

Peri-Coronary Sinus flutter: a unique form of atypical atrial flutter. A case report.

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May 12, 2022

Abstract

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Funding: None

Conflict of Interest: None

ABSTRACT

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KEY WORDS

macroreentrant tachycardia, atrial flutter, catheter ablation, atypical flutter, biatrial flutter, coronary sinus flutter, ultrahigh-resolution mapping

TEXT

Introduction

In recent years, flutter circuits utilising epicardial structures have been a focus of interest, as their occurrence has become more frequent after endocardial catheter ablation and understanding the mechanisms of complex

macroreentrant tachycardias has become deeper through high resolution mapping. As already known from several anatomical and invasive studies, the coronary sinus (CS) musculature connects to the right atrium (RA) as well as the left atrium (LA) forming electrical interatrial connections. The connections to the LA can vary from 1 or 2 up to a wide continuum.

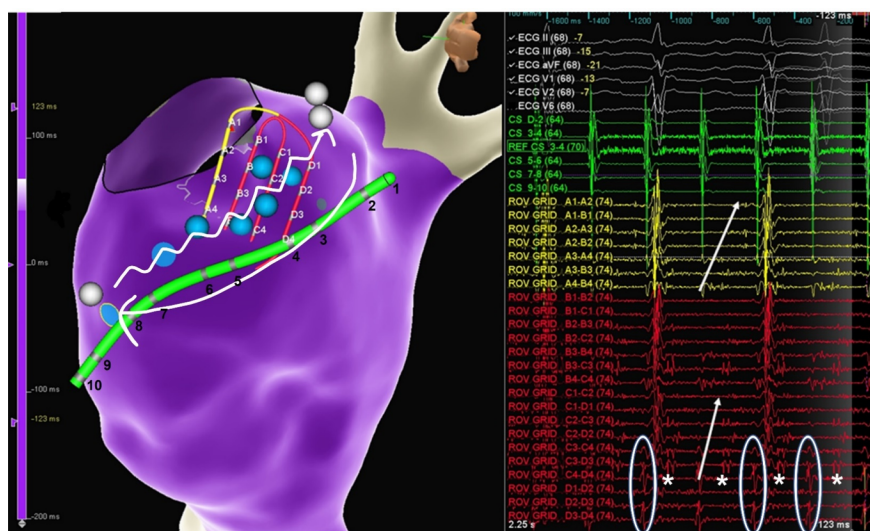
The coronary sinus often is a critical part of various forms of left atrial macroreentrant tachycardias, mostly of common perimitral flutter, and biatrial tachycardias, such as large circuits around both mitral and tricuspid valves or biatrial flutter with perimitral circuit using only the right atrial septum.

The CS musculature with its connections to RA and LA and the adjacent myocardium of LA can serve substrate for a macroreentrant circuit without utilizing other atrial areas. However, this kind of tachycardia has only been reported once.

We herein report a rare case of a peri-CS atrial flutter in a female patient after pulmonary vein isolation (PVI) four years ago and roof line ablation due to roof-dependent atypical flutter during the same procedure.

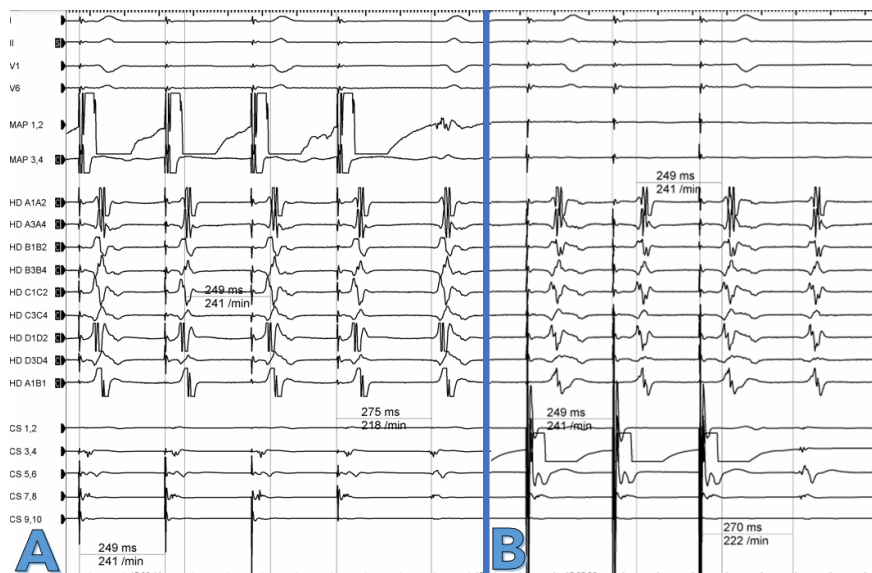
Case presentation

A 77-year-old female patient presented with shortness of breath and tachycardia of 125bpm. The electrocardiogram showed \soutan atypical flutter with atrial cycle length (CL) of 240msec. The flutter waves were monophasic positive in II, III, aVF and V1-V2, negative in aVL and isoelectric in I. A PVI-only procedure was performed four years ago. We performed a new electrophysiological study upon admission. Written informed consent was obtained. The procedure was performed under conscious sedation. A steerable decapolar catheter (Inquiry, Abbott, spacing 2-5-2) was introduced into the coronary sinus and showed a “chevron” pattern. An activation map of the left atrium (NavX Ensite Precision, Abbott, St. Paul, MN) was created using a high-density mapping catheter (HD Grid catheter SE, Abbott) with support from a steerable long sheath (Agilis, Abbott). LAT mapping and post pacing interval (PPI) mapping revealed a roof dependant atrial flutter with a “figure of 8” propagation around both pulmonary vein pairs. The pulmonary veins were persistently isolated. Low voltage was documented in the roof of LA. A roof line led to abrupt cycle length prolongation to 270msec with CS propagation distal to proximal. The P-Wave was biphasic negative in II, III, aVF, positive in V1-V2 and isoelectric in I. The decapolar catheter had one large sharp signal per cycle on each bipole *Figure 1*. An ultrahigh density activation mapping from both LA and RA was performed. In the LA it seemed that the tachycardia was centrifugal propagating from a focal exit in the inferolateral mitral annulus with highly fractionated signals. The entrance of the tachycardia to the RA was at the CS ostium. A focal mechanism of the tachycardia was primarily assumed.



Nevertheless, thorough examination of the signals of the bottom of the LA showed double signals directly opposite to the entire CS: one sharper signal simultaneous to the recorded signal in CS (encircled in Figure 1) and in addition highly fractionated signals, which presented a sequence proximal to distal (see stars and arrows in Figure 1). Examination of the propagation pattern of the adjacent LA led to the conclusion that the sharper component presented a near field signal of the LA endocard and the low voltage fractionated signal presented a far field signal of epicardial myocardial sleeves, i.e. CS musculature.

Figure 1 Signals on the grid catheter on the bottom of the LA opposite to CS with two components. Sharp signals simultaneously to the signals on the decapolar catheter (rather posteriorly, encircled). Highly fractionated signals with a sequence proximal to distal (stars and arrows). Blue globes represent exemplary points with double signals adjacent the entire CS. White globes show the exits into the LA and RA.



Extensive PPI mapping confirmed the exact macroreentrant circuit: The PPI-TCL in the entire CS, including the CS ostium and the inferolateral exit to the LA with fractionated signals, was <10msec Figure 2 .

Moreover, the PPI at several positions in the LA and RA (see exemplary white crosses in Figure 3) excluded a perimitral, a roof-dependant, and a biatrial atrial flutter utilizing only the LA and right atrial septum or the LA and the tricuspid valve.

The true circuit was a peri-coronary sinus flutter with an activation sequence proximal to distal in the coronary sinus musculature. LA excitation begins inferolateral and proceeds endocardially through the inferior LA back to the CS ostium. *Figure 3*

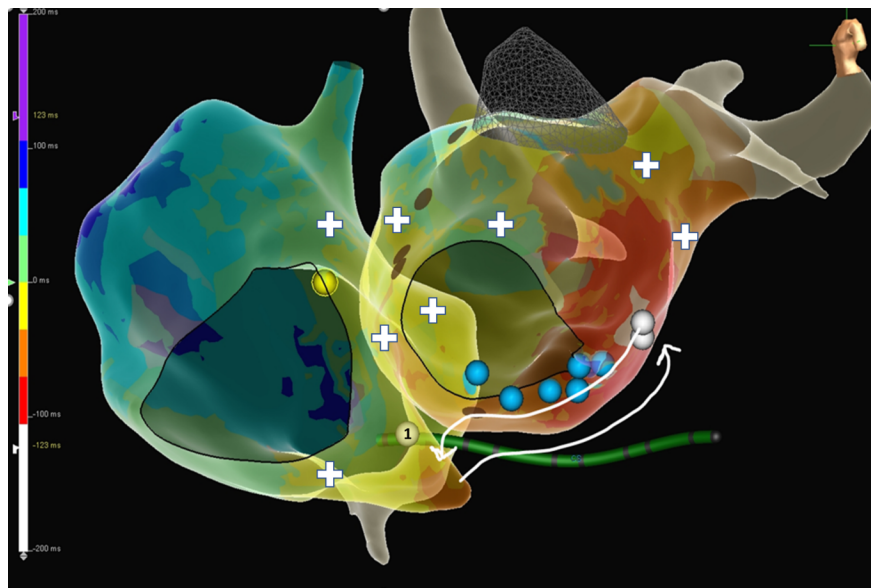


Figure 3 Propagation map of both atria with marked circuit of the peri-CS flutter. Blue spheres show exemplary areas with double signals with one highly fractionated component, white ones exits in the right and left atrium with a PPI equal to the tachycardia cycle length (CL). PPI-CL from all decapolar catheter bipoles was <20msec. White crosses represent sites with PPI-TCL>50msec.

Radiofrequency energy was delivered endocardial in LA at the area of fractionated signals at the inferolateral exit of the CS using an irrigated 3,5mm-tip ablation catheter (TactiCath SE, St Jude Medical) with a power of 40 Watts and a cut-off temperature of 43°C terminating the tachycardia at the first application.

On completion of the procedure, bidirectional conduction block at the roof line without further ablation was proven. Noninducibility of the tachycardias was also confirmed by burst pacing down to a CL of 200msec.

Discussion

Atrial arrhythmias involving the musculature of the coronary sinus are common after ablation for atrial fibrillation but also in patients without prior procedures, mostly in the context of a perimitral flutter with a critical isthmus in the LA. Less commonly, but with increasing incidence after atrial ablation, a biatrial flutter that utilizes the CS musculature is diagnosed. The increasing incidence may partly be due to a better understanding of tachycardia mechanisms through high density activation maps.

We here describe a rare type of biatrial flutter that utilizes the CS musculature, the adjacent myocardium of the LA and the right atrium in the CS orifice only. To the best of our knowledge, this type of macroreentrant tachycardia has only been described once before. Maruyama et al described a case of peri-coronary sinus flutter after ablation in the proximal CS in the context of AVNRT ablation. The CS musculature was activated from proximal to distal during the tachycardia exiting in the inferolateral LA and reaching the CS orifice endocardially again. The decapolar catheter showed double potentials in the coronary sinus with a dull second component.

In our case, the decapolar catheter in coronary sinus with its larger cylindric electrodes, despite the spacing of 2 mm, showed the endocardial activity, epicardial CS sleeves activity was not detected. This could also be due to anteroposterior position discrepancy (see Figure 1). The HD-mapping catheter with the flat small electrodes, though endocardial, was capable of showing signals of the CS musculature. Hence, the signals on the linear catheter in the coronary sinus should not be equated to the activity of the CS musculature, as

they can be far field signals of the numerous LA endocardial myocardial cells. The amplitude of a bipolar electrogram depends on the electrode size, the electrode spacing, the angle of incidence between the catheter and tissue, and the orientation of the bipole relative to the wavefront propagation. It has been previously shown in ventricular mapping that the grid catheter with a smaller electrode area, increased electrode density and orthogonal bipole orientations, was capable of detecting activation at the same anatomic sites where a linear catheter failed to record any activity.

A recent study showed that depending on the adjacent tissue EGMs acquired from larger electrodes may be composed of far-field components without near-field components.

Our case is the first case proving this unique form of peri-CS flutter through extensive biatrial ultra high-density mapping with a multipolar HD-mapping catheter. In our case there was no previous ablation within or in vicinity of the coronary sinus.

Unawareness of the possibility of such a flutter circuit could lead to underdiagnosis. Proof of participation of CS musculature in the macroreentrant circuit is ordinarily used as equivalent to confirmation of a perimitral flutter. Extensive activation and entrainment mapping including the coronary sinus and the LA bottom is of substantial importance to verify the exact tachycardia mechanism. The LA bottom, despite the distance to the pulmonary veins and the often present difficult anatomical conditions, should not be neglected.

Such a circuit could probably also coexist with a perimitral flutter as a dual loop tachycardia. Especially if during a perimitral flutter double signals with inverse activation sequences are detectable throughout the coronary sinus, such as the cases of perimitral flutter reported from Olgin et al and Jimenez et al.

Conclusion

The CS musculature with its connections to both atria can provide the anatomic substrate for macroreentrant atrial tachycardias. The peri-coronary sinus atrial flutter without utilizing a larger left atrial or biatrial circuit, although rare, should be included in the differential diagnosis of an atypical flutter. Awareness of the existence of this type of atrial flutter is required to prevent underdiagnosis. Despite the precision of the current electroanatomical maps, basic electrophysiological maneuvers such as entrainment mapping should be, if possible, part of every electrophysiological study of a stable macroreentrant tachycardia. A multipolar HD-mapping catheter with small flat electrodes can record crucial low voltage electrocardiograms. This underlines the use of HD-mapping catheters and systems to directly address the tachycardia pathomechanism and prevent needless ablation due to misunderstanding of the tachycardia.