



# Transesophageal echocardiographyfor detection of left atrial appendage thrombi: Is it good enough?

Aazda Biria<sup>1</sup>, Thomas Rosamond MD<sup>1</sup>, Dhanunjaya Lakkireddy<sup>1</sup>, Louis Wetzel, MD<sup>2</sup>.

Department of Cardiovascular Disease (Mid-America Cardiology), 2Department of Radiology, The University of Kansas Hospital

## Introduction

Transesophageal echocardiography (TEE) has beenconsidered the gold standard for visualization of left atrial appendage thrombiprior to electrocardioversion in patients with atrial fibrillation1. We report two cases in which 64-slice computed tomographic angiography (CTA)demonstrated prominent left atrial appendage thrombi in spite of a negativetransesophageal echocardiogram.

#### Case I

A 65-year old gentleman with a 10 year historyof paroxysmal atrial fibrillation and left ventricular dysfunction was beingprepared for 64-slice cardiac CTA prior to planned pulmonary venous isolation. In the interim however, he presented with symptomatic persistent atrialfibrillation and was submitted for elective cardioversion after transesophagealechocardiography was performed and failed to demonstrate a left atrialappendage thrombus (Figure 1). He was successfully cardioverted with a sub-therapeuticINR. A subsequent 64-slice CT angiogram was performed the same day as part of preparation for radiofrequency ablation. Sixty-four slice CTA demonstrated alarge thrombus in the left atrial appendage (Movie 1).

#### Case II

A 66-year old gentleman with a history of persistentatrial fibrillation for six months with therapeutic INRs was admitted to thehospital for direct current electrical cardioversion. A previous cardioversionhad failed to maintain sinus rhythm. Transesophageal echocardiogram wasperformed prior to cardioversion and did not show evidence for left atrialappendage thrombus (Figure 2). The patient was successfully cardioverted tonormal sinus rhythm without complications. The INR remained therapeuticthroughout the hospital stay. A 64-slice CTA was obtained the next day inorder to identify the pulmonary venous anatomy in preparation for pulmonaryvenous isolation but demonstrated the presence of a large mobile thrombus in he left atrial appendage (Movie 2).

#### Discussion

Transesophageal echocardiography has recognizedlimitations in the visualization of the left atrial appendage due to chambersize and shape variability.<sup>2</sup> Nevertheless, studiesemploying transesophageal echocardiography have been said to

**Corresponding Address** : Thomas L. Rosamond, M.D., Clinical Assistant Professor of Medicine, Co-Director of Cardiovascular Computed Tomography/Magnetic Resonance Imaging, Department of Cardiovascular Disease (Mid-America Cardiology), The University of Kansas Hospital, Mail Stop 4023, 3901 Rainbow Boulevard, Kansas City

show 100%sensitivity for the detection of left atrial thrombi.3 The chanceof thrombus formation in atria is high after cardioversion due to myocardial stunningand it has been considered as the cause of CVA/TIA after cardioversion. The twocases presented in this report however, demonstrate the limitation of transesophagealechocardiography to detect even large mobile thrombi in the left atrialappendage. Subtherapeutic INR in the first case may explain presence of thrombiin the left atrium at the time of CTA. However, in the presence of therapeuticanticoagulation with warfarin in the second case, left atrial appendage thrombuswas unrecognized by TEE. None of these cases had a known history of thrombosisthat suggests hypercoagualable state. Studies have shown limitations of CTA in diagnosis of left atrial appendage due to high interobserver variability,<sup>4</sup> inability of distinguishing between thrombus and slow blood flow in the appendage,<sup>5</sup> and low sensitivity in comparison to TEE5,<sup>6</sup> Left atrial appendage sometimes is difficult to visualize in its entirety by TEE as was in the two mentioned cases. The tip of the LAA if very long, curved and with greater branching could pose a problem in identifying clots with TEE.

#### Discussion

We present a patient with co-occurrence of speech and respiratory induced AT, implying a common etiology most probably involving the ANS. The origin of arrhythmia was mapped to the SVC and RSPV area, similar to the findings of Yamamoto et al in seven patients with RCAT.<sup>2</sup> The epicardial adipose tissue in this area contains a network of nerves and ganglia comprising most of the ANS traffic to the heart . Using electron micrographs from human preparations, Pausa et al demonstrated nerve fibers in a sulcus between the SVC and the RSPV, which synapse within the closely located ARGP with other nerve fibers apparently innervating the sinus node and other RA tissue .6 This was the anatomical ablation area in our patient. Biopsies of epicardial fat collected at this location from eight human patients during cardiothoracic surgery were analyzed for different neurotransmitters.<sup>7</sup> It was found that the GP has a complex anatomy, which includes cholinergic and nitrergic phenotype for most of the neurons, noradrenergic markers in some neurons, and other neurochemical inputs. Nevertheless, electrical GP stimulation in animals or humans elicits a strong vagal response.<sup>8</sup>

Near the ARGP location, the heart and the great vessels are attached to the mediastinum by the pericardial reflections, and hence may be susceptible to local stretch, like movements of the diaphragm or the trachea while speaking or breathing. Notably, in our patient, the arrhythmia started literally with the first word and subsided even between sentences. It was initiated at midinspiration as well. It is possible that activation of stretch receptors in the great vessels or in the

Figure 1: Transesophagealechocardiogram negative for presence of thrombus.



carotid sinus induced a fast hypothalamic reflex within the nucleus of the solitary tract, with efferent parasympathetic discharge through the dorsal medulla to the cardiac ganglia.9 Local physical irritation of a ganglion cannot be excluded as well. Studies in dogs demonstrate that electrical and cholinergic stimulation of the ARGP cause shortening of the ERP in the SVC and the RSPV myocardial sleeves, resulting in early after-depolarizations, rapid local firing and atrial tachyarrhythmia .<sup>10,11</sup> This appears to be the mechanism of arrhythmia in our patient, who had clinically identical ATs originating from both the SVC and the RSPV. Interestingly, two out of the seven patients with RCAT reported by Yamamoto et al also had two different arrhythmias, initiating from the same locations.<sup>2</sup> It is likely that a common trigger induced both ATs.

The arrhythmia in our patient occurred only during talking or breathing, had different morphologies, did not respond to AAD and could not be induced during EPS by programmed electrical stimulation. All of these suggest triggered activity as the mechanism of the arrhythmia. The lack of response to beta-blockers and the inability to induce AT under isoproterenol infusion exclude a sympathetic etiology. Vagal response was evidenced during RF ablation at the site of AT origin by sinus pauses and increased sinus cycle length. Such a response to electrical stimulation is used to identify ganglion sites during AF ablation.<sup>3,8</sup> Similarly, the ARGP was probably stimulated by the RF energy, being in close proximity to the tip of the catheter and therefore, ablation at this location was expected to involve the ganglion. Sinus tachycardia, which occurred after the ablation and lasted for a few months, is compatible with the expected vagal withdrawal after ablation of the ARGP, which mediates the parasympathetic traffic to the SA node .<sup>6</sup> Similarly, inappropriate sinus tachycardia has been reported in 23.5% of patients who underwent successful GP ablation as an adjunct to AF ablation.<sup>3</sup>

Balloon cryo-ablation has been developed as an alternative to RF ablation for PV isolation in AF patients. To the best of our knowledge, this is the first report of using a cryo-balloon to treat AT near a single PV, or of manipulating this balloon through a PFO. The large contact area with the atrial wall adjacent to the ARGP location probably caused plexus ablation, involving the underlying pre-ganglionic vagal nerves running between the RSPV and the SCV.<sup>6</sup> A reduction in heart rate variability parameters compatible with modulation of the intrinsic cardiac ANS has been reported after cryo-balloon PV isolation for the treatment of AF

## Conclusions

We present a patient with AT induced by speech and by respiration. The trigger of the arrhythmia appears to be parasympathetic discharge from the ARGP, mediated either by a fast ANS reflex or by

Figure 2: Transesophagealechocardiogram in Case 2 was also negative for presence of thrombus.



another local mechanism. These vagal stimuli induced rapid firing in the SVC and the RSPV myocardial sleeves and initiated ATs in both sites. Balloon cryo-ablation around the orifice of the RSPV, probably involving the ARGP and the pre-ganglionic vagal nerves, abolished the arrhythmia. Thus, targeting the cardiac ANS appears to have an important role in AT/AF ablation. Other mechanical stimuli have been reported as triggers of atrial tachyarrhythmias, including cough, swallowing or esophageal reflux.<sup>1,13</sup> As in our patient, these maneuvers may stretch the ARGP area, induce vagal discharge and initiate AT/AF. This possible etiology should be kept in mind while ablating arrhythmia in these patients.

We showed that balloon cryo-ablation might be used for the treatment of AT originating near the orifice of a single PV if a large contact area with the atrial wall is needed or if focal ablation failed. The feasibility of handling a large deflectable 15F (outer diameter) sheath and a 28 mm diameter Arctic Front cryo-balloon through a PFO is hereby demonstrated, as well as the good tolerability of the procedure in a non-sedated patient. by the authors.

#### References

1. Klein AL,Grimm RA, Murray RD, Apperson-Hansen C, Asinger RW, Black IW, Davidoff R, ErbelR, Halperin JL, Orsinelli DA, Porter TR, Stoddard MF; Assessment ofCardioversion Using Transesophageal Echocardiography Investigators. Use of transesophageal echocardiography to guide cardioversion in patients withatrial fibrillation. N Engl J Med. 2001 May 10;344(19):1411-20.

Stollberger C, Ernst G, Bonner E, Finsterer J,Slany J. Left atrial appendage morphology: comparison of transesophagealimages and postmortem casts. Z Kardiol. 2003 Apr; 92 (4):303-8.

Manning WJ, Weintraub RM, Waksmonski CA,Haering JM, Rooney PS, Maslow AD, Johnson RG, Douglas PS. Accuracy oftransesophageal echocardiography for identifying left atrial thrombi. Aprospective, intraoperative study. Ann Intern Med. 1995 Dec. 1:123(11):817-22.

Gottlieb I, Pinheiro A, Brinker JA, Corretti MC, Mayer SA, Bluemke DA, Lima JA, Marine JE, Berger RD, Calkins H, Abraham TP, Henrikson CA. Diagnostic accuracy of arterial phase 64-slice multidetector CT angiography for left atrial appendage thrombus in patients undergoing atrial fibrillation ablation. J Cardiovasc Electrophysiol. 2008 Mar;19(3):247-51.

Kim YY, Klein AL, Halliburton SS, Popovic ZB, Kuzmiak SA, Sola S, Garcia MJ, Schoenhagen P, Natale A, Desai MY. Left atrial appendage filling defects identified by multidetector computed tomography in patients undergoing radiofrequency pulmonary vein antral isolation: a comparison with transesophageal echocardiography. Am Heart J. 2007 Dec;154(6):1199-205.

# Disclosures

No disclosures relevant to this article were made can Movie 1: CTAngiogram from Case 1 showing presence of thrombus.



Shapiro MD, Neilan TG, Jassal DS, Samy B, Nasir K, Hoffmann U, Sarwar A, Butler J, Brady TJ, Cury RC. Multidetector computed tomography for the detection of left atrial appendage

thrombus: a comparative study with transesophageal echocardiography. J Comput Assist Tomogr. 2007 Nov-Dec;31(6):905-9

Movie 2: CTAngiogram from Case 2 showing presence of thrombus.

