

Frequent Asymptomatic VPCs in a Young Competitive Athlete

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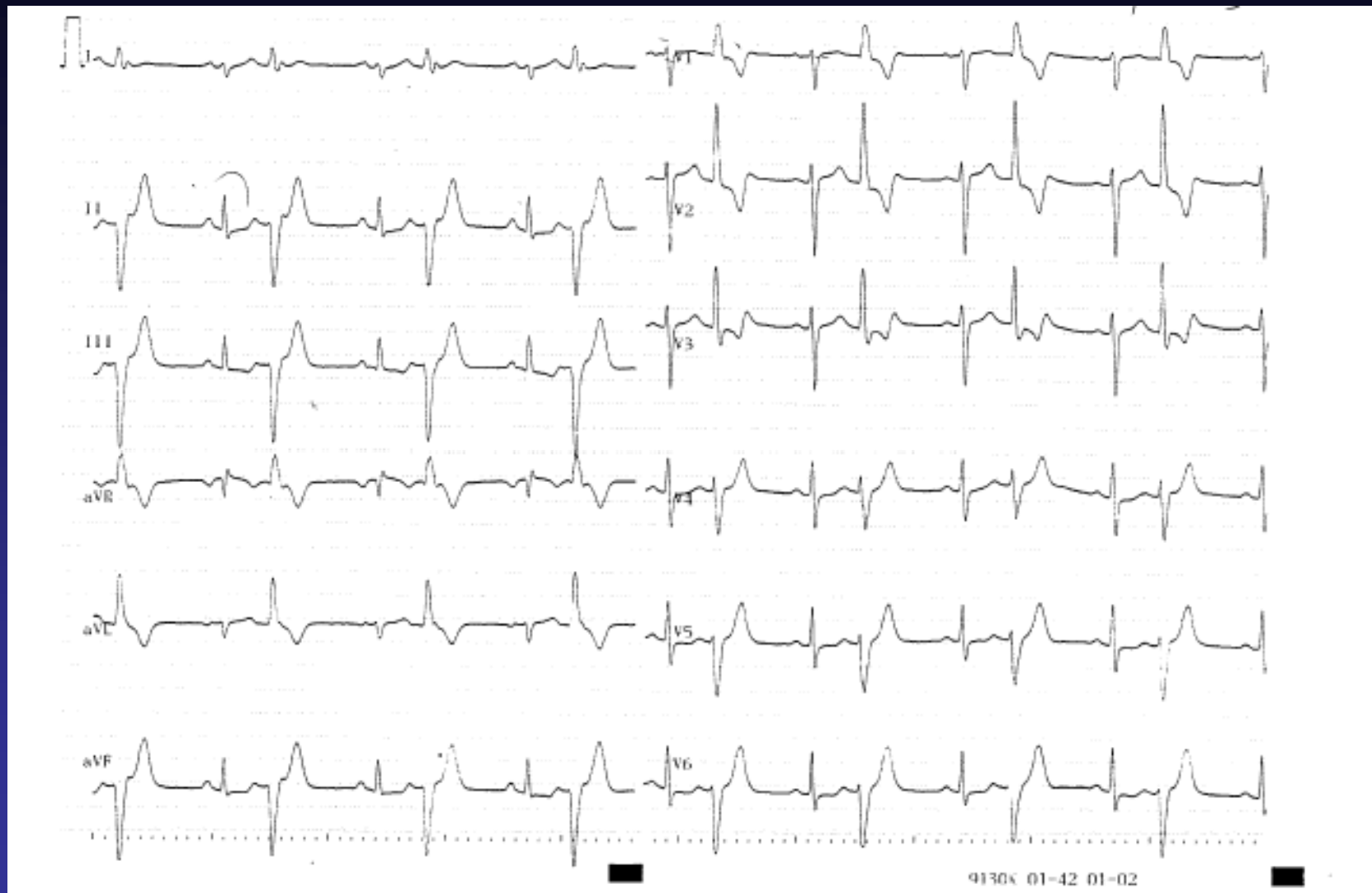
- 15 y/o F with normal past medical history.
- Competitive basketball player.
- Found to have multiple VPCs during a normal checkup.
- Asymptomatic.
- Failed betablockers (Normiten) Tx.



- PE: within NL.
- ECG: bigeminy rhythm, VPCs- *RB NW morphology*.
- Echo- No structural or vavlvular abnormalities.
- Holter: VPCs: 35% mostly unifocal.
- ET- no increase in number of VPCs.



12 lead ECG:



1. What is the diagnosis?

2. What should be done?



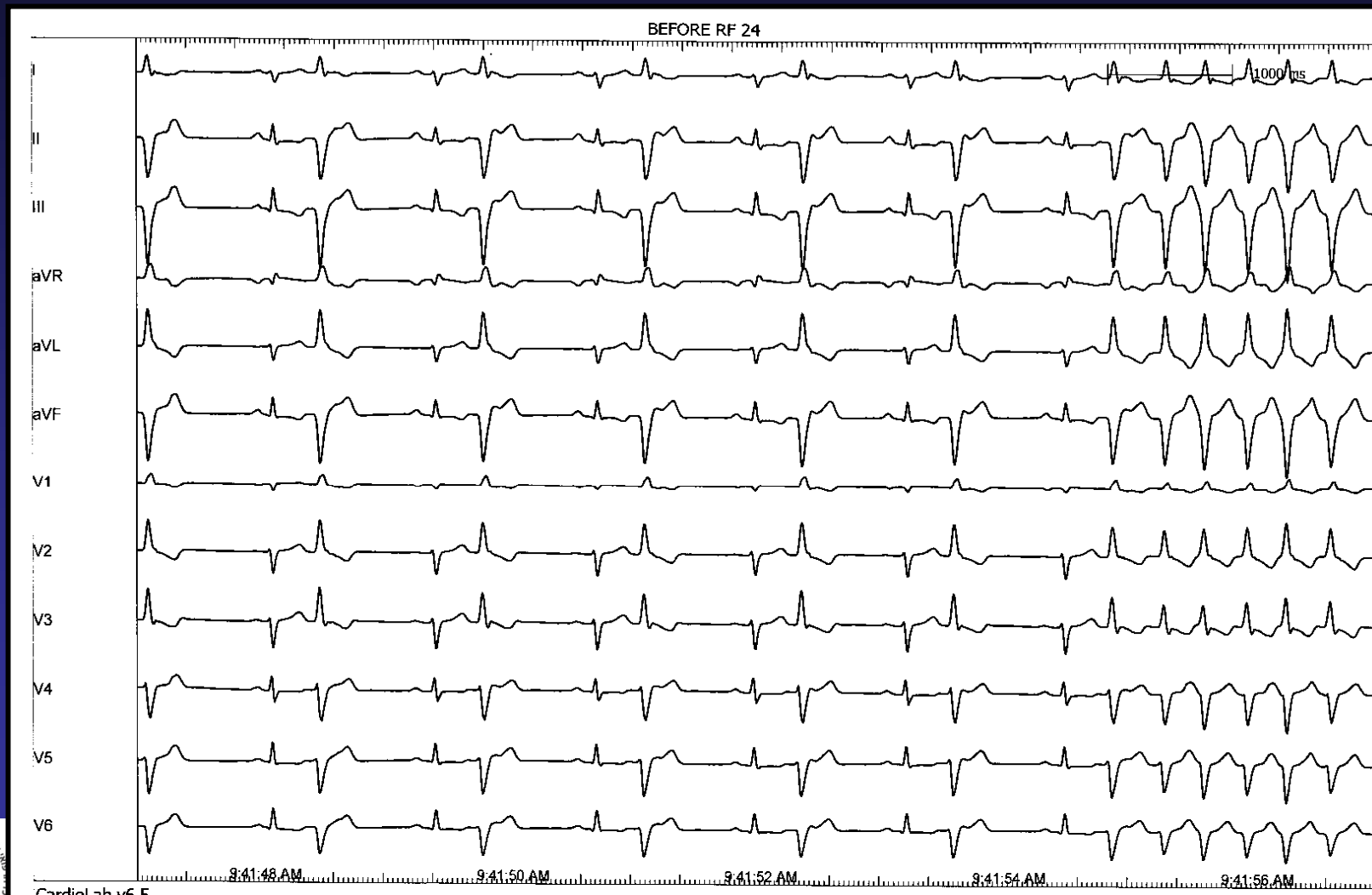
- DD:
 1. Fascicular (Belhassen) VT
 2. Papillary muscle VT
 3. Other focal? CMP?

- Tx options:
 1. Nothing.
 2. BB/ CCB
 3. AAD
 4. Ablation

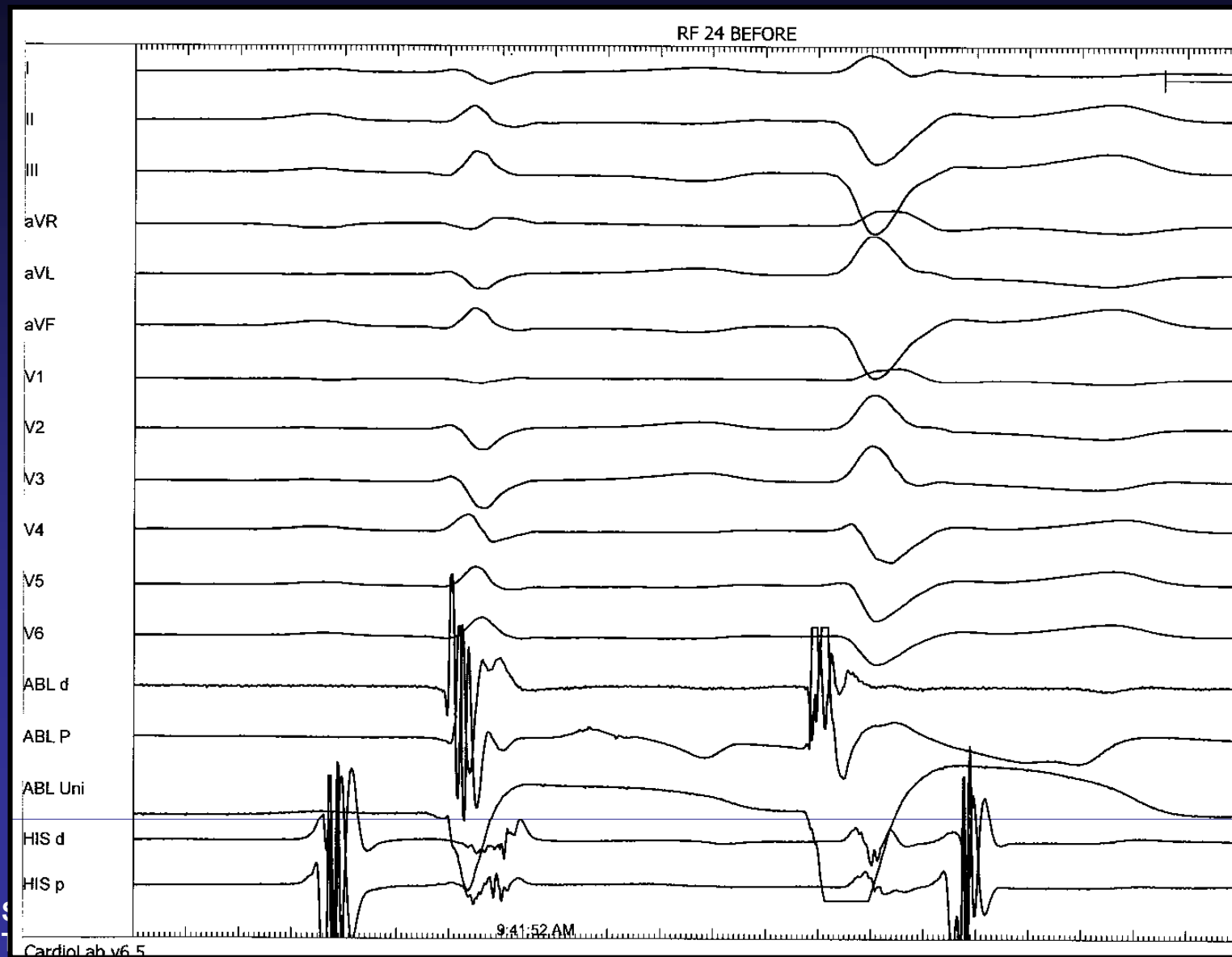


Referred to another center for ablation of Fascicular VT:

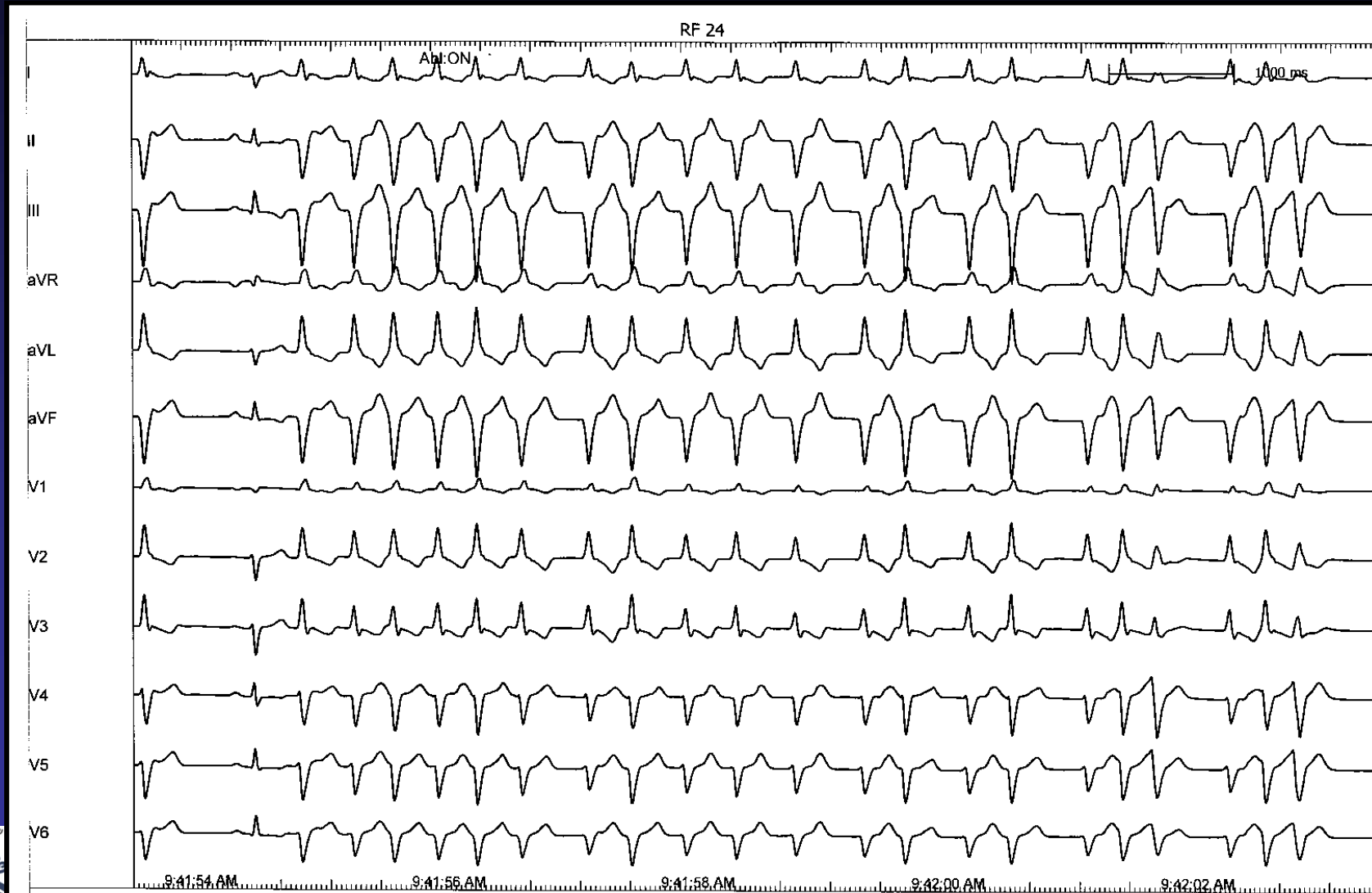
Baseline:



Catheter ablation located on Lt. post fascicle preceding PVC by 20 msec and
Identical 12/12 pace map- of note- *no recording of a fascicular potential
preceding the VPC at that site:*



Ablation on (standard EPT 4 mm catheter): acceleration of VPCs.
During the RF pulse the VT including the VPC's disappeared during a 2-min period (no such arrhythmia-free period was yet observed in this patient).



- Ablation failed and she was started on CCB (Verapamil 40mg*3/d).
- Holter on CCB: 450 VPCs/ day.
- Banned from any competitive sports activity.
- The pt was referred to our center .

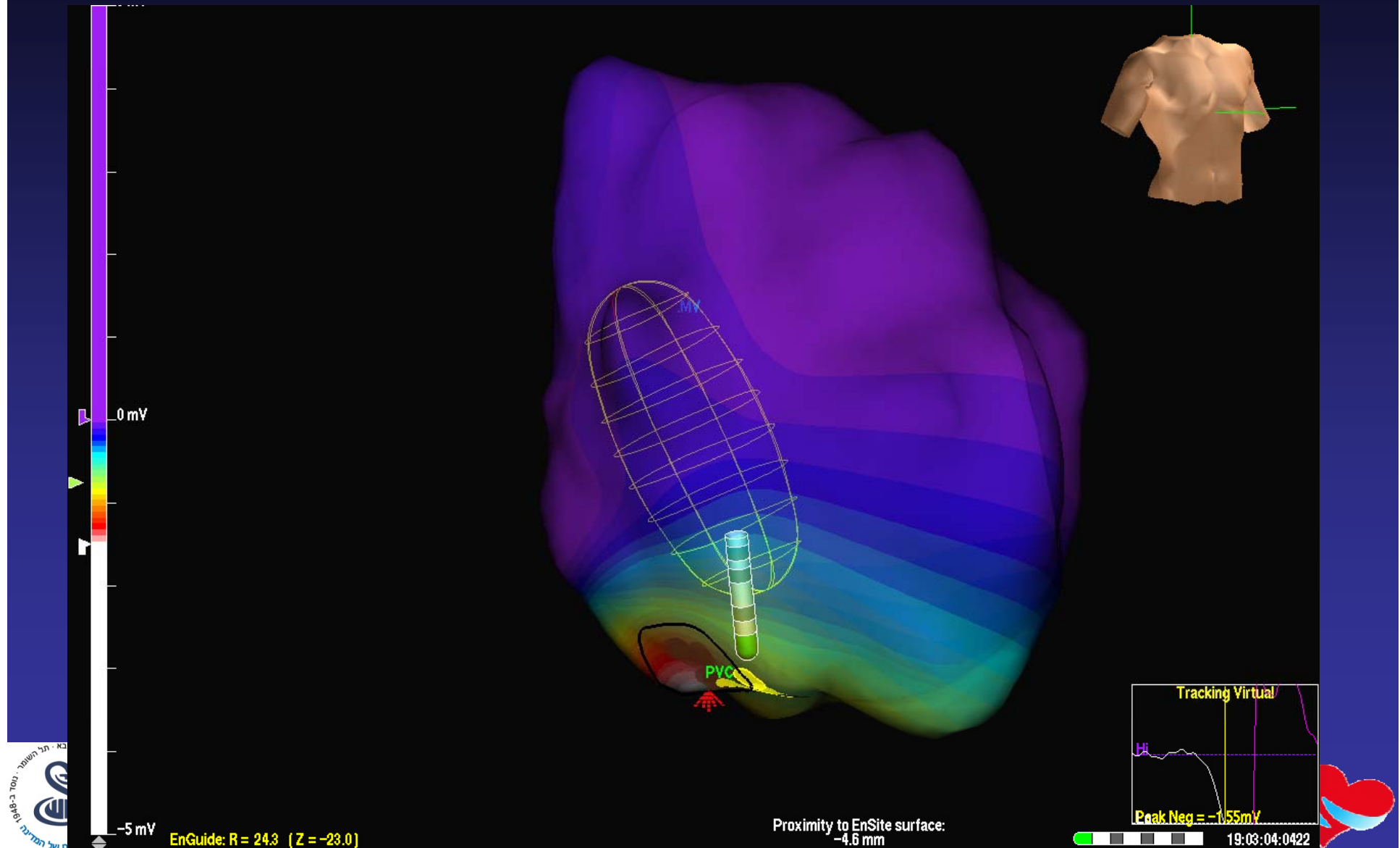


- Verapamil dosage increased to 240 mg/ day.
- Holter: 3% VPCs;
- ET: No VPCs at baseline but bigeminy rhythm at maximal HR.
- Cardiac MRI: very mild decrease in LVEF (52%); LVEDV: 152 ml ; LVESV 73 ml (mildly increased).
- 1st attempted ablation aborted > no spontaneous VPCs after insertion of catheters.
- A few weeks later referred for another attempt.



Ablation using ESI mapping system:

Earliest origin of VPCV located at inf basal LV:



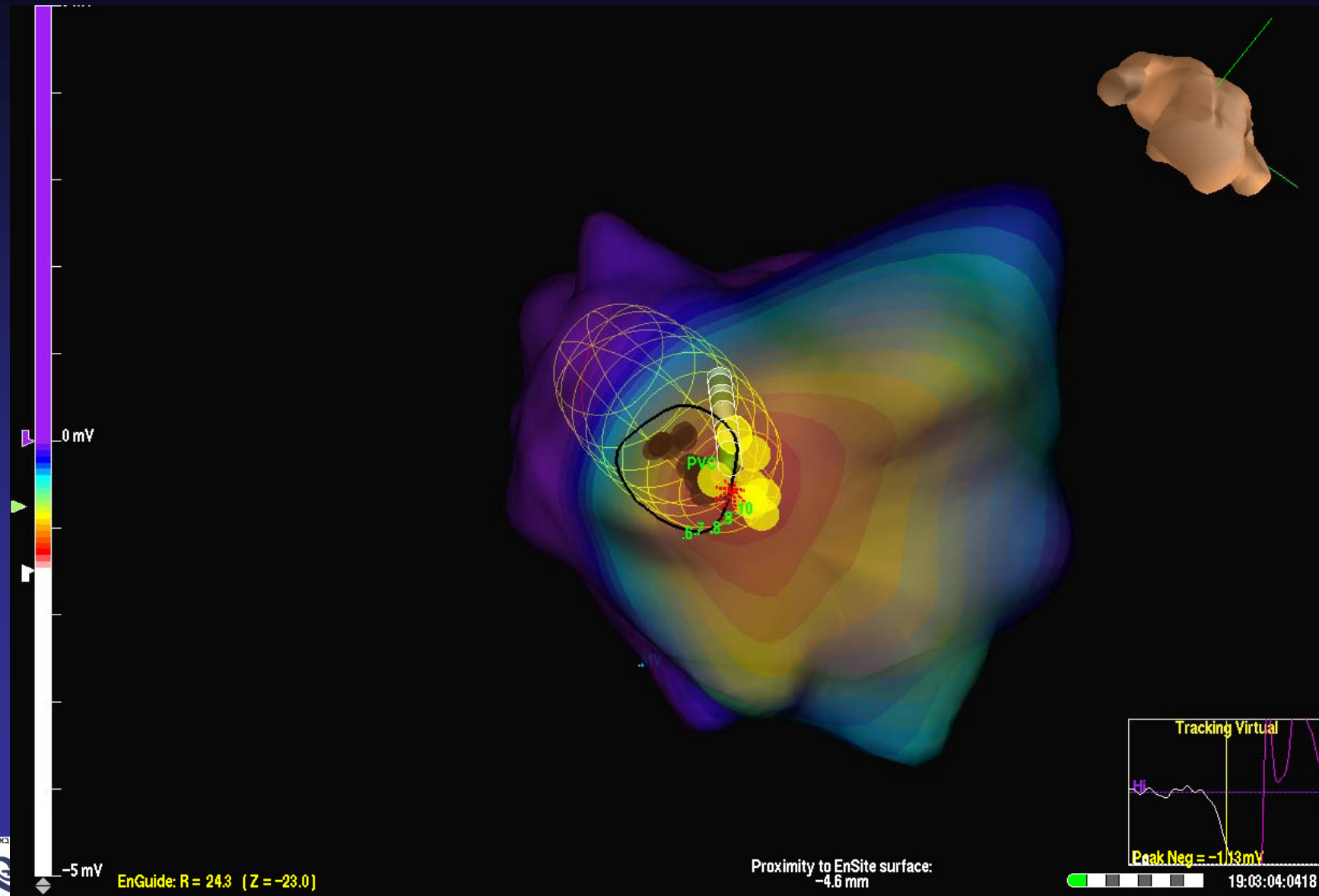
At this site a perfect pace map was observed
(no pre-potential was observed at this site):



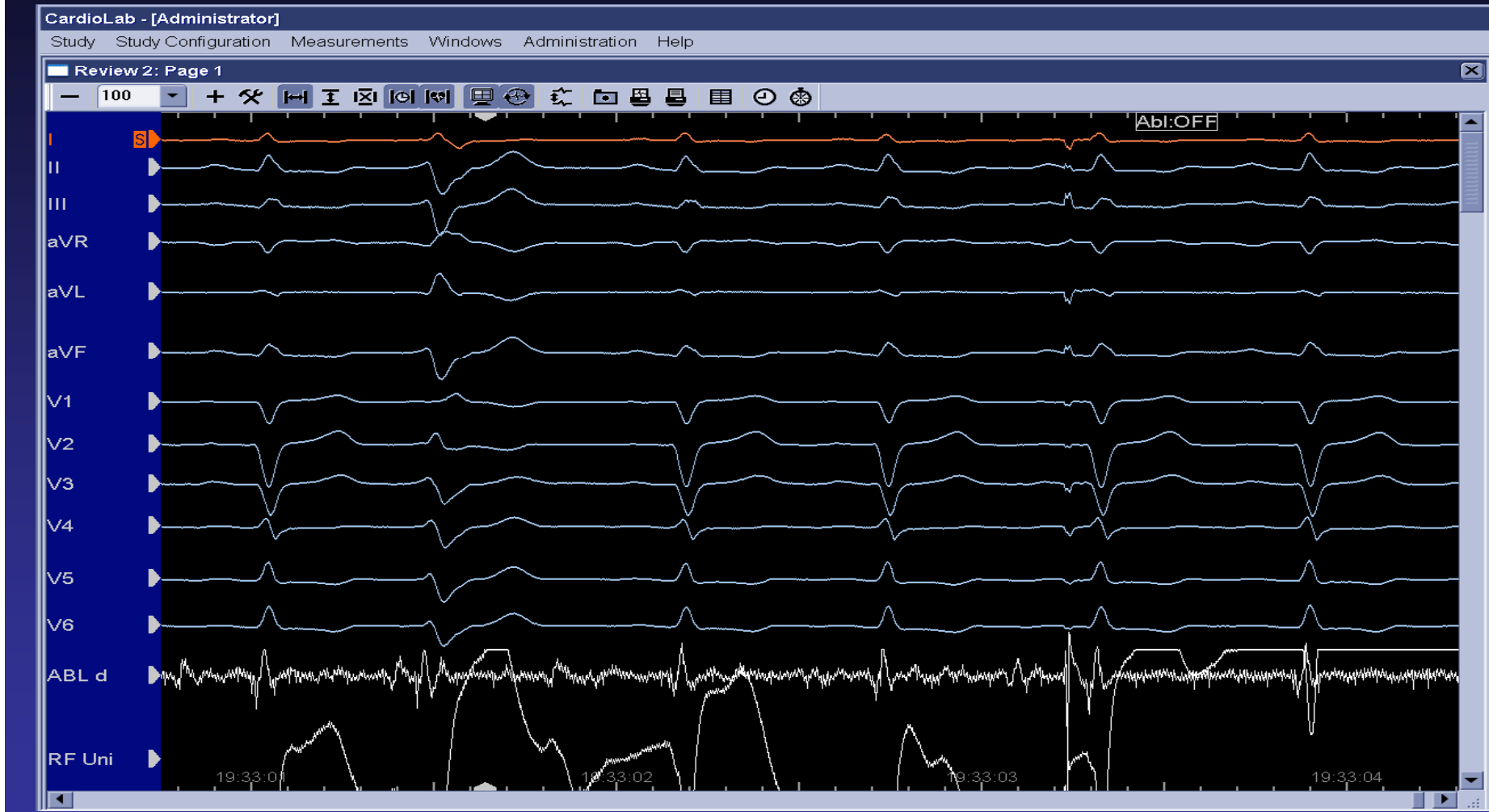
Ablation ON (irrigated catheter):



Catheter ablation at earliest site by ESI activation map:



Ablation OFF



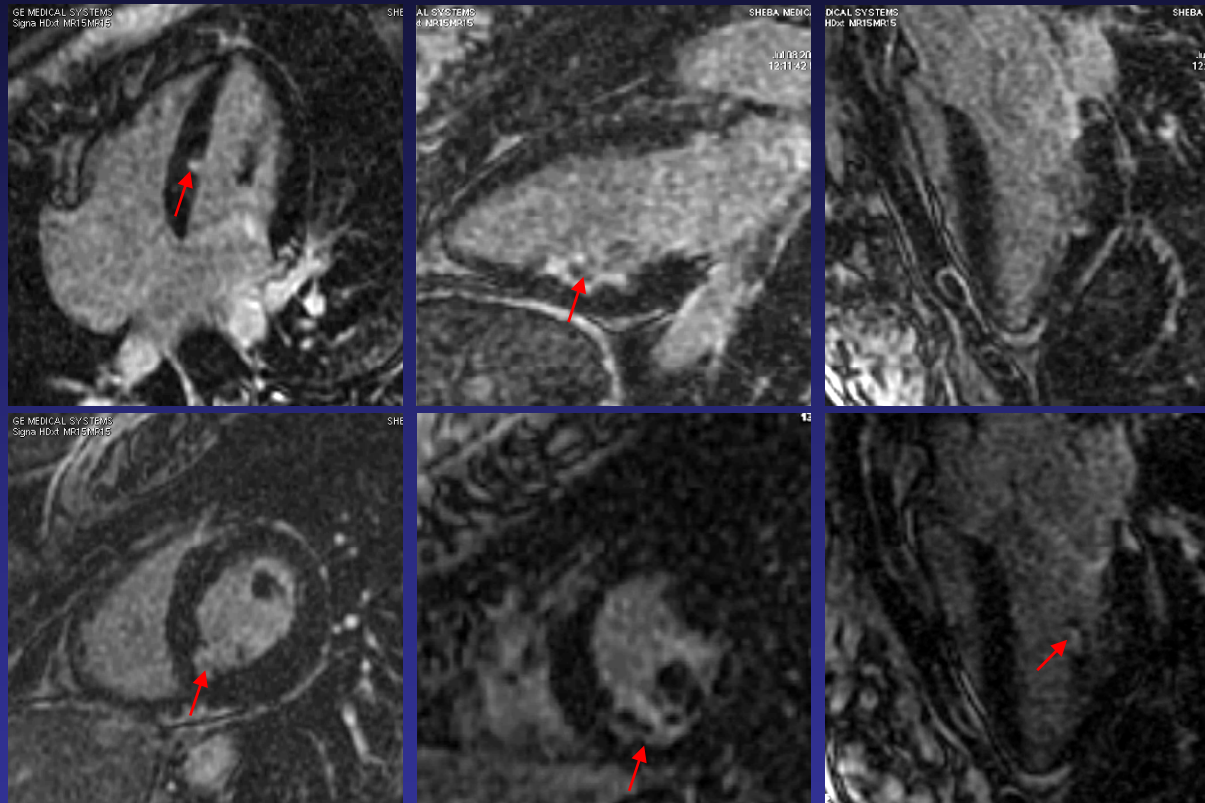
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Post ablation: VPCs not inducible.



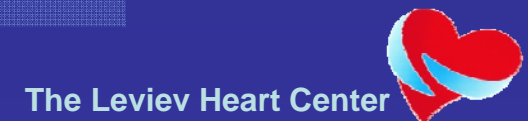
MRI: delayed enhancement at site of ablation located on LV posterior papillary muscle.



Final Diagnosis: Papillary Muscle VT!

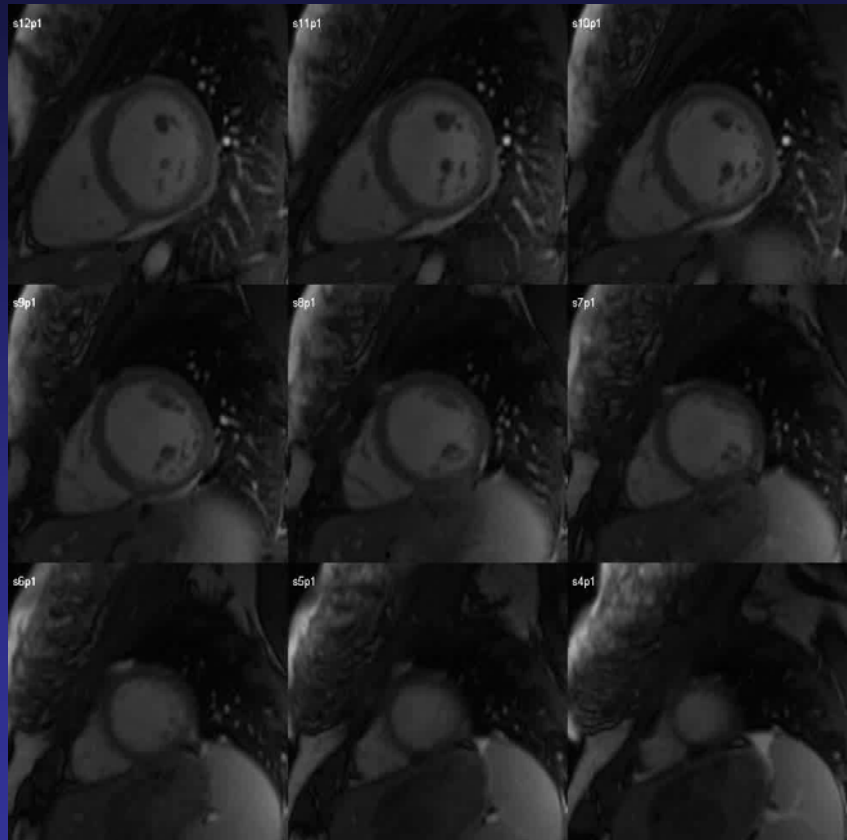


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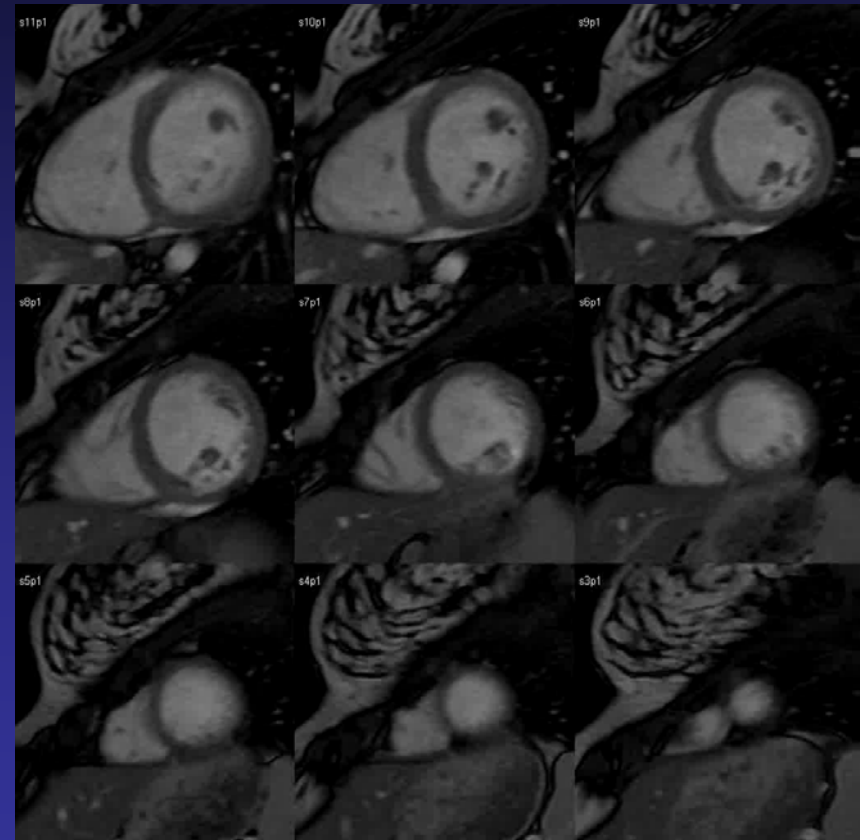


However on retrospect MRI demonstrated Lt. post papillary delayed enhancement also prior ablation.

Before ablation

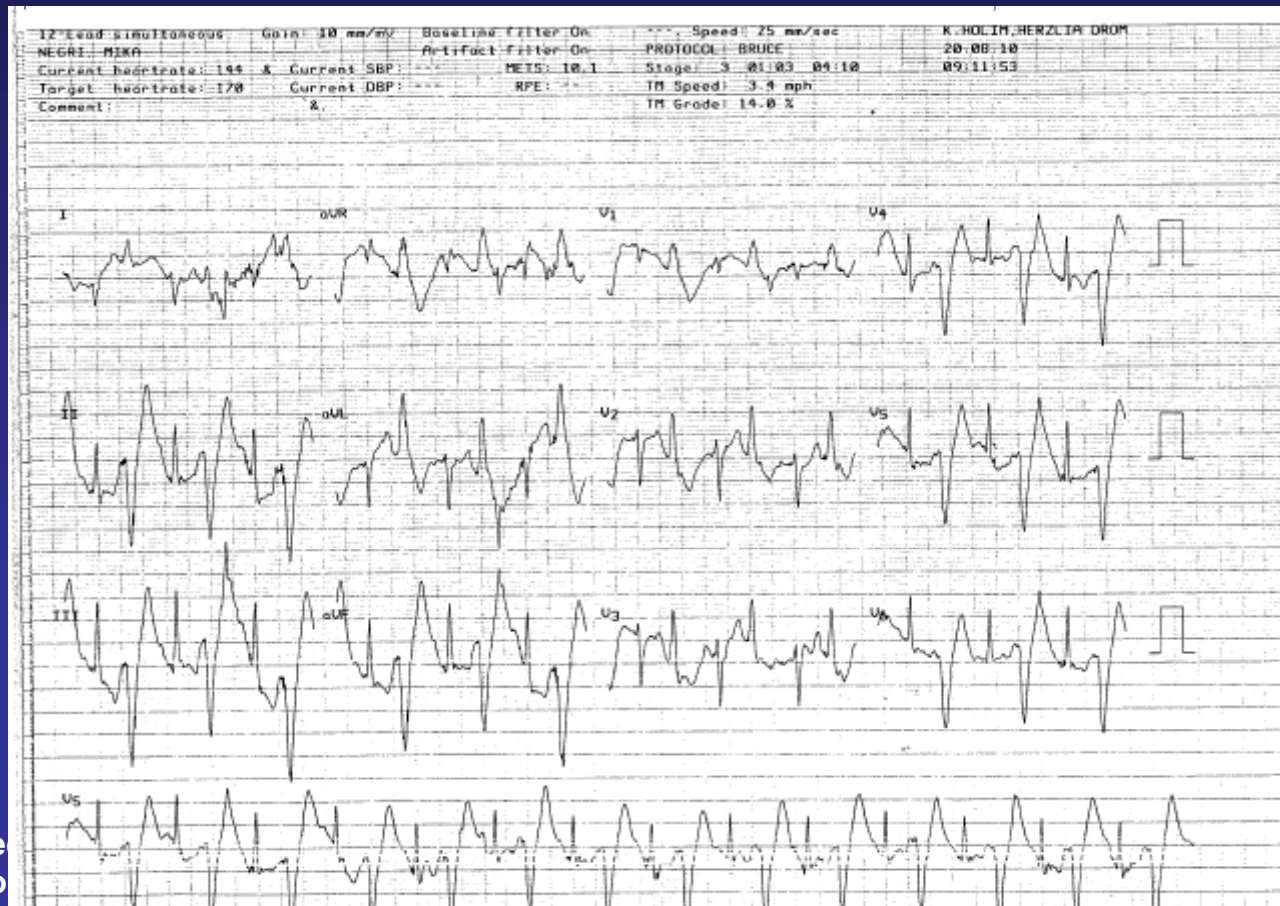


After ablation



Follow up:

- A few weeks later: No VPCs on Holter or ET.
- But another month later she performed another ET demonstrating...



Discussion:

- Final diagnosis: Papillary muscle (PAP) VT.
- Described as a distinct clinical entity by Doppalapudi et al. (Circ Arrhythmia Electrophysiol. 2008;1:23-29.):
 1. All had normal LVEF.
 2. More frequently located in the left posterior than left anterior papillary muscle.
 3. Non of them experienced syncope or SCD.



4. PAP arrhythmias were not inducible by programmed atrial or ventricular stimulation.
 5. Sustained VT, if inducible, was provoked by isoproterenol or burst pacing, suggesting that the underlying mechanism is triggered activity.
- Papillary VPCs vs. Fasc. VPCs (Good et al. Heart Rhythm 2008;5:1530 –1537) :
 1. Might have similar surface ECG (specifically post.medial papillary muscle). However:
 - a. QRS tended to be broader in PAP compared to Fasc. VPCs.
 - b. All of the fascicular had an rsR' morphology pattern in lead V1;this pattern was not present in PAPs group, in which a monophasic R and qR pattern predominated.



2. 2/7 pts with PAPs showed focal, delayed enhancement on MRI compared to non of the fasc. VT pt group.

3. EPS: Presystolic Purkinje potentials were identified at all effective ablation sites for fascicular arrhythmias, but in arrhythmias originating from PAPs, no or more distal Purkinje potentials were recorded.



- Activation map is the most reliable method.
- Ablation at site with excellent pace maps failed to terminate the tachycardia (albeit change in QRS morphology..)
- Several further RFA were usually.
- 80% RFA at both sides of the PAP were required.
- *The above suggests that the origin is located in the subendocardial or deep regions of the PAM (Yamada et al. (Circ A+E Aug.2010).*



Conclusion:

1. Papillary muscle VT is a new clinical entity with distinct electrocardiographic and electrophysiologic features.
2. Advanced mapping tools such as ESI/ Carto or ICE are very helpful.
3. Several RFA are usually needed in order to eliminate the arrhythmia.
4. Some pts exhibit focal areas of delayed enhancement suggesting some degree of predisposal jeopardized myocardium.

