
SHORT COMMUNICATION

Can a central stitch over the Arantius' nodules provide a solution for preoperative severe native AI in LVAD patients?

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ABSTRACT

Purpose: To evaluate the evolution of aortic valve insufficiency (AI) after Park's central stitch in patients with severe, preoperative, native aortic valve insufficiency.

Methods: We retrospectively studied 71 continuous flow LVAD patients between January 2004 and December 2010. Four patients with $AI \geq 3/4$ were treated with a central stitch. An intensive review of the literature was performed to debate the use of the central stitch in this population.

Results: The AI at baseline ($AI = 2.75 \pm 0.5$) and AI at last measurement ($AI = 0.75 \pm 0.65$) is statistically different after central stitch ($p < 0.05$) with mean follow up of 198.25 (± 146.70) days. Total cross clamp-time during the placement of the stitch was 15.5 minutes (± 13.062). CVA was not diagnosed in our cohort.

Conclusions: Park's central stitch can be successfully performed on patients with severe native AI ($\geq 3/4$) with good long-term results. Short ischemic time and simple application of the stitch are the biggest advantages. Due to the progression of AI in longstanding LVAD, the central stitch may be beneficial for LVAD in destination therapy. Since this is a small group of patients and also an early experience, more cases will be necessary to confirm these positive results.

KEY WORDS: Aortic Valve Insufficiency, Heart-Assist Devices, Heart Failure

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INTRODUCTION

Aortic valve insufficiency (AI) is generally considered a relative contraindication for LVAD implantation. Nowadays, different solutions are proposed. Replacement with a bio-prosthetic valve, closure of the aortic root with a patch or closure of the commissures along the coaptation lines are suggested (1-3). Bioprostheses imply some threats like thromboembolic events, early sclerosis, and progressive AI (1, 2). In 2003, Park mentioned a partial closure of the aortic valve with the placement of a central stitch over the nodules of Arantius in moderate AI (4). We evaluated the long term

evolution of AI after Park's stitch in 4 patients with severe preoperative native aortic valve insufficiency.

CASE REPORTS

Patient 1

A 58-year-old man with idiopathic cardiomyopathy (CMP) was presented for implantation of a LVAD. He had moderate to severe aortic valve insufficiency (3/4) due to restrictive cusp motion (El Khoury functional classification type III

(5)). Because of the rapid progression towards heart failure, the patient was scheduled for and Incor[®] implantation (Berlin Heart, Berlin, Germany). After cardiopulmonary bypass (CPB) was started, the aorta was clamped under retrograde cardioplegia (NIH-2) for 10 minutes. The aortic valve was inspected through a small transversal incision in the ascending aorta and we placed a central stitch over the nodules of Arantius with Prolene 4/0 (without pledgets). After closing the aorta with Prolene 4/0, the Incor[®] LVAD was placed under fibrillation and the outflow cannula was connected on a separated incision higher up the ascending aorta. The patient was weaned with the help of a temporary right heart support (Impella[®]; Abiomed, Danvers, MA, USA). During follow-up, postoperative ultrasound showed complete resolution of the aortic insufficiency. At 97 days after the operation, the patient was successfully transplanted. Examination of the explanted heart could not find any thrombus in the left ventricle or in the aortic valve.

Patient 2

A 39-year-old patient with severe ischemic CMP was referred in sustained cardiogenic shock. After installing CPB, retrograde cardioplegia and cross-clamping, a small transversal incision was made to evaluate the aortic valve. The cusps did not show any structural problems but we could see a flattened and dilated sinotubular junction and a dilated ascending aorta (45 mm) (El Khoury functional classification type Ib). With a simple central stitch of Prolene 5/0, the Arantius' nodules were approximated. The incision in the aorta was closed and the cross clamp was released after 35 min. A HeartMate II (HMII) axial flow LVAD (Thoratec, Pleasanton, USA, CA) was implanted using standard procedure under fibrillation with a separated incision for the outflow tract of the HMII. During Follow up, ultrasound showed regression of the aortic insufficiency from severe to mild-moderate. At 21 months after implantation, the patient was successfully transplanted and study of the explanted heart did not show any thrombi in the ventricular cavity or the aortic valve.

Patient 3

A 71-year-old patient in NYHA class 4 with idiopathic dilated CMP was suited for destination therapy. Preoperatively, renal insufficiency with a creatinine level of 1.79 mg/dl was diagnosed. Prolapse of the non-coronary cusp caused

an AI of 3/4 (type II). We performed an exploration of the aortic valve after administration of retrograde cardioplegia (9 min). A central stitch was placed with Prolene 4/0. An HMII device was placed as described above. There were no problems during the weaning process. Ultrasound during follow-up showed a stable reduction of the AI to 1/4 at 191 days after implantation. Renal function was improving with a final creatinine level of 1.07 mg/dl. He is now in NYHA class 2.

Patient 4

A 53-year-old patient with ischemic CMP and CRT-D was evaluated for LVAD implantation. Dobutamine stress echocardiography showed severe AI at 3/4 (baseline 2/4). Implantation of and HMII was scheduled. We performed a transversal aortotomy under retrograde cardioplegia to evaluate the aortic valve. No dilatation of the aortic root was found but there were fibrotic and thickened cusps, causing restrictive movement to the cusps (type III). A central stitch with Prolene 4/0 was performed. Total ischemic time was 8 minutes. The HMII was implanted with a separate incision on the ascending aorta. Follow-up showed a stable reduction of the patient's AI, with measurements not higher than 0.5/4. At 108 days after implantation, the patient received an orthotopic heart transplantation. The explanted heart did not contain any thrombi.

DISCUSSION

Aortic valve insufficiency is a concern in long-term mechanical support. It has generally been considered a relative contraindication to LVAD implantation due to the adverse impact of regurgitation on pump efficiency and ventricular unloading. When insufficiency is severe, the pump must not only maintain the systemic circulation but also recirculate the regurgitant volume. The inability of the pump to meet these increased demands may result in poor systemic perfusion and inadequate left ventricular unloading (6). The only solution to tackle this problem is to create a proper working "valve" during the implantation of the LVAD.

As we know from various studies, the normal evolution of AI in axial flow LVADs is a gradual increase in insufficiency (2, 7-14). Different mechanisms are described for the progression of AI. Fusion of leaflets and dilation of the aortic root are mostly seen as the culprits. In the small series

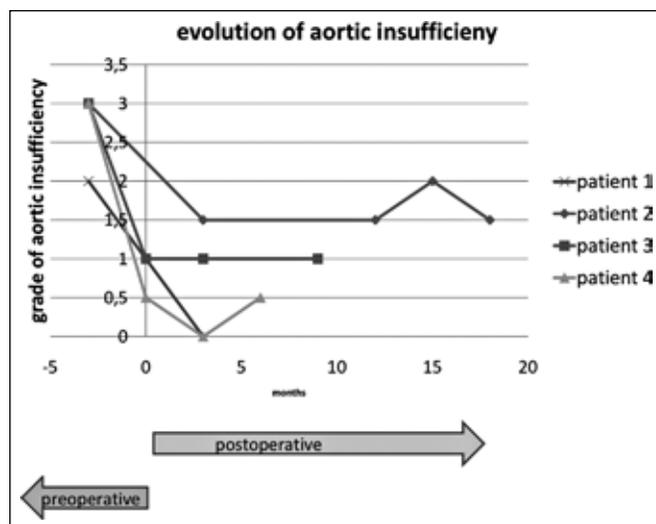


Fig. 1 - Evolution of aortic insufficiency in our cohort.

studied by Mudd et al, it appears that LVAD with continuous flow causes fusion of the aortic leaflet commissures (8). This valvular fusion is the endpoint in a degenerative process leading to malcoaptation. Moreover, there is also continuous stress from the LVAD above which induces improper movement of the leaflets. This induces thickening, loss of pliability and degeneration of the leaflets, finally leading to (mal) fusion of the cusps.

By doing Park's stitch with approximation of the fibrous nodules of Arantius we force correct central coaptation. During the process towards fusion, we expect fusion in a correct position starting from the central stitch and from the annulus. This explains the reasonably good results in our group (Fig. 1). With a mean follow-up of 198.25 (± 146.70) we saw a mean decrease in aortic regurgitation from 2.75 ± 0.5 to 0.75 ± 0.65 at the end of the observation ($p < 0.05$).

Pak et al hypothesized that the same hemodynamic alterations that lead to commissural fusion – loss of pulsatility and persistent elevation of aortic root pressure – cause aortic root dilation in those patients with an underlying predisposition (10). As far back as 1984 and 1987, Olsen et al and Roman et al stated that aortic root dilatation is the primary cause of AI due to a decrease in coaptation height (15, 16). Schafers et al observed that decreased effective height difference (axial distance between the central portion of the free cusp margin and the aortic insertion line) was associated with the development of aortic insufficiency (17).

These findings lead us to two remarks. First of all, patients without AI have normal coaptation heights. In this group,

we can force central coaptation with Park's stitch before dilation of the aortic root starts. Fusion from this stitch leads to strengthening of the aortic root and so decreases dilation and AI. We think that the above-mentioned aspects of Park's stitch can be used in our destination population with perfect functioning valves. Good symmetrical coaptation in an intact, good working native valve is very easy to achieve. The central stitch can bring a solution in this group to prevent further evolution to insufficiency. Secondly, if aortic root dilatation exists preoperatively, we need to be aware of the already small coaptation height. This was certainly the case in patient 2, where AI was caused by a decreased coaptation height (type 1 El Khoury functional classification). The central stitch forced central coaptation at the level of the Arantius' nodules but did not influence the coaptation height in this patient. This caused a lot of stress on the nodules, resulting in the disruption of one of the leaflets from the stitch. We think that aortic root dilation can better be seen as a relative contraindication for the central stitch. An additional subcommissural plasty can offer a solution for this group of patients.

Total aortic valve closure or aortic valve replacement might increase the risk for thrombus formation (1, 2). In our small group, thrombus formation was not diagnosed during echographic evaluation or during post-explanation studies. We did not face stroke. When evaluating the echographic data, we measured bidirectional flow over the aortic valve in three cases. Only Patient 4 had no opening during systole and there was also no antegrade flow measured over the aortic valve; on the other hand, minimal AI caused small backflow towards the ventricle. We believe that this bidirectional flow over the aortic valve reduces the thrombogenic risk due to the continuous wash-out of blood.

Moreover, to reduce thrombogenicity, foreign material was strongly avoided. We only used Prolene monofilament thread without pledgets to close the native aortic valve. Normally, Prolene 4/0 was used, but in Patient 2, the stitch was performed with Prolene 5/0. The fine stitch tore out and caused the initial AI. The fusion of the other two leaflets probably explains the reduction of the AI over time (Fig. 2). Prolene 4/0 seems to be the most appropriate gauge, but the quality of the tissue needs to be considered and might require additional reinforcement with pledgets.

When the LVOT is completely closed, the chance decreases for a patient to maintain hemodynamic stability if the device fails. (2) The heart can pump through the LVAD, but it

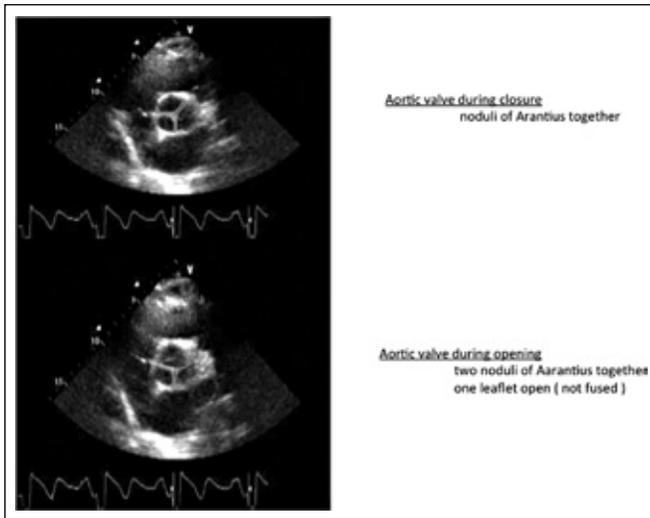


Fig. 2 - Failure of stitch in Patient 2.

will be doubtful that a poor left ventricle function can maintain sufficient output without a valved conduit. Three patients had flow over the aortic valve and in this subgroup, ejection fraction (EF) was above 10%. The EF was very low in Patient 4 (5%), and the valve seemed to be almost completely fused. Anterograde flow seems to protect the aortic valve against the progression towards complete fusion (18). Only a sufficient EF ensures flow through the native valve and this may be enough to serve as an escape mechanism during pump failure.

Another issue with the classic treatment of AI is the significant time span of the procedure. We performed all of our stitches within a short ischemic interval (15.5 ± 13.062 minutes) to minimize the deleterious effects on the myocardium. Preservation of the right ventricular function is

essential for optimal hemodynamics. It can spare us from right (temporary) ventricular support. Nevertheless, we needed to use a right-sided Impella® device (Abiomed, Danvers, MA, USA) in Patient 1 with dilated cardiomyopathy. In spite of the short ischemic interval (10 min), the reduced right ventricular function that was moderate preoperatively became severe. The temporary right ventricular support resolved the problem after 2 days.

CONCLUSIONS

A central stitch approximating Arantius' nodules is a stable and feasible solution for patients with severe AI who are scheduled for LVAD implantation. The placement of the stitch is less time consuming in comparison with classic solutions and the thrombogenicity seems to be low. Choosing the appropriate suture material is crucial for the success of the stitch. Patients with aortic root dilation are less suitable for a central stitch, but an additional subcommissural plasty can be beneficial. Placement of the central stitch in patients with destination therapy can provide a safe solution for the progression in AI. Since this is a small group of patients and also an early experience, more cases will be necessary to confirm these positive results.

Conflict of Interest Statement: None of the authors has a conflict of interest.

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REFERENCES

1. Feldman CM, Silver MA, Sobieski MA, Slaughter MS. Management of aortic insufficiency with continuous flow left ventricular assist devices: bioprosthetic valve replacement. *J Heart Lung Transplant.* 2006;25(12):1410-1412.
2. Cohn WE, Demirozu ZT, Frazier OH. Surgical closure of left ventricular outflow tract after left ventricular assist device implantation in patients with aortic valve pathology. *J Heart Lung Transplant.* 2011;30(1):59-63.
3. Adamson RM, Dembitsky WP, Baradarian S, et al. Aortic valve closure associated with HeartMate left ventricular device support: technical considerations and long-term results. *J Heart Lung Transplant.* 2011;30(5):576-582.
4. Park SJ, Liao KK, Seguro R, Madhu KP, Miller LW. Management of aortic insufficiency in patients with left ventricular assist devices: a simple coaptation stitch method (Park's stitch). *J Thorac Cardiovasc Surg.* 2004;127(1):264-266.
5. El Khoury G, Glineur D, Rubay J, et al. Functional classification of aortic root/valve abnormalities and their correlation

- with etiologies and surgical procedures. *Curr Opin Cardiol.* 2005;20(2):115-121.
6. Rao V, Slater JP, Edwards NM, Naka Y, Oz MC. Surgical management of valvular disease in patients requiring left ventricular assist device support. *Ann Thorac Surg.* 2001;71(5):1448-1453.
 7. Rose AG, Connelly JH, Park SJ, Frazier OH, Miller LW, Ormaza S. Total left ventricular outflow tract obstruction due to left ventricular assist device-induced sub-aortic thrombosis in 2 patients with aortic valve bioprosthesis. *J Heart Lung Transplant.* 2003;22(5):594-599.
 8. Mudd JO, Cuda JD, Halushka M, Soderlund KA, Conte JV, Russell SD. Fusion of aortic valve commissures in patients supported by a continuous axial flow left ventricular assist device. *J Heart Lung Transplant.* 2008;27(12):1269-1274.
 9. Cowger J, Pagani FD, Haft JW, Romano MA, Aaronson KD, Kollas TJ. The development of aortic insufficiency in left ventricular assist device-supported patients. *Circ Heart Fail.* 2010;3(6):668-674.
 10. Pak SW, Uriel N, Takayama H, et al. Prevalence of de novo aortic insufficiency during long-term support with left ventricular assist devices. *J Heart Lung Transplant.* 2010;29(10):1172-1176.
 11. Bryant AS, Holman WL, Nanda NC, et al. Native aortic valve insufficiency in patients with left ventricular assist devices. *Ann Thorac Surg.* 2006;81(2):e6-e8.
 12. Miller LW, Pagani FD, Russell SD, et al. HeartMate II Clinical Investigators. Use of a continuous-flow device in patients awaiting heart transplantation. *N Engl J Med.* 2007;357(9):885-896.
 13. Pagani FD. Continuous-flow rotary left ventricular assist devices with "3rd generation" design. *Semin Thorac Cardiovasc Surg.* 2008;20(3):255-263.
 14. Topilsky Y, Oh JK, Atchison FW, et al. Echocardiographic findings in stable outpatients with properly functioning HeartMate II left ventricular assist devices. *J Am Soc Echocardiogr.* 2011;24(2):157-169.
 15. Olson LJ, Subramanian R, Edwards WD. Surgical pathology of pure aortic insufficiency: a study of 225 cases. *Mayo Clin Proc.* 1984;59(12):835-841.
 16. Roman MJ, Devereux RB, Niles NW, et al. Aortic root dilatation as a cause of isolated, severe aortic regurgitation. Prevalence, clinical and echocardiographic patterns, and relation to left ventricular hypertrophy and function. *Ann Intern Med.* 1987;106(6):800-807.
 17. Schäfers HJ, Bierbach B, Aicher D. A new approach to the assessment of aortic cusp geometry. *J Thorac Cardiovasc Surg.* 2006;132(2):436-438.
 18. Mudd JO, Cuda JD, Halushka M, Soderlund KA, Conte JV, Russell SD. Fusion of aortic valve commissures in patients supported by a continuous axial flow left ventricular assist device. *J Heart Lung Transplant.* 2008;27(12):1269-1274.