

Supplementary material is available at European Heart Journal - Case Reports online.
Slide sets: A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data. Consent

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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