The left main coronary artery (LM) originates from the left coronary sinus of Valsalva and gives origin to the left anterior descending coronary artery (LAD) and left circumflex coronary artery (LCX). The LAD courses in the anterior epicardial ventricular septum and gives origin to various diagonals and septal perforators. The LAD is divided into proximal, mid, and distal segments. The first septal perforator generally divides the proximal and mid-segments of the LAD. The diagonals are varied in number and caliber and are labeled from proximal to distal, D1, D2, D3, and so forth. The LCX runs in the left atrial–ventricular sulcus and gives origin to obtuse marginal branches (OM). The OMs are labeled from proximal to distal, OM1, OM2, OM3, and so forth. Ostium refers to the segment of origin of the artery (Figure 1.1A–Z).

The right coronary artery (RCA) originates from the right coronary sinus and is divided in proximal, mid, and distal segments. The proximal segment of the RCA is from the ostium to the origin of the first acute marginal artery. In the majority of patients, the conus artery originates from the ostium of the RCA or separately from the right coronary sinus and is generally the first visualized branch. The conus artery has a superior and anterior course. The sinoatrial (SA) artery is generally the second artery to be visualized and originates from the proximal RCA and has a posterior course. The RCA gives origin to acute marginal (AM) branches, which vary in size and number and are labeled from proximal to distal, AM1, AM2, AM3, and so forth.

Dominance refers to whether the posterior descending artery (PDA) originates from the RCA (right dominant), LCX (left dominant), or both (codominant). Approximately 80% of humans are right dominant. In right dominance, the distal RCA at the level of the crux of the heart typically bifurcates into the PDA and a posterolateral branch. The PDA courses in the posterior ventricular septum giving origin to the SA nodal artery and posterior ventricular branch. In left dominance, the PDA originates from the distal LCX. In co-dominance, there are right and left PDAs originating from the RCA and LCX.

The coronary venous system is variable. Generally, the great cardiac vein (GCV) and the middle cardiac (MCV) vein are present. The GCV runs parallel to the LAD and then courses superiorly, crossing the LCX and posteriorly draining into the coronary sinus. The MCV runs inferiorly at midline parallel to the PDA and drains into the coronary sinus.
Figure 1.1. (A–F) Axial. (G–I) Coronal. (O–T) Sagittal. (U–Y) VR. (Z) 2D composite. A, anterior; AA, ascending aorta; APM, anterior papillary muscle; AR, aortic root; AV, aortic valve; CA, conus artery; CS, coronary sinus; D1, diagonal 1; D2, diagonal 2; DA, descending aorta; ES, esophagus; GCV, great cardiac vein; I, inferior; IVC, inferior vena cava; LA, left atrium; LAA, left atrial appendage; LAD, left anterior coronary artery; LCX, left circumflex coronary artery; LIMA, left internal mammary artery; LIPV, left inferior pulmonary vein; LM, left main coronary artery; LMSB, left main stem bronchus; LSPV, left superior pulmonary vein; LV, left ventricle; MB, moderator band; MCV, middle cardiac vein; MV, mitral valve; OM1, obtuse marginal 1; P, posterior; PA, pulmonary artery; PAB, pulmonary artery branch; PDA, posterior descending artery; PM, papillary muscles; PV, pulmonic valve (axial C and coronal D); pulmonary vein (sagittal D); PVB, pulmonary vein branch; RA, right atrium; RAA, right atrial appendage; RCA, right coronary artery; RIMA, right internal mammary artery; RIPV, right inferior pulmonary vein; RMPA, right main pulmonary artery; RPV, right pulmonary veins; RPA, right pulmonary artery; RMSB, right main stem bronchus; RSPV, right superior pulmonary vein; RV, right ventricle; RVOT, right ventricular outflow tract; S, superior; SA, sinoatrial artery; SVC, superior vena cava; STE, sternum.
Figure 1.1. (Continued)
Pearls and Pitfalls

The coronary anatomy has great variability. The arteries vary in size, length, course, and branches. It is important to understand the individual anatomy of a patient in order not to mistakenly report disease or occlusion of an artery. Close observation must be made as to how the myocardium is receiving its blood supply in normal and diseased arteries. This will be of great importance in the subsequent chapters that illustrate normal coronary variants, congenital coronary anomalies, coronary artery disease (CAD), and collateral pathways.

If the IV contrast bolus is timed correctly, you will note that the veins are less dense and generally larger than the adjacent coronary arteries. The veins should not be mistaken for diseased or occluded arteries. If in doubt, follow the vessel to its origin (artery) or drainage (vein).

Case 1.1

History

A 47-year-old asymptomatic man presented with strong family history of coronary disease.

Findings

The study demonstrated a right dominant coronary anatomy and without disease (Figure 1.2A–I).

Diagnosis

Normal coronary CTA was acquired with prospective gated axial technique (PGA), also known as step and shoot.

Discussion

Radiation exposure is of significant concern, particularly in younger patients. The cardiac CTAs are most commonly acquired using a multicyle retrospective technique. Multiple cardiac cycles are acquired with radiation exposure during the entire cycle (approximately 8–30 mSv). Techniques have been developed to reduce radiation exposure by decreasing the x-ray tube output in the nondiastolic phase of the cycle. This is also known as dose modulation, which can achieve, depending on the heart rate, a reduction of the radiation of up to 40%.

In the last quarter of 2007, PGA acquisition became commercially available. The technique is similar to that of a calcium score in which a single mid diastolic phase (typically 75%) of the cardiac cycle is targeted and the x-ray tube only exposes during this segment (20% of the cycle). This is also called step and shoot since in a 64-slice CT scanner, a nonhelical single cycle is acquired per gantry rotation followed by several other steps in the Z (direction 19.5–40 mm), in order to include the entire heart (approximately 12 cm). PGA allows up to 80% reduction of the radiation dose (approximately 3 mSv), similar to a calcium score. Even greater radiation reduction can be achieved with the use of a lower keV (80–100).
FIGURE 1.2. (A–D) VR: Normal PGA CCTA. (E) 2D composite. PGA normal coronary arteries. (F–I) cMPR of the coronary arteries. With appropriate patient selection and acquisition, the quality of the PGA images is excellent. Also, the spatial resolution is the same as a retrospective acquisition.
Currently, we use PGA in younger patients for coronary artery disease assessment and who have no documented history of coronary obstruction, prior coronary intervention, or surgical revascularization.

In order to acquire a diagnostic study, a low stable heart rate is required, that is, under 65 bpm. The use of oral or IV beta blockers is recommended in most patients. In our laboratory, the success rate of PGA has been greater than 95%.

**Pearls and Pitfalls**

We currently perform the PGA with an IV dose of 60–70 mL of high-density low-osmolar contrast (350 mg/mL). Following the acquisition, the reconstructed images are immediately assessed to determine whether these are diagnostic. If the study is not considered diagnostic, a second acquisition is quickly performed, unless contraindicated; with a second IV injection of contrast, switch to the standard retrospective dose-modulated technique. The combined total iodine dose does not exceed 50 g. This protocol avoids the patient having to be rescheduled. With PGA, only a single phase is acquired, consequently cardiac wall motion cannot be assessed.

**Case 1.2**

**History**

A 47-year-old man presented with dyslipidemia and a strong family history of CAD.
Findings

The RCA and LCX are short arteries of small caliber. The posterior descending artery was not identified. The LAD and ramus intermedius (RI) arteries are long and have large caliber (Figure 1.3A and B).

Diagnosis

The diagnosis is normal variant of left coronary dominant anatomy.

Discussion

It is important to differentiate normal variant coronary anatomy from acquired and congenital abnormalities. The LAD and RI in this case provide flow to the inferior and posterior wall of the left ventricle, compensating for the lack of a posterior descending artery originating from the RCA or LCX.

Pearls and Pitfalls

The differentiation between an acquired obstruction of the PDA with compensatory hypertrophy of the LAD and RI versus a normal variant can be ascertained by the identification of a very small RCA and LCX.

Case 1.3

History

A 59-year-old woman presented with atypical chest pain and a normal cardiac perfusion scan result.

Findings

There is absence of the right coronary artery. Mild disease is seen in the mid-LAD. Coronary circulation is left dominant, with the left circumflex extending into the right atrial–ventricular sulcus (Figure 1.4A–C).
FIGURE 1.4. (A) 2D map. Left anterior coronary artery and left circumflex coronary artery. (B) Axial. Absent right coronary artery (arrow). (C) Globe sphere: Left circumflex coronary artery extension into the right atrial-ventricular sulcus.

**Diagnosis**

The diagnosis is congenital absence of the right coronary artery, with a super-dominant left circumflex.

**Discussion**

Knowledge of the cardiac physiology, normal, variant anatomy, and anomalies of coronary circulation is an increasingly vital component in managing congenital and acquired heart disease. In this case, the absence of the right coronary artery is well compensated by the LAD and a long large-caliber left circumflex coronary artery (LCX).

**Pearls and Pitfalls**

The lack of visualization of the RCA originating from the right sinus of Valsalva, with a dominant left circumflex coronary artery, usually indicates an anatomic variant rather than an occluded RCA.
Suggested Readings


Kini S, Bis KG, Weaver L. Normal and variant coronary arterial and venous anatomy on high-resolution CT angiography. AJR Am J Roentgenol 2007;188(6):1665–1674.


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