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Relationship between significant coronary artery disease and coronary artery anomalies

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Aim: In the present study we aimed to investigate the frequency and types of coronary artery anomalies (CAAs) and their correlation with coronary artery disease (CAD).

Materials and methods: We assessed retrospectively the coronary angiography records of 8120 adult patients. We defined and classified the CAAs according to the classification system described by Angelini et al. Significant CAD was defined as the presence of angiographic coronary stenosis of >50% of the luminal diameter in at least 1 of the epicardial coronary arteries. Moreover, we compared the frequencies and features of the CAAs among CAD cases.

Results: We detected coronary artery anomalies in 3.32% and 64.4% of the patients with significant CAD. The percentage of anomalous coronary artery origin was significantly higher in CAD (–) patients than CAD (+) patients. The incidence of absence of left main coronary artery (LMCA) and the left anterior descending artery originating from the right sinus of Valsalva or the right coronary artery was significantly higher in CAD (–) patients than CAD (+) patients.

Conclusion: The development of coronary artery disease might be associated with coronary artery anomalies. In particular, the lack of LMCA seems to restrict the development of CAD.

Key words: Coronary artery anomaly, coronary artery disease

1. Introduction

Coronary artery disease (CAD) and its complications are the major cause of death worldwide (1–4). The diagnosis of CAD is made by the detection of luminal stenosis during coronary angiography in most of the patients. In general, CAD occurs when plaques build up in multiple segments of coronary arteries, which supply oxygen-rich blood to myocardium. Coronary artery anomalies (CAAs) are commonly isolated in congenital cases with no additional cardiac pathologies and they are accidentally encountered during coronary angiography, which is frequently employed to investigate myocardial ischemia (5–9). The majority of CAAs are benign in nature and do not cause hemodynamic deterioration; nevertheless, some of CAAs are shown to trigger myocardial ischemia, myocardial infarct, and sudden cardiac deaths through restricting the flow of oxygenated blood to the myocardium or altering flow dynamics of blood (10,11).

There are few studies assessing the relationship between coronary artery disease and coronary artery anomalies. In the present study, we therefore aimed to assess the role of CAAs in the progression of CAD.

2. Materials and methods

We evaluated retrospectively the angiography records of 8120 adult patients who received coronary angiography at our hospital between February 2007 and September 2011. The angiographic movies were reviewed by an interventional cardiologist for the types of CAA and the distribution of CAD. Presence of significant CAD was defined as the presence of angiographic coronary stenosis of >50% of the luminal diameter in at least 1 of the 3 coronary arteries. CAAs were classified according to the classification defined by Angelini et al. (12). We detected coronary arteries with 1) anomalous origin, 2) anomalous course (myocardial bridge [MB]), and 3) aberrant termination (coronary artery fistula [CAF]). The patients who had undergone a previous bypass surgery, with one or more totally occluded epicardial coronary artery, or with poor angiography quality were excluded from the present

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study. We determined the number of coronary arteries with anomalous origin, course, and termination and calculated the frequency of them among the CAA cases and the whole angiographic population. We then detailed the frequency of CAD (+) and CAD (–) cases with respect to the types of CAAs. We finally studied the incidence of CAD in normal and anomalous vessels of the patients with CAA. Overall, CAD (+) and CAD (–) patients were compared with respect to CAAs.

2.1. Statistical analysis

Continuous variables were expressed as means ± standard deviations; categorical variables were defined as percentages. To compare continuous variables, we used Student's t-test or the Mann–Whitney U test, where appropriate. Categorical variables were compared via the chi-square test. For all of the tests, $P < 0.05$ was considered to be statistically significant. SPSS 16.0 for Windows (SPSS Inc., Chicago, IL, USA) was used to perform all statistical calculations.

3. Results

The mean age of the patients whose digital angiography records were reviewed for the present study was 57.7 ± 13 years (range: 19–102 years). Of the patients, 73% were males and 27% were females. Overall, we detected CAAs in 270 cases (3.32%), and 64.4% of the patients had CAD. Representative coronary angiography images pertaining to normal and anomalous coronary arteries are illustrated in Figures 1 and 2, respectively. The sum of CAAs and their classification according to their anomalous origin, course, and termination are summarized in Table 1.

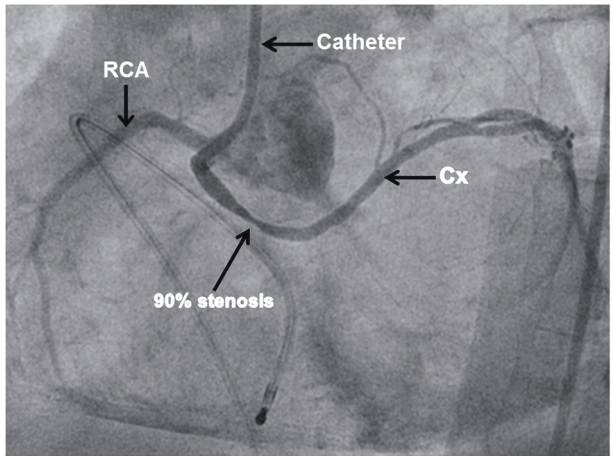

Figure 2. View of the anomalous Cx artery arising from the RCA; note that it is accompanied with CAD. CAD: RCA: the right coronary artery, Cx: circumflex artery.

We checked whether the presence of CAAs has a relationship with the development of CAD by comparing the CAD (+) and CAD (–) patients with CAAs. Our results revealed that the frequency of CAAs was markedly higher in CAD (–) patients than in CAD (+) patients (5.33% vs. 2.21%, $P < 0.01$). The incidences of CAFs and MBs were similar between CAD (–) and CAD (+) patients ($P > 0.05$). The percentage of anomalous coronary artery origins

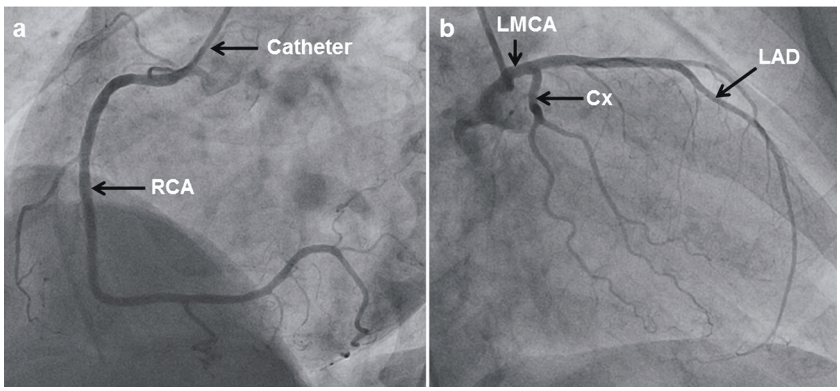

Figure 1. Angiography belonging to normal coronary arteries of the right and left systems (a and b). RCA: right coronary artery, LMCA: left main coronary artery, Cx: circumflex artery, LAD: left anterior descending artery.

Table 1. Total CAAs and their classifications according to types. CA: coronary artery, CAA: coronary artery anomaly.

	Number	Frequency among CAAs (n = 270) (%)	Frequency among the whole angiographic population (n = 8120) (%)
Sum of anomalous CAs	270	100	3.32
Anomalous CA origin	134	49.6	1.65
Myocardial bridge	109	40.4	1.34
Fistula	27	10	0.33

(ACAOs) was significantly elevated in CAD (–) patients than in CAD (+) patients (3.32% vs. 0.72%, P < 0.01). Moreover, akin to the increased frequency of the ACAOs, the lack of the left main coronary artery (LMCA) and the incidence of the left anterior descending artery (LAD) originating from the right sinus of Valsalva (RSV) or the right coronary artery (RCA) (for both P < 0.05) were noticeably higher in CAD (–) participants than in CAD (+) patients. Other ACAOs were comparable between the 2 groups. The comparisons of CAAs according to the presence or lack of CAD are summarized in Table 2.

In addition, we examined the distribution of CAD in normal and anomalous coronary arteries of the patients diagnosed with CAAs. The percentage rate for the presence of CAD was 58.7% in MBs, 51.9% in CAFs, and 27.8% in ACAOs, indicating that more than half of the MBs contained CAD. We further noted that 10 of the 15 patients with the lack of the LMCA had CAD in their anomalous coronary artery (the LAD and/or circumflex artery [Cx]). Of these, while 2 lesions were located at the proximal part of the anomalous coronary artery, 8 lesions were situated at the distal part of the anomalous coronary artery. The distribution of CAD in normal and anomalous coronary arteries is summarized in Table 3.

4. Discussion

Coronary artery anomalies are accidentally discovered during angiography or autopsies; however, their association with certain pathologies, e.g., coronary artery disease, has not been well studied. Although CAAs are rarely seen in

Table 2. The incidence of CAAs with regard to the presence of CAD. CAD (+): coronary artery disease-positive, CAD (–): coronary artery disease-negative, LMCA: left main coronary artery, RCA: right coronary artery, LSV: left sinus of Valsalva, Cx: circumflex artery, LAD: left anterior descending artery, RSV: right sinus of Valsalva, CA: coronary artery.

	CAD (+) (n = 5232)	CAD (–) (n = 2888)	P-value
Sum of anomalous CAs, n (%)	116 (2.21)	154 (5.33)	<0.001
Anomalous CA origin, n (%)	38 (0.72)	96 (3.32)	<0.001
Lack of LMCA, n (%)	15 (0.29)	64 (2.22)	<0.001
RCA from LSV, n (%)	4 (0.07)	8 (0.28)	0.19
RCA from Cx, n (%)	0 (0)	1 (0.03)	0.35
Cx from RSV or RCA, n (%)	11 (0.21)	8 (0.28)	0.24
LAD from RSV or RCA, n (%)	1(0.02)	5 (0.17)	0.02
Split RCA, n (%)	7 (0.13)	9 (0.31)	0.07
Single CA, n (%)	0 (0)	1 (0.03)	0.35
Myocardial bridge, n (%)	64 (1.22)	45 (1.56)	0.12
Fistula, n (%)	14 (0.27)	13 (0.45)	0.12

Table 3. Distribution of coronary artery disease in normal and anomalous coronary arteries. ACA: anomalous coronary artery, NCA: normal coronary artery, CA: coronary artery, LMCA: left main coronary artery, RCA: right coronary artery, LSV: left sinus of Valsalva, Cx: circumflex artery, LAD: left anterior descending artery, RSV: right sinus of Valsalva.

	Total	Lesion at ACA	Lesion at NCA	Lesion at ACA + NCA
Anomalous CA origin, n (%)	38	11 (29)	7 (18.4)	20 (52.6)
Lack of LMCA, n (%)	15	10 (66.7)	0 (0)	5 (33.3)
RCA from LSV, n (%)	4	0 (0)	0 (0)	4 (100)
RCA from Cx, n (%)	0 (0)	0 (0)	0 (0)	0 (0)
Cx from RSV or RCA, n (%)	11	1 (9.1)	2 (18.2)	8 (72.7)
LAD from RSV or RCA, n (%)	1	0 (0)	1 (100)	0 (0)
Split RCA, n (%)	7	0 (0)	4 (57.1)	3 (42.9)
Single CA, n (%)	0	0 (0)	0 (0)	0 (0)
Myocardial bridge, n (%)	64	9 (14.1)	35 (54.7)	20 (31.2)
Fistula, n (%)	14	4 (28.6)	6 (42.8)	4 (28.6)

coronary angiographies, their frequencies are interestingly considerably higher in the autopsies of persons who died suddenly. Particularly, the second most important cause of sudden death of young persons is CAA, after hypertrophic cardiomyopathy (13–15). Nevertheless, the relationship between the potentially lethal CAAs and the development of CADs has not been fully described so far; therefore, we planned to examine the possible relationship between the frequency of CAAs determined with angiography and CADs.

The frequency of CAAs is shown to range between 0.6% and 5.6% in the general population (7-9,12,16–19). In the present study, we detected CAAs in 3.32% of our study population (270 of 8120 patients). This rate is higher than the reports of previous studies, except for the work of Angelini et al. (12). The relatively higher frequency of the CAAs in the present study in comparison to other studies might stem from the methodological approaches and the classification system we used. Overall, we assessed anomalous origin, course, and termination of CAA throughout the study (12). In contrast, few of the previous studies evaluated all of these anomalies in the context of CAAs (8,19). In addition, we obtained the types of CAAs by watching the coronary artery angiography movies, but not researching angiographic records. Most of the studies examining the frequency of the CAAs have acquired their data from searching the angiographic records (7,9,17–19). The data generated from angiographic records as to the CAAs possess a number of drawbacks, some of which can be listed as follows: 1) since the CAAs are rarely encountered entities, the physician reporting them may fail to accurately recognize and describe them in the records; 2) because most of the CAAs are usually considered benign in nature, a variance to normal, the specialist may not register them at all times; 3) in most of the published studies, angiographic records were evaluated by various cardiologists whose criteria for the classification of the CAAs may well greatly vary, thereby yielding nonhomogeneous data concerning the CAAs. Further factors that may confuse the various frequencies of the CAAs can spring from geographic variations and genetic backgrounds. All of these parameters can play a role in the divergent range reported for the frequency of the CAAs in the previous studies and in the present study.

Moreover, two-thirds of the patients in the present study population possessed CAD. This markedly elevated rate of CAD may come mainly from the fact that the majority of these patients underwent angiography due to exploration of myocardial ischemia. The rate of CAD in the present study is consistent with those of earlier studies (7,9,20). The frequency of anomalous coronary artery course and termination was similar between CAD (+) and CAD (–) patients. In contrast, the percentage of anomalous coronary artery origins was significantly higher in CAD (–) patients than in CAD (+) patients. Furthermore, the absence of the LMCA and the incidence of the LAD originating from the RSV or the RCA were noticeably higher in CAD (–) participants than in CAD (+) patients. Other anomalous coronary artery origins were similar between the 2 groups. In this regard, previous studies have reported no relationship between the anomalous coronary artery origin and CAD (7–9).

The lack of the LMCA is considered a benign anomaly and is not reported to cause hemodynamic deterioration such as myocardial ischemia. In the present study, the most common anomalous coronary artery origins was the lack of the LMCA, both in CAD (+) and CAD (–) patients. The lack of the LMCA was considerably higher in CAD (–) patients than in CAD (+) patients (2.22% vs. 0.29%). In other words, it is probable that the lack of the LMCA might hinder the development of CAD. Most of the CADs build up owing to the occurrence of atherosclerosis, accumulating typically at the sites of bifurcations of the vessels. The alteration of laminar blood flow to turbulent blood flow and formation of low endothelial shear stress at bifurcation sites are critical factors for the development of endothelial damage and the buildup of atherosclerosis (21,22). The LAD and Cx arise directly from the aorta at the lack of the LMCA and a more laminar flow occurs through the course of the coronary arteries. In this case, the proximal parts of the LAD and Cx arteries seemed to be protected from the adverse effect of turbulent blood flow occurring at bifurcation sites. The observation of a lesion at the proximal parts of the LAD and Cx in only 2 of the patients with a lack of the LMCA further supports our remark here. To the best of our knowledge, the present study is the first study examining a potential association between the lack of the LMCA and the development of CAD. The report of Yamanaka and Hobbs is the largest study that has been performed so far regarding CAAs and indicates that the most common type of CAA is anomalous coronary artery origin at 0.41%; however, they did not investigate the relationship between the lack of the LMCA and CAD formation in their study (7). Since this type of CAA can create difficulty during angiographic intervention, awareness of the infrequent presence of this type of anomaly is critical for optimizing catheterization in the patients with this anomaly. Moreover, contrast injection into the left sinus Valsalva at the left anterior oblique to caudal projection provides the best view for making the distinction of absent LMCA during angiography. While the anterior direction of the catheter selectively visualizes the LAD, its posterior direction selectively envisages the Cx. Sometimes, distinguishing the absent LMCA angiographically from the too-short LMCA can be challenging. Occasionally, selective placement of the catheter in the LAD or Cx can lead to false assessment of an absent LMCA.

Moreover, the frequency of the LAD arising from the RSV or the RCA was higher in CAD (–) patients than in CAD (+) patients. This type of anomaly is also called type IV dual LAD. In this coronary artery anomaly, there are 2 LAD arteries, the first of which is short and the other is long. While the short LAD is the continuation of the LMCA and terminates at the proximal end of the anterior interventricular groove, the long LAD branches off the RCA or the RSV and ends at the apex of the left ventricle (23,24). Furthermore, this type of anomaly is benign in general and does not cause hemodynamic corrosion unless the LAD originating from the RCA or the RSV does not run between the aorta and the pulmonary trunk. The interarterial course (between the aorta and the pulmonary artery) of the LAD arising from the RCA or the RSV could trigger myocardial ischemia and myocardial infarction (25). Therefore, determining whether the abnormal LAD traces an interarterial course is critical for reducing such risk. The interarterial course of the abnormal LAD can be demonstrated via inserting a catheter to the root of the aorta and at the right ventricular outflow tract during angiography. However, we were unable to determine whether they coursed between the aorta and the pulmonary artery since our study was performed retrospectively. In their study, Chaitman et al. reported that the left coronary artery in 75% of the cases possessing this anomaly showed an interarterial course (26). In the present study, the LAD of 6 patients (0.07%) was arising from the RCA or the RSV. In this anomaly, interarterial course of the LAD between the aorta and pulmonary artery can be the reason for performing angiography for these patients by causing ischemia without the presence of CAD. The observation of the LAD not reaching the apex of the left ventricle during the angiography of the left coronary system should arouse physicians' suspicion regarding the presence of this anomaly. If enough attention is not paid, the short LAD can be assumed as an LAD totally occluded at its proximal portion. Consequently, this erroneous interpretation might lead to the diagnosing of the patient with a coronary artery disease and provoke the wrong treatment approach for the patient. Nonselective application of radiopaque substances at the RSV level during the coronary angiography can facilitate easy recognition of anomalous LADs.

MBs in coronary angiography are characterized with the narrowing of a coronary artery during systole and the returning to normal during diastole (milking effect). Blood flow creates stronger hemodynamic stress at the vessel wall in the presence of MB. This is reported to accelerate the accumulation of atherosclerosis at the proximal of the MB. However, conflicting results are reported regarding the effect of MB on the formation CAD. While some studies indicate a relationship between the MB and CAD, others do not point out such an association (27–29). Our current finding showed that the frequency of MB between CAD (–) and CAD (+) patients was similar, indicating that there is no marked causative relationship between the presence of MB and the development of CAD.

Although small coronary artery fistulas usually do not cause any hemodynamic compromise, the larger fistulae can cause coronary artery steal phenomenon, which leads to ischemia of the segment of the myocardium perfused by the coronary artery. The pathophysiologic mechanism of coronary artery fistula is myocardial stealing or reduction in myocardial blood flow distal to the site of the coronary artery fistula connection. In addition, a left-to-right shunt can lead to right heart failure and to the development of pulmonary hypertension (30). Few studies are available regarding the relationship between CAFs and CAD. In the present study, the frequency of CAFs was similar in CAD (–) and CAD (+) patients. However, we assume that coronary artery angiography is not very sensitive in showing fistulas. While the angiographies performed under high pressure would visualize a fistula as if it existed, the angiographies performed under low pressure would not envisage a fistula, as if it did not exist. Fistulas considered being hemodynamically active should be closed with percutaneous or surgical approaches (31).

We are aware that the most significant limitation to the present study is the use of a small patient population. CAAs are rarely encountered congenital anomalies among the general population; therefore, the present results remain to be further verified with future studies using larger populations. Another limitation to the present study is failing to include the analyses of the patients with one or more major epicardial coronary artery occlusions. These limitations should be considered when the present results are interpreted.

In conclusion, the development of coronary artery disease might be associated with CAAs. The present study showed that the lack of LMCA and the LAD arising from the RCA or the RSV were higher in CAD (–) patients than in CAD (+) patients. The present results indicate that CAA, particularly the lack of LMCA, seems to restrict the development of CAD.

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