What's New in MCS

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Best Papers

Dunlay SM, Deo SV, Park SJ. Impact of Tricuspid Valve Surgery at the Time of Left Ventricular Assist Device Insertion on Postoperative Outcomes. ASAIO J 2015; 61:15–20

The authors published the first meta-analysis to evaluate the impact of tricuspid valve surgery (TVS) performed at the time of LVAD implantation on early postoperative outcomes.

They found that addition of TVS prolonged cardiopulmonary bypass times by an average of 31 minutes. There was no difference in need for right ventricular assist device (six studies, HR 1.42, 95% CI 0.54–3.76), acute renal failure (four studies, HR 1.07, 95% CI 0.55–2.10), or early mortality (six studies, HR 1.28, 95% CI 0.78–2.08) in patients treated with LVAD + TVS versus LVAD alone.

However, the authors admit that that there are limited observational data available on this topic, and that there is insufficient information to draw definitive conclusions on the impact of TVS on early postoperative outcomes. As such, further data are needed in order to inform surgeons as to the best practice for these patients.

Since there is always the question to correct a moderate or higher grade tricuspid insufficiency during LVAD implantation, this article may help in the decision making, rather not to do TVS than to do.

Bartoli CR, Restle DJ, Zhang DM, Acker MA, Atluri P. Pathologic von Willebrand factor degradation with a left ventricular assist device occurs via two distinct mechanisms: Mechanical demolition and enzymatic cleavage. J Thorac Cardiovasc Surg 2015;149:281-9

The authors addressed the major role of von Willebrand factor for bleeding complications in patients with rotary LVADs. They investigated whether LVAD–related shear stress and ADAMTS-13, the von Willebrand factor protease, altered von Willebrand factor metabolism.

Their findings demonstrate for the first time that there is both a mechanical and a separate enzymatic mechanism of vWF degradation during mechanical circulatory support. Supraphysiologic shear stress itself caused moderate degradation of vWF multimers but did not generate vWF fragments. In the presence of supraphysiologic shear stress, ADAMTS-13 (the vWF protease) eliminated high-molecular-weight vWF multimers and generated vWF degradation fragments. As such, ADAMTS-13 is the major mechanism of vWF degradation during mechanical circulatory support. However, both mechanistic pathways likely contribute to bleeding episodes in patients with an LVAD.

The authors conclude that ADAMTS-13 may be a therapeutic target to reduce vWF degradation and predisposition to bleeding in patients with an LVAD. They suggest that ADAMTS-13 inhibitors should be investigated as a potential intervention to reduce pathologic degradation of vWF during mechanical circulatory support.

If one considers the importance of bleeding complications for the morbidity of LVAD patients, this article may have a remarkable impact for a reduction of such events in the future.

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None

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None