# Isolated Pulmonic Valve Endocarditis: A Rare Clinical Entity

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**ABSTRACT**

**BACKGROUND:** Isolated pulmonic valve endocarditis is a rare heart valve infection, and constitutes about 1% to 2% of all infective endocarditis cases. Modified Duke's criteria were used to diagnose culture negative pulmonic valve endocarditis.

**CASE PRESENTATION:** A 52-year-old male patient presented with generalized body swelling of 1 month duration associated with prolonged fever, malaise, fatigue, and lassitude. He had productive cough, dyspnea on mild exertion, and reddish discoloration of urine. Upon physical examination, blood pressure (BP) = 140/90 mmHg, pulse rate (PR) = 104 beats per minute, respiratory rate (RR) = 26 breaths per minute, temperature ($T^0$) = 38.3°C, and $Sp0_2$ = 90% at ambient air. He had signs of bilateral pleural effusion. Cardiovascular examination revealed tachycardia, raised jugular venous pressure, murmurs of pulmonic regurgitation, and tricuspid regurgitation. There was grade 2 ascites and bilateral leg edema. On laboratory investigation, there were normochromic, normocytic anemia; raised ESR; positive Rheumatoid factor, elevated serum creatinine; and active urinary sediments on urinalysis. Two sets of blood culture were negative on days 1, 5, and 7. Chest-X-ray showed cardiomegaly with bilateral pleural effusion. ECG revealed sinus tachycardia with regular P-waves and QRS complexes. 2D Transthoracic echo showed vegetation on pulmonic valves, pulmonary valve lesions, dilated right atrium and right ventricle, and elevated right ventricular systolic pressure. Abdominal ultrasound revealed enlarged and echogenic kidneys, and ascites. Definitive diagnosis of PVE was made using modified Duke's criteria which was evidenced by 1 major (echo-proven vegetation on pulmonic valve), and 3 minors (suspected congenital pulmonic stenosis, fever, and immunologic phenomena [acute glomerulonephritis, positive rheumatoid factor]). The patient's clinical condition markedly improved after 2 weeks of intravenous antibiotics and loop diuretics, and discharged home after completing 6 weeks of parenteral antibiotics.

**CONCLUSION:** Modified Duke's criteria could play a major role in the management decision about diagnosis and empiric treatment of infective endocarditis in the absence of positive bacterial cultures.

**KEYWORDS:** Pulmonic valve, infective endocarditis, rare clinical entity, Ethiopia

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## Introduction

Isolated pulmonic valve endocarditis (PVE) is a rare heart valve infection and constitutes about 1% to 2% of all infective endocarditis cases.[1-3] It is often noticed in intravenous drug users, or in those who have congenital valve disease, central venous catheters, or intra-cardiac devices.[2-6] Here, we present a case of PVE, who might have congenital pulmonic valve stenosis as a risk factor. Diagnosis of PVE was made using modified Duke's criteria.

## Case Presentation

A 52-year-old male patient was admitted to medical ward of University of Gondar Hospital after presenting with generalized body swelling of 1-month duration, which started from the lower extremities and later involved the abdomen and face. He had associated prolonged fever, malaise, loss of appetite, fatigue, and lassitude. He had cough with phlegm but no chest pain or hemoptysis. He had shortness of breath on mild exertion, but no orthopnea, paroxysmal nocturnal dyspnea, or palpitation. He noticed reddish discoloration of urine, but no flank pain or change in urine volume. There was no preceding septic focus in the body. There were no preceding dental, gastrointestinal, and genitourinary instrumentations. He had no history of intravenous drug use. He was diagnosed to have hypertension 6 months back and was put on hydrochlorothiazide 25 mg po daily. No history of diabetes or chronic kidney disease. On physical examination, he was acutely sick looking, with vital signs, blood pressure (BP) = 140/90 mmHg, pulse rate (PR) = 104 beats per minute, respiratory rate (RR) = 26 breaths per minute, temperature (T0) = 38.3°C, and oxygen saturation ($Sp0_2$) = 90% at ambient air. He had conjunctival pallor but no icteric sclerae. Chest examination revealed reduced air entry with crackles in basal lung fields. On cardiovascular system, he had tachycardia (104 bpm) and raised jugular venous pressure (JVP). Apical impulse was located at 6th intercostal space lateral to the left midclavicular line. He had early diastolic murmur at left upper parasternal area. He had holosystolic murmur at left lower parasternal area, but there

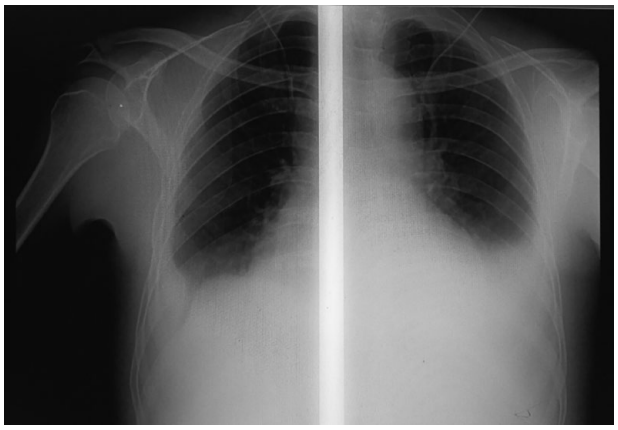

**Figure 1.** Chest-X-ray showing cardiomegaly with bilateral pleural effusion.
Abbreviations: AO, aorta; PA, pulmonary artery; RVOT, right ventricular outflow.

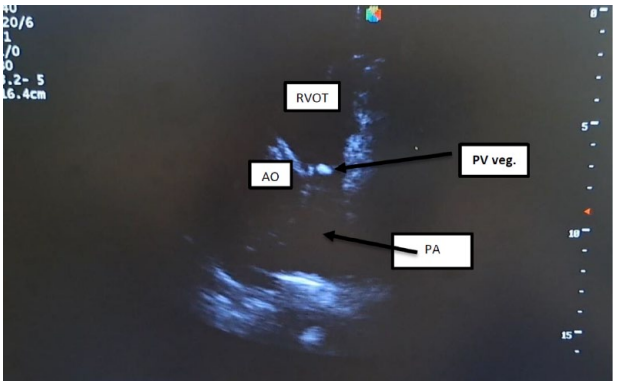


**Figure 2.** 2D parasternal short axis view at the great vessels level showing 12 mm × 10 mm mobile pulmonary valve vegetation, and mildly thickened pulmonic valves.
Abbreviations: AO, aorta; PA, pulmonary artery; RVOT, right ventricular outflow.

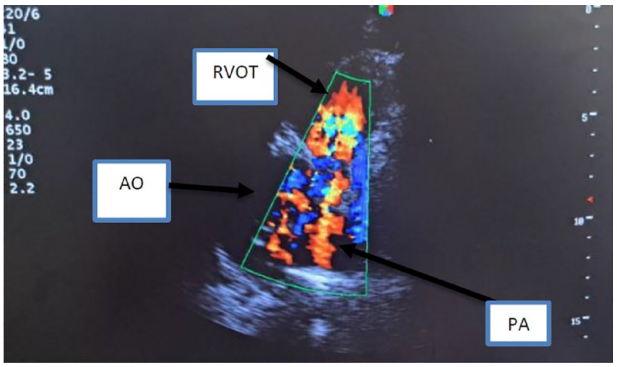


**Figure 3.** Parasternal short axis view at the great vessels level: color flow doppler at the pulmonary valve showing turbulent flow showing both pulmonary regurgitation and pulmonary stenosis.

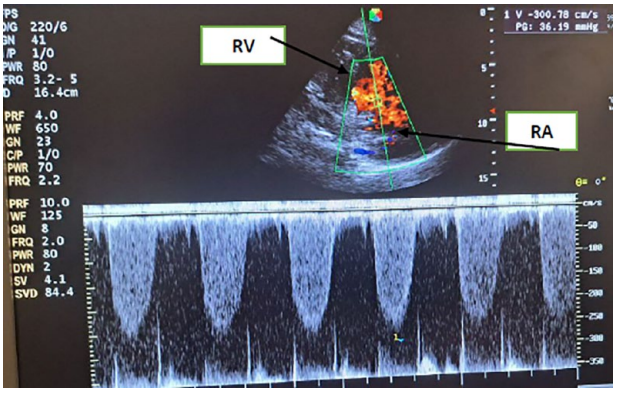


**Figure 4.** Parasternal long axis right ventricular inflow view showing tricuspid regurgitation with TV velocity 3 m/s, and right ventricular systolic pressure of 46 mmHg.

was no summation gallop. There were signs of peritoneal fluid collection (ascites), but no ballotable organ on abdominal examination. He had no costovertebral angle (CVA) tenderness. There was bilateral pitting leg edema. There were no petechial rashes, Osler's nodes, Janeway lesions, splinter hemorrhages, or clubbed fingers on integumentary system. Neurologic examination was unremarkable. Roth spots were not visualized on fundoscopic examination. On laboratory investigation, WBC = 4600/µl (normal range = 4500–10 000/µl) (neutrophils = 60%, lymphocytes = 30%), Hgb = 7g/dl (normal range = 12–16 g/dl), platelets = 244 000/µl (normal range = 150 000–450 000/µl), and ESR = 93mm in first hour (normal range 0–22 mm/hr). On urinalysis, protein +2, blood +3, many red blood cells/hpf, few granular casts/lpf. Serum creatinine = 2.9 mg/dl (normal range = 0.6–1.2 mg/dl), and blood urea nitrogen (BUN) = 54 mg/dl (normal range = 6–24 mg/dl). Serum total protein = 6.5 g/dl (normal range = 6.0–8.0 g/dl) and serum albumin = 3.5 g/dl (normal range = 3.5–5.0 g/dl). Serum rheumatoid factor (RF) was positive, while antinuclear antibody (ANA) was negative. Liver biochemical tests and serum electrolytes were within normal limits. Serological tests for hepatitis B, hepatitis C, and HIV were negative. No prolongation of coagulation profiles (PT, PTT, and INR). Two sets of blood culture were negative on days 1, 5, and 7. Chest-X-ray revealed cardiomegaly with bilateral pleural effusion (Figure 1). ECG revealed sinus tachycardia (rate = 110 bpm) with normal P-waves and QRS complexes. Trans thoracic Echocardiography (TTE) showed there was 10 mm × 12 mm sized vegetation on pulmonic valve (Figure 2). There was thickened and scalloped pulmonic valve suggestive of pulmonary stenosis, and moderate pulmonary regurgitation on color flow study (Figure 3). Right ventricular systolic pressure was 46 mmhg (Figure 4). There were dilated right atrium and right ventricle with interatrial septal deviation to the left atrium. Right ventricular systolic function was normal (TAPSE = 19 mm) and had normal right ventricular wall motion and contractility. The inferior venacava demonstrated partial (50%) collapse. There was left ventricular hypertrophy but no wall motion abnormalities. There were no abnormalities of mitral and aortic valves. LVEF was 65%. Abdominal ultrasound revealed slightly enlarged and echogenic kidneys, and anechoic fluid collection in the peritoneal space. Based on

clinical, laboratory, and imaging evaluation, diagnosis of stage C, NYHA class III, heart failure secondary to pulmonic valve infective endocarditis was made. He had fulfilled 1 major (pulmonic valve vegetation on echocardiography) and 3 minor criteria (congenital pulmonary valve anomaly, fever, and immunologic phenomena [acute glomerulonephritis, positive rheumatoid factor]) for the diagnosis of infective endocarditis using Modified Duke's criteria. The patient was put on vancomycin 1 g IV twice daily and ceftriaxone 1 g IV twice daily for 6 weeks. Furosemide 40 mg IV three times daily was given for 2 weeks and de-escalated on second week of admission. He had significant improvement after 2 weeks of parenteral antibiotics and loop diuretic use. Renal function tests and urinalysis abnormalities normalized on fourth week of admission. He was discharged home after 1 week of completion of antibiotics, and appointed to cardiac clinic of the hospital for regular follow-up. There was marked regression of vegetation size (5 mm × 6 mm) on repeat echocardiography on third month of hospital discharge.

## Discussion

Right-sided endocarditis is less common than left-sided endocarditis, and it accounts for 5% to 10% of all infective endocarditis cases. Majority (90%) of right-sided IE involve the tricuspid valve. Isolated pulmonic valve endocarditis (PVE) is extremely rare, affecting 1% to 2% of all infective endocarditis cases.[1,2] The possible reasons for low incidence of right-sided IE include lower prevalence of congenital malformations or acquired valve abnormalities, difference in endothelial lining and vascularity of right-sided valves, lower pressure gradient and jet velocities across right sided valves, and lower oxygen content of venous blood.[2,3] Right-sided IE is more frequently observed among intravenous drug users, patients with central intravenous catheters or intra-cardiac implantable devices, patients with congenital heart disease, and immunosuppression or comorbidities (HIV, diabetes, or chronic kidney disease). Almost one-third (28%) of pulmonic valve endocarditis cases have been reported with no identifiable risk factors.[4,6] Our patient might have congenital pulmonic valve stenosis on echocardiography, which showed scalloped and thickened pulmonic valve in the absence of left-sided valvular lesions. Modified Duke's criteria were applied to confirm diagnosis of infective endocarditis.[7] Our patient had one major (echo-proven vegetation on pulmonic valve), and 3 minors (suspected congenital pulmonic valve anomaly, fever, and immunologic phenomena [acute glomerulonephritis, positive rheumatoid factor]). Medical literatures described true culture-negative infective endocarditis is mainly caused by, Coxiella burnetii, Bartonella spp, Legionella spp., and Chlamydia spp. These organisms require special enriched culture media to grow. Serologic tests and PCR of tissue material alsohelp to diagnose IE caused by these organisms. Other reasons for culture-negative IE could be prior antibiotic exposure before blood culture and endocarditis caused by fastidious organisms like HACEK group organisms and nutritional variant streptococci such as Abiotrophia defectiva. Trans-thoracic echocardiography (TTE) was the imaging modality in our case. Trans-esophageal echocardiography (TEE) would be used in negative transthoracic imaging with high clinical suspicion of IE.[8] The echocardiographic differential diagnoses of pulmonic valve vegetation are non-bacterial thrombotic endocarditis, cardiac myxoma, and cardiac papillary fibroelastoma.[8-10] Non-bacterial thrombotic endocarditis is a competing differential diagnosis for culture negative infective endocarditis, and affects those with advanced malignancy, systemic hypercoagulable state, and systemic lupus erythematosus. Most of the thrombotic vegetation is located in left-sided valves. Two-thirds involve the mitral valve and a quarter is sited in the aortic valve.[8] Most cardiac myxoma occurs in the atrium. Of all cardiac myxomas, three-fourths is located in the left atrium, and the remaining is situated in the right atrium. Myxoma involving heart valves is rare.[9] Most cardiac papillary fibroelastoma are located on left-sided valves. Right sided valves are rarely involved. It is diagnosed incidentally without symptoms or with symptoms of embolization (ischemic stroke, myocardial infarction, pulmonary embolism), or syncope, heart failure or sudden death (cardiac obstruction).[10] Since blood culture was negative in our case, empirical antibiotics was started with intravenous vancomycin and ceftriaxone for 6 weeks to cover both staphylococcal spp. and streptococcal spp.[4,6] The patient improved markedly with medical treatment. He had significant improvement on the second week of parenteral antibiotics use. Renal function tests and urinalysis abnormalities normalized on fourth week of therapy. Repeat echo showed marked regression of vegetation size on third month of hospital discharge. The patient had no clear-cut indications for surgery. Surgical indications include severe valvular regurgitation with refractory heart failure, persistent bacteremia, or recurrent emboli despite appropriate antibiotic use, IE complicated by heart block or abscess formation, severe regurgitation with large mobile vegetation, or the presence of multi-drug resistant organisms.[4-6]

## Conclusion

Modified Duke's criteria could play a major role in the management decision about diagnosis and empiric treatment of infective endocarditis with suspected or known risk factors in the absence of positive bacterial cultures in limited clinical settings.

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## Author Contributions

All authors contributed to data analysis, drafting or revising the article, have agreed on the journal to which the article will be submitted, gave final approval of the version to be published, and agreed to be accountable for all aspects of the work.

## Availability of Data and Materials

All data sets of the case report are available from the corresponding author, and on reasonable request from the editors.

## Ethical Approval

Our institution does not require ethical approval for reporting individual case reports.

## Informed Consent

Written informed consent was obtained from the study subject.

## Consent for Publication

Written informed consent was obtained from the patient for publication of the case report and any accompanying images. A copy of the written consent was available for review by the Editor-in-Chief of this journal.

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## REFERENCES

1. Ramadan FB, Beanlands DS, Burwash IG. Isolated pulmonic valve endocarditis in healthy hearts: a case report and review of the literature. *Can J Cardiol*. 2000;16:1282-1288.
2. Ranjith MP, Rajesh KF, Rajesh G, et al., Isolated pulmonary valve endocarditis: a case report and review of literature. *J Cardiol Cases*. 2013;8:161-163.
3. Samaroo-Campbell J, Hashmi A, Thawani R, Moskovits M, Zadushlivy D, Kamholz SL. Isolated pulmonic valve endocarditis. *Am J Case Rep*. 2019;20:151-153.
4. Zhang MX, Zhang WM, Yu C, et al., Isolated pulmonary valve endocarditis with rapid progression: a case report and literature review. *J Cardiothorac Surg*. 2021;16:16.
5. Shmueli H, Thomas F, Flint N, Setia G, Janjic A, Siegel RJ. Right-sided infective endocarditis 2020: challenges and updates in diagnosis and treatment. *J Am Heart Assoc*. 2020;9:e017293.
6. Bayer AS, Bolger AF, Taubert KA, et al. Diagnosis and management of infective endocarditis and its complications. *Circulation*. 1998;98:2936-2948.
7. Evangelista A, Gonzalez-Alujas MT. Echocardiography in infective endocarditis. *Heart*. 2004;90:614-617.
8. Zmaili M, Alzubi J, Lo Presti Vega S, Ababneh E, Xu B. Non-bacterial thrombotic endocarditis: a state-of-the-art contemporary review. *Prog Cardiovasc Dis*. 2022;74:99-110.
9. Tanabe S, Yano K, Mizunaga T, et al. Pulmonary valve myxoma requiring pulmonary valve replacement: a case report. *Surg Case Rep*. 2022;8:68.
10. Sun JP, Asher CR, Yang XS, et al. Clinical and echocardiographic characteristics of papillary fibroelastomas: a retrospective and prospective study in 162 patients. *Circulation*. 2001;103:2687-2693.