PICTORIAL ESSAY

Non-Coronary Cardiac Findings and Pitfalls in Coronary Computed Tomography Angiography

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ABSTRACT

Non-coronary incidental findings are not rare. Kirsch et al found 67% non-coronary abnormalities with coronary computed tomography angiography (CCTA). Radiologists are expected to identify the extracoronary, intra- and para-cardiac anatomical structures and distinguish them from pathologic processes in CCTA. We have reviewed 2000 CCTA studies done at our institution with 64-MDCT. This pictorial essay presents case studies of non-atherosclerotic cardiovascular findings to recognize cardiac anatomic structures and how to distinguish them from pathologic processes. Correct interpretation of benign, clinically insignificant findings is crucial to avoid unnecessary additional imaging tests.

Key words: Coronary computed tomography angiography, incidental non-coronary cardiac findings, intra-cardiac shunt, paracardiac structures, non-coronary cardiac structures

INTRODUCTION

Coronary computed tomography angiography (CCTA) is widely used in clinical practice. Clinicians, especially radiologists are expected to evaluate all extra-coronary, intra-, and para-cardiac anatomical structures and distinguish them from pathologic processes in coronary CCTA. Non-coronary incidental findings are not rare. Kirsch et al found 67% non-coronary abnormalities with CCTA.[1] Understanding the anatomy of the cardiac and para-cardiac structures is a key in assessing non-coronary cardiac incidental findings. It is important to identify the intra-cardiac anatomical structures that can mimic a disease and distinguish them from pathologic processes using reformatted images. This pictorial essay describes the anatomy of the cardiac and para-cardiac structures and reviews the appearance of non-coronary incidental findings and pitfalls in CCTA.

METHODS

We have reviewed 2000 CCTA studies performed at our

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Coronary computed tomography angiography scan protocol

Dose of the non-ionic contrast used intravenously was 350 mgI/mL over a duration of 12 s. The average flow rate was 4.1 ± 2.8 mL/s and total amount of the contrast was 50.2 ± 10.3 mL. This was followed by 0.9% saline solution at a fixed duration of 7 s and the same flow rate as the contrast material.

If the heart rate was less than 60 beats per minute (bpm), CCTA was performed using the step and shoot (Snapshot Pulse) sequence. In patients with heart rate of more than 60 bpm, CCTA was performed using the helical technique with low helical pitch (cardiac helical). Patients with irregular heart rates or heart rates more than 60 bpm received intravenous beta-blocker. The dose administered was IV metoprolol in 5 mg doses, up to a maximum of 20 mg. The aim was to keep the heart rate below 60 bpm.

Coronary computed tomography angiography parameters used

Snapshot pulse

120 kV variable mA 0.35 s/rot. temporal resolution 230 ms, 0.625 mm slice, 0.625 mm interval, R-R interval 75% (center) padding time 150 ms.

Cardiac helical

120 kV variable mA 0.35 s/rot. temporal resolution variable, 0.625 mm slice, 0.625 (or 0.312) mm interval, beam pitch variable (0.16–0.20) HR60-65: ECG dose modulation (R-R 65–85%).

Images were analyzed on a CT post-processing workstation (Advantage Windows, GE Healthcare) using commercially available software. Standard axial images acquired during end-diastole, volume rendering images and corresponding 2D multiplanar reformations were used for image interpretation.

Non-coronary-related intra-cardiac findings

It is important to pay attention to the cardiac chambers and myocardium in addition to coronary arteries to assess for congenital and anatomical variants that may mimic an abnormality. There are several normal variants as described below.

Cor triatriatum: mimicking the left atrial mass

This congenital anomaly was first described by Church in 1868, as a rare congenital heart defect, accounting for 0.1% of all congenital heart diseases.[2] It is characterized by the presence of a fibromuscular membrane [Figures 1a and 1b] dividing the left atrium into two chambers: A posterosuperior chamber into which the pulmonary veins drain, and an anteroinferior chamber that communicates with the left atrial appendage and the mitral valve.[3]

Crista terminalis: mimicking the right atrial mass

A vertically orientated fibromuscular ridge at the posterolateral region of the right atrium [Figure 2], formed by the junction of the sinus venous and primitive right atrium. Besides mimicking right atrial mass, crista terminalis is an important anatomic structure responsible for paroxysmal atrial fibrillation and atrial flutter by initiating ectopic atrial beats.[4]

Ventricular diverticula

The incidence of congenital ventricular diverticula was reported to be as high as 0.4% in an autopsy series of patients with cardiac diseases.[5] The diverticula ranged from 0.2 to 1.4 cm in maximum dimension. The diverticula showed contractile properties, appearing smaller in systolic phases
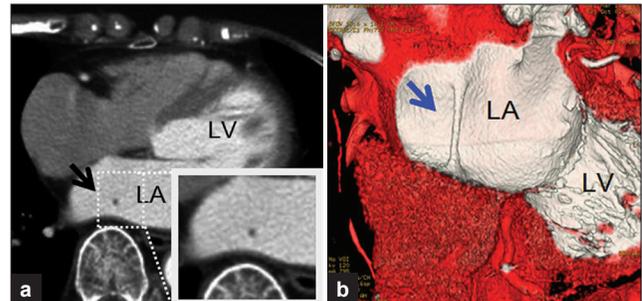

Figure 1: (a) 47-year-old woman with suspected coronary artery disease. Axial CT image shows spotty low density area in the left atrium. (b) 3D endocardial view shows membrane.
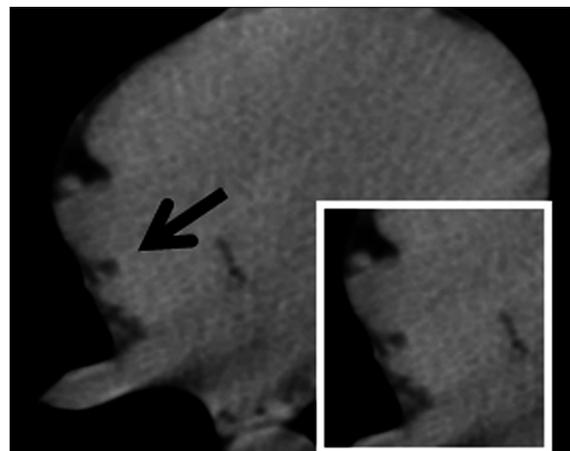

Figure 2: 42-year-old man with no symptom. Axial CT image shows spotty low density area on the right wall in the right atrium.

than in diastolic phases, which indicated that the tissue surrounding the diverticula was primarily muscle tissue.[6]

On the other hand, ventricular septal aneurysms show a paradoxical expansion in systole. Diverticula can be seen in both ventricles [Figures 3 and 4].[7]

Ventricular septal aneurysm

Ventricular septal aneurysms are defined by bowing of the septum 10–15 mm to either side. The size of the base of the aneurysm is important and must be more than 15 mm but not involve the entire septum.[8]

The prevalence of cardiac septal aneurysms is 0.2–3% in the general population. Myocardium surrounds the contrast media indicating aneurysm [Figures 5a and 5b], while ventricular septal defect [Figure 6] shows left to right contrast shunt without muscle wall.

Patent foramen ovale

A patent foramen ovale (PFO) is a persistent valvular-like connection between the left and right atrium [Figures 7a and 7b]. During the first months of life, adhesions between the septum primum and secundum form, closing this embryological connection. In approximately 25% of the general population, this connection persists into adult life, resulting in a potential right-to-left shunt.[9] In some patients, the PFO may be the pathway through which thrombotic emboli, air emboli, desaturated blood are shunted into the left-sided cardiac chambers. ECG-gated CCTA performed with a 64-MDCT scanner can be used to reliably detect and evaluate size and shunt grade of the PFO.[10,11]

Williamson et al described the CT criteria for diagnosing PFO. The criteria are:
1. Presence of a distinct flap in the left atrium at the expected location of the septum primum.
2. Presence of a continuous column of contrast material between the septum primum and septum secundum.
3. Presence of a jet of contrast material from the column into the right atrium.

Using above described three CT criteria results in 100% specificity for the diagnosis of PFO.[10]

Left appendage / Left atrial thrombus

Rhythm abnormalities, such as atrial fibrillation, and various cardiac chamber and valvular abnormalities have been associated with the formation of left atrial appendage thrombi.[12] An intracardiac thrombus is depicted as a
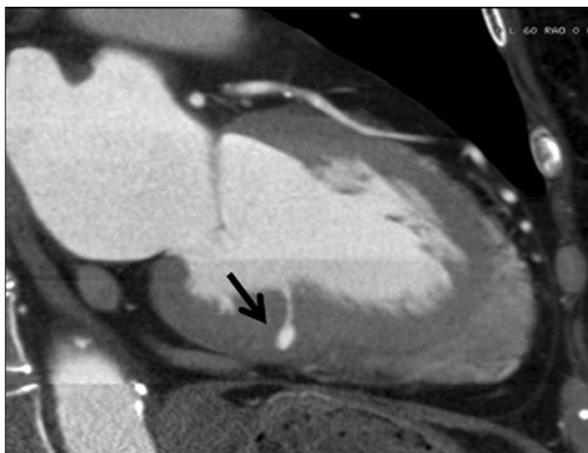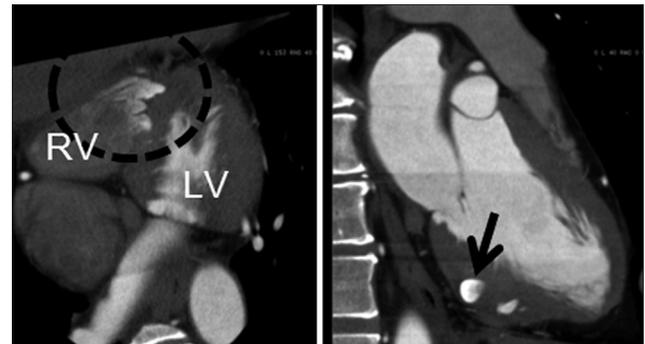

Figure 4: 53-year-old man with angina. MPR images show protrusion of the contrast media to the septum, representing the right ventricular diverticulum.

Figure 3: 70-year-old woman with angina. MPR image shows long-axis view of diverticulum in diastolic phase.
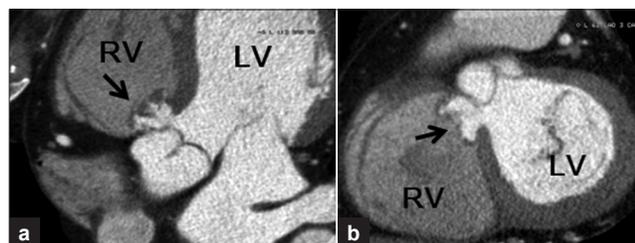

Figure 5: 81-year-old man with suspected coronary artery disease, (a) Four chamber view, (b) Short axis view of the ventricles. Myocardium surrounds the contrast media protruding toward right ventricle (RV) indicates aneurysm.
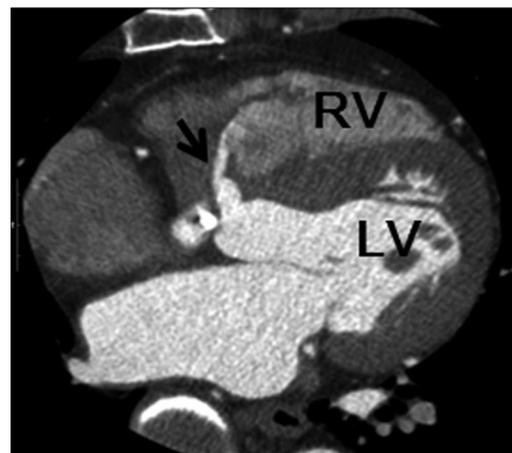

Figure 6: 62-year-old man with known ventricular septal defect. Ventricular septal defect shows left ventricle (LV) to right ventricle (RV) contrast shunt (arrow) without muscle wall.

filling defect on CCTA [Figures 8a and 8b]. The pseudo-filling defects seen on CT images represent incomplete mixing of CT contrast material and blood [Figure 9a and 9b]. Two-phase cardiac CT angiography can be used to differentiate thrombus from circulatory stasis, which may cause a pseudo-filling defect on early-phase CT images.[13]

Coronary artery related, but non stenotic findings

It is also important to pay attention to assess variants of coronary arteries in a CCTA study.
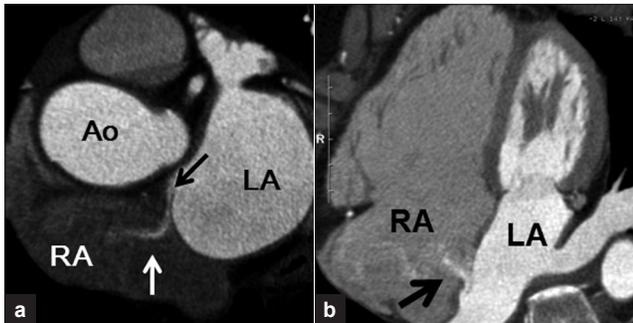

Figure 7: 49-year-old woman with angina pectoris. (a) Coronal oblique view. Coronal oblique CT image shows characteristic findings of patent foramen ovale (PFO) with left atrial flap (arrow), continuous contrast column connecting the left (LA) and right atria (RA), (b) Four chamber view. A left-to-right PFO shunt is visible with contrast media jet (arrow) through foramen ovale.
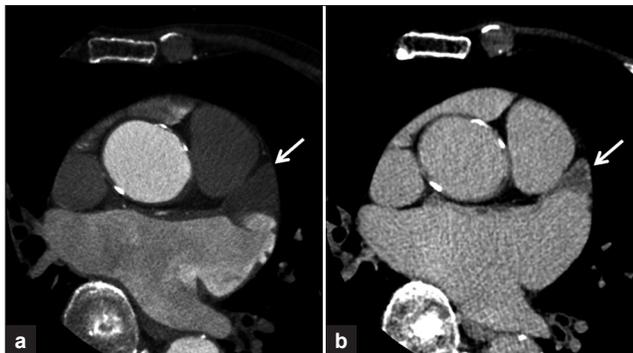

Figure 8: 69-year old man with chronic atrial fibrillation showed consistent filling defect in LAA, indicating LAA thrombus (a) Early-phase CT image shows triangular filling defect (arrow) in LAA, (b) Late-phase CT angiographic image shows no filling defects in LAA.
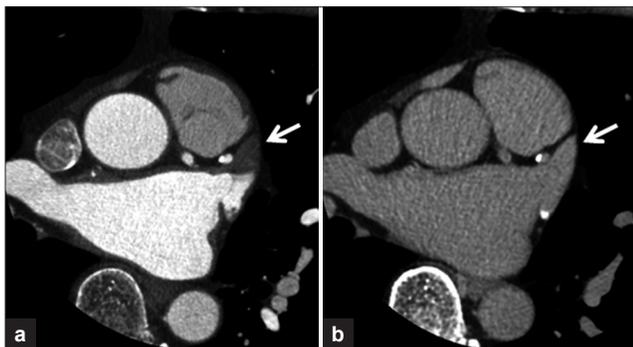

Figure 9: Pseudo-filling defect: 51-year-old man with chronic atrial fibrillation (a) Early-phase CT image shows triangular filling defect (arrow) in LAA, b) Late-phase CT angiographic image shows no filling defects in LAA.

Anomalies of the coronary arteries may be found incidentally in 0.3%–1% of healthy individuals.[14]

Kim et al classified the coronary artery anomalies into anomalies of origin, course, and termination.[15]

Anomalous origin

High take off: "High take off" refers to the origin of either the RCA [Figure 10a] or the LCA at a point above the junctional zone between its sinus and the tubular part of the ascending aorta.[15]

Multiple ostia: Separate ostia (multiple ostia) of the LCA and LCx artery may occur in a small percentage (0.41%) of individuals with otherwise normal anatomy [Figure 11].[16]

Single coronary artery: In the anomalous situation of a single coronary artery, only one coronary artery arises with a single ostium from the aortic trunk. This is an extremely rare congenital anomaly that is seen in only 0.0024%–0.044% of the population.[17]

Anomalous origin and course

In terms of clinical importance, recognized hemodynamically significant coronary artery anomalies are anomalous origins arising from the pulmonary artery; inter-arterial courses that indicate malignancy between the aorta and pulmonary artery [Figures 10a–10c]. Hemodynamically insignificant or courses that indicate benignity of the coronary arteries are retroaortic, prepulmonic, and subpulmonic locations.[15]

Myocardial bridging

Myocardial bridging is a congenital anomaly that results in abnormal positioning of the coronary artery and most commonly occurs in the mid LAD [Figure 12]. Jacobs et al reported the prevalence of 10.4% on CCTA.[18,19] Myocardial bridging is generally regarded to be a benign condition; however, myocardial bridging has been reported in association with myocardial ischemia and infarction, conduction abnormalities, and sudden death.[20] The length and depth of the tunnelled arterial segment have been shown to correlate with the presence of myocardial ischemia and sudden death.[21]

Anomalous termination

Coronary artery fistula

Coronary artery fistula is a condition in which a communication exists between one or two coronary arteries and either a cardiac chamber [Figures 13a and 13b], the coronary sinus, the superior vena cava, or the pulmonary artery [Figures 14a and 14b].[15] Coronary artery

Figure 10: 61-year-old woman with suspected coronary artery disease (a) On VR, the RCA demonstrates a high take-off above the sinotubular junction, (b) On axial CT image, the RCA (arrow) shows inter-arterial course between aorta (Ao) and pulmonary artery (PA), (c) The stretch view (left) and the trans-axial view (right) show narrowing of the proximal RCA.
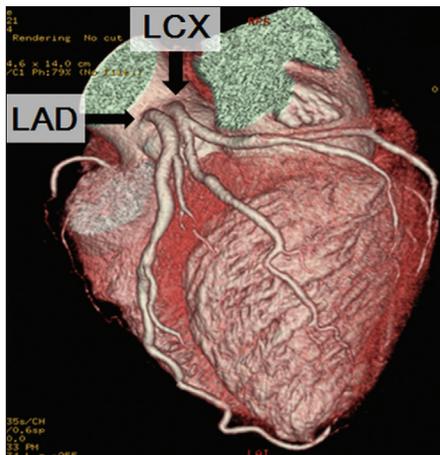

Figure 11: 70-year-old woman with suspected coronary artery disease. VR image shows separate ostia of the LAD and LCX arteries.

Figure 12: 62-year-old woman with suspected coronary artery disease. Curved MPR shows long segment of myocardial bridging of mid LAD.

fistulas are found in 0.1–0.2% of patients who undergo coronary angiography.[22]

Coronary artery aneurysms

Coronary artery aneurysms are defined as segments with a diameter greater than 1.5 times the normal adjacent artery segment and can be classified as fusiform or saccular.[23] Coronary artery aneurysms are small and thick walled and have a low risk of spontaneous rupture.[24] Giant aneurysms are rare and usually congenital in origin. Another cause of giant aneurysms is Kawasaki's disease [Figures 15a and 15b]. Kawasaki's disease is the most frequent cause of coronary aneurysms worldwide, whereas atherosclerotic coronary disease is the most common cause in the United States. The prognosis of coronary artery aneurysm is related to the severity of concomitant obstructive coronary disease.[25]

Other non-coronary findings

Pericardial disease

A spectrum of abnormalities involving pericardium exists, ranging from small defects to total absence of the pericardium. Left-sided absence of the pericardium allows interposition of lung tissue between the aorta and the main segment of the pulmonary artery.[26]

Pericarditis

The causes of pericarditis are numerous, including postsurgical, postradiation, posttraumatic, and postinfectious. However, idiopathic pericarditis is most common. Thickening of the pericardium, often with and without calcifications, can be clearly depicted with CT.[26] Pericardial enhancement may indicate active inflammation [Figure 16]. Pericardial calcifications, bi-atrial enlargement, and pleural effusions are highly suggestive of constrictive pericarditis resulting in impaired ventricular filling.

Abnormal coronary sinus

The unroofed coronary sinus (UCS) is a spectrum of cardiac anomalies in which part or entire common wall between the coronary sinus (CS) and the left atrium is absent [Figures 17a and 17b].[27,28] In a normal case, a normal-sized coronary
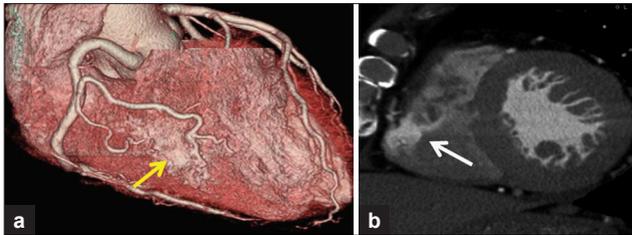
Figure 13: 79-year-old man with suspected coronary artery disease (a) VR image shows tortuous right ventricular branch (arrow) from the RCA, (b) Short axis image of the ventricles shows contrast flow into the right ventricle (arrow: shunt sign).
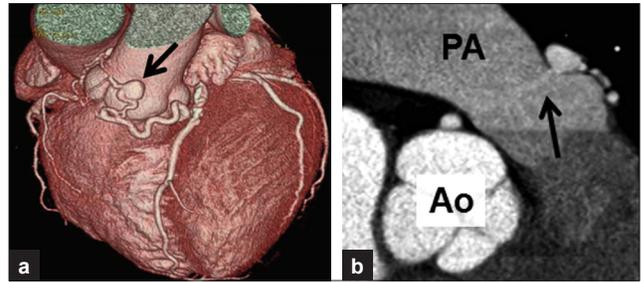

Figure 14: 65-year-old man with diabetes mellitus (a) VR image shows multiple tortuous communicating vessels originating from the proximal LAD artery, (b) The communicating vessels drain into the main pulmonary artery (PA). Note that contrast flow into the PA (arrow: shunt sign).
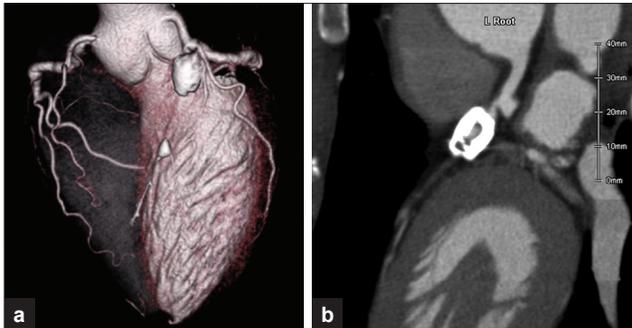

Figure 15: 26-year-old man with Kawasaki disease. VR (Figure 15a) and CPR (Figure 15b) images show partly calcified thrombosed saccular aneurysm in proximal LAD. He has another fusiform aneurysm in the right coronary artery.
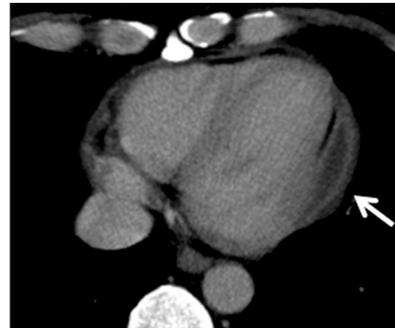

Figure 16: 55-year-old man with fever up. Axial CT shows enhanced pericardial thickening without calcification (arrow). Open biopsy of pericardium revealed tubercular pericarditis.
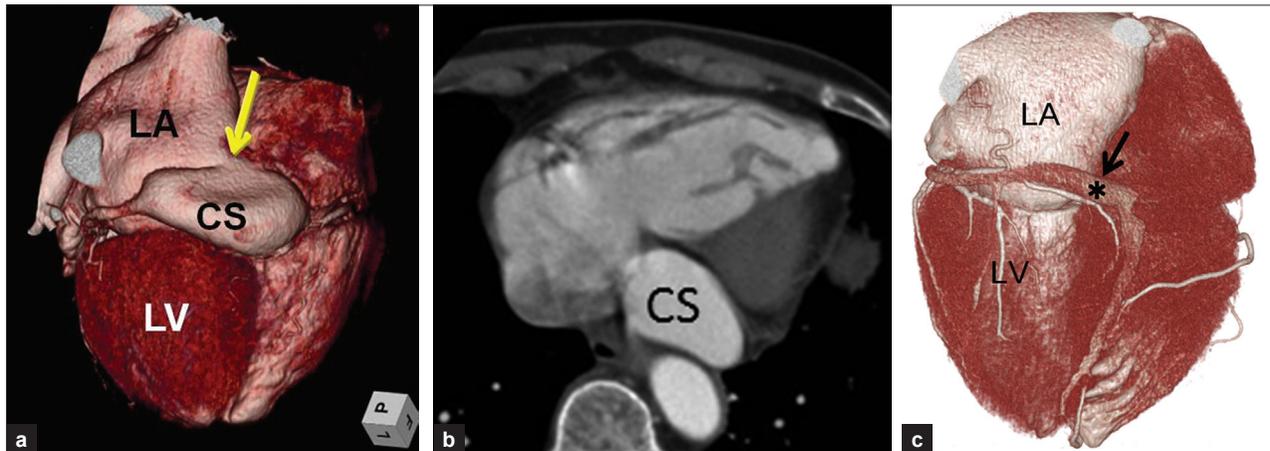

Figure 17: 71-year-old woman with pulmonary-to-systemic blood flow ratio (Qp:Qs) more than 3 (a) The roof of the coronary sinus (CS) was partially absent (arrow). VR image shows dilated, partially unroofed CS and its relationship to the left atrium (LA). Note the distal CS is normal in size, (b) Axial CT image shows the dilated CS, (c) Another 76-year-old man with coronary artery disease. VR shows normal sized CS (arrow).

sinus runs parallel to the posterolateral branch [Figure 17c]. A dilated CS and its relationship with coronary arteries are clearly depicted by CCTA.

CONCLUSION

It is important for the practicing radiologist to be familiar with the imaging appearances of unusual cardiac anomalies during the interpretation of CCTA. Normal anatomical variants can cause confusion to the inexperienced observer and must be recognized to avoid unnecessary intervention. Knowledge of the CCTA appearance of various coronary artery and non-coronary artery cardiac anomalies is essential for accurate diagnosis.

REFERENCES

1. Kirsch J, Araoz PA, Steinberg FB, Fletcher JG, McCollough CH, Williamson EE. Prevalence and significance of incidental extracardiac findings at 64-multidetector coronary CTA. J Thorac Imaging 2007;22:330-4.
2. Thakrar A, Shapiro MD, Jassal DS, Neilan TG, King ME, Abbara S. Cor triatriatum: The utility of cardiovascular imaging. Can J Cardiol 2007;23:143-5.
3. Ferreira F, Ferreira AM, Lopes RS, Ferreira J, Correia Mda G, Gil VM.

Asymptomatic cor triatriatum in an adult--diagnosis by cardiac CT angiography. Rev Port Cardiol 2011;30:363-6.

4. Akcay M, Bilen ES, Bilge M, Durmaz T, Kurt M. Prominent crista terminalis: As an anatomic structure leading to atrial arrhythmias and mimicking right atrial mass. J Am Soc Echocardiogr 2007;20:197.e9-10.

5. Ichikawa K, Makino K, Futagami Y, Fujioka H, Ito M, Hamada M, et al. Isolated congenital left ventricular diverticulum in an adult. A case report. Angiology 1994;45:743-7.

6. Srichai MB, Hecht EM, Kim DC, Jacobs JE. Ventricular diverticula on cardiac CT: More common than previously thought. AJR Am J Roentgenol 2007;189:204-8.

7. Lee WJ, Chen SJ, Wang TD. Right ventricular diverticula. AJR Am J Roentgenol 2008,190:W170.

8. Dodd JD, Aquino SL, Holmvang G, Cury RC, Hoffmann U, Brady TJ, et al. Cardiac septal aneurysm mimicking pseudomass: Appearance on ECG-gated cardiac MRI and MDCT. AJR Am J Roentgenol 2007;188:550-3.

9. Beacock DJ, Watt VB, Oakley GD, Al Mohammad A. Paradoxical embolism with a patent foramen ovale and atrial septal aneurysm. Eur J Echocardiogr 2006;7:171-4.

10. Williamson EE, Kirsch J, Araoz PA, Edmister WB, Borgeson DD, Glockner JF, et al. ECG-gated cardiac CT angiography using 64-MDCT for detection of patent foramen ovale. AJR Am J Roentgenol 2008;190:929-33.

11. Saremi F, Channual S, Raney A, Gurudevan SV, Narula J, Fowler S, et al. Imaging of patent foramen ovale with 64-section multidetector CT. Radiology 2008;249:483-92.

12. Hart RG, Pearce LA, Rothbart RM, McAnulty JH, Asinger RW, Halperin JL. Stroke with intermittent atrial fibrillation: Incidence and predictors during aspirin therapy. Stroke Prevention in Atrial Fibrillation Investigators. J Am Coll Cardiol 2000;35:183-7.

13. Hur J, Kim YJ, Lee HJ, Ha JW, Heo JH, Choi EY, et al. Left atrial appendage thrombi in stroke patients: Detection with two-phase cardiac CT angiography versus transesophageal echocardiography. Radiology 2009;251:683-90.

14. Angelini P, Velasco JA, Flamm S. Coronary anomalies: Incidence, pathophysiology, and clinical relevance. Circulation 2002;105:2449-54.

15. Kim SY, Seo JB, Do KH, Heo JN, Lee JS, Song JW, et al. Coronary artery anomalies: Classification and ECG-gated multi-detector row CT findings with angiographic correlation. Radiographics 2006;26:317-33.

16. Danias PG, Stuber M, McConnell MV, Manning WJ. The diagnosis of congenital coronary anomalies with magnetic resonance imaging. Coron Artery Dis 2001;12:621-6.

17. Desmet W, Vanhaecke J, Vrolix M, Van de Werf F, Piessens J, Willems J, et al. Isolated single coronary artery: A review of 50,000 consecutive coronary angiographies. Eur Heart J 1992;13:1637-40.

18. Jacobs JE, Bod J, Kim DC, Hecht EM, Srichai MB. Myocardial bridging: Evaluation using single- and dual-source multidetector cardiac computed tomographic angiography. J Comput Assist Tomogr 2008;32:242-6.

19. Dodd JD, Ferencik M, Liberthson RR, Nieman K, Brady TJ, Hoffmann U, et al. Evaluation of efficacy of 64-slice multidetector computed tomography in patients with congenital coronary fistulas. J Comput Assist Tomogr 2008;32:265-70.

20. de Winter RJ, Kok WE, Piek JJ. Coronary atherosclerosis within a myocardial bridge, not a benign condition. Heart 1998;80:91-3.

21. Goitein O, Lacomis JM. Myocardial bridging: Noninvasive diagnosis with multidetector CT. J Comput Assist Tomogr 2005;29:238-40.

22. Said SA, el Gamal MI, van der Werf T. Coronary arteriovenous fistulas: Collective review and management of six new cases-changing etiology, presentation, and treatment strategy. Clin Cardiol 1997;20:748-52.

23. Robinson FC. Aneurysms of the coronary arteries. Am Heart J 1985;109:129-35.

24. Daoud AS, Pankin D, Tulgan H, Florentin RA. Aneurysms of the coronary artery. Report of ten cases and review of literature. Am J Cardiol 1963;11:228-37.

25. Murthy PA, Mohammed TL, Read K, Gilkeson RC, White CS. MDCT of coronary artery aneurysms. AJR Am J Roentgenol 2005;184:19-20.

26. Oyama N, Komuro K, Nambu T, Manning WJ, Miyasaka K. Computed tomography and magnetic resonance imaging of the pericardium: Anatomy and pathology. Magn Reson Med Sci 2004;3:145-52.

27. Raghib G, Ruttenberg HD, Anderson RC, Amplatz K, Adams P Jr., Edwards JE, et al. Termination of Left Superior Vena Cava in Left Atrium, Atrial Septal Defect, and Absence of Coronary Sinus; a Developmental Complex. Circulation 1965;31:906-18.

28. Oyama N, Ooka T, Sasaki T, Kubota S, Onodera Y, Matsui Y, et al. Volume-rendering and endocardial views of partially unroofed coronary sinus with 64-slice multidetector CT. J Cardiovasc Comput Tomogr 2009;3:346-7.