Reviews:


Aortic insufficiency (AI) is a common, well known occurrence in LVAD supported patients. The effect of AI on clinical outcomes in these patients is less well known. With this in mind, Dr. Cowger and her colleagues performed this study to characterize the development and progression of AI during VAD support, define the burden of reoperation for AI, and evaluate the effects of AI on the right ventricle (RV), mitral regurgitation (MR), hemolysis, and survival.

In this study, the authors retrospectively evaluated aortic insufficiency by echocardiogram before VAD insertion and at routine intervals thereafter for all patients (n=166) implanted with a HeartMate II LVAD at the University of Michigan between 2000 and 2011. They found mild or less AI in 156 patients (94%) prior to VAD insertion, and moderate to severe in only 1 (0.6%). During 291.4 person-years of follow-up, moderate or worse AI developed in 36 patients (0.17 events per patient year (PPY)) at a median 273 days after implant, moderate to severe or worse AI developed in 11 (0.039 PPY) at 564 days, and severe AI or worse developed in 2 (0.0069 PPY) at 414 days. At 2 years post-implant, moderate or higher AI was present in 33% of patients. Adjusted analysis demonstrated smaller BSA and age at VAD implant were associated with worse AI at follow-up. When factoring in duration of support, the group also found closed AV valve during systole, smaller LVdD, and higher LVAD rpm to be post-op predictors of AI. AI was not found to be associated with worsening MR, RV function, or pump thrombosis. Finally, the
authors found no difference in overall survival in patients with mild-moderate or worse AI or those with moderate or worse AI compared with patients with less severe degrees of AI.

In this large, single-center, retrospective study, Dr. Cowger and colleagues detail the course of AI and related outcomes in 166 patients after implantation of a HeartMate II LVAD. In doing so, the authors report a prevalence of 33% of at least moderate AI at two years of support and also found no association between the presence of moderate or worse AI and worsening MR, RV function, pump thrombosis, or survival. These findings stand in contrast to previously published studies that have reported worse survival and HF symptoms in those with AI. As noted by the authors, confirming these findings in other cohorts may allow clinicians to change from device management strategies focusing on preserving AV opening to optimizing LV decompression.


Since it was first reported in the New England Journal of Medicine, the recent increase in the risk of VAD thrombosis in the US has generated much discussion and concern in the MCS community. While many potential explanations have been offered, the issue remains surrounded by uncertainty. While anticoagulation strategies, patient selection, and device issues have been hypothesized as possible culprits, little attention has been given to the accuracy of measuring anti-coagulation. Citing evidence that the anti-factor Xa (anti-Xa) assay may more accurately reflect the degree of anticoagulation from unfractionated heparin than the more commonly measured activated partial thromboplastin time (aPTT), Dr. McIlvennan and colleagues at the University of Colorado hypothesized that the aPTT may not adequately reflect the level of anti-coagulation with UFH in LVAD patients. With this concern, they performed a prospective, single-center quality improvement project to compare the relationship between aPTT levels and anti-Xa levels between a group of patients with LVAD support and another group of heart failure patients admitted with acute decompensated heart failure (ADHF).

The group identified 19 LVAD patients and 10 ADHF patients receiving UFH (after device implantation, bridging for sub-therapeutic INR, or active thrombosis) and obtained simultaneous anti-Xa and aPTT levels after a minimum of 6 hours of UFH. Of the 19 LVAD patients, 10 had therapeutic aPTTs, but none of those 10 had a therapeutic anti-Xa level. 6 LVAD patients had a supra-therapeutic aPTT, with 2 of those having a therapeutic anti-Xa level and the remaining 4 being sub-therapeutic. In the ADHF group, 6 of the 10 patients had a therapeutic aPTT, with 4 of them also having a therapeutic anti-Xa level and 1 having a supra-therapeutic INR. The authors also plotted Anti-Xa activity and aPTT by group and compared the resulting line slope. Comparison between slopes found a statistically significant difference when removing a single outlier from the ADHF group. From this data, the authors suggest that aPTT overestimates the level of UFH anti-coagulation in LVAD patients compared with patients hospitalized for ADHF without LVADs.
In their Research Correspondence, Dr. McIlvennan and colleagues provide preliminary evidence that aPTT may overestimate the degree of anti-coagulation in LVAD supported patients. They posit a combination of the effect of aquired von Willebrand’s syndrome lowering Factor VIII levels and under-dosing UFH due to the effect of warfarin on aPTT levels as the most likely explanation for this finding. While based on single measurements from a small number of patients at a single center, confirmation of these results may substantially alter the way bridging anticoagualtion is monitored and the efficacy thereof. The findings merit further, in-depth study across numerous centers.

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European Heart Journal

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