Pericardial Agenesis: When Should This Diagnosis Be Considered?

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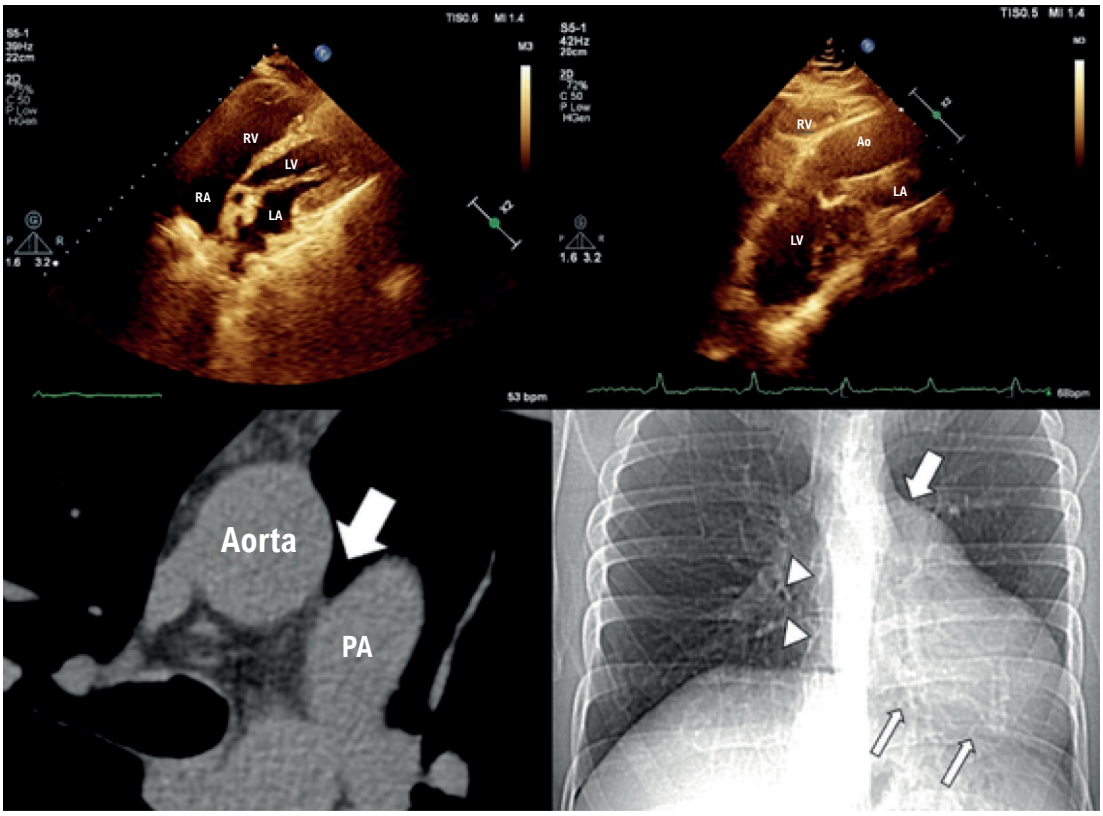
Central Illustration: Pericardial Agenesis: When Should This Diagnosis Be Considered?



Arq Bras Cardiol: Imagem cardiovasc. 2025;38(1):e20250004

The importance of multimodality in the diagnosis of PA. Ao: aorta; PA: pericardial agenesis; LV: left ventricle; RV: right ventricle; LA: left atrium.

Keywords

Pericardium; Pericarditis; X-Ray Computed Tomography; Magnetic Resonance Imaging

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Manuscript received January 29, 2025; revised January 30, 2025; accepted February 3, 2025
Editor responsible for the review: Marcelo Tavares

DOI: https://doi.org/10.36660/abcimg.20250004i

Abstract

Pericardial agenesis (PA) is a rare condition characterized by the partial or complete absence of the pericardium. The consequences associated with this condition can be quite serious. Therefore, understanding the peculiarities of this disease is essential for accurate diagnosis and treatment.

Introduction

PA prevalence is estimated at approximately 0.002% to 0.004% in the general population, often diagnosed incidentally.[1] Studies indicate that this condition occurs more frequently in

males, with a 3:1 ratio between males and females, and may be associated with other congenital anomalies.[2]

Although there is no specific gene directly responsible for the PA, this disease is associated with several genetic anomalies and structural congenital malformations,[2] which suggests a possible genetic predisposition.[2]

The consequences associated with this diagnosis can be serious and include ventricular herniation (which occurs in partial agenesis) and sudden death. To know the peculiarities of such disease is crucial, as the symptoms are nonspecific. Imaging tests such as chest X-ray, Doppler echocardiogram, computed tomography (CT), and cardiac magnetic resonance imaging (CMRI) play a key role in confirming the diagnosis (Central Illustration).

Embryogenesis

The pericardium is formed by the visceral and the parietal layers. It acts as a barrier against infections, maintaining the heart in a stable position and limiting cardiac volume.[3] It is innervated by the phrenic nerve, whose nociceptors are involved in pericardial pain transmission reflexes, and secretes prostaglandins and similar substances that modulate coronary tone and neural traffic.[3]

In the first week of fetal development, cavities are formed, which permeate the mesodermal tissue, which will later unite to form three primitive cavities: pericardium, peritoneum, and pleura.[4]

During the fourth week of gestation, a fragment of the mesoderm (transverse septum) separates the primitive pericardium from the peritoneum.[5] This separation completes between the fourth and fifth weeks when the right and left pleuropericardial membranes fuse, driven by the growth of the Ducts of Cuvier (common cardinal veins).[5,6] The pleuropericardial membrane later forms the fibrous pericardium. Failure in the closure of these membranes results in partial or total PA.[4]

One of the theories to explain AP is that the developing heart would stretch the pericardium. If the pleuropericardial membranes do not fuse before the heart enlarges, a pericardial defect is generated.[7] Another theory proposes that a traction-induced tear may develop in the pleuropericardial membrane, resulting in pericardial defect.[7] To illustrate this situation, Kaneko et al. describe a case of pericardial absence associated with the division of the phrenic nerve with its two bundles passing ventrally and dorsally to the defect.[8]

A third hypothesis suggests that pericardial membrane development fails due to premature atrophy of the cardinal veins (duct of Cuvier), which supply the pleuropericardial folds.[7,9] This theory explains the rarity of right-sided pericardial defects, as the right cardinal vein remains as the superior vena cava.[9] A percentage of pericardial defects may be attributed to each of these mechanisms.

Classification

PA is a rare anomaly that can be classified as total or partial based on the extent of pericardial absence. This condition results from failures in embryonic development, specifically during fusion of the pleuropericardial primordia.[7] Its classification is based primarily on the extent of the pericardium absence, in addition to its clinical and anatomical implications (Table 1).[10,11] The main categories are:

Total agenesis:

- Complete absence of the pericardium.
- The heart may have an abnormal position in the thorax (levoposition) and greater mobility.
- It is usually asymptomatic, but may be associated with a left side displacement of the heart and an increased risk of cardiac torsion.

Partial agenesis:

- Usually involves the absence of the left or right portion of the pericardium, with the first one being more common.
- It may cause herniation or strangulation of cardiac structures, leading to more serious complications.
- It is more associated with symptoms, such as chest pain or arrhythmias, due to compression or herniation of cardiac structures through the pericardial defect.

Circumscribed or localized defects:

- A subtype of partial agenesis affecting specific areas, such as the pericardium over the left atrium or the great vessels. Small defects in the pericardium, which may allow protrusion of structures such as the atrium or ventricle.

Clinical Presentation

The clinical presentation of PA is often heterogeneous, ranging from nonspecific symptoms to fatal complications. Although it is commonly asymptomatic, precordial pain

Table 1 – Prevalence, complications, and symptoms by category of PA.[8, 10-15]

	Partial on the right or left	Complete bilateral	Complete on the right	Complete on the left
Prevalence	3–4%	9%	17%	70%
Complications	Frequent	Rare	Rare	Rare
Symptoms	Chest pain, dyspnea, arrhythmias	Asymptomatic in most cases	Asymptomatic or with mild symptoms	Generally asymptomatic
Published cases	Balaji A et al.[12]	Pedrotti P et al.[13]	Minocha GK et al.[14]	Kaneko Y et al.[8]

may occur and is the most common symptom, followed by palpitations.[16-18] Other symptoms attributed to PA, such as dyspnea, trepopnea (dyspnea related to the side on which one lies, which is a characteristic feature of partial defects), arrhythmias (sick sinus syndrome and syncope are probably due to increased vagal tone or intermittent atrial herniation, which resolves with atropine or changing to lateral decubitus), and dizziness, are signs and symptoms normally attributed to increased cardiac mobility, anomalous heart positioning and herniation of cardiac tissue.[7,17] Moreover, increased cardiac mobility may produce a displaced and vigorous apical impulse, leading to the impression of cardiomegaly.[19] Patients with complete defects may present with precordial pain due to pleuropericardial adhesions, absence of the pericardial cushion, and torsion of the great vessels.[20]

The most severe documented manifestation is sudden death, caused by strangulation of the heart through an incomplete pericardium, as observed in patients with partial defects.[21] Strangulation typically leads to regional myocardial ischemia, resulting in precordial pain and electrocardiographic changes.[22] Cardiac displacement may cause severe tricuspid regurgitation due to the stretching of the anterior wall of the right ventricle (RV).[23]

Although rare, PA should be considered in the differential diagnosis of patients with atypical cardiovascular symptoms that are unusual for their age, particularly when echocardiographic images are acquired from nonstandard locations. Early identification is essential to avoid more serious complications and direct appropriate clinical management. Differential diagnoses of PA include post-surgical or post-traumatic defects, the latter after blunt thoracic trauma, which may result in pericardial rupture, herniation, and torsion of the cardiac cavities.[24]

Diagnosis: The role of multimodality

Chest X-ray

Chest X-ray may be altered in large pericardial defects, where the cardiac silhouette is displaced excessively to the left in the posteroanterior projection, and posteriorly in relation to the mediastinum. There is a loss of the right border of the heart, which overlaps the spine. Radiolucency and increased distance between the aorta and pulmonary artery, as well as between the lower heart border and diaphragm, occur due to lung tissue insinuation into these areas. The "Snoopy Sign" refers to leftward cardiac displacement, elongation of the left heart border with a rounded apex resembling Snoopy's nose, and prominence and convexity of the left atrial appendage resembling Snoopy's ear (Figure 1).[25]

Doppler echocardiogram

The Doppler echocardiogram is a widely-available non-invasive, low-cost exam and the first-line choice in the diagnostic suspicion not only for AP, but also for the evaluation of pericardial diseases.[26]

The first indication suggesting agenesis during examination relates to image acquisition in nonstandard positions due to abnormal cardiac positioning (Figures 2A and 2B). In the long parasternal plane, the apex deviates posteriorly, revealing a large segment of the RV, creating an impression of cavity enlargement[7,27] (Figures and Videos 1 and 2). The acquisition of the apical 4-chamber plane will occur beyond or in the posterior midclavicular line, different from the commonly located in the 5th intercostal space, in the mid-midclavicular line. (Figure and Video 3 and 4).

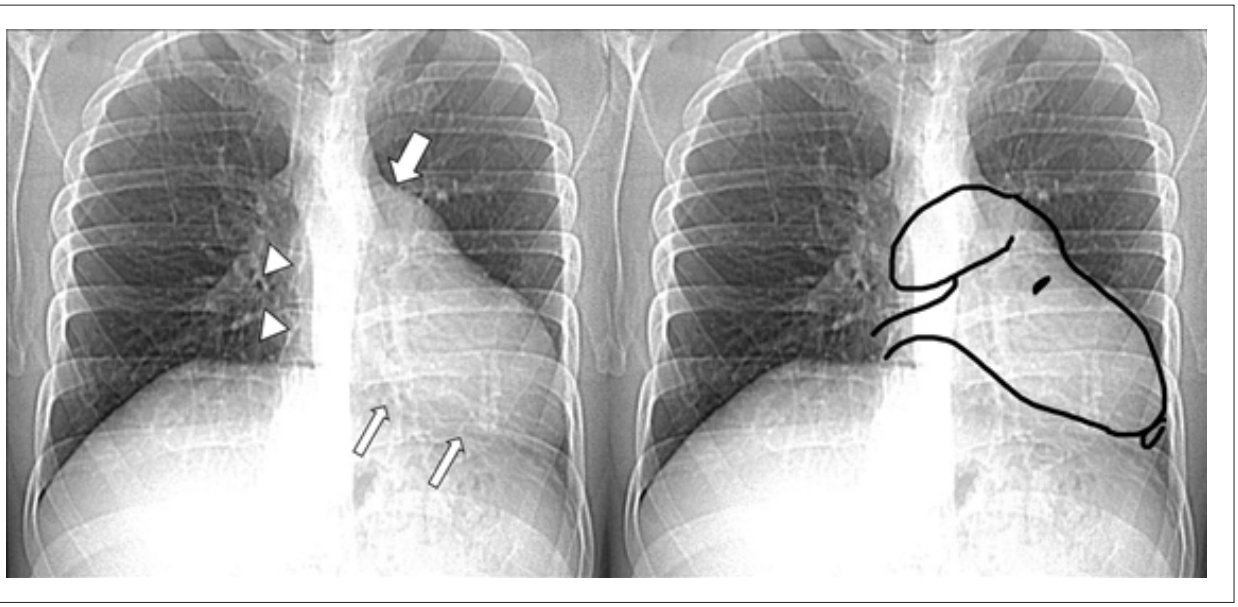


Figure 1 – *The "Snoopy sign" on chest radiography is composed of multiple abnormalities, namely: excessive levoposition with elongation of the LV edge, radiolucency separating the aorta and pulmonary artery (thick arrow), radiolucency separating the LV and diaphragm (thin arrows), and loss of the right edge of the heart (arrowheads).*

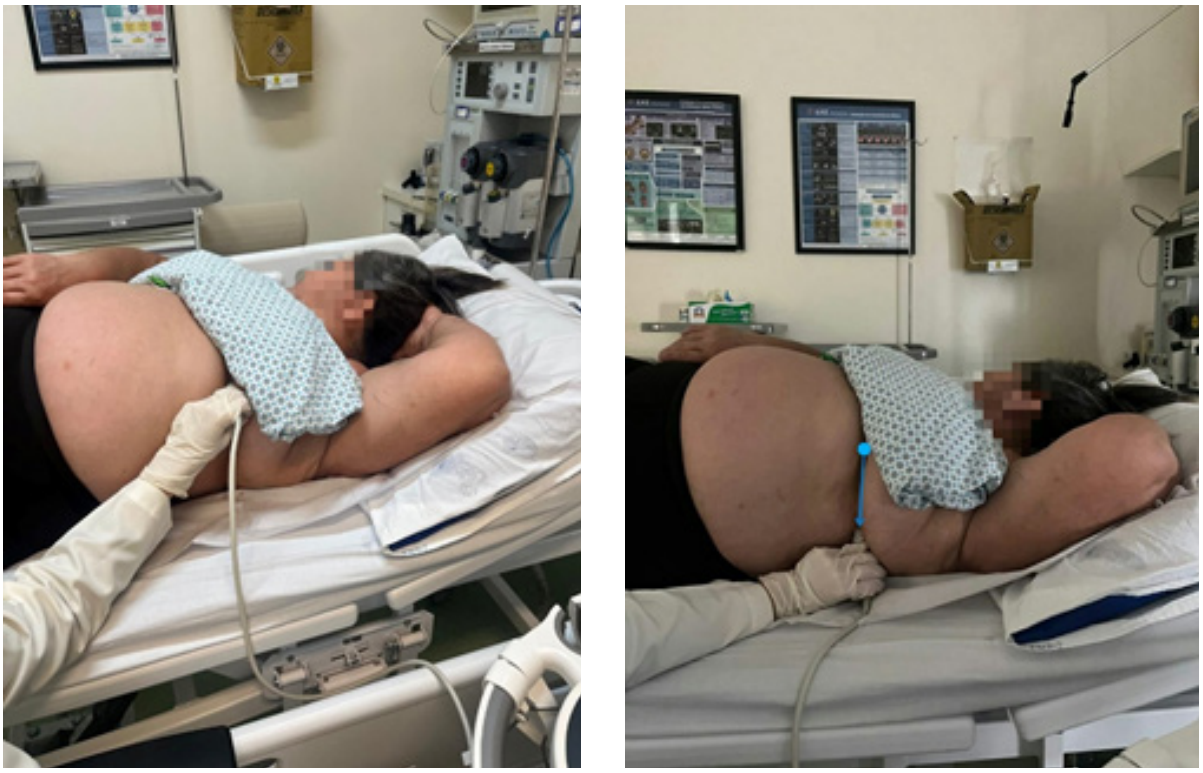


Figure 2 – *A) This image illustrates the positioning of the transducer on the patient, where the apical 4-chamber plane would normally be located. B) In partial PA, note the significant displacement through the inserted line (blue), from the site to the new location of the apical 4-chamber plane.*

In cases of left partial PA, 63% of patients present an apparent RV enlargement due to leftward cardiac displacement.[28] This finding can lead to a misdiagnosis of interatrial communication or arrhythmogenic cardiomyopathy. Additional findings relating to PA include exaggerated cardiac motion and paradoxical ventricular septum movement due to increased inferior wall motion. A teardrop-shaped heart results from atrial elongation, ventricular widening, and inferior wall bulging.[2,7,28] Right-sided agenesis may lead to RV and right atrium (RA) enlargement, along with severe tricuspid regurgitation due to chordal traction. Stress Doppler echocardiography may demonstrate a pendulum-like movement of the heart. Another significant spectral Doppler finding is reduced systolic flow and a lower systolic-diastolic ratio in the pulmonary vein as a result of the absence of negative intrapericardial pressure.[26]

However, this method depends on the quality of the acoustic window, especially in obese patients or those with lung disease.[26]

CT

CT imaging, with its high resolution and volumetric acquisition, provides excellent contrast between the pericardium's dense thin line and the low-density epicardial and mediastinal fat, making it highly effective for pericardial delineation. However, some pericardial segments in the left silhouette of the heart may be poorly delimited, even when present.

On CT, PA can be confirmed by the partial or complete absence of the thin dense line corresponding to the pericardium around the heart. There is excessive levoposition of the heart, which touches the rib cage. There may be an absence of hypodense fatty tissue between the myocardium and the costal arches. There is insinuated lung tissue between the aorta and the pulmonary arterial trunk, a site commonly covered by pericardium and containing fatty tissue that obliterates this space. Occasionally, there is bulging of the left atrial appendage through the defect (Figure 5).[28]

CMRI

CMRI is currently the gold standard for pericardial assessment thanks to its superior spatial resolution and inherent tissue characterization. T1- and T2-weighted "black blood" morphological acquisitions and cine steady-state-free-precession (SSFP) functional acquisitions are useful for delimiting the hypointense linear image corresponding to the pericardium, between the mediastinal and epicardial fat with hyperintense signal. However, similar to CT, some segments of the pericardium adjacent to the left ventricle (LV) may be difficult to individualize

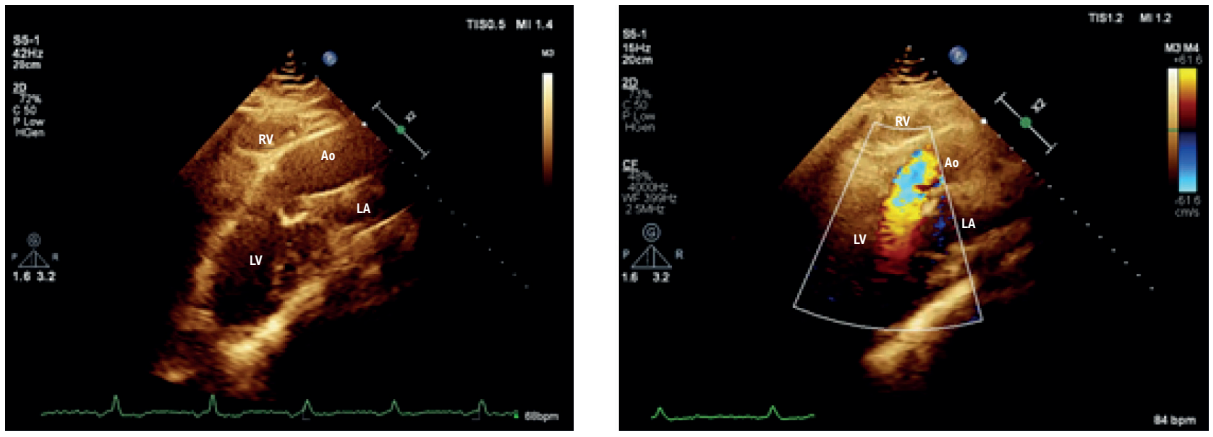
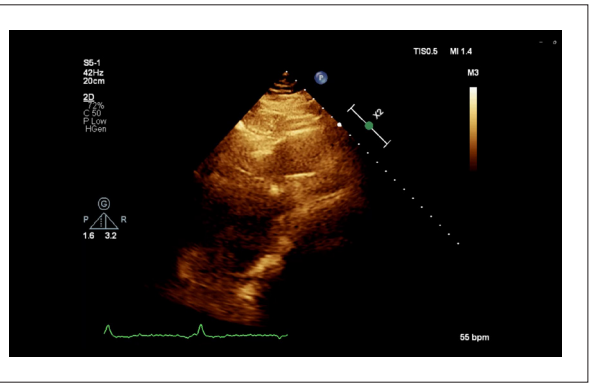
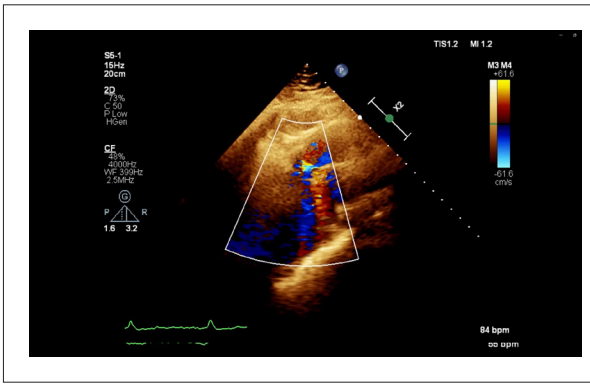


Figure 3 – *A) Visualization of the completely distorted longitudinal parasternal plane, with the apex deviated posteriorly (videos attached). B) The flow through the LV outflow tract confirms the position of the ascending aorta, LV and LA in a patient with partial PA. Ao: aorta; LV: left ventricle; RV: right ventricle; LA: left atrium.*



Video 1 – *Visualization of the completely distorted longitudinal parasternal plane, with the apex deviated posteriorly. Link: http://abcimaging.org/supplementary-material/2025/3801/2025-0004_video_1.mp4*



Video 2 – *The flow through the LV outflow tract confirms the position of the ascending aorta, LV and LA in a patient with partial PA. Link: http://abcimaging.org/supplementary-material/2025/3801/2025-0004_video_2.mp4*

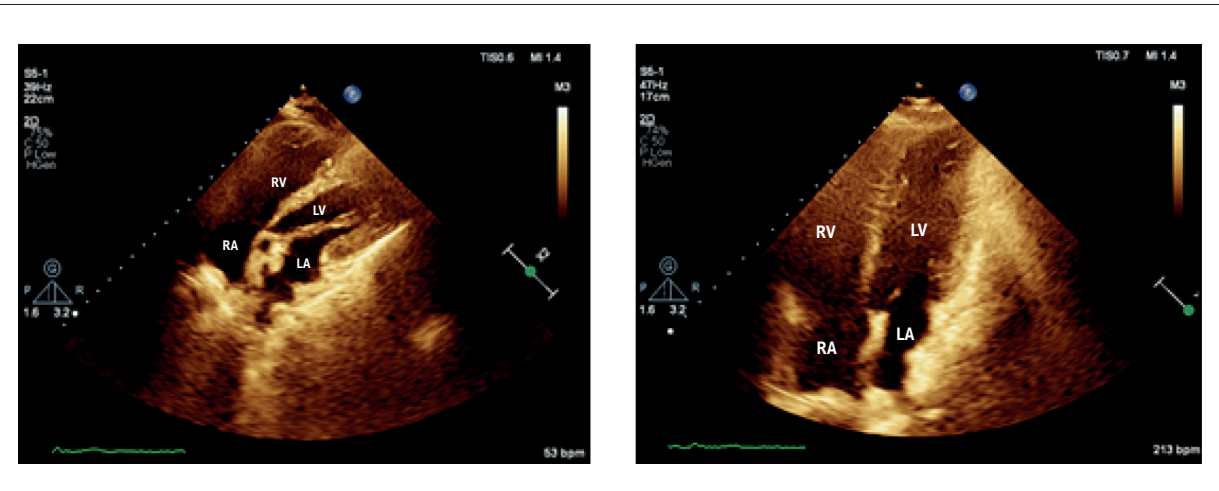
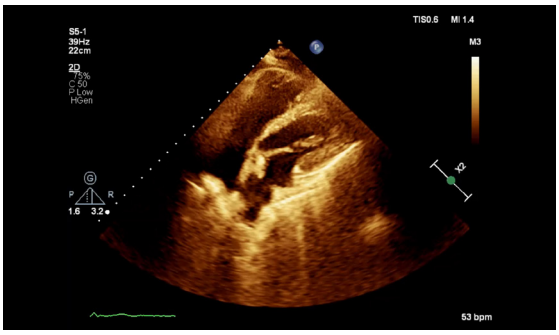
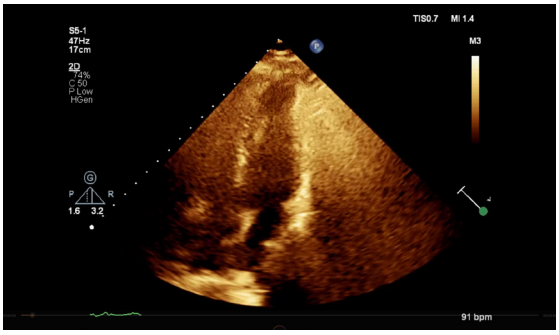


Figure 4 – *A) Case of partial PA showing the impression of enlarged right cavities due to the deviation caused by partial left agenesis (video attached). B) Case of partial PA showing the teardrop-like appearance of the heart (video attached). LV: left ventricle; RV: right ventricle; RA: right atrium; LA: left atrium.*



Video 3 – *Case of partial PA showing the impression of enlarged right cavities due to the deviation caused by partial left agenesis. Link: http://abcimaging.org/supplementary-material/2025/3801/2025-0004_video_3.mp4*



Video 4 – *Case of partial PA showing the teardrop-like appearance of the heart. Link: http://abcimaging.org/supplementary-material/2025/3801/2025-0004_video_4.mp4*

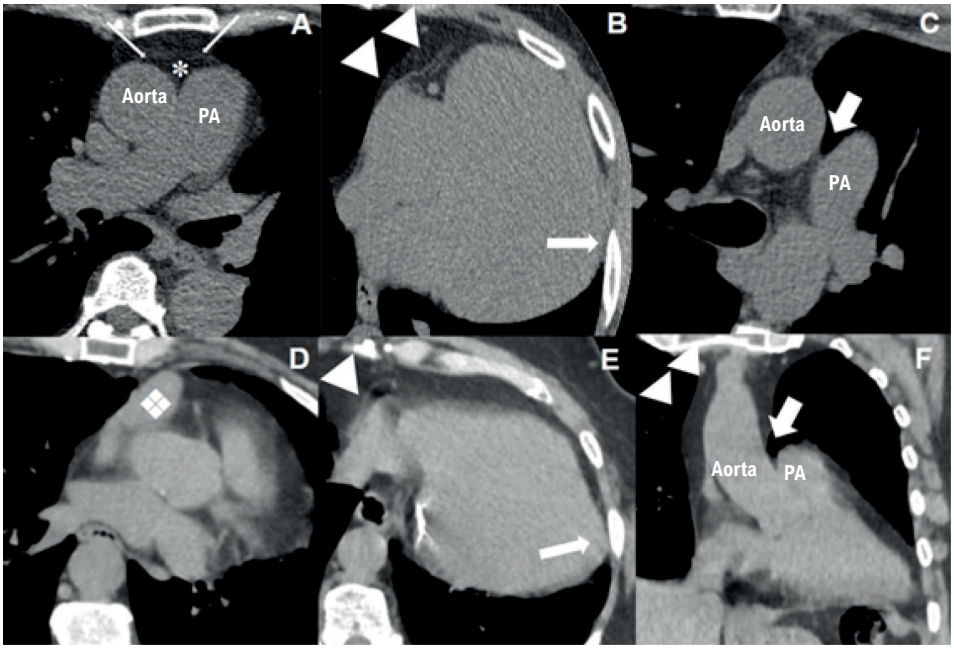


Figure 5 – *A) The non-contrast CT in the axial plane demonstrates the normal anatomy of the pericardium (thin arrows) outlining the fatty tissue (✻) between the aorta and the pulmonary trunk. (B-F) Non-contrast CT images in two patients with partial left pericardial agenesis, respectively, ♂, 41 years (B/C), and ♀, 58 years (D-F). The absence of pericardial covering is observed over almost the entire extent, visible only along the right atrial border (arrowheads in B, D, and E). There is excessive leftward positioning of the heart and posterior deviation of the apex touching the chest wall (thin arrows in B and E). D) Projection of the right atrial appendage (❖). (C/F) Lung interposition between the aorta and the pulmonary trunk (thick arrows). AP: pericardial agenesis.*

by the method. The absence of pericardial visualization, combined with excessive leftward cardiac displacement, hypermobility (particularly of the posterior LV wall), and lung interposition between the aorta and pulmonary trunk, are CMRI diagnostic criteria for PA (Figure 6). The presence of focal indentations in the ventricular walls suggests the presence of partial PA.[2, 28]

Treatment

Watchful waiting is the preferred approach for most patients with PA, as the majority remain asymptomatic.[26] Surgical treatment relies on small, uncontrolled observational studies.[26]

Surgical intervention (Table 2) is recommended for symptomatic patients with high-risk complicated partial defects or refractory symptoms, such as pain caused by abnormal cardiac motion.[10] Left partial defects presenting with compression, herniation, or strangulation of major vessels or coronary arteries also warrant surgical consideration.[29]

Recently, the detection of high-risk anatomic features by multimodal cardiovascular imaging, including (1) LV myocardial fold or hinge point on echocardiography, CMR, or

CT; (2) coronary compression on CT; (3) inducible ischemia on stress perfusion imaging – Single Photon Emission Computed Tomography (SPECT), Positron Emission Tomography (PET) or CMR – and (4) evidence of left atrial appendage herniation have been proposed for risk stratification.[30] Detection of these red flags can be used to tailor surgical planning.

Specific surgical options for pericardial defects vary, with pericardioplasty being the most common procedure. As an alternative to pericardial reconstruction, pericardiectomy with pericardioplasty, to enlarge the defect and prevent strangulation, has also been described.

Author Contributions

Conception and design of the research and acquisition of data: Almeida BBC, Jácome BZ, Lourenço BN, Gripp EA; analysis and interpretation of the data and statistical analysis: Gripp EA; writing of the manuscript and critical revision of the manuscript for intellectual content: Almeida BBC, Jácome BZ, Lourenço BN, Seixas AZ, Silveira JS, Gripp EA.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

There were no external funding sources for this study.

Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

Table 2 – Relationship between surgical intervention, possible indications and complications.[7,15]

Surgical interventions	Possible indications	Potential complications
Pericardioplasty	Major congenital defects	Failure to correct the defect
Pericardiectomy with pericardioplasty	Risk of strangulation	Formation of pericardial adhesions
Left atrial appendage resection/ligation	Left atrial appendage herniation	Damage to adjacent structures
Right atrial appendage correction	Right atrial appendage herniation	Right atrial appendage rupture
Video-assisted thoracic surgery (VATS)	Pneumopericardium/pneumothorax	Perforation of thoracic structures

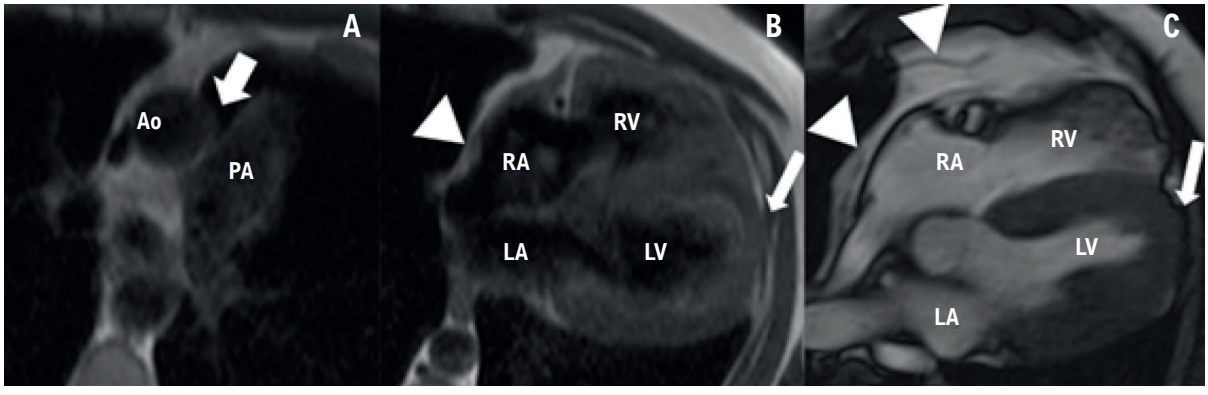


Figure 6 – *Black-blood T2-weighted CMR images in the axial plane (A,B) and cine SSFP (C) in a patient with left partial PA. A) Interposition of lung tissue between the aorta and pulmonary artery (thick arrow). (B,C) The pericardium is partially visible as a thin hypointense line along the right atrial border (arrowheads in B and C). It is observed excessive levoposition of the heart and posterior deviation of the apex, touching the rib cage (thin arrows in B and C). PA: pericardial agenesis; LV: left ventricle; RV: right ventricle; RA: right atrium; LA: left atrium: Ao: Aorta.*

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