Atrial arrhythmias arising from the superior vena cava presenting as paroxysmal atrial fibrillation, flutter and focal atrial tachycardia

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ABSTRACT

The superior vena cava (SVC) harbours about 25% - 40% of the non-pulmonary vein foci in atrial fibrillation (AF) and could manifest itself as paroxysmal atrial tachycardia (AT). AF ablation focusing on pulmonary vein isolation alone could miss SVC ectopy and result in failure of the procedure. Successful ablation is usually curative in SVC ectopy-induced AT / AF, however, potential complications include injury to the phrenic nerve, vagus nerve or the sino-atrial node. A focal ablation approach or SVC isolation are both proven options in the management of SVC tachycardia. In this article, we report SVC ectopy with variable conduction into the right atrium mimicking sinus rhythm, AT, atrial flutter (AFL) or AF. SA Heart® 2024;21:58-65

INTRODUCTION

Pulmonary vein (PV) isolation has been proven to be a useful strategy for paroxysmal atrial fibrillation (AF) with origin in the PVs worldwide. However, non-PV foci play an important role in initiating and maintaining AF in about 20% of patients. Non-PV foci are located at sites including the superior vena cava (SVC), left atrial (LA) posterior wall, the crista terminalis, the coronary sinus, the ligament of Marshall, the inter-atrial septum, the left atrial appendage.[1,2] In particular, the SVC harbours 25% - 40% of the non-PV foci for AF and is the most common non-PV source.[3] Atrial myocardial sleeves into the superior vena cava (SVC) are well known to cause focal ectopy which can induce AT / AF. We report our experience with SVC ablation performed as a stand-alone procedure for patients demonstrated to have focal ectopy from the SVC and presenting with focal AT / AFL / AF.

METHOD

A total of 323 patients who underwent ablation for atrial tachycardia and atrial fibrillation were analysed retrospectively and 3 patients with SVC-origin ectopy-induced AT / AFL / AF encountered between 2009 and 2023 in Christian Medical College, Vellore, India, were included in the study. All 3 patients presented with AT / AFL or AF were proven to be SVC ectopy initiated at cardiac electrophysiological study (EPS) and followed up after successful RFA. St Jude Medical EnSite NavX 3D mapping system was used for the creation of geometry, activation mapping and RFA.

CASES

Case 1:

A 52-year-old male presented to us with chief complaints of paroxysmal palpitations and chest pain for 3 months. The 12 lead ECG obtained during symptoms showed narrow QRS complex regular tachycardia which was initially diagnosed as AFL with 2:1 atrio-ventricular (AV) conduction (Figure 1). The patient was taken for an electrophysiological study (EPS) followed by radio-frequency ablation as appropriate. At EPS, the diagnostic EP catheters (Dua-Decapolar around the tricuspid annulus, Decapolar in the CS, 2 quadri-polar catheters in the right atrium – RA and the right ventricle – RV respectively) and a mapping and ablation catheter (St Jude Medical 8mm tip non-irrigation catheter) were utilised. The initial electrograms deceptively suggested an atrial flutter (AFL) rhythm with 2:1 A-V conduction and demonstrated a sequential activation around the lateral RA. The His-channel "A", however, was earlier, compared to the "A" in the CS 9/10 channels (Figure 2) unlike what is expected in typical cavo-tricuspid isthmus (CTI) dependent macro-re-entrant counter-clockwise right atrial flutter. The CS atrial activation progressed in a proximal to distal sequence. Catheter-induced RBBB was evident at this stage. The rhythm spontaneously degenerated into AF which

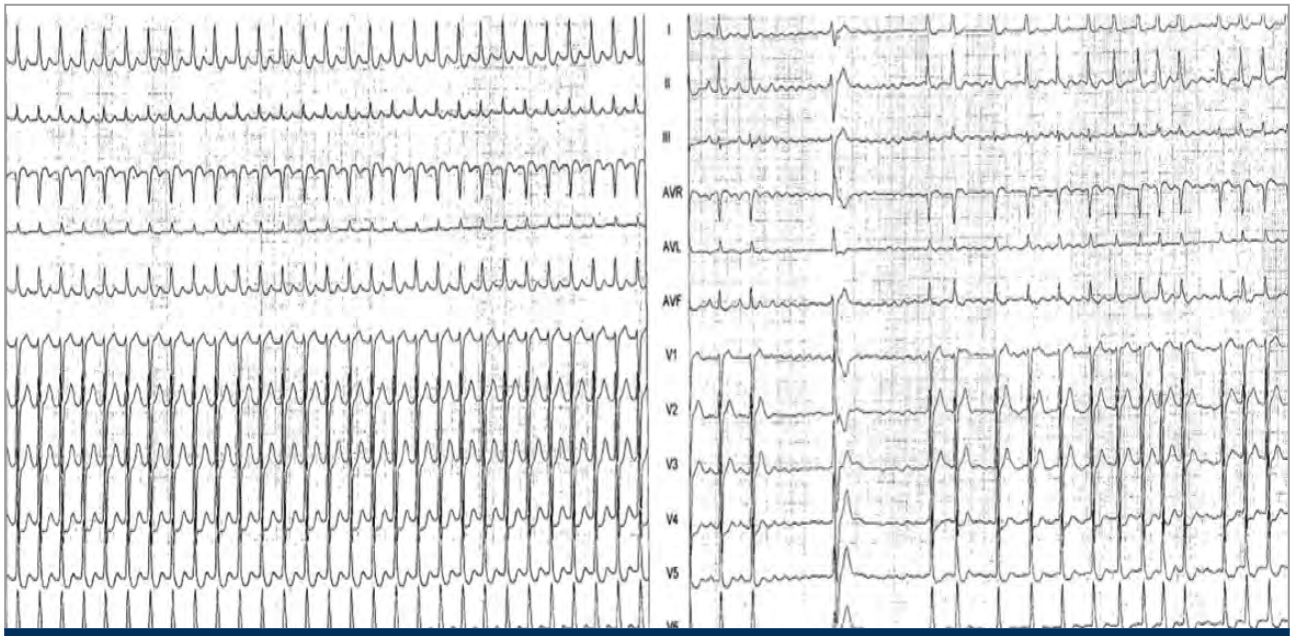

FIGURE 1: On the left side ECG shows narrowing complex short RP tachycardia on the left side which frequently degenerates into atrial fibrillation on adenosine administration as seen on the right side ECG.

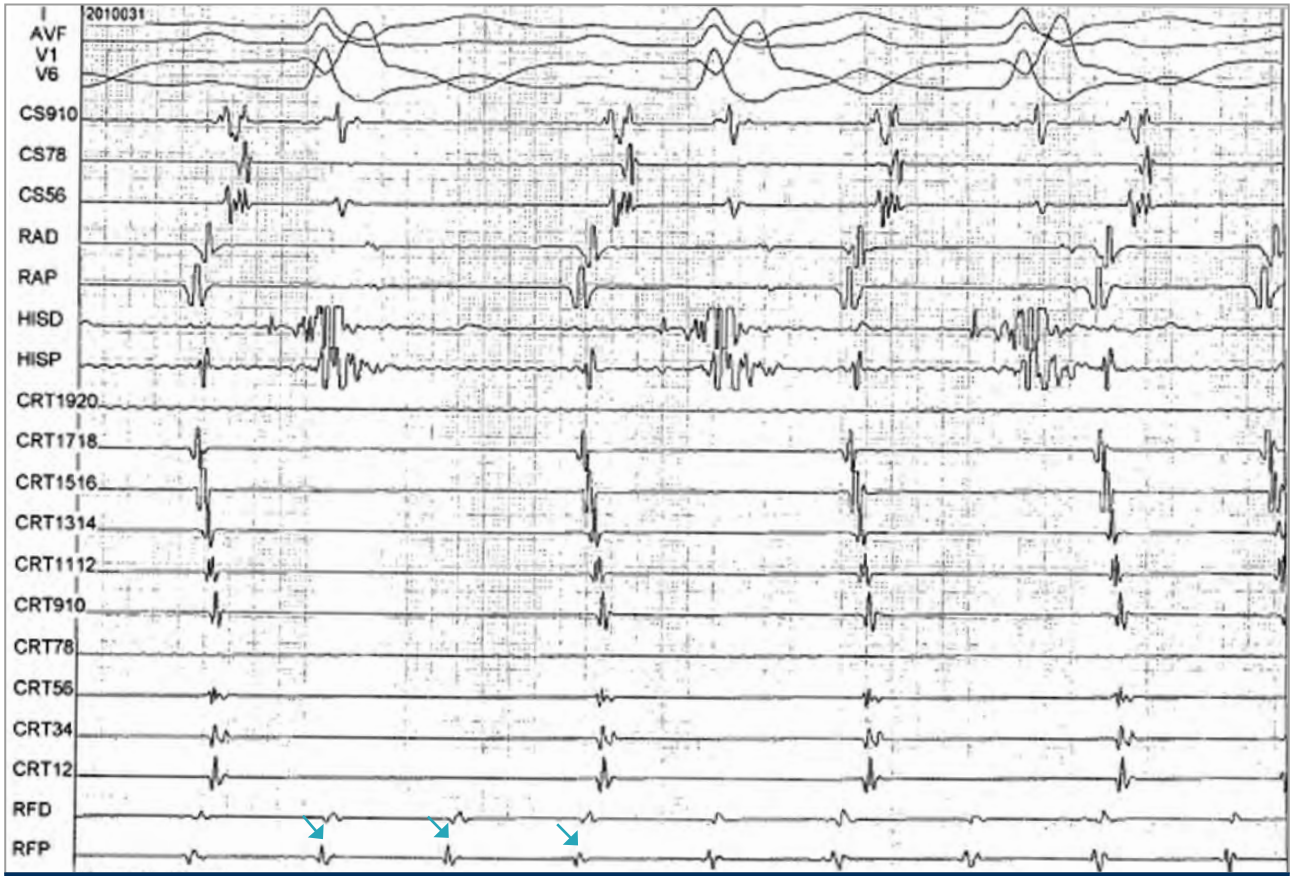

FIGURE 2: Shows superior to inferior activation of the lateral RA and bidirectional activation wavefront in coronary sinus with ablation catheter in SVC showing continuous firing of SVC (blue arrows) with variable conduction into RA and LA. Also, note the "A" signal on the His channel (HisD and HisP) was earlier than the "A" signal on the proximal coronary sinus (CS910) suggesting against typical Cavo-tricuspid dependent flutter.

then terminated to demonstrate sinus tachycardia-like P waves. Repeatedly the rhythm abruptly accelerated to AF before switching back to 1:1, 2:1 or variable A-V node conduction with AT / AFL pattern on ECG, however, the intracardiac EGMs during the AF and AT were consistently showing organised and regular firing from the SVC indicating a focal mechanism for the tachycardia during all the different manifestations on ECG described above (Figure 3 & 4). The earliest atrial EGMs were evident in the high RA (RA proximal) and the Dua-Decapolar catheter was exchanged for a Crista 2-2-2 spacing catheter and a superior to inferior activation pattern was evident straight-away. The ablation catheter was also exchanged for a Biosense Webster 4mm non-irrigated mapping and ablation catheter and was positioned in the SVC. High up in the SVC, rapid regular potentials were evident, present all the time with variable degrees of decrement sequentially, at the SVC-RA junction, and variably, at the A-V node (Figure 3). The beats originating high up from SVC also showed rapid firing with a cycle length of 145 - 170ms with a fairly constant rate and a variable SVC to RA exit block. These potentials were mapped to the postero-lateral SVC and RF energy application was initiated at this site, without termination of the tachycardia. At this stage, a linear RF lesion set was attempted to connect to the SVC-RA junction and the adjoining superior RA. As this lesion set progressed, dissociation was noted between the distal to proximal RF channels and slowing of the CL resulted in termination. No further tachycardia occurred on testing with atrial burst pacing, decrementing up to 200ms CL. The patient developed symptoms of vagus nerve dysfunction in the form of oesophageal and gastric symptoms which subsided gradually over the course of 2 years after the procedure. The patient complained of symptom recurrence after 2 weeks without any documented arrhythmia and he was taken back for EPS which however did not reveal any SVC firing. SVC isolation was attempted due to the recurrence of the symptoms, although no potential could be demonstrated in SVC (no demonstration of SVC isolation possible). At his last visit the patient had been free of symptomatic arrhythmia for 12 years.

Case 2:

A 31-year-old male who underwent a successful radiofrequency ablation procedure for a left-sided accessory pathway in a different centre 5 years prior and presented to us with chief complaints of recurrent palpitations and 1 episode of syncope. Each episode of palpitations was sudden in onset and offset, increasing with exertion. The ECG taken immediately after the syncope demonstrated focal AT with no pre-excitation (Figure 5). Exercise stress test (EST) showed bursts of narrow complex tachycardia with a 1:1 A-V relationship. The bursts occurred at about 200bpm and progressed to a sustained tachycardia demonstrating slowing of the tachycardia before termination. The tachycardia P waves were almost identical (in

FIGURE 3: Shows the continuous firing as detected by the ablation catheter (RFD) in the SVC with a decrement in RA. Note the highlighted ECG (blue colour) suggesting an AT with 1:1 AV node conduction. The ECG varied markedly and could be read as AT or AF while the intracardiac EGMs showed persistent firing from SVC.

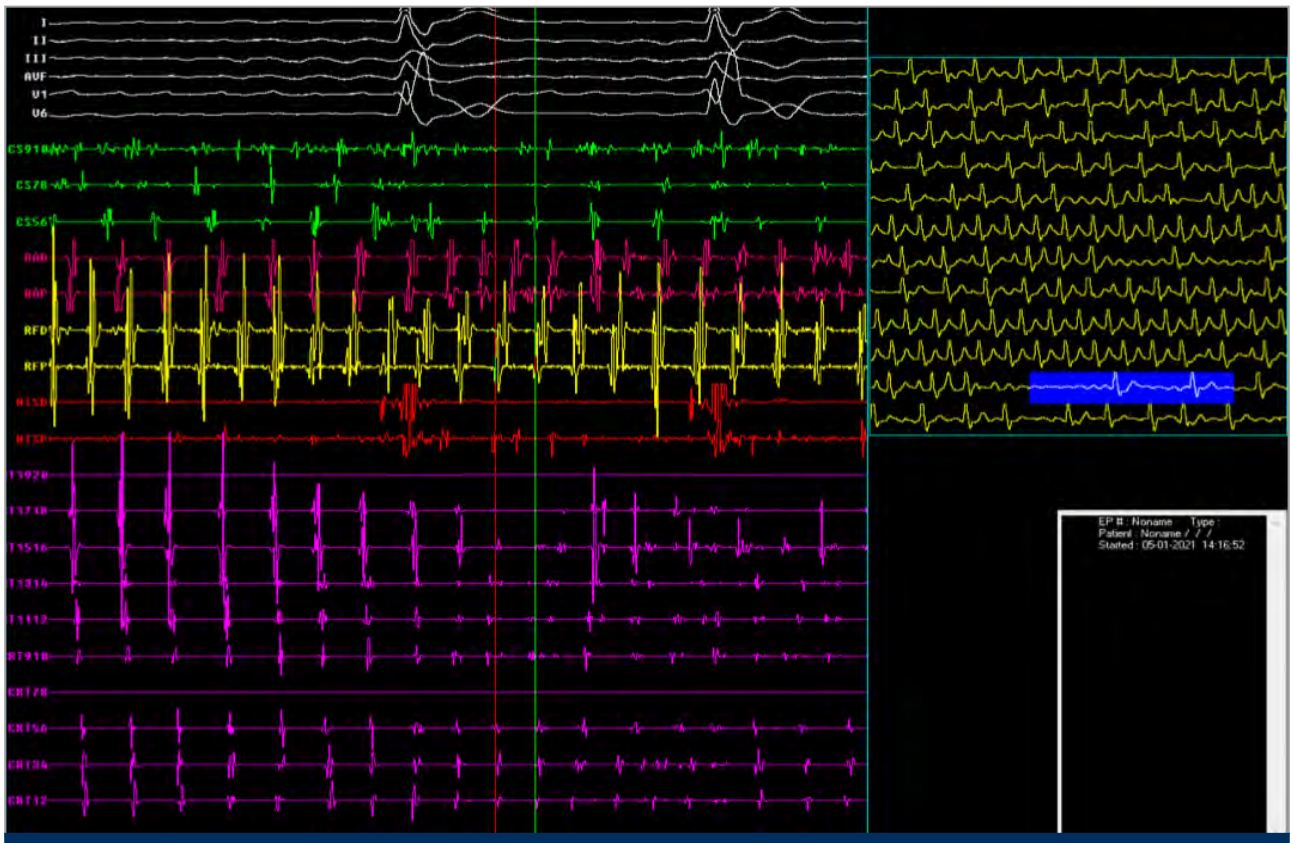

FIGURE 4: The Holter on the right side shows ECG suggestive of atrial fibrillation (see the closest showing highlighted ECG (in blue) and the corresponding EGMs show sharp EGMs (bright yellow colour) arising from SVC (ablation catheter in SVC).

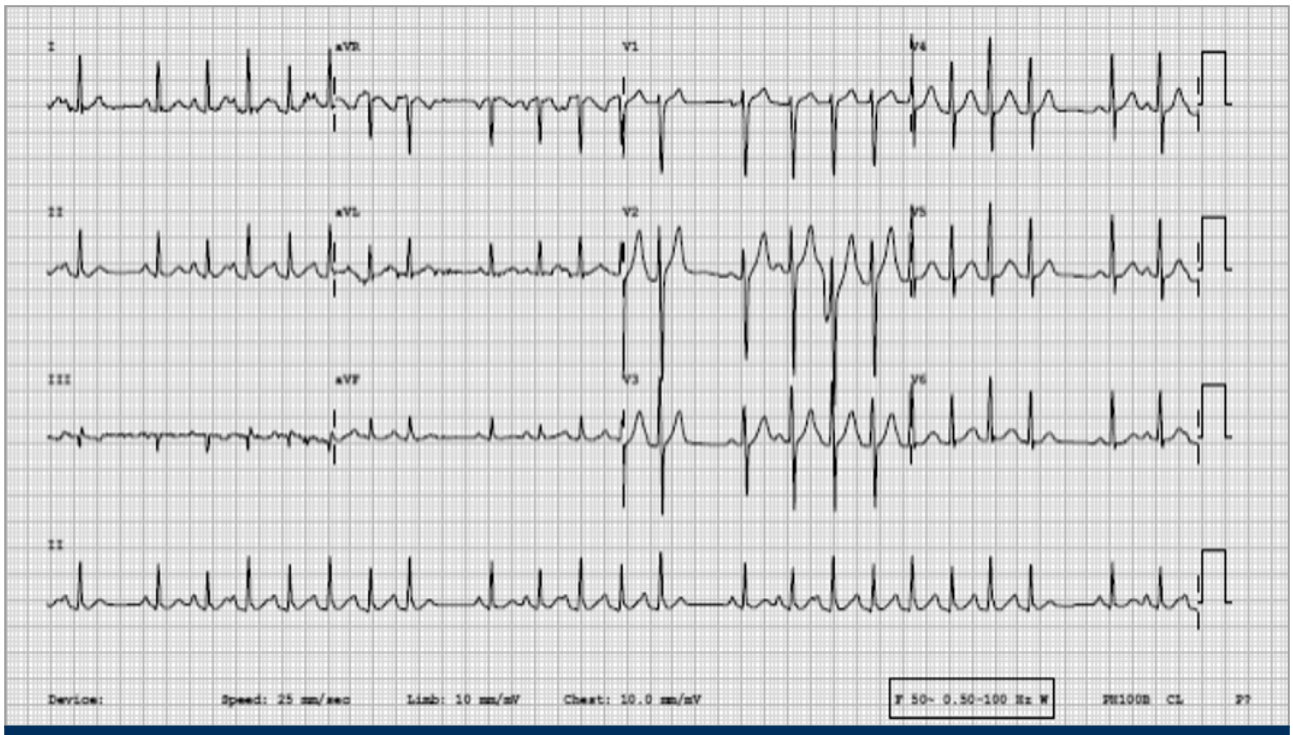

FIGURE 5: Shows bursts of focal AT with P waves positive in limb leads LI and LII, and negative in lead aVR suggestive of origin from the high right atrium.

morphology and axis) to the sinus P waves except that they were slightly larger with a sharper peak in lead II. He underwent EPS and radio-frequency ablation with 3D mapping (St Jude Medical Ensite NavX). At EPS frequent focal atrial premature contractions (APC) in the form of atrial bigeminy occurred with isoproterenol infusion. The earliest atrial activation was in the HRA catheter. Within 2 - 3 minutes of the appearance of APCs, repeatedly, runs of long RP narrow complex tachycardia (variable conduction) were induced with intermittent termination and re-initiation after 2 - 3 sinus beats. Tachycardia could not be entrained, and all the atrial EGMs fell within a narrow window suggesting a focal AT with the earliest atrial activation mapped to the postero-medial aspect of SVC, 1 - 2cm superior to the SVC- RA junction. This was 100ms earlier compared to the proximal CS reference EGM (CS 9-10), but just 40ms earlier compared to the right upper PV. The tachycardia terminated spontaneously and was no longer inducible, hence the early site in the SVC obtained from the activation map on the 3D mapping system was used for successful RFA lesion delivery (Figure 6) on the same day. Exercise stress testing 2 days after did not show any tachycardia and there was no recurrence on follow-up after 5 years.

Case 3:

A 46-year-old female presented with a history of recurrent episodic palpitations associated with pre-syncope for one year. Her ECG and trans-thoracic echocardiogram were normal and the Holter study showed multiple runs of very rapid atrial tachycardia with P waves very similar to the sinus morphology. She underwent EPS which revealed fast AT with the origin from SVC with SVC potentials and this was confirmed with a 15 - 25mm variable loop multi-polar Lasso catheter. Pacing from high up in the SVC at a low voltage output captured the RA myocardium indicating the connection of the SVC muscle sleeves with the RA (Figure 7). A 3D mapping system (St Jude Ensite NavX) was used for creating RA geometry and activation mapping of frequent APCs which revealed a focal origin in the SVC with the EGMs in SVC preceding P wave by 47ms

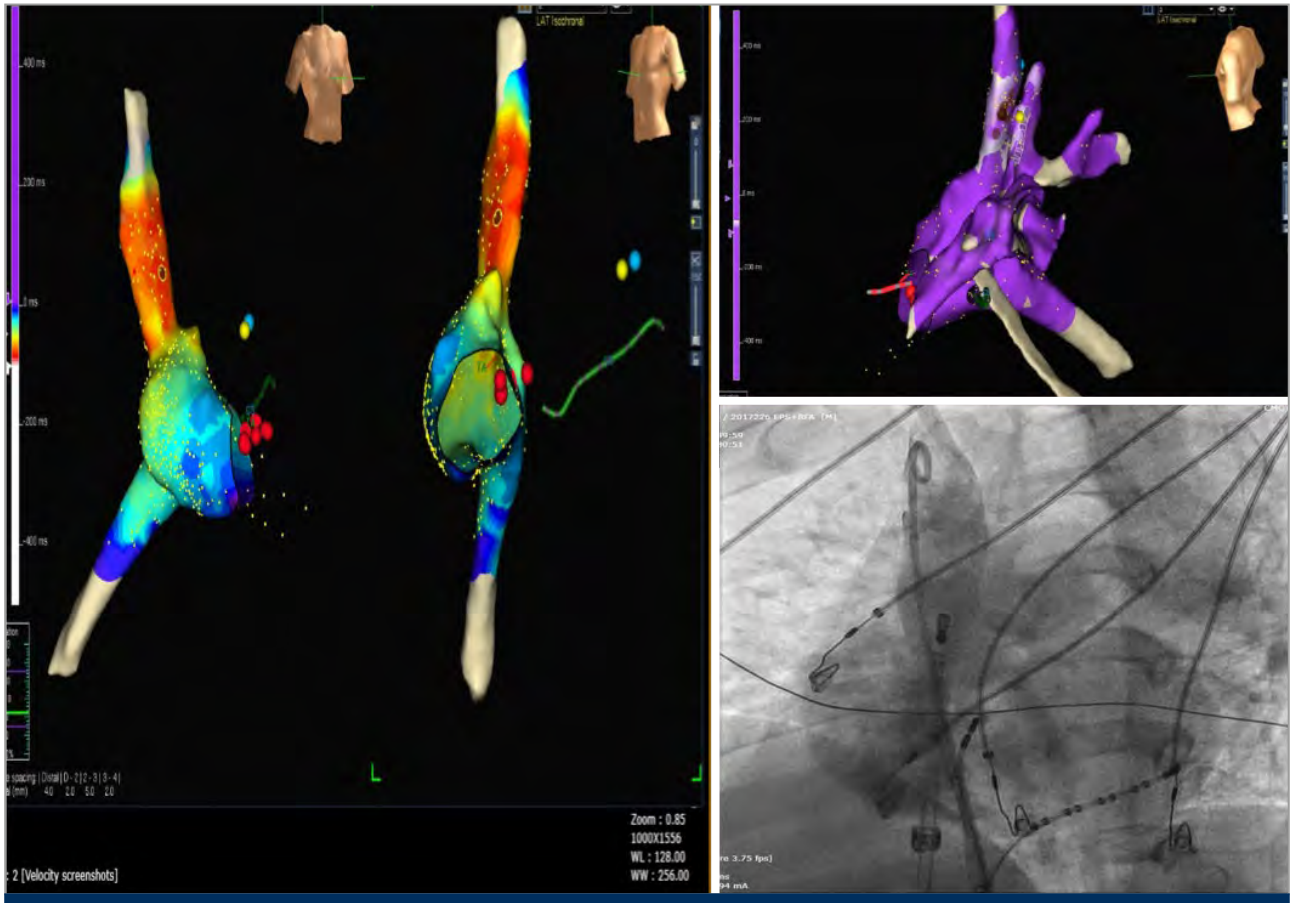

FIGURE 6: Shows on activation mapping the earliest signals were noted in SVC with late activation of the right upper and lower pulmonary veins (left and upper right). SVC EGMs preceded the P wave by 30ms. In the right lower part of the figure, the angiogram shows a pigtail catheter in high up in SVC with an ablation catheter in SVC antrum. Also seen in the Figure are His catheter and Decapolar catheter in the coronary sinus.

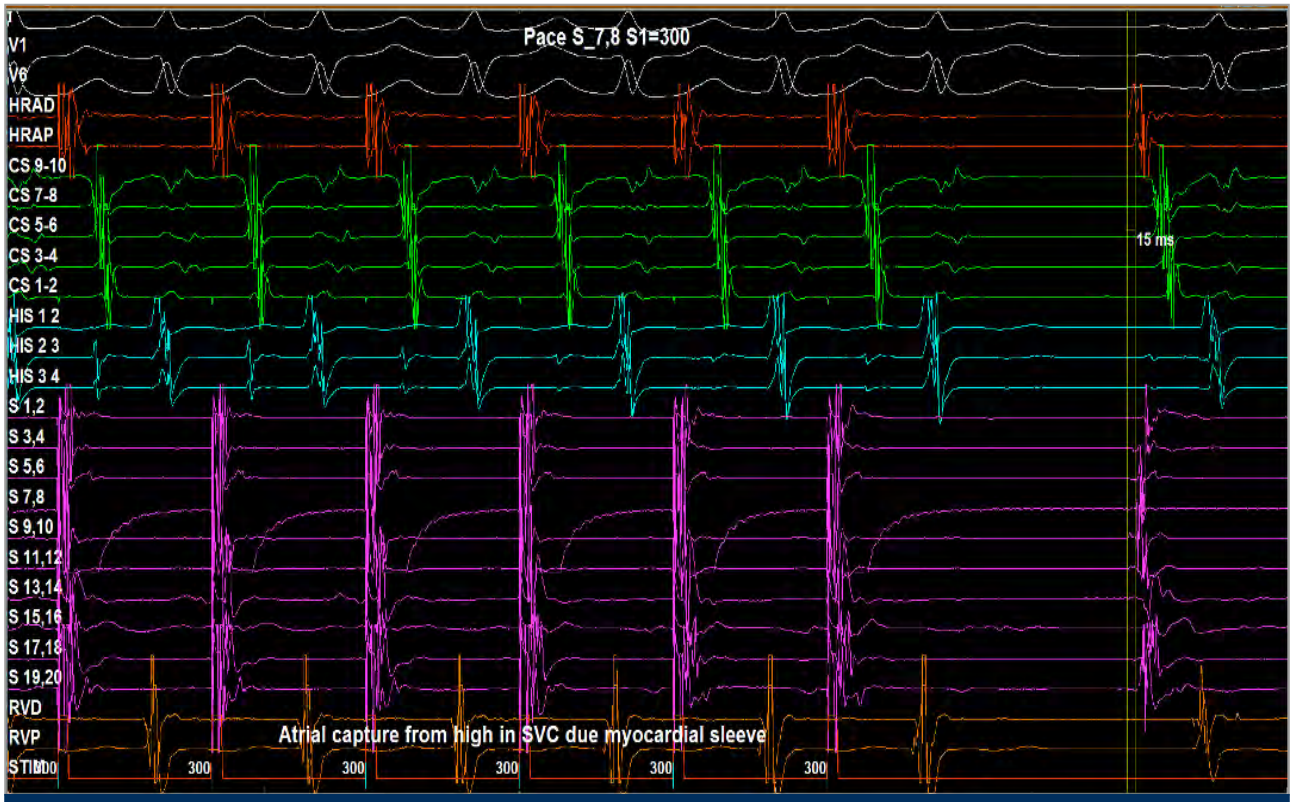

FIGURE 7: Shows atrial capture on pacing at low output from a Lasso catheter placed high up in SVC indicating SVC myocardial capture with connection with RA.

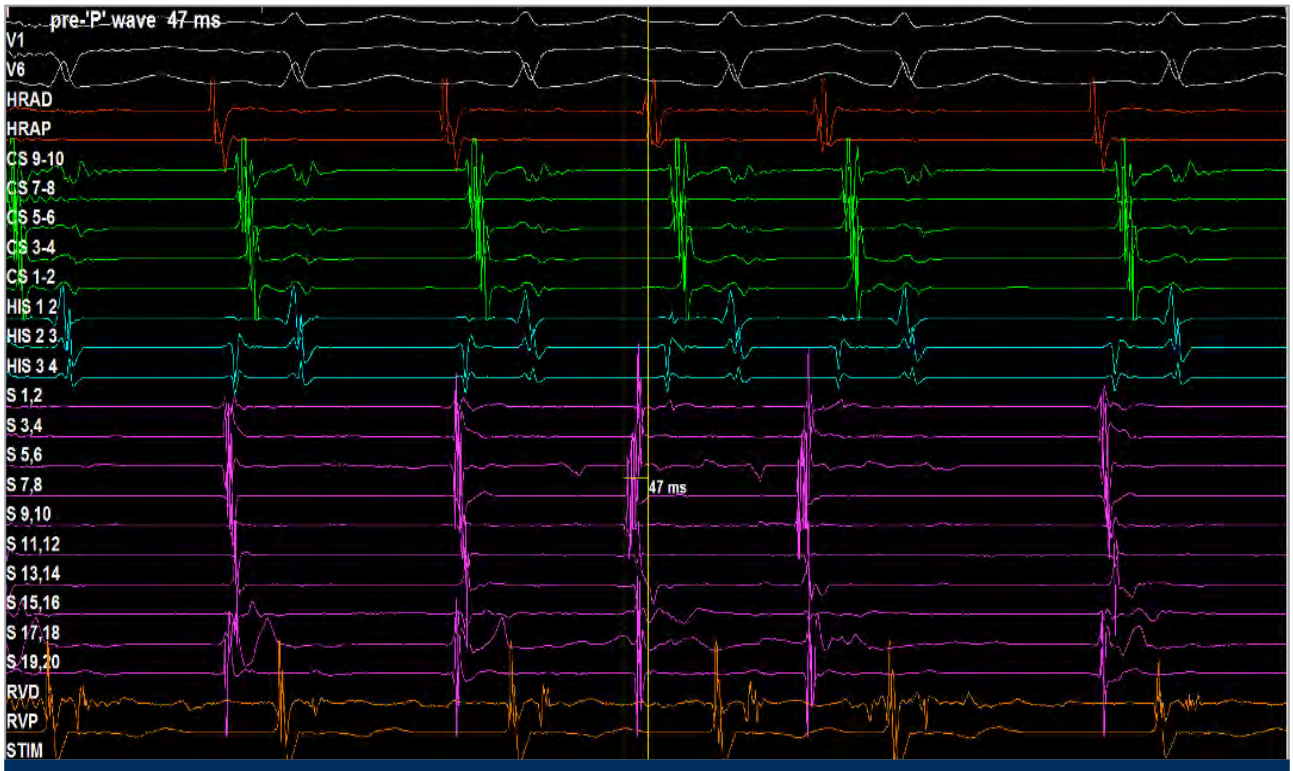

FIGURE 8: Shows activation mapping during APCs with the earliest signal from SVC preceding P wave by about 47ms. Note that the Duo-Decapolar catheter shows activation in the lateral RA.

(Figure 8). SVC tachycardia was spontaneously induced during isoprenaline infusion and the reversal of atrial activation on the St Jude Medical Tacticath Quartz catheter ablation catheter (i.e. proximal to distal in sinus rhythm, distal to proximal during tachycardia) indicating the origin for the respective rhythm. Focal ablation targeting the earliest signal in the postero-superior SVC was not successful. Progressive RF applications proceeding from a superior to the inferior direction for eliminating all potentials guided by the Lasso were attempted with no success. Finally, in the SVC / RA junction, a segmental approach was used, guided by the Lasso catheter channel EGMs and RF applications in the multiple RF applications were required linearly towards the SVC-RA junction anterolaterally for the termination of the tachycardia. After the procedure, the patient developed asymptomatic sinus bradycardia due to inadvertent sinus nodal damage. The patient was asymptomatic on follow-up at 4 years with a peak exercise heart rate of 118 beats per minute on the treadmill test

RESULTS

All 3 patients found to have SVC-origin atrial tachycardia in our series had successful ablation of SVC-origin tachycardia. One of these patients presented with paroxysmal AF and atrial tachycardia at other times and the AF was proven to have a trigger from SVC and the other 2 patients presented with focal atrial tachycardia. During follow-up no patient had recurrent symptoms after SVC ablation. One of the patients in our series developed gastroparetic symptoms which resolved gradually over 2 years, quite likely due to reversible right Vagal nerve damage and another patient developed asymptomatic Sinus Node dysfunction. Our study shows the curative ablation of paroxysmal AF initiated by SVC ectopy. For focal AT with P wave morphology like the sinus P wave and the importance of SVC mapping as well as the long-term success of ablation in SVC alone was also demonstrated. The possibility of damage to the adjacent structures like the phrenic nerve, sinus node and vagal inputs to the oesophagus are potential complications while ablating in SVC.

DISCUSSION

The incidence of tachycardia of SVC origin has been reported variedly and in 1 series SVC induced AF has been reported as 5% in patients with paroxysmal AF by Miyazaki, et al.[4] The mechanism of AF originating from the SVC is thought to be similar to typical PV-related muscle sleeve firing triggered AF. Pulmonary veins are the commonest triggers of AF and the isolation of pulmonary veins is well established in the treatment of AF.[3,5] The role of SVC triggers have not been studied as well as those of the pulmonary veins. However, it has been reported in some cases by various authors.[6] Interestingly in our series of 3 patients, 2 patients presented with focal AT and 1 patient presented with paroxysmal AFL / AF which was proved to be triggered by SVC ectopy. The SVC ectopy was very rapid, conducting with variable decrement into the RA and at times it induced AF. During AF, the fast regular and organised activity in SVC was characteristic of triggered AF. In 1 of our patients, a long muscle sleeve was demonstrated, and the atrium could be captured from this relatively remote site from the atrium. Interestingly the superior extent of such muscle sleeves could be quite long, as demonstrated in our patients. Tsai, et al.[7] reported SVC muscle sleeve extensions of 33 ± 7mm above the SCV-RA junction.

SVC ectopy may present clinically as AT / AFL or AF. The fast ectopy usually conducts very rapidly in the atrium and the patient may present with paroxysmal AF due to fibrillatory conduction in the atria. Hence a high clinical suspicion of SVC ectopy is pivotal in such cases so that appropriate ablation strategies can be formulated in the management of paroxysmal AF rather than a conventional pulmonary vein isolation.[7] In our series, finding a P wave morphology similar to the sinus rhythm P wave morphology was a useful clue to SCV ectopy while there was variable ECG presentation of the arrhythmia as was seen in our first case. When Atrial activation progresses in a supero-inferior direction in the Atria, looking for the most superior extent of origin is necessary, followed up with ruling out activation potentials in the SVC using catheters such as the variable loop Lasso catheters. For mapping these SVC potentials, the direction of the activation on the EGM's obtained from any straight EP catheter in the SVC during the tachycardia, looking for a distal to proximal activation, with the catheter placed deep in the SVC and gradually withdrawn into the RA will be helpful as well. A deflectable multi-polar EP catheter may also help ensure better contact with the SVC. Just rotating it completely round at the same level while maintaining gentle flexion to ensure contact gives an estimate of the possible location of the muscle sleeve extension. Further, if left-atrial mapping in the right-sided PVs, especially the right inferior PV is pivotal to rule out earlier activation from those sites, in which case achieving PV Isolation would be the ablation strategy. Once SVC triggers are identified as the triggers of AT / AFL / AF, one could use either a focal, a segmental or a complete SVC-RA junction Isolation approach depending on the extent of clear SVC potentials. Thick or wide muscle sleeves would require more RF energy applications. Usually, there is a single breakthrough site from SVC into RA and a focal ablation may be undertaken.[8] SVC Isolation will be required if multiple or

wide sleeves are considered possible and during total isolation or a wide segmental approach (guided by potentials recorded using a Lasso catheter or similar such catheters, to avoid a complete encircling lesion set in the SVC-RA junction) potential damage to the SA node and the phrenic nerve in the immediate vicinity should be considered. Damage to the right vagus nerve is also a potential complication and at present there is no specific easy-to-use technology available for locating the right vagus nerve (which lies posteromedial to the SVC). The phrenic nerve (which lies in the lateral aspect of the SVC) however is easily located by pacing close to it in the SVC.[4] One of our patients developed vagal nerve damage symptoms that improved gradually over 2 years.

CONCLUSION

SVC-triggered AT / AFL / AF is a well-known entity and a high clinical suspicion is required to diagnose it. SVC ablation is safe and effective, however, the risk to the sino-atrial node, phrenic nerve and right vagus nerve needs to be borne in mind. The monitoring of phrenic nerve function and sinus nodal function in the form of sinus rhythm cycle length during RF application would help reduce the complications.

CLINICAL IMPLICATIONS

While dealing with focal AT / AFL or AF, SVC ectopy triggers should be considered and ruled out in the appropriate setting. Ablation in SVC alone could suffice in such situations saving the patient the risks of more complex procedures.

Conflict of interest: none declared.

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