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# Cardiac CT Angiography of a Membranous Ventricular Septal Aneurysm Short Title: Ventricular Septal Aneurysm

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The most frequent congenital heart defects in the neonatal period are ventricular septal defects. Ventricular septal aneurysms can rarely develop from an interventricular septal (IVS) defect in adults. We describe a 47-year-old man with an aneurysm in the IVS growing towards the right ventricle, which was confirmed by cardiac computed tomographic angiography and was missed by echocardiography.

*Keywords:*Cardiac; Angiography; Ventricular; Defects; Aneurysm

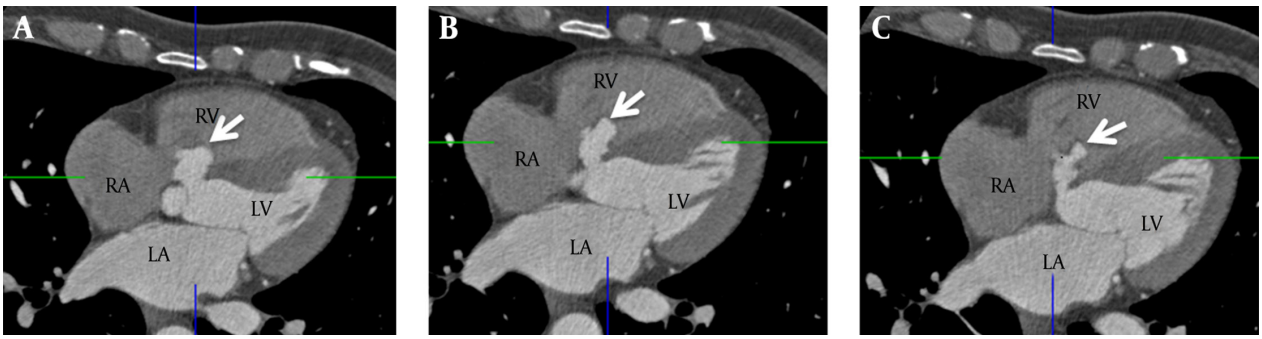
## 1. Introduction

Interventricular septal (IVS) defects are among the most frequent cardiac anomalies seen in childhood (1). The system developed by Soto et al. is frequently used to classify IVS defects (1). The IVS has two components: the small membranous septum and the much larger muscular septum (1). The most frequent congenital heart defects in the neonatal period are ventricular septal defects (VSDs), which usually occur in the muscular septum. In adults, IVS defects are rare and are usually seen in the membranous septum. Ventricular septal aneurysms (VSAs) can rarely develop from an IVS defect in adults (2). They are usually detected using cardiac echocardiography or angiography or at autopsy. Recently, the increasing number of cardiac multidetector computed tomography (MDCT) and magnetic resonance imaging (MRI) studies has enabled the detection of asymptomatic VSAs in vivo.

## 2. Case Presentation

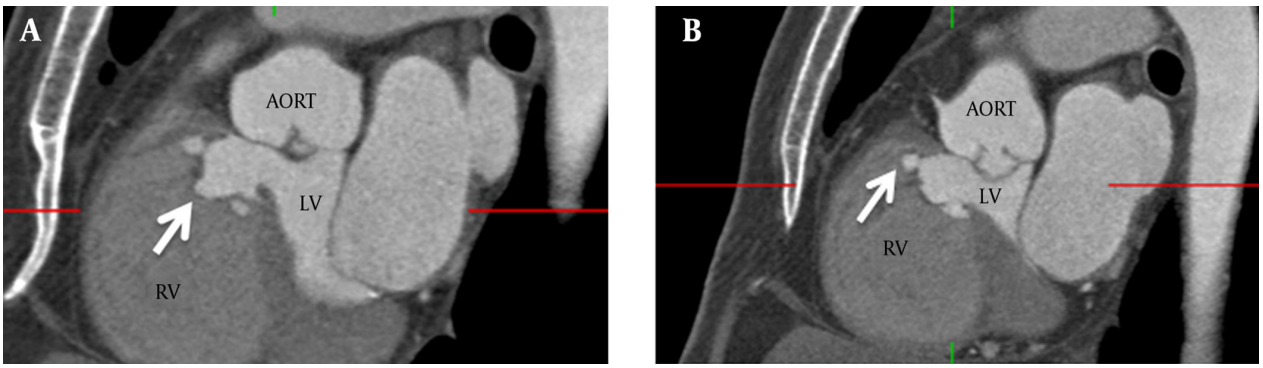
A 47-year-old man presented to the cardiology clinic complaining of exertional chest pain. No abnormalities were found on echocardiography. The resting electrocardiogram (ECG) and laboratory results were normal. There were signs of ischemia on stress testing, so cardiac MDCT was planned. An ECG-triggered cardiac MDCT study was performed after injecting 75 mL of a non-ionic contrast fluid via an antecubital vein with a 64-detector row Toshiba Multiline Aquiline 64 System (Toshiba Medical Systems, Otawara, Japan). The images were reviewed on a Vitrea Workstation (Vital Images, Fairfield, IA); they showed a 30×25×30-mm aneurysm in the IVS growing towards the right ventricle (RV). The contrast material filled the aneurysm (Figures 1 A, B, and c and Figures 2 A, B, and C). No thrombi were detected within the aneurysm. The coronary arteries showed no pathology. No narrowing of the RV outlet was seen on echocardiography.

**Figure 1.** Axial Images (A, B, and C), Showing a Lobulated Out-Pouching Lesion Measuring Approximately




25 mm at its longest diameter in the membranous portion of the ventricular septum; Abbreviations: LA; left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

**Figure 2.** Cardiac Computed Tomography, Showing a Sagittal View of the Aneurysm of the Membranous Septum (arrow)




In this image, the left atrium, left ventricle, and ascending aorta are opacified with iodinated contrast medium; Abbreviations: LV, left ventricle; RV, right ventricle.

## 3. Discussion

The cardiac chambers develop *in utero*. The ventricles are divided by the IVS, the development of which ends at birth (1). Problems with the IVS development can cause defects in the muscular or membranous septum seen after birth. In the neonatal period, most IVS defects are located in the muscular ventricular septum (1). Kirklin et al. classified VSDs as anterior, mid-muscular, apical, and posterior; moreover, 50% of neonatal heart defects are VSDs. In 80% of VSD cases, there are other congenital heart defects, while 20% of the cases are isolated VSDs. Most VSDs close spontaneously during the first year of life (1).

In adulthood, IVS defects are rarely diagnosed in the membranous septum (3). In an MDCT study of 402 cardiac patients, Choi et al. reported only eight VSAs, for an incidence rate of 2.4% (2, 3). In 1408 cardiac MDCT studies, we have found only one case of VSA.

In adulthood, IVS defects are seen in the membranous septum. Given the lower pressure in the RV compared to the left ventricle (LV), aneurysms grow from the IVS defect into the RV (2). A VSA can cause clinical findings depending on its location and size (2). The size of VSAs arising from an IVS defect in adulthood depends on the pressure gradient across the IVS (3). VSAs tend to grow toward the RV and can cause RV-outlet narrowing as they grow (2). Other causes of RV-outlet narrowing include pulmonary stenosis, double-chambered RV, double-outlet RV complex, and aortic regurgitation. Pulmonary stenosis is the most common RV-outlet pathology (2, 4). Such patients can be diagnosed easily with echocardiography (4). Not all patients with IVS defects develop a VSA. In patients with open defects, a shunt can develop between the RV and LV (2, 4). The clinical complaints depend on the size of the IVS defect. Larger defects cause double-outlet RV complex (4). In these patients, hemodynamic problems arise from the large connection between the RV and LV. Other cases might be asymptomatic for years. In adults, membranous septal defects can cause paradoxical embolisms (4). A VSA can appear as a "pseudo mass" in routine CT and MRI studies depending on the contrast material timing (2). Closed atrial septal defects and VSAs might cause atrial and intraventricular filling defects. Primary cardiac tumors and metastatic lesions are usually seen within the atria (4). In cancer patients who develop a VSA, routine CT and MRI studies might suggest false metastasis (2). VSAs should be included in the differential diagnosis in patients with a cardiac mass, and CT and MRI can be used to diagnose VSAs (1, 3). In cardiac MRI, four-chamber images and sequenced contrast imaging are very helpful in diagnosing aneurysms. The diagnostic superiority of cardiac MRI is because the lesion contents show different signal properties in different sequences and contrast patterns in contrast sequences (2). The rapid filling and emptying of the contrast material from the lesion on MRI and the absence of solid components are typical of aneurysms (4). The movement of the contrast material within the ventricles in cardiac MRI perfusion sequences is helpful for diagnosing aneurysms. On MRI, thrombi in a VSA appear as filling defects (2).

In cardiac MDCT studies, images are obtained when the coronary arteries fill with contrast material or when the contrast material is within the LV or aorta. VSAs grow into the RV because of the higher pressure in the LV (1). Therefore, the ideal time for VSA imaging is when the LV is full of the contrast material, but the RV has none (2). That is why cardiac MDCT is useful for VSA imaging. ECG-synchronized techniques enable high-resolution cardiac MDCT images. Cardiac MDCT can also be used to study the morphology of VSAs and the aneurysm lumen (2-4). In our patient, the IVS defect grew into an aneurysm in the RV. VSAs growing into the RV can cause the narrowing of the ventricular outlet and hemodynamic problems. Our patient had no clinical RV-outlet pathology despite the large size of his VSA (2).

## Authors' Contributions

Sedat Altay reported the case. All the authors assisted in the writing and translating of the manuscript. Orhan Oyar was the senior author.

## References


1. Minette MS, Sahn DJ. Ventricular septal defects. *Circulation.* 2006;**114**(20):2190–7.
2. Choi M, Jung JI, Lee BY, Kim HR. Ventricular septal aneurysms in adults: findings of cardiac CT images and correlation with clinical features. *Acta Radiol.* 2011;**52**(6):619–23.
3. Ammash NM, Warnes CA. Ventricular septal defects in adults. *Ann Intern Med.* 2001;**135**(9):812–24.
4. Bashore TM. Adult congenital heart disease: right ventricular outflow tract lesions. *Circulation.* 2007;**115**(14):1933–47.