PO-642-01

ESOPHAGO-PERICARDIAL FISTULA AS A RARE AND LIFE-THREATENING COMPLICATION OF EPICARDIAL VENTRICULAR TACHYCARDIA ABLATION

Justin Edward MD, MS; Shu Cheong Chang MD; Edward Gill MD; Matthew M. Zipse MD; Michael A. Rosenberg MD; Alexis Z. Tumolo MD; James Arthur Mann MD; Syed Rafay Ali Sabzvari MBBS, MD; Lukasz Cerbin MD; Christopher Barrett; Amneet Sandhu MD; Paul D. Varosy MD, FhRS; Jason West; Lohit Garg MBBS, MD; Wendy S. Tzou MD, FhRS and Ryan G. Aleong MD, FhRS

Background: Esophago-pericardial fistula is a rare but potentially devastating complication of epicardial ventricular tachycardia ablation. This case highlights the importance of recognition of this potential risk, as well as timely treatment.

Objective: To highlight the potential rare complication of fistula formation that can result from epicardial ventricular tachycardia ablation.

Methods: N/A

Results: A 59-year-old man presented with 10 days of pleuritic left chest discomfort two weeks following ablation for ventricular tachycardia during which epicardial ablation at the inferior basal left ventricle was performed without acute procedural complication. His symptoms worsened despite treatment with colchicine, and he subsequently developed elevated white blood cell count (14.1 x 10⁹/L) and anuric renal failure (serum creatinine 3.4 mg/dL). Echocardiogram revealed a moderate, circumferential pericardial effusion with pneumopericardium (Figure A). Chest CT showed pneumopericardium and evidence of esophageal fistula (Figure B). On pericardiocentesis, purulent fluid was drained. The patient underwent surgical repair through a left thoracotomy approach and was found to have a quarter-sized fistula connecting the esophagus and the pericardium that was repaired using an intercostal muscle flap. The patient recovered postoperatively and passed a swallow evaluation without dysphagia over 12 months of follow up.

Conclusion: This case demonstrated a rare complication of epicardial ventricular tachycardia ablation, which resulted in the formation of an esophago-pericardial fistula. The epicardial aspect of the basal-posterior left ventricle is in close proximity to the esophagus, and this risk must be considered when ablation is performed in this region. Monitoring of esophageal temperature in such instances may help to reduce the risk of this rare complication.

Figure A: Chest CT coronal view showing pneumopericardium

Figure B: Chest CT axial view showing esophageal fistula

PO-642-02

WIDE AND NARROW COMPLEX TACHYCARDIA AFTER TRANSCATHETER AORTIC VALVE REPLACEMENT

Luke Chong MD; Matthew Kalscheur MD and Daniel S. Modaff MD

Background: High grade AV block is a known complication after transcatheter aortic valve replacement (TAVR). The incidence of tachyarrhythmias following TAVR is not as well documented.

Objective: We performed an electrophysiology study (EPS) in a patient with symptomatic tachycardia after TAVR.

Methods: N/A

Results: An 88-year-old man who recently underwent TAVR presented with two days of palpitations. ECG revealed a wide-complex tachycardia which spontaneously converted. Later, tachycardia recurred with ECG showing a narrow-complex tachycardia that terminated with vagal maneuvers. The patient continued to have frequent episodes of tachycardia during admission. History was notable for occasional palpitations to a far lesser extent over the last eight years. On EPS, a narrow complex tachycardia was readily induced by atrial pacing with extra-stimuli. Spontaneous oscillation between narrow and wide complex with left bundle morphology and 1:1 AV relationship were seen, with no change in cycle length. Features indicating this tachycardia was consistent with AV nodal re-entry tachycardia (AVNRT) included: septal VA time < 70 ms with a concentric pattern of atrial activation; ventricular overdrive pacing advancing the atrial electrogram after the transition zone; VAHV response after cessation of ventricular overdrive pacing. Ablation of the rightward inferior extension of the slow pathway was performed. Following ablation, no tachycardia was inducible. The patient was discharged with an event monitor. He had no recurrent symptoms, and no arrhythmias were detected on his monitor.

Conclusion: We postulate that the TAVR valve impingement on AV nodal extensions provoked changes in slow and fast pathway refractoriness resulting in incessant AVNRT.

Figure 1: Electrophysiology study showing wide-complex tachycardia

Figure 2: Electrogram showing transition zone

Figure 3: Ventricular overdrive pacing advancing the atrial electrogram after the transition zone

PO-642-03

SILENT PROGRESSION OF CALCIFIC AORTIC STENOSIS DETECTED BY ARTIFICIAL INTELLIGENCE ELECTROCARDIOGRAM: A CASE REPORT

David Harmon MD; Awais Malik MD; Michal Shelly BSc; Zachi Itzhak Attia MSEE, PhD; Jae K. Oh MD; Saki Ito MD; Paul A. Friedman MD, FhRS and Rick A. Nishimura MD

Background: We have previously developed an algorithm to detect moderate to severe aortic stenosis by application of
artificial intelligence (AI) to a standard 12 lead ECG using a convolutional neural network.

**Objective:** This exemplary case demonstrates the potential use of the algorithm to identify aortic stenosis one decade before clinical detection.

**Methods:** N/a

**Results:****Case Description:** An 81-year-old man with a history of hypertension and a long-standing heart murmur presented to the hospital with one month of worsening dyspnea on exertion and a recent syncopal event. He had been previously in satisfactory health.

Physical exam demonstrated significant carotid upstroke delay, loud (3/6) late-peaking systolic ejection murmur at the right upper sternal boarder which extended into the second heart sound with radiation to the carotid arteries. Lungs had rales at the bases bilaterally.

An ECG showed sinus tachycardia with a first-degree AV block, left anterior fascicular block, lateral T-wave inversions with lateral ST-depressions, meeting criteria for left ventricular hypertrophy (Figure 1). Compared to prior ECGs, worsening ST-T-wave abnormalities and left ventricular hypertrophy were noted (Figure 1). Review of prior ECGs with the AI ECG algorithm for aortic stenosis revealed an increased probability of clinically relevant valve disease since 2010 with increasing likelihood of disease each subsequent ECG (Figure 2).

Echocardiography identified critical, calcific aortic valve stenosis with a mean systolic gradient of 78 mmHg and valve area of 0.56 cm² with a left ventricular ejection fraction of 51% (Figure 2F). NT-proBNP was 7086 pg/mL. Moderate coronary disease identified on angiography. The patient underwent transcatheter aortic valve replacement and PCI with good outcome.

**Conclusion:** This case demonstrates the potential of the AI ECG to detect clinically relevant valve disease years before clinical presentation. Though this patient had regular contact with the healthcare system, his historical murmur did not trigger advanced imaging. Though multiple ECGs were obtained over years, all changes were assumed to be benign and minimal (Figure 1). Use of the AI ECG algorithm may have expedited identification of this valve disease nearly a decade prior to his critical presentation (Figure 2).

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**PO-642-04**

HALF-NORMAL SALINE IRRIGATION AS A METHOD FOR PHRENIC NERVE DISPLACEMENT DURING EPICARDIAL ABLATION OF VENTRICULAR TACHYCARDIA

Jeffrey Lin MD; Paul J. Wang MD, FHRS and Duy Thai Nguyen MA, MD, FHRS

**Background:** Phrenic nerve displacement can be necessary during epicardial catheter ablations. High impedance irrigants can be used to direct radiofrequency (RF) energy toward myocardium.

**Objective:** We present the novel use of half-normal saline (HNS) within the pericardial space to displace the phrenic nerve during ventricular tachycardia (VT) ablation.

**Methods:** N/A

**Results:** A 39 year-old male with non-ischemic cardiomyopathy and idiopathic VT previously underwent an endocardial VT ablation five years ago targeting the left ventricular (LV) anterolateral papillary muscle, and again one year ago targeting a similar VT felt to be from the anterolateral LV. A cardiac MRI showed thinning and akinesis of the basal to mid anterolateral, lateral, and inferolateral segments, with delayed gadolinium enhancement. A mid-myocardial stripe was present. LV ejection fraction was 35%. An implantable cardioverter-defibrillator was implanted, after which he experienced a shock for VT. He was started on sotalol and referred to our institution for repeat ablation. Under general anesthesia, anterior epicardial access and vascular access were obtained. An endocardial LV voltage map showed no scar. Epicardial voltage mapping showed anterolateral and inferolateral scar. A dense cluster of late potentials (LPs) was present within a deceleration zone identified by isochronal late activation mapping. VT was induced and the cluster of LPs became mid-diastolic. Attempts to entrain were unsuccessful. Substrate homogenization was performed. However, additional LPs persisted directly under the course of the phrenic nerve identified by pace mapping, and VT remained inducible. HNS was infused to create a pericardial effusion and displace the phrenic nerve; high output pacing was performed until loss of phrenic nerve capture, at which point ablation was performed to abolish the remaining LPs. The pericardial effusion was drained and phrenic capture re-confirmed. VT was thereafter non-inducible on isoproterenol, and the patient has remained arrhythmia-free.

**Conclusion:** Intra-pericardial fluid infusion can be used to displace the phrenic nerve. Low ionic irrigants can preferentially direct RF to myocardium. To our knowledge, this is the first reported case of HNS use for epicardial phrenic nerve displacement.