# Transcatheter Tricuspid Valve Replacement for Anderson Fabry Disease With Severe Tricuspid Regurgitation

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## INTRODUCTION

Anderson Fabry disease (AFD) is an X-linked recessive metabolic disorder characterized by deficient activity of the lysosomal hydrolase, $\alpha$-galactosidase A. Clinical manifestations of AFD are diverse and vary greatly depending on age and gender. These can be multiorgan involving the kidney, brain, heart, eye, and skin, and hence the diagnosis of AFD may be missed.[1]

Treatment depends on early recognition of the disease and timely institution of enzyme replacement therapy (ERT).[2] A severe form of valvular regurgitation that would require surgical correction is relatively rare in AFD. The patient in this report had been followed for many years for renal dysfunction, mitral regurgitation, and hypertrophic cardiomyopathy (HCM), but AFD was not recognized until recently. Our patient had severe tricuspid regurgitation (TR) and implanted transcatheter tricuspid valve replacement (TTVR) and accepted ERT for further treatment. In this report, we described echocardiographic imaging and clinical observations to further understand AFD, help avoid misdiagnosis, and provide evidence for its clinical treatment and prognosis.

## CASE PRESENTATION

A 54-year-old man was admitted with bilateral lower extremity edema and abdominal distension that persisted over a year but worsened within the last 3 months. The patient had a history of cardiac pacemaker implantation, renal replacement, and mitral valve replacement (MVR), which were performed in 2006, 2009, and 2015, respectively. They denied any family history of cardiovascular or kidney disease. After being admitted, physical examination showed blood pressure of 112/70 mm Hg, heart rate of 60 bpm, and body surface area of 1.4 $m^2$. On auscultation, there was a diastolic murmur over the right lower sternal border. A blood test showed high level of creatinine, 159 $\mu$mol/L; glomerular filtration rate, 27.9 mL/min; trioxypurine 724, $\mu$mol/L; N-terminal b-type natriuretic peptide precursor, 6,050 pg/mL, B-type natriuretic peptide, 1,491.38 pg/mL, and international standardized ratio of prothrombin time, 2.0.

The transthoracic echocardiogram (TTE), which was performed 1 day after the patient was admitted to our hospital, revealed markedly increased wall thickness of the left (LV) and right ventricular (RV) myocardium (interventricular septum, 2.3 cm; post wall, 1.7 cm; RV free wall, 1.3 cm), enlargement of the left and right atrium (left atrial [LA] volume index [LAVi], 69.2 mL/$m^2$; right atrial [RA] volume index, 51.4 mL/$m^2$), LV ejection fraction (LVEF) of 67%, RV fractional area change of 55%, mild increased pulmonary artery systolic pressure (37 mm Hg), and normally functioning mechanical mitral valve (mean transvalvular gradient, 6.0 mm Hg); transesophageal echocardiography (TEE) showed severe TR with vena contracta (VC) of 0.7 cm (Figure 1, Videos 1 and 2). An electrocardiogram revealed atrial fibrillation and ventricularly paced rhythm. Chest radiography showed postoperative MVR, cardiomegaly, and dual-chamber pacemaker implantation (Figure 2). An overall evaluation showed that the Society of Thoracic Surgeons score was 8.07%, clinic risk score was 7, Kansas City Cardiomyopathy Questionnaire (KCCQ) score was 58.33, and 6-minute walking distance (6MWD) was 308 m. The patient was informed that due to their surgical risk, they were not eligible to undergo a second thoracotomy but met the inclusion criteria for interventional tricuspid valve surgery for severe TR (VC $\geq$ 0.7 cm), as their LV systolic function was normal (LVEF $\geq$ 50%), the pulmonary artery systolic pressure was $\leq$55%, and there was no coexistent severe valve dysfunction. After obtaining consent, TTVR was performed.

LuX-Valve is a novel TTVR system that consists of the following 4 components: (1) a trileaflet prosthetic valve with bovine pericardium; (2) a self-expandable nitinol valve stent consisting of an atrial disc; (3) 1 interventricular septal anchor "tongue"; and (4) 2 expanded polytetrafluoroethylene-covered graspers (Figure 3A). It can be delivered via a 32-F catheter (Figure 3B and C) through a minimally invasive right thoracotomy under anesthesia without cardiopulmonary bypass using TEE and fluoroscopy guidance (Figure 3D and E).

The total procedure went smoothly. The TEE showed that the position and function of the implanted valve prosthesis were normal, while color Doppler showed no obvious valve regurgitation or paravalvular leakage (Figure 4, Videos 3 and 4). Right ventricular angiography was not performed because of a history of kidney transplantation and unilateral renal insufficiency on admission. The patient's symptoms improved to mild exertional dyspnea (New York Heart Association functional class I), the edema disappeared in both legs, and there were no TTVR-related complications. Compared to pre-TTVR, TTE showed that TR disappeared, the LAVi increased (82.1 vs 69.2 mL/$m^2$), RA volume index decreased (42.8 vs 51.4 mL/$m^2$) before discharge, and TV mean gradient was improved (2.5 vs 1.3 mm Hg).

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## VIDEO HIGHLIGHTS

**Video 1:** Two-dimensional TTE, apical 4-chamber view, demonstrates severe biventricular myocardial wall thickness with normal systolic function. Also noted is a mechanical MVR and pacemaker wire.

**Video 2:** Two-dimensional TTE, parasternal short-axis mid-ventricular display, demonstrates severe biventricular myocardial wall thickness and normal global LV systolic function.

**Video 3:** Two-dimensional TEE with color Doppler, mid-esophageal window, biplane (0° and 90°) display demonstrates dilated right atrium with severe TR.

**Video 4:** Two-dimensional TEE with color Doppler, multiplanar view (0°/60°/120°) demonstrates no significant valvular or paravalvular TR after TTVR implantation.

**Video 5:** Two-dimensional TTE, apical 4-chamber view demonstrates progressive thickening of the biventricular myocardial walls, reduction in right heart dimensions, and further dilatation of the left atrium 1 year following the TTVR compared with baseline.

**Video 6:** Two-dimensional TTE, parasternal short-axis mid-ventricular display demonstrates persistently severe biventricular myocardial wall thickness and normal global LV systolic function 1 year following the TTVR compared to baseline.

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Nearly 1 year after TTVR, the patient began to feel fatigue and shortness of breath after activity, which would subside after rest (New York Heart Association class II) and was associated with elevated B-type natriuretic peptide (1,099 pg/mL) and reduced 6MWD (320 m), but a good KCCQ score (81.67). Routine echocardiographic parameters suggested that the volume of the left atrium was twice as large as before TTVR and the thickness of the RV wall remained 1.3 cm, while interventricular septal diameter (IVSd) and posterior wall diameter (PWd) were thicker than pre-TTVR, that is, 2.9 and 2.0 cm, respectively (Videos 5 and 6).

Future studies should investigate whether LV muscle thickening and LA enlargement are related to clinical symptoms. Furthermore, the mechanism of the continuously increased LV myocardial wall thickness should be further examined. The diagnosis of AFD should be raised if there are 2 or more clinical problems from among the following: acroparesthesia or neuritic pain in hands or feet, persistent proteinuria of unknown cause, progressive renal impairment of obscure cause, unexplained HCM, or the presence of angiokeratomas. Galactosidase A activity assays should be checked for AFD screening after clinical suspicion is raised. Our patient had many characteristic features of AFD including progressively increased LV and RV wall thickness, which was initially but incorrectly presumed to be due to aging and renal dysfunction. The diagnostic criteria for AFD are based on guidelines recommended by the National Association of Genetic Counselors and the consensus of Chinese experts on the diagnosis and treatment of AFD and requires one of the following: (1) abnormal X chromosome GLA gene detection; (2) decreased blood $\alpha$-galactosidase A enzyme activity; (3) increased plasma Lyso-GL-3 content; or (4) pathological examination of kidney, skin, myocardium, and nerve tissue showing corresponding vacuolar changes in histiocytes under a light microscope and a cytoplasm that is full of osmium-loving "myeloid bodies" under an electron microscope. In the present case, DNA analysis as the gold standard was performed and confirmed the diagnosis of AFD 1 year after TTVR. The results revealed a heterozygote $\alpha$-galactosidase mutation at chrX: 100653895 [c.679 C>T(p.R227X)] (Figure 5). The detection result of deacetyl GL-3 (Lyso-GL-3) was 87.73 ng/mL (reference value < 1.11 ng/mL).

After the diagnosis, the patient's history and physical findings were meticulously reviewed. Since the age of 13, the patient had suffered stabbing pain in the palms and soles (acroparesthesia) during the period of heavy stress associated with school requirements or they would come down with a fever, hypohidrosis, intolerance to heat and cold, and intermittent seizures. Their symptoms improved with age without any specific therapy. Due to unknown reasons, proteinuria and renal impairment began to appear at middle-young age, which was treated conservatively and eventually required renal transplantation in 2009. At the age of 39, the patient was implanted with a permanent pacemaker for sinus bradycardia and recurrent cardiogenic syncope. Nine years later, they were diagnosed with mitral valve prolapse as well as asymmetric nonobstructive HCM and underwent mechanical MVR and tricuspid valvuloplasty. We reviewed in detail the changes that occurred within 1 year of echocardiography, that is, from October 2020 to November 2021. Compared to pre-TTVR, the size of the left atrium significantly increased at 1 year after TTVR (LAVi, 162.8 vs 69.2 mL/m$^2$), as well as LV wall thickness (IVSd, 2.9 vs 2.3 cm; PWd, 2.0 vs 1.7 cm, respectively). Although LVEF (70% vs 67%) and RV fractional area change (63% vs 55%) remained normal and stable, comprehensive, multichamber two-dimensional speckle-tracking echocardiography (STE) showed the following: LA reservoir function (peak positive strain) of 8% (vs 6%); LA conduit function (early diastolic strain) of −8% (vs −7%); RA reservoir function (peak positive strain) of 5% (vs 8%); RA conduit function (early diastolic strain) of −8% (vs −7%); RV global longitudinal strain (LS) of −8.8% (vs −8.8%), RV free-wall strain of −11.8% (vs −12.9%), LV global LS of −9.4% (vs −8.9%), and circumferential strain (CS) of −12.1% (vs −14.5%; Figure 6). The strains of all the chambers were lower than normal reference values both before and after TTVR (LV LS, −20%; RV LS, −21%; RV free-wall strain, −20%; LA strain, 32%; RV strain, 60%, respectively). Also, there was no obvious strain improvement after TTVR and continuously enhanced oral diuretic therapy (antisterone 10 mg twice a day and furosemide 10 mg twice a day) during the follow-up period. Meanwhile, tissue Doppler recording of the RV free wall at the tricuspid annular level showed abnormal systolic annular velocity (Figure 1C, white arrow: RV peak systolic velocity of the tricuspid annulus = 8 cm/sec), which was slightly lower than the normal reference (>9.5 cm/sec). Nevertheless, 6MWD and KCCQ scores improved compared with before TTVR (326 vs 308 m; 81.67 vs 58.33, respectively), especially the latter one (Figure 7). The patient started to take ERT in January 2022: $\beta$-Galrch (FABRAZYME) 45 mg four times a day by venous channel as the routine method to cure AFD. Six months later (July 2022), due to the impact of the COVID epidemic, the patient was treated and followed up locally. After ERT, the patient's fatigue, asthma, functional capacity, lower extremity edema, and shortness of breath improved. The detection results of deacetyl GL-3 (Lyso-GL-3; 47.99 vs 87.63 ng/mL), creatinine (143 vs 159 $\mu$mol/L), glomerular filtration rate (45.9 vs 27.9 mL/min), and trioxypurine (613 $\mu$mol/L vs 724 $\mu$mol/L) each demonstrated progressively

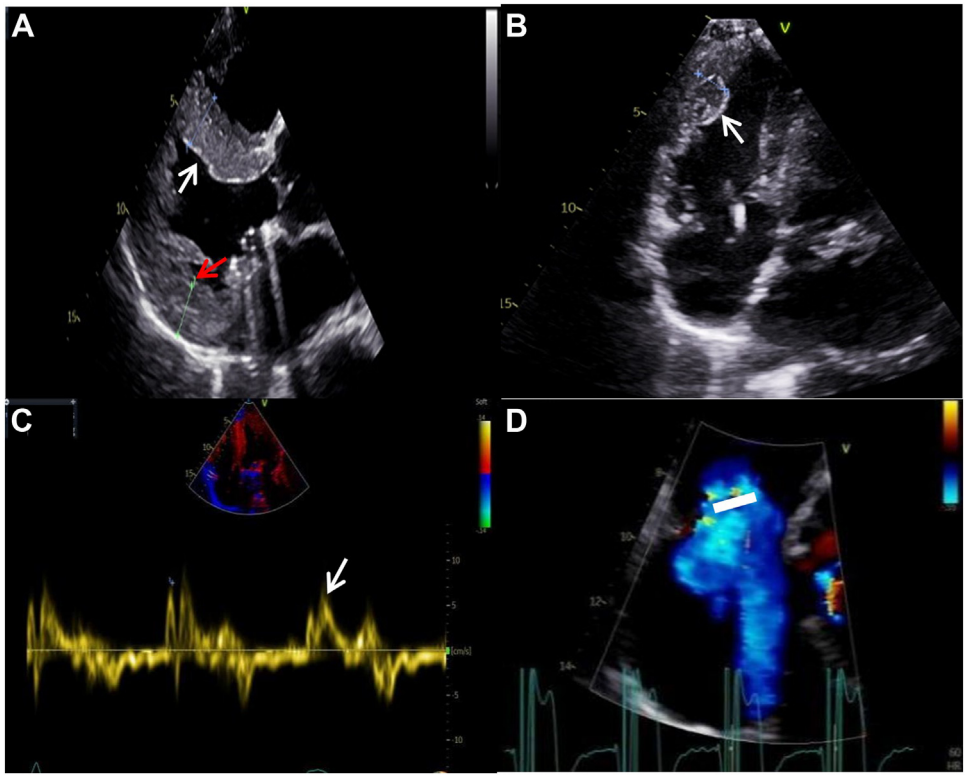


Figure 1 **(A)** Parasternal long-axis view shows severe LV hypertrophy (*white arrow* points to the interventricular septum measuring 2.3 cm; *red arrow* points to the inferolateral wall measuring 2.0 cm). **(B)** Right ventricular focused view shows RV hypertrophy (*white arrow* points to the RV free wall measuring 1.3 cm). **(C)** Tissue Doppler of the free wall at the tricuspid annular level demonstrates abnormal annular velocity: RV peak systolic velocity of the tricuspid annulus (*white arrow*) = 8 cm/sec. **(D)** Severe TR showing a VC (*white line*) = 0.8 cm.

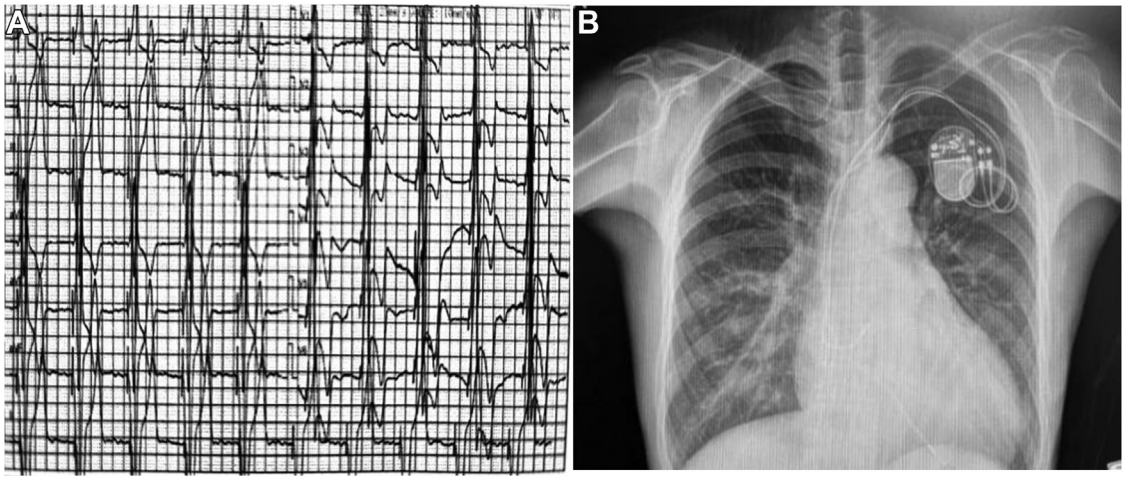


Figure 2 **(A)** A 12-lead electrocardiogram showed atrial fibrillation with a paced rhythm with a heart rate of 60 bpm. **(B)** Chest radiography revealed MVR, cardiomegaly, and a dual-chamber pacemaker implantation.

improved values. Comparison TEE demonstrated the following findings: LVEF, 62% vs 67%; LAVi, 78.5 vs 162.8 mL/m$^2$; IVSd, 2.5 vs 2.9 cm; PWd, 2.0 vs 2.0 cm; and LV global LS, −7.4% vs 9.4%. In conclusion, ERT treatment had an important role in improving the prognosis of this patient with respect to clinical symptoms and cardiac and renal functions.

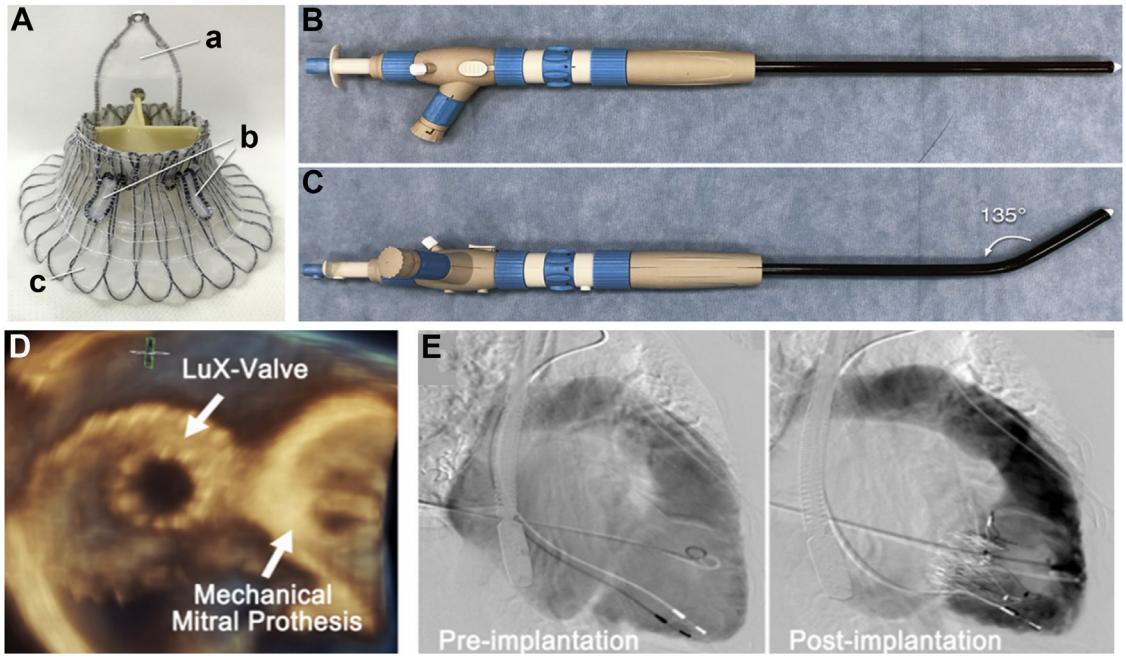


Figure 3 LuX-Valve is a radial force-independent orthotopic TTVR device for severe TR. **(A)** The LuX-Valve consists of an interventricular septal anchor (a), 2 graspers (b), and a valve disk (c). **(B and C)** General view of the delivery catheter. **(D)** Three-dimensional TEE TTVR en face view shows the device was deployed in targeted position. **(E)** Right ventricular angiography showed TR significantly improved from baseline (*left panel*) compared with after TTVR (*right panel*). Panels A-C are reprinted from Lu *et al*[3] with permission of Elsevier; panels D-E are reprinted from Lu *et al*[4] with permission from BMJ Publishing Group Ltd.

## DISCUSSION

Most patients with AFD have cardiac manifestations; however, symptoms of the cardiovascular system manifest much later than Gb3 accumulation, which remains one of the most important causes of morbidity and mortality in affected patients. The cardiac symptoms include increased LV wall thickness, binary sign, prominent papillary muscles, RV and functional impairment, atrial enlargement, and reduced compliance. Valvular heart disease includes leaflet thickening and redundancy and even prolapse, aortic dilatation, and conduction disturbances.[5] Increased LV wall thickness is the hallmark characteristic of AFD. In cohorts of patients initially diagnosed with HCM, 3.9% to 12% are eventually found to have AFD,[6] so differential diagnosis for patients with HCM or unexplained features of hypertrophy on echocardiography should include AFD, particularly with various late-onset diseases.

Echocardiography is an effective noninvasive method for assessing the structural and functional consequences of AFD on the heart. Two-dimensional STE offers additional advantages and can assess regional function in all myocardial segments. Patients with AFD have significantly lower magnitudes of global LS and CS compared to healthy control groups.[7] Anderson Fabry disease with left ventricular hypertrophy (LVH) demonstrates significantly lower absolute values of global LS compared to those without LVH but display similar global CS and a similar loss of their normal base-to-apex CS gradient. In the present case, the LV LS and CS results had much lower absolute values than healthy control groups, which is consistent with the results of the previous study. In contrast, patients with nonobstructive HCM have significantly higher global CS and maintain a normal base-to-apex CS gradient compared with patients with AFD.[8] Myocardial fibrosis, which can be detected by cardiovascular magnetic resonance imaging, finally ensues, causing progressive deterioration in longitudinal and radial function.[9] In the present case, the right ventricle was also involved, suggesting that the patient had a great extent of myocardial fibrosis, although this was established without the use of cardiovascular magnetic resonance imaging.

Histologic studies showed glycolipid deposition in atrial cells, which may cause atrial enlargement and predisposition to atrial arrhythmias.[5] In addition, in some cohort studies, the mean LA size on echocardiography was greater in patients with AFD than in age-matched control groups, with the prevalence reaching up to 30%.[10] Increased LAV occurs more frequently in patients with increased LV mass and myocardial fibrosis.[11] Another study showed that reduced LA reservoir, conduit, and contractile functions were characterized by a decrease in peak positive, early diastolic, and late diastolic strain and strain rates by STE.[12] Our case showed an underlying atrial myopathy related to glycolipid deposition in the atria that may occur independently of LVH and the diastolic dysfunction.

The conduction abnormalities could be seen in AFD patients, such as sinus node dysfunction, atrioventricular (AV) block, and bundle branch block. In addition, myocardial fibrosis can extensively impair biventricular and atrial function and hence lead to atrial fibrillation.[13] Our patient had a dual-chamber pacemaker because of sinus node dysfunction or AV block. Although the initial rhythm was sinus, the patient demonstrated persistent recordings of atrial fibrillation and high-degree AV block with 100% ventricular pacing.

Although current AFD specific treatment includes ERT and migalastat, there are new therapeutic approaches that are in development. Enzyme replacement therapy is effective when AFD is diagnosed early and the amount of myocardial fibrosis is still low.[5] Several other

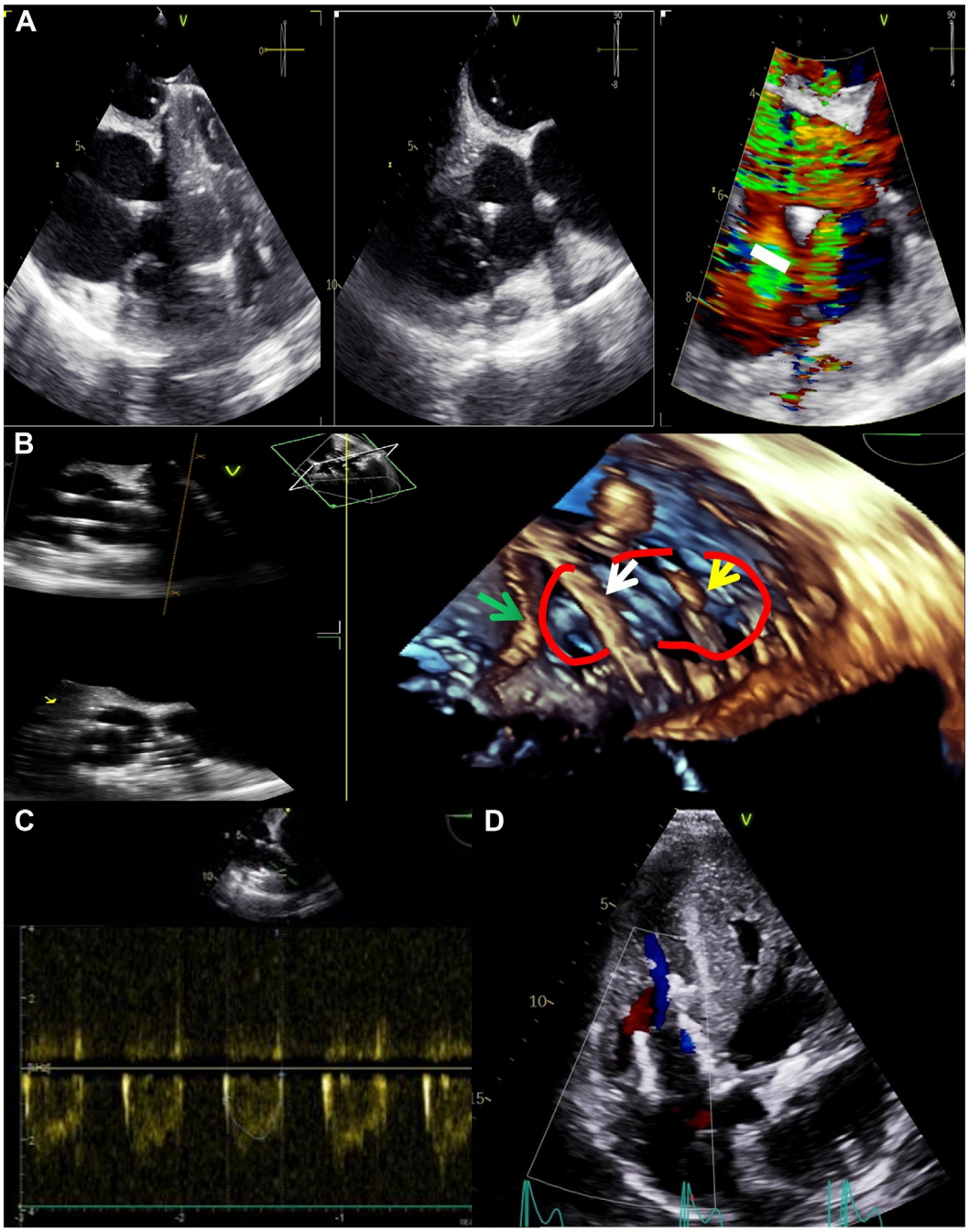


Figure 4 LuX-Valve Implantation. **(A)** Midesophageal biplane views showing severe TR (*white line*: VC = 0.8 cm). **(B)** Midesophageal biplane view and three-dimensional en face view shows the system implantation (*green arrow*: interatrial septum; *white arrow*: pacemaker electrode; *yellow arrow*: catheter sheath, *red circle*: the edge of atrial disk). Pacemaker electrode was located at the junction of the posterior valve and septal valve, under the edge of the atrial disk, and had no effect on pacemaker function. **(C)** Pulsed-wave Doppler showed the postoperative tricuspid transvalvular mean gradient was 1.43 mm Hg. **(D)** The transthoracic RV focused view of the percutaneous tricuspid valve was seen without paravalvular leakage visualized.

factors impact the cardiac response to ERT, including sex, phenotype, timing and dosage of ERT, and antidrug antibody development against exogenous $\alpha$-galactosidase A. Moreover, chaperone therapy was proved by clinical trials, and open-label extension studies showed that the treatment with migalastat was associated with a significant decrease in the LV mass index. Nevertheless, recent real-world data suggested that biochemical and clinical responses to chaperone therapy must be carefully monitored to confirm its clinical effificacy.[14] The patient in our case had undiagnosed AFD for a long time, whose clinical manifestations related to myocardial reverse remodeling and

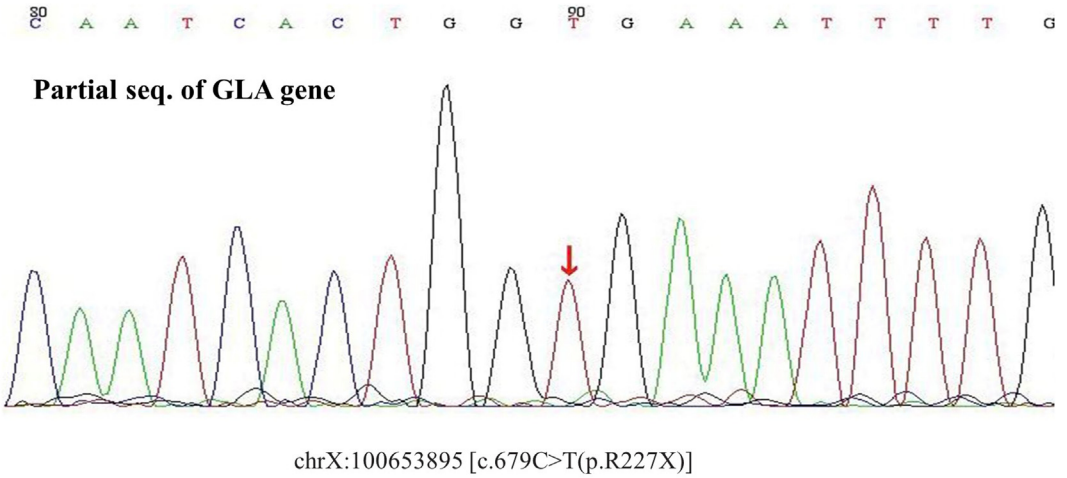


Figure 5 Automated sequencing profile of genomic DNA revealed a heterozygote $\alpha$-galactosidase A mutation at chrX:100653895.

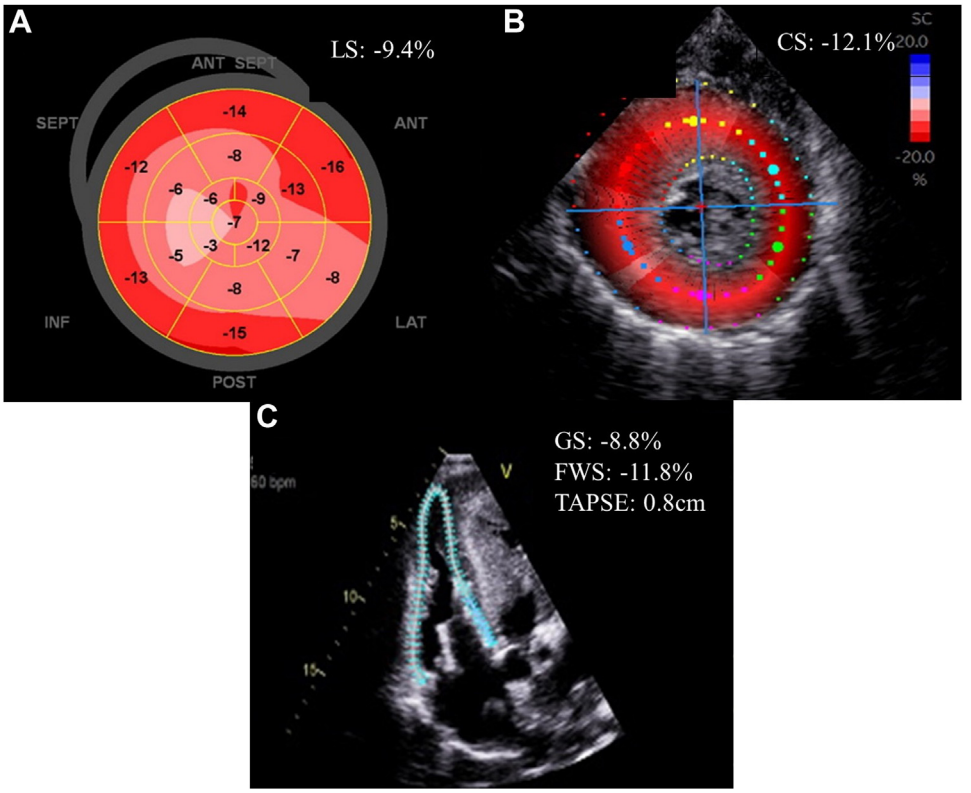


Figure 6 Biventricular involvement prior to ERT. **(A)** Left ventricular global LS, **(B)** CS, and **(C)** RV global GS and free wall strain (FWS) and tricuspid annular plane systolic excursion (TAPSE) demonstrated abnormal strain imaging.

cardiac as well as renal functions notably improved after only half a year of ERT.

Histopathologic studies demonstrated valvular glycolipid deposition in patients with AFD causing leaflet thickening and redundancy. Other case reports described patients with aortic, mitral, and even TR who needed valve surgery for their symptoms.[5,15] In our case, the severe TR was related to 3 possible mixed aspects: (1) right atrium enlargement due to the long history of atrial fibrillation caused by myocardial fibrosis due to AFD, (2) long-term interaction between endocardial lead and leaflet or chordal structure leading to loss of leaflet mobility or coaptation, and (3) TR secondary to pulmonary hypertension that resulted from a long history of left-sided heart valve replacement. Three years ago, we developed a radial force-independent TTVR device that was successfully implanted in patients with severe TR.[3] It was found that TTVR was feasible, safe, and with low complication rates in patients with severe TR[4] who were at high risk for traditional cardiac surgery. The patient in our case had severe TR

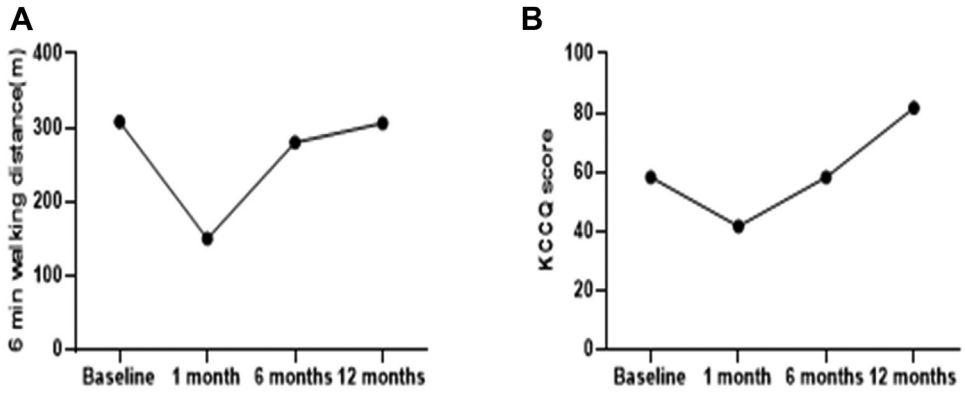


Figure 7 A graph showing the change in 6MWD **(A)** and KCCQ score **(B)** over 1 year. A brief decline was seen 1 month after TTVR, followed by an improvement thereafter.

due to AFD with significantly decreased cardiac function and high risk for a second thoracotomy. After TTVR, the KCCQ score and 6MWD demonstrated gradual improvement in exercise capacity and quality of life and may be better after additional ERT.

## CONCLUSION

The lessons from this case are that the constellation of unexplained progressive LV wall thickness or LV hypertrophy, valvular abnormalities, and multiorgan involvement should trigger the differential diagnosis of AFD. Besides that, using two-dimensional STE, the LV LS and LV CS in AFD were significantly lower than in healthy control groups. These findings suggest that STE should be performed when AFD is being considered. This case also demonstrated that treatment of severe TR with a novel interventional device (LuX-Valve) was feasible.

## ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

## CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

## FUNDING STATEMENT

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## DISCLOSURE STATEMENT

The authors report no conflict of interest.

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## SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2022.10.008.

## REFERENCES

1. Desnick RJ, Brady RO. Fabry disease in childhood. J Pediatr 2004;144(5 Suppl):S20-6.
2. Biegstraaten M, Arngrimsson R, Barbey F, Boks L, Cecchi F, Deegan PB, et al. Recommendations for initiation and cessation of enzyme replacement therapy in patients with Fabry disease: the European Fabry Working Group consensus document. Orphanet J Rare Dis 2015;10:36.
3. Lu FL, Ma Y, An Z, Cai CL, Li BL, Song ZG, et al. First-in-Man experience of transcatheter tricuspid valve replacement with LuX-valve in high-risk tricuspid regurgitation patients. JACC Cardiovasc Interv 2020;13:1614-6.
4. Lu FL, An Z, Ma Y, Song ZG, Cai CL, Li BL, et al. Transcatheter tricuspid valve replacement in patients with severe tricuspid regurgitation. Heart 2021;107:1664-70.
5. Patel V, O'Mahony C, Hughes D, Rahman MS, Coats C, Murphy E, et al. Clinical and genetic predictors of major cardiac events in patients with Anderson-Fabry disease. Heart 2015;101:961-6.
6. Sachdev B, Takenaka T, Teraguchi H, Tei C, Lee P, McKenna WJ, et al. Prevalence of Anderson-Fabry disease in male patients with late onset hypertrophic cardiomyopathy. Circulation 2002;105:1407-11.
7. Gruner C, Verocai F, Carasso S, Vannan MA, Jamorski M, Clarke JT, et al. Systolic myocardial mechanics in patients with Anderson-Fabry disease with and without left ventricular hypertrophy and in comparison to nonobstructive hypertrophic cardiomyopathy. Echocardiography 2012;29:810-7.
8. Labombarda F, Saloux E, Milesi G, Bienvenu B. Loss of base-to-apex circumferential strain gradient: a specifific pattern of Fabry cardiomyopathy? Echocardiography 2017;34:504-10.
9. Weidemann F, Breunig F, Beer M, Sandstede J, Stork S, Voelker W, et al. The variation of morphological and functional cardiac manifestation in

Fabry disease: potential implications for the time course of the disease. Eur Heart J 2005;26:1221-7.
10. Senechal M, Germain DP. Fabry disease: a functional and anatomical study of cardiac manifestations in 20 hemizygous male patients. Clin Genet 2003;63:46-52.
11. Pieroni M, Chimenti C, Ricci R, Sale P, Russo MA, Frustaci A. Early detection of Fabry cardiomyopathy by tissue Doppler imaging. Circulation 2003;107:1978-84.
12. Pichette M, Serri K, Page M, Di LZ, Bichet DG, Poulin F. Impaired left atrial function in Fabry disease: a longitudinal speckle-tracking echocardiography study. J Am Soc Echocardiogr 2017;30:170-1792.
13. Acharya D, Robertson P, Kay GN, Jackson L, Warnock DG, Plumb VJ, et al. Arrhythmias in Fabry cardiomyopathy. Clin Cardiol 2012;35:738-40.
14. Lenders M, Nordbeck P, Kurschat C, Karabul N, Kaufeld J, Hennermann JB, et al. Treatment of Fabry's disease with migalastat: outcome from a prospective observational multicenter study (FAMOUS). Clin Pharmacol Ther 2020;108:326-37.
15. Cho SC, Yoo HW, Lee JW, Jang JY, Heo R, Song JM. Fabry disease presenting with hypertrophic cardiomyopathy and tricuspid regurgitation. J Cardiovasc Ultrasound 2016;24:324-8.