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CASE REPORT

A case of sinus venosus atrial septal defect misdiagnosed as primary pulmonary hypertension

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Electrocardiogram

Abstract We present a case of sinus venosus atrial septal defect in a patient who was previously diagnosed as having primary pulmonary hypertension in a tertiary care center. Our findings are based on 2-dimensional trans-thoracic echocardiography, chest X—ray and surface electrocardiogram. A 26-year-old man, previously diagnosed as a case of primary pulmonary hypertension, presented to the emergency department (ED) with chest pain and breathlessness on exertion. Cardiac biomarkers were within their normal ranges. Surface electrocardiogram showed right atrial and ventricular overload with right axis deviation. Chest imaging noted enlarged central pulmonary vascularity with bilateral plethoric lung fields.

Trans-thoracic echocardiography showed a dilated right atria and ventricle with severe tricuspid regurgitation and severe pulmonary artery hypertension with an intact atrial septum. Surprisingly, the transoesophageal echocardiogram revealed the presence of a sinus venous superior vena cava-type atrial septal defect with the right pulmonary vein draining into the right atria.

In this full-text version, we present a more detailed discussion of sinus-venous atrial septal defect associated with partial anomalous pulmonary venous return that was wrongly diagnosed as a case of primary pulmonary hypertension in a tertiary care center.

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1. Introduction

Sinus venous atrial septal defects (ASD) vary in that the atrial septum is intact except in the superior portion adjacent to the superior vena cava and can coexist with partial anomalous pulmonary venous connections. Diagnosis by trans-thoracic echocardiography (TTE) is difficult, although trans-oesophageal echocardiography (TEE) can contribute to the diagnosis of sinus venous defects and assessment of associated anomalies. However, even in tertiary care centers, facilities in developing countries may lack the ability to perform trans-oesophageal echocardiography (TEE) and cardiac catheterization, and sinus venous ASD is often misdiagnosed as primary pulmonary hypertension. We report a case of superior vena caval sinus venous ASD that was misdiagnosed and wrongly treated as primary pulmonary hypertension based on 2-dimensional trans-thoracic echocardiography (TTE), chest X-Ray and electrocardiographic (ECG) findings.

2. Case report

A 26-year-old male presented to the emergency department with chief complain of acute onset chest pain for 4 hours. The pain was retrosternal without any typical radiation and was associated with uneasiness and chest heaviness. On inquiring about past history, the patient noted that he had experienced breathlessness for the previous 3—4 months. This was gradual in onset and progressive in nature; initially he felt breathlessness on usual ordinal outdoor activities with slight limitations of his physical activities, and at present he became breathless on less than ordinal activities with marked limitation of physical activities. There is no history of seasonal or diurnal variation of his breathlessness. That patient had no history of cough, hemoptysis, back pain, chest trauma, orthopnea or paroxysmal nocturnal dyspnea. There is no childhood history of acute rheumatic fever. The patient is non-hypertensive, non-diabetic, a non-smoker, a non-alcoholic and vegetarian in diet. There is no past history of pulmonary tuberculosis, systemic hypertension or diabetes mellitus. The patient belongs to a lower socioeconomic stratum. After receiving his medical records from a tertiary care centre in eastern Uttar Pradesh in north India, it was found that he was on oral diuretics, calcium channel blockers and sildenafil citrate for primary pulmonary hypertension for the last 2 years with some degree of symptomatic relief in between. However, he had left the treatment for the previous 6 months due to financial issues.

On examination, the patient was hemodynamically stable with a pulse rate of 110 per minute that was regular, normovolumic, and normal in character without any radio radial or radio femoral delay. All peripheral pulses are equally palpable with normal condition of the arterial wall. The patient's blood pressure was 128/78 mm Hg in the right arm while in a sitting position using an adult blood pressure measuring cuff. There was no significant difference between the upper limbs or between the upper and lower limbs. The patient was of adequate build and nutrition with a body mass index of 21. He was afebrile at the time of presentation, with an arterial oxygen saturation of 98% measured by a pulse oxymeter. Pallor, cyanosis, clubbing, icterus, edema and lymph node enlargement were absent. The jugular venous pressure was normal with prominent v wave and y descent. Hepatojugular reflux was absent. On examination of the cardiovascular system, we found that the S1 was normal and the S2 was normally split with a loud P2 and a grade III pansystolic murmur present at lower left parasternal area that increased in intensity on inspiration.

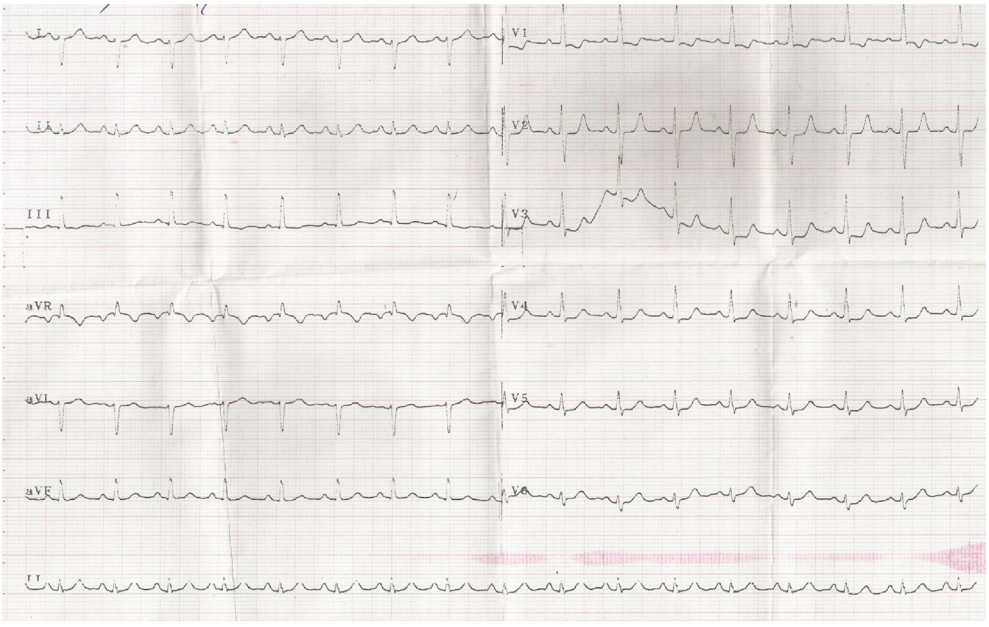

Figure 1 Electrocardiogram of patient showing right axis deviation with RAA and RVH. RAA-Right atrial abnormality, RVH-Right ventricular hypertrophy.