Coronary artery anomalies: what we know and what we have to learn. A proposal for a new clinical classification

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The anatomic details and pathophysiological patterns of most coronary artery anomalies (CAAs) are presently well known. On the contrary, few data exist on the clinical relevance of different CAAs which necessitate a proper management and follow-up protocol. Clinical, invasive and interventional cardiologists often continue to encounter CAAs as incidental findings during routine diagnostic work up for other cardiac diseases and are sometimes unable to fit them into a specific pathophysiological context and a corresponding management protocol. In describing CAAs the authors have focused their attention on the clinical relevance in order to suggest a practical clinical classification based on four classes of clinical significance: I-benign, II-relevant (related to myocardial ischemia), III-severe (related to sudden death), IV-critical (association of classes II and III with superimposed coronary artery disease).

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Introduction

The anatomic details and pathophysiological patterns of most coronary artery anomalies (CAAs) are presently well known. On the contrary, few data exist on the clinical relevance of different CAAs which necessitate a proper management and follow-up protocol. The pathophysiological complexity, non-specific manifestations and unclear relationship with myocardial ischemia, sudden death and coronary artery disease (CAD) limit a comprehensive classification and management of such congenital heart diseases. In this brief review of the literature on the topic, the authors describe the anatomy, pathophysiology and clinical relevance of the most important CAAs and suggest a new possible clinical classification which could help both invasive and clinical cardiologists when assessing the risks, type of follow-up or surgical treatment.

What we know

Epidemiology and clinical manifestations. CAAs occur in 0.64 to 5.6% of patients undergoing coronary angiography[1-9].

Although patients with CAAs are usually asymptomatic, clinical symptoms may include angina pectoris, syncope, ventricular tachycardia, cardiac arrest, myocardial infarction and sudden death in the absence of manifest CAD, depending on the anatomo-morphologic abnormalities correlated with the pathophysiology underlying the specific anomaly (Table I).

Anatomy and pathophysiology. The anatomical details of almost all CAAs as well as the majority of the underlying pathophysiological characteristics are well known: the anomalies of the origin, course, and distribution and the relative pathophysiology are shown in table II.

Diagnosis. CAA detection techniques include color-flow Doppler echocardiography, especially in case of an ectopic origin of the left main or right coronary artery and of a coronary artery fistula[10]. Magnetic resonance and computed tomography imaging have recently been found to be very useful, non-invasive, and effective imaging techniques for the detection of major CAAs[11]. Selective coronary arteriography is the gold standard for the evaluation of CAAs and of their anatomic and morphologic definition prior to any treatment.

The differential diagnosis should include other coronary abnormalities such as atherosclerotic coronary ectasia and aneu-

Table I. Incidence of the most common coronary artery anomalies in the authors' experience and their possible clinical manifestations.

Coronary artery anomaly	Incidence (%)	Possible clinical manifestations
Separate origin of the LAD and LCx	0.31	–
Ectopic origin of the LCx from the RCA	0.25	–
Ectopic origin of the LCx from the right sinus	0.13	–
Myocardial bridge	0.11	UA, AMI, MA, SD
Ectopic origin of the LCA from right sinus	0.098	UA, AMI, MA, SD
Single coronary artery	0.098	SA, UA, SD
Atresic coronary artery	0.039	SI
Dual LAD type IV	0.039	–
Ectopic origin of the RCA from the left sinus	0.039	UA, AMI, MA, SD
Coronary artery fistula	0.039	HF, UA, AMI, SYC
Ectopic origin of the RCA from the PA	0.020	SD
Ectopic origin of the LCA from the PA	0.020	HF, SA, UA
Total	1.21	

AMI = acute myocardial infarction; HF = heart failure; LAD = left anterior descending coronary artery; LCA = left coronary artery; LCx = left circumflex coronary artery; MA = malignant arrhythmias; PA = pulmonary artery; RCA = right coronary artery; SA = stable angina; SD = sudden death; SI = silent ischemia; SYC = syncope; UA = unstable angina.

rysm, coronary lesions associated with Kawasaki and other arteritis[12], and iatrogenic coronary aneurysms and fistulas following percutaneous coronary interventions.

What we have to learn

Proposal for a new classification. In order to facilitate clinical management and follow-up and to standardize clinical communication, we propose a classification based on clinical relevance and including four classes as described in table III.

The aim of the following section is to describe each class.

Class I: benign coronary artery anomalies. The most frequent CAAs are usually clinically silent[7] and are unrelated *per se* to myocardial ischemia and sudden death, although they were often found to be associated with various degrees of atherosclerotic disease[13,14].

An ectopic coronary origin from the ascending aorta is suspected when the angiographer is unable to locate a coronary artery within the sinuses of Valsalva. This anomaly is normally benign, its presence being important only during cardiac surgery due to the danger of accidentally cross-clamping or transecting the vessel[15].

Intercoronary communication is a rare anomaly usually occurring in the absence of CAD and it may be mistaken for a functioning collateral vessel indicative of an unrecognized coronary obstruction. Normally, this CAA is totally benign.

A separate origin of the left anterior descending coronary and circumflex arteries is a relatively frequent anomaly[6]. Its incidence is increased in case of aortic valve disease and in case of dominance of the left coronary artery: this anomaly causes no hemodynamic impairment and should be considered "benign".

Similarly, an ectopic origin of the left circumflex coronary artery from the right coronary artery or right sinus of Valsalva, probably the most frequent CAA, can be considered benign and its relevance is related to the possibility of accidental compression during surgery for valve replacement.

A dual descending coronary artery, distinguished, in the classification of Spindola-Franco et al.[16], into the I to IV subtypes depending on the distribution of the septal and diagonal branches and on its course within the interventricular sulcus, is not associated with unfavorable events in the absence of coronary atherosclerosis[17,18].

Myocardial bridges are classifiable depending on their length (< 1 or > 1 cm) and luminal narrowing during milking (< 25, 50 or > 75%) with a score of 1 to 5, with a score of 5 being allocated to the most severe type[19]. Despite the possibility of episodic ischemia, myocardial bridges with a score < 4 are likely to be benign: in our series no patient with this anomaly developed serious cardiovascular event.

In the authors' series including about 100 CAAs, more than half of the patients had CAD of varying degrees: this high percentage probably depends on the high mean age of our population and it is thus probably best explained by referral bias. As reported in a previous study[13,14], patients with CAAs and without CAD had few cardiovascular events, suggesting the independence of the two phenomena: in our experience the presence of a benign coronary anomaly did not really influence survival which is probably only impaired by CAD.

CAAs may become more relevant when associated with other congenital heart diseases: tetralogy of Fallot[20], D-transposition of the great arteries[21] and pulmonary atresia with an intact ventricular septum[22] are not infrequently associated with CAAs and the success of surgical treatment partly depends on the preservation of the blood supply to the coronary circulation. For ex-

Table II. Anatomical and pathophysiological characteristics of the most important coronary artery anomalies.

Coronary artery anomaly	Anatomy	Pathophysiology
Separate origin of the LCx and LAD	LAD and LCx arise from adjacent separate ostia in the LS	No hemodynamic impairment
Ectopic origin of the LCx from the RS Ectopic origin of the LCx from the RCA	The LCx arises from the RS or proximal RCA posterior to the AO course	No hemodynamic impairment. Accidental compression during valve replacement
Ectopic coronary origin from the AO	Origin from the proximal 2 cm of the AO	Accidental cross-clamping or transection during surgery
Intercoronary communication	Contiguity of the AV branches of the RCA and LCx resulting in a bidirectional flow	May serve as collateral source in case of coronary obstruction
Dual LAD		
Type I	The short LAD gives off the septal branches, the long LAD runs in the AIVS	No hemodynamic impairment
Type II	The long LAD descends on the right ventricular side before reentering into the AIVS	Misinterpretation during bypass surgery
Type III	The long LAD travels intramyocardially in the ventricular septum	
Type IV	The long LAD arises from the RCA	
Atresic/hypoplastic coronary artery	Congenitally absent or hypoplastic LCx or LM	Fixed myocardial ischemia
Myocardial bridge	Intramyocardial tunneling of the epicardial coronary segments	Fixed and episodic myocardial ischemia; increased risk of CAD
Coronary artery fistula	Vessel arising from a coronary artery branch and draining into a single chamber	Fixed myocardial ischemia; right ventricular overload in case of drainage into the RV
Single coronary artery		
R	Ostium in the RS	Potential compression of the single coronary vessel if it passes between the aorta and the pulmonary artery with episodic ischemia, myocardial infarction and sudden death
L	Ostium in the LS	
I	Anatomical course of a normal RCA or LCA	
II	Single vessel arising from the proximal segment of the RCA or LAD	
III	LAD and LCx arise separately from the proximal normal RCA	
Ectopic origin of the LCA from the PA	Blood flows from the RCA and passes via collaterals to the LCA where it flows in a retrograde fashion into the PA	Fixed and episodic myocardial ischemia. Volume overload
Ectopic origin of the RCA from the PA	Flow from the LCA via collaterals into the RCA and retrograde into the PA	Congestive heart failure. Sudden death
Ectopic origin of LCA from the RS	LCA arises form the RS and passes anterior or posterior to the aorta or between the AO and PA or intramyocardially	Potential compression of the single coronary vessel if it passes between the AO and PA with episodic ischemia, myocardial infarction and sudden death
Ectopic origin of RCA form the LS	RCA arises from the LS and passes between the AO and PA or posterior	

AIVS = anterior intraventricular sulcus; AO = ascending aorta; CAD = coronary artery disease; LAD = left anterior descending coronary artery; LCA = left coronary artery; LCx = left circumflex coronary artery; LM = left main branch; LS = left sinus; PA = pulmonary artery; RCA = right coronary artery; RS = right sinus; RV = right ventricle.

Table III. Clinical relevance-based classification of coronary artery anomalies in the adult.

Class	Coronary artery anomalies
I-Benign	Ectopic origin of the LCx from the RS Separate origin of the LCx and LAD Ectopic origin of the LCx from the RCA Ectopic coronary origin from the AO Dual LAD type I-IV* Myocardial bridge (score ≤ 5)** Intercoronary circulation
II-Relevant	Coronary artery fistula Single coronary artery R-L, I-II-III, A-P§ Ectopic origin of the LCA from the PA Atresic coronary artery Hypoplastic coronary artery
III-Severe	Ectopic origin of the LCA from the RS Ectopic origin of the RCA form the LS Ectopic origin of the RCA from the PA Single coronary artery R-L, I-II-III B§ Myocardial bridge (score 5)**
IV-Critical	Class II and superimposed CAD Class III and superimposed CAD

AO = ascending aorta; CAD = coronary artery disease; LAD = left anterior descending coronary artery; LCA = left coronary artery; LCx = left circumflex coronary artery; LS = left sinus; PA = pulmonary artery; RCA = right coronary artery; RS = right sinus. * according to the classification of Spindola-Franco et al.[16]; ** according to Angelini et al.[19]; § according to the classification of Lipton et al.[28].

ample, as reported by Pasquali et al.[23], intramural coronary arteries are associated with an increased risk of perioperative death during repair of transposition of the great arteries.

Class II: coronary artery anomalies related per se to myocardial ischemia. Clinically relevant CAAs are usually correlated with fixed or episodic myocardial ischemia in the absence of CAD.

The presence of a coronary artery fistula is clearly correlated with the development of myocardial ischemia and congestive heart failure: a fistula draining into the right heart results in right ventricular volume overload[24,25].

An ectopic left coronary origin from the pulmonary artery was recognized to influence survival during childhood and up to 90% of affected children require prompt surgical correction[26]. In the adult this anomaly may be related to fixed myocardial ischemia, to an increased risk of CAD, to volume overload and to ischemic cardiomyopathy[27].

Single coronary arteries subtype R or L, I-II-III, A or P constitute single coronary arteries with an anterior or posterior route to the aorta and are involved in the development of myocardial ischemia[28]: in the authors' experience various degrees of myocardial ischemia may be observed when the single coronary artery becomes insufficient to support the coronary circulation. In our series single coronary arteries were of the R-II subtype and except one, all had a benign prognosis.

An atresic coronary artery is normally correlated with congenital syndromes such as Rubeolla syndrome, Hurler's syndrome, Friederich's ataxia syndrome and normally have a benign course, although they may result in myocardial ischemia[29,30].

CAAs related to fixed ischemia usually have a benign course and clinical management depends on the extent of CAD and other comorbidities.

Class III: association between coronary artery anomalies and sudden death. CAAs in origin, course and distribution have recently been suggested as the main cause of 5-35% of sudden death in young people[27], whereas an acute take-off angle and the presence of coronary ostial valve-like ridges have been proposed as important causes of sudden death in the absence of CAD[31-33]. Since 19% of sudden deaths in young athletes are due to CAAs[34], the association of class III CAAs and extreme exertion may be a catastrophic event and it should require prompt and precise recommendations.

Single coronary arteries of the L II-III B subtypes are a very rare condition in which the coronary circulation is totally supported by an anomalous right coronary artery originating from a normal left coronary artery and usually passing between the pulmonary artery and aorta constituting a potential risk[35,36].

The significance of an ectopic origin of the left coronary artery from the right sinus of Valsalva depends on the site of origin and on its anatomical relationship with the aorta and pulmonary artery: the "septal" subtype is the most common finding while the "between" one is rare but often dangerous[37]. In our experience this anomaly was associated with myocardial infarction and sudden death.

In case of an ectopic origin of the right coronary artery from the left sinus of Valsalva, the vessel passes between the aorta and pulmonary arteries. Owing to possible right coronary artery occlusion during expansion of the aorta, this anomaly is potentially dangerous[38,39]. In the authors' practice patients with this anomaly had clinical manifestations of ventricular malignant arrhythmias and were, in agreement with the caring physician, promptly operated without complications.

An anomalous origin of the right coronary artery from the pulmonary artery is extremely rare: its clinical course is usually benign but it can be related with sudden death[40,41]. No serious event was observed among the patients suffering from this anomaly in our series.

Myocardial bridges represent a clinical challenge: they are correlated with both fixed and episodic myocardial ischemia and with sudden death[42]: coronary spasm or intravascular clotting are likely to be due to added pathophysiological changes capable of exacer-

bating neurogenic or autocrine changes in the vessels. Myocardial bridges with a score > 5 are likely to cause myocardial infarction and sudden death.

Class IV: association between coronary artery anomalies and superimposed coronary artery disease. Although there are no studies which demonstrate that CAAs are more susceptible to CAD, a number of authors report some interesting observations. Samarendra et al.[43] found that an anomalous left circumflex coronary artery had an earlier and greater degree of atherosclerosis than non-anomalous vessels, although this predilection has been observed only for vessels arising from the right side and pursuing a retroaortic route.

As recently observed by Feldman et al.[44], although the coronary arteries are equally exposed to systemic risk factors, coronary atherosclerosis is focal and eccentric, and each lesion evolves independently. Variations in shear stress elicit markedly different humoral, metabolic, and structural responses in endothelial cells. In the great arteries, such as the carotid arteries and the aorta, areas of high fluid (acting perpendicularly to the vessel surface as pressure) shear stresses are thought to play the greatest role in promoting atherosclerosis[45]. On the contrary, in the coronary tree, areas of low mechanical shear stress (acting tangentially to the vessel surface) promote atherosclerosis, whereas areas of high flow shear stress prevent this disease. This evidence should be stronger in the presence of CAAs in which acute take-off angles or flow turbulence due to an increased blood flow may contribute to the endothelial injury and to the development of CAD.

In their series of 24 959 patients, Click et al.[4] were the first to suggest that an anomalous circumflex coronary artery had a greater degree of stenosis than non-anomalous arteries but that the location and degree of stenosis in the anomalous artery did not influence survival. Similarly, Garg et al.[8] and Topaz et al.[5] respectively found an incidence of 28-33% of CAD in the anomalous arteries and an overall incidence of CAD in patients with CAA of 66-68%, suggesting that these two conditions are independent.

Clinical management. As written by Angelini et al.[34], angiographic documentation of CAAs has not led to any effective or widely agreed-upon recommendations for functional testing, exercise tests or nuclear imaging examination. This has often led to false-negative or confusing results. Moreover, because some CAAs may manifest only under exceptional conditions such as extreme exertion, probably only long-term Holter monitoring might be informative. To identify coronary spasm related to some CAAs an ergonovine or acetylcholine challenge may be essential. Intravascular ultrasound may help during evaluation of coronary spasm.

Similarly, no established recommendations have been made for treatment: benign CAAs without CAD probably do not need any pharmacological treatment; a careful follow-up may help good decision-making for class II CAAs, whereas prompt correction is probably required for class III and IV CAAs only.

Conclusions and a proposal for a new classification

CAAs constitute a highly variable and complex phenomenon. Probably most CAAs do not impact significantly on the patient's survival and clinical history but this practical classification may be useful. Obviously, further studies are required to assess the real effectiveness of the new classification but for the time being the suggested classification may help clinical cardiologists who are not involved in coronary arteriography to rapidly familiarize themselves with the management of most CAAs and address them with appropriate follow-up and treatment.

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