

Research Article

A Novel Approach for Transcatheter Management of Perimembranous Ventricular Septal Defect with a Subaortic Ridge

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Introduction. Surgical closure of the perimembranous ventricular septal defect (PM VSD) and resection of the subaortic ridge are the standard methods of management, but there is no definitive agreement regarding the timing of surgery. **Objectives.** To evaluate the safety and efficacy of the management of patients with PM VSD and subaortic ridge with or without AR via transcatheter closure of the defect and compressing the ridge against the ventricular septum using Amplatzer ductal occluder type I (ADO-I). **Patients and Methods.** We introduced a new approach for transcatheter management of PM VSD and subaortic ridge by closing the VSD and capturing or compressing the ridge against the interventricular septum (IVS) using the ADO-I device. Thirty-eight (9.5%) of 398 patients with a PM VSD were found to have subaortic ridge and were enrolled in this study from August 1, 2014, to February 1, 2018, at the Ibn Albitar Center for Cardiac Surgery, Baghdad, Iraq. **Results.** The ages and weights of patients ranged from 1.5 to 25 years and 7 to 73 kg, respectively. The male-to-female ratio was 2.2 : 1. The VSD sizes ranged from 4 to 8 mm, and the median distance of the ridge from the proximal edge of the VSD was 2.5 mm. Prior to closure, 13 patients (34.2%) had mild and mild-to-moderate aortic regurgitation (AR), and nine patients (23.7%) had mild-to-moderate left ventricular outflow tract (LVOT) obstruction. The mean AR pressure half-time increased significantly after intervention (from 385 ± 38 ms to 535 ± 69 ms (significant P value, 0.001)), and the mean of the peak pressure gradient across the LVOT decreased from 33 ± 7 mmHg to 15 ± 2.4 mmHg (significant P value, 0.001). Successful procedures were achieved in 33 patients (86.8%). **Conclusion.** Transcatheter management of patients with PM VSD and subaortic ridges with or without AR is feasible and effective.

1. Introduction

Subvalvular aortic stenosis represents approximately 15% of left ventricular outflow tract (LVOT) stenosis [1, 2]. Perimembranous ventricular septal defect (PMVSD) is found in about one-fifth of the cases of subvalvular aortic stenosis [1, 3, 4].

The subaortic ridge is defined as a ridge-like protrusion into the subaortic area from the crest of the ventricular septum that extended towards the mitral valve apparatus with or without Doppler evidence of obstruction [5, 6]. Any fibrous or membranous echo-dense protrusion into the

LVOT was considered by some authors as part of the spectrum of subaortic stenosis [6]. Subaortic ridge is best visualized in the parasternal long axis transthoracic echocardiographic view [1, 4, 6].

Subaortic ridge in patients with VSD is either caused by a fibrous, fibromuscular, or membranous ridge that is located at the lower border of the VSD or malalignment or deviation of the right ventricular outflow tract anteriorly or posteriorly [5, 7]. Posterior deviation occurs when the outlet septum is displaced posteriorly into the LVOT resulting in narrow LVOT and obstruction above the VSD in the majority of patients. Alternatively, anterior deviation occurs when the

outlet septum is displaced anteriorly into the right ventricle and is associated with aortic override and LVOT obstruction [7–9].

The progression of subaortic ridge in the presence of VSD is highly variable and unpredictable during childhood [1, 10–12]. Although progression is rapid and aggressive in some patients, others may exhibit slow and mild increase in LVOT gradient over the years, thereby mandating a prolonged follow-up of these patients [1, 11, 12].

Aortic regurgitation (AR) that occurs in cases of subaortic ridge and VSD may develop as a complication of VSD itself or due to progressive tethering and encroachment of the ridge onto the aortic valve leaflets. The frequency and progression of AR are variable depending on the age of the patient, anatomy of the ridge, and associated cardiac lesions [11, 13–15].

Surgical intervention is often required at some point in the clinical course of VSD and subaortic ridge. Surgery may be recommended in case of an LVOT pressure gradient >40 mmHg, LV volume overload, or aortic valve insufficiency. Some physicians prescribe surgery immediately after diagnosis, regardless of the LVOT pressure gradient, due to the progressive and nonbenign nature of this disease [10, 15, 16]. Early surgical intervention is negated by the high recurrence rate, reoperation risk, and development of aortic regurgitation [15, 17, 18].

2. Aim of the Study

The aim of the study was to evaluate the safety and efficacy of the management of patients with PM VSD and subaortic ridge with or without AR using transcatheter closure of the defect and compressing the subaortic ridge against the ventricular septum using Amplatzer ductal occluder type I (ADO-I). The goal of our technique is to protect the aortic valve and prevent progression of preexisting aortic regurgitation by ameliorating the subaortic ridge.

3. Patients and Methods

This prospective study was performed at the Ibn Albitar Center for Cardiac Surgery, Baghdad, Iraq, from August 1, 2014, to February 1, 2018. Thirty-eight (9.5%) of 398 patients with PM VSD were found to have subaortic ridge and were enrolled in this study. The inclusion criteria allowed patients with PM VSD and significant subaortic ridge causing LVOT flow disturbance with mild-to-moderate LVOT obstruction and mild and mild-moderate AR or LV volume overload. The exclusion criteria included patients with tunnel type and ring-like subaortic obstruction, severe prolapse of the right coronary cusp (RCC), moderate and severe AR, VSD with septal aneurysm, or VSD with malalignment of more than 25%, patients with a distance between the ridge and the proximal border of the VSD more than 5 mm, and patients who had other cardiac defects that required surgical intervention.

Informed consent was obtained from all patients or their guardians. Procedures were performed under deep sedation and fluoroscopic and transthoracic echocardiographic

guidance. Patient data such as age, sex, weight, and TTE measurements were recorded. Echocardiographic assessment included defect size, location, distances of the ridge from the aortic valve and the VSD borders, presence of LV volume overload, peak and mean pulmonary arterial pressures, aortic cusp prolapse, and AR severity. In all included patients, the anatomical characteristics of the lesions as clarified by TTE evaluation were PM VSD with a significant subaortic membranous, fibrous, or fibromuscular ridge below the VSD that protrudes into the LVOT, with or without mild-to-moderate LVOT obstruction and mild-to-moderate AR.

Right and left heart catheterization was performed in all patients to evaluate the pulmonary and systemic pressures, magnitude of the left-to-right shunt, and measurement of the pressure gradient across the LVOT, which were obtained by catheter pullback from the left ventricle to the aorta. The patients were heparinized to achieve an activated clotting time of more than 200 seconds at the time of device implantation. A modified pigtail or Judkins right catheter was used to cross the VSD from the left ventricle with a 0.035 inch hydrophilic guidewire to either the pulmonary artery or the superior vena cava, which was snared and exteriorized from the femoral vein to create an arteriovenous loop. An appropriately sized delivery sheath was advanced from the femoral vein to the ascending aorta. The appropriate device size was chosen to be 1–4 mm larger than the VSD size as measured by the left ventricular angiogram at end-diastole, taking into consideration the anatomical characteristics of the subaortic stenosis as evaluated by TTE (the distance between the subaortic ridge and VSD border should not be more than 5 mm for effective capturing or compressing the ridge by the aortic disc of the ADO-I) (Figure 1(a)).

The aortic disk of the ADO-I was deployed completely just under the aortic valve and pulled toward the defect, capturing and compressing the subaortic ridge against the ventricular septum, which was confirmed by continuous TTE guidance. The waist and the remainder of the device were deployed into the VSD and subsequently into the right ventricle (RV) side of the septum (Figure 1(d), Figures 2(c) and 2(d), and Figures 3(c)–3(f)). When the TTE and left ventricular angiogram confirmed a good device position, aortic and tricuspid valve integrity, and capture or compression of the subaortic ridge, the device was released (Figure 1(d), Figures 2(d), 2(g) and 2(h), Figures 3(f), 3(h) and 3(i), Figures 4(e) and 4(f), and Figures 5(c) and 5(d)).

All patients were discharged a day after the procedure and received aspirin (3–5 mg/kg) daily for six months. Most patients were followed up at one, three, six, and twelve months after the closure and then annually with a TTE study evaluating the device position, direction of the subaortic ridge, residual shunt, right ventricular systolic pressure, or any degree of AR. The mean follow-up interval was 27 ± 7 months (range: 17–41 months). The data are expressed as means \pm standard deviations, medians, percentages, and ranges obtained using the IBM/SPSS statistical program, version 24 (IBM Corporation, Armonk, NY).

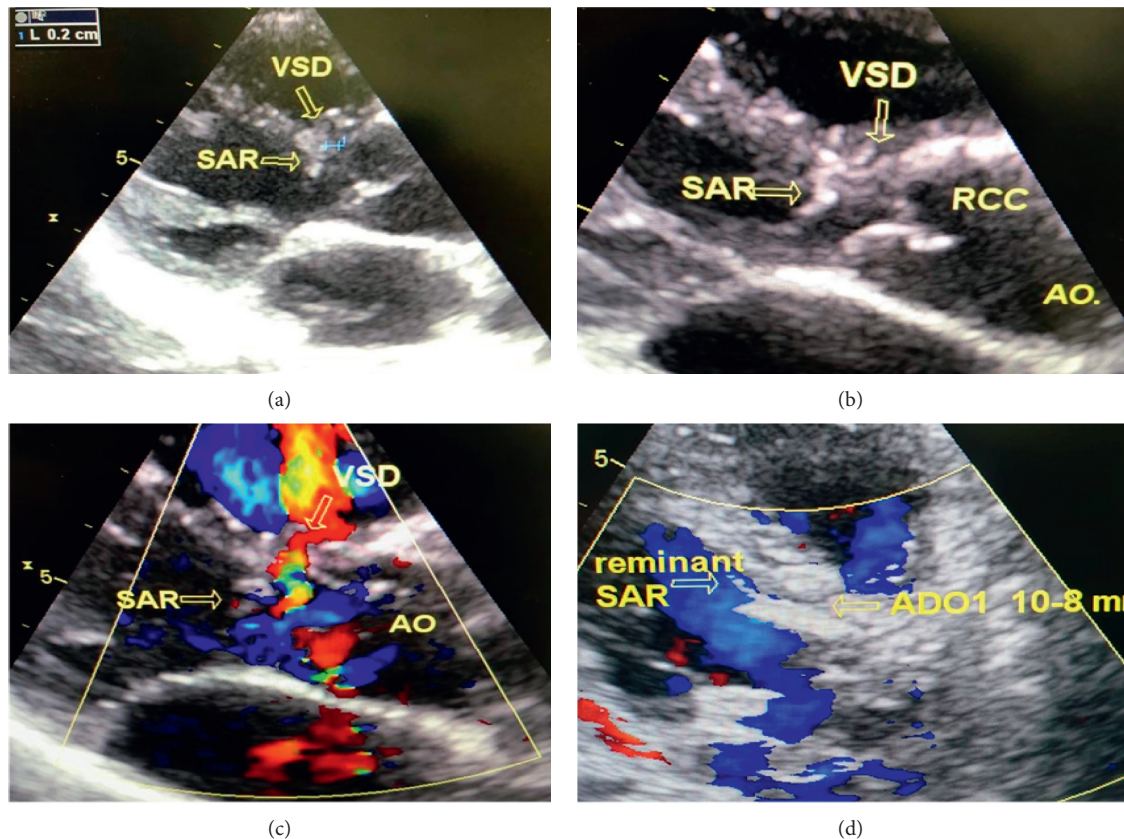


FIGURE 1: (a) and (b) Parasternal long axis 2D echocardiographic images of a 10-year-old female patient with a moderately sized perimembranous subaortic VSD with mild prolapse of RCC and subaortic stenosis (fibromuscular ridge 2 mm below the defect) with a systolic pressure gradient across the LVOT of 35 mmHg. (c) Color flow imaging from the long axis parasternal view demonstrates the location of the defect with a left-to-right shunt and a long and thick subaortic ridge. (d) Modified parasternal long axis echocardiographic image with color flow mapping demonstrates ADO-I (10–8 mm) closing the defect and compressing the ridge against the IVS without residual shunt or AR. The peak systolic pressure gradient decreased to 14 mmHg.

4. Results

Thirty-eight (9.5%) of 398 patients with perimembranous VSD were found to have subaortic ridge and were enrolled in this study. Their ages ranged from 1.5 to 25 years (mean = 6.2 ± 4 years, median = 4.5 years), and their body weights ranged from 7 to 73 kg (mean = 19.7 ± 12 kg, median = 15.2 kg). Most of the patients (26, 68.4%) were men, with a male-to-female ratio of 2.2:1.

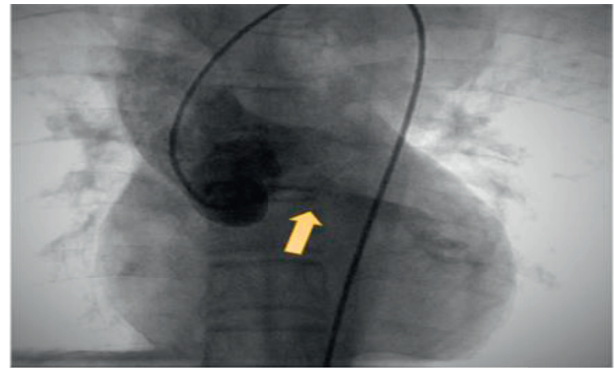
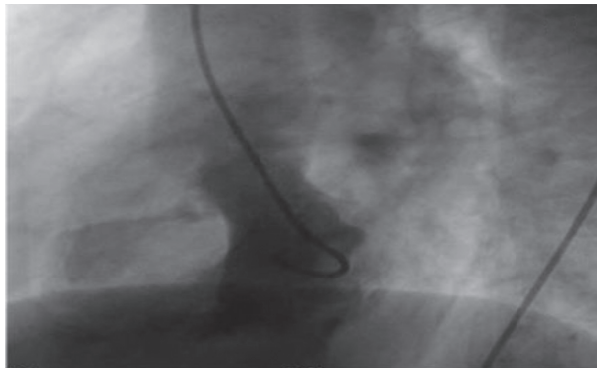
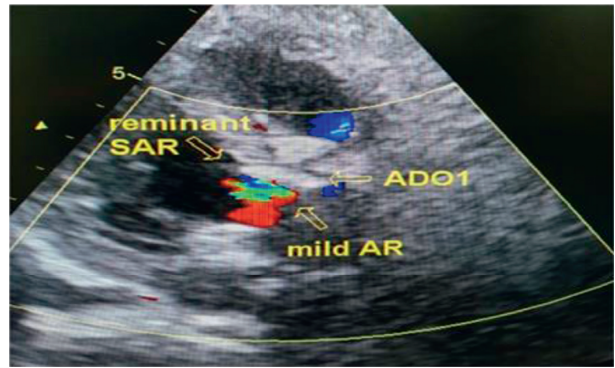
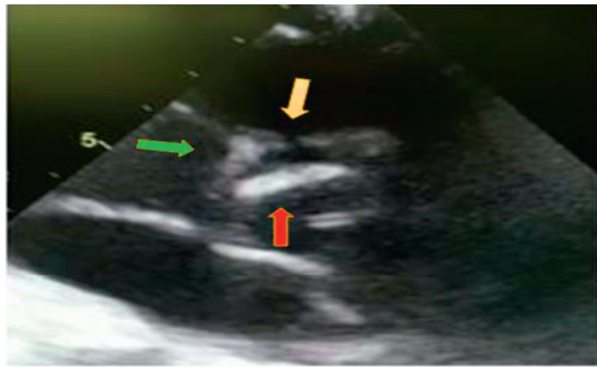
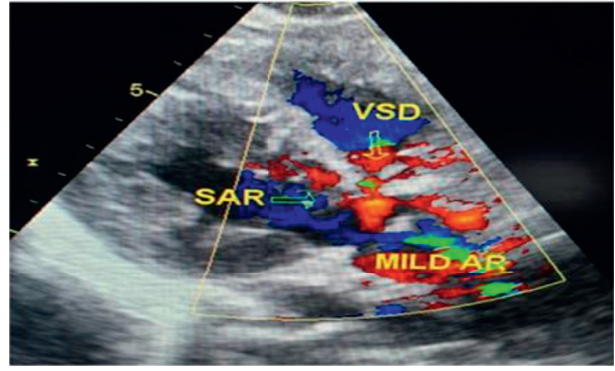
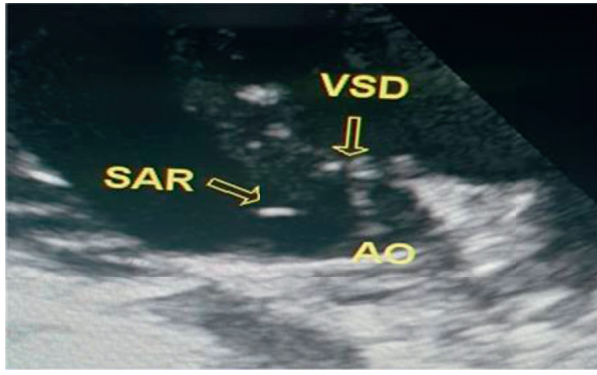
The VSD sizes ranged from 4 to 8 mm (Figure 5(a) and Figures 4(a) and 4(d)), and the median distance of the subaortic ridge from the proximal edge of the VSD was 2.5 mm (range: 1–5 mm) as measured by TTE in parasternal long axis view (Figure 1(a)). The Qp/Qs values ranged from 1.6 to 3.1 (mean = 2.2 ± 0.4), and the left ventricular diastolic dimensions which have been measured by TTE ranged from 24 mm to 59 mm (mean = 37.6 mm, median = 35 mm), which were significant in all patients.

The mean of peak systolic pulmonary artery pressure was 30 ± 6 mmHg (range: 20–45 mmHg), which declined to 19 ± 2.6 mmHg after transcatheter closure of the VSD. None

of the patients exhibited evidence of pulmonary hypertension on follow-up.

Eight (21%) of the patients were found to have mild aortic override (VSD with malalignment of 15–25%) due to anterior deviation of the outlet septum and/or right ventriculo-infundibular fold into the right ventricle without compromising the right ventricular outflow tract (Figure 1(a), Figures 2(a), 2(c), 2(e) and 2(g), Figures 4(a), 4(d), 4(e), and 4(f)), while 30 patients (79%) had VSD without malalignment (Figures 3(a), 3(b), 3(e), and 3(g) and Figures 5(a) and 5(c)).

Twenty-four (63%) patients were found to have mild and moderate prolapsed noncoronary aortic cusp (NCC) and/or right coronary cusp (RCC) (Figure 1(b), Figures 2(a) and 2(h), Figure 3(i), and Figure 4(f)), and the remaining 14 patients (37%) had no cusp prolapse. The study showed that 25 patients (65.8%) had no AR, whereas 13 patients (34.2%) were found to have mild and mild-to-moderate AR angiographically (3 patients <6 years, 3 patients, 6–12 years, 5 patients, 12–18 years, and 2 patients, more than 18 years) (Figures 2(b) and 2(f) and Figure 5(b)). The mean AR



(a)

(b)

(c)

(d)

(e)

(f)

FIGURE 2: Continued.

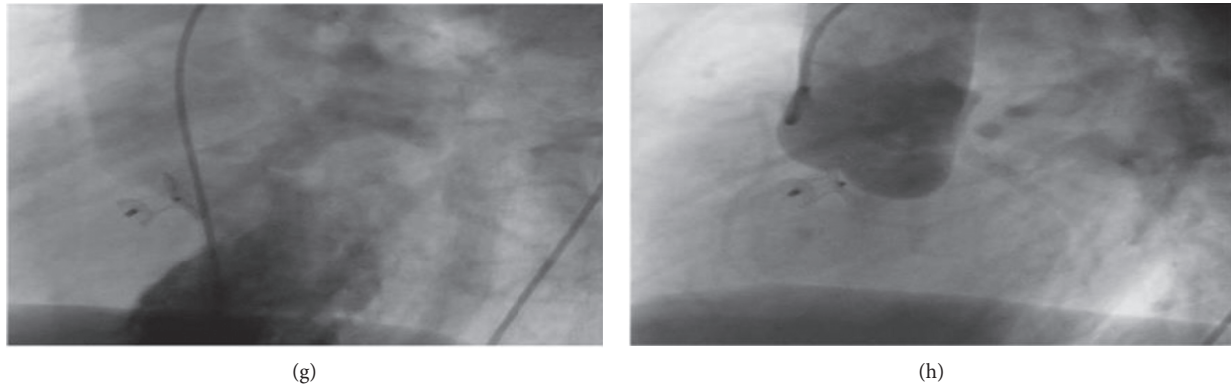


FIGURE 2: (a) Cross-sectional echocardiogram in the long axis parasternal view for a 21-year-old female patient showing a significant subaortic stenosis with broad-based fibromuscular ridge below a perimembranous subaortic VSD with about 20% aortic override. (b) Color flow imaging from the long axis parasternal view demonstrates the location of the defect with left-to-right shunt and long broad-based subaortic ridge with peak systolic pressure gradient of 36 mmHg across the LVOT and mild-to-moderate AR. (c) Parasternal long axis 2D echocardiographic image demonstrates the aortic disc of ADO-I; 10–8 mm (red arrow) was pulled towards the subaortic fibromuscular ridge (green arrow) and VSD (yellow arrow). (d) Parasternal long axis 2D echocardiographic image with color flow mapping demonstrates excellent position of the device closing the VSD and compressing the ridge against the interventricular septum (IVS) with mild AR. The peak systolic pressure gradient across the LVOT decreased to 17 mmHg. (e) Left ventricular angiogram in the left anterior oblique (70°) and cranial (30°) projection demonstrates a small-sized subaortic perimembranous VSD with a mild aortic override of approximately 20%. The peak systolic pressure gradient across the LVOT was 31 mmHg. (f) Aortogram in the left anterior oblique (60°) projection revealing a prolapse NCC with mild-to-moderate AR (yellow arrow). (g) Left ventricular angiogram in the left anterior oblique (70°) and cranial (30°) projection demonstrating no residual shunt across the VSD and obvious aortic override. (h) Aortogram in lateral (90°) and cranial (20°) projections demonstrating proper device position with trivial AR. The peak systolic pressure gradient across the LVOT declined to 12 mmHg.

pressure half-time before intervention was 385 ± 38 ms, which increased significantly after intervention to 535 ± 69 ms (vena contracta from 0.3–0.6 to \sim 0.3) with a significant *P* value (0.001).

Significant peak systolic pressure gradient across left ventricular outflow tract (LVOT) was found in nine patients, 23.7% (two patients less than 6 years, 3 patients 6–12 years, 2 patients 12–18 years, and 2 patients >18 years). The mean of the LVOT peak and mean systolic pressure gradient before intervention were 33 ± 7 mmHg and 15 ± 3 mmHg, which decreased after the intervention to 15 ± 2.4 mmHg and 11 ± 2 mmHg, respectively, with a significant *P* value (0.001). Patients with peak systolic pressure gradient of ≤ 20 mmHg were not included. The hemodynamic characteristics of patients with LVOT obstruction and AR are demonstrated in Table 1.

Five patients with mild AR showed improvement immediately after the procedure, while patients with mild-to-moderate AR (no. = 4) showed regression of the AR to a mild degree in two patients and to a trivial degree in the other two patients during the follow-up period (Figures 2(d) and 2(h) and Figure 5(d)).

The Amplatzer ductal occluder type one (ADO-I) (sizes: 6/4 (6 patients), 8/6 (19 patients), 10/8 (11 patients), 12/10 (1 patient), and 16/14 (1 patient)) was used to close the VSD and capture and compress the subaortic ridge in this study. We have chosen ADO I device because we have managed a lot of cases of subaortic PM VSD using this device (in more than 400 cases) with satisfying results without major complications like complete heart block and aortic regurgitation through long-term follow-up period. We had used ADO II device in one patient with unfortunately an unsuccessful result as the device is too soft

and flexible with a waist between two equal discs that make the compression of the ridge with closure of the VSD not applicable, as the soft left ventricular disc cannot compress the ridge and will not be well aligned with the IVS, making the residual shunt more significant. This is in contrary to the harder LV disc of ADO I device, which is capable of compressing the ridge effectively and would be well aligned with the IVS, making the residual shunt negligible.

Successful procedures were achieved in 33 patients (86.8%), in whom the VSD had been closed without residual shunt and the subaortic ridge had been captured and compressed against the interventricular septum toward the apex by the aortic disc of the ADO-I. The procedures failed in 5 patients due to a deficient aortic rim in three patients and increased severity of the AR in the other two patients.

At the mean of the follow-up period (27 ± 7 months), the highest LVOT systolic pressure gradient as detected by TTE was 22 mmHg, and there was no worsening in the degree of aortic regurgitation. All patients completed the follow-up period of 12 months, four patients missed the follow-up during the 2nd year, and three patients missed the follow-up during the 3rd year. All patients with AR and/or LVOT obstruction completed the follow-up period because we contacted them at the scheduled follow-up time. We encountered no significant complications acutely or on follow-up, and no patients developed heart block.

5. Discussion

We have introduced a new approach for the management of patients with PM VSD and subaortic ridge before and after

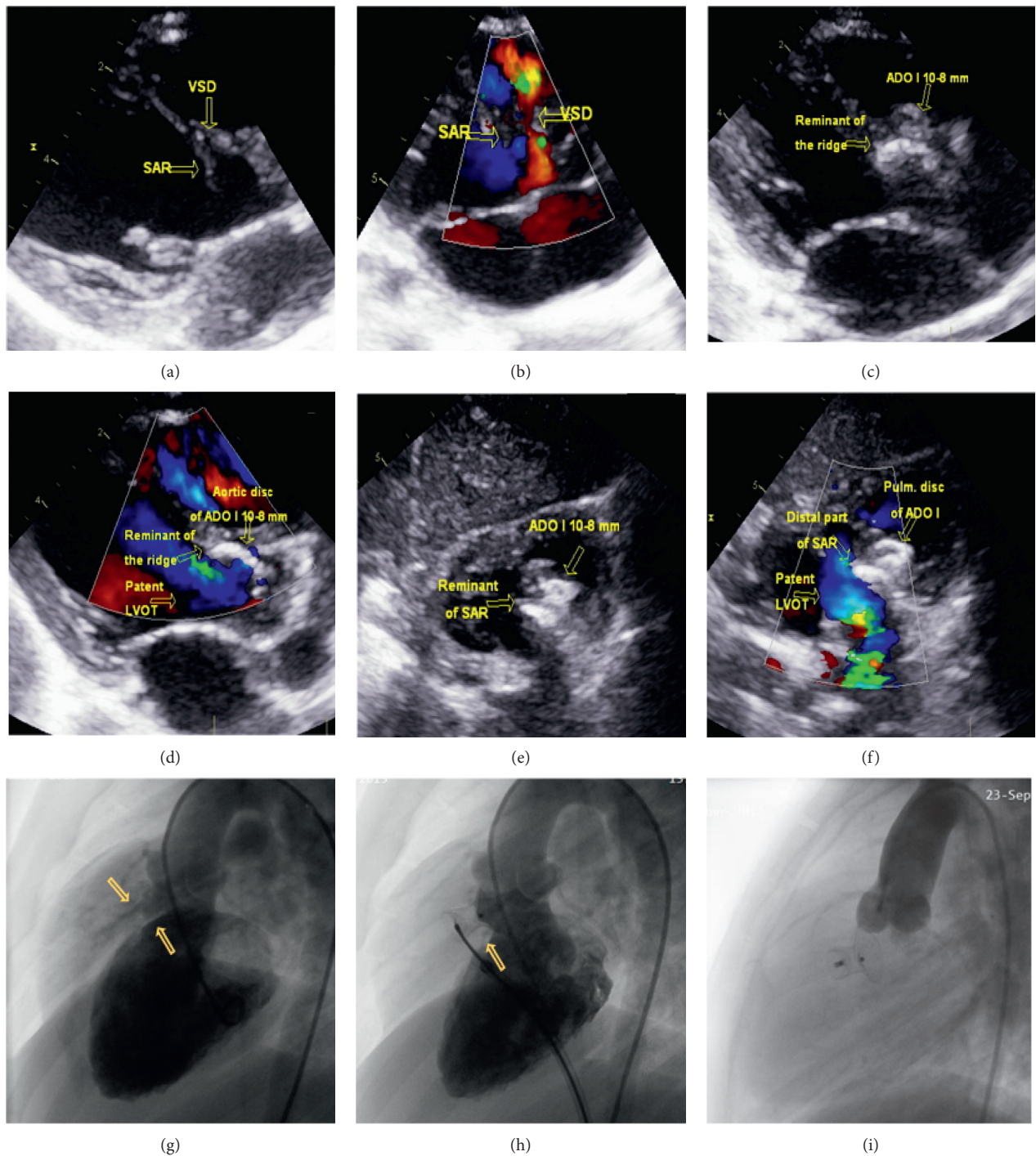


FIGURE 3: (a) Cross-sectional echocardiogram in the long axis parasternal view of a four-year-old male patient demonstrates perimembranous subaortic VSD (upper arrow) distal to the subaortic ridge (lower arrow). (b) Parasternal long axis 2D echocardiographic image with the color flow mapping demonstrating significant left-to-right shunt across the defect (arrow) with the subaortic ridge. (c) Echocardiographic parasternal long axis view shows ADO-I (10–8 mm) closing the defect and compressing the subaortic ridge against the IVS. (d) Parasternal long axis 2D echocardiographic image with color flow mapping demonstrating the optimum device position with neither residual shunt nor AR. (e) Echocardiographic subcostal view demonstrating proper position of ADO-I closing the defect and compressing the subaortic ridge against the IVS with patent LVOT. (f) Modified subcostal 2D echocardiographic image with color flow mapping demonstrating ADO-I closing the defect and compressing the subaortic ridge against the IVS with no residual shunt, patent LVOT, and no AR. (g) Left ventriculogram in the left anterior oblique (70°) and cranial (20°) projection demonstrates a small-to-moderate size subaortic VSD (upper arrow) with a filling defect below the VSD that represents the subaortic ridge (lower arrow). The peak systolic pressure gradient across the LVOT was 16 mmHg. (h) Left ventriculogram in the left anterior oblique (70°) and cranial (20°) projection documents good device deployment with no residual shunt. (i) Aortogram in lateral (90°) and cranial (20°) projection, demonstrating no AR despite prolapse of the NCC. The peak systolic pressure gradient across the LVOT decreased to 10 mmHg.

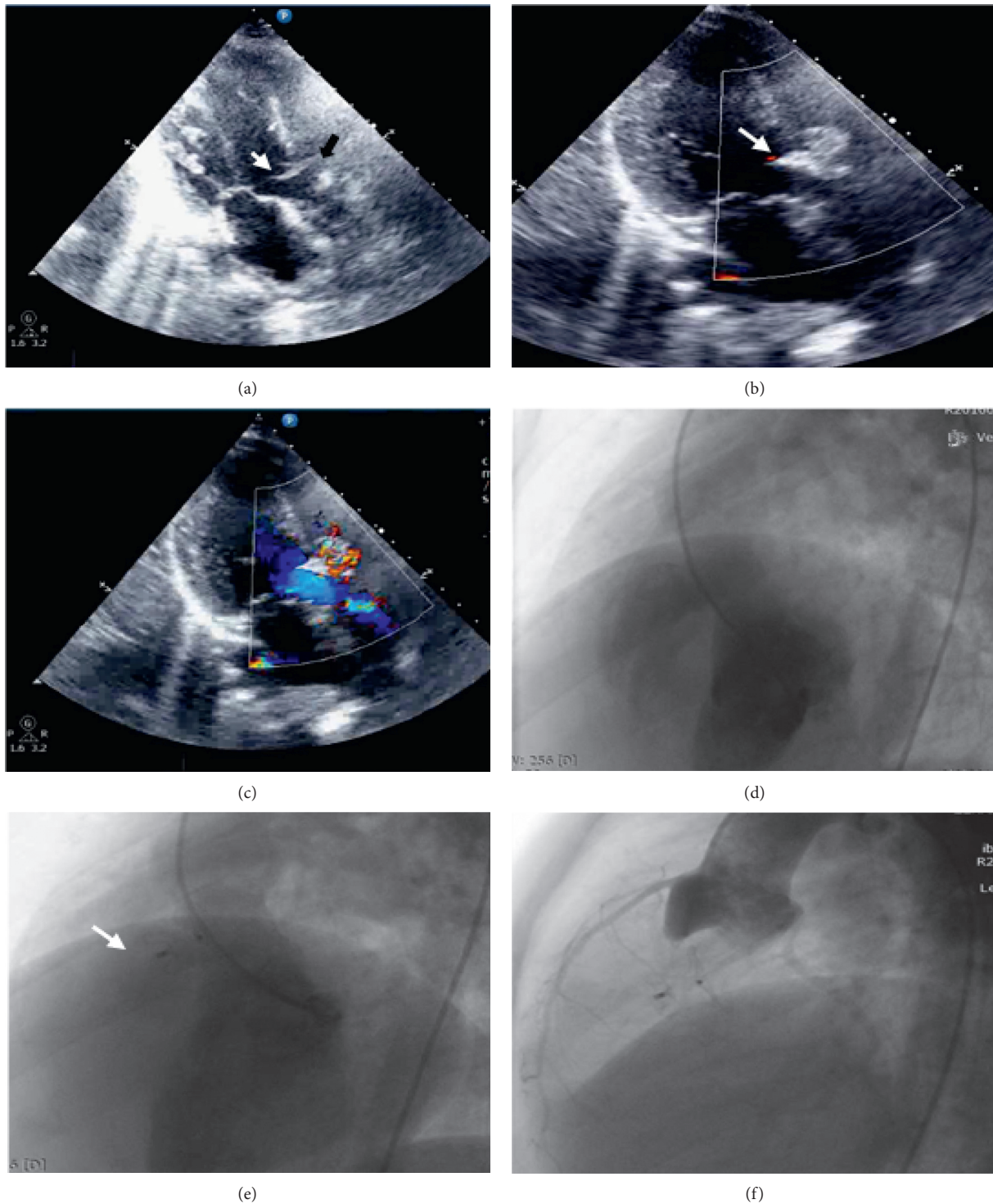


FIGURE 4: (a) Parasternal long axis 2D echocardiographic image of a 13-year-old female patient demonstrates moderate-to-large subaortic perimembranous VSD with 15% aortic override (black arrow) distal to a significant subaortic ridge (white arrow). (b) Cross-sectional parasternal long axis 2D echocardiographic image shows ADO-I (16–14 mm) closing the defect and compressing the subaortic ridge toward the IVS. (c) Modified parasternal long axis 2D echocardiographic image with color flow mapping documenting that ADO-I compresses the subaortic ridge successfully with complete closure of the VSD with no AR. (d) Left ventriculogram in the left anterior oblique (70°) and cranial (20°) projection demonstrating a moderate-to-large sized subaortic VSD with 15–20% aortic override and peak systolic pressure gradient across the LVOT of 20 mmHg. (e) Left ventriculogram in the left anterior oblique (70°) and cranial (20°) projection documents excellent device position (arrow) with a small residual shunt through it (which completely disappeared at follow-up). (f) Aortogram in the lateral (90°) and cranial [20] projection demonstrating no AR despite mild prolapse of the RCC. The peak systolic pressure gradient across the LVOT decreased to 10 mmHg.

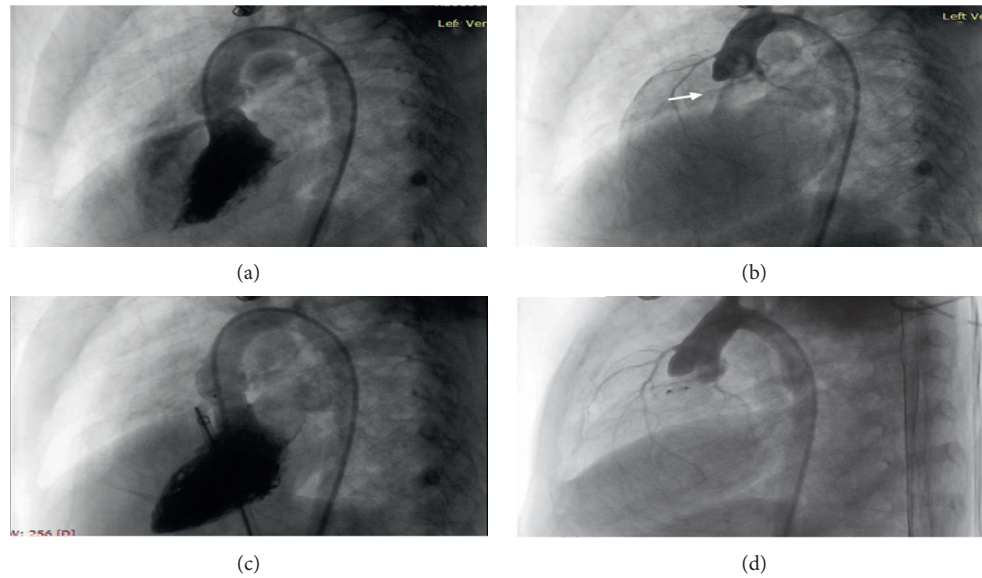


FIGURE 5: (a) Left ventriculogram in the left anterior oblique (70°) and cranial (20°) projection of a 2.5-year-old female patient demonstrates a small subaortic VSD. (b) Aortogram in the left anterior oblique (60°) projection reveals mild AR. (c) Left ventriculogram in the left anterior oblique (70°) and cranial (20°) projection documents excellent device deployment with no residual shunt. (d) Aortogram in the lateral (90°) and cranial (20°) projection demonstrating proper device positioning with no AR.

TABLE 1: Hemodynamic characteristics of patients with LVOT obstruction and AR.

No.	Age/yr	Wt/kg	Qp:Qs	AR PHT		LVOT PG		PPAP	
				Pre	Post	Pre	Post	Pre	Post
1	7.2	28	2.9	390	555	28	12	27	14
2	4	12	1.6	433	522	26	14	41	24
3	6.2	17	2.2	366	611	27	18	29	18
4	25	73	1.7	322	655	48	16	45	16
5	12.3	35	2.3	355	499	33	15	30	21
6	6.1	18	2	411	455	35	14	31	17
7	18.2	35	2.4	344	555	41	20	37	23
8	12	27	2.2	388	577	30	13	36	18
9	3.7	14	3	422	426	32	15	37	15
10	13.1	24	2.7	355	488			25	19
11	12.4	22.5	1.9	399	633			33	20
12	12.1	43	2	377	488			30	21
13	2.8	13	2.2	455	499			26	14
Mean ± SD	10.39 ± 6.3	27.80 ± 16.5	2.23 ± 0.4	385.9 ± 38	535.6 ± 69	33.33 ± 7.1	15.22 ± 4.2	32.84 ± 6.0	18.46 ± 3.2
Median	12	24	2.2	388	522	32	15	31	18

AR PHT: aortic regurgitation pressure half-time, LVOT PG: left ventricular outflow tract pressure gradient, PPAP: peak pulmonary artery systolic pressure, Pre: preintervention, Post: postintervention.

development of AR via transcatheter closure of the VSD and concomitant capturing or compressing the subaortic ridge against the ventricular septum by the aortic disk of the ADO-I.

The incidence of subaortic ridge in our study was 9.5% (38/398), which is comparable to those reported by Zielinsky et al. [5] (10.5%, 32/295) and Cassidy et al. [6] (7.2%, 16/223). Gabriels et al. [19] reported a higher incidence (14%), which may be due to the older age of their patients (median age = 29 years), including postoperative cases.

The progressive nature of the subaortic ridge was confirmed by many investigators, such as Vogel et al. [12] and

Horta et al. [10], who reported progression of LVOT pressure gradient in 63.89% of cases during the follow-up period. The preoperative development and progression of AR has also been mentioned by authors like Rohlicek et al. [15] (the incidence of AR increased from 18% (12/68) to 53% (36/68) during the follow-up period of 3.6 years), Babaoglu et al. [14] (91% of patients), and others [10, 12].

Many authors documented the development and progression of AR postsurgical resection of the subaortic ridge, even in patients without preexisting detectable AR, such as Babaoglu et al. [14], Rohlicek et al. [15], Tefera et al. [20], and Donald et al. [18], who documented progression of aortic

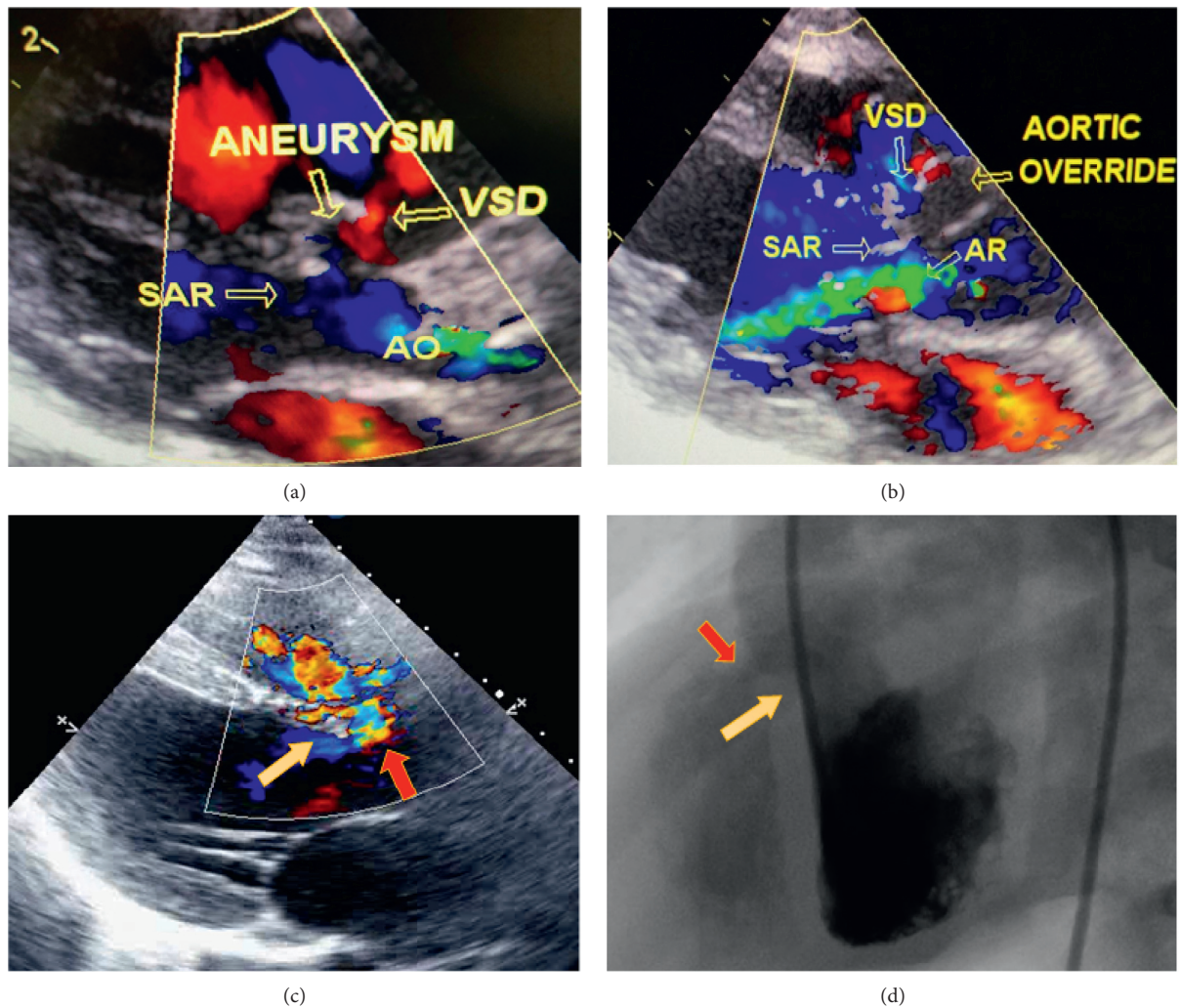


FIGURE 6: Exclusion criteria. (a) Echocardiographic parasternal long axis view with color flow mapping of a 10-year-old male patient demonstrated a moderately sized perimembranous subaortic VSD with septal aneurysm and subaortic ridge below the aneurysm and the VSD. (b) Cross-sectional echocardiogram in the long axis parasternal view with color flow mapping of an eight-year-old female patient demonstrating a moderately sized subaortic VSD abutting severely prolapsed RCC with significant subaortic stenosis (fibromuscular ridge) associated with a moderate-to-severe AR. (c) Cross-sectional echocardiogram in the long axis parasternal view of a 3.5-year-old male patient with two holes of perimembranous subaortic VSD (red arrow) with anterior deviation of the outlet septum into the right ventricle (yellow arrow) causing subaortic obstruction below the VSD. (d) Left ventricular angiogram in the left anterior oblique (70°) and cranial (30°) projection showing a moderate sized subaortic VSD (red arrow) with anterior deviation of the outlet septum into the right ventricle without compromising the right ventricular outflow tract (yellow arrow) producing an aortic override of 40% and subaortic obstruction below the VSD (narrow LVOT).

regurgitation in 38.9% (28/72) of postoperative patients. In our study, no patients developed AR after closure of the VSD and compressing the subaortic ridge by ADO-I immediately and during the follow-up period, while patients with mild-to-moderate AR showed either significant improvement or no progression of AR during the follow-up period.

Surgical resection of the subaortic ridge with or without extensive myectomy carries the risk of development of complete heart block, which has been mentioned in many studies like that of Parry et al. [21] (5 patients, one initially and 4 after reoperation), Serraf et al. [22], and Drolet et al. [23] (6%, 3/49), while, with our technique, no patient developed heart block throughout the follow-up period.

Patients with VSD and subaortic ridge without AR were treated successfully using this technique with amelioration of the LVOT obstruction without interference with the aortic valve function (Figures 1, 3, and 4). The regression of AR in some patients after device closure of the VSD and compression of the subaortic ridge is not completely understood and may be related to multiple factors (Figures 2(b), 2(d), 2(f), and 2(h); Figures 5(b) and 5(d)). We thought that closure of the VSD with abolishment of the left-to-right shunt effect on the aortic valve leaflets and the restoration of the LVOT anatomy (after closure of the VSD and compressing the subaortic ridge against the ventricular septum) greatly decreased the blood flow disturbance through the LVOT, a finding that

was also observed after closure of the VSD with AR and without an associated subaortic ridge. If device implantation induces a new onset or worsening of preexisting AR, the device was retrieved and the patient was referred for surgery.

In this study, most patients had a prolapse of the noncoronary cusp (NCC), which did not interfere with the device implantation or resulted in induction or worsening of AR (Figures 2(a) and 2(h) and Figure 3(i)). Patients with severe prolapse of the right coronary cusp (RCC) were excluded from this study as transcatheter closure of the VSD was unsuitable because the RCC was prolapsing into the defect (Figure 6(b)). All patients with prolapsing cusps (24/38, 63%) who underwent successful procedures without induction of AR continued to have no AR during the follow-up period of 12 months. Three patients missed follow-up during the next two years.

In patients with LVOT obstruction (no. = 9), it was found that the severity of obstruction increased with age of the patients (7 patients were more than 6 years), a finding that was reported by other authors, such as Shem-Tov et al. [11], and Rohlicek et al. [15].

Perimembranous subaortic VSD with malalignment associated with subaortic ridge has been described by many authors. Zielinsky et al. [5] reported malalignment VSD in all patients (100%), and Kitchiner et al. [7] reported nearly equal percentages between subaortic ridge with malalignment VSD (33/65, 50.8%) and those with short-segment fibromuscular ridge without malalignment (32/65, 49.2%). In our study, only eight patients (21%) had VSD with mild malalignment due to anterior deviation of the outlet septum that resulted in aortic override of 15–25% according to our global assessment, which did not interfere with our technique to close the VSD and compress the subaortic ridge that was abutting the lower border of the defect (Figures 2(a)–2(c), 2(e) and 2(g), Figures 4(a), 4(d), 4(e) and 4(f), and Figure 1(a)). Patients with malalignment VSD \geq 30% were excluded from this study because we found that our technique cannot be applied in such cases (Figures 6(c) and 6(d)). Patients with associated septal aneurysm were excluded because our technique cannot be applied to close the defect and compress the subaortic ridge against the septal aneurysm (Figure 6(a)).

6. Conclusion

Transcatheter management of patients with PM VSD and subaortic ridge is feasible and effective in protecting the aortic valve by ameliorating the blood flow disturbance across the LVOT created by the left-to-right shunt across the VSD and the presence of subaortic ridge with regression of the preexisting AR. In addition, this technique was found to be effective in relieving LVOT obstruction by compressing the subaortic ridge against the interventricular septum without creating a new AR.

Data Availability

The data used in this study are available within the article.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

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