Early Structural Valve Deterioration of the Mitroflow Aortic Bioprosthesis

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There has been a trend toward more frequent use of bioprosthetic valves, especially in the young generations, over the last decade. According to the Society of Thoracic Surgeons database, use of bioprosthetic valve increased from 44% in 1996 to 78% in 2006 in North America. Freedom from warfarin use and restrictions on diet and activities make bioprosthetic valves more attractive and popular, and multiple reports have shown that choosing a bioprosthetic valve does not decrease survival despite the increased rate of reoperation.

The Mitroflow aortic prosthesis (Sorin Group Inc) is one of the most frequently used bioprostheses, with >100,000 implanted worldwide. The bovine pericardium is mounted externally around the stent, which maximizes the flow relative to the stent size. The valve is placed in the supra-annular position compared with the intra-annular position in some of the other bioprostheses. These characteristics allow superior valve hemodynamics in the Mitroflow aortic valve, especially in those with a small aortic annulus (19 and 21 mm); therefore, the Mitroflow aortic valve is considered an ideal valve for patients with a small aortic root.

The Achilles heel of the bioprosthetic valve is structural valve deterioration (SVD). Cusp tears and thickening, calcification, pannus formation, and thrombus lead to deterioration of the valve, which is the leading reason for reoperation in patients with a small aortic root, as mentioned earlier. The Mitroflow aortic valve is considered an ideal valve in patients who have a small prosthesis and gradient >30 mm Hg. Recently, Saleeb et al have reported this accelerated degeneration of the Mitroflow valve in a young patient population (<30 years of age). Freedom from valve failure was 53% at 2 years and 18% at 3 years in this series. Life-threatening SVD was detected at a median of 6 months after a normal or mild gradient on a previous echocardiogram. Pathological examination showed that intrinsic calcification causing valve fixation was the main reason for this accelerated degeneration. The Sénage et al report gives us more insight into this phenomenon; accelerated SVD was seen more often in patients with a small aortic prosthesis (76.9%) and in patients with a prosthesis gradient >30 mm Hg. This subgroup had 46.2% valve-related death rate after the occurrence of accelerated SVD. SVD had the strongest correlation of mortality, with an increased risk of death of 7.7. This highlights the importance of frequent monitoring with echocardiograms in patients who have a small prosthesis and gradient >30 mm Hg across the bioprosthesis. Given these data, echocardiogram surveillance should be performed at ≤6 months for early detection.

Another important finding in this article was the underreported incidence of SVD. In many articles, SVD is reported as the rate of reoperation and explantation of the old prosthesis. This underestimates the incidence of SVD because many patients may develop SVD but few may receive surgical treatment. In this series, an
echocardiogram was used to follow up these patients, and an increased gradient and valve insufficiency were used as the criteria for SVD. Only 10.3% of patients with SVD underwent reoperative aortic valve replacement; 11.4% died suddenly and another 11.4% died while on the wait list. There is a question of why 51.4% were not referred to surgery, but this shows the underreported incidence of SVD if reoperation alone is used as the criterion.

The externally mounted pericardial valve poses problems for valve-in-valve transcatheter solutions. Because the internal diameter of Mitroflow valve is only 15.4 mm for the 19-mm valve and 17.3 mm for the 21-mm valve, no transcatheter valve currently is recommended because of the likelihood of a postprocedural gradient.

There is also a concern about coronary obstruction when valve-in-valve replacement is performed because of the externally placed valve leaflet pushing into the ostium. For the US Food Drug and Administration in April 2014. Approved for CE marking in 2011 in Europe, and it was approved to remove phospholipids from the tissue. This new valve was modified from the original Mitroflow A11 by reversing the external cloth so that the ribbed side was external. This valve was used widely in Europe and Canada and was subsequently approved in the United States in 2007. The Mitroflow LX is a variation of the A12 and had minor revisions such as the use of an automatic sewing machine, reduction of sewing ring seams to 1 seam, and prefixation by glutaraldehyde rather than post-fixation. Both Mitroflow A11/12 and Mitroflow LX do not undergo antimineralization treatment. The most recent modification, Mitroflow with phospholipid reduction therapy, added this step. This chemical process uses long-chain alcohol solution to remove phospholipids from the tissue. This new valve was approved for CE marking in 2011 in Europe, and it was approved by the US Food Drug and Administration in April 2014.

Although antimineralization treatment is still a hypothetical explanation for the accelerated degeneration seen in the Mitroflow valve, this report warns about a potential epidemic of SVD in patients who had Mitroflow A12 and LX implanted. It is unclear whether the Mitroflow with phospholipid reduction therapy will be able to prevent this type of SVD, but this is a separate issue. Monitoring patients who received older-generation Mitroflow with frequent echocardiography is mandatory to prevent undesired complications, especially in high-risk patients. Aggressive treatment is needed even in asymptomatic patients once the gradient becomes severe.

Disclosures

None.

References


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