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CASE REPORT

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Unusual presentation of a patient with partial anomalous pulmonary venous connections without a septal defect: a case report and literature review

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Abstract

Introduction Partial anomalous pulmonary vein connections (PAPVC) are rare congenital abnormalities in which one or more pulmonary veins drain into the right atrium. This pathological condition may present in various ways, such as chest pain and dyspnea, or it may be diagnosed incidentally. Consequently, missed or late diagnoses are common, highlighting the importance of optimal diagnostic modalities. This study presents a case of PAPVC that remained undiagnosed despite two years of symptomatic evaluations.

Case Presentation The patient was a 58-year-old woman who presented with chest pain and dyspnea, New York Heart Association Functional Class (NYHA FC) II, which had started 2 years before. She had been evaluated with a transthoracic echocardiogram, coronary angiography, and chest imaging, none of which resulted in a diagnosis or effective treatment. After being referred for evaluation by a cardiac imaging specialist, the diagnosis of PAPVC was revealed. The patient underwent corrective surgery, resulting in complete recovery and symptom improvement. Post-surgical cardiac overload caused pleural effusion and dyspnea, which were managed with diuretics. Four- and six-month follow-ups showed no abnormal findings on examination and imaging, and the patient reported no new complaints.

Conclusion The findings in this structural cardiac abnormality are nonspecific and can be missed, and misdiagnosis is relatively common. However, patients with symptoms such as chest pain, dyspnea, increased pulmonary artery pressure, and right ventricle dilation should undergo surgical treatment to avoid more serious complications, such as heart failure.

Key clinical point Detection of PAPVC is not straightforward in many cases and often requires evaluation with various imaging techniques for accurate diagnosis. Therefore, physicians encountering undefined causes of chest pain or dyspnea should consider multiple imaging modalities. Follow-up is also important, as certain groups of patients may require surgical treatment to prevent complications.

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Introduction

Partial Anomalous Pulmonary Vein Connections (PAPVC) are rare congenital defects in which one or more pulmonary veins drain into the right atrium or its tributaries. Different types have been observed in 0.5-1% of autopsies. However, fewer presentations throughout a patient's lifetime are largely attributed to the asymptomatic condition. This anomaly often results in a left-to-right shunt (LTRS), which may require surgical correction. Another reason for fewer presentations is that the diagnosis is not straightforward and may require multimodality imaging [1]. Based on the drainage location, it can be categorized into four groups: supra-cardiac, cardiac, infra-cardiac, and mixed-type [2]. This condition might be isolated or occur with other concomitant structural disorders such as an atrial septal defect (ASD) [3]. Both lungs are involved in very rare variations of PAPVC [4]. A dual drainage variation of PAPVC, similar to Total Anomalous Pulmonary Vein Connections (TAPVC), occurs when the pulmonary veins drain into systemic veins at two different levels, most commonly at the cardiac and supra-cardiac levels [5]. Treatment depends on the clinical and hemodynamic condition and findings from cardiac imaging evaluations. While some patients are asymptomatic and may live without intervention, others may require immediate surgical or transcatheter interventions to prevent serious complications [6 7]. Although there is a trend toward transcatheter correction of the anomaly, surgical treatment remains the gold standard for treating PAPVC [8].

This study presents a patient with undiagnosed chest pain for two years despite several evaluations, who was finally diagnosed as PAPVC after being referred for TEE and PCTA. This case, in addition to highlighting the existence of this rare condition, underscores the importance of comprehensive evaluation of undiagnosed cases with cardiac or respiratory complaints and referral to more specialized centers when necessary.

Case presentation

History and physical examination

The patient is a 58-year-old woman who presented to a tertiary cardiology center with ongoing and progressive dyspnea, which began as exertional dyspnea 2 years ago and worsened over time. Two weeks ago, she experienced breathlessness and severe chest pain at rest. The patient reported a history of a complete cardiology workup in their town, which was inconclusive and did not result in any definitive diagnosis. The patient also had a history of uncontrolled blood pressure and poor adherence to antihypertensive therapy. She was taking a combination tablet of Valsartan (160 mg) and Amlodipine (5 mg) twice daily, Aspirin (81 mg) daily, and Atorvastatin (40 mg) each night.

During her physical examination, her general appearance was anxious. On vital sign examination, the respiratory rate was elevated (22/minute); the oxygen saturation on room air was also measured to be 98%, and her blood pressure in both arms was 170/100 mmHg in a supine position. The patient was not febrile. The respiratory physical examination showed no abnormalities. On the other hand, the cardiac examination raised concern for cardiac abnormalities due to the presence of fixed S2 splitting on cardiac auscultation, which prompted a plan for temporary admission and further evaluation, including an electrocardiogram (ECG), transthoracic echocardiogram (TTE), and a probable transesophageal echocardiogram (TEE).

Diagnostic and therapeutic procedures

The patient's ECG showed no abnormal findings. (However, TTE revealed Right ventricular dilation and dysfunction without any other abnormalities. The QP/ QS ratio was 2.1. Other significant findings on TTE **included** marginally increased pulmonary arterial pressure (PAP), moderate tricuspid regurgitation (TR), and Mild to moderate pulmonary valve insufficiency (PI) (Table 1).

TEE examination revealed that the upper right pulmonary vein appeared anomalous and drained into the superior vena cava (SVC). (Fig. 1) Given the dilated right ventricle observed on TEE, more than one pulmonary vein was suspected to be involved. Following American Heart Association (AHA) guidelines, the patient underwent a pulmonary CTA, which revealed that two pulmonary veins were affected. The right upper lobe (RUL) pulmonary vein drained into the SVC, and the left pulmonary vein drained into the brachiocephalic vein—findings consistent with PAPVC (Fig. 2). The sagittal plane also showed a vertical vein that abnormally connected the left pulmonary vein to the left brachiocephalic vein (Fig. 3). She underwent surgery, during which the presence of the second anomalous vein was confirmed, validating the conclusions of the CTA. The coronary arteries appeared normal on the CAG. The videos of axial, coronal, and sagittal views are attached to the manuscript (Supplementary videos 1, 2 and 3).

The surgery was successful, and the patient was transitioned from the care of cardiology and cardiac surgery services to the ICU.

The patient showed poor cooperation with deep breathing exercises and was unwilling to move. Within 24 h after surgery, the patient experienced reduced oxygen

Table 1 Findings of initial transthoracic echocardiogram

Index	Result
Left ventricle (LV)	Normal LV size and systolic function, ejection fraction (EF); 55%
	Mild Diastolic dysfunction
	Mild septal hypertrophy (LVH)
	No right wall motion abnormality
Mitral Valve	No mitral stenosis (MS), mild Mitral regurgitation (MR)
	No Mitral valve prolapse (MVP)
Aortic Valve	Trileaflet AV, No aortic stenosis (AS)
	No aortic insufficiency (AI)
Ascending Aorta	Normal with a 3 cm diameter
Right Ventricle	Moderately dilated (RVEDD:44 mmm) with mild dysfunction (TAPSE: 15 mm, Velocity: 7 cm/s, FAC: 32%)
Main pulmonary artery (PA)	Dilated (35 mm)
Systolic pulmonary artery pressure (SPAP)	Mildly increased (45 mmHg)
Tricuspid Valve	Moderate TR, no TS

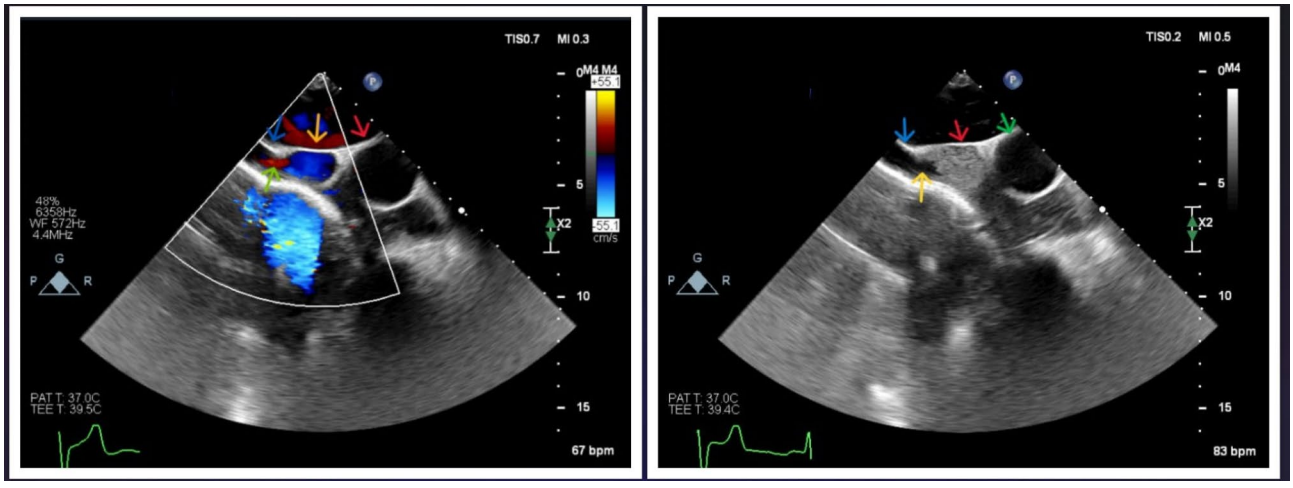

Fig. 1 **Left**: Color study TEE with an upper esophageal view illustrates flow (green arrow) from RUPV (blue) to SVC (orange). The red arrow indicates the ascending aorta; **right**: Contrast study with the upper esophageal view highlights the ascending aorta (green arrow), the SVC filled with contrast (red arrow), and negative contrast (yellow arrow) caused by flow from the RUPV (blue arrow)

saturation (79%) and dyspnea caused by postoperative lung atelectasis. A series of diagnostic tests, including a chest CT, chest X-ray (Fig. 4), pleural ultrasound, and transthoracic echocardiography (TTE), were performed. Based on the pleural findings, the patient underwent diagnostic thoracentesis, which removed 10 cc of fluid from each pleural cavity. Fluid analysis revealed a transudative nature, suggesting fluid overload as the underlying cause. The patient was subsequently started on Lasix (Furosemide) at a rate of 5 mg per hour. She also received chest physiotherapy and prophylactic antibiotics to prevent post-surgical infections. Fluid intake and output were monitored for three days, and daily TTE was performed to ensure appropriate cardiac function. After three days, no abnormalities were detected in the cardiorespiratory examination. To confirm anatomical integrity before discharge, the cardiologist ordered a PCTA, which revealed the results in Table 2.

Conclusion and follow-up

The patient was discharged from the hospital five days after the operation. At her four- and six-month follow-up visits, she reported no new symptoms or complaints. TTEs performed at both follow-ups revealed normal findings, indicating a stable postoperative recovery.

Discussion

The study presents a patient with PAPVC, which was initially missed during evaluations conducted for her chest pain. An initial TTE was performed in the patient's hometown; however, the anomaly was not diagnosed. After being referred by a cardiac imaging specialist for additional evaluations, supplementary tests such as TEE and PCTA revealed the diagnosis of PAPVC. Surgical management was performed due to symptomatic presentation and abnormal imaging findings. The patient's recovery involved a minor and relatively common complication of cardiac function overload, which

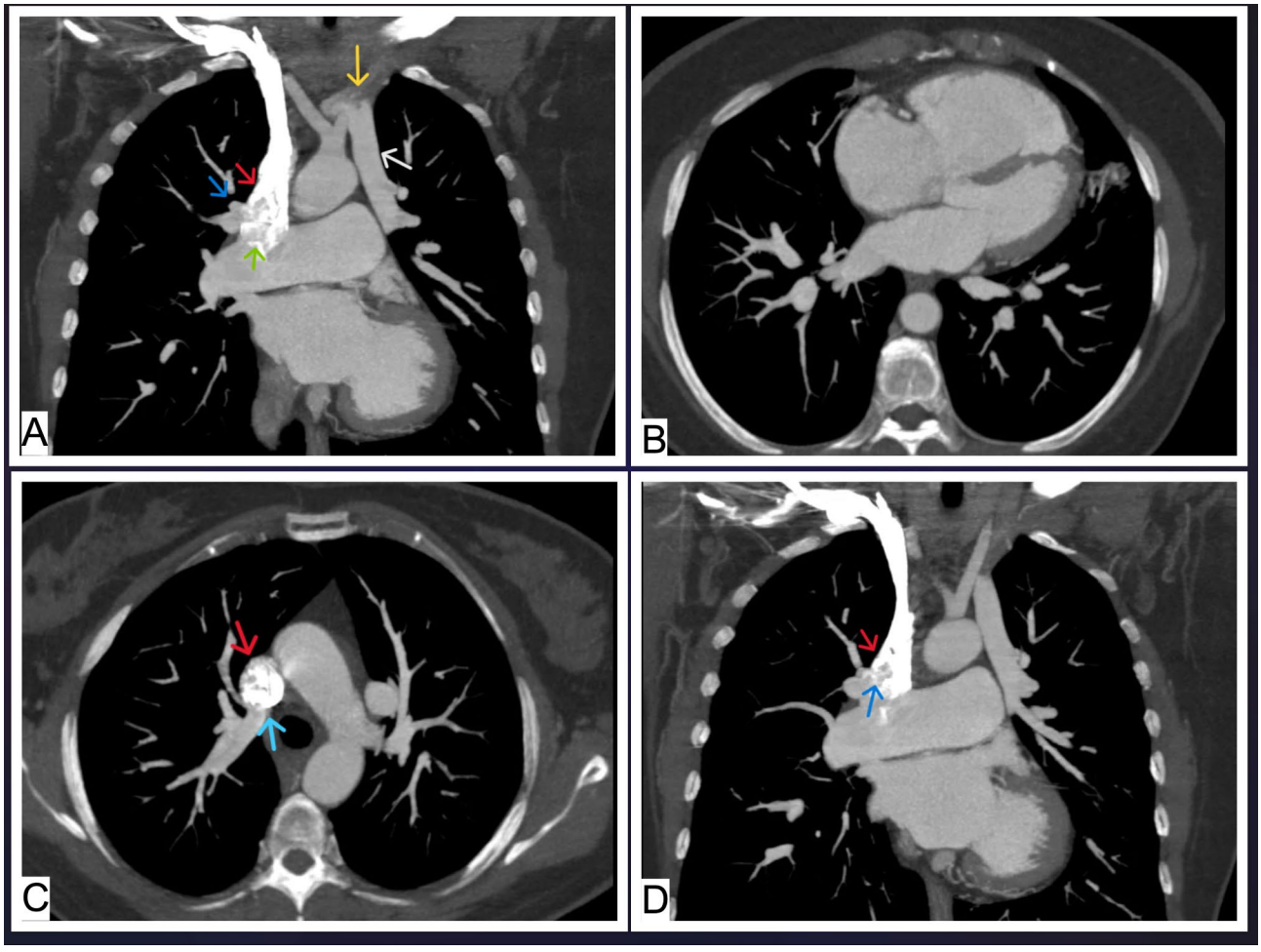

Fig. 2 **A**: The chest computed tomography angiography in the coronal plane reveals abnormal pulmonary vein connections. The vertical vein (white arrow) connects the left pulmonary vein to the left brachiocephalic vein (orange arrow). The right upper pulmonary vein (blue arrow) also connects to the superior vena cava (red arrow). The green arrow highlights the mixture of blood and dye at the site of the connection. **B**: The axial plane at the ventricular level shows an intact interatrial septum and significant right atrium and ventricle dilation. **C**: The axial plane at the superior vena cava level reveals an abnormal connection of the right upper pulmonary vein to the superior vena cava, indicated by the blue arrow and red arrow, respectively, resulting in a mixture of blood and contrast. **D**: The coronal plane shows an abnormal connection between the right upper pulmonary vein (blue arrow) and the superior vena cava (red arrow)

was managed smoothly with medications. Follow-up was uneventful and showed normal findings.

PAPVC has been diagnosed in approximately 0.4-0.7% of the normal population during autopsy evaluations. The prevalence of symptomatic patients is nearly equal to that of the lower range of this spectrum (0.41%) [9]. Therefore, it is considered a rare anomaly that cardiologists should keep in mind [10]. This case report underscores the fact that diagnosis was missed for almost two years after symptomatic diagnosis due to the limitations of TTE in diagnosing PAPVC. This structural anomaly is present in 0.5-1% of the population. This suggests that many patients live their entire lives with this anomaly without symptoms or a diagnosis [11]. Although there are various subtypes of this condition, nearly four-fifths of diagnosed cases involve anomalous pulmonary veins draining into the RA via a sinus venosus connection with an ASD. In contrast, the diagnosis of pure PAPVC without a septal defect is seen in approximately one-tenth of cases and is substantially rare [12]. The presented case demonstrated no septal defect.

The presentation of PAPVC is considerably nonspecific and depends on the magnitude of the intracardiac shunt and the anomalous drainage of the PV. Therefore, diagnosis is not straightforward and requires a thorough evaluation using multimodal imaging [13]. However, regardless of the type of PAPVC, it causes left-to-right shunts in pre-tricuspid level, which result in recirculation of the oxygenated blood into the pulmonary circulation, increased pulmonary blood flow, and increased volume load on the right heart. Consequently, right atrial enlargement (RAE) and right ventricular enlargement

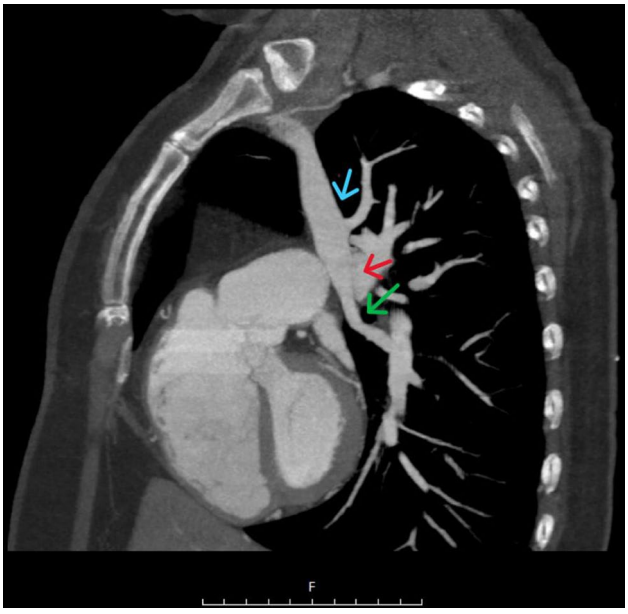

Fig. 3 The chest computed tomography angiography in the sagittal plane shows a vertical vein (blue arrow) that abnormally connects the left pulmonary vein (red and green arrows) to the left brachiocephalic vein

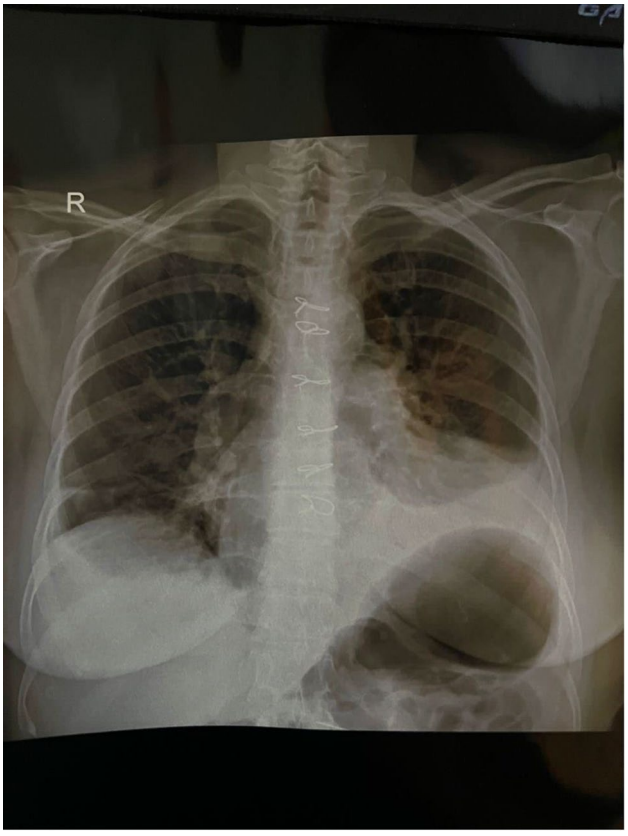

Fig. 4 Pericardial effusion caused by cardiac overload after the surgery, which was managed with diuretics

(RVE) inevitably result from these functional abnormalities [14]. In summary, it is important to note that aging and its associated effects, such as HTN and diastolic dysfunction, contribute to increased pressure in the LA compared to the RA. This, in turn, can lead to increased blood flow to the right heart and a greater left-to-right shunt. However, the amount of blood that enters the right heart is influenced by the number of abnormal connections present. For instance, it has been observed that a single anomalous pulmonary vein connection may not cause significant overload on the right side of the heart.

Additionally, an atrial septal defect can facilitate diagnosis, as imaging techniques make it more easily visualized. This condition may also be associated with greater blood shunting and an increased likelihood of developing symptoms [14]. Cyanosis is an uncommon manifestation of this condition, occurring when pressure on the right side of the heart exceeds that on the left. This pressure difference can reverse blood flow, resulting in the development of Eisenmenger's syndrome. While PHTN is an indication for the surgical or interventional correction of PAPVC, surgery is contraindicated in cases of Eisenmenger's syndrome, as it may worsen the patient's condition. Standard medications are used to manage pulmonary hypertension (PHTN) [15 16].

Diagnostic procedures when encountering CHD usually start with CXR and an echocardiogram. Although increased heart size, abnormally located pulmonary vessel structure, and interstitial changes in the lungs might suggest abnormalities in the cardiovascular system, the first choice for detecting anatomical heart defects is an echocardiogram. TTE and TEE are used, with TEE offering more sensitivity and specificity, especially for visualization of the entire atrial septum and PV connections [10]. A limitation of TEE is its requirement for sedation, which entails specific requirements and potential risks [14]. Overall, TEE and TTE are suboptimal for diagnosing PAPVC, and multidetector computed tomography (MDCT) or magnetic resonance imaging (MRI) angiography provides significantly higher accuracy. Newer-generation three-dimensional MDCT has proven to be an excellent diagnostic modality [14].

The management of PAPVC depends on clinical findings, hemodynamic characteristics, and anatomical features. Therapeutic options include (1) follow-up, (2) medical management, (3) surgical treatment, and (4) transcatheter interventional procedures [17]. Corrective intervention is indicated in the following situations: the presence of significant symptoms related to this anomaly, a significant left-to-right shunt indicated by a Qp/Qs ratio greater than 2, or imaging evidence showing right ventricular (RV) dilation or RV volume overload (evidenced by a D-shaped septum during diastole). Additionally, surgical repair may be necessary alongside the

Table 2 Findings of chest computed tomography angiography before and after the surgery

Anatomical Feature	Finding before the surgery	Findings after the surgery
Left Upper Pulmonary Vein	Drainage to Brachiocephalic vein compatible with PAPVC	Connected to a vertical vein leading to LAA, the connection is patent
Pulmonary arteries tree	Normal	No filling defect
Main Pulmonary Artery (MPA)	36 mm	36 mm
Right Pulmonary Artery (RPA)	32 mm	Diameter = 32 mm, dilated
Left Pulmonary Artery (LPA)	22 mm	Diameter = 25 mm
Right Upper Pulmonary Vein	Drainage to SVC compatible with PAPVC	Reconnected to LA
Right Subclavian Artery	An aberrant right subclavian artery is seen	An aberrant right subclavian artery is seen
Atrial and Ventricular Septum	No ASD, no VSD	No ASD, no VSD
Right Atrium (RA)	Enlarged	Enlarged
Right Ventricle (RV)	Enlarged	Enlarged
Pleural Cavity	Normal	Mild bilateral pleural effusion
Heart	Cardiomegaly seen	Cardiomegaly seen
Superior Vena Cava (SVC)	RUL is drained to SVC	
Inferior Vena Cava (IVC)	Drains to Right Atrium (RA)	Drains to Right Atrium (RA)
Coronary Sinus (CS)	Drains to Right Atrium (RA)	Drains to Right Atrium (RA)
Thoracic Aorta	Normal	Normal
Pericardial Cavity	Normal	Mild pericardial effusion seen
Left Atrium	Normal size, no left atrial appendage filling defect	Normal
Left Ventricle	Ventricular cavity size within normal limits, no stigmata of prior infarction, no abnormal filling defect	Normal

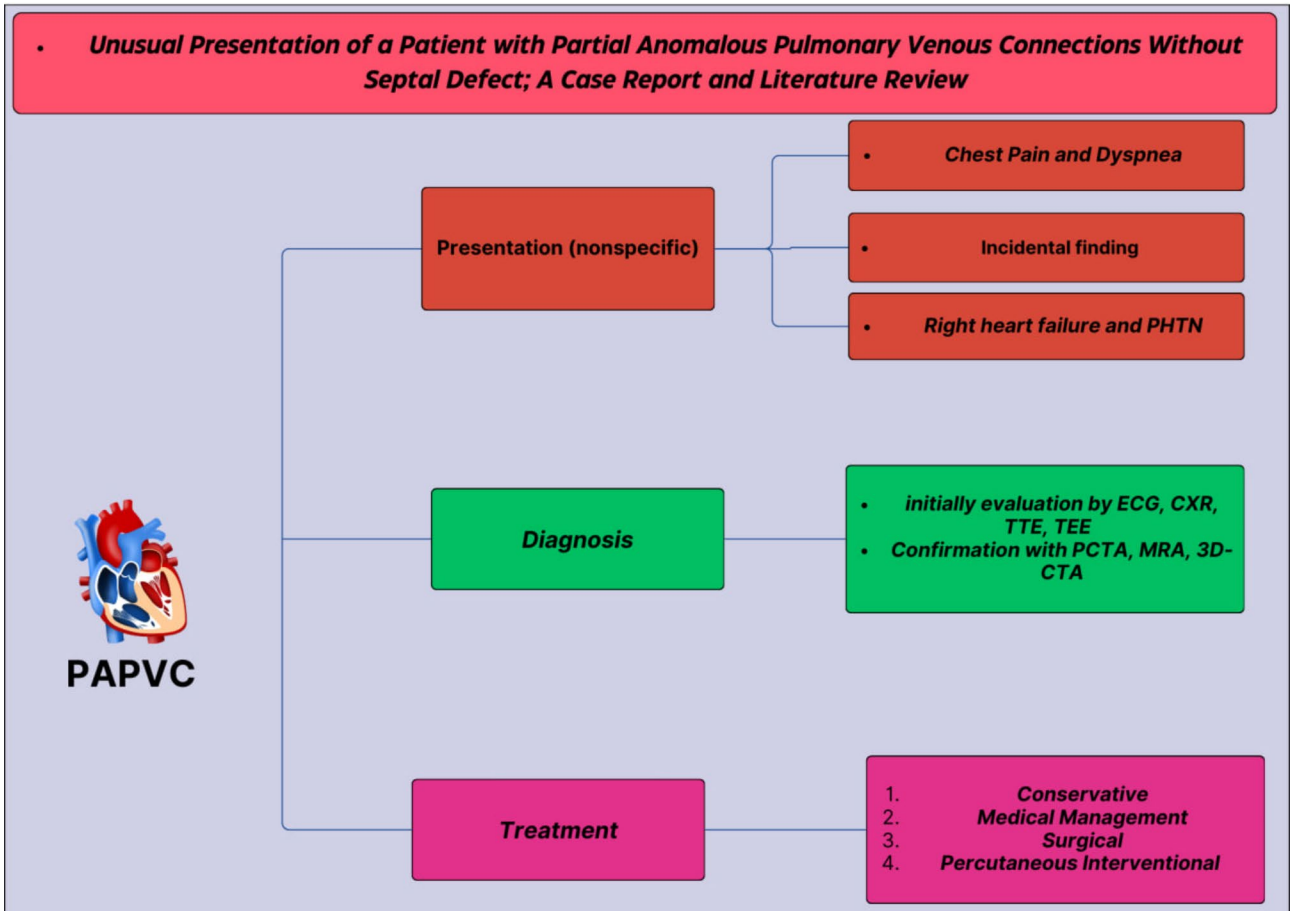

Fig. 5 Graphical abstract of Partial Anomalous Pulmonary Venous Connection (PAPVC)

Table 3 Presentation of a patient with partial anomalous pulmonary venous connections and their natural history

	First Author and YOP	Gender and Age	Presentation and History	Diagnostic and therapeutic interventions
1	Tarun Kumar. et al. (2014) [20]	18-year-old female	CC: Effort intolerance and easy fatigability NYHA FC: II PMH: None. PFH: None. LAB: Increased urea (48), Creatinine (2) Abdominal US: grade I renal parenchymal disease ECG: 'P' + right axis deviation TTE: dilated RA & RV + mild to moderate TR + TR jet gradient of 42 mm of Hg TEE: Obstructed PAPVC connecting to the IVC without any ASD or VSD Chest CT: W/O contrast due to renal dysfunction showed no pulmonary abnormality	DX: PAPVC + PHT TX: Sildenafil & low-dose diuretic to decline PHT and planned surgical anomaly correction. However, the patient did not follow up
2	Iqra Qamar. et al. (2018) [1]	58-year-old-female	CC: central chest pain PMH: NSTEMI PH/EX: MRA: Revealed PAPVC, left UPV drained into the brachiocephalic vein◊ No substantial LTRS (Qp/Qs < 1.5). Delayed hyper-enhancement of the septal regions indicated an MI despite unremarkable LV and LR.	TX: Despite being asymptomatic, this patient was followed up with informed consent, and if symptoms developed, she would be considered for PAPVC surgery.
3	Hiroki Wakamatsu. et al. [3]	42-year-male	CC: progressive exertional dyspnea PH/EX: An accentuated 2nd heart sound and no heart murmur were audible. NYHA FC: III CXR: Bilateral pulmonary arteries dilatation ECG: RAD and RVH and complete RBBB TTE: RVE & IVS flattening CECT: right upper and middle PVs drained into the SVC, and Pas and RV were dilated RHC: (Qp/Qs) 1.4, PAP:91 mmHg, PCWP: 12,	DX: Increased PAP due to PAPVC TX: PAH-specific drugs. Macitentan (20 mg/day) and tadalafill (10 mg/day) were immediately administered. In addition, selexipag (increasing from 2 to 10 mg/day at our outpatient clinic) Progress: The patient improved, but after a while, due to nonadherence, the surgery was successfully performed.
4	Pratap Upadhya et al. (2024) [21].	41-year-old Female	CC: exertional dyspnea, mMRC FC II, and dry cough for 3 years PH/EX: increased JVP HRCT: Non-fibrotic HP TTE: RA and RV dilatation, Mod TR with an RVSP of 48 mmHg + RAP, and a nor-mal LVEF, PCTA: NO PE but incidentally found left superior pulmonary vein draining into the left brachiocephalic vein	DX: PAPVC Treatment: Ambrisentan and tadalafil were used for 3 months, and the condition improved.
5	Aprateem Mukherjee et al. 2024 [4]	27-year-old female	CC: dyspnea on exertion was TTE: ostium secundum ASD and PAPVC with RIPV draining into the coronary sinus. The pulmonary vein was seen draining into the left brachiocephalic vein. The RSVP drains normally into the LA.	DX; Double drainage PAPVC TX: Surgical treatment

Table 3 (continued)

	First Author and YOP	Gender and Age	Presentation and History	Diagnostic and therapeutic interventions
6	Yuuki Matsui. et al. 2024 [22]	74-year-old female	CC: Asymptomatic before chest trauma, Incidental finding of a 24 mm tumor in the right lower lobe + PAPVC CECT: PAPVC: right upper lobe pulmonary vein into the SVC Intraoperative FNA: (Qp/Qs) of 0.98	DX; papillary adenocarcinoma with a maximum diameter of 2.1 × 1.5 cm and invasive diameter of 1.5 cm, at pathological stage IA2 with T1bN0M0 PLAN: Successful lobectomy + Not manipulating PAPVC + Good condition at 1 year FU
7	Gengxu He. et al. 2024 [23]	59-year-old male	CC: short of breathing and palpitation for 3 months PH/EX: A mid-systolic 2/6 murmur over the precordium in the mitral area. TTE: RVE and PAP: 51 mmHg + Mild TR CTPA: Dilation of PA + RA + RV LSPV connected to the LBCV lateral to the aortic arch, right PVs, and LIPV were seen draining normally into the LA	DX: PAPVC TX: Anastomosis of left superior vein and LA appendage through video-assisted thoracoscopy without cardiopulmonary bypass.

RVE: right ventricle enlargement, IVS: interventricular septum, MRA: magnetic resonance angiogram, RAD: Right axis deviation, RVH: right ventricular hypertrophy, RBBB: Right bundle branch block, mMRC: modified medical research council HP: hypersensitivity pneumonitis, RVSP: Right ventricular systolic pressure, RIPV: Right inferior pulmonary vein, RSPV: right superior pulmonary vein, LSPV: left superior pulmonary vein, LBCV: left brachiocephalic vein, LIPV: left inferior pulmonary vein,

simultaneous correction of other serious heart lesions and in cases of frequent respiratory infections. Other indications for intervention include persistent pulmonary hypertension (PHTN) and recurrent paradoxical emboli [18]. Open surgical correction remains the gold standard treatment for these patients. However, selected patients undergoing percutaneous transcatheter modification have demonstrated excellent outcomes. The selection is based on age, anatomical features, available expertise or devices, and clinical conditions [19]. Similar cases from the literature are summarized in Table 3 for reference. A graphical abstract illustrating and summarizing different aspects of this pathology is included in this manuscript (Fig. 5).

Clinical key message (conclusion)

Although rare, PAPVC can be a potentially debilitating anomaly if it is not diagnosed on time. The presentation is nonspecific, and accurate diagnosis primarily relies on multimodal imaging techniques, especially PCTA or PMRA. Many of these patients are misdiagnosed or experience delayed diagnosis, resulting in complications and long-term impairments in cardiac function, such as congestive heart failure. Therapeutic options are determined by clinical presentation, anatomical characteristics, and the severity of the shunt. Surgical management, if indicated, is the gold standard. However, new percutaneous intervention techniques have been applied successfully and are increasingly favored for selected patients.

Abbreviations

PAPVC	Partial Anomalous Pulmonary Vein Connections
NYHA FC	New York Heart Association Functional Class
RTLS	Right-To-Left Shunt
ASD	Atrial Septal Defect
TAPVC	Total Anomalous Pulmonary Vein Connections
PV	Pulmonary Vein
RAE	Right Atrium Enlargement
RVE	Right Ventricle Enlargement
PHTN	Pulmonary Hypertension
PAP	Pulmonary Arterial Pressure
CHD	Congenital Heart Disease
CCHD	Cyanotic Congenital Heart Disease
ICU	Intensive Care Unit
MDCTA	Multidetector Computed Tomography Angiography
PMRA	Pulmonary Magnetic Resonance Angiography

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12245-025-00809-3.

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

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None

Author contributions
P.E., T.A., N.G., and M.H.M contributed to the conceptualization, resource data curation and analysis, project administration, and writing of the initial draft. P.R. and F.N. contributed to the supervision, validation, visualization, investigation, methodology, software, and revision of the final draft of the manuscript. All authors read and approved the final manuscript.

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Data availability
Data is available on request due to privacy/ethical restrictions.

Declarations

Ethical approval
The study was performed in accordance with the ethical standards outlined in the 1964 Declaration of Helsinki and its later amendments. The Ethics Committee of the Day Hospital of Tehran approved the study. Considering that no identifying patient information or breach of confidentiality was involved, the committee waived the requirement for an ethics code.

Consent statement
The patient provided written informed consent to publish this report, following the journal's patient consent policy. The procedure was performed in accordance with the center's ethical policy.

Competing interests
The authors declare no competing interests.

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