E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/15.01.147

Case Report

The Critical Role of Clinical Examination in Diagnosing Atrial Septal Defect in an Elderly Male with Haemoptysis: A Case Report

Dr.Krishna Harsha Bhaskara[1*], Dr.M A Uma[2]
[1*]Post Graduate, Internal Medicine, P.E.S. Institute of Medical Sciences and Research, Kuppam.
[2]Professor and Head, Department of Internal Medicine, P.E.S. Institute of Medical Sciences and Research, Kuppam.
*Corresponding author: Krishna Harsha Bhaskara
Email: bvkh2297@gmail.com
Received: 07.03.25, Revised: 30.04.25, Accepted: 24.05.25

Abstract
Atrial septal defects (ASDs) are among the most prevalent congenital heart defects and often remain undiagnosed until adulthood. They may present later in life with a diverse range of symptoms, including arrhythmias, thromboembolic events, heart failure, and haemoptysis. Although diagnosis can typically be established through echocardiography, limitations in technical expertise in resource-constrained settings may necessitate reliance on clinical examination. Here, we report a case of an elderly male presenting with haemoptysis, whose initial echocardiogram failed to identify any congenital heart defect. However, clinical findings raised suspicion of an ASD, later confirmed by transoesophageal echocardiography as a 22-mm ostium secundum defect.

INTRODUCTION

Atrial septal defect (ASD) is one of the most common congenital heart anomalies, with the ostium secundum type being the most frequently encountered. This defect results from inadequate development of the septum secundum, excessive resorption of the septum primum, or a combination of both. Atrial septal defect (ASD) represents a direct communication between atrial chambers, allowing shunting of blood between the systemic and pulmonary circulation.[1] While ASDs are usually detected during childhood, some cases present later due to the gradual development of right ventricular remodelling and enlargement of the right heart chambers.[2]Untreated late presentations can lead to irreversible pulmonary vascular remodelling and advanced right ventricular failure, underscoring the importance of timely diagnosis and intervention [2].[2]

Case Presentation

A 72-year-old male, a former smoker of 20-25 years who quit 15 years prior, presented with a one-week history of grade 2 dyspnoea and 1-2 episodes of streaky haemoptysis. The patient had no prior comorbidities or associated symptoms such as fever, weight loss, chest pain, palpitations, or orthopnoea. On examination, he had bradycardia (heart rate: 45 bpm), blood pressure of 124/72 mmHg, respiratory rate of 17-19 breaths/min, and oxygen saturation of 90-92% on room air. No cyanosis, clubbing, or lymphadenopathy was noted.

Respiratory examination revealed bilateral rhonchi with normal vesicular breath sounds, while cardiac assessment demonstrated a parasternal heave, palpable P2, and a loud P2 with a wide A2P2 split. A grade 3 ejection systolic murmur was heard in the pulmonary area.

Considering the history and clinical findings, a provisional diagnosis of chronic obstructive pulmonary disease (COPD) or bronchogenic carcinoma was made, with pulmonary embolism included as a differential. The patient was started on oxygen therapy, bronchodilators, and corticosteroids.

Initial investigations revealed first-degree heart block with sinus bradycardia on ECG. A chest X-ray showed significant pulmonary artery enlargement. A 2D echocardiogram indicated concentric left ventricular hypertrophy, moderate mitral and tricuspid regurgitation, severe pulmonary artery hypertension, and right ventricular dysfunction, without evidence of regional wall motion abnormalities. The initial echocardiography did not report of any congenital heart defects.

A contrast-enhanced CT pulmonary angiogram revealed a dilated pulmonary artery (4.8 cm diameter) and severe pulmonary hypertension, with mild COPD changes but no evidence of pulmonary embolism, (Figure 1). The

haemoptysis was attributed to pulmonary hypertension; however, the severity of pulmonary hypertension was deemed disproportionate to the observed respiratory findings. Given these discrepancies, an evaluation for congenital heart disease was pursued.

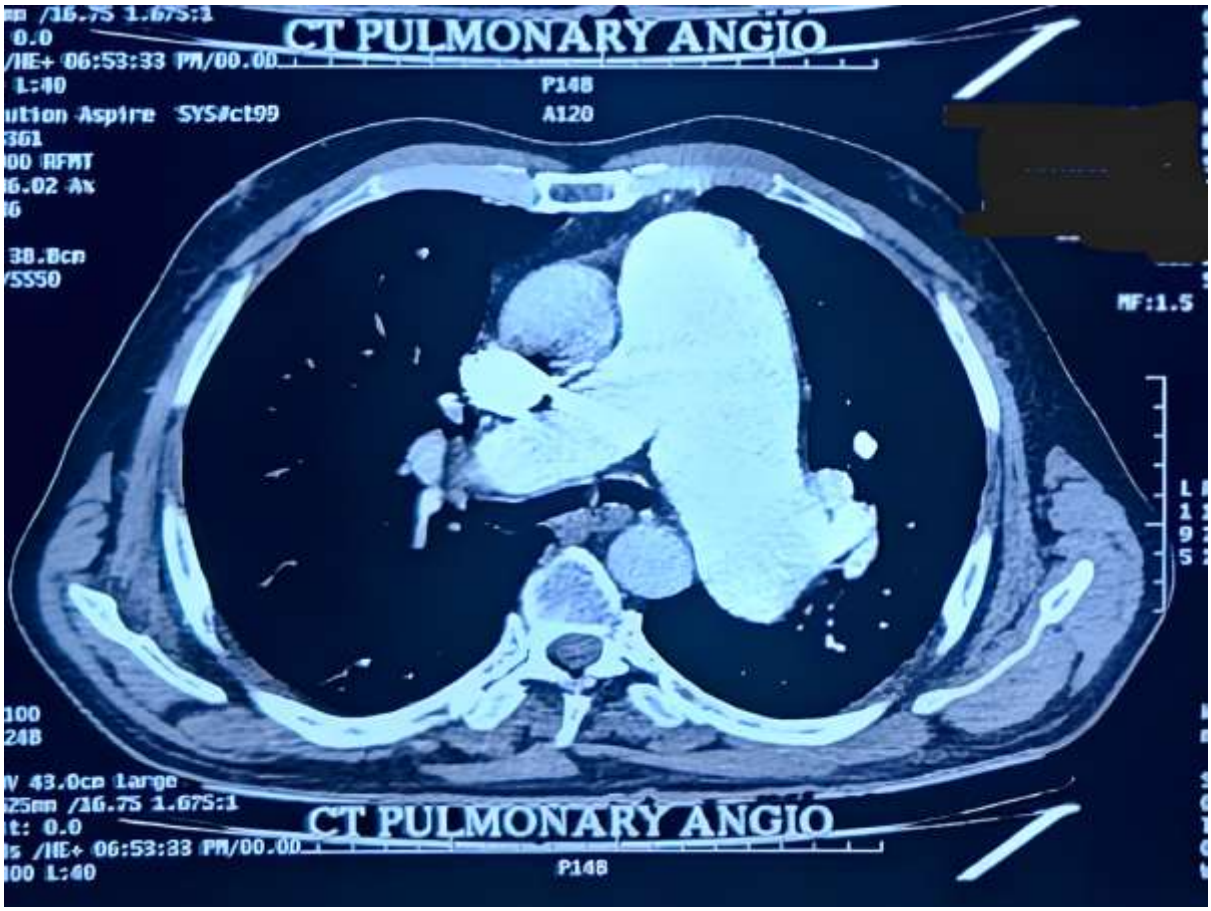

FIGURE 1: CT image showing dilated pulmonary artery

Despite the findings on initial 2D echocardiogram, clinical suspicion of ASD persisted due to the presence of a wide and fixed S2 split. The cardiologist was requested to review the echocardiogram. A saline contrast echocardiogram revealed evidence of septal flow, prompting a transoesophageal echocardiogram (TEE), which confirmed a 22-mm ostium secundum ASD with a left-to-right shunt, a floppy superior rim, and deficient retro-aortic rim (Figure 2). The patient was referred for a cardiac catheterization study to evaluate the reversibility of pulmonary hypertension and was initiated on medical therapy.

FIGURE 2: Transoesophageal Echocardiography showing 22mm Atrial Septal Defect

E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/15.01.147

DISCUSSION

Pulmonary artery hypertension (PAH) is observed in 6-35% of patients with ostium secundum ASDs and is defined by a pulmonary artery systolic pressure (PASP) ≥40 mmHg.[3] Very small ASDs (diameter 10 mm) typically present with symptoms in the third decade of life.[4]

Echocardiography remains the cornerstone of ASD diagnosis, providing critical information about the defect's size, shunt magnitude, and hemodynamic impact.[4,5] When initial echocardiographic findings are inconclusive, transoesophageal echocardiography or saline contrast studies can confirm the diagnosis. A well-structured clinical examination remains essential, even in the current era of advanced diagnostic investigations. Investigations should serve as confirmatory tools rather than being relied upon blindly. In this case, TEE played a pivotal role in identifying the ASD after initial investigations failed to do so.

While surgical or transcatheter closure is the definitive treatment for symptomatic ASDs, considerations must be made for complications such as atrial fibrillation or acute left ventricular failure. There have been no large, truly randomized comparisons of surgery with transcatheter closure of ASDs as the design of such a study is problematic partly because, given a choice between surgery and device closure, parents and patients often prefer the latter method. Nevertheless, surgery is the gold standard against which transcatheter closure of ASDs has been and should be judged, not least because longer term follow up is available for surgical treatment.[6]

Individuals with impaired left ventricular diastolic function may manifest abrupt rise in left atrial and left ventricular filling pressures once the ASD is completely occluded, which may result in pulmonary oedema, pulmonary hypertension and atrial or ventricular arrhythmias. This phenomenon has been mainly described in older patients with diastolic dysfunction undergoing transcatheter closure of secundum ASD.[7] Strategies such as preconditioning the left ventricle through balloon occlusion can mitigate these risks.

Exercise capacity of patients with unrepaired ASDs depends on the importance of the shunt, the right ventricular (RV) function and volume overload, the level of pulmonary arterial pressure, and the occurrence of arrhythmias. For repaired ASDs, exercise capacity also depends on the delay before closure and the type of procedure (catheter or surgery).[8] Patient selection is critical in determining post-procedural outcomes. Although most patients achieve symptomatic relief and improved exercise capacity after ASD closure, those with preoperative severe pulmonary hypertension or reduced oxygen uptake may experience persistent limitations.[8]

CONCLUSIONS

In elderly patients presenting with haemoptysis, congenital heart defects such as ASDs should remain part of the differential diagnosis, particularly when initial findings suggest severe pulmonary hypertension. This case highlights the importance of thorough clinical evaluation and the utility of advanced imaging modalities in identifying ASDs in adults. In our case, conducted in a resource-poor setting, the initial echocardiography reported no congenital heart defects. However, the presence of a loud P2, a wide fixed split S2, and a grade 3 murmur prompted us to seek further evaluation by a cardiologist. A saline contrast echocardiogram and transoesophageal echocardiography subsequently confirmed a 22mm atrial septal defect. The patient was started on medical therapy and eventually advised to undergo a cardiac catheterization study at discharge. Timely diagnosis and management are essential for improving long-term outcomes.

REFERENCES

1. Brida M, Chessa M, Celermajer D, Li W, Geva T, Khairy P, et al. Atrial septal defect in adulthood: a new paradigm for congenital heart disease. Eur Heart J. 2022;43(28):2660-2671.
2. Lopes AA, Mesquita SM. Atrial septal defect in adults: does repair always mean cure? Arq Bras Cardiol. 2014;103(6):446-8.
3. Jain S, Dalvi B. Atrial septal defect with pulmonary hypertension: when/how can we consider closure? J Thorac Dis. 2018;10(Suppl 24):S2890-S2898.
4. Martin SS, Shapiro EP, Mukherjee M. Atrial septal defects - clinical manifestations, echo assessment, and intervention. Clin Med Insights Cardiol. 2015;8(Suppl 1):93-8.
5. Diaconu CC. Atrial septal defect in an elderly woman-a case report. J Med Life. 2011;4(1):91-3.

6. Spence MS, Qureshi SA. Complications of transcatheter closure of atrial septal defects. Heart. 2005;91(12):1512-4.
7. Torres AJ. Hemodynamic assessment of atrial septal defects. J Thorac Dis. 2018;10(Suppl 24):S2882-S2889.
8. Barron AJ, Wensel R, Francis DP, Malik I. The role for cardiopulmonary exercise testing in patients with atrial septal defects: a review. Int J Cardiol. 2012 Nov 15;161(2):68-72.