Prevalence and Anatomic Characteristics of Single Coronary Artery Diagnosed by Computed Tomography Angiography

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Single coronary artery (SCA) is a rare congenital anomaly. We assessed the prevalence and anatomic characteristics of SCA diagnosed with coronary computed tomography angiography and compared the dimensions of the proximal SCA trunk with a reference group of 199 subjects with normal coronary arteries. We screened 30,230 patients who underwent coronary computed tomography angiography from 2008 to 2018 to identify 17 with SCA (age 55 ± 19.0 years, 8 men [47%]). The prevalence of SCA was 0.056%. SCA originated from the right sinus of Valsalva in 11 patients (65%) and from the left sinus of Valsalva in 6 subjects. According to Lipton's classification, the 17 SCAs were L1 (n = 5, 29%), L2-A (n = 1, 6%), R2-A (n = 2, 12%), R2-B (n = 6, 35%), R2-P (n = 2, 12%), and R3 (n = 1, 6%). (Lipton's classification consists of 3 groups and the division is based on the site of origin of SCA ["R" − right, "L" − left sinus of Valsalva] and its anatomical course relating to the ascending aorta and pulmonary trunk ["A" - anterior to the pulmonary trunk, "B" − between the aorta and pulmonary trunk, "P" − posterior to the aorta].) As compared with the reference group, SCA patients had shorter proximal trunks (5.0 ± 3.6 mm vs 8.6 ± 4.8 mm, p = 0.0012). The lumen area (LA) and lumen diameter of the proximal trunk in patients with SCA were larger than the LA and lumen diameter of the left main coronary artery from the reference group (49.5 ± 18.0 mm2 vs 21.3 ± 6.5 mm2, p <0.0001, and 7.8 ± 1.6 mm vs 5.1 ± 0.75 mm, p <0.0001, respectively). Moreover, the LA of the proximal SCA trunk was larger than the sum of respective measurement performed in left main coronary artery and proximal right coronary artery segments in the control group (49.5 ± 18.0 mm2 vs 34.0 ± 7.9mm2, p = 0.0001). In conclusion, the incidence of SCA is very low; but this condition is associated with significant enlargement of the proximal vessel segment. **(Am J Cardiol 2019;124:939−946)**

Single coronary artery (SCA) − the presence of only 1 coronary trunk arising from the aorta − is a rare congenital coronary artery anomaly with a prevalence of 0.024% to 0.066%.[1−5] Approximately 20 possible variations of SCA have been described.[6] The most widely used classification of SCA was proposed by Lipton et al.[2] However, due to the low prevalence, clinical and imaging data on this anomaly remain scarce. The aims of our study were to (1) assess the prevalence and anatomic characteristics and clinical correlates of SCA diagnosed with coronary computed tomography angiography (CCTA) and (2) compare the dimensions of the proximal SCA trunk with a reference group of normal coronary arteries free of atherosclerosis.

Methods

From February 2008 to September 2018 there were 30,230 CCTA examinations performed at Institute of Cardiology, Warsaw, Poland. The electronic database of all CCTA reports was manually screened to identify subjects with SCA. A comparison reference group of subjects with absolutely normal atherosclerosis-free coronary arteries has been described previously by Skowronski et al.[7] SCA was defined as the common origin of a single proximal trunk giving rise to both the left and right coronary artery (RCA), supplying the whole myocardium, and arising from either the right or left sinus of Valsalva. Separate origins of both coronaries from the same sinus of Valsalva were not considered as SCA.

All patients with SCA were analyzed according to Lipton's angiographic classification[2] by a radiologist (IM) and cardiologists experienced in cardiovascular imaging (JS and JP). Lipton's classification consists of 3 groups; and the division is based on the site of origin of the SCA ("R" − right, "L" − left sinus of Valsalva) and its anatomic course relating to the ascending aorta and pulmonary trunk ("A" − anterior to the pulmonary trunk, "B" − between the aorta and pulmonary trunk, "P" − posterior to the aorta; Figure 1). In Group 1 the SCA follows the course of a normal right or left coronary

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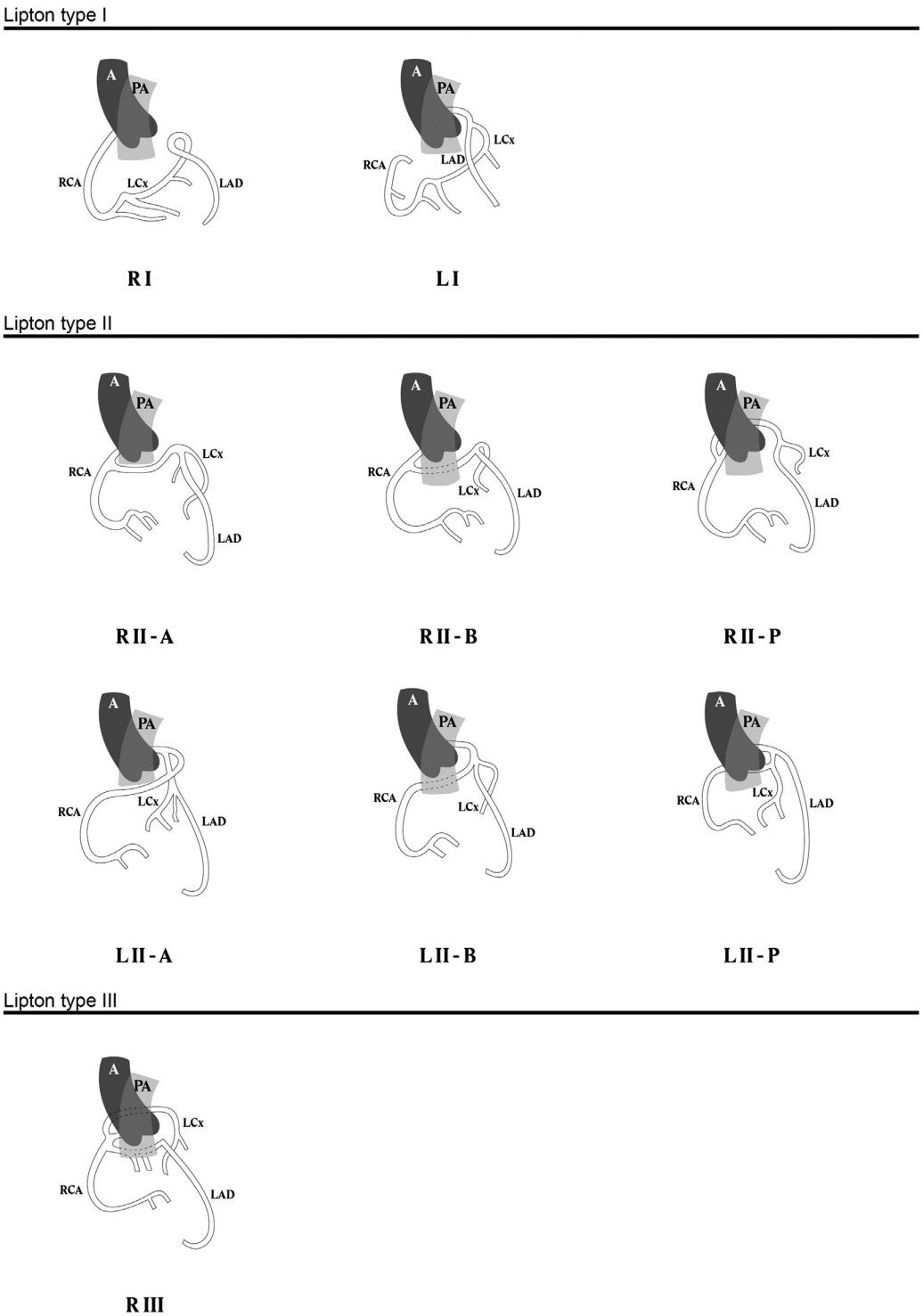

Figure 1. The scheme of Lipton's classification (adapted from Aldana-Sepulveda N et al.[3] A = aorta; LAD = left anterior descending artery; LCx = left circumflex artery; PA = pulmonary artery; RCA = right coronary artery.

artery (R1, L1; Figure 2). In Group 2 the SCA originating from right or left coronary sinus of Valsalva (R2, L2) crosses the base of the heart to approach the vicinity of the anatomically normal contralateral artery (R2-A [Figure 3], R2-B [Figure 4], R2-P [Figure 5], L2-A [Figure 6], L2-B, L2-P). In Group 3 the left anterior descending and left circumflex share a common trunk with RCA, that arises from the right coronary sinus of Valsalva (RIII) [Figure 7].

Due to the complexity of congenital heart defects such as univentricular heart and transposition of the great arteries, 2 patients (12%) were previously analyzed using the modified Leiden Convention[8] – the coding of coronary arterial origin

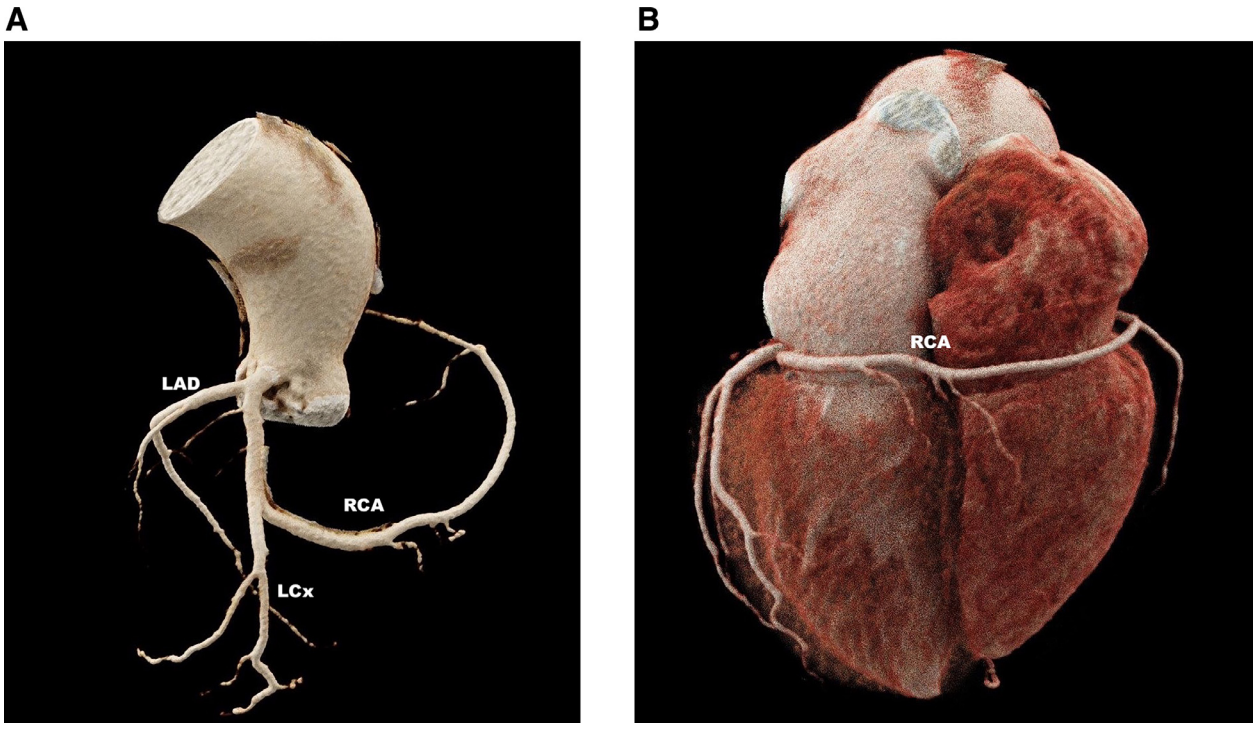

Figure 2. Single coronary artery (SCA) type Lipton L1. Coronary computed tomography, volume-rendered reconstruction *(A)* Lateral view, image shows a SCA originating from the left sinus of Valsalva. *(B)* Posterior view shows the right coronary artery taking-off from the left circumflex coronary artery. LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery.

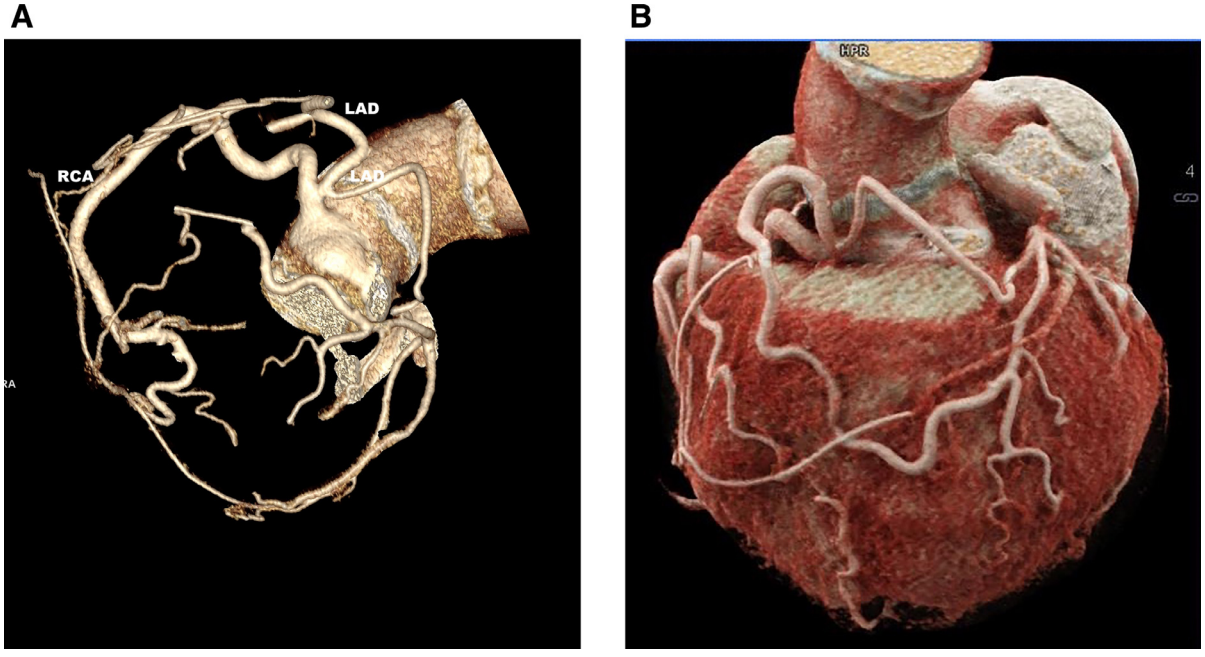

Figure 3. Single coronary artery type Lipton type R2-A. *(A, B)* Coronary computed tomography angiography, volume-rendered reconstructions show the left anterior descending artery taking-off from the right coronary artery. LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery.

and branching in congenital heart disease system – to assess the sinus of Valsalva.

Studies conducted from February 2008 to May 2011 (5,894 patients [19.5%]) were performed with a first-generation dual-source computed tomography (DSCT) scanner (Somatom Definition; Siemens Healthcare, Forchheim, Germany); from June 2011 to May 2015 (12,269 patients [40.5%]) with a use of a second-generation DSCT scanner (Somatom

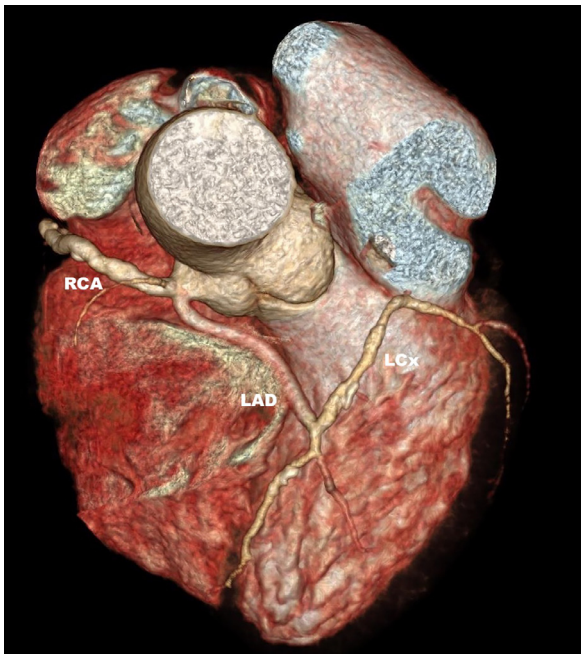

Figure 4. Single coronary artery (SCA) type Lipton type R 2B.
Coronary computed tomography angiography, volume-rendered reconstruction shows a SCA originating from the right sinus of Valsalva. LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery.

Definition Flash); and CCTA from June 2015 to September 2018 (12,067 patients [40%]) were performed using third-generation DSCT scanner (Somatom Force). CT acquisition parameters for first-generation DSCT were as follows: slice collimation 64 × 0.6 mm, a gantry rotation time of 330 ms, tube voltage of 80 to 140 kV, pitch of 0.2 to 0.3. CT acquisition parameters for the second-generation DSCT were slice collimation 128 × 0.6 mm, gantry rotation time 280 ms, tube voltage 100 to 120 kV. CT acquisition parameters for third-generation DSCT were slice collimation 192 × 0.6 mm, gantry rotation time 250 ms, tube voltage 70 to 100 kV, tube current 320 to 500 mAs (depending on the patient body mass). A retrospective or prospective electrocardiographically gated acquisition protocol was used at the operator's discretion. To minimize radiation exposure, electrocardiographically gated tube current modulation was applied in all patients. Scan data were reconstructed routinely in mid- to end-systole and diastole (35% to 45% and 65% to 75% of the RR interval).

Unless contraindicated, an intravenous bolus of metoprolol (sequential 2.5 mg doses) was administered to target a heart rate <65 to 75 beats/min (depending on the generation of the DSCT scanner); and sublingual nitroglycerin (0.8 mg) was given directly before computed tomography. For acquisition of the volume data set, a bolus of 60 to 80 mL iodinated contrast material (Iomeron 400; Bracco Altana Pharma, Konstanz, Germany, Ultravist 370 Bayer Pharma AG) was administered through an antecubital vein at a rate of 4.5 to 6 mL/s (depending on the generation of the DSCT scanner). The computed tomographic protocol was extended to the measurement of a coronary artery calcium score if clinically indicated.

The curved multiplanar reconstructions of all SCA were automatically created with commercially available software (Syngo-Via software, Siemens, Germany). The SCA trunk length was defined as the distance between the SCA ostium and the origin of the first side branch and was measured manually. Within the length of the SCA trunk, the maximal and minimal cross-sectional lumen area (LA) and lumen diameter (LD) were measured. All measurements were performed by the single experienced observer (JS).

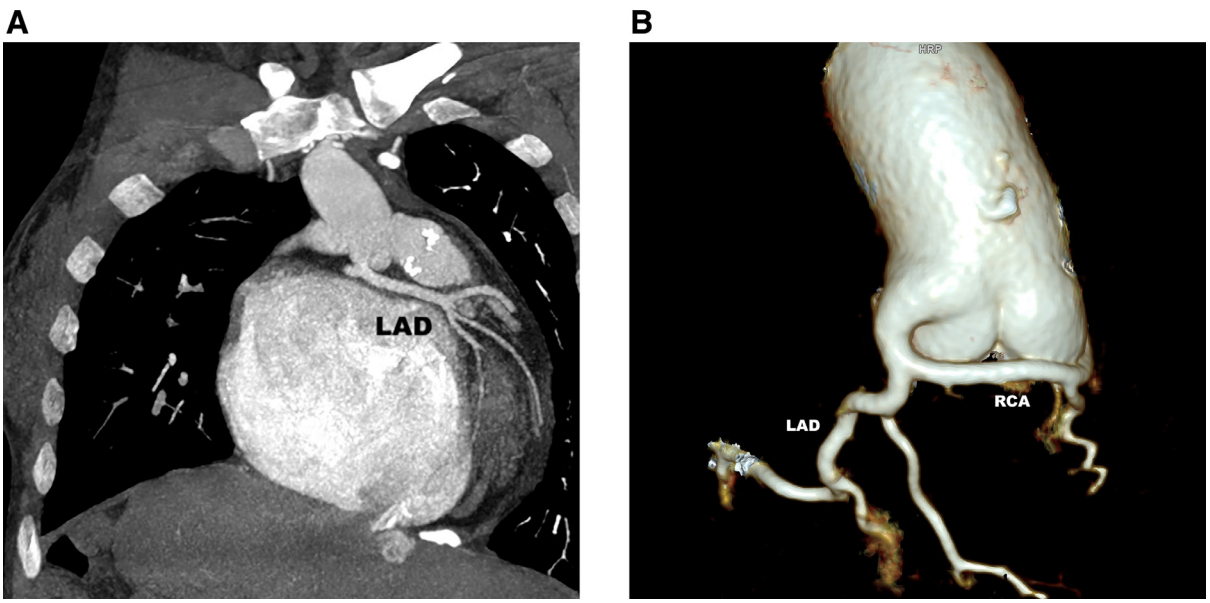

Figure 5. Single coronary artery (SCA) type Lipton R 2P. *(A)* Coronary computed tomography angiography, maximum intensity projection reconstruction, *(B)* volume-rendered reconstruction shows a SCA originating from the right sinus of Valsalva and dividing into the right coronary artery and left anterior descending artery. LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery.

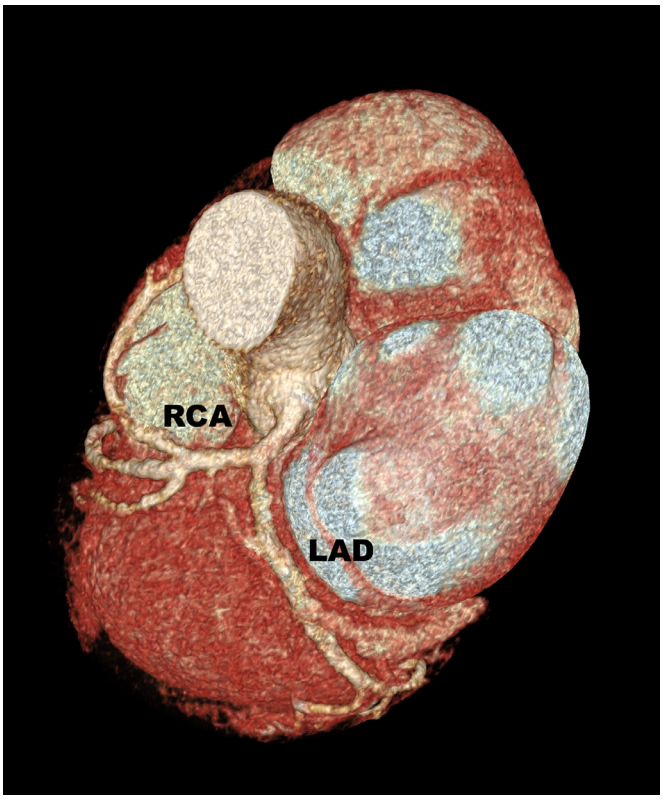

Figure 6. Single coronary artery (SCA) type Lipton type L2-A. Coronary computed tomography angiography, volume-rendered reconstruction shows a SCA originating from the left sinus of Valsalva and dividing into the right coronary artery and left anterior descending artery. LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery.

The measurements performed in the reference group were described previously.[7] Briefly, we measured maximal and minimal LA and LD in the left main coronary artery (LMCA) and in the proximal segment of the RCA.

Data are presented as absolute numbers and/or percentages, or as median followed by interquartile range, where applicable. The Kolmogorov-Smirnov test was used to test for normal distribution. Data with nonparametric distribution were compared using the U-Mann Whitney test. Categorical variables were compared using the chi-squared test. p values below 0.05 were considered significant. All tests were 2-sided. Statistical analysis was prepared using MedCalc 9.3.8.0 Software (MedCalc, Marierkerke, Belgium).

Results

We identified 17 patients with SCA. Thus, the prevalence of SCA in our population was 0.056% (17 of 30,230). The age of patients with SCA was 55 ± 19 years. There were 8 men (47%). The reference group of patients with normal coronary arteries consisted of 199 subjects. The clinical characteristics of the patients with SCA and control subjects have been shown in Table 1. The SCA and control group had similar demographics and clinical characteristics except for a higher prevalence of diabetes mellitus in the SCA group.

Sixteen of 17 patients had other cardiovascular disease (Table 2). Four patients (24%) underwent CCTA because of a congenital heart defect, 9 patients (53%) due to typical angina, 2 (12%) had atypical chest pain, and 2 (12%) underwent CCTA because of a suspicion of coronary artery disease. Overall, 9 of 17 patients (53%) had coronary artery disease and 2 underwent in the past percutaneous coronary intervention of a distal vessel. Only 1 of 9 patients with coronary artery disease (11%) had a significant stenosis (>70%), whereas 8 (89%) had nonsignificant stenosis (<50%). All SCA trunks and ostia were free of atherosclerotic changes. Three of 9 patients (33%) had moderately disseminated atheromatous changes with the plaque burden <50%. Stress tests for ischemia were performed only in 2 subjects (1 cardiac magnetic resonance with perfusion and 1 single-photon emission computed tomography); and both were negative.

In our study, the most common origin of the SCA was from the right sinus of Valsalva (n = 11, 65%), whereas in 6 patients (35%) the origin was from the left sinus of Valsalva. According to Lipton's classification, the 17 SCA were L1 (n = 5, 29%), L2-A (n = 1, 6%), R2-A (n = 2, 12%), R2-B (n = 6, 35%), R2-P (n = 2, 12%), and R3 (n = 1, 6%).

The most serious clinical presentation is usually associated with the SCA course between the ascending aorta and the pulmonary trunk (Lipton type 2 B). In our group, 6 patients had this potentially malignant anatomic variant. Nevertheless, in these 6 patients the clinical presentation did not differ from the rest of the group (Table 2).

Congenital heart defects accompanying the SCA included univentricular heart, transposition of great arteries, pulmonary stenosis, persistent ductus arteriosus, ventricular septal defect, and bicuspid aortic valve (Table 2).

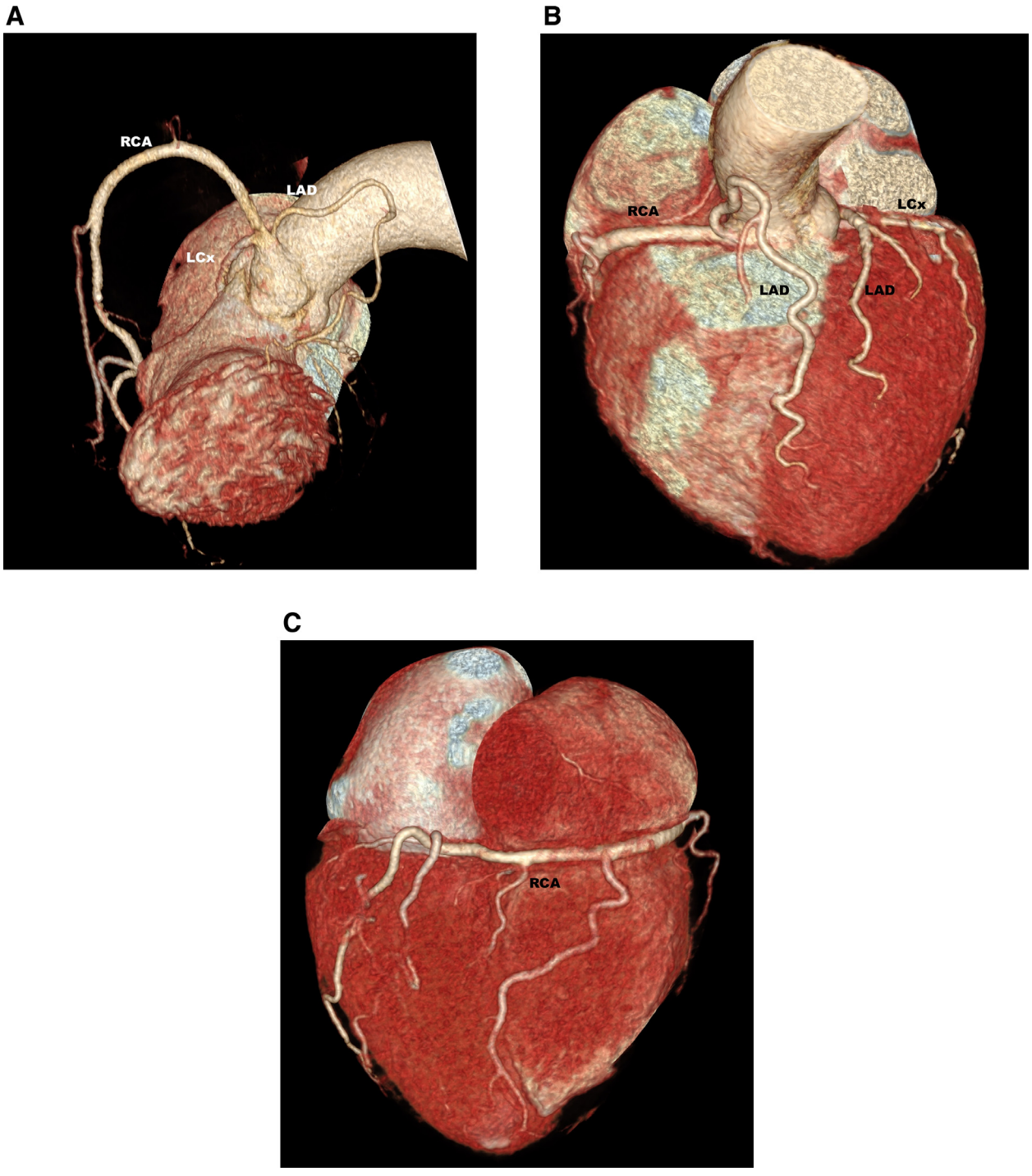

Figure 7. Single coronary artery type Lipton type R3. *(A* to *C)* Coronary computed tomography angiography, volume-rendered reconstructions, oblique, anterior and posterior views, respectively. LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery.

The mean length of the SCA trunk was 5.0 ± 3.6 mm. Thirteen of 17 trunks (76%) were longer than 2 mm; and the LD and LAs of the SCA were 7.8 ± 1.6 mm and 49.5 ± 18.0 mm2, respectively. As compared with a reference group of 199 patients with normal coronaries, SCA patients had shorter proximal trunks (5.0 ± 3.6 mm vs 8.6 ± 4.8 mm, p = 0.0012). The LA and LD of the proximal trunk in patients with SCA were larger than the LA and LD of the LMCA from the reference group (49.5 ± 18.0 mm2 vs 21.3 ± 6.5 mm2, p <0.0001, and 7.8 ± 1.6 mm vs 5.1 ± 0.75 mm, p <0.0001, respectively). What is more, the LA and LD of the proximal trunk in patients with SCA and without CAD were larger than the LA and LD of the LMCA from the reference group (52.3 ± 19.3 mm2 vs 21.3 ± 6.5 mm2, p <0.0001, and 8.1 ± 1.5 mm vs 5.1 ± 0.8 mm, p <0.0001, respectively). Moreover, the LA of the proximal SCA trunk was larger than the sum of the respective measurement performed in LMCA and proximal RCA

Table 1
The comparison of the patients with single coronary artery versus control group of subjects without coronary atherosclerosis

Variable	Subjects with single coronary artery (n = 17)	Control group (n = 199)	p values
Age, years ± mean	55 ± 19	50 ± 13	0.1
Male	8	76	0.48
Diabetes Mellitus	6	16	0.0001
Hypertension	11	105	0.34
Previous or active smokers	1	22	0.54
Hyperlipidemia	7	111	0.23

Diabetes mellitus was diagnosed if the patient was on insulin or oral antidiabetic medication. Hypertension was defined as blood pressure ≥140/90 mm Hg on several readings or if the patient was on antihypertensive medication. Hyperlipidemia was defined as LDL level ≥115 mg/dL or if the patient was on lipid lowering therapy.

segments in patients from the control group. (LA 49.5 ± 18.0 mm2 vs 34.0 ± 7.9 mm2, p = 0.0001).

Follow-up was available for 15 of 17 patients (median duration of 25 [2 to 51] months). One patient with complex congenital heart defect died due to progression of heart failure. Two patients were operated on for aortic stenosis, 1 patient for severe tricuspid regurgitation, and 1 for both mitral and tricuspid regurgitation. The remaining patients were managed conservatively. No patient underwent coronary artery surgery.

Table 2
Classification of the single coronary artery type and its association with concomitant heart diseases

Patient number	Age (years)	Sinus of Valsalva	Lipton's classification	Concomitant heart diseases
				Patients with noncongenital HD
1	40	L	L1	CAD, HF
2	63	L	L1	AF, TR
3	69	R	R2-A	AS
4	59	R	R2-B	CAD
5	46	R	R2-B	AR, HF, MR, TR
6	67	L	L1	CAD
7	69	L	L2-A	AS, CAD, HF
8	62	R	R2-B	-
9	69	R	R2-B	CAD
10	79	R	R2-P	AS, CAD
11	64	R	R3	CAD
12	81	R	R2-B	CAD
				Patients with congenital HD
13	51	R	R2-B	ASD, CAD
14	43	L	L1	AS, BAV
15	28	R	R2-P	PS, TGA, UVH
16	15	L	L1	AS, BAV, HF, MR, PH, TR
17	21	R	R2-A	PDA, PS, TGA, UVH, VSD

AF = atrial fibrillation; AR = aortic regurgitation; AS = aortic stenosis; ASD = atrial septal defect; BAV = bicuspid aortic valve; CAD = coronary artery disease; HD = heart disease; HF = heart failure; L = left; MR = mitral regurgitation; PDA = persistent ductus arteriosus; PH = pulmonary hypertension; PS = pulmonary stenosis; R = right; TGA = transposition of great arteries; TR = tricuspid regurgitation; UVH = univentricular heart; VSD = ventricular septal defect.

Discussion

The major findings of our study were as follows. The prevalence of SCA diagnosed with CCTA was very low. The proximal trunk (before the origin of the first side branch) of the SCA was enlarged as compared with normal subjects whether compared with the LMCA or to the sum of the LMCA and RCA.

Although rare, SCA prevalence data have differed according to analytic methods. In angiographic studies, the prevalence ranged from 0.024% to 0.066%.[1–5] The prevalence described in the tomographic study by Graidis et al[9] was 0.12% (3 of 2,572) whereas in the autopsy study of Patel et al[10] the prevalence was 0.48% (1 of 210). Noteworthy, only about 45 cases of SCA had been discovered and described at autopsy until 1950.[11] The SCA prevalence in our study was 0.056%, in line with previous big angiographic studies. Mandal et al reported 22 cases (14 children and 8 adults) of SCA based on CCTA or/and MRI.[12] The systematic review by Kumar et al[13] identified 713 adults with SCA diagnosed with the use of invasive coronary angiography (ICA) [n = 391 (55%)], CCTA [n = 52 (7%)], ICA + CCTA [n = 191 (27%)], at autopsy [n = 54 (7%)], ICA + MRI or CCTA + MRI or ICA + CCTA + MRI [n = 25 (4%)].

The most widely used classification for SCA was proposed by Lipton et al. In our study, the most common SCA anatomy fulfilled their criteria of Group 2, similar to Lipton's study.

SCA has been associated with other congenital anomalies in 18% to 40% of the reported cases (i.e., tetralogy of Fallot, bicuspid aortic valve, aortic arch atresia, transposition of great arteries).[1,3,14–17] In our study, additional congenital heart abnormalities were found in 30% of patients, in line with previous reports and the modified Leiden Convention. Of note, aortic stenosis was relatively common in our study group (5 of 17 patients, and 2 patients had bicuspid aortic valve).

To our knowledge, our study is the first report showing that the common SCA trunk is larger as compared either with the normal LMCA or even to the sum of luminal dimensions of LMCA and RCA in an atherosclerosis-free reference population. Conversely, Lipton's angiography-based study showed that LA within the SCA was smaller than in normal right and left main coronary arteries, perhaps related to the different imaging methods (CCTA vs angiography) and the 4 times larger size of our quantitative study population (17 vs 4 patients). We assume that according to the medical application of Poiseuille's equation which states that flow rate is proportional to the radius to the fourth power, the bigger radius of SCA provides increased blood flow in the vessel in comparison with the anatomically normal coronary arteries. Compensatory enlargement of the SCA trunk might also have important clinical implications. Namely, percutaneous coronary intervention in a patient with SCA should be performed under intravascular imaging guidance, as possible complications might be even more devastating than percutaneous coronary intervention of LMCA.

Although SCA is usually asymptomatic, a patient may experience chest pain, dyspnea, palpitations, syncope, sudden death, ventricular fibrillation, or myocardial infarction, especially after exercise.[16] According to Yamanaka et al, anomalous coronary artery origins can be divided into 2 groups: benign anomalies and potentially malignant anomalies that include ectopic coronary origin from the pulmonary artery, coronary origin from the opposite sinus of Valsalva, and SCA.[4] Clinical manifestations might also be caused by underlying coronary atherosclerosis. Of note, 53% of patients with SCA in our study had CAD. According to some authors SCA does not increase the risk of coronary atherosclerosis, but Porto et al were concerned that an acute-angle take-off malformation may increase the risk of atherosclerosis in these patients.[2,18,19] Nevertheless, we did not find the correlation in our study group. The clinical symptoms of angina might be caused not only by the atherosclerotic disease, but also by the anomalous pattern, acute angle take-off, and/or narrowed slit-like orifice as well as interatrial/intramural course[8,11,20,21] although there were no apparent correlations between the type of anomalous courses and the symptoms of angina.[2] The intraseptal course was a high-risk factor of ischemic myocardial injuries.[12,22,23] Nevertheless, in our study this potentially malignant anatomical variant (Lipton type 2B) did not impact adversely clinical presentation.

Finally, a coronary artery anomaly such as SCA should be suspected in any young patient with exertional syncope, exercise-induced arrhythmias, myocardial infarction, or cardiac arrest.[8]

There were some limitations. The reference group represented patients without atherosclerosis whereas disease was present in 53% of SCA patients. One cannot exclude the influence of distal atherosclerotic lesions on the proximal trunk diameter, but we did not observe obvious atherosclerotic lesions in the SCA trunk. However, the differences in the proximal segment dimension were so large that, in our opinion, cannot be attributed only to remodeling. Diabetes mellitus was found more often in the SCA group, but otherwise both SCA and control patients were similar.

In conclusion, the prevalence of SCA was very low, but this condition was associated with significant enlargement of the proximal vessel segment which should be considered by an interventional dealing with this rare subset of patients.

Disclosures

The authors have no conflicts of interest to disclose.

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