Why do some conduits get infected and others don't?

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Bovine jugular vein (BJV) grafts increasingly have been used for right ventricular outflow tract reconstruction. Stent-mounted BJV grafts now also are implanted widely as percutaneous valves in different positions.

Despite an initial enthusiasm with the use of BJV conduits, our group¹ and others²⁻⁴ have reported a high risk of late endocarditis. In our series,¹ which included 245 patients with BJV conduits, 6% of conduits developed endocarditis with a median follow-up of 7 years, and it was estimated that as many as 17% of patients would develop endocarditis 10 years after conduit placement. BJV grafts were associated with a 9-fold increased risk of endocarditis compared with homografts. Interestingly, the risk of endocarditis increased with time, and most of the cases happened after 7 years from implantation. Similar concerns have been raised for percutaneous BJV valves.⁵

The mechanism behind the high incidence of endocarditis in BJV grafts is unclear. Some of the theories include increased susceptibility of these conduits to bacterial adherence, increased thrombogenicity, turbulent flow, and late tissue degeneration.¹ In this issue of the *Journal*, Veloso and colleagues⁶ assessed the in vitro adherence of 3 bacterial strains (*Staphylococcus aureus*, *Staphylococcus epidermidis*, and *Streptococcus sanguis*) to small pieces of bovine pericardial patch, BJV, and cryopreserved homograft under both static and simulated shear stress conditions. In general, bacterial adherence was relatively similar for all tissues. The authors concluded that the surface composition of the conduits, bacterial surface proteins, and shear forces were not the most important determinants of the greater risk of endocarditis in BJV conduits.

The results of this study somewhat contradict a recent study by Jalal and colleagues,⁷ in which the authors found greater bacterial adherence in percutaneous BJV valves compared with homemade stents with bovine and porcine pericardium. Interestingly, bacterial adherence significantly increased in traumatized BJV valve leaflets. The main differences between these 2 studies include the use of actual strains isolated from patients with endocarditis in the study by Jalal and colleagues⁷ compared with commercial

Necrotic and poorly incorporated explanted bovine jugular conduit.

Central Message

The mechanism for increased risk of endocarditis in bovine jugular grafts is poorly understood.

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bacterial isolates⁶ and the use of stented and traumatized valves by Jalal and colleagues⁷ instead of intact tissue.⁶

Even though these studies represent an important step in trying to define the mechanism of endocarditis in BJV grafts, it is important to note that in vitro bacterial adherence to fresh tissue is likely significantly different than in vivo bacterial adherence and invasion over time. Most cases of endocarditis in BJV conduits occur years after implantation of the conduit. In our experience, BJV conduits seem to be poorly incorporated, and sometimes clearly necrotic, when explanted years after placement (Figure 1). It is thus feasible that poor incorporation of the conduit associated with worsening shear stress and thrombi from

FIGURE 1. Necrotic and poorly incorporated bovine jugular conduit explanted 1 year after implantation due to branch pulmonary artery compression caused by a sterile pseudocyst around the conduit.

degeneration of the conduit may prompt bacterial adherence and invasion of this necrotic tissue. These late conditions are obviously difficult to simulate in an in vitro setting. Nonetheless, we applaud these early efforts to define the mechanism of endocarditis and hope that further studies can get us closer to determine why some conduits get infected and others don't.

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