Cureus
Part of SPRINGER NATURE

Open Access Case Report

# Double-Outlet Left Atrium in a One-Month-Old Boy: A Rare Heart Anomaly in a Resource-Limited Setting

**Review began** 10/28/2026
**Review ended** 11/07/2026
**Published** 11/13/2026

© **Copyright** 2026
Nakato et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

**DOI:** 10.7759/cureus.119586

Aisha Nakato [1], Emma Tina Ndagire [1], Hilda Tumwebaze [1], Gloria Owomugisha [1], Twalib Aliku [1], Sulaiman Lubega [1]

1. Pediatric Cardiology, Uganda Heart Institute, Kampala, UGA

**Corresponding author:** Aisha Nakato, nakaisha@gmail.com

## Abstract

Double-outlet left atrium (DOLA) is an exceedingly rare congenital cardiac anomaly caused by ventriculoatrial malalignment. Its presentation ranges from subtle cyanosis to delayed manifestations resembling an atrial septal defect. We report a one-month-old male infant who presented with cyanosis and symptoms of heart failure. Echocardiography showed a rare variant of double-outlet atrium, in which the right atrium emptied solely through a patent foramen ovale (PFO) into the left atrium, which drained into both ventricles through a common atrioventricular (AV) valve. Treatment with intravenous furosemide and supplemental oxygen did not produce significant improvement. He was too unstable for surgical intervention, deteriorated, and died three days after diagnosis. The presumed cause of death was refractory heart failure or an arrhythmia. His parents declined a postmortem examination, so pathological confirmation was not possible. This case illustrates the diagnostic challenges of DOLA, which can mimic a complete AV canal defect, and shows that careful echocardiographic assessment of the AV junction is key to diagnosis. Clinical instability at diagnosis limited surgical options in our patient, highlighting the potential value of early fetal and neonatal cardiac screening.

**Categories:** Radiology, Cardiology, Pediatrics
**Keywords:** case report, common atrioventricular valve, cyanosis, double-outlet left atrium, heart failure

## Introduction

Double-outlet atrium is an extremely rare congenital cardiac malformation in which the atria are malaligned with the ventricles. It is thought to arise during embryogenesis when the ventricles and ventricular septum are displaced excessively relative to the atria and atrial septum [1]. Double-outlet left atrium (DOLA), in which the left atrium empties into both ventricles, is considerably less common than double-outlet right atrium (DORA), and only a handful of cases have been described [2].

The term “double-outlet atrium” has been applied to a heterogeneous group of lesions. Some have the morphology of an atrioventricular (AV) septal defect with malalignment between the atrial and ventricular septal structures. Others show straddling and overriding of an AV valve when one AV connection is absent [3]. The nature of the AV valves forming the left atrial outlet is still debated: they may represent a single straddling valve or distinct mitral and tricuspid valves. In right atrial outlet atresia, the right AV valve is atretic, so the remaining left AV valve cannot be said to guard a common AV junction and is not a true common AV valve. Following Van Mierop’s reasoning, this valve has been divided embryologically into right and left components that resemble the tricuspid and mitral valves [4]. The anomaly may coexist with complex intracardiac anatomy and can be detected prenatally. Vaksmann et al. reported a fetus with DOLA and complex anatomy that was identified on echocardiography, with a normal karyotype on amniocentesis [5].

Most published reports describe two AV valves, including one case with right atrial outlet atresia and parachute left-sided AV valve leaflets [6]. Patients typically present with cyanosis due to obligatory mixing of systemic and pulmonary venous blood in the left atrium, and management is surgical, tailored to ventricular size and associated intracardiac lesions [7]. Because of its rarity and its resemblance to more common shunt lesions, DOLA is easily missed. We report a one-month-old infant who presented with heart failure and cyanosis and was found to have DOLA with a common AV valve, adding to the limited literature on this entity.

## Case Presentation

A one-month-old male infant, born at term, presented to a peripheral hospital with fast breathing since birth. His parents had also noted bluish discoloration of the lips when crying, poor weight gain, excessive sweating (especially on the forehead), and a suck-rest-suck feeding pattern suggestive of heart failure. There were no significant prenatal or perinatal events. The mother had only one obstetric ultrasound scan, done in the first trimester. He was the second-born of two siblings; his birth weight was 3.2 kg; and there was no

known family history of congenital heart disease. He was referred to our institution, the Uganda Heart Institute (UHI), for echocardiography.

On examination, he weighed 2.8 kg with a length of 50 cm, and his weight-for-length z-score was -2 SD. He had no obvious dysmorphic features. There was central cyanosis on crying, but no conjunctival pallor or peripheral edema, and the chest wall had no deformities. He was tachypneic, with a respiratory rate of 70 breaths/min, and postductal oxygen saturation (right foot) was 85% on room air. Air entry was equal bilaterally, with bronchovesicular breath sounds and bilateral fine crepitations. Cardiovascular examination revealed tachycardia (heart rate: 162 beats/min) and a blood pressure of 93/59 mmHg (mean arterial pressure: 69 mmHg), which was within normal limits. Heart sounds were normal, and there was a grade 2/6 pansystolic murmur at the left lower sternal border and mitral area. The abdominal examination was normal.

Laboratory investigations showed a normal total white blood cell count of $8.9 \times 10^9$/L, with an absolute neutrophil count of $1.6 \times 10^9$/L and a lymphocyte count of $6.18 \times 10^9$/L. The hemoglobin level was 14.8 g/dL (MCV: 85.4 fL; MCH: 27.1 pg; MCHC: 31.7 g/dL), and the platelet count was $345 \times 10^9$/L.

A babygram performed at the referring facility demonstrated cardiomegaly, with a double-density sign along the right cardiac border and an upwardly displaced cardiac apex suggestive of right ventricular hypertrophy, together with pulmonary plethora. The abdominal situs was normal, and there were no features suggestive of a diaphragmatic hernia (Figure *1*). Electrocardiography demonstrated a sinus tachycardia with first-degree AV block, a superior QRS axis, left atrial enlargement, and right ventricular hypertrophy (Figure *2*).

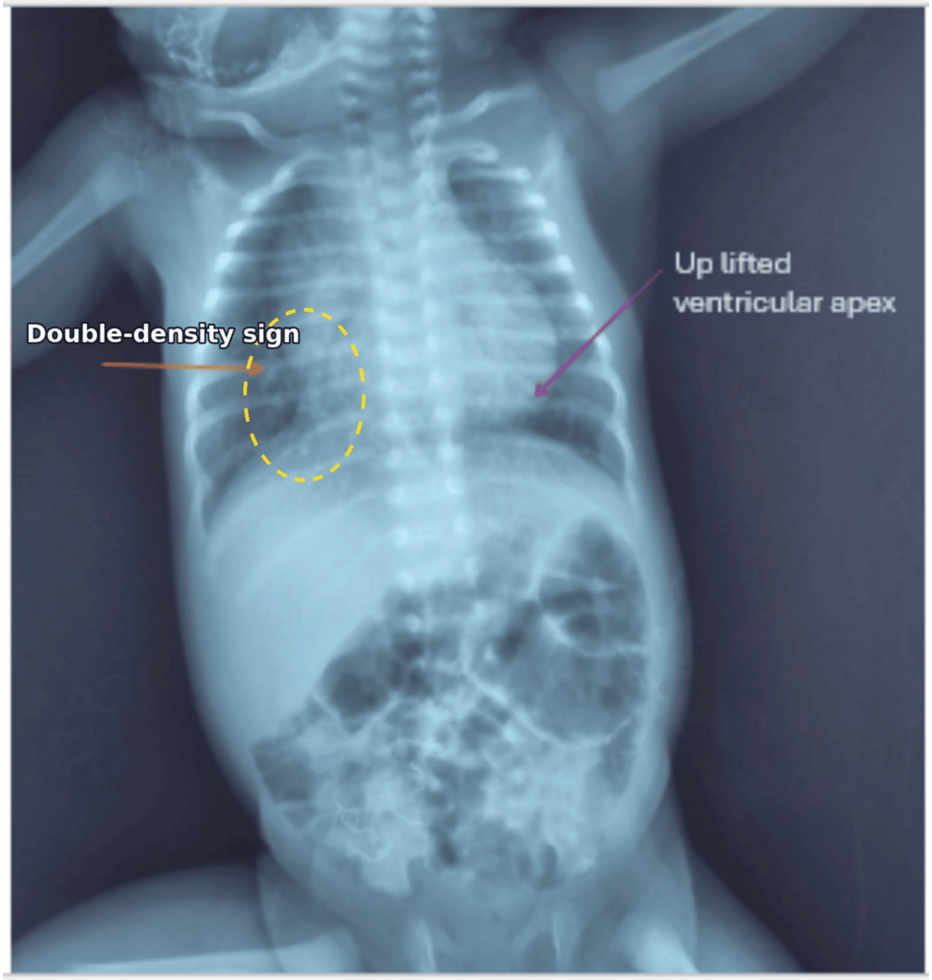



**FIGURE 1: Babygram showing cardiomegaly with pulmonary plethora (orange arrow), a double-density sign along the right cardiac border (yellow outline), and an upwardly displaced cardiac apex (purple arrow)**

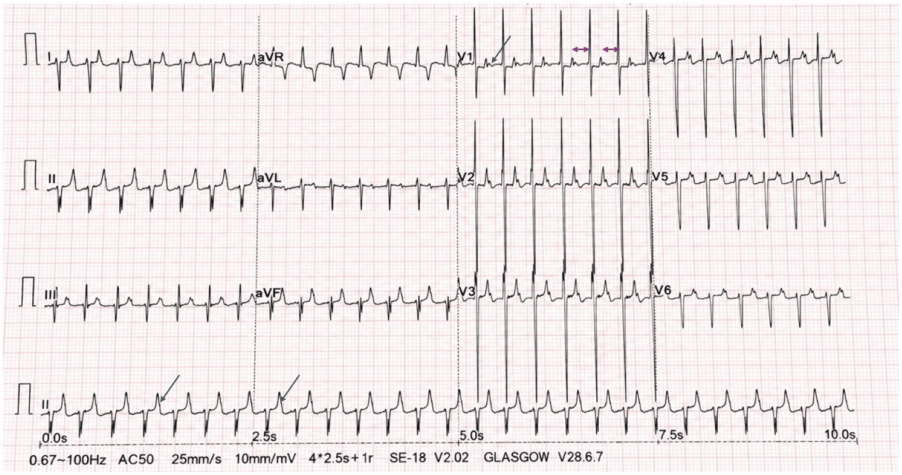



**FIGURE 2: A regular, narrow-complex tachycardia at a rate of 162 beats/min (sinus tachycardia) (blue arrow). A superior axis in limb leads (II, aVF). Prolonged PR interval (lead V1) (purple double arrow); a dominant R-wave in leads (V1 and aVR) and mid-precordial leads (V2-V4), which show very tall complexes, are suggestive of right ventricular hypertrophy. Left atrial enlargement in precordial lead V1**

Transthoracic echocardiography showed atrial situs solitus, levocardia, and D-looped ventricles with normal systemic and pulmonary venous drainage. There was no right atrium (RA) to right ventricle (RV) connection, and the only egress of blood from the right atrium was through a moderately sized, nonrestrictive patent foramen ovale (PFO) with a right-to-left shunt into the left atrium. The dilated left atrium drained into both the right and left ventricles through a common AV valve with chordal attachment to the crest of the ventricular septum, consistent with DOLA (Figure *3*). There was moderate regurgitation of the left AV valve component and mild regurgitation of the right. The ventricles were balanced, with an atrioventricular valve index (AVVI) of 0.47. Biventricular systolic function was preserved, with a left ventricular ejection fraction (LVEF) of 84% and fractional shortening (FS) of 49%. The left ventricular internal diameter was 1.4 cm in diastole (LVIDd) and 0.7 cm in systole (LVIDs). A small inlet ventricular septal defect (VSD) of 4 mm with a left-to-right shunt was also found. The great arteries were normally related, and there was no evidence of right or left ventricular outflow tract obstruction (Videos *1*-*5*).

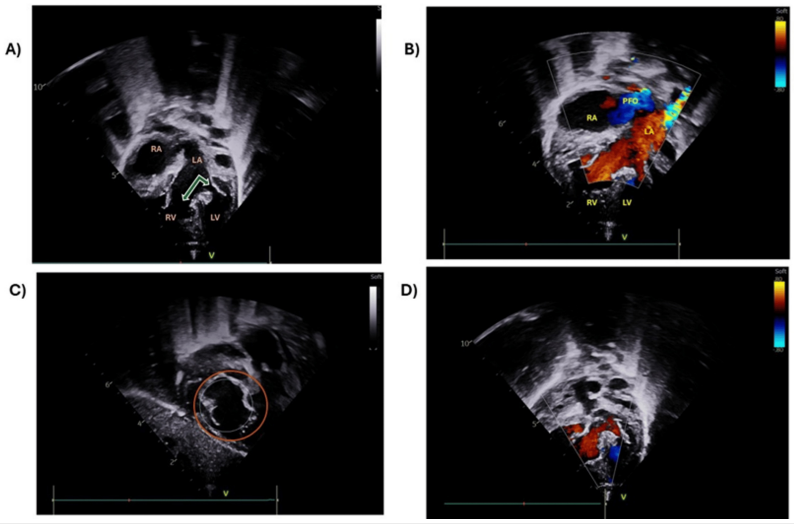



**FIGURE 3: Two-dimensional and color Doppler echocardiography: apical four-chamber view showing (A) a moderately sized nonrestrictive PFO 4 mm with a right-to-left shunt; (B) right atrial outlet atresia, with the left atrium opening into both the right and left ventricular inflows, hence DOLA (green double arrow); subcostal views (two-dimensional and color Doppler) showing (C) a common atrioventricular valve (orange ring); (D) chordal attachment to the ventricular crest, and balanced ventricles**

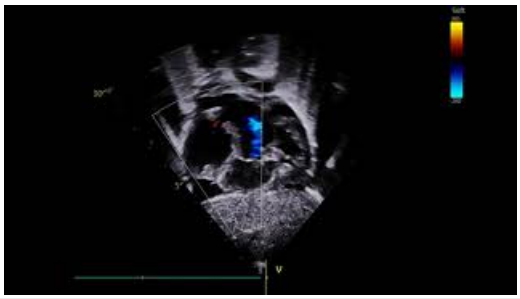
RA: right atrium; RV: right ventricle; LA: left atrium; LV: left ventricle; PFO: patent foramen ovale; DOLA: double-outlet left atrium



**VIDEO 1: Subcostal view 2D echocardiography showing a moderately sized nonrestrictive PFO (4 mm)**

PFO: patent foramen ovale

View video here: https://youtu.be/Rtj0vCMIEkk

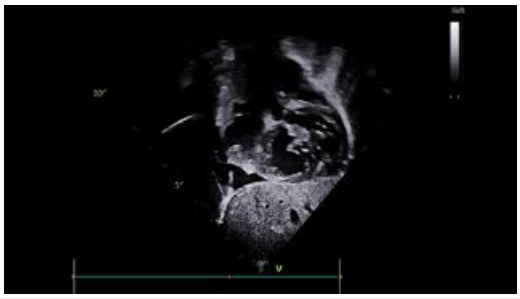


**VIDEO 2: Subcostal view color flow Doppler echocardiography showing**

**a moderately sized nonrestrictive PFO with a right to left shunt**

PFO: patent foramen ovale

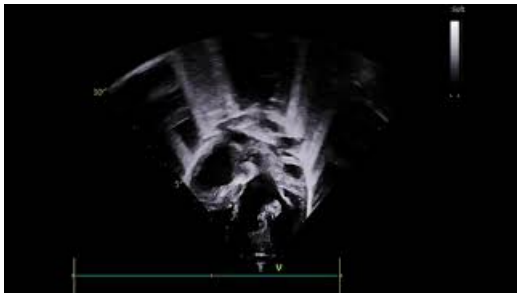
View video here: https://youtu.be/njjvVpMvRLs



**VIDEO 3: Subcostal view 2D shows a common AV valve with chordal attachment to the ventricular crest and balanced ventricles**

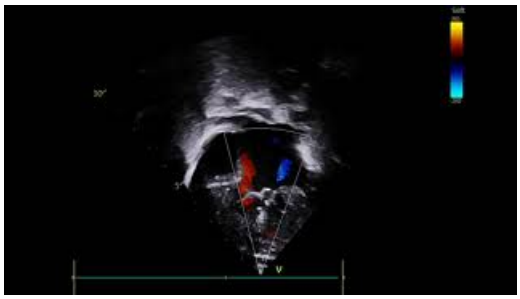
AV: atrioventricular

View video here: https://youtu.be/G-whtStvZiE



**VIDEO 4: Apical four chamber view 2D shows right outlet atresia with the left atrium opening into the right and left ventricle**

View video here: https://youtu.be/uHKXO8f-T4s



**VIDEO 5: Apical four chamber view color Doppler shows right outlet atresia with the left atrium opening into the right and left ventricle**

View video here: https://youtu.be/NiuQ8RhPUR4

Following the diagnosis, the patient was admitted and commenced on intravenous furosemide (3 mg twice daily, approximately 1 mg/kg per dose), antibiotics (piperacillin-tazobactam 280 mg every eight hours and gentamicin 14 mg once daily) for presumed pneumonia, and supplemental oxygen via nasal prongs, with oxygen saturation improving to 93-95%. Despite medical management, there was no significant clinical improvement. He remained critically ill, with severe respiratory distress, intermittent fevers, bilateral crepitations, tachycardia (heart rate: 176-217 beats per minute), and progressive hepatomegaly, increasing from 2 cm to 6 cm below the costal margin. He was deemed too unstable for surgical intervention, and management remained medical.

On the third day of admission, he suffered a cardiac arrest. The ward team commenced cardiopulmonary resuscitation (CPR), comprising bag-valve-mask ventilation, chest compressions at a compression-to-ventilation ratio of 15:2, adrenaline boluses at 0.01 mg/kg every four minutes, and a 10% dextrose bolus. Despite 30 minutes of resuscitation, return of spontaneous circulation was not achieved, and he was pronounced dead. The presumed cause of death was refractory heart failure or an arrhythmia. His parents declined a postmortem examination, which limited further pathological evaluation. The timeline of clinical events is summarized in Table *1*.

| Time point | Clinical events and findings |
|---|---|
| **Prenatal period** | **One obstetric ultrasound scan in the first trimester; no febrile illness reported** |
| Birth and early history (birth to 1 month) | Term male infant; birth weight 3.2 kg; no significant perinatal events |
| Presentation (1 month of age) | Presented at a peripheral hospital with fast breathing, bluish discoloration of the lips on crying, poor weight gain, and excessive sweating |
| Physical examination | Central cyanosis, tachypnea, and oxygen saturation of 85% in room air; heart rate 162 beats/min; blood pressure 93/59 mmHg. Bronchovesicular breath sounds with bilateral fine crepitations. Normal heart sounds and a grade 2/6 pansystolic murmur at the left lower sternal border and mitral area. Normal abdominal examination |
| Chest radiography (referring facility) | Babygram showed cardiomegaly, features suggestive of right ventricular hypertrophy, and pulmonary plethora (Figure 1); referred to UHI for echocardiography |
| Electrocardiography | Sinus tachycardia with first degree atrioventricular block, superior QRS axis, left atrial enlargement, and right ventricular hypertrophy (Figure 2) |
| Transthoracic echocardiography | At UHI, echocardiography demonstrated double outlet left atrium with a common AV valve (Figure 3) (Videos 1-5) |
| Management following diagnosis | Admitted and started on intravenous furosemide, antibiotics for presumed pneumonia, and oxygen supplementation by nasal prongs |
| Clinical course and outcome | No significant clinical improvement. The infant deteriorated and died three days after diagnosis; refractory heart failure or an arrhythmia was the presumed cause of death |
| Postmortem evaluation | The parents declined consent for a postmortem examination |

**TABLE 1: Timeline of clinical events, diagnostic evaluation, management, and outcome**

AV: atrioventricular; UHI: Uganda Heart Institute

## Discussion

Double-outlet atria were first described by Van Mierop as a variant of endocardial cushion defect resulting from extreme malalignment of the atrial septum [4]. More recently, Van Praagh proposed that double-outlet atria represent a ventriculoatrial malalignment defect caused by excessive displacement of the ventricles and ventricular septum relative to the atria and atrial septum during embryogenesis [1].

Embryologically, during the fifth week of fetal development, the ventricular septum normally shifts leftward to align with the atrial septum. This allows the tricuspid valve to communicate with the right ventricle and establishes concordant AV connections between the right atrium and right ventricle and between the left atrium and left ventricle. In DOLA, abnormal ventriculoatrial alignment results in drainage of the left atrium into both ventricles [8].

Under normal conditions, the atrial and ventricular septa intersect at an angle of approximately 15°. Deviation from this relationship has been described in association with common AV canal defects, leading to extreme rightward displacement of the atrial septum. Instead of growing centrally within the AV canal, the atrial septum fuses with the right margin of the canal, resulting in right atrial outlet atresia. Consequently, the only egress of blood from the right atrium is through an atrial septal defect (ASD) [9].

To date, nine cases of DOLA have been documented in the literature [2,3,5-8,10-14], with patient ages ranging from one month to 32 years. All reported patients had an interatrial communication, most commonly an ostium secundum ASD. Similar to our case, three cases have been reported with a common AV

valve [7,8,10], whereas the remainder had two distinct AV valves. Importantly, every previously reported case was associated with additional congenital cardiac anomalies, including pulmonary stenosis or atresia, whereas our patient had only a VSD, with no outlet obstruction or other anomalies [7].

Similar to our case, Espinola-Zavaleta et al. described a one-month-old infant who presented with generalized cyanosis exacerbated by crying and recurrent chest infections. The patient was found to have DOLA with pulmonary atresia, for which a Blalock-Taussig-Thomas shunt was considered; however, the parents declined the procedure [14]. Likewise, Patel et al. documented a 16-month-old child with DOLA who presented with failure to thrive and cyanosis and who underwent successful intracardiac repair [7].

Patients with DOLA may present later in infancy with cyanosis and tachypnea, frequently in association with a large interatrial communication [2,7]. With disease progression, older children and adults may develop exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. Diuretic therapy, such as furosemide, may provide temporary symptomatic relief; however, outcomes are often poor, particularly where follow-up is lost.

As in our patient, diagnosis is challenging when a nonrestrictive interatrial communication is present, because the clinical features may mimic those of an isolated ASD despite persistent cyanosis [2]. Cyanosis is a consistent finding because of obligatory mixing of systemic and pulmonary venous blood within the left atrium. Associated anomalies, including VSD and AV valve regurgitation, can increase pulmonary blood flow and lead to earlier presentation with congestive heart failure, as observed in our patient.

In the initial diagnostic evaluation, a babygram was obtained to provide a broad overview of the cardiothoracic anatomy. The radiograph demonstrated an enlarged cardiac silhouette, raising suspicion for congenital heart disease, and the pulmonary vascular markings were variably increased, consistent with pulmonary overcirculation, as expected from the increased pulmonary blood flow across the VSD in DOLA. The babygram also excluded major thoracic anomalies, thereby ruling out noncardiac causes of neonatal cyanosis such as diaphragmatic hernia.

His electrocardiogram showed a superior axis, which is abnormal for a one-month-old, a pattern that is classically seen with AV septal and common AV valve anatomy. The rate was also within what an infant in heart failure could reach in sinus tachycardia. Tall biphasic complexes across precordial leads (V2-V4) are also seen with large left-to-right shunts and biventricular volume load, which fits the small inlet VSD. First-degree AV block in DOLA is probably related to the underlying anatomical malformation, which may disrupt the conduction system and delay impulse propagation from the right atrium through the AV node to the bundle of His, prolonging the PR interval. While not diagnostic of DOLA, these findings supported the presence of a complex congenital cardiac lesion and underscored the need for definitive characterization by echocardiography.

Key echocardiographic clues for diagnosing DOLA include a left atrium that connects to both ventricles, while the right atrium has no direct outlet except through an ASD or PFO. The hallmark is mixing of systemic and pulmonary venous blood at the atrial level with abnormal AV valve alignment.

Complete AV canal defect (CAVCD) is the principal differential diagnosis for DOLA, as the two conditions share overlapping anatomical and electrocardiographic features. In its extreme form, the ventriculoatrial malalignment underlying DOLA may occur within the AV septal defect spectrum, in which a straddling AV valve closely resembles a common AV valve with bridging leaflets [1,15]. Consequently, a superior QRS axis, classically associated with CAVCD, may also be present in DOLA and cannot reliably differentiate the two. Distinction therefore depends on echocardiographic assessment of the AV junction, specifically whether the right atrium connects to either ventricle and whether the entire left atrium drains across both ventricles.

Tricuspid atresia can be distinguished from DOLA by its pathophysiology. In tricuspid atresia, cyanosis results from complete agenesis of the tricuspid valve, leading to absence of communication between the right atrium and right ventricle. In contrast, DOLA is characterized by either a common AV valve or two separate AV valves, allowing atrial inflow into both ventricles. Ventricular morphology in DOLA varies from a hypoplastic right ventricle to adequately balanced ventricles, unlike the uniformly hypoplastic right ventricle seen in tricuspid atresia. Clinical presentation and severity of cyanosis in tricuspid atresia depend on the degree of pulmonary blood flow obstruction, the presence of a VSD, and the relationship of the great arteries [16].

Management is primarily surgical and depends on ventricular adequacy and associated intracardiac anatomy [12]. Patients with adequately sized ventricles may undergo biventricular repair, whereas those with inadequate ventricular anatomy may require single-ventricle palliation through a cavopulmonary pathway [7,12]. In selected cases, catheter-based interventions such as balloon atrial septostomy may be performed when the ASD or PFO is restrictive, to improve right atrial decompression and systemic oxygenation as a bridge to definitive intracardiac repair, or because the patient is otherwise at high risk of early neonatal death [2].

If there is no VSD or other intracardiac abnormality in DOLA, surgical repair typically involves redirecting the right atrial blood flow to the right ventricle by creating an intra-atrial baffle or by patch closure of the ASD, while preserving the dual outlet of the left atrium to both ventricles [8]. This separates systemic and pulmonary venous return without the need for complex intracardiac rerouting.

Successful intracardiac repair of DOLA has been described by Patel et al. [7] and Bhat et al. [8] via right atriotomy. In these cases, the thickened interatrial septum was excised, and the two AV valves were separated using autologous pericardium to direct the right and left atrial components into their respective ventricles. The atrial patch was fenestrated in view of a hypoplastic right ventricle. Both patients had an uneventful postoperative course, underscoring the feasibility of surgical correction in selected anatomies [7,8].

Patients with DOLA may die from progressive cyanosis, heart failure, arrhythmias, or surgical complications. Outcomes are best when the anomaly is recognized early and repaired in centers experienced in complex congenital heart disease. Similar to our case, a four-month-old female infant with DOLA and a restrictive VSD was scheduled for pulmonary artery banding but died at home following a chest infection [2]. This highlights the vulnerability of infants with complex physiology and the impact of intercurrent illness and delayed definitive intervention on survival.

### Strengths and limitations

The main strength of this report is the detailed echocardiographic characterization of an exceptionally rare congenital cardiac malformation, which illustrates the distinctive intracardiac anatomy and associated abnormalities that contributed to the infant's early presentation. The integration of clinical findings, electrocardiography, chest radiography, and transthoracic echocardiography also illustrates the diagnostic approach to complex congenital heart disease in a resource-limited setting.

This report also has limitations. The mother had only one obstetric ultrasound scan, done in the first trimester, and no fetal echocardiography was performed. This was a missed opportunity for antenatal diagnosis and for planning life-saving interventions in the immediate postnatal period. The diagnosis could not be confirmed by pathological examination because the parents declined a postmortem examination. Advanced cardiac imaging and invasive hemodynamic assessment were not performed, which limited further anatomical and physiological characterization.

The infant's death three days after diagnosis precluded assessment of the response to sustained medical therapy and of the feasibility of definitive surgical intervention, and the presumed cause of death, refractory heart failure or an arrhythmia, could not be confirmed. Finally, the single-case design limits generalizability and precludes conclusions on the optimal management and prognosis of this rare anomaly.

## Conclusions

We report a rare variant of DOLA, characterized by right atrial outlet atresia with no direct communication between the right atrium and right ventricle, such that the left atrium drained into both ventricles. Obligatory interatrial shunting, a ventricular septal defect, and AV valve regurgitation produced cyanosis and congestive heart failure. Because DOLA can mimic a complete AV canal defect, including on electrocardiography, careful echocardiographic assessment of the AV junction is essential for diagnosis. Our patient was too unstable for surgery and died three days after diagnosis. This case highlights the potential value of prenatal screening for congenital heart disease, which may allow planned delivery at a cardiac center and timely surgical assessment, and the importance of meticulous echocardiography in infants with unexplained cyanosis and heart failure. It also adds to the limited literature on this exceptionally rare malformation.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

**Concept and design:** Aisha Nakato, Emma Tina Ndagire, Gloria Owomugisha, Twalib Aliku, Sulaiman Lubega

**Acquisition, analysis, or interpretation of data:** Aisha Nakato, Hilda Tumwebaze

**Drafting of the manuscript:** Aisha Nakato

**Critical review of the manuscript for important intellectual content:** Aisha Nakato, Emma Tina Ndagire, Hilda Tumwebaze, Gloria Owomugisha, Twalib Aliku, Sulaiman Lubega

**Supervision:** Twalib Aliku

## Disclosures

**Human subjects:** Informed consent for treatment and open access publication was obtained or waived by all participants in this study. N/A issued approval N/A. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

## Acknowledgements

I would like to express my sincere gratitude to Dr. Sulaiman Lubega, Dr. Owomugisha, Dr. Emma Tina Ndagire, Dr. Aliku Twalib, Dr. Hilda Tumwebaze, and the entire Pediatric Cardiology Department at the Uganda Heart Institute for their invaluable support and encouragement throughout the preparation of this case report.

# References

1. Praagh RV: What are double-outlet left atrium and double-outlet right atrium? . Ann Pediatr Cardiol. 2013, 6:155-7.
2. Shetkar SS, Kothari SS: Double-outlet left atrium: ventriculo-atrial malalignment defect . Ann Pediatr Cardiol. 2013, 6:158-61. 10.4103/0974-2069.115266
3. Gnanapragasam JP, Houston AB, Anderson RH: Double outlet left atrium with co-existing double inlet left ventricle and discordant atrioventricular connections. Cardiol Young. 1991, 1:155-7. 10.1017/S1047951100000299
4. Van Mierop LH: Pathology and pathogenesis of endocardial cushion defects. Surgical implications . Second Henry Ford Hospital International Symposium on Cardiac Surgery. Davila JC (ed): Appleton-Century-Crofts, New York; 1977. 201-7.
5. Vaksmann G, Bouzguenda I, Houyel L: Antenatal diagnosis of double-outlet left atrium . Cardiol Young. 2017, 27:816-8. 10.1017/S104795111600250X
6. Agrawal G, Chaurasia AK: Unusual case of absent right-sided atrioventricular connection with double outlet left atrium with parachute left-sided atrioventricular valve leaflets. IHJ Cardiovasc Case Rep. 2018, 2:214-7. 10.1016/j.ihjccr.2018.07.001
7. Patel R, Katewa A, Haranal M, Saxena S, Patel P, Patel K: Double outlet left atrium: successful management of a rare entity. Indian J Thorac Cardiovasc Surg. 2023, 39:626-8. 10.1007/s12055-023-01550-3
8. Bhat YA, Pratap H, Dagar KS, Awasthy N: Double-outlet left atrium: successful repair of an extremely rare anomaly. Ann Pediatr Cardiol. 2018, 11:204-6. 10.4103/apc.APC_136_17
9. Coto EO, Calabro R, Marsico F, Lopez Arranz JS: Right atrial outlet atresia with straddling left atrioventricular valve. A form of double outlet atrium. Br Heart J. 1981, 45:317-24. 10.1136/hrt.45.3.317
10. Horiuchi T, Saji K, Osuka Y, Sato K, Okada Y: Successful correction of double outlet left atrium associated with complete atrioventricular canal and l-loop double outlet right ventricle with stenosis of the pulmonary artery. J Cardiovasc Surg (Torino). 1976, 17:157-61.
11. Bini RM, Pellegrino PA, Mazzucco A, Gallucci V, Milanesi O, Maddalena F, Thiene G: Tricuspid atresia with double-outlet left atrium. Chest. 1980, 78:109-11. 10.1378/chest.78.1.109
12. Suzuki Y, Hamada Y, Miura M, Haneda K, Horiuchi T, Ogata H: Double-outlet left atrium with intact ventricular septum. Ann Thorac Surg. 1988, 45:332-4. 10.1016/S0003-4975(10)62476-9
13. Naik A, Nabar A, Dalvi B: Double outlet left atrium . Indian Heart J. 1997, 49:418-20.
14. Espinola-Zavaleta N, Vargas-Barrón J, Hernández-Morales G, Keirns C, Romero-Cárdenas A, Roldán J: Clinical and echocardiographic aspects of double-outlet left atrium . J Am Soc Echocardiogr. 2002, 15:86-9. 10.1067/mje.2002.115888
15. Moss AJ, Adams FH, Emmanouilides GC: Heart Disease in Infants, Children and Adolescents . Williams & Wilkins, Baltimore, MD; 1977.
16. Hofbeck M, Wiegand G, Michel J, Schlensak C: Ventriculoatrial malalignment in atrioventricular septal defect resulting in uniatrial biventricular connection: surgical options. J Cardiothorac Surg. 2020, 15:59. 10.1186/s13019-020-01099-x