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Case Reports

Double-Chambered Right Ventricle in Adults: Characteristic Echocardiographic Features from an Incidental Case

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Abstract

Background: A Double Chambered Right Ventricle (DCRV) is an uncommon congenital heart defect in which an abnormal muscular bundle divides the Right Ventricle (RV) into two chambers with different pressure levels. Diagnosing DCRV in adults is challenging because it is often asymptomatic and may be misdiagnosed.

Case Illustration: We present a 37-year-old male with a history of an uncorrected Ventricular Septal Defect (VSD), who was incidentally found to have type 2 DCRV on echocardiography during evaluation for abdominal pain. No residual VSD was detected, possibly due to spontaneous closure. Electrocardiography (ECG) revealed first-degree Atrioventricular (AV) block. Chest radiography showed cardiomegaly with a Cardiothoracic Ratio (CTR) of 0.56. Transthoracic Echocardiography (TTE) demonstrated an anomalous muscular bundle that divided the RV into a high-pressure proximal chamber and a low-pressure distal chamber, accompanied by left atrial and ventricular enlargement.

Conclusions: DCRV in adults is often misdiagnosed as pulmonary stenosis, especially in patients with mild or no symptoms. TTE and Doppler imaging are key diagnostic modalities for accurate diagnosis; however, advanced imaging techniques such as Transesophageal Echocardiography (TEE) may be required in complex cases. Management includes symptom control, such as beta-blockers, and further evaluation to determine the need for surgical intervention.

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Keywords: Adult congenital heart disease, double-chambered right ventricle, echocardiography, ventricular septal defect

Introduction

Double Chambered Right Ventricle (DCRV) is an uncommon congenital heart defect, representing only about 0.5-2.0% of all congenital cardiac anomalies.[1] It is defined by hypertrophy of muscular bundles within the Right Ventricle (RV) that partition the chamber into two cavities, a high-pressure proximal chamber and a low-pressure distal chamber.[2] Most patients are diagnosed during childhood or adolescence, commonly before the age of 20. Nevertheless, some individuals may remain asymptomatic until adulthood, which can result in a delayed diagnosis.[3]

Reports of DCRV in adult patients are rare, especially in Indonesia. Furthermore, diagnosis in adults is challenging, and DCRV is often misdiagnosed as pulmonary stenosis.[4] We present a case of DCRV in a 37-year-old male in Indonesia who was detected incidentally during evaluation of a non-cardiac complaint. Several adult cases of DCRV have been reported, most commonly presenting with exertional dyspnea, chest pain, or during evaluation of a Ventricular Septal Defect (VSD) after a cardiac murmur was incidentally detected on routine examination. In contrast, our patient, who had a history of congenital heart disease during childhood, was diagnosed incidentally while being evaluated for a non-cardiac complaint. This report aims to emphasize the importance of considering DCRV as a differential diagnosis in adult individuals with prior congenital heart disease presenting with a systolic murmur, even in the absence of typical symptoms. Additionally, this case highlights the crucial role of echocardiography as an essential tool for accurate diagnosis of DCRV.

Case Illustration

A 37-year-old male was admitted to the cardiology department for further assessment of a prominent systolic heart murmur. He initially came with complaints of intermittent sharp pain in the epigastrium extending to the lower abdomen for one week, accompanied by nausea, vomiting, weakness, and sudden episodes of palpitations without clear triggers. On physical examination, a prominent ejection systolic murmur graded 5/6 with a palpable thrill was detected, most prominent along the left sternal border. The patient was diagnosed with a VSD in early life, which had been managed conservatively with daily medication and without corrective surgery. Twelve years earlier, the patient had been hospitalized for 40 days due to infective endocarditis. After a treadmill test showed good tolerance, he discontinued follow-up and cardiac medications. He had no complaints of chest pain, breathlessness, excessive sweating, or loss of consciousness. However, he occasionally experienced dizziness and blurred vision, which he attributed to a long-standing history of hypotension. There was no history of drug allergies, chronic or infectious diseases, or growth and developmental issues during childhood. Family history revealed that his mother and first brother had died of heart disease, though not related to congenital heart defects.

On physical examination, the patient was comfortable at rest with a New York Heart Association (NYHA) functional class II status. Vital signs demonstrated normotension with Blood Pressure (BP) of 107/66 mmHg, a regular Heart Rate (HR) of 74 beats/min, afebrile temperature of

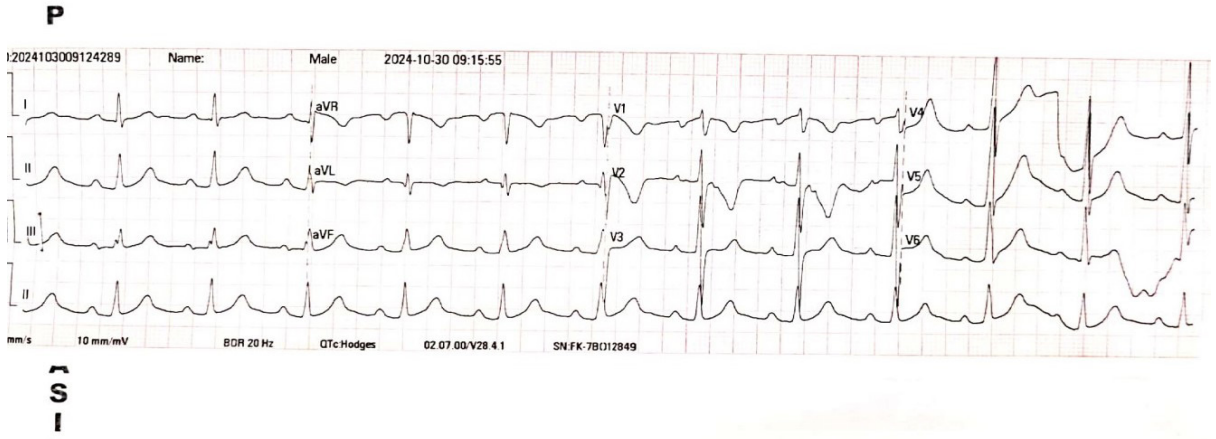

Figure 1. The electrocardiogram shows a prolonged PR interval and T-wave inversion in leads V1-V2.

36.5°C, respiratory rate 20 times/min, and oxygen saturation on room air was 92%, which increased to 94% with supplemental oxygen at 3 L/min via nasal cannula. Exercise tolerance was mildly reduced; the patient experienced shortness of breath after climbing one flight of stairs or walking approximately 100–200 meters at a normal pace. The patient's Body Mass Index (BMI) was 21.6 kg/m^2, which falls within the normal range according to the Asia-Pacific classification for Asian populations. Thoracic examination revealed pectus excavatum, symmetrical chest movements, and no visible ictus cordis. Chest palpation showed good fremitus conduction bilaterally and a palpable thrill on the left sternal border. On percussion, the upper left cardiac margin was identified at the second intercostal space along the left parasternal line. At the same time, the lower border was found at the sixth intercostal space at the left midclavicular line, and the right margin at the fifth intercostal space near the right parasternal line. Cardiac auscultation demonstrated a harsh grade 5/6 systolic ejection murmur that was audible across the precordium, with maximal intensity at the left parasternal area, particularly at the second to fourth intercostal spaces. The murmur could also be heard along the right sternal border. Lung auscultation revealed normal vesicular breath sounds bilaterally. Examination of the mucous membranes revealed no central cyanosis, and all four extremities were warm with good perfusion.

On Electrocardiography (ECG), a prolonged PR interval with a duration of 0.22 – 0.24 seconds, together with T-wave inversion, was noted in leads V1-V2. Additionally, QRS complex spikes were observed in lead III (Figure 1). Chest radiography demonstrated an enlarged cardiac silhouette, with a Cardiothoracic Ratio (CTR) of 0.56 (Figure 2). Laboratory evaluation was generally unremarkable. Nonetheless, a reduced Mean Platelet Volume (MPV) (8.2 fL, reference 9.0-13.0) and a mildly decreased serum creatinine (0.7 mg/dL, reference 0.9-1.3) were observed.

Two-dimensional transthoracic echocardiography was performed using a 3.0-MHz phased-array transducer on a LOGIQ P7 ultrasound system (GE Healthcare). Parasternal long-axis and short-axis views revealed an abnormal muscular bundle that separated the RV into two cavities. Both the left atrium and left ventricular appeared dilated, while the interventricular septum and posterior left ventricular wall were non-thickened. The interatrial septum and interventricular septum were intact, with a normal Ejection Fraction (EF) of 69%, a Tricuspid Annular Plane Systolic Excursion (TAPSE) value of 20 mm, and normokinetic wall motion. No valvular abnormalities or VSD were observed. Continuous wave Doppler across the muscular septation demonstrated a holosystolic antegrade jet with a peak velocity of approximately 2.0 m/s, corresponding to an estimated pressure gradient of 16 mmHg, consistent

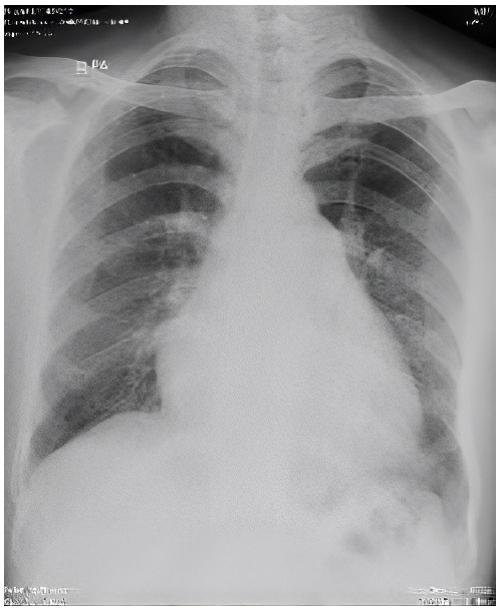

Figure 2. Chest X-ray (posteroanterior view) showing cardiomegaly.

with mild right-ventricular outflow tract obstruction (Figures 3 and 4). Overall, Transthoracic Echocardiography (TTE) indicated preserved left-ventricular diastolic function and normal right-ventricular systolic performance. Based on these results, a diagnosis of DCRV was made. He was referred to Dr. Sardjito General Hospital for further evaluation and was started on beta-blocker therapy to help reduce cardiac workload. However, he declined further evaluation or routine follow-up and surgical consultation, choosing instead to continue with medical management. Consequently, no advanced imaging or surgical intervention was performed.

On follow-up echocardiography performed one year later, TTE demonstrated preserved left-ventricular systolic function (Left Ventricular Ejection Fraction [LVEF] 72%) and normal right-ventricular performance (TAPSE 20 mm). The right atrium appeared dilated with a diameter of 54 mm, while the RV measured 40 mm in true dimension and 26 mm in the mid-cavity region. No new valvular abnormality or residual ventricular septal defect was identified (Figures 5). Continuous-wave Doppler aligned to the mid–right-ventricular jet recorded a peak velocity of 3.29 m/s and an estimated pressure gradient of 43 mmHg (calculated by $4v^2$; sweep speed 50 mm/s), indicating moderate right-ventricular outflow tract obstruction (Figure 6).

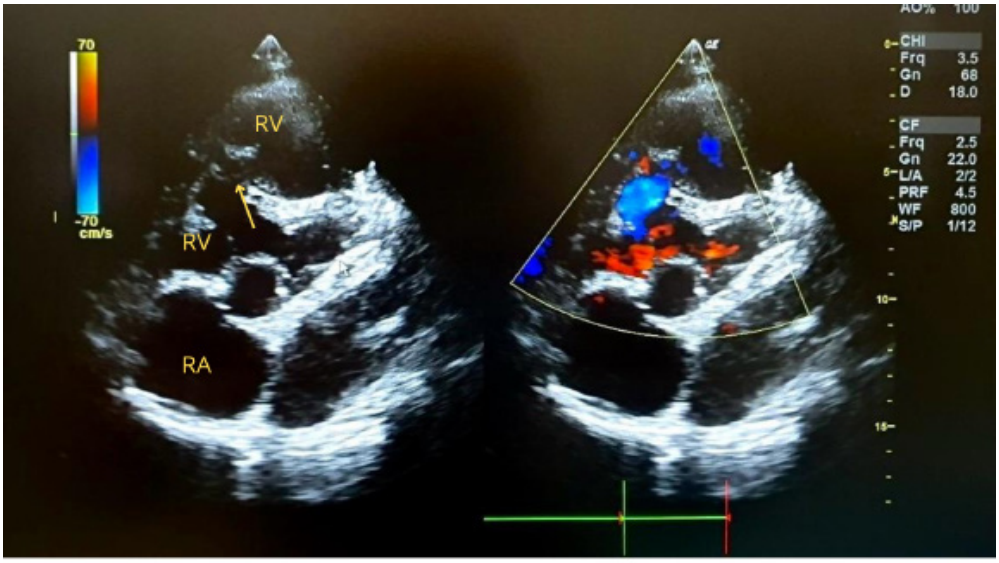

Figure 3. Two-dimensional transthoracic echocardiography (parasternal short-axis view at the level of the mitral valve leaflets) without (left) and with (right) color-flow Doppler demonstrates an abnormal muscular bundle that separates the RV into two cavities.

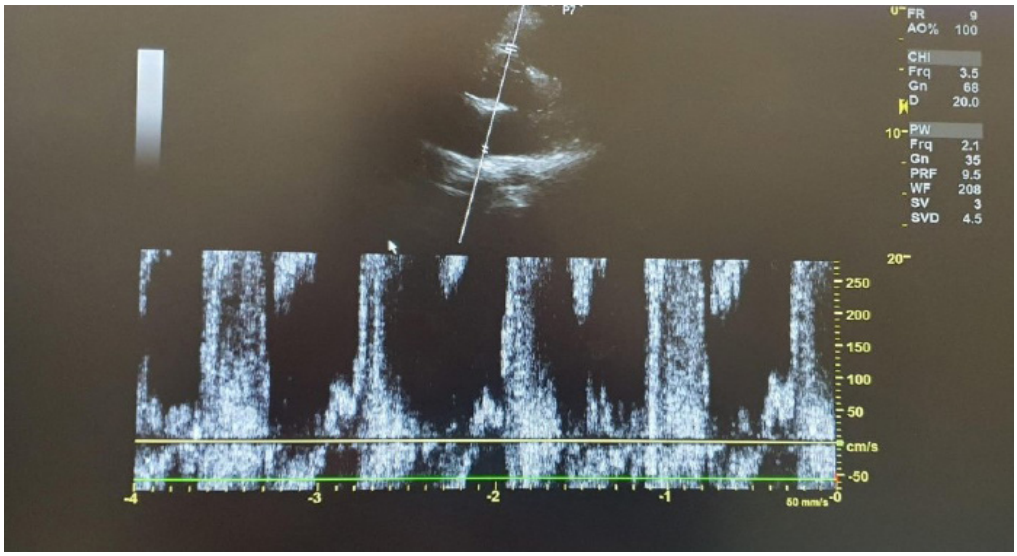

Figure 4. Continuous-wave spectral Doppler across the muscular septation demonstrates a peak holodiastolic antegrade continuous flow gradient, indicative of RV outflow obstruction.

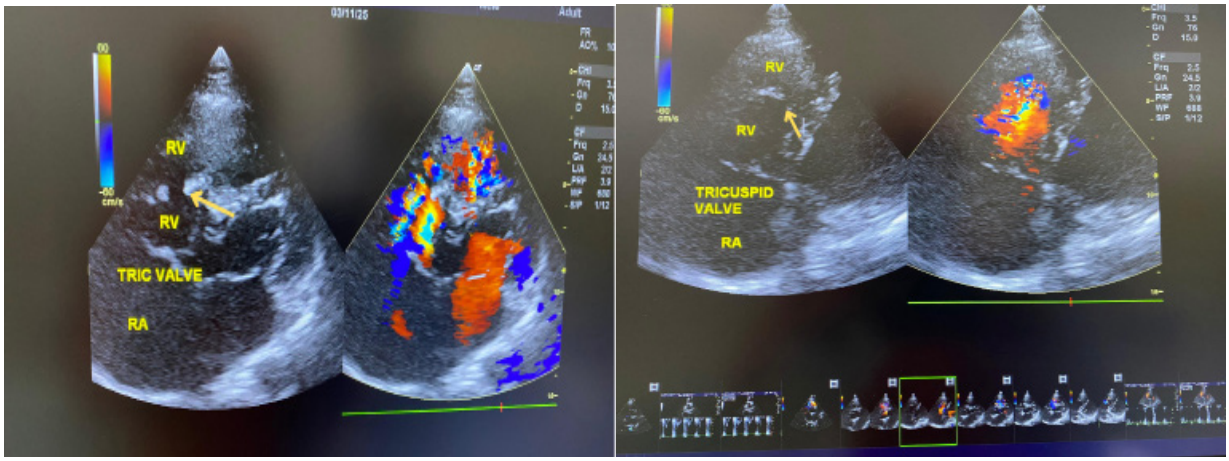

Figure 5. Follow-up TEE shows RV-focused four-chamber and RV-inflow views demonstrate a hypertrophied mid-RV muscular bundle with color-Doppler aliasing across the obstruction. Continuous-wave Doppler aligned through the jet (parasternal short-axis at the great vessels) recorded Vmax 3.29 m/s (peak ≈43 mmHg, $4v^2$), indicating moderate intracavitary obstruction consistent with DCRV.

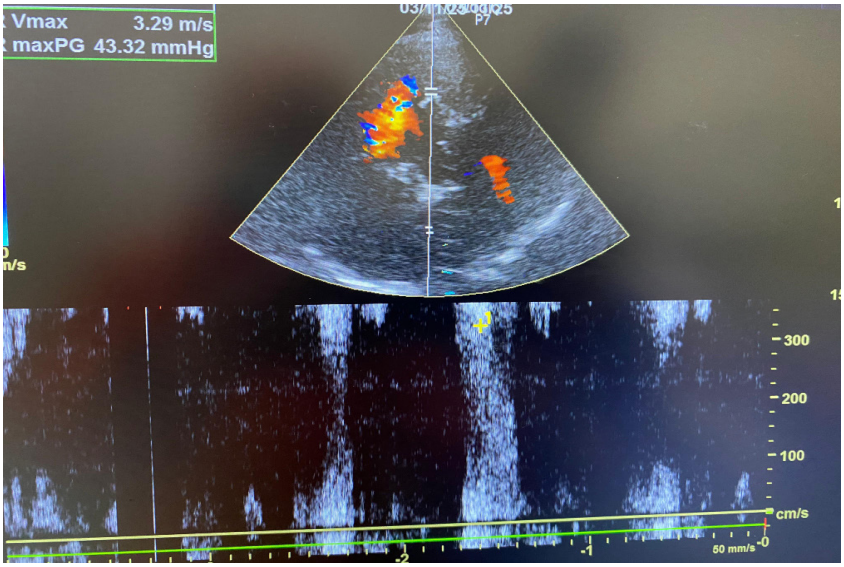

Figure 6. CW-Doppler through the mid-RV obstruction showing Vmax 3.29 m/s and peak gradient 43.3 mmHg (sweep 50 mm/s), consistent with moderate mid-RV obstruction in DCRV.

Discussion

DCRV is a congenital heart defect in which the RV is partitioned into two cavities: a proximal chamber with elevated pressure and a distal chamber with lower pressure.[5] Ventricular muscle fibers generally extend from the Atrioventricular (AV) junction to the ventriculo-arterial junction. The RV has three components: the inlet, apical trabecular, and outlet regions. The inlet segment accommodates the tricuspid valve and its supporting apparatus, which regulate appropriate valve opening and closure. The trabecular region extends to the ventricular apex and is typified by thin myocardial walls and prominent trabeculations. In contrast, the outlet region, also termed the infundibulum, arises from the ventricular base as a uniform muscular layer that supports the pulmonary valve leaflets. This region is smoother than the other regions because it lacks trabecular muscles.[4]

Based on these anatomical features, several hypotheses have been proposed to explain the development of DCRV. In DCRV, abnormal hypertrophy of muscle bundles occurs in certain parts of the RV, particularly in the trabecular or outlet regions. One of the main causes is the adaptation of the RV to increased flow through a VSD, leading

to hypertrophy of the supraventricular crest and the formation of an obstruction. In addition, displacement of the septomarginal trabeculation or moderator band to a higher position also contributes to hypertrophy. This displacement results in abnormal thickening of the muscle, which forms a new muscular layer connecting the septoparietal trabeculation with the apical trabecular region of the RV. The thickened muscle forms a muscular bundle that obstructs blood flow, separating the RV into proximal and distal chambers with different pressures. As a result, obstruction occurs and disrupts normal blood flow within the RV.[5]

Garg et al. (2020) reported that DCRV can be classified into two patterns of intracavitary obstruction. Type 1 is defined by the presence of an anomalous muscular bundle crossing the RV cavity, considered the main cause of intraventricular obstruction. In contrast, type 2 is primarily related to marked hypertrophy of the parietal and septal walls. The pressure gradient within the ventricle tends to be greater in type 1 compared with type 2. An underlying VSD is most often linked with type 2 DCRV.[6] This case is classified as type 2 DCRV based on the patient's history of VSD during childhood. The absence of valvular abnormalities or VSD on TTE can be explained by the fact that VSDs are known to undergo spontaneous closure as children grow, especially in small defects, through various mechanisms. One possible mechanism is the proliferation of fibrous tissue surrounding the defect margins, which gradually results in septal closure. In membranous VSD, closure can occur through attachment of the septal leaflet of the tricuspid valve to the defect margin, a mechanism known as endocardial adhesion. Turbulent blood flow through the VSD may also damage the endocardium, producing a jet lesion that contributes to defect closure. In muscular VSDs, myocardial growth around the defect may contribute to defect closure.[7]

DCRV can develop progressively postnatally. Hypertrophy of the muscular bundles driven by high velocity jet flow may lead to mid-cavitary obstruction that becomes clinically apparent only in adulthood. Even when the VSD later closes, the established muscular remodeling can sustain or further the progression of the obstruction.[3] In our patient, the persistent anomalous right-ventricular muscular bundle and the absence of a VSD are consistent with this trajectory. Several studies have reported that asymptomatic adults with an anomalous muscular bundle and an intact ventricular septum might have previously had a VSD that spontaneously closed.[8] DCRV has been reported to occur many years after the closure of a VSD. It is speculated that the persistent effect of high-velocity VSD jets on the RV wall may, over time, activate genetic factors that promote the release of growth signals and cause thickening of the heart muscle, even after the VSD has already closed.[9] Accordingly, a prior VSD that closed spontaneously remains plausible, and the absence of a current shunt is consistent with this. Nevertheless, in this patient, the mid-ventricular obstruction may still progress because jet-driven muscular hypertrophy can develop postnatally and

Table 1. Comparison with previously published adult DCRV cases.

Study (Author, Year)	Age/Sex	Mode of Detection & Symptoms	Obstruction Site	Imaging	Hemodynamics	ECG	Management
Garg et al., 2020	18-year-old male	Progressive shortness of breath for the past 2 months	A muscle band protruding from the RV free wall to the interventricular septum	Two-dimensional TTE using a 3.0 MHz transducer and a Vivid S5 cardiac ultrasound system (GE Healthcare, Milwaukee, WI, USA)	Continuous-wave Doppler across this turbulent jet revealed flow acceleration of 6.0 m/s, corresponding to a pressure gradient of approximately 144 mmHg	Increased amplitude of the R wave on V1, inverted T waves on V1–V6, and right-axis deviation	Refused for operative correction.
Malone et al., 2023	40-year-old patient	Routine evaluation of VSD, asymptomatic	RV septation by muscular bundles and flow acceleration. The pulmonic valve is normal, with no evidence of VSD	TTE, Cardiac catheterization, and ventriculogram	TTE on the parasternal short-axis view was suggestive of DCRV, with the highest intracavity gradient measured at 39 mm Hg	Not available	Surgical resection for DCRV

Malone et al., 2023	20-year-old patient	Severe pulmonary stenosis and RVH with concerns for tetralogy of Fallot	RVH with hypertrabeculation	TTE, CMR	A severe gradient across the RVOT. Peak and mean gradients 96 and 41 mmHg	RVH & RV strain pattern	Surgical closure of the VSD and resection of the RV muscle bundles and outflow tract obstruction.
Romano et al., 2007	29-year-old man	Mild exertional dyspnea, dizziness, and chest pain during the last 3 years	A 17 mm perimembranous VSD. Slow velocity flow from left to RV; the aortic root was slightly deviated to the right. RV hypertrophy and a muscular septation inside this cavity	TTE	A peak gradient of 80 mmHg.	Sinus rhythm, RAD, right ventricular overload and a minor degree of Right Bundle Branch Block (RBBB)	Surgical resection
Satria et al., 2021	36-year-old woman	Pulmonary stenosis	Perimembranous outlet (PMO) VSD with left-toright shunts	TEE & TTE	Left-to-right shunts of 0.7-0.8 cm in diameter, pressure gradient of 100 mmHg, visible bulkhead at 1/3 distal RV, turbulence (+) with a pressure gradient of 130 mmHg between the two walls	Not available	Surgery with VSD closure and resection of the septum.

ECG: Electrocardiogram; RV: Right Ventricle; VSD: Ventricular Septal Defect; DCRV: Double-Chambered Right Ventricle; RVH: Right Ventricular Hypertrophy; CMR: Cardiac Magnetic Resonance; RVOT: Right Ventricular Outflow Tract; TTE: Transthoracic Echocardiography; RAD: Right Axis Deviation; TEE: Transesophageal Echocardiography.

persist after the defect has closed. Management should therefore be guided by the severity and progression of right-ventricular obstruction and patient symptoms, rather than shunt status alone.

The history of infective endocarditis in this patient may be related to the hemodynamic disturbances and pressure gradients within the cardiac chambers. Vegetations on the endocardium usually form in areas with pressure gradients that cause turbulent blood flow. Congenital heart disease with high-velocity blood flow, with or without prosthetic material, increases the risk of infective endocarditis. Any lesion that causes turbulent blood flow, regardless of the presence of a shunt, can predispose to infective endocarditis. Individuals with congenital heart disease are at particularly high risk for endocarditis. Turbulent blood flow from a high-pressure chamber to a low-pressure chamber, or through a narrow defect, can injure the endothelium and promote thrombus formation, resulting in a sterile platelet and fibrin clot known as nonbacterial thrombotic endocarditis. This condition provides an ideal environment for bacterial adherence and the formation of infected vegetations. Endothelial lesions are usually located on the low-pressure side, so vegetations are often found on the atrial side of the AV valves or distal to the descending aorta in cases of aortic coarctation.[10]

In this case, TTE also showed left atrial and left ventricular dilation. These findings may be related to chronic hemodynamic disturbances in congenital heart disease. In VSD, abnormal shunting can increase pulmonary blood flow and pulmonary venous return, resulting in left-sided volume

overload. As tissue metabolic demands increase, the left ventricle may compensate by increasing stroke volume and heart rate determines LA and LV dilation.[11]

Diagnosing DCRV in adulthood is particularly challenging and frequently misinterpreted as pulmonary stenosis. Symptoms are often absent, leading to misdiagnosis. Patients with DCRV are reported to remain asymptomatic until they become symptomatic due to progressive obstruction, which eventually creates a high gradient between the two chambers.[7,12] In this case, the patient reported no symptoms and maintained normal activities, only occasionally experiencing palpitations and blurred vision due to his history of hypotension. DCRV was incidentally discovered when the patient presented with abdominal pain. A study by Malone et al. (2024) also reported that two-thirds of DCRV patients were asymptomatic.[2] Two-dimensional TTE and Doppler imaging are capable of reliably identifying DCRV in both pediatric and adult populations. In one series, TTE confirmed the diagnosis in 26 out of 32 patients (81%), demonstrating its value in characterizing structural abnormalities and estimating intraventricular pressure gradients.[2] Nevertheless, despite its utility, TTE does not always provide optimal anatomical visualization. In such situations, Transesophageal Echocardiography (TEE) offers more detailed imaging of cardiac structures, subpulmonic gradients, valve motion, and subinfundibular narrowing. Nevertheless, TTE remains the preferred initial modality for detecting DCRV.[9]

Surgical resection of intraventricular obstruction is an effective and durable treatment for patients with DCRV.[2] According to the latest American Heart Association and American College of Cardiology (AHA/ACC) guidelines, operative intervention is recommended for symptomatic adults with congenital heart disease who present with moderate Right Ventricular Outflow Tract (RVOT) obstruction, indicated by a Doppler velocity of ≥3.0 m/s or a peak gradient of ≥36 mmHg. Evidence from other studies also highlights that surgery is commonly undertaken in patients with DCRV when the Right Ventricular Pressure (RVP) gradient exceeds 40 mmHg, or in the presence of significant left-to-right shunting due to severe VSD (Qp/Qs ≥ 2.0). Surgical repair should aim to eliminate the obstruction by resecting the hypertrophied muscular bundles in the RVOT to reduce pressure gradients. The obstruction in DCRV can lead to increased proximal chamber pressure, resulting in significant clinical symptoms such as dyspnea or heart failure. Therefore, the most common management is surgical resection of the abnormal muscular bundle causing the obstruction.[9]

At the 12-month follow-up, continuous-wave Doppler across the mid-RV obstruction showed Vmax of 3.29 m/s (peak = 43 mmHg), meeting commonly cited operative thresholds for DCRV (≥40 mmHg). This was discussed with the patient, and surgery was declined. Because TEE adequately delineated the obstructing bundle with reproducible Doppler measurements, and the patient declined further work-up and surgery, TEE/CMR/CT Cardiac was not performed. Acknowledging that Doppler may overestimate intracavitary gradients relative to catheterization, invasive confirmation will be obtained if a future operative decision hinges on gradient magnitude.

However, since the obstruction in DCRV is dynamic, pharmacological treatment can also be used to help manage this condition. Beta-blockers and calcium channel blockers can help reduce RVP by decreasing myocardial contractility and slowing the HR. Nevertheless, the effectiveness of these medications is limited, and they are generally considered as temporary management or for patients who cannot undergo immediate surgery.[9]

Beta blockers are recommended as first-line therapy for patients with Heart Failure and Reduced Left Ventricular Ejection Fraction (HFrEF), including those with Dilated Cardiomyopathy (DCM). For DCRV, the recommendation is similar, but caution is advised in cases of severe RV dysfunction or acute heart failure, as beta blockers may worsen hemodynamics in these settings. Several landmark trials and meta-analyses have shown that beta blockers improve survival and reduce hospitalizations in patients with HFrEF, including those with DCM.[13]

Because DCRV obstruction has a dynamic component, pharmacologic therapy may assist symptom control when surgery is deferred. β-blockers can control DCRV with dynamic obstruction. In this patient who declined surgery and had preserved biventricular function (LVEF 72%, TAPSE 20 mm), we initiated a low-dose beta blocker to reduce HR/contractility and thereby blunt the dynamic gradient.[3] Given a PR interval of 0.22–0.24 s (first-degree AV block), β-blocker therapy was started cautiously, with ECG/clinical monitoring and avoidance of additional AV nodal blocking agents, recognizing that first degree AV block is not an absolute contraindication, yet AV

nodal drugs can worsen conduction or precipitate higher-degree block, treatment would be withheld if PR prolongation, symptomatic bradycardia, or hypotension occurred. In line with the 2018 ACC/AHA/HRS bradycardia guideline, first-degree "marked" AV block is defined as a PR interval > 0.30 ms. Because β-blockers are listed among medications that can induce/exacerbate bradycardia or conduction disorders, their use in first-degree AV block is not absolutely contraindicated but warrants careful ECG surveillance and continued avoidance of other AV-nodal blockers.[14]

Conclusion

DCRV is an unusual congenital heart condition where abnormal thickening of the muscular bundles within the ventricle divides the RV into two chambers that operate under different pressures. In this case, a 37-year-old male with a history of unrepaired VSD was incidentally found to have type 2 DCRV on echocardiography, without evidence of a VSD, possibly due to spontaneous closure. Diagnosis of DCRV in adulthood is often mistaken for pulmonary stenosis, especially in patients with mild or asymptomatic presentations. TTE and Doppler studies are essential for identifying and confirming the diagnosis, although TEE or advanced imaging may be needed in more complex cases. Management includes symptomatic treatment, such as the use of beta-blockers, and further evaluation to determine the need for surgical intervention. This case emphasizes the value of prompt diagnosis and comprehensive assessment of congenital heart disease in adults.

List of Abbreviations

AV Atrioventricular
BMI Body Mass Index
BP Blood Pressure
CTR Cardiothoracic Ratio
DCM Dilated Cardiomyopathy
DCRV Double-Chambered Right Ventricle
ECG Electrocardiography
EF Ejection Fraction
HFrEF Heart Failure and Reduced Left Ventricular Ejection Fraction
HR Heart Rate
MPV Mean Platelet Volume
NYHA New York Heart Association
RBBB Right Bundle Branch Block
RV Right Ventricle/Ventricular
RVOT Right Ventricular Outflow Tract
TAPSE Tricuspid Annular Plane Systolic Excursion
TEE Transesophageal Echocardiography
TTE Transthoracic Echocardiography
VSD Ventricular Septal Defect

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors Contributions

DAY conceptualized and designed the study, conducted clinical data acquisition, drafted and critically revised the manuscript; PDA drafted and critically revised the abstract and manuscript; PIMY provided clinical data support, conducted the literature review, and revised the manuscript; RRI assisted with the discussion section, refined, proofread, and revised the manuscript; NN provided clinical supervision, validated supporting diagnostic findings, and critically revised the manuscript. All authors approved the final version

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Conflict of Interest

The authors declare no conflicts of interest.

Availability of Data and Materials

Not applicable.

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Generative AI and AI-Assisted Technologies in the Writing Process

Generative AI was used solely to improve the language (grammar and phrasing). The authors reviewed, edited, and take full responsibility for the content, including the accuracy of all clinical information, interpretations, and references.

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