ISSN: 3049-7361

Case Report

Journal of Clinical Surgery and Anesthesia

Surgical Strategy in Partial AVSD and Mitral Cleft Repair: The Experience of a Failed Annuloplasty Ring and the Importance of 'Ring-less' Repair

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Received: November 19, 2025; **Accepted:** November 25, 2025; **Published:** December 02, 2025

ABSTRACT

Mitral valve cleft is a progressive congenital anomaly that can lead to severe mitral regurgitation and pulmonary hypertension. Mitral valve cleft is often associated with complete atrioventricular canal defects. However, its association with partial defects is less common and presents unique technical challenges for surgical repair due to distorted annular geometry.

The etiology of this lesion is unknown, and diagnosis of mild cases may be difficult at an early age. Although, the severity of regurgitation and valve thickening tend to show a progressive course over time. This case report presents the clinical presentation and a critical surgical challenge treatment of a 26-year-old female patient diagnosed with mitral cleft in conjunction with a partial atrioventricular canal defect, representing an advanced example of this rare association.

Keywords: Mitral Cleft, Atrial Septal Defect, Mitral Regurgitation

Introduction

Mitral valve cleft is a congenital division defect in one of the mitral valve leaflets that leads to mitral regurgitation [1]. Mitral clefts may exist in isolation or can be seen in association with atrioventricular canal defects [2,3]. The anterior (aortic) leaflet of the mitral valve is the most involved in most cases of mitral cleft [1,4]. Posterior leaflet involvement may occur, but it is very rare and usually acquired; congenital posterior mitral clefts are mostly isolated and not associated with atrioventricular canal defects [5,6]. A slit-like defect in the valve leaflet disrupts the closure of the anterior and posterior leaflets and improper coaptation of the leaflets [1,6]. This leads to mitral regurgitation, which over time progresses to pulmonary hypertension and eventually heart failure [2,7].

This case report presents the clinical presentation and treatment of a 26-year-old female patient diagnosed with mitral cleft in conjunction with a partial atrioventricular canal defect.

Case Presentation

A 26-year-old female patient referred to institutional hospital with complaints of dyspnea on exertion and reduced exercise capacity. Her past medical history was remarkable with congenital atrioventricular septal defect that was diagnosed during pregnancy 4 years ago. She successfully completed her pregnancy without any hemodynamic and cardiovascular complication. Her ventricular septal defect spontaneously underwent spontaneous closure over time. The patient's physical examination was in stable general condition. Pulmonary and cardiac auscultation were unremarkable. A 2–3/6 systolic murmur was noted at the mesocardiac region without associated thrill. Heart sounds were normal no audible murmurs, and no peripheral edema was observed. Her echocardiography

Citation: Emre Öteyaka, Mustafa Sedat Yıldız, Hasan Erdem, Didem Melis Öztaş, Murat Uğurlucan, et al. Surgical Strategy in Partial AVSD and Mitral Cleft Repair: The Experience of a Failed Annuloplasty Ring and the Importance of 'Ring-less' Repair. J Clin Surg Anesth. 2025. 3(4): 1-5.
DOI: doi.org/10.61440/JCSA.2025.v3.42

demonstrated a preserved left ventricular ejection fraction (EF 55%). Moderate mitral regurgitation (MR) was detected, and pulmonary artery pressure was estimated at 40 mmHg also right ventricle was dilated. Additionally, echocardiographic evaluation revealed thickening of the anterior mitral leaflet with central mitral regurgitation. A cleft was noted in the anterior mitral leaflet. Posterior mitral annular dilation was present. A large atrioventricular canal defect with a primum atrial septal defect (ASD) was observed. Tricuspid annular dilation was also detected, resulting in mild-to-moderate tricuspid regurgitation (TR). A surgical repair of anterior mitral valve cleft and closure of atrial septal defect with pericardial patch was planned. The patient was scheduled for corrective surgery fallowing the consent of the family after being informed about risks and benefits of the treatment in details.

Surgical Treatment

The patient was placed under general anesthesia, with right internal jugular vein (IJV) and left radial artery cannulation, followed by urinary catheter placement. Median sternotomy was performed to access the heart. The aorta, superior vena cava (SVC), and inferior vena cava (IVC) were mobilized; Ao-SVC-IVC cannulation was performed, and the patient was placed on extracorporeal circulation (ECC) and cooled to 28°C. The heart was freed from surrounding tissues, the aorta was clamped, and cardiac arrest was achieved using antegrade Delnido cardioplegia at +8°C, 20 ml/kg. The right atrium was opened, and the mitral valve was evaluated through the primum atrial septal defect (ASD). Clefts were observed in the anterior and posterior leaflets (Figure 1). The clefts were closed at the annular level using 5/0 Prolene, and augmentation of the anterior leaflet was performed with fresh autologous pericardium (Figure 2). A 28-size Edwards annuloplasty ring was initially placed, but it was removed due to increased regurgitation and deformation of the valve. Posteromedial commissural annuloplasty was then performed; mild mitral regurgitation was left untreated to avoid further stenosis.

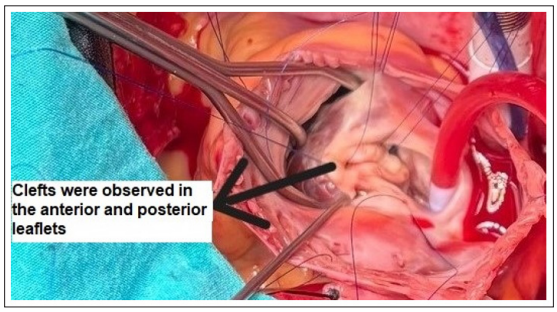

Figure 1: Clefts were Observed in the Anterior and Posterior Leaflets

The primum ASD was closed using fresh autologous pericardium, which was extended from the tricuspid to the mitral annulus, keeping the coronary sinus on the right (Figure 3). Tricuspid annular dilation was observed; posteroseptal annuloplasty directed toward the coronary sinus and antero-septal commissural annuloplasty were performed. The atriotomy was closed appropriately, and air was evacuated before releasing the aortic clamp. The heart resumed spontaneous contraction and returned to sinus rhythm.

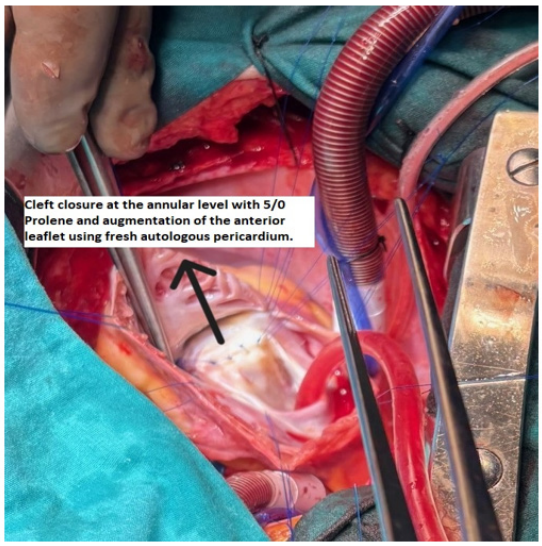

Figure 2: Cleft Closure at the Annular Level with 5/0 Prolene and Augmentation of the Anterior Leaflet Using Fresh Autologous Pericardium.

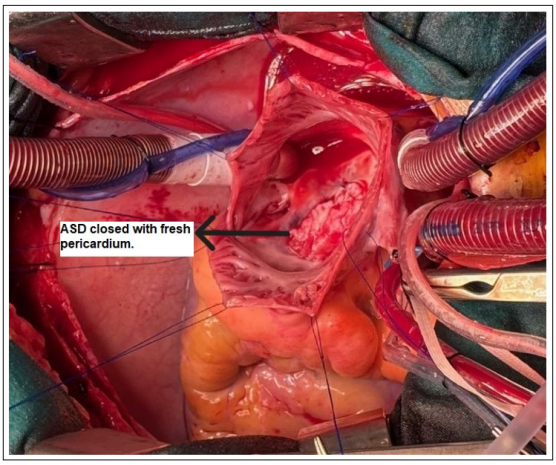

Figure 3: The Primum ASD was Closed.

Weaning from ECC was performed. Transesophageal echocardiography after surgery was (TEE) demonstrated mild mitral regurgitation and mild mitral stenosis; no further intervention was made. Temporary pacemaker wires were placed on the right heart. The posterior mediastinum was opened toward the left pleura; one 32F drain was placed in the mediastinum and one 32F drain in the left thorax. Sternotomy was closed with wires, and the subcutaneous tissue and skin were closed. The patient was transferred to the intensive care unit (ICU) in sinus rhythm.

Discussion

Mitral valve cleft is a congenital division abnormality of one of the mitral leaflets. Both mitral valve leaflets can be affected by mitral clefts; however, the anterior leaflet of the mitral valve is the most affected leaflet [1,2]. It usually coexists with congenital atrioventricular canal defects, aorta and coronary artery defects, or endocardial cushion defects [3,4]. It may also occur in isolation, but this is rare [5]. The anterior mitral leaflet cleft mostly originates from the intervalvular fibrosa (between the aortic and mitral annulus) and is usually directed toward the aorta [6]. This leads to improper coaptation of the mitral leaflets and

incomplete closure of the mitral valve during systole, causing mitral regurgitation [1,7]. There is no strong gender predilection for anterior mitral leaflet cleft, although some studies show a slight female predominance [3]. Our patient was a 26-year-old female, and her cleft was in the anterior leaflet, which coexisted with an atrial septal defect [8].

The diagnosis of the mitral cleft may be easily missed in neonatal screening and juvenile ages [1]. Mitral cleft diagnosis is usually confirmed by echocardiographic evaluation of the mitral valve [2]. However, 2-dimensional echocardiographic evaluation does not always provide accurate information about the structural integrity of the valve because of the anatomical location of the mitral valve in the heart [3]. For this reason, using 3-dimensional Doppler transesophageal echocardiography for diagnosis is more useful and provides more accurate information about the characteristics of the mitral cleft [4,5]. Nevertheless, intracardiac imaging and evaluation of the mitral valve through the atrium is considered the most reliable method for diagnosis [6]. In some cases, the exact features of the mitral cleft are detected only during surgery, despite all pre-operative screening tests, and this may affect the surgical method of mitral repair [7]. Additionally, cardiac magnetic resonance imaging (MRI) may be used in selected cases to evaluate chamber volumes and regurgitant fraction [8]. We used 2D and 3D transthoracic and transesophageal echocardiography for evaluation of the mitral valve integrity [5,7].

Mitral valve cleft (MVC) is a congenital pathology that is inherently progressive and frequently leads to mild to severe mitral regurgitation (MR) [1,2]. The severity of MR tends to progress over time [3], and this progression forms the basis of the patient's clinical presentation. The severity of symptoms is influenced by the anatomical location, division, size, and accompanying atrioventricular valve abnormalities of the cleft [2,4]. Our patient's association with partial AVSD (pAVSD) is an example of this course; the natural course of pAVSD may allow patients to remain asymptomatic until adulthood [8,9]. Treatment methods of the mitral clefts are associated with anatomical location and size of the cleft [10]. The surgical repair of the mitral cleft has several techniques that depend on severity of the symptoms [11]. The main aim of the treatment is to achieve proper leaflet coaptation, maintain structural integrity, preserve subvalvular apparatus, adjust annular size, and avoid mitral valve replacement as much as possible [12].

As MR caused by cleft progresses, it inevitably leads to volume overload, left atrial and ventricular enlargement, and ultimately hemodynamic changes such as pulmonary hypertension (PH) [4,6,7]. This situation causes symptoms such as dyspnea, decreased exercise capacity, fatigue, and palpitations [2,5,8]. Our patient's clinical presentation provides a striking example of this classic progressive course. Although she was diagnosed four years ago during her pregnancy, the fact that she survived that period without experiencing any hemodynamic complications suggests that the lesion was better tolerated at that time. Over the intervening four years, the patient's current exertional dyspnea, PH estimated at 40 mmHg, and the enlargement of the left heart chambers detected on echocardiography clearly demonstrate that the MR has progressed silently but rapidly during this period. It is known that untreated, this progressive MR leads to PH and ultimately heart failure [7,9].

Surgical repair of a mitral valve cleft (MVC) in the setting of a partial AVSD (pAVSD) presents more complex challenges than repairing an isolated cleft. The primary goal of surgical treatment is to achieve adequate leaflet coaptation and eliminate mitral regurgitation (MR) through direct suture or patching of the cleft and correction of annular dilation [13]. Direct suture closure of the cleft is applied in suitable cases by approximating the cleft edges using interrupted sutures [14,15]. However, if the cleft is large, if there is tissue deficiency, or if coaptation will be insufficient after simple closure, the "patch augmentation" technique is employed [16,17]. In this technique, autologous pericardium is often used to restore the normal anatomy of the valve [16,17]. In our case, augmentation with autologous pericardium was also preferred, specifically to prevent tension on the anterior leaflet and to increase the coaptation surface area.

If regurgitation persists after these repairs, adjunctive annuloplasty (ring or band) is often recommended to provide annular support by reducing annular dilation and improving coaptation [14,18]. The most instructive point of our case was the challenge encountered during this annuloplasty repair strategy. Initially, a size 28 annuloplasty ring was placed, but this intervention resulted in "increased regurgitation and deformation of the valve," leading to the ring's removal. This experience strongly highlights that the mitral valve annulus (left atrioventricular valve or LAVV) associated with pAVSD differs from the standard mitral annulus geometry [19,20]. In these patients, due to abnormal commissural placement, imposing a rigid or complete ring can force the subvalvular apparatus into an unnatural position and worsen coaptation rather than improving it [21].

Following this unsuccessful ring attempt, the surgical team shifted to a more anatomical and "ring-less" repair technique. The posteromedial commissural annuloplasty that was performed is a classic and effective suture annuloplasty method aimed at directly narrowing the dilated segment without deforming the annulus with a ring [22]. The presence of mild MR and mild stenosis on postoperative TEE is a common finding in these complex repairs. This is the result of "surgical judgment," where an adequate repair is preferred over the risk of unacceptable mitral stenosis that a more aggressive repair might cause [23].

Residual mitral regurgitation (MR) is one of the most recognized complications following mitral cleft repair [24] and is cited as the primary driver for reoperation in patients with partial atrioventricular septal defect (AVSD) [25]. Achieving durable, long-term leaflet coaptation is critical, as inadequate closure may necessitate future re-repair or valve replacement [26]. Even with modern patch techniques, such as the "diamond-shaped patch," which show good early results (approx. 90% with no/mild MR), a subset of patients still experiences moderate (6.6%) or severe (3.3%) MR within the first year, highlighting the technical challenge [26]. The presence of a cleft also complicates newer techniques like edge-to-edge repair, often requiring more devices and yielding less favorable outcomes [24].

This pursuit of eliminating regurgitation, however, must be balanced against the risk of iatrogenic mitral stenosis. As noted in our case, the final surgical outcome was a balance, accepting mild MR and mild stenosis. This reflects the known surgical dilemma: an "overzealous suture closure or patch insertion" to achieve zero MR can restrict leaflet mobility, reduce the effective orifice area, and ultimately impair long-term valve function by causing stenosis [26]. Therefore, the mild residual findings in our patient represent an accepted compromise to avoid both significant residual regurgitation and prohibitive stenosis.

Surgical repair in infants and those with associated congenital anomalies presents additional challenges due to fragile tissue, complex anatomy, and subvalvular apparatus distortion, increasing the risk of chordal injury or repair failure [27]. Even with seemingly optimal cleft closure, uncorrected annular dilatation or abnormal annular geometry may compromise durability, underscoring the importance of proper annuloplasty support [28]. Although isolated cleft repair tends to have favorable outcomes in high-volume centers, technique standardization is limited, and outcomes are strongly influenced by surgeon experience and anatomical complexity [29]. Preoperative imaging, particularly 3D transesophageal echocardiography, is indispensable for delineating cleft anatomy, leaflet quality, subvalvular apparatus, and annular dimensions [30]. Whenever feasible, repair is preferred over replacement in congenital cases to preserve native valve and subvalvular apparatus, facilitate ventricular growth (in children), and optimize left ventricular function [30]. Long-term follow-up is essential to monitor for recurrent MR, valve stenosis, annular dilatation, ventricular dysfunction, and potential reintervention [30].

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