

Atrial septostomy as a therapeutic option in stiff left atrial syndrome

Mehmet Eren, Yalcin Velibey[✉], Erkan Kahraman

Siyami Ersek Thoracic and Cardiovascular Surgery Center, Training and Research Hospital, Department of Cardiology, Istanbul, Türkiye

✉ Correspondence to: dr_yalchin_dr@yahoo.com.tr

<https://doi.org/10.26599/1671-5411.2026.08.005>

The stiff left atrial syndrome (SLAS) is a relatively rare but well-established cause of heart failure (HF), typically occurring several years after catheter or surgical ablation for atrial fibrillation (AF) or mitral valve replacement (MVR) due to scarring with reduction or loss of left atrium (LA) compliance and contractility.^[1] In patients with SLAS, because of compliance, contractility and reservoir are significantly impaired due to extensive LA fibrosis, LA pressure increases significantly during hemodynamic stress, consequently leading to pulmonary venous hypertension and subsequent pulmonary congestion, and eventually results in intractable HF.^[2] Treatment of SLAS is quite challenging and restricted to the use of diuretics only, which has limited efficacy.^[1,2] We report a clinical case of SLAS after MVR and Cox-Maze IV procedure for longstanding AF, in which the patient reported immediate improvement after atrial septostomy. The aim of this case presentation is to address interventional treatment methods of SLAS and especially highlight the importance of atrial septostomy in symptomatic patients despite optimal medical therapy.

A 75-year-old female patient was admitted to our hospital with New York Heart Association Class III-IV dyspnea. Her history revealed MVR for severe mitral regurgitation, Cox-Maze IV procedure, LA plication, and appendage ligation 15 years ago, percutaneous coronary intervention to the right coronary artery, hypertension, diabetes mellitus, and persistent AF. She was on long-term warfarin, spironolactone (50 mg/day), diltiazem (180 mg/day), metformin, digoxin, and furosemide (120 mg/day) treatment. Physical examination was notable for an irregular heart rate at 75 beats/min and crackles in both lungs. Electrocardiography revealed AF rhythm. Transthoracic echocardiography (TTE) demonstrated a functioning

prosthetic mitral valve (peak pressure gradient: 10 mmHg; mean pressure gradient: 4 mmHg; effective orifice area: 2.8 cm²; pressure half-time: 110 ms) severe dilatation of LA (LA diameter in the parasternal long axis plane was 50 mm), mild aortic regurgitation, elevated (70 mmHg) pulmonary artery systolic pressure (PASP), moderate tricuspid regurgitation, dilated and noncollapsing inferior vena cava. Left ventricular ejection fraction was 55%. In TTE recordings obtained before and one year after the MVR and Cox-Maze IV procedure, the estimated PASPs were documented as 52 mmHg and 38 mmHg, respectively, accompanied by mild tricuspid regurgitation. Routine laboratory tests revealed normal electrolyte levels and normal renal, liver and thyroid functions. The international normalized ratio was 2.75. Transesophageal echocardiography (TEE) demonstrated a functioning prosthetic mitral valve in the regular position and normal opening movement without a central or paravalvular regurgitation, severe dilatation of LA and a marked left-to-right shunt through a patent foramen ovale on a color flow Doppler (Figure 1, Video 1). Coronary angiography revealed nonstenotic atherosclerotic plaques in coronary arteries with a patent stent in right coronary artery. Cardiac catheterization demonstrated normal mean right atrial (RA) pressure (7 mmHg), elevated mean pulmonary arterial pressure (34 mmHg), and elevated pulmonary capillary wedge pressure (PCWP) (Table 1). LA pressures were assessed by direct catheterization of the LA via a patent foramen ovale. The LA pressure tracing demonstrated a markedly elevated mean LA pressure (26 mmHg) accompanied by prominent giant V waves (Figure 2A). In contrast, the left ventricular end-diastolic pressure was within normal limits (10 mmHg) (Figure 2B). The transpulmonary gradient, systemic and pulmonary vascular resistance were 9 mmHg, 19 Wood units, and 4.25 Wood units, respectively. There was

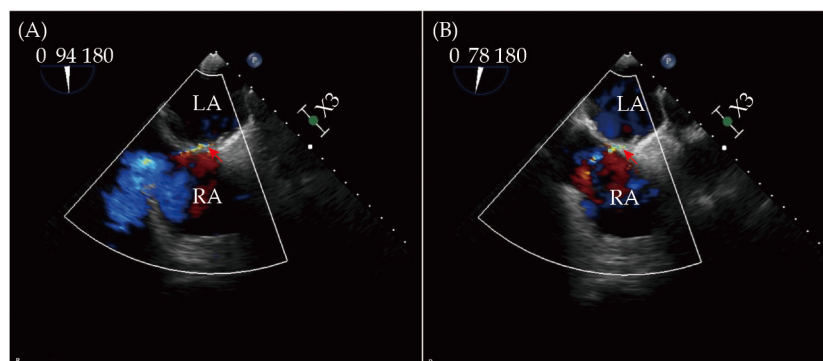


Figure 1 Two-dimensional transesophageal echocardiographic imaging (A & B): marked left-to-right shunt (red arrows) through a patent foramen ovale on a color flow Doppler is shown. LA: left atrium; RA: right atrium.

Table 1 Cardiac catheterization findings before and after atrial balloon septostomy.

Parameters	Prior to septostomy	After septostomy
Heart rate, beats/min	85	70
Mean right atrium pressure, mmHg	7	11
RV pressures (systolic/diastolic), mmHg	67/5	50/10
Pulmonary artery pressures (systolic/diastolic/mean), mmHg	70/16/34	50/15/25
PCWP, mmHg	26	16
Mean LA pressure, mmHg	26	15
Left ventricle end-diastolic pressure, mmHg	10	9

LA: left atrium; PCWP: pulmonary capillary wedge pressure; RV: right ventricle.

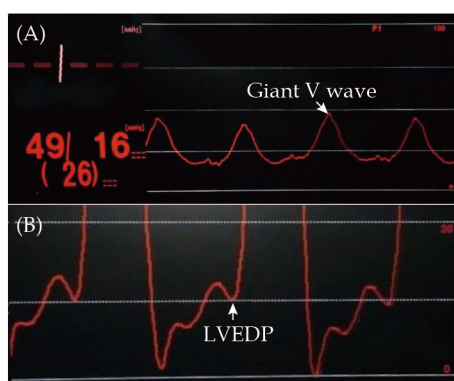


Figure 2 Elevated left atrial pressure with giant V waves in the left atrium pressure tracing (A), with normal LVEDP (B) is shown. LVEDP: left ventricular end-diastolic pressure.

no evidence of right-sided HF. Contrast-enhanced computed tomography angiography demonstrated the absence of both pulmonary embolism and pulmonary vein stenosis (PVS). A large V waves on PCWP or LA pressure tracings in the absence of significant valve disease, heart failure with preserved ejection fraction (HFpEF) or PVS is consistent with severely depressed LA compliance because of stiff LA syndrome. Given the lack of response to optimal medical therapy and as symptoms we-

re difficult to manage medically, an interventional course was pursued, and the patient underwent atrial septostomy using a 6 mm × 10 mm peripheral percutaneous transluminal angioplasty balloon, 8 mm × 20 mm peripheral cutting balloon and 15 mm × 40 mm Z-MED II non-compliant balloon, respectively (Figure 3A, Video 2). Balloon dilation was performed in a stepwise fashion under continuous hemodynamic monitoring. After each dilation, LA pressure, PCWP, pulmonary artery pressure, and RA pressure were reassessed. The procedure was terminated when a satisfactory reduction in LA pressure was achieved without an excessive increase in RA pressure, while maintaining stable systemic hemodynamics and oxygen saturation. No predefined numerical pressure threshold was used; rather, the endpoint was determined by the overall hemodynamic response and procedural safety. Following successful atrial septostomy, TEE demonstrated 5 mm × 6 mm iatrogenic atrial septal defect (ASD) after dilatation (Figure 3B, Video 3). 30 min after the atrial septostomy procedure, the mean LA pressure was found to be 15 mmHg which decreased by 42% (Table 1). The patient experienced immediate symptomatic improvement and was discharged two days after the procedure.

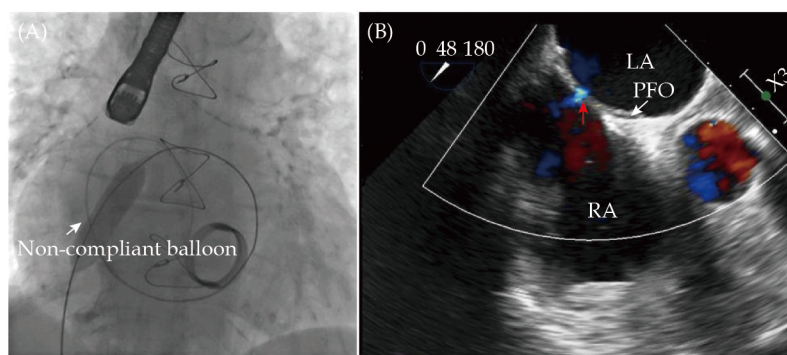


Figure 3 Atrial septostomy procedure using a non-compliant balloon (A) and two-dimensional transesophageal echocardiographic imaging after atrial septostomy procedure (B): left-to-right shunt (red arrow) through an iatrogenic atrial septal defect on a color flow Doppler is shown. LA: left atrium; PFO: patent foramen ovale; RA: right atrium.

During the 2-year follow-up, she remained clinically stable in New York Heart Association functional class II, with no re-hospitalization for HF and no evidence of right-sided HF. In addition, her diuretic therapy was successfully reduced, with the furosemide dose decreased from 120 mg/day to 40 mg/day and the spironolactone dose from 50 mg/day to 25 mg/day. TTE performed at the second year after the procedure demonstrated a functioning prosthetic mitral valve, mild aortic regurgitation, and a left ventricular ejection fraction of 55%–60%. Moderate tricuspid regurgitation was present, with an estimated PASP of 45 mmHg. Right ventricular (RV) dimensions and systolic functions were within normal limits (RV basal diameter: 38 mm, tricuspid annular systolic velocity: 13 cm/s, tricuspid annular plane systolic excursion: 19 mm). The inferior vena cava measured 18 mm in diameter with > 50% inspiratory collapse.

The SLAS (also called pulmonary hypertension with LA diastolic dysfunction or isolated LA hypertension) is defined as the combination of HF symptoms and/or pulmonary hypertension and large V wave in the PCWP or LA pressure tracings in the absence of alternative explanations such as a significant mitral valve pathology, HFpEF and PVS.^[1,2] In the present case, the markedly elevated V wave in the PCWP tracing with normal left ventricular end-diastolic pressure makes HFpEF diagnosis less likely. Prosthetic mitral valve dysfunction was excluded by either TTE or TEE. Cardiac computed tomography was normal, and no significant pressure gradient was observed between the PCWP and the LA pressure, ruling out PVS. Despite the fact that we have not proven the presence of atrial fibrosis by cardiac magnetic resonance imaging, the combination of the patient's history of MVR and surgical ablation, non-invasive findings, and the hemodyn-

amic assessment (Figure 4) were highly suggestive of the diagnosis of a fibrotic non-compliant LA in our case.

SLAS was first described in 1988 by Pilote, *et al.*^[3] as a complication after MVR for longstanding mitral valve diseases. They described this syndrome in a patient with pulmonary edema and intractable HF seven years after MVR. In recent years, it has also been recognized as a potential sequela of catheter ablation or surgical maze procedures (surgical ablation) for atrial arrhythmias, and approximately 1.4% of patients undergoing radiofrequency catheter ablation for AF developed SLAS due to extensive LA scarring,^[1] thus, gained significant prominence. Severe LA scarring, obstructive sleep apnea syndrome, diabetes mellitus, small LA (≤ 45 mm) and high LA pressure are clinical variables that predict the development of this syndrome.^[1,4,5] In our case, several important factors were supposed to be superimposed, and this ultimately resulted in a non-compliant LA. Firstly, chronic pressure overload and overstretch imposed on the LA myocardium due to chronic AF and longstanding severe mitral valve stenosis presumably resulted in considerable LA fibrosis. In addition to the preoperative LA fibrosis, an extensive Cox-Maze IV procedure appeared to precipitate fibrotic derangement further. Finally, surgical appendage ligation would further reduce LA compliance because the LA appendage is proposed to be a compliant portion with great reservoir capacity, protecting from elevation in LA internal pressure.

In stiff LA syndromes, the walls of the LA, which have reduced elasticity, are unable to expand and adapt to the excessive volume load on the LA during ventricular systole, especially during exercise, and LA pressure increases excessively.^[4] Thus, the main rationale for the treatment of stiff LA syndromes should be to reduce LA volume. Although diure-



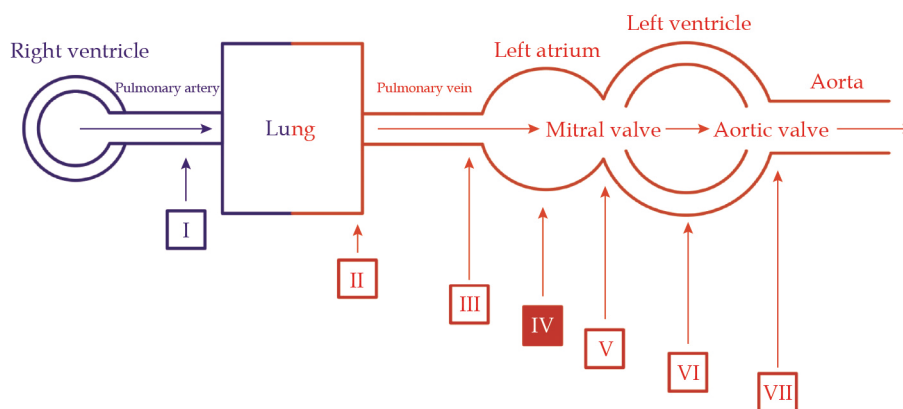


Figure 4 Schematic representation of the anatomic levels of 'obstruction' (A-G) in different forms of pulmonary hypertension, each characterized by a distinct hemodynamic profile (modified from Maeder, *et al.*^[6]). (I) Pulmonary arterial hypertension or pulmonary hypertension associated with lung disease: high mPAP, low mPAWP, and low LVEDP; (II) pulmonary veno-occlusive disease: high mPAP, normal mPAWP, and low/normal LVEDP; (III) pulmonary vein stenosis: high mPAP, high mPAWP, low/normal LVEDP, normal left atrial size, and evidence of pulmonary vein stenosis; (IV) (Present situation, red), stiff left atrium: high mPAP, high mPAWP, low/normal LVEDP, dilated left atrium, normal mitral valve, and exclusion of pulmonary vein stenosis; (V) mitral stenosis: high mPAP, high mPAWP, low/normal LVEDP, dilated left atrium, and abnormal mitral valve; (VI) heart failure with reduced or preserved ejection fraction: high mPAP, high mPAWP, high LVEDP, and no aortic valve pathology; and (VII) aortic stenosis: high mPAP, high mPAWP, high LVEDP, and aortic valve stenosis. LVEDP: left ventricular end-diastolic pressure; mPAP: mean pulmonary artery pressure; mPAWP: mean pulmonary artery wedge pressure.

tics have long been used to reduce LA volume,^[1] since left ventricular filling pressures are normal or near normal, yet pulmonary artery and venous pressures are elevated owing to the increased V wave amplitude,^[6] treatment for stiff LA syndrome is challenging, and diuretics are often proved to be unsuccessful in improving symptoms and generally results in intractable HF.^[1,25] Pulmonary vasodilators have also been employed but have not been formally studied.^[5] Therefore, new treatment methods are needed in cases where diuretic treatment is inadequate or not tolerated. LA volume can be reduced by diverting to the RA by percutaneous atrial septostomy through a new small defect in the interatrial septum (iatrogenic ASD – 'neo-ASD'). The concept for this approach comes from the observation that patients with mitral stenosis and an ASD fair better than those with isolated mitral stenosis. It has also been shown that in the presence of a significant 'post-puncture ASD' developed after catheter ablation for AF in some patients with SLAS, LA decompensation is less frequent and these patients are oligosymptomatic despite impressive hemodynamics.^[6] On the other hand, interatrial shunt devices have been shown to successfully reduce the increased LA pressures in patients with HFpEF by creating an ASD.^[7]

In the vast majority of cases of stiff LA syndrome reported in the literature, diuretic treatment was used to improve symptoms. In addition, heart transplantation was performed in some patients in whom diuretic treatment was insufficient to

reduce symptoms.^[8] Although interatrial shunt devices have been shown to reduce LA pressures in patients with HFpEF successfully, there are no studies on the efficacy of atrial septostomy in the treatment of patients with SLAS with HF symptoms refractory to diuretic therapy. To date, the literature has reported only a limited number of cases (Table 2) in which atrial septostomy was performed to reduce LA volume by redirecting blood flow to the RA through a newly created small interatrial defect, owing to inadequate symptom control with diuretic therapy; in these cases, patients experienced significant improvement in HF symptoms.^[5,9] In addition, another case of SLAS has been reported in which repeat cardiac catheterization was required, and an atrial septal stent (16-mm IntraStent Mega LD balloon-expandable biliary stent) was implanted one week after the initial atrial septostomy due to spontaneous closure of the defect, resulting in marked clinical improvement and no evidence of right-sided HF at 12-month follow-up.^[10]

In present case, atrial septostomy was performed using a stepwise balloon dilation strategy, with balloon sizes tailored according to the patient's hemodynamic response during the procedure. This approach aimed to optimize LA decompression while minimizing the risk of excessive interatrial shunting. During follow-up, patients were closely monitored for potential complications, such as clinical and hemodynamic deterioration, with particular attention to right-sided volume ov-

Table 2 Reported case reports of stiff left atrial syndrome treated with atrial septostomy or interatrial stenting.

Study	Age/Sex	Prior procedure	Clinical features	Initial cardiac catheterization findings	Cardiac catheterization findings after atrial septostomy	Cardiac catheterization findings after interatrial stenting	Follow-up course
Chandrasekar, <i>et al.</i> ^[5]	71-year-old Male	Paroxysmal AF/atrial flutter treated with 3 ablation procedures in the preceding 9 years	Dyspnea	Mean RA pressure: 10 mmHg Mean PAP: 39 mmHg PVR: 3.3 Wood units LA pressure at mid C wave: 16 mmHg LV end-diastolic pressure: 16 mmHg A giant LA V wave: 39 mmHg	LA V wave: 20 mmHg		Four months later, he continued to report symptomatic improvement. One year after discharge, symptoms remained minimal
Zaidi, <i>et al.</i> ^[10]	73-year-old Female	Mitral valve annuloplasty ring for regurgitation 11 years prior, three LA ablations for atrial arrhythmias in the preceding 10 years	NYHA Class III-IV dyspnea; lower extremity swelling; and recurrent bilateral, transudative pleural effusions refractory to diuresis	Mean RA pressure: 4 mmHg Mean PAP: 33 mmHg PCWP: 25 mmHg Mean LA pressure: 17 mmHg LA-RA gradient: 13 mmHg LV end-diastolic pressure: 9 mmHg PVR: 2.12 Wood units	Mean RA pressure: 7 mmHg LA pressures: a-9 mmHg, v-16 mmHg, mean: 8 mmHg (v-27 after 8 mm balloon → v-16 after 12 mm balloon) LA-RA gradient: 13 → 1 mmHg	Mean RA pressure: 4 mmHg LA pressures: a-8 mmHg, v-15 mmHg, mean: 8 mmHg (v-40 mmHg before stent → v-25 mmHg after stent → v-15 mmHg after stent dilation) LA-RA gradient: 12 → 8 → 4 mmHg	Significant clinical improvement and no right HF after twelve months
Aslam, <i>et al.</i> ^[1]	Mean age of 66.5-year-old Male (45%) and Female (55%)	AF (63.6% experiencing paroxysmal AF) treated with ablation procedure.		Mean LA pressure: 18.6 mmHg Mean LA V-wave pressure: 35.4 mmHg Mean RA pressure: 7.9 mmHg	Mean LA pressure: 16.4 mmHg ($P=0.01$) Mean LA V-wave pressure: 30 mmHg ($P=0.02$) Mean RA pressure: 10.8 mmHg ($P=0.02$)		Marked symptom improvement was seen after undergoing atrial septostomy, as evidenced by a decrease in mean NYHA class from 3 to 1.6

AF: atrial fibrillation; HF: heart failure; LA: left atrium; LV: left ventricle; NYHA: New York Heart Association; PAP: pulmonary arterial pressure; PCWP: pulmonary capillary wedge pressure; PVR: pulmonary vascular resistance; RA: right atrium.

erload. Serial TTE evaluations were used to assess RV dimensions, systolic function, and the severity of tricuspid regurgitation. We believe that the present case report and literature review will be helpful and salutary for the progress of the literature and will contribute to increasing physician awareness that atrial septostomy may provide symptomatic benefits in patients with SLAS who fail optimal medical therapy.

ACKNOWLEDGMENTS

This research did not receive any specific grant from funding agencies. All authors had no conflicts of interest to disclose.

REFERENCES

- [1] Gibson DN, Di Biase L, Mohanty P, *et al.* Stiff left atrial syndrome after catheter ablation for atrial fibrillation: clinical characterization, prevalence, and predictors. *Heart Rhythm* 2011; 8: 1364–1371.
- [2] Senda A, Matsumoto K, Hoshijima M, *et al.* Lessons from a case with stiff left atrial syndrome complicated by iatrogenic inter-atrial shunts: a case report. *Eur Heart J Case Rep* 2023; 7: ytaad306.
- [3] Pilote L, Hüttner I, Marpole D, *et al.* Stiff left atrial syndrome. *Can J Cardiol* 1988; 4: 255–257.
- [4] Reddy YNV, El Sabbagh A, Packer D, *et al.* Evaluation of shortness of breath after atrial fibrillation ablation: is there a stiff left atrium? *Heart Rhythm* 2018; 15: 930–935.
- [5] Chandrasekar P, Park JY, Al-Hijji MA, *et al.* Atrial septostomy to treat stiff left atrium syndrome. *Circ Heart Fail* 2017; 10: e004160.
- [6] Maeder MT, Nägele R, Rohner P, *et al.* Pulmonary hypertension in stiff left atrial syndrome: pathogenesis and treatment in one. *ESC Heart Fail* 2018; 5: 189–192.
- [7] Shah SJ, Feldman T, Ricciardi MJ, *et al.* One-year safety and clinical outcomes of a transcatheter interatrial shunt device for the treatment of heart failure with preserved ejection fraction in the reduce elevated left atrial pressure in patients with heart failure (REDUCE LAP-HF I) trial: a randomized clinical trial. *JAMA Cardiol* 2018; 3: 968–977.
- [8] Kronenberger R, Tanaka K, de Asmundis C, *et al.* Stiff left atrial syndrome with pulmonary veins occlusion after percutaneous radiofrequency ablation: a life-long complication that can lead to heart transplantation. *J Cardiothorac Surg* 2023; 18: 181.



- [9] Aslam FN, Schneider A, Sabbagh AE. Atrial balloon septostomy for stiff left atrial syndrome secondary to catheter ablation: a case series. *J Am Coll Cardiol Interv* 2024; 17: S79–S80.
- [10] Zaidi D, Williams D, Zhao D, *et al*. Interatrial stent to treat stiff left atrium syndrome. *Cardiovasc Revasc Med* 2022; 40S: 337–340.

Please cite this article as: Eren M, Velibey Y, Kahraman E. Atrial septostomy as a therapeutic option in stiff left atrial syndrome. *J Geriatr Cardiol* 2026; 23(8): 532–537. DOI: 10.26599/1671-5411.2026.08.005

