Bioprosthetic Pulmonary Valve Endocarditis: Incidence, Risk Factors, and Clinical Outcomes

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Background: Pulmonary valve replacement (PVR), with either a bioprosthetic valve or right-ventricle-to-pulmonary artery conduit, is often required for congenital heart disease (CHD) as part of pulmonary outflow tract reconstruction. There are a growing number of case reports about infective endocarditis (IE) after PVR, but the overall incidence and risk factors for IE after PVR are unknown. Furthermore, the utility of transthoracic (TTE) vs. transesophageal (TEE) echocardiogram in diagnosis of IE, and the clinical outcomes (including need for surgical intervention vs. antibiotics alone for treatment of IE in this setting) are also unknown. The aim of this study was to assess the incidence, risk factors, and clinical outcomes of IE after surgical PVR in patients with CHD.

Methods: We performed a retrospective analysis of all cases of PVR performed at Children's Hospital of Wisconsin between 1975 and 2016. Patients that died < 30 days after PVR were excluded. All cases of IE after PVR were identified and clinical and imaging data were obtained by review of medical records. Percutaneous PVRs (i.e. Melody valve) were excluded as duration of follow up is short.

Methods and results: Out of a total of 924 surgical PVRs, there were 19 (2.0%) cases of IE that occurred in 16 individual patients. The median age at diagnosis of IE was 21 years (range=1.2 yrs to 34 yrs) and the median follow-up interval from PVR to diagnosis of IE was 9.4 years (range=5 mo to 21 yrs). The overall freedom from IE was 99.1%, 96.9%, 93.4%, and 91% at 5, 10, 15, and 20 years, respectively, after PVR. There was no significant difference in freedom from IE in homograft vs. heterograft PVR (93.6% vs. 97.3% at 15 years, p=0.14), or in bovine vs. porcine heterograft PVR (96% vs. 99.1% at 15 years, p=0.26). PVR performed as a reoperation had significantly higher risk for IE compared to PVR performed during initial CHD repair (HR 3.6, 95% CI 1.3-9.8; p=0.01). Age at time of PVR, gender, and CHD diagnosis were not significant risk factors for IE in multivariable analysis. In the 19 cases of IE, the most common infective organisms were *Streptococcus viridans* in 5 (26%), *Staphylococcus aureus* in 4 (21%), and *Enterococcus faecalis* in 3 (16%). TTE alone identified a pulmonary valve vegetation in 7 (37%) cases, while TEE following a non-diagnostic TTE identified a vegetation in 4 (21%) additional cases. The remaining 8 were diagnosed with modified Duke criteria and/or positive blood cultures associated with change in prosthetic pulmonary valve function. Septic pulmonary emboli were identified in 4 (21%) cases. Ten (53%) cases of IE were treated with surgical PVR + 6 weeks of intravenous (IV) antibiotics, 8 (42%) were successfully treated with ≥ 6 weeks of IV antibiotics alone, and 1 (5%) case was treated with surgical debridement without PVR + 6 weeks of IV antibiotics. There were no deaths and no recurrences of IE after treatment.

Conclusion: The overall risk for IE after PVR is low. There was no association between age, CHD diagnosis, or type of pulmonary valve on the risk of IE. TEE does provide additional value in identifying prosthetic pulmonary valve vegetations, but overall echocardiography was diagnostic in only 58% of cases. Outcomes after either surgical or IV antibiotics alone are excellent with no mortality or recurrences of IE.