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Percutaneous closure of an iatrogenic ventricular septal defect associated with transcatheter aortic valve implantation

Transkateter aort kapak implantasyonu sonrasında görülen iatrojenik ventriküler septal defektin perkütan kapatılması

CASE REPORT
OLGU SUNUMU

ABSTRACT

An 81-year-old man who had dyspnea was admitted to our hospital with a diagnosis of severe aortic stenosis. A transcatheter aortic valve implantation was successfully performed with a 29 mm Edwards Sapien XT valve using a transfemoral access. After the procedure, the echocardiography showed a restrictive ventricular septal defect (VSD) in the membranous septum. As the patient had no symptoms, it was decided to follow him up conservatively. However, he was readmitted within three weeks with symptoms suggestive of biventricular failure. A control echocardiography revealed a membranous VSD, 8 mm in size, right chambers dilatation with moderate tricuspid regurgitation, and systolic pulmonary artery pressure of 60 mm Hg. The previously deployed aortic valve was normal in function. The decision to perform a percutaneous VSD closure was made. The defect was then closed with a 10 mm muscular VSD occluder. During and after the procedure, there was no dysfunction in the bioprosthetic aortic valve. At the one-year follow-up, the patient was still asymptomatic.

Keywords: Iatrogenic disease, ventricular septal defect, transcatheter aortic valve replacement

ÖZET

81 yaşında erkek hasta nefes darlığı şikayeti ile başvurduğu hastanemize ileri aort darlığı tanısıyla yatırıldı. Transfemoral yolla, 29 mm Edwards Sapien XT kapakçığı başarıyla implante edildi. İşlemden sonra, ekokardiyografide membranöz septumda restriktif bir ventriküler septal defekt (VSD) görüldü. Hastanın semptomu olmadığı için konservatif takip kararı verildi. Ancak hasta 3 hafta sonra biventriküler yetersizlik düşündüren nefes darlığı ve periferik ödem şikâyetleriyle tekrar başvurdu. Kontrol ekokardiyografisinde 8 mm boyutunda membranöz VSD, orta derecede triküspit yetersizliği ile sağ kalp boşluklarında dilatasyon ve 60 mm Hg sistolik pulmoner arter basıncı görüldü. Önceden yerleştirilmiş biyoprotez aort kapağı fonksiyone idi. Hastaya perkütan VSD kapatma kararı verildi. Defekt 10 mm'lik bir VSD Occluder ile kapatıldı. İşlem sırasında ve sonrasında biyoprotetik aort kapağında herhangi bir işlev bozukluğu olmadı. Bir yıllık takipte hasta hala asemptomatikti.

Anahtar Kelimeler: İyatrojenik, ventriküler septal defekt, transkateter aortik kapak replasmanı

Transcatheter aortic valve replacement (TAVR) has emerged as an alternative treatment for patients with severe, symptomatic aortic stenosis who have a high surgical risk.[1] The data from a multicenter, randomized trial have also recently supported TAVR as an alternative to surgery in low-risk patients.[2] Evidence-based clinical data have led clinicians to expand this procedure to nearly all patients who have severe aortic stenosis. Technological improvements, increased operator experience, and pro-

Göktuğ Savaş M.D.[1]
Selçuk Yazıcı M.D.[1]
Özgür Kılıç M.D.[2]
Sait Terzi M.D.[1]

[1]Department of Cardiology, Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Turkey
[2]Department of Coronary Care Unit, Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Turkey

Corresponding Author:
Göktuğ Savaş
✉ goktug_savas@hotmail.com

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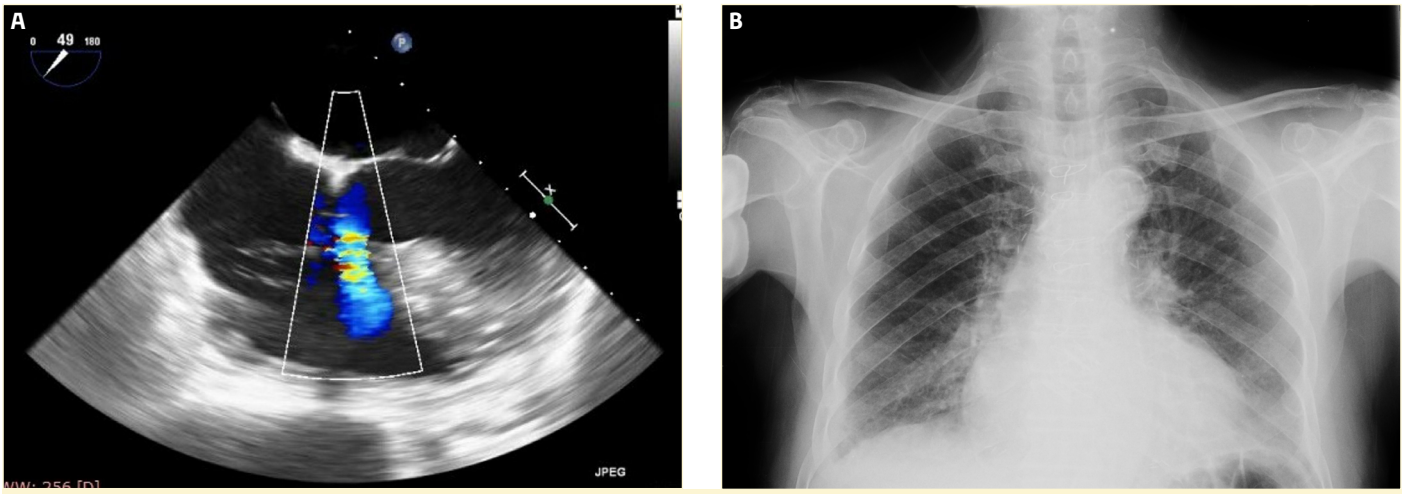

Figure 1. (A) Transesophageal echocardiographic view of the ventricular septal defect. (B) Chest radiography showed pulmonary congestion.

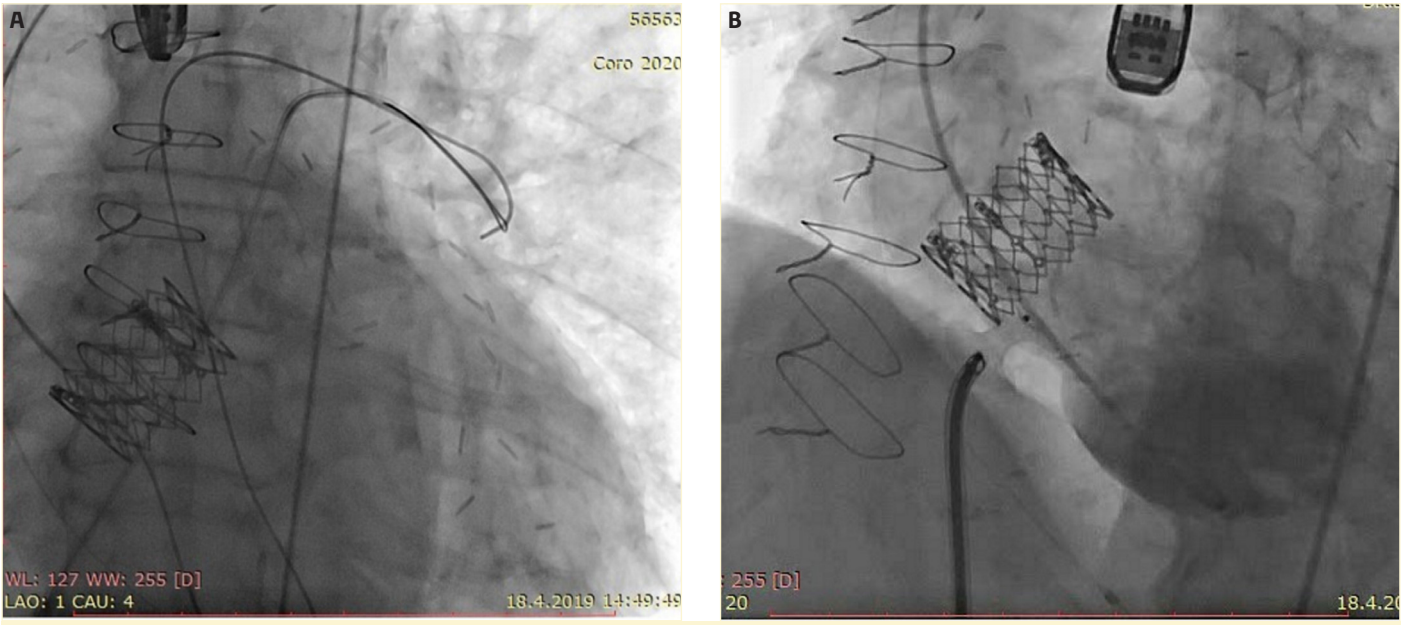

Figure 2. (A) Wire captured with snare in the pulmonary artery. (B) Defect closure with a 10 mm muscular VSD occluder.

cedural simplification have contributed to decreased complication rates in TAVR; however, broader use of this procedure in patients with aortic stenosis who often have coexisting conditions has resulted in an increased cumulative number of complications, such as stroke, major vascular complications, coronary occlusion, and aortic annular rupture or ventricular septal defect (VSD).[3,4]

Diagnosing, preventing, and treating these procedure-related complications have gained importance, and more patients with severe aortic stenosis now undergo TAVR than surgical aortic valve replacement. Here, we describe the case of an 81-year-old patient with iatrogenic VSD following TAVR who went on to have a successful percutaneous VSD closure.

ABBREVIATIONS

LVOT	Left ventricular outflow tract
STS	Society of Thoracic Surgeons
TAVR	Transcatheter aortic valve replacement
VSD	Ventricular septal defect

CASE REPORT

An 81-year-old man with a history of coronary bypass surgery, hypertension, and diabetes mellitus was admitted with dyspnea. As the New York Heart Asso-

Table 1. Clinical features of reported cases

Publication Author/ Year	Age/Sex	Comorbidities	STS/Logistic Euro Score (%)	Annulus Size - CT (mm)	LVOT Calcification Severity	Prosthesis	Valve type/Size of valve	Access	Predilatation/ Postdilatation (mm)
Tzikas A et al. 2009[4]	89/M	None	NR; 15	NR	NR	Self- expandable	Core Valve /29-mm	transfemoral	23/25
Al-Attar N et al. 2009[4]	NR	NR	NR	NR	NR	NR	NR	transapical	NR/NR
Massabuau P et al. 2011[4]	86/M	CAD, HLD	7;28	NR	NR	Balloon-expandable	Edwards Sapien/ 26-mm	transapical	NR/NR
Martinez R et al. 2012[4]	76/F	COPD,HTN	NR;17	NR	moderate	Self- expandable	Core Valve /26-mm	NR	22/25
Gerckens U et al. 2013[15]	86/M	AF,CKD, HF	NR; 21	27,2	severe	Self- expandable	Core Valve /31-mm	transfemoral	22/28
Aminian A et al. 2013[4]	85/F	CAD, CKD	NR; 18.5	NR	NR	Balloon-expandable	Edwards Sapien XT/ 23-mm	transfemoral	20/None
Rodgers V et al. 2014[4]	82/F	NR	NR	17x20	severe	Balloon-expandable	Edwards Sapien XT/ 23-mm	transfemoral	18/None
Baruteau AE et al. 2014[4]	89/M	NR	NR	NR	NR	Balloon-expandable	Edwards Sapien/ 26-mm	NR	NR/NR
Salizzoni S et al. 2014[4]	88/F	HF	10.9; 26.64	NR	severe	Balloon-expandable	Edwards Sapien XT/ 26-mm	transfemoral	20/25
Yanes-Bowden G et al. 2014[4]	87/M	None	NR; 13.92	21x23	NR	Balloon-expandable	Edwards Sapien XT/ 26-mm	transfemoral	NR/NR
Patel Y et al. 2014[4]	85/F	HTN,CAD, HLD	>10; NR	NR	NR	Balloon-expandable	Edwards Sapien XT/ 23-mm	transfemoral	20/None
Patel Y et al. 2014[4]	89/F	HTN,CAD, HLD,DM	16; NR	NR	NR	Balloon-expandable	Edwards Sapien/ 26-mm	transfemoral	22/None
Patel Y et al. 2014[4]	88/F	HTN, CKD, AF	16; NR	NR	NR	Balloon-expandable	Edwards Sapien/ 23-mm	transfemoral	20/None
Garrido JM et al. 2014[4]	79/F	HTN, AF, COPD	NR;11.66	NR	moderate	Balloon-expandable	Edwards Sapien XT/ 23-mm	transfemoral	NR/NR
Mauri L et al. 2014[16]	78/F	HF	NR;NR	NR	NR	Balloon-expandable	Edwards Sapien XT/ 23-mm	transfemoral	NR/NR
Dursun H et al. 2015[4]	73/F	NR	NR;33.8	NR	mild	Balloon-expandable	Edwards Sapien XT/ 26-mm	transfemoral	23/None
Levi DS et al. 2015[4]	57/F	MVR	NR;NR	NR	NR	Balloon-expandable	Edwards Sapien/ 23-mm	transapical	20/NR
Shakoor MT et al. 2015[4]	89/M	COPD,CAD, Aorta Valvuloplasty	NR;NR	26x28	NR	Balloon-expandable	Edwards Sapien/ 29-mm	transfemoral	NR/NR
Mark SD et al. 2015[4]	89/M	NR	5.6;NR	NR	NR	Balloon-expandable	Edwards Sapien/ 29-mm	NR	NR/NR
Lee CW et al. 2015[4]	80/F	COPD	NR;NR	NR	NR	Balloon-expandable	Edwards Sapien/ 23-mm	NR	NR/NR

Table 1. Clinical features of reported cases (continued)

Publication Author/ Year	Age/Sex	Comorbidities	STS/Logistic Euro Score (%)	Annulus Size - CT (mm)	LVOT Calcification Severity	Prosthesis	Valve type/Size of valve	Access	Predilatation/ Postdilatation (mm)
Rene AG et al. 2016[6]	89/M	AF	5.7;NR	24x30	severe	Balloon-expandable	Edwards Sapien/ 29-mm	transfemoral	NR/NR
Rene AG et al. 2016[6]	86/F	COPD,HTN, CKD	4.8;NR	23x28	moderate	Balloon-expandable	Edwards Sapien XT/ 29-mm	transfemoral	NR/NR
Rene AG et al. 2016[6]	82/F	COPD, CAD, AF	14.2;NR	26.2x29	severe	Balloon-expandable	Edwards Sapien XT/ 29-mm	transaortic	NR/NR
Rene AG et al. 2016[6]	93/M	CKD, HF,AF, HTN	9.37;NR	18.6x27	severe	Balloon-expandable	Edwards Sapien XT/ 26-mm	transfemoral	NR/NR
Sardar MR et al.2017[4]	87/M	AF, CAD, CKD	6.1;17.5	23.6x29	mild	Balloon-expandable	Edwards Sapien XT/ 29-mm	transfemoral	22/None
Sardar MR et al.2017[4]	90/F	AF	8.1;15.8	23x25	severe	Balloon-expandable	Edwards Sapien/ 26-mm	transfemoral	22/None
Ohlow MA et al.2017[9]	83/F	HF	NR;27	NR	severe	Balloon-expandable	Edwards Sapien XT/ 26-mm	transfemoral	NR/NR
Almanfi A et al. 2017[8]	92/M	CAD,HF, obesity	NR;NR	NR	NR	Balloon-expandable	Edwards Sapien XT/ 29-mm	transfemoral	NR/NR
Zeniou V et al.2018[7]	83/F	NR	NR;NR	NR	severe	Balloon-expandable	Edwards Sapien XT/ 26-mm	NR	NR/NR
Zeniou V et al.2018[7]	87/F	NR	NR;NR	NR	severe	Self- expandable	Core Valve /26-mm	NR	NR/NR
Zeniou V et al.2018[7]	82/M	NR	NR;NR	NR	moderate	Balloon-expandable	Edwards Sapien/ 29-mm	NR	NR/NR
Zeniou V et al.2018[7]	79/M	NR	NR;NR	NR	severe	Self- expandable	Core Valve Evolut/26-mm	NR	NR/NR
Zeniou V et al.2018[7]	84/F	NR	NR;NR	NR	severe	Self- expandable	Core Valve Evolut/26-mm	NR	NR/NR
Zeniou V et al.2018[7]	81/M	NR	NR;NR	NR	severe	Balloon-expandable	Edwards Sapien/ 29-mm	NR	NR/NR
Ancona M et al. 2018[10]	87/F	CKD	8;NR	NR	NR	Self- expandable	Core Valve Evolut/29-mm	NR	None/24
Chourdakis E et al. 2018[11]	86/M	AF, HTN, HF, CKD	NR;41	NR	NR	Balloon-expandable	Edwards Sapien/ 29-mm	transfemoral	20/None
Kooistra NHM et al. 2018[12]	90/F	HTN, AF, DM, CKD	NR;3.74	22.9x26.5	NR	Self- expandable	Core Valve /29-mm	NR	25/None
Our Case	81/M	HTN, DM, CAD	NR;25.4	24x26	severe	Balloon-expandable	Edwards Sapien XT/ 29-mm	transfemoral	25/None

* NR: not reported, HTN: Hypertension, AF: Atrial Fibrillation,CAD: Coronary artery Disease, CKD: Chronic Kidney Disease, COPD: Chronic Obstructive Pulmonary Disease,HF: Heart Failure, DM: Diabetes Mellitus,HLD: Hyperlipidemia, MVR:Mitral Valve Replacement

Table 2. Clinical characteristics and follow-up data of reported cases

Publication Author/ Year	Symptoms	Time of Diagnosis	VSD location	VSD shunt	VSD size	VSD severity (Qp/Qs or RVSP)	Treatment	Follow up
Tzikas A et al. 2009[4]	NR	Peri-procedural	Membranous	LV to RV	NR	NR	conservative	at 12 days still alive
Al-Attar N et al. 2009[4]	NR	Postmortem	NR	NR	small	NR	NR	death (at hospital)
Massabuau P et al. 2011[4]	Dyspnoea	12 months	Apical	LV to RV	NR	RVSP=65 mm Hg	conservative	at one year still alive
Martinez R et al. 2012[4]	Heart failure	Peri-procedural	Membranous	LV to RV	small	NR	conservative	at one year still alive
Gerckens U et al. 2013[15]	Clinical deterioration	7 days	Peri-membranous	LV to RV	NR	RVSP=65 mm Hg	percutaneous VSD closure	at 6 months still alive
Aminian A et al. 2013[4]	Dyspnoea	6 weeks	Membranous	LV to RV	small	mild	conservative	at 6 months still alive
Rodgers V et al. 2014[4]	Heart failure	10 days	Peri-membranous	LV to RA	NR	Qp/Qs ratio=2.0	conservative	at 9 months still alive
Baruteau AE et al. 2014[4]	Clinical deterioration	6 months	Peri-membranous	LV to RV	NR	Qp/Qs ratio=2.0	percutaneous VSD closure	at 2 years still alive
Salizzoni S et al. 2014[4]	NR	7 days	Atrio-ventricular septum	LV to RA	NR	Qp/Qs ratio=2.0	conservative	death (at 35 th day)
Yanes-Bowden G et al. 2014[4]	Asymptomatic	Peri-procedural	Membranous	LV to RV	6 mm	Peak interventricular gradient 125 mm Hg	conservative	at 2 years still alive
Patel Y et al. 2014[4]	NR	Peri-procedural	Peri-membranous	LV to RV	NR	NR	conservative	at one month still alive
Patel Y et al. 2014[4]	Asymptomatic	Peri-procedural	Peri-membranous	LV to RV	small	NR	conservative	death (at hospital)
Patel Y et al. 2014[4]	Asymptomatic	Peri-procedural	LVOT	LV to RA	NR	39 mm Hg gradient	conservative	alive (NR)
Garrido JM et al. 2014[4]	Asymptomatic	Peri-procedural	Membranous	LV to RV	1x2mm	severe	surgery	at 9 months still alive
Mauri L et al. 2014[16]	Dyspnoea	3 weeks	sub aortic	LV to RV	10x15mm	Qp/Qs ratio=3.0	percutaneous VSD closure	at 6 months still alive
Dursun H et al. 2015[4]	Asymptomatic	4 days	Membranous	LV to RV	5mm	Qp/Qs ratio=1.5	percutaneous VSD closure	at 9 months still alive
Levi DS et al. 2015[4]	Dyspnoea	Few days	Peri-membranous	LV to RV	8x5 mm	NR	percutaneous VSD closure	at 3 months still alive
Shakoor MT et al. 2015[4]	Dyspnoea	30 days	Membranous	LV to RV	small	NR	conservative	death (few days later)
Mark SD et al. 2015[4]	Dyspnoea	Peri-procedural	Membranous	LV to RV	NR	Qp/Qs ratio=1.6	percutaneous VSD closure	alive (NR)
Lee CW et al. 2015[4]	NR	Peri-procedural	Peri-membranous	LV to RV	small	NR	conservative	at 2 months still alive
Rene AG et al. 2016[6]	Dyspnoea	1 day	LVOT	LV to RV	NR	Qp/Qs ratio=2.3	percutaneous VSD closure	death (four months after)

Table 2. Clinical characteristics and follow-up data of reported cases (continued)

Publication Author/ Year	Symptoms	Time of Diagnosis	VSD location	VSD shunt	VSD size	VSD severity (Qp/Qs or RVSP)	Treatment	Follow up
Rene AG et al. 2016[6]	Asymptomatic	Peri-procedural	Peri-membranous	LV to RV	NR	124 mm Hg gradient	conservative	death (at one month)
Rene AG et al. 2016[6]	Dyspnoea	3 days	Peri-membranous	LV to RV	NR	Qp/Qs ratio=1.6	percutaneous VSD closure	at 14 months still alive
Rene AG et al. 2016[6]	Asymptomatic	Peri-procedural	Peri-membranous	LV to RV	small	94 mm Hg gradient	conservative	at one month still alive
Sardar MR et al.2017[4]	Ventricular tachycardia	9 days	Membranous	LV to RV	4 mm	NR	conservative	at 6 months still alive
Sardar MR et al.2017[4]	Clinical deterioration	Peri-procedural	Membranous	LV to RV	large	severe	valve in valve	death on the third day
Ohlow MA et al.2017[9]	Heart failure	Peri-procedural	Membranous	LV to RV	NR	Qp/Qs ratio=1.7	percutaneous VSD closure	At 16 day still alive
Almanfi A et al. 2017[8]	Asymptomatic	1 day	Aortic root-RV	Aorta to RV	small	NR	conservative	at one year still alive
Zeniou V et al.2018[7]	NR	NR	Membranous	LV to RV	<2 mm	140 mm Hg gradient	conservative	at one year still alive
Zeniou V et al.2018[7]	NR	NR	Membranous	LV to RV	small	minimal	conservative	at one year still alive
Zeniou V et al.2018[7]	NR	NR	Membranous	LV to RV	4 mm	130 mm Hg gradient	conservative	at one year still alive
Zeniou V et al.2018[7]	NR	NR	Muscular-near the apex	LV to RV	NR	66.5 mm Hg gradient	conservative	at one year still alive
Zeniou V et al.2018[7]	NR	NR	Muscular-near the apex	LV to RV	6 mm	88 mm Hg gradient	conservative	at one year still alive
Zeniou V et al.2018[7]	NR	NR	Membranous	LV to RV	NR	64 mm Hg gradient	conservative	at one year still alive
Ancona M et al. 2018[10]	Clinical deterioration	Peri-procedural	İnterventricular septum	LV to RV	10 mm	NR	surgery	at 14 day still alive
Chourdakis E et al. 2018[11]	Asymptomatic	Peri-procedural	Aortic root-RV	Aorta to RV	small	Qp/Qs ratio=1, 75 mm Hg gradient	conservative	at 25 day still alive
Kooistra NHM et al. 2018[12]	Heart failure	12 days	LVOT	LV to RV	5 mm	64 mm Hg gradient	percutaneous VSD closure	at one year still alive
Our Case	Heart failure	3 weeks	Membranous	LV to RV	8 mm	100 mm Hg gradient, Qp/Qs ratio=1.6	percutaneous VSD closure	at one year still alive

* NR: not reported, LVOT:left ventricular outflow tract, LV: left ventricular, RV: right ventricular

ciation Class III symptoms and systolic murmur in the aortic area were suggestive of an aortic stenosis, the patient underwent a transthoracic echocardiography that revealed decreased left ventricular ejection fraction (30% by Modified Simpson's rule) and severe aortic stenosis with peak and mean aortic valve gradients of 62 mm Hg and 41 mm Hg, respectively. The calculated aortic valve area was 0.89 cm^2. The mean diameter of the aortic annulus was 26 mm by multi-slice computer tomography. The subsequent coronary angiogram revealed that all bypass grafts were still patent. His calculated Society of Thoracic Surgeons (STS) risk score was 25.4%. The patient was evaluated by the heart team and referred for TAVR.

The procedure was performed using a transfemoral access. The aortic valve was pre-dilated with a ZMED II 25 mm×5 cm×100 cm valvuloplasty balloon. A 29 mm Edwards Sapien XT valve was deployed during rapid ventricular pacing. Post-procedural echocardiography showed a well-seated aortic valve with a mild paravalvular leak, a peak transvalvular gradient of 9 mm Hg, and a new VSD on the membranous septum (Figure 1A). The defect was 8 mm in size, and there was a shunt directed from the left to the right ventricle with a peak systolic velocity of 5 m/s across the defect. The decision to manage the patient conservatively was made by the heart team. Subsequently, the patient showed symptomatic improvement and was discharged home on medical therapy after five days.

Three weeks after discharge, he was admitted with symptoms suggestive of biventricular failure (shortness of breath and peripheral edema). On examination, he was found to be in marked respiratory distress with oxygen saturation of 85%. The chest radiography showed pulmonary congestion (Figure 1B). The transthoracic echocardiography revealed membranous VSD, 8 mm in size, dilatation of the right chambers with moderate tricuspid regurgitation, and systolic pulmonary artery pressure of 60 mm Hg. The previously deployed aortic valve was normal in function. A subsequent transesophageal echocardiography confirmed the VSD (Video 1*). Cardiac catheterization revealed a large VSD with pulmonary to systemic blood flow ratio (Qp/Qs) of 1.6 (Video 2*). The decision to perform a percutaneous VSD closure was made. Under general anesthesia, the right femoral artery and vein were cannulated. The defect was crossed from the LV side with a Judkin's 6 Fr right catheter and Terumo wire. This wire was advanced into the left pulmonary artery, snared, and externalized from the right femoral vein to form a wire rail (Figure 2A). A 7 Fr delivery catheter was advanced through the defect from the femoral vein to the aorta. A 10 mm VSD Occluder (AGA Medical Corp, Plymouth, MN, USA) was loaded onto its delivery system, and the distal tip of the device was partially unloaded in the ascending aorta. The entire delivery system was withdrawn until the device was placed at an appropriate site across the defect and then released (Figure 2B, Video 3*). The transesophageal echocardiography confirmed there was no residual shunting through the device. The patient reported pronounced improvement in his symptoms and was discharged home after three days. He remained stable at the one-year follow-up.

DISCUSSION

With favorable results from sequential randomized clinical trials, TAVR has been increasingly performed in nearly 66,000 patients annually in USA, and the occurrence of rare complications has become more apparent.[5] In retrospective analyses, iatrogenic VSD occurs in 0.4% to 1.5% following TAVR.[6-7] However, to date, only 37 cases of those have been published in English literature.[4-12] Owing to late or uncertain presentation and lack of suspicion, the diagnosis of VSD after TAVR in most patients is thought to have been missed. Here, we report a case of VSD after TAVR, the first event in a 220 case series performed at our center, which was successfully treated by percutaneous VSD closure. We have also sought to describe reported literature, the patient characteristics, possible risk factors, procedural details, and clinical outcomes. The clinical features of the reported cases are summarized in Tables 1 and 2.

The patients were between 57 and 93 years old with a median age of 86 years, and 60% of them were women. The mean STS and Logistic Euro Scores risk scores were 9.4±3.9, n=13 and 21.1±9.4, n=16, respectively. In patients for whom access site data were available (n=26), the femoral artery was the most widely (n=22, 84%) used access site for TAVR. The balloon-expandable valves were predominantly used than self-expandable valves (75% vs. 25%, respectively). Post-dilatation data were available for 16 patients where post-dilatation was performed in five of them (31%).

Several potential risk factors have been implicated for VSD following TAVR including elliptic aortic annulus; severe, asymmetric calcification of the aortic annulus; oversizing or higher placement of the valve; and direct trauma of the rigid wire.[13] In most cas-

es, it is thought to emerge by perforation of the interventricular septum after implantation of the rigid stent frame into the aortic annulus. The injury can be associated with a wide variety of manifestations including extension into the RV outflow tract and the right or left atria and focal rupture of the annulus underneath the left coronary artery in which an aorta-ventricular gap occurs.[4,8,13] The most vulnerable sections to injury can be predicted by assessing the severity and symmetry of calcification of the aorta and the adjacent structures.[13] In a previous study evaluating the variables that increase the risk for aortic root rupture in patients who had undergone TAVR, the authors reported that the risk of sub-annular rupture was high if a narrow and bulky calcification of the left ventricular outflow tract (LVOT) was present.[14]

After reviewing the previous cases, 19 patients had data on the grade of LVOT calcification of whom 13 (68.4%) had severe calcification. Both annulus and valve size were reported in 16 patients; however, oversizing had been detected in only three (18%) of them. The higher placement of the aortic valve was speculated to be a predisposing factor in only one case.[15]

The location of VSD was predominantly membranous (n=16, 42%) or peri-membranous (n=10, 26%), and most of those (n=15, 39%) were diagnosed during or immediately after the procedure. Nine patients were asymptomatic at the time of diagnosis; however, dyspnea was the most common symptom (n=14, 36%). Management was generally conservative (n=23, 60%). One patient was treated by valve in valve therapy and two were treated by open heart surgery.[4] In 11 (28%) patients, including our case, the preferred therapy was a percutaneous VSD closure, performed four days to six months following TAVR. An antegrade approach was used more than retrograde approach in patients treated with percutaneous closure (n=8 vs. n=3, respectively). Only in one patient with percutaneous closure, the femoral artery occluded during the procedure, and that patient was successfully treated with peripheral bypass surgery.[16] The rest of the patients who underwent percutaneous closure had no complications of either vascular access or valve dysfunction.

In previous reports, follow-up periods for patients diagnosed with VSD following TAVR were variable in length, 12 days to two years. Total mortality was 18% (n=7) of which most were reported as in-hospital mortality (n=4). At the end of the follow-up periods, median nine months, 81% of the patients were still alive. Mortality was 9% (n=1) in patients treated with percutaneous VSD closure.

In this case report, we present a patient with iatrogenic VSD, a rare complication following TAVR, who was successfully treated by percutaneous VSD closure. We also reviewed the reported literature and the patient characteristics, possible risk factors, procedural details, and clinical outcomes. It is, now, the current review on iatrogenic VSD following TAVR. The insights gained from this report are that most patients with iatrogenic VSD following TAVR were hemodynamically stable and did not require VSD closure. The management was generally conservative; however, percutaneous closure seemed to be an applicable strategy.

*Supplementary video files associated with this article can be found in the online version of the journal.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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