Focal Source of Atrial Fibrillation Arising From the Ostium of the Inferior Vena Cava

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A patient presented with paroxysmal atrial fibrillation (AF) caused by focal rapid discharges arising from the ostium of the inferior vena cava (IVC). Surface ECG showed typical features of AF and flutter when rapid activation at the IVC ostium was conducted to the right atrium in a 1-to-1 and 2-to-1 fashion, respectively. Discrete radiofrequency energy applications at the medial ostium of the IVC completely eliminated the atrial tachyarrhythmias. (Circ J 2005; 69: 756–759)

Key Words: Atrial fibrillation; Catheter ablation; Inferior vena cava

Although it has been established that the initiation and maintenance of paroxysmal atrial fibrillation (AF) is frequently associated with ectopic beats inside the thoracic veins (pulmonary veins: PVs, superior vena cava: SVC, coronary sinus: CS),1–3 the inferior vena cava (IVC) has been regarded as exempt from arrhythmogenesis. We present a patient with paroxysmal episodes of AF that were initiated and maintained by focal rapid discharges arising from the ostium of the IVC and successfully eliminated by discrete radiofrequency (RF) applications.

Case Report

A 57-year-old man with a 3-year history of palpitations was referred for curative treatment of drug-resistant paroxysmal AF. Twelve-lead ECGs recorded before admission demonstrated typical features of both AF and atrial flutter (AFL, suggestive of counterclockwise typical flutter). Other examinations on admission, such as 2-dimensional echocardiogram, chest radiogram, and laboratory tests, were all unremarkable.

Mapping of the left atrium and PVs was performed through the patent foramen ovale using a 7-Fr roving ablation catheter and a 7-Fr steerable circular catheter equipped with 10 electrodes. During a period of spontaneous ectopic beats, endocardial recordings in the left atrium, including each of the 4 PVs, did not show any earlier activation relative to the surface P wave.

The site of the earliest activation of the ectopic beats was mapped to the antero-medial ostium of the IVC, and this arrhythmogenic focus behaved exactly like those of the PVs (Fig 1). During sinus rhythm (SR), a sharp potential representing the passive IVC activation appeared at the terminal portion of the atrial electrogram and was followed by an ectopic discharge either without conduction to the atrium or provoking a train of spike discharges, which conducted to the remaining atria in an approximately 2–3-to-1 fashion. As demonstrated in Fig 2A, endocardial recordings in both atria showed rapid and organized activity during sustained episodes of AF, preceded by generally regular spike-potentials at the IVC ostium (cycle length: 140–160 ms). Local conduction block (Wenckebach or 2-to-1 block) between the IVC and right atrium (RA) frequently occurred spontaneously or by catheter-tip pressure during sustained tachycardias, producing surface ECG features almost identical to common AFL (Fig 2B).

The first application of RF energy at the antero-medial ostium of the IVC (Fig 3) immediately terminated the tachycardia. After additional RF application at the contiguous site, it appeared that IVC pacing only captured the local myocardium without conducting to the atrium, suggesting the electrical isolation of the IVC from the remaining atrium (Fig 4). Because neither ectopic beats nor atrial tachyarrhythmias reappeared, even under isoproterenol infusion and vigorous rapid atrial stimulations, the session was ended with no further RF lesions. The patient has experienced no symptoms or ECG evidence of tachycardia during a 12-month follow-up.

Discussion

Although it is well recognized that the thoracic veins (PVs, SVC, CS and its branches) are the major source of atrial tachyarrhythmias (AF and atrial tachycardia)1–3 the IVC has been thought exempt from arrhythmogenesis. However, recently Mansour et al reported 2 cases of AF initiated by ectopic beats originating from the posterolateral ostium of the IVC4 and Šcavee et al also described an AF case initiated by repetitive firing inside the IVC.5 In the present case, focal rapid discharges at the IVC ostium were responsible not only for the initiation but also for the maintenance of the atrial tachyarrhythmias. The surface ECG patterns of AF and AFL were respectively produced by a 1-to-1 and 2-to-1 conduction of focal discharges at the IVC ostium to the RA, and both were simultaneously eliminated by discrete RF energy application. To our knowledge, this is the first reported case of a focal source of AF in the IVC. 

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Fig 1. Initiation and maintenance of atrial fibrillation by ectopic beats originating from the ostium of the inferior vena cava (IVC). A sharp potential appeared at the terminal portion of the atrial electrogram during sinus rhythm (arrow), and was followed by an ectopic discharge either without conduction to the atrium (arrowhead), or provoking a train of spike discharges that conducted to the remaining atria in an approximately 2–3-to-1 fashion (double arrowheads and asterisks).

II and aVF, recordings from 12-lead surface ECG; CS, proximal coronary sinus recording near its ostium; RSPV and IVC, recordings inside the right superior pulmonary vein (#1–2, #5–6 and #9–10 represent 3 bipoles recorded by the circular mapping catheter) and at the ostium of the inferior vena cava, respectively.

Fig 2. Recordings during tachycardia show the ECG patterns of atrial fibrillation (AF) and atrial flutter (AFL). (Panel A) Although the surface ECG demonstrated disorganized AF, the endocardial recordings from the RSPV and IVC ostium showed mostly regular, repetitive rapid firing. Recordings inside the coronary sinus (CS) showed irregular and disorganized activity. (Panel B) When the focal firing at the IVC ostium conducted to the remaining atria in a 2-to-1 fashion, all the endocardial recordings showed organized activity, producing the surface ECG pattern of AFL. II, III and V1, recordings from 12-lead surface ECG; CSp and CSd, proximal and distal coronary sinus recordings, respectively; RSPV and IVC, recordings inside the right superior pulmonary vein and at the ostium of the inferior vena cava, respectively.
Fig 3. (Panel A) Angiography of the IVC shows the position of the successful ablation site (arrowhead) just below the IVC–right atrium junction (Upper: right anterior oblique view; Lower: left anterior oblique view). (Panel B) Angiography of the CS performed at the beginning of the session (RAO and LAO views). Comparison of the results suggested that the successful ablation site was located laterally and posteriorly to the CS ostium and faced an upright structure at the labium of IVC ostium (possibly the Eustachian ridge).

Fig 4. Electrical disconnection of the IVC from the remaining atrium. (Panel A) ECG and intracardiac recordings after the initial application of radiofrequency (RF). Although ectopic beats were eliminated by the initial RF delivery, the IVC potential (arrowhead) was passively activated at the terminal portion of the atrial electrogram during sinus rhythm. (Panel B) Another RF application disconnected the IVC from the remaining atrium. IVC pacing from bipoles #5–6 (arrow) on the circular catheter only captured the local myocardium (arrowhead), but could not affect the regular sinus rhythm. As demonstrated in the magnified traces, a sharp potential just after the atrial activation observed in Panel A disappeared in B, and a similar sharp potential could be detected following the pacing stimulus. II, recording from surface ECG lead II; CS (#1–2 to 9–10), 5 bipolar recordings inside the coronary sinus (CS7–8 and CS9–10 located at the CS ostium). ABL, recording from the mapping catheter at the IVC–RA junction; IVC (#1–2, 3–4, to 9–10), 5 bipolar recordings with a 10-electrode circular mapping catheter inside the IVC.
contributing to the initiation and maintenance of AF.

The difference in the electrical and mechanical activity between the SVC and IVC has been described in both humans and animals. Spach et al reported that the excitation extended 2–5 cm into the SVC during SR, but no evidence of electrical activity could be found inside the IVC in either humans or animals. These differences may relate to different developmental processes between the two venous systems; the SVC is embryologically derived from the anterior cardinal vein, whereas the terminal segment of the IVC is derived from the right vitelline vein. Extension of myocardial sleeves into both the SVC and IVC in humans has been well described by Hashizume et al who showed that the length of the myocardial extension into the IVC was much shorter, and there were less longitudinal fibers than in the SVC (18 mm vs 45 mm in the IVC and SVC, respectively). Recently Kholová et al reported that although the myocardial extensions were almost always found in both the SVC and IVC in human autopsied hearts, frequent absence of an electrical connection between the IVC and RA myocardium would make the IVC electrically more silent than the SVC. Although these anatomical studies support that the IVC is more silent than the SVC, the present and previous cases support the possibility that the IVC can become arrhythmogenic.

There may be debate concerning the precise location of the focal source of arrhythmia in this case. Because the successful ablation site was not located deep inside the IVC but at its ostium, any structure around the IVC ostium (i.e., RA tissue or the ostium of CS) could be a candidate for the focus. In fact, it may be difficult to precisely localize the successful ablation site as either at the ostium or just on the labium of IVC (Eustachian ridge) from angiographic views. However, the electrophysiological features of a focal source of discharges with or without conduction to the atrium initiating and perpetuating AF closely resemble those of a PV or CS source and suggest that the origin in the present case was related to a venous structure.

References