Relationship Between VAD Implantation and Ventricular Tachycardia

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Ventricular Tachycardia

Ventricular Tachycardia is an abnormal heart rhythm - cardiac arrhythmia - in which the ventricles beat faster than they should. It is caused by scar tissue in and around the ventricles. This scar tissue can be a result of lack of blood flow through the coronary arteries (caused by blockages), acute injury to the heart muscle such as a heart attack, or from intrinsic myocardial disease; all of which lead to a weak heart condition known as cardiomyopathy. Mild arrhythmias oftentimes can be treated medically by antiarrhythmic medications like amiodarone. More severe or persistent Ventricular require a more invasive intervention known as an ablation procedure which utilizes catheter directed heat energy to remove the arrythmia's focus.

Left Ventricular Assist Device (LVAD)

LVAD is a device that acts as a replacement for the nonfunctioning left ventricle in patients with cardiomyopathy. The implantation procedure is extremely invasive as its an open heart procedure. A hole is cut for the inflow cannula to be placed in the left ventricle. Another hole is made in the aorta and the outflow tube is placed here. The LVAD bypasses the aortic valve in pumping blood from the LV through the aorta to the body since depressed left ventricle function as a result of cardiomyopathy leaves it unable to do so.

LVAD is usually used as a bridge to transplant in patients with extreme heart failure (NYHA Class IV). Many patients who receive LVAD go on to develop severe ventricular arrythmias. The main cause of this is thought to be because of the scar around the apex of the heart caused by the implantation of the device.

Our research is seeking to prove that the main cause is preexisting arrythmias that just get exacerbated after the procedure and that the best treatment for this is ablation two weeks after implantation.

Experiment Protocol

- This is a retrospective clinical study. All of the patients have already received the procedures prior to the start of the study.
- All of the data is obtained through the Electronic Medical Records systems at Memorial Hermann Med Center.
- For each patient in the study 14 points of data are recorded ranging from prescribed medicines to kidney function.
- So far only 7 patients have been found for which enough data is present to use in the study.
- The purpose of this study is to determine whether ablation following LVAD implantation helps prevent incessant post-procedural VT and to determine the cause of the VT that follows LVAD implantation for majority of LVAD recipients.
- Despite the invasive nature of LVAD implantation, we've found that most patients are healthy enough two weeks after the procedure to undergo a less invasive ablation procedure.

<u>VT Procedure</u>

- Patients are sedated and access to the heart is gained through the groin.
- A catheter is placed which travels through the inferior vena cava to the right atrium. It is then placed through the tricuspid valve or passes transeptally to the left atrium and then through the mitral value into the left ventricle depending on which ventricle the arrythmia is coming from.
- The scar tissue is ablated around to prevent these extraneous signals from causing premature ventricle contractions.

Results

This project is currently underway and so far, the sample size is too small for any conclusive results to be found. Results will be finalized in 2022 and published in the HRS journal.

Conclusion and Recommendations

If our hypothesis that VT ablation can prevent persistent VT in LVAD recipients is true then it will become common practice to ablate on this specialized group of patients.

A study done by physicians at the University of Virginia shows that concomitant cryoablation (ablation done using extreme cold rather than heat) at the time of LVAD implantation has excellent results for preventing persistent post-procedural VT. This was also a limited study as only 7 patients in the study received both procedures concurrently. If this study was reproduced with a larger sample size, it could prove that this is the best method to prevent ventricular arrythmias in LVAD recipients.

Our current research is to conduct a study with a larger sample size to prove the hypothesis that ablation following LVAD implant is an effective method for prevention of the development of ventricular arrythmias in LVAD recipients.

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Age	Etiology	ICD date/type	VAD Date	VT occurance (post VAD)	Pre VaD VT	VT Characteristics	Ablation Date	Access	Scar Location	Outcome	Meds (most recent follow up)	VT survival	LVAD
59	Nonischemic	BiV	2/25/21	Day 18 & month 7, 9/26/21	NA	Slow relatively stable sustained monomorphic, 60 episods, rare Polymorphic	3/17/21, 10/27/21	transseptal	apical septal LV and around inflow canula	Non inducible/inducible poly	Mexiletine	VT free till 2/15/2022	HeartWare
71	Combined	Dual	6/29/16	Day 11	9/26/13	ICD shock, hemodynamically stable monomorphic, 5 morpholgies, 310-390 msec	10/24/16, PVC 10/18/19, 10/27/20	Retrograde	anterior septum and LV apex peri-LVAD cannula scar/Posterolateral scar-LVAD	Non inducible/inducible poly	Mexiletine & Sotalol	VT free till 12/1/21	HMII
59	Nonischemic	BiV	Feb-19	18 months	NA	Monomorphic 360msec, RBB, unstable	10/14/20	retrograde	posterior/posterolateral scar	Non inducible/inducible poly	Amiodarone	VT free till 2/11/22	HMIII
73 D	Nonischemic	BiV	7/6/16	50 months	NA	Polymorphic at 260 msec, unstable	9/11/20	retrograde	mid anteroseptal LV	scar modification/inducible poly	Amiodarone	VT free from 9/11/2020 to death due to GI bleeding and shock in Dec-2020	HMII
60 D (deceased on 8-6-21)	Nonischemic	Dual	May-17	48 months	NA	Monomorphic RBB, 450 msec & 620 msec	5/3/2021, 5/26/2021	retrograde	basal anterior anterolateral and later during second ablation	VT1 ablated endocardially, VT 2 remained inducible	Amiodarone, mexiletine	Died at 8/6/21 due to multiple VT episodes & shocks leading to RV failure	HMII
64 D (deceased Feb 2022)	nonischemic	Dual	9/7/18	Epicardial VT ablation at the time of chest closure next day post VAD and endocardial portion was ablated on 9/14/2018	NA but VT prior to VAD	5 monomorphic VTs CL 330-440 msec stable +VF, several ICD shocks prior to VAD hospitalization. Did not have VT/VF after ablation	9/8/2018 epi, 9/14/2018 endo	retrograde/Epicardial	mid lateral and inferolateral scar	Scar modification/inducible	Amiodarone	VT free till death in Feb 2022	HeartWare
60	Nonischemic	Dual	Dec-21	Day 13	NA	Stable monomorphic at 150 bpm, VT-2 Polymorphic	1/28/22	Retrograde	BBVT	Non inducible/inducible poly	Mexiletine	No VT (34 days)	HMIII