The prevalence of coronary artery anomalies in Qassim Province detected by cardiac computed tomography angiography

Osama A. Smettei a,⇑, Sawsan Sayed a, Rami M. Abazid a

a Department of Cardiology, Cardiac Imaging Department, Prince Sultan Cardiac Center Al-Qassim, Buraydah

Background: Coronary artery anomalies (CAAs) affect about 1% of the general population based on invasive coronary angiography (ICA) data, computed tomography angiography (CTA) enables better visualization of the origin, course, relation to the adjacent structures, and termination of CAAs compared to ICA.

Objective: The aim of our work is to estimate the frequency of CAAs in Qassim province among patients underwent cardiac CTA at Prince Sultan Cardiac Center.

Methods: Retrospective analysis of the CTA data of 2235 patients between 2009 and 2015.

Results: The prevalence of CAAs in our study was 1.029%. Among the 2235 patients, 241 (10.78%) had CAAs or coronary variants, 198 (8.85%) had myocardial bridging, 34 (1.52%) had a variable location of the Coronary Ostia, Twenty two (0.98%) had a separate origin of left anterior descending (LAD) and left circumflex coronary (LCX) arteries, ten (0.447%) had a separate origin of the RCA and the Conus artery. Seventeen (0.76%) had an anomalous origin of the coronaries. Six (0.268%) had a coronary artery fistula, which is connected mainly to the right heart chambers, one of these fistulas was complicated by acute myocardial infarction.

Conclusions: The incidence of CAAs in our patient population was similar to the former studies, CTA is an excellent tool for diagnosis and guiding the management of the CAAs.

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Keywords: Coronary artery anomalies, Cardiac computed tomography, Prevalence

Introduction

The prevalence of coronary artery anomalies (CAAs) is reported to be 0.3% to 2% of the general population [1–3]. Asymptomatic CAAs are more common and have a better prognosis. Nevertheless, some of these anomalies are linked with symptoms such as syncope, chest pain, and sudden cardiac death [4,5]. CAAs include abnormalities of either number, origin course, termination, or structure of the coronary arteries [6]. CAAs...
are detected usually as incidental findings during invasive coronary angiography or at postmortem examination. Various imaging modalities are available for assessment of CAAs. Invasive coronary angiography cannot define CAAs in detail, especially with the complex anatomy. By contrast computed tomography angiography (CTA) is a noninvasive tool that permits a clear visualization of CAAs better than invasive coronary angiography. CTA has recently become the test of choice for diagnosing CAAs [7,8]. The aim of our study is to evaluate the prevalence of CAAs in the Qassim area using CTA.

Patients and methods

A retrospective analysis of 2235 coronary CTA scans was performed in our center between 2009 and 2015 to identify patients with CAAs. Most of the patients were referred for evaluation of chest pain. Other indications included: evaluation of congenital heart disease; determining the three-dimensional anatomy of the CAAs detected by invasive coronary angiography; evaluation before coronary artery bypass surgery; detection of coronary artery disease before noncardiac surgery; anatomic mapping before atrial fibrillation ablation; and excluding of coronary artery disease in patients with cardiomyopathy (Table 1). Patients with uncontrolled arrhythmia, previous allergic reaction to the iodinated contrast material, pregnancy, renal impairment (serum creatinine >3.0 mg/dL), and an inability to hold breath were excluded. The patients with CAAs were selected, and each CAA was reviewed for origin, course, relation to adjacent structures, and the termination of these anomalies. All patients had signed informed consent to be a part of this study. The study was approved by the ethics committee of Prince Sultan Cardiac Center Al-Qassim.

Data acquisition and reconstruction protocol

Using a dual-source 256 slice scanner (Siemens Flash Definition CT scanner; Siemens, Berlin, Germany), rotation time 280 ms, and GE Light Speed VCT 64 slice CT Scanner (GE Healthcare, Chicago, IL, USA), all imaging started with a scout image and then a calcium score scan with 3-mm slice thickness and prospective gating at 75% of the cardiac cycle. We used a 0.6-mm slice thickness and electrocardiography (ECG) gating with either prospective, retrospective during the breathing hold in inspiration for enhanced CTA scan, we used ECG-dependent tube current modulation of 40% to 75% of R–R internal in retrospective scanning to minimize radiation exposure. We used a test bolus technique with a 4-second delay time after the peak contrast enhancement of a region of interest in ascending aorta, using 15 mL of contrast agent (Xenetix 350; Guerbet, Roissy, France; 350 mg iodine/mL), then 20 mL normal saline. The CTA scan was performed by injecting of 75 mL of contrast and 45 mL of saline solution at a rate of 6 mL/s. Prior to CTA, all patients with a baseline heart rate of >65 beats/min received 5–20 mg of metoprolol intravenously. Sublingual nitroglycerin (0.5 mg) was given to all the patients during the scan unless contraindicated. Medium smooth kernels (B26f) were reconstructed for postprocessing using a Multimodality Workplace (Siemens Medical Solutions, Erlangen, Germany). The axial, coronal, sagittal, and oblique multiplanar reconstruction; thin-slab maximum intensity projection; and volume-Rendering images were reformatted. Different retrospective ECG-gated reconstruction temporal window settings (in retrospective scan), usually between 40% and 75% of R–R intervals in gated ECG were applied, and we did the reconstruction at 75% window in the prospective ECG gated scans.

Results interpretation

CTA images were interpreted independently by two cardiologists who had at least 6 years’ experience in cardiac CTA. The origin, course, relation to adjacent structures, termination, branches, and

| Indication                          | n (%) |
|------------------------------------|-------|
| All patients                       | 2235 (100) |
| Atypical chest pain                | 1947 (87.1) |
| Non cardiac chest pain             | 99 (4.42)  |
| Typical chest pain                 | 15 (0.67)  |
| Before coronary artery bypass surgery | 35 (1.56)  |
| Before atrial fibrillation ablation | 44 (1.96)  |
| Congenital heart disease           | 13 (0.58)  |
| Before noncardiac surgery          | 18 (0.8)   |
| Syncope                            | 5 (0.22)   |
| Coronary artery anomalies          | 9 (0.4)    |
| Other indications                  | 50 (2.2)   |
territories of CAAs were retrospectively determined in all patients. In our review, the CAAs were divided into four primary groups: anomalies of origin, course, termination, and intrinsic [8–10]. Classification of CAAs and coronary variants is shown in Table 2.

### Results

A comprehensive classification of CAAs has been utilized and described in Table 2. Of our patients, 241 (10.78%) had CAAs or normal coronary variants. The prevalence of CAAs using the mentioned classification is presented in Table 2. Three patients were referred after invasive coronary angiography, one patient with congenital heart disease for further assessment while the remaining CAAs was diagnosed primarily by CTA.

Myocardial bridging (Fig. 1B, C) was the most common variant (198 patients, 82.15% of all anomalies and variants, 8.85% of all patients), with more involvement of the middle part of the left anterior descending artery (LAD; 163 out of 198 patients). Seventeen patients (7.05% of CAAs), had anomalies of the origin (Fig. 2), five patients had right coronary artery (RCA) arising from the left SV; three of them had intra-arterial course between the aortic root and the pulmonary artery, four patients (23.5%) had the left main coronary artery (LMCA) arising from the right SV; three had intra-arterial course (Fig. 2B, G, H); and the remaining two had a benign course (anterior to the Pulmonary artery). Four patients (23.52%) had a left circumflex coronary artery (LCX) that arose from the right SV and passed posterior to the aorta; two patients (11.76%) had a single coronary artery, both arising from the right SV, with an anterior course of LMCA (Fig. 2E, F) for the first patient, and an intra-arterial course of the LAD and the LCX passed posterior to the aorta in the second patient. Separate ostia of the LAD and the LCX (Fig. 1A) were seen in 22 patients (9.12% of all CAAs). Variant coronary ostia were seen in 34 patients (14.1% of all CAAs), LMCA from noncoronary sinus of valsalva in 14 patients, RCA from noncoronary sinus of valsalva in nine patients, and high take off of the coronary artery in 14 patients. Coronary artery fistulas (CAFs) were seen in six patients (2.48% of CAAs, 0.268% of all patients), mostly connected to the right

### Table 2. The classification and prevalence of coronary artery anomalies (CAAs) and variants.

| Classification of CAAs and coronary variants and prevalence: n (%) | Saudi patients (n = 1933) | CAAs, Saudi patients (n = 212) | All patients (n = 2235) | CAAs patients (n = 241) |
|---|---|---|---|---|
| **Anomalies of origin** | | | | |
| Number of coronary ostia | 28 (1.448%) | 28 (13.2%) | 32 (1.43%) | 32 (13.27%) |
| Anomalous location of ostia | 27 (1.2%) | 27 (12.73) | 34 (1.52%) | 34 (14.1%) |
| Anomalous origin of CA from opposite sinus | 15 (0.775%) | 15 (7.07%) | 17 (0.76%) | 17 (7%) |
| Single coronary artery | 2 (0.103%) | 2 (0.94%) | 2 (0.0894%) | 2 (0.82%) |
| **Anomalies of course** | | | | |
| Myocardial bridge | 179 (8%) | 179 (84.45) | 198 (8.85%) | 198 (82.15) |
| **Anomalies of termination** | | | | |
| Coronary arteriovenous fistula | 5 (0.258%) | 5 (2.358%) | 6 (0,268%) | 6 (2.48%) |
| Extracardiac connections | 2 (0.1%) | 2 (0.943%) | 2 (0.08%) | 2 (0.82%) |
| **Intrinsic CAs** | | | | |
| Atresia of the left main CA | 2 (0.1%) | 2 (0.943%) | 2 (0.08%) | 2 (0.82%) |
| CA ectasia or aneurysm | 4 (0.2%) | 4 (1.88%) | 6 (0.268%) | 6 (2.48%) |

CA = coronary artery.

### Table 3. The baseline clinical characteristics of the patients with CAAs (n = 241).

| Age (y) | 52 (24–77) |
|---|---|
| Sex | | |
| Male | 166 |
| Female | 75 |
| Presenting symptoms | | |
| Chest pain | | |
| Typical | 12 |
| Atypical | 180 |
| Noncardiac | 34 |
| Syncope | 3 |
| Palpitation | 29 |
| Dyspnea | 28 |
| Risk factors | | |
| Diabetes | 34 |
| Hypertension | 56 |
| Dyslipidemia | 29 |
| Smoker | 55 |
chambers, with one connection between aneurysmal LMCA and right atrium complicated by myocardial infarction due to LCX occlusion by the thrombus. We did a subgroup analysis to look into the prevalence of CAAs in Saudi patients (1933 patients, 86.49% of all patients) and we found CAA prevalence of 1.034% (20 patients out of 1933) (Table 2).

Figure 1. (A) Maximum intensity projection image showing separate origin of left anterior descending artery (LAD) and left circumflex coronary artery (LCX). (B) Multiplanar reformation image and showing a mid-LAD bridge with significant narrowing at the bridge site. (C) Volume rendering image showing mid-LAD bridge and the intramyocardial segment of the LAD. (D) Multiplanar reformation image showing ectatic LCX artery affecting its proximal part. (E) Multiplanar reformation image of a thrombosed coronary artery fistula (CAF) between LM and right atrium. (F) Multiplanar reformation image from the same patient as D showing occluded LCX at the ostium (arrow) by thrombus, mid and distal thromboses. (G) Volume rendering image from the same patient showing the three-dimensional orientation of the CAF, its course and the connections between left main coronary artery and right atrium. (H, I) Volume rendering images showing complex CAF between right coronary artery, pulmonary artery, and left main coronary artery.

Figure 2. (A) Axial image showing anomalous left anterior descending artery from right SV with intra-arterial course and normal course of the left circumflex coronary artery. (B) Volume rendering image showing anomalous left main coronary artery from right SV and intra-arterial course. (C, D) Volume rendering images from different patients showing anomalous left anterior descending artery from right SV and the intra-arterial course. (E, F) Volume rendering images showing a single coronary artery arise from the RSV and its anterior course. (G, H) Multiplanar reformation and Volume rendering images showing anomalous left main coronary artery from right SV with along intra-arterial course and then the intraseptal course.
Discussion

The prevalence of CAAs in our survey was 1.029% (1.034% in Saudi patients). Which is close to the prevalence of CAAs in the literature [1–3]. To our knowledge, this is the first survey to look into the prevalence of CAAs in the Saudi population using CTA. Invasive coronary angiography is usually unable to offer sufficient information about the complex anatomy of CAAs compared with the CTA. The primary disadvantage of CTA is the high exposure to irradiation, although 256 slice-CT has decreased the radiation exposure for patients compared with 46 slice-CT or earlier design scanners. Increased radiation exposure with 46 slice-CT might be unacceptable when screening young individuals with suspected coronary anomalies [11].

The etiology of CAAs is not well known. There is no definite inheritance pattern and no sex predominance. In the previous studies, the most frequent coronary variant was multiple ostia due to the conus branch arising separately from the aorta, and separate ostia for the LAD and LCX (0.5–8% of the population) [10,12,13]; in our review, this variant was seen in 32 patients (0.98% of all patients). Variant coronary ostia were seen in 34 patients (1.52% of all patients), LMCA from non-coronary sinus of valsalva in 14 patients, RCA from non-coronary sinus of valsalva in nine patients, and high coronary ostia take off in 11 patients. Myocardial bridging is defined as an intramyocardial segment in the course of a major epicardial coronary artery, myocardial bridging can be seen as a normal variant, without clinical significance. It has been infrequently linked with ischemia, tachycardia-induced ischemia, conduction disturbances, myocardial infarctions, and even sudden cardiac death [7,14,15]. The bridging can be superficial or deep and is most commonly seen in the mid LAD (80%) [7,16]. Prevalence of myocardial bridging has been reported to vary from 0.5% to 16% in angiographic series, from 40% to 80% in postmortem studies [17], and 26% in CTA data [14]. In our review the incidence was 82.15% of all CAAs (8.859% of all included patients), with more involvement of the middle part of the LAD (163 out of 198 patients 82.3%). In anomalous origin of the coronary artery from opposite sinus, the coronary artery arises from the opposite sinus and then takes one of four paths: (1) an interarterial course (which carries a high risk of sudden cardiac death. Several pathologic processes have been implicated such as a narrow slit-like orifice, an acute angle of the ostium, and an intramural course.) (2) transseptal (subpulmonic); (3) retroaortic; and (4) prepulmonic (anterior to the Pulmonary Artery or right ventricle). The most common anomalous origin is the RCA arises from the left SV [18,19]. In our review, the anomalous origin of the RCA from the left SV was the most common anomaly (5 patients, 29.4%), and the anomalous origin of LMCA from the right SV is the next most common (4 patients, 23.52%); then anomalous origin of the LCX coronary artery from the right SV (4 patients, 23.5%). Anomalous origin of the LAD from the right SV was seen in 2 patients (11.76%). A single coronary artery is extremely rare, with only one coronary artery arising from a single ostium (0.0024–0.044% of the population) [8]. In our study, two patients had a single coronary artery (0.0894% of all patients).

CAF is defined as a direct precapillary connection between a branch of a coronary artery and other vascular structure [20,21]. CAFs have an estimated prevalence of 0.002% in the general population, but they are present in 0.05% to 0.25% of patients who undergo invasive coronary angiography [22]. CAF arises from the RCA in approximately 50% of patients, the left coronary artery in approximately 42% of patients, and both the RCA and left coronary artery in approximately 5% of patients [23]. There is no race or sex predilection for CAF. More than 90% of the fistulas drain into the venous circulation. The majority of adult patients are usually asymptomatic. The minority of symptomatic CAFs originates from the left coronary artery [24,25]. The clinical presentation of CAFs is mainly dependent on the severity of the left-to-right shunt. If symptoms come up, most patients present later in life with dyspnea and right ventricular enlargement or dysfunction. Other presentations include fatigue, orthopnea, chest pain, endocarditis, arrhythmias, stroke, myocardial ischemia, or myocardial infarction. In our study, CAFs were seen in six patients (2.489% of all CAAs, 0.2684% of all patients). The most common termination of CAFs was between the CAF and the Pulmonary artery or right atrium; one CAF was between LMCA and right atrium, which complicated with acute myocardial infarction due to blockage of the LCX by a thrombus extending from the thrombosed fistula (Fig. 1E, F), another fistula showed a complex anatomy with a connection between right atrium, Pulmonary artery, and LMCA and extra connection to Pulmonary artery at a higher level (Fig. 1H, I), this fistula with the remaining CAFs were asymptomatic.
Conclusions

The prevalence of CAAs in our study was similar to previous studies and the literature data. Most of CAAs are asymptomatic and has no clinical significance, but some can be symptomatic and even life threatening. The most common anomaly is myocardial bridging followed by anomalous of the ostia (separate ostia and variable location) and anomalous origin of the coronary artery. CTA is a noninvasive method and an excellent tool to diagnose and guide the management of CAAs. It enables excellent three-dimensional visualization and provides a clear delineation of the origin, course, relation to the adjacent structures, and termination compared with invasive coronary angiography. In the future, with attempts to reduce radiation dose, CTA can overcome the current limitation and may become the screening test of choice for CAAs.

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