Back to basics: Coronary arteries anatomical variants and anomalies - A pictorial review using coronary CT angiography

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Learning objectives

- To describe the coronary arteries (CAs) normal and variant anatomy.
- To illustrate the CAs anomalies and discuss their clinical implications.

Background

Normal and variant anatomic findings are found in >1% of the general population, whereas anomalous features are defined to be present in <1% of the same population [1]. CA anomalies are the result of disorders that evolve during the third week of fetal development [2], through aberrations in embryonic cell lineage commitment, peripheral conduction system, diversification, cell migration, transition and cell differentiation, vasculogenesis, neural crest cells, alterations in growth factors and genes [3,4]. CA anomalies should be suspected in any young patient with symptoms of angina pectoris, near fatal arrhythmias, or recurrent syncope [5]. According to the Sudden Death Committee of the American Heart Association, up to 19% of sudden death in athletes could be related to CA anomalies [6]. Moreover, a wrong interpretation of a coronary variant or anomaly might cause technical difficulties during interventional procedures, lead to clinical misdiagnosis or major complications during graft surgery [7].

Coronary computed tomography angiography has entered the daily clinical practice in many institutions worldwide, after becoming feasible as temporal and spatial resolution improved with the development of electrocardiographically gated multi-detector row computed tomography [5,8]. It enables rapid coverage of the coronary territory in a single breath hold and improved longitudinal resolution, approaching the ideal of "isotropic" spatial resolution [5]. Furthermore, it offers many advantages over invasive coronary angiography by providing an accurate and comprehensive evaluation of the CAs due to its excellent spatial and temporal resolution, 3-dimensional depiction, with the possibility of performing a flexible post-processing - multiplanar reformation (MPR), three-dimensional (3D) volume-rendering (VR), and maximum intensity projection (MIP) techniques [7,9,10]- and a large field of view that provides reference landmarks for accurate anatomical assessment of coronary anomalies, with the benefit of noninvasiveness and minimal risk to patients [9].

The radiologist should have knowledge of the normal CAs anatomy and variations, and understand the different types of anomalies and their implications in order to provide a correct diagnosis.
The four main CAs - the right coronary artery (RCA), the left main coronary artery (LMA), the left anterior descending (LAD) artery, and the left circumflex (LCx) artery - and their branches have a specific course along the epicardial surface (Table 1) [11-13]. On an axial cross-sectional image through the aortic root, the ostium of the RCA arises in the right (anterior) sinus of Valsalva, at approximately the 10- to 12-o'clock position; the LMA arises in the left (posterior) sinus of Valsalva, at the 3- to 5-o'clock position. There is a third aortic sinus, referred to as the noncoronary sinus [3]. According to Angelini [14], the normal features of human CA anatomy include: 2-4 ostia, with location at upper midsection of the right and left anterior sinuses, with an orientation at 45° and 90° off the aortic wall, a common trunk only on the left, extramural, subepicardial course direct from the ostium to destination - the capillary bed.

Normal variants of CAs anatomy are benign entities with limited clinical significance [9]. However, CAs anomalies range from benign entities to those associated with hemodynamically significant abnormalities, leading to malignant arrhythmias, syncope, angina pectoris, myocardial infarction and sudden cardiac death [9]. The CAs anomalies are classically divided into anomalies of origin and course (absent LMA, anomalous ostium outside of the aortic sinuses, and anomalous ostium at an improper sinus), of intrinsic coronary anatomy (congenital ostial stenosis or atresia, coronary ectasia and aneurysm, myocardial bridging, duplicated arteries, subendocardial coronary course, coronary crossing), and termination (CA fistulas) (Table 2)[1,15,16]. Malignant CAs anomalies are: an abnormal location of coronary ostium at improper sinus coursing between the aorta and pulmonary artery, an anomalous origin of a CA from the pulmonary artery, a single CA with an interarterial course, occasional myocardial bridge, coronary ostial atresia and congenital CA fistula [5,15,16].

The artery which crosses the crux of the heart and gives off the posterior descending branches (the posterior descending artery and posterior left ventricular branch) is considered to be the dominant CA. Approximately 80-85% of individuals have a right coronary circulation dominance, ~8% have left coronary dominance (Fig. 1), while 7-8% have codominance (balanced dominance), meaning the posterior descending artery arising from the RCA and the posterior left ventricular branch arising from the LCx [11-13]. The LMA, LCx, and LAD have greater cross-sectional areas in left dominant and co-dominant hearts than in right dominant hearts [17].

Anomalies of origination and course

Absent left main trunk/multiple ostia
The absence of the LMA is characterized by a separate origin of the LAD and LCx from the left coronary sinus (Fig. 2), which is not a clinically significant anomaly [6,18].

**Fig. 2:** Volume-rendered CT image of heart in left anterior oblique projection shows absent LMA and separate origin (arrow) of the LAD and LCx

**References:** Radiology Department, Guy’s and St Thomas’ Hospital, London, UK

**Significance:**

- Catheter insertion in coronary angiography is often very difficult [6].
- If not recognized, it may be misinterpreted as occlusion [18].

- Risk of compression/occlusion of the LCx by a percutaneous mitral annuloplasty device [19].

- They may allow alternate collateral sources in patients with proximal coronary artery disease [13].

Other multiple ostia, refers to an aberrant conus artery arising separately from the RCA [13]. (Fig. 3)

**Significance:**

- Presence of multiple ostia in the right sinus Valsalva may lead to surgical problems in cases requiring right ventriculotomy for ventricular septal defect or pulmonary stenosis [10,20].

**Anomalous location of coronary ostium within aortic root or near proper aortic sinus of Valsalva**

The origin of a CA within the proximal 1 cm segment of the ascending aorta might be considered as a normal variant [20]. A **high takeoff** CA is considered as an anomaly and refers to an ostium located approximately 10 mm or more above the sinotubular junction, while a low CA origin refers to an ostium located at the lower end of the aortic sinus. A commissural ostium refers to a CA ostium located within 5 mm of the aortic valve apposition at the aortic annulus [3].

**Significance:**

- Awareness of the presence of high takeoff abnormalities is important because they may cause problems in interventional procedures [18].

**Anomalous origin of a CA from the main pulmonary artery**

The anomalous origin of a CA from the pulmonary artery is one of the most serious and rare CA anomalies, with the LMA originating from the pulmonary artery (Bland-White-Garland Syndrome) being the most common form [18].

**Significance:**

- Anomalous origin of the LMA from the pulmonary artery often results in death during infancy, with few patients surviving to adulthood [21].

- Anomalous origin of the RCA from the pulmonary artery is a relatively benign anomaly and may rarely lead to sudden death in case of right dominant coronary circulation [21].
A higher contrast medium in the RCA than in the pulmonary artery and identical to the enhancement in the aorta and the left heart, suggests collateral filling from the left and reversal flow in the RCA toward the pulmonary artery, causing coronary steal and left-to-right shunt [17,22]. The imaging features include tortuosity of the CA and multiple dilated intercoronary collaterals [21].

**Anomalous CA originating from the opposite sinus of Valsalva**

There are 4 patterns of origination with this anomaly: LMA origination from the right coronary sinus; RCA origination from the left coronary sinus (Fig. 4); LCx or LAD origination from the right coronary sinus; and CA origination from a noncoronary sinus. An anomalous CA originating from the opposite coronary sinus may follow 4 main courses: an *interarterial* course, between the aorta and pulmonary artery at the level of pulmonary valve or right ventricular outflow; a *retroaortic* course, posterior to the aorta between the aortic root and left atrium; a *prepulmonic* course, anterior to the pulmonary artery or right ventricular outflow tract; or a *transseptal* (subpulmonic) course, traversing the upper interventricular septum [3,6,18,21]. LAD arising from the right coronary sinus takes either interarterial or prepulmonic course [6].
Fig. 5: Oblique axial maximum intensity projection image of aortic root shows RCA (arrow) arising from left sinus of Valsalva and coursing between the aorta and the pulmonary artery trunk (malignant course).

**References:** Radiology Department, Guy’s and St Thomas’ Hospital, London, UK

**Significance:**

- The interarterial course is also referred to as malignant anomaly (Fig. 5) [3,18]. (details in Table 3)

- The retroaortic course is important to recognize in patients with aortic valve disease because the artery may be compressed during aortic valve or aortic root surgery [23].
- The transseptal course is considered to be only relatively benign due to reports suggesting a potentially fatal outcome [23].

- The prepulmonic course is considered clinically insignificant (Fig. 6) [23].

- Unstable hemodynamics cause blood flow turbulence and injury to vascular endothelium leading to arteriosclerosis [6].

- An LCx originating from the right coronary sinus or proximal RCA is a benign anomaly that usually takes a retroaortic course and has not been associated with sudden death and ischemic heart disease [6,18]; however it may predispose to atherosclerosis [21]. Knowledge of this coronary anomaly by cardiac surgeons is important to prevent compression of the LCx by prosthetic valve fixation rings during valve surgery [18,21].

- There is a higher prevalence of chest pain in patients with anomalous RCA compared to those with anomalous LMA due to a more frequent intramural course of the RCA, higher values of the coronary orifice area and less common ostia stenosis of the LMA [30].

**Single coronary artery**

Single CA is particularly rare among anomalies, characterized by a single CA ostium from an aortic sinus and associated with congenital heart disease in up to 40% of cases [6,18]. There are 5 patterns of distribution: a single CA supplying the entire myocardium; a single artery with 2 major branches, 1 of which has a retroaortic course (Fig. 7); 2 major branches, 1 of which has an interarterial course; 2 major branches, 1 of which has a prepulmonic course; 3 equally dominant major branches [3].

*Significance:*

- Benign or associated with ischemic heart disease or sudden death when the origin of the CA is on the opposite side to its perfusion region and branches turn in acute angle at the base of the heart. Depending on how the CA courses, it may be squeezed among cardiac muscles or compressed between the aorta and the pulmonary artery [6,18,31]. In the case of a single CA with an interarterial course, the lack of collateral flow from the opposite side makes this a more critical anomaly [31].

- Endothelial injury occurs due to high flow load on one CA [6].

**Anomalies of intrinsic coronary arterial anatomy**

**Congenital ostial atresia** is characterized by an ostial dimple in a coronary sinus without a patent arterial lumen. More commonly, the proximal LMA ends blindly with blood flowing from the RCA to the left coronary circulation via collateral arteries [4].
The average LMA length has a reported range of 10+/-5mm. Left dominance is associated with shorter LMAs. The longer the LMA, the greater the angle of bifurcation [17].

Significance:
- To avoid misdiagnoses of diseases and for proper stent placement during percutaneous coronary intervention in the area of bifurcation [17].

Occasionally, the LMA trifurcates, with the middle branch known as the **ramus intermedius** (Fig. 8) [11]. The ramus intermedius courses laterally, similar to the first diagonal branch of the LAD, to supply the anterior left ventricle wall [12].

Significance:
- Decreased number of diagonal branches [7].
- An occlusion in the ramus intermedius in patients with no collaterals from the LCx and LAD supplying the region of the ramus intermedius may be as dangerous as an occlusion in the LCx or the LAD [17].

**A CA aneurysm** is defined as a focal saccular or fusiform dilatation of the CA that is 1.5x or more the diameter of the adjacent normal arterial segment. Diffuse dilation of an entire artery is termed "ectasia". CA aneurysm of congenital etiology is rare, most common in the RCA; more frequently it is associated with Kawasaki disease, lupus, and atherosclerotic CA disease [3,10].

Significance:
- Risk of thrombosis, rupture, myocardial ischemia, and fistulous communication with adjacent structures [10].
- CA ectasia may lead to increased risk of coronary atherosclerotic disease or ischemic cardiomyopathy [3].

**A myocardial bridge** (MB) is a congenital condition in which a segment of CA goes intramurally through the myocardium beneath a muscle bridge [32]. The MB is most commonly seen in the diagonal branches and the mid segment of the LAD [32,33]. The vessel may return to an epicardial position after the muscle bridge, or have an intramyocardial course, defined as a vessel running and ending in the myocardium [33]. Myocardial bridging with partial encasement is defined as the LAD being within the interventricular gorge and in direct contact with left ventricular myocardium. Myocardial bridging with full encasement is defined as the CA being surrounded by the myocardium with or without measurable overlying muscle (Fig. 9, 10) [34]. Comparison of the images
obtained during the systolic and diastolic phases allows the assessment of the luminal narrowing during the systolic phase [21].

Fig. 9: Oblique reformatted maximum intensity projection images of mid RCA show long segment of intramyocardial course (arrows) preceded and succeeded by epicardial course.

References: Radiology Department, Guy's and St Thomas' Hospital, London, UK

Significance:

- Generally, it is a benign condition, but has been associated with angina, myocardial ischemia or infarction, arrhythmia, and sudden death. The symptoms are considered to be caused by coronary ischemia attributed to a reduction in blood flow subsequent to coronary compression by the MB during systole, by delayed arterial relaxation in diastole, or both [32].
- The thickness and length of MB in patients with culprit lesions in LAD are significantly larger than those in patients without myocardial infarction in the cardiac area supplied by LAD. The MB length cut-off value was estimated as 18.55 mm [35].

- Tunneled segments are known to be spared by atherosclerotic change, whereas the latter are more frequent in the segment proximal to the MB [32]. (details in Table 4)

- Complications, such as perforation of the right ventricular wall, when exposure of the intramuscular arterial segment is attempted [37].

An intracavitary course of an intact epicardial CA is rare. Two variants have been described: an intracavitary course of the proximal LAD into the right ventricle (Fig. 11) and an intracavitary course of the RCA into the right atrium (Fig. 12) [38].
Fig. 11: Curved reformatted maximum intensity projection images (upper row), oblique long-axis reformatted image (left lower image) and short-axis reformatted image (right lower image) demonstrate an intracavitary course of the LAD into the right ventricle (arrows).

References: Radiology Department, Guy’s and St Thomas' Hospital, London, UK

Significance:

- Accidental disruption of an intracavitary artery resulting in left-to-right shunting or distal myocardial ischemia can occur at the time of invasive coronary angiography, pacemaker implantation, right heart catheterization or invasive electrophysiology testing.

- Intra-atrial CA damage may occur during intracardiac electrode placement for the diagnostic electrophysiology study, during transseptal puncture for left atrial access, during radiofrequency ablation of ventricular tachyarrhythmias originating in the right ventricle (intracavitary LAD) or atrial tachyarrhythmias arising in the right atrium (intracavitary RCA).

- An intracavitary course can complicate CA bypass surgery, with difficulties in vessel localization and bypass grafting.

- Risk of direct trauma at the time of inferior vena cava or coronary sinus cannulation for cardiopulmonary bypass or retrograde delivery of cardioplegia.[38]

Duplication of the CAs can be seen with the LAD, LCx, or RCA. Duplication of the LAD is more commonly reported, while LCx duplication is the least common. The duplicated artery may arise from the corresponding CA of the same name, directly from the same aortic sinus, ectopically from another aortic sinus or CA, or from a pulmonary artery [3].

There are 4 types of double LAD. In types 1 and 2, a long LAD originates as a branch from the proper LAD, takes a course parallel to the short LAD in its proximal course on either the left ventricle (type 1) or the right ventricle (type 2), and reenters the anterior interventricular groove in the distal aspect. Type 3 duplication of the LAD is characterized by a proximal intramyocardial course of the long LAD and appears on the epicardial surface in the distal part of anterior interventricular sulcus. In type 4 the long LAD arising from the right coronary sinus or RCA (Fig. 13) takes an anomalous course and enters the anterior interventricular groove [10,21].

A double RCA is an extremely rare variant and often found incidentally because they do not cause any clinical signs or symptoms [39]. There are 3 types of double RCAs: type 1 - arising from 2 separated orifices in the right sinus of Valsalva; type 2 - two separate RCAs originated from a common orifice in the right sinus of Valsalva (Fig. 14); type 3 - double RCAs coming off a relatively long common segment of the main RCA [38].

Significance:
- The first two types of double RCA might be vulnerable to atherosclerosis [39].

- ECG features of isolated RCA occlusion in type 3 double RCA can mimic left coronary occlusive disease [39].

- In type 1 it is important to have anatomical details about an ectopic opening of the CA prior to intervention for proper management [40].

**Anomalies of coronary termination**

A **CA fistula** is an abnormal termination of the CA characterized by a communication between the CAs and either a cardiac chamber, systemic vein, or the pulmonary artery. CA fistula drainage sites are clinically important and are mostly represented by the right ventricle, the right atrium (Fig. 15), the pulmonary artery, the coronary sinus, the left atrium and left ventricle [18,21]. Fistulas more commonly involve the RCA than the LMA. In <5% of the cases, fistulas originate from both the LMA and the RCA [21]. A fistulous communication between a CA and cardiac chamber is known as **coronary arterio-cameral fistula**. The involved CA is often dilated and tortuous, due to increased blood flow and shunting [3]. In the majority of cases, the fistula has a single communication and is not clinically important, but the minority of cases has multiple communications with a diffuse network that contains aneurysms [18].
Fig. 15: Volume-rendered anterior oblique image shows RCA fistula draining into the right atrium (arrow) - coronary arterio-cameral fistula; note the dilated and tortuous RCA.

References: Radiology Department, Guy's and St Thomas' Hospital, London, UK

Significance:

- CA fistula may lead to increased risk of coronary atherosclerotic disease.

- Surgical correction is indicated when a high flow fistula, multiple communications, a complex network, multiple terminations, or significant aneurysm formations is detected [18].
- High-flow fistulas and enlarged vessels may develop myocardial ischaemia and congestive cardiac failure.

- Small CA fistulas that drain into left-sided heart chambers or the pulmonary artery are more common and relatively benign.

- Larger CA fistulas draining into a left heart chamber can lead to left ventricular overload and may clinically mimic aortic insufficiency.

- When a CA fistula drains into a right-sided chamber, the clinical presentation is a left-to-right shunt, dilatation of right heart chambers, right ventricular overload, and coronary steal phenomenon within larger fistulas.

- Other complications include endocarditis and the development of mural thrombus in the dilated CA due to aneurysm formation, with potential for rupture or side branch obstruction.[3]

Extracardiac termination

Congenital communications between the CAs and extracardiac vascular structures such as the bronchial, internal mammary and phrenic arteries, intercostals and esophageal arteries [3]. These pathways become functionally significant only when a pressure gradient exists between the two arterial systems.

Significance:

- Generally associated with atherosclerotic coronary artery disease, leading to blood flow from the bronchial artery to the coronary arteries [13].

Images for this section:

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<th>CORONARY ARTERY</th>
<th>BRANCHES</th>
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<td>RCA</td>
<td>conus artery, sinoatrial artery, acute marginal artery, atrioventricular nodal artery, PDA, PLB</td>
<td>courses anteriorly and laterally (between the right auricle and pulmonary trunk) from its ostium at the right sinus of Valsalva and runs in the right atrioventricular groove, curving posteriorly at the acute margin of the RV, bifurcating into the PDA and the PLB of the LV at the crux of the heart</td>
<td>the RV outflow tract, the right and left atrium, the RV free wall</td>
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<td>LMA</td>
<td>PDA, PLB, LAD, LCx</td>
<td>the PDA runs in the posterior interventricular groove, the PLB courses on the posterolateral surface of the LV, from the left sinus of Valsalva passes leftward posterior to the pulmonary trunk and divides into the LAD and LCx</td>
<td>the posterior 1/3 of the septum, the posterior surface of the LV</td>
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<td>LAD</td>
<td>septal perforators, diagonalis (D1, D2, ...)</td>
<td>LAD courses anteriorly and inferiorly in the anterior interventricular groove to the apex of the heart, run perpendicular to the LAD, run on the epicardial surface of the heart</td>
<td>the left atrium, the apex of the heart, the anterior 2/3 of the septum, the anterolateral part of the LV</td>
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<td>LCX</td>
<td>obtuse marginal arteries (OM1, OM2, ...)</td>
<td>LCx runs along the left atrioventricular groove and in ~80% of the population terminates at the obtuse margin of the heart, or may continue posteriorly to the crux of the heart</td>
<td>the lateral aspect of the LV, the anterolateral LV papillary muscle</td>
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* LAD - left anterior descending; LCx - left circumflex artery; LMA - left main coronary artery; LV - left ventricle; PDA - posterior descending artery; PLB - posterolateral branches; RCA - right coronary artery; RV - right ventricle; ** source [11-13]
### Table 1: Normal coronary arteries anatomy

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<td>Decreased number of arteriosar/capillary ramifications</td>
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### Table 2: Classification of coronary artery anomalies
**Fig. 1:** Volume-rendered posterior view of a left dominant heart shows the distal LCx giving rise to the posterior descending artery (arrow) and the posterior left ventricle branches.
Fig. 2: Volume-rendered CT image of heart in left anterior oblique projection shows absent LMA and separate origin (arrow) of the LAD and LCx
Fig. 3: Oblique volume-rendered image of the top of the heart shows a common ostium (arrow) of the RCA and the conus artery
**Fig. 4:** Volume-rendered CT images in left anterior oblique projection (A) and of the top of the heart (B), (C), with windowing adjusted to "see through" translucent right ventricular outflow tract, demonstrate the RCA originating from the left coronary sinus (arrows).
Fig. 5: Oblique axial maximum intensity projection image of aortic root shows RCA (arrow) arising from left sinus of Valsalva and coursing between the aorta and the pulmonary artery trunk (malignant course).
Syncope, chest pain [24], ischemic heart disease [6], myocardial infarction and sudden cardiac death result from impairment flow by collapse of a slit-like coronary orifice in a valve-like manner, acute take off at the origin, an intramural course within the aortic wall (the CA and the aorta share the same media without the interposition of adventitia, resulting in systolic compression of this segment [25]), and transient occlusion of the anomalous CA between the aorta and pulmonary artery which expand as pressure of both vessels increases during exercise [6,18,25,26], 
- A CA ostium located above the pulmonary valve, followed by a high interarterial course, results in compression of the interarterial segment, because during systole both the aorta and the pulmonary artery are distended. The right ventricular outflow tract contracts during systole, therefore a CA ostium below the pulmonary valve, with a low interarterial course, would be less compressed [25,27].
- Other features for increased risk of cardiac events include: a coronary minimal lumen area ≤4mm², an area stenosis ≥50 %, a longer intra-arterial course (>10mm) and a smaller width of proximal segment [25].
- Direct visualization of an intramural course is not reliable with the current CT technology, however an acute angle of origin from the aorta, a slit-like orifice, an elliptical cross-sectional shape with vessel height/width ratio >1.3 and a tangential initial coronary course are indirect signs of intramural origin of the CA [25,28]. Symptoms seem to be correlated with imaging measurements of the intramural CA length: 7.8±2.8 mm in positive symptoms vs. 5.3±0.8 mm in cases with no symptoms [29].

**Table 3:** Physiopathology in interarterial course of coronary arteries
Fig. 6: Volume-rendered right anterior oblique image demonstrates a common origin of the LAD and the RCA from the right coronary sinus (arrow) and shows a prepulmonic course of the LAD.
Fig. 7: Right posterior oblique volume-rendered image shows a single coronary artery (arrow) originating in the noncoronary sinus, with two major branches, one - the RCA - having a retroaortic course.
Fig. 8: Volume-rendered images of heart in left anterior oblique projections show trifurcation anatomy (A) and quadrifurcation anatomy (B) of LMA (arrows).
Fig. 9: Oblique reformatted maximum intensity projection images of mid RCA show long segment of intramyocardial course (arrows) preceded and succeeded by epicardial course.
Fig. 10: Volume-rendered images of heart in left anterior oblique projections show myocardial bridging (arrows) of the ramus intermedius (A), diagonals (B), obtuse marginal branch (C).

The intima beneath the bridged segments consists only of contractile-type smooth muscle cells, and lacks synthetic-type smooth muscle cells. The synthetic-type smooth muscle cells usually proliferate and produce collagen fibrils and elastic fibers in the intima as atherosclerosis progresses. Myocardial contraction causes increased shear stress in the arterial wall which in the tunneled artery is believed to have a protective effect against atherosclerosis, a low shear stress in the arterial segments proximal to MB might induce release of endothelial vasoactive agents leading to increased atherosclerosis [32]. Such susceptibility to atherosclerosis in the LAD intima proximal to MB originates from the retrograde blood flow toward proximal LAD by MB compression force at cardiac systole because this abnormal blood flow causes the increases in local wall tension and stretch in LAD segments, which induces functional damage of the endothelial cells [35]. MB causes the compression of the tunneled segment during systole, enhancing the lymph drainage of the vessel wall that is important for the prevention of lipid accumulation and disease development [36].

Table 4: Physiopathology of atherosclerosis in myocardial bridging

Fig. 11: Curved reformatted maximum intensity projection images (upper row), oblique long-axis reformatted image (left lower image) and short-axis reformatted image (right
lower image) demonstrate an intracavitary course of the LAD into the right ventricle (arrows).

Fig. 12: Oblique reformatted maximum intensity projection images show an intracavitary course of the RCA into the right atrium (arrows).
Fig. 13: Volume-rendered anterior oblique image demonstrates type 4 double LAD (arrows): a shorter branch arising from the LMA and a longer LAD originating from the same ostium as RCA, in the right coronary sinus.
Fig. 14: Oblique volume-rendered image of the top of the heart shows type 2 double RCA arising from a common ostium in the right sinus of Valsalva (arrow).
Fig. 15: Volume-rendered anterior oblique image shows RCA fistula draining into the right atrium (arrow) - coronary arterio-cameral fistula; note the dilated and tortuous RCA.
Conclusion

Familiarity of radiologists with each specific variant and anomaly of the CAs and its spectrum of potential implications is essential in making a correct diagnosis.

References


