Clinical Case Report

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Anomalous right coronary artery in a middle-aged patient

A case report and review of the literature

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Abstract

Background: An anomalous right coronary artery originating from the left sinus of Valsalva is a rare, but often incidental, finding in middle-aged to elderly people. Prevalence is difficult to define, as well as determining potential harmful hemodynamic consequences. Moreover, the optimal treatment remains debatable.

Case summary: The authors present a case of a middle-aged patient diagnosed with an anomalous right coronary artery causing ischemia, who was treated surgically.

Conclusion: By reviewing literature, the authors conclude that choice of treatment depends on age, symptoms, and certain anatomic features of this anomaly. However, there are no randomized trials available in this field.

Abbreviations: ALCA = anomalous left coronary artery, ARCA = aberrant right coronary artery, CA = coronary angiography, CMP = cardiac magnetic resonance, ECG = electrocardiogram, IVUS = intravascular ultrasound, MACE = major adverse cardiac event, MDCT = multidetector computed tomography, MRA = magnetic resonance angiography, RCA = right coronary artery, RVOT = right ventricular outflow tract, SCD = sudden cardiac death, SPECT = single photon emission computed tomography.

Keywords: aberrant right coronary artery, ARCA, case report, coronary anomaly, sudden cardiac death

1. Introduction

In middle-aged to elderly patients, a coronary anomaly is often an incidental finding. The clinical presentation varies from asymptomatic up to presentation as an acute myocardial infarction or even sudden cardiac death (SCD). Coronary anomalies are the second most frequent cause of SCD in young athletes.[1,2]

Many coronary anomalies are truly benign. Nevertheless, in a large group of anomalies, it is difficult to determine if and how dangerous they are. There is no strict consensus about which treatment should be recommended.

We present a case of an aberrant right coronary artery (ARCA) and reviewed the literature concerning the best diagnostic and therapeutic strategy.

Editor: Manal Elshmaa.

Patient consent was not given due to practical reasons since follow-up takes place in a referring center. Neither institutional review board nor ethics approval was asked for this case report.

The authors have no funding and conflicts of interest to disclose.

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Medicine (2016) 95:49(e5508)

Received: 1 July 2016 / Received in final form: 1 November 2016 / Accepted: 8 November 2016

http://dx.doi.org/10.1097/MD.0000000000005508

2. Case

A 40-year-old male farmer attended the outpatient clinic complaining of chest pain during work. When he tilted heavy straw bales he felt a piercing and oppressive left-sided thoracic pain, which ceased at rest. The episodes tended to increase in frequency. Syncope or dyspnea was present.

Cardiovascular risk factors consisted of a familial predisposition for coronary artery disease (father had coronary bypass at age of 65) and hypercholesterolemia. His medical history was uneventful.

Physical examination was unremarkable. Resting electrocardiogram (ECG) showed a regular sinus rhythm at 65 bpm, without further abnormalities.

During his first visit, a treadmill exercise test and echocardiography were performed. During the maximal treadmill test (Fig. 1) up to 200 W, he mentioned the chest pain, he felt when working and tilting heavy weights. This was associated with ST depression up to 1.5 mm in leads V5 and V6 and some solitary monomorphic premature ventricular complexes. Echocardiography (Fig. 2) revealed a normal left and right ventricular function with normal function of the heart valves. The right ventricle showed a mild degree of hypertrabeculation.

A further diagnostic work-up was initiated with stress echocardiography and cardiac magnetic resonance (CMR).

Supine bicycle stress echocardiography once again provoked recognizable chest pain, electrocardiographic ST depression in leads V5 to V6, and revealed severe hypokinesia in the inferoposterior region. A coronary angiography (CA) was subsequently planned.

Angiography revealed nonsignificant atherosclerosis in the 3 main coronary arteries in a right dominant coronary system. However, an anomalous take off of the right coronary artery (RCA) in the left sinus of Valsalva was diagnosed (Fig. 3).

CMR confirmed some hypertrabeculation of the right ventricle, without signs of arrhythmogenic right ventricular

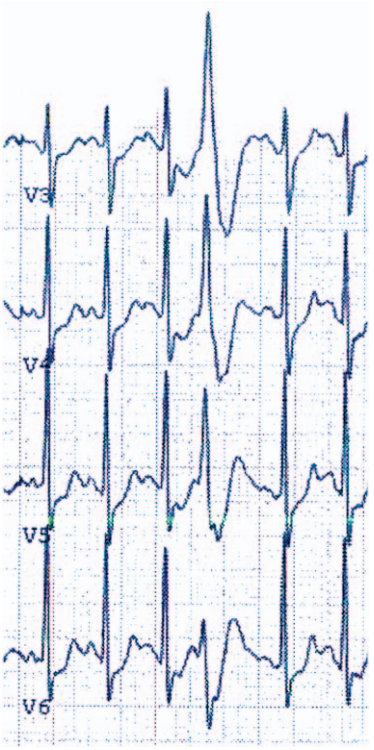

Figure 1. Treadmill test showing 1.5 mm ST-segment depression in leads V5 to V6 and a solitary ventricular extrasystole.

dysplasia or noncompaction syndrome. The left and right ventricular function was preserved and no delayed enhancement or myocardial fibrosis was visualized. This imaging technique confirmed the aberrant origin of the RCA, originating from the left coronary cusp and with a course of the proximal RCA between the pulmonary artery and aorta.

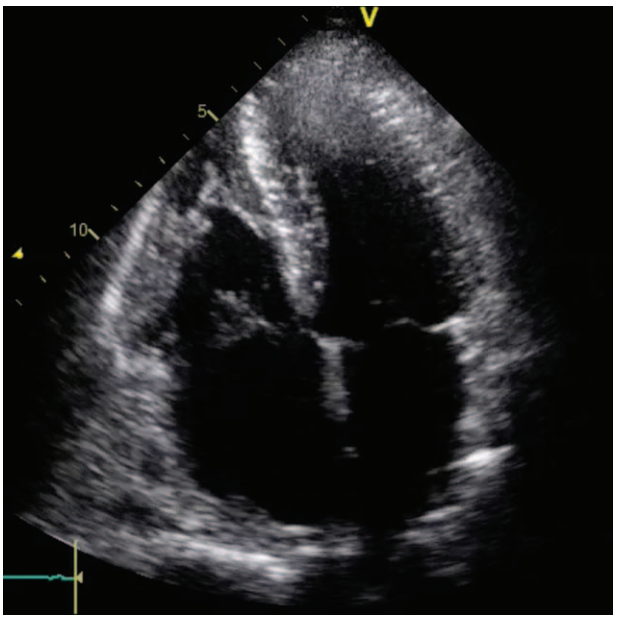

Figure 2. Transthoracic echocardiography apical 4-chamber view showing normal left ventricle function, and moderate right ventricle hypertrabeculation.

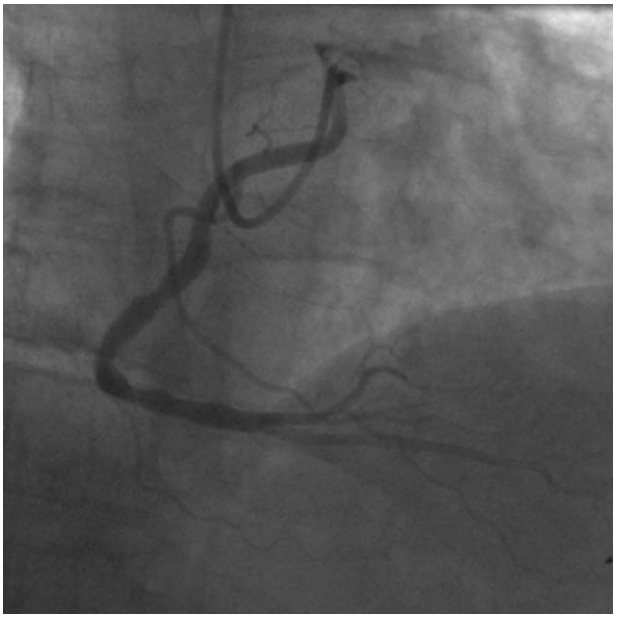

Figure 3. Coronary angiography showing the anomalous origin of the right coronary artery from the left sinus of Valsalva.

In conclusion, our patient presented with recurrent stable angina and only an ARCA could explain the symptoms. This was clearly confirmed by the abnormal myocardial ischemia tests (treadmill exercise ECG and stress echocardiography) in the territory of the RCA. The case was debated in the heart team, and the patient was referred for a surgical correction.

Uneventful reimplantation of the RCA was performed. Medication at the time of discharge included low-dose aspirin, a statin, and a beta-blocker. After 4 weeks the patient was already slowly resuming daily life, including his work as a farmer.

Four months after the operation, the patient consulted because of exertional shortness of breath (New York Heart Association Class II). There were no obvious signs of ischemia during exercise treadmill test. Coronary multidetector computed tomography (MDCT) (Fig. 4) showed a patient reimplanted RCA without significant coronary artery disease. Repeat stress echocardiography was normal, confirming the treadmill and MDCT scan findings. Cardiac rehabilitation was prescribed. During all follow-up visits, the patient remained asymptomatic and 5 years after presentation there were no cardiac events.

3. Discussion

Several forms of anatomical variants in coronary arteries exist. Some are believed to be potentially dangerous, others are benign.

Potential malignant coronary artery anomalies are an ectopic coronary origin from the pulmonary artery, an ectopic coronary origin from the opposite sinus of Valsalva, a single coronary artery, and large coronary fistulae or muscular bridging.[3]

3.1. Aberrant right coronary artery

The proximal course of an ARCA may vary: preaortic, interarterial (between aorta and pulmonary artery, as in our patient), retrocardiac, retroaortic, intraseptal, and precardiac (prepulmonary).

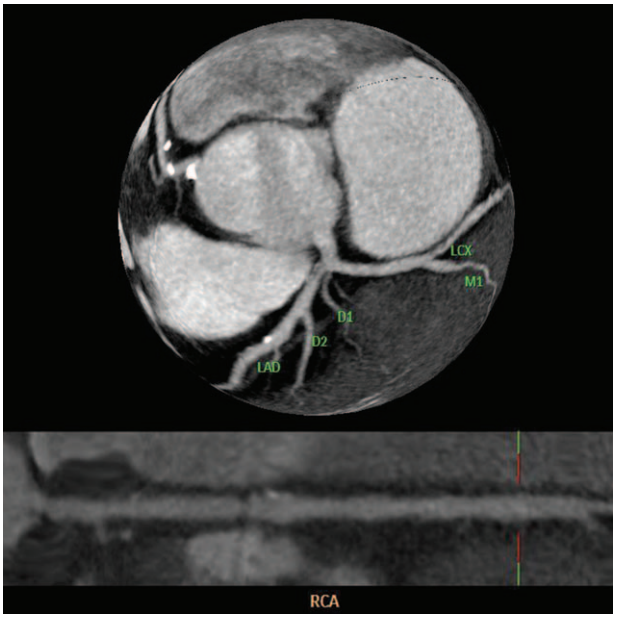

Figure 4. Multidetector computed tomography of coronary arteries after reimplantation of right coronary artery.

The hemodynamic important variant (i.e., interarterial course) runs between the aorta and pulmonary artery, either intramural (within the aortic wall) or separated from the aortic wall (like 2 distinct arteries). It is supposed that only this interarterial course can cause symptoms or is potentially dangerous.[4]

With newer imaging techniques, newer anatomical variants of the proximal course of an ARCA were distinguished. The coronary takeoff can be high (above the level of the pulmonary valve with a course between the aorta and pulmonary artery) or low (below the level of the pulmonary valve with a course between the aorta and right ventricular outflow tract [RVOT]). This high takeoff variant is believed to be more hostile than an ARCA with low takeoff.[5] Post hoc analysis of the CMR images of our case, revealed a low takeoff.

3.2. Prevalence and mortality

During CA studies, the incidence of an ARCA ranges from 0.09% to 0.92%.[6–8] An ARCA has a higher incidence than an anomalous left coronary artery (ALCA) (0.02–0.15%) and is presumed to be the most common type of hemodynamically significant coronary anomalies.[9,10]

In MDCT coronary studies, the incidence of an ARCA is comparable (0.54%).[5]

Among 18,950 autopsy cases in a Los Angeles hospital, 54 cases of coronary anomalies were detected of whom 39 with anomalies of the coronary ostia, totaling to an incidence of 0.206%.[11] In young athletes, 12% of SCDs is caused by an anomalous coronary artery from the opposite aortic sinus.[12] In young athletes, coronary anomaly is the second most common cardiac cause of SCD. However, with increasing age of athletes, coronary anomaly becomes a less frequent cause of SCD.[13]

Mortality data derived from autopsy studies (risk on SCD in ARCA 0–57%, in ALCA 30–100%) are far too high when converted to the general population. These numbers can obviously not be extrapolated from autopsy studies to the general population and are probably severely overestimated as mortality rates are biased as we are not aware of the exact prevalence of an ARCA in asymptomatic patients.[14,15] Notwithstanding, it is assumed that there is a 3- to 6-fold increased risk for SCD in people with an ARCA doing physical activity.[14]

3.3. Pathophysiology

The exact pathophysiological mechanism remains unclear. There are several hypotheses: a slit-like orifice caused by an acute angle of takeoff, which could cause reduced coronary flow during exercise[16,17]; compression of the interarterial segment caused by systolic compression between the aorta and pulmonary artery, aggravated during increased flow, for example, with exercise; ventricular arrhythmias caused by ischemia; acute or repetitive ischemia provoking myocardial fibrosis or reperfusion; intramural proximal intussusception of the anomalous artery at the aortic-root wall, as proposed by Angelini et al during intravascular ultrasound (IVUS) studies.[18] Presumably it is even a combination of these different mechanisms. On the other hand, studies showed that a coronary anomaly is not associated with an increased risk for development of coronary atherosclerosis.[19]

It would be interesting to identify specific anatomical or clinical risk factors for SCD in order to predict the possible hemodynamic significance. Logically, the dominance of an anomalous coronary artery is an important risk factor.[20] In a large pathology study of 30 cases of anomalous arteries, it was not possible to identify a certain anatomical feature related to increased mortality.[21] In an MDCT study, clinical symptoms were not related to the relative luminal narrowing nor angle of takeoff.[17,22] However, a significant difference in the prevalence of major adverse cardiac events (MACE) and typical angina was observed in a retrospective review of 22,925 consecutive MDCT scans, in which 124 cases with an interarterial anomalous coronary artery were found. They differentiated the anatomical takeoff in a high and a low takeoff (above or below the level of pulmonary valve). The group with high takeoff (coursing between aorta and pulmonary artery) proved to have a significant higher prevalence of typical angina (43% vs 6%, $P=0.001$) and MACE (28% vs 6%, $P=0.012$) compared to those with a low takeoff (coursing between aorta and RVOT). Age is an important clinical parameter that is related with the risk of SCD. Under the age of 30, there is an increased risk for SCD in patients with a coronary anomaly. This risk decreases with age.[23] It has been hypothesized that the aortic wall stiffens with age, which reduces compression.[10,21] As mentioned above, exercise is a risk factor for SCD in people with an ARCA.[14]

3.4. Clinical presentation and diagnosis

In a necropsy study of 242 patients who died suddenly, 62% of the patients were asymptomatic until the event.[23]

In a Japanese review of 56 patients with coronary anomalies of whom 44 with an ARCA and with a mean age of 55.9 years old, clinical patterns were reviewed. Of 22 patients with an ARCA who had undergone a treadmill test, 10 proved abnormal. Three out of 4 had a positive myocardial perfusion single photon emission computed tomography (SPECT) exercise test. Two patients even suffered ventricular tachycardia during stress testing.[24] These latter results show a much higher rate of positive stress testing than seen in other studies. Stress testing is often inadequate to identify ischemia. This is why a negative stress test

does not exclude a potentially dangerous coronary anomaly.[25] When a stress test proves negative but symptoms are suspicious (e.g., exertional syncope or chest pain) anatomical examinations (i.e., MDCT scan) should be considered.

In young patients, or echogenic patients, a transthoracic echocardiography could be useful in determining the proximal coronary origin.[26] One study showed significant 2D strain impairment in 25 patients with a coronary anomaly (of whom 15 with ARCA), suggesting subtle left ventricular contractility disorder in these patients.[27]

One study evaluated the accuracy of coronary artery calcium scanning for detecting coronary anomalies, which was found out to have a great diagnostic accuracy.[28] However, nowadays, MDCT CA is accepted as the "gold standard" for the evaluation of coronary anomalies.[29]

Magnetic resonance angiography (MRA) is similar successful, but the identification of the distal coronary course can be more difficult.[30] Some disadvantages of MRA are also to be considered: availability is less, MRA is not useful in patients with pacemakers or claustrophobia, and total study time takes much longer. One large benefit of MRA is that it can assess and locate scarred tissue and viability in the course of the affected artery, which could have an important prognostic value.[31]

A conventional CA is an invasive test. During CA it is not always possible to define the exact proximal anatomical course of the coronary anomaly. In a CA study, only in 53% of the cases the exact proximal course could be defined.[32] IVUS showed in some studies intussusception or lateral compression during systole, aggravated by saline, atropine, or dobutamine infusion.[10,33] Fractional flow reserve (FFR) was tested by Angelini et al, and showed results within normal limits during adenosine provocation (FFR $>$ 0.9, cutoff 0.8),[10] indicating that FFR is not a good parameter to diagnose hemodynamic significance in this setting.

Stress-rest myocardial perfusion SPECT can be used to detect reversible perfusion defects,[34] although results are often negative, as it is for stress ECG.

Long-term Holter monitoring can be helpful in screening for arrhythmia, although being an aspecific tool.

In summary, MDCT CA seems to be the best examination for anatomical diagnosis and ischemic testing proves often negative.

3.5. Treatment

The 2008 American Heart Association guidelines for the management of adults with congenital disease,[35] recommend that surgical coronary revascularization should be performed when there is evidence of ischemia in an anomalous RCA coursing between aorta and pulmonary artery (level of evidence class I, B).

There are no prospective studies available to determine the best treatment option. Indication to treat should be individualized, and depends on age, symptoms, the anatomical variant, and ischemic testing.

In literature, patients are separated by age (younger and older than 35 to 40 years old). New anatomical understandings separate high and low coronary takeoff of the ARCA (above or under level of pulmonary valve) as a possible prognostic finding.

Lee et al suggest to operate all patients, younger than 40 years old, with high takeoff, regardless of symptoms. Symptomatic patients older than 40 years old and with high takeoff should be operated if symptomatic, following their ideas. In patients with a low takeoff, close observation is suggested, unless when related symptoms are present surgery might be considered.[5]

In a Japanese retrospective study of 56 patients (mean age 55.9 years old), with anomalous origin of coronary artery (78% ARCA) and without coexisting atherosclerosis, a conservative treatment was applied (nitrates, calcium channel blockers, beta-adrenergic antagonists, or antiarrhythmic drugs).[24] During follow-up (2 months to 14.5 years), there were no cardiac-related deaths. The authors concluded that the prognosis of middle-aged to elderly patients with an anomalous origin of the coronary artery is relatively good. A few other studies proved comparable results, suggesting a conservative approach (beta-blockers and physical restriction) may be safe.[13,36,37]

This somehow conflicting evidence illustrates that further research is warranted, matching anatomical with clinical and functional test data.

3.6. Invasive management

A percutaneous approach has been applied in the past. One case series of 14 patients with proximal coronary artery stenting showed a normalization of stress test results and angiographical patency 6 months after the percutaneous coronary intervention was demonstrated.[38]

There are also different surgical strategies: reimplantation of the RCA (as in our case), unroofing of the intramural course with creation of a neo-orifice, the modified unroofing technique, patch augmentation, and classical bypass grafting. With patch augmentation, the proximal interarterial course is not relieved. Bypass grafting demonstrated graft failure because of competitive flow.[39,40]

The unroofing technique is mostly applied in children. Unroofing of the anomalous artery can be applied when the proximal course runs intramural, and when there is no involvement of aortic valve commissures, which otherwise could create aortic insufficiency. This technique relieves the ostial stenosis, creates a large neo-orifice, and removes the intramural segment. In the modified unroofing technique, the anomalous orifice is closed, a neo-orificium is created in the appropriate sinus, without extensive unroofing of the proximal intramural part of the anomalous coronary artery. This technique is used to avoid aortic regurgitation when there is commissural involvement.

4. Conclusion

The interarterial form of an anomalous RCA (coursing between the aorta and pulmonary artery) can lead to symptoms or even SCD. A high takeoff of the ARCA is a high-risk anatomical feature.

The incidence of an ARCA varies from 0.09% to 0.92%. Coronary anomaly is the second most common cause of cardiac sudden death in young athletes, which demonstrates that young age and vigorous exercise are risk factors for sudden death in patients with an ARCA.

Coronary MDCT is the gold standard for anatomical diagnosis. Further ischemic testing can help to guide the therapy strategy, although functional tests often prove negative.

In middle-aged patients, this anomaly is often an incidental finding. Notwithstanding the lack of randomized trials, we consider it advisable to operate when an ARCA is seen in young patients ($<$40 years old) or, in older patients with proven related symptoms or positive ischemic testing. Reimplantation of the RCA or unroofing the proximal course of the anomalous artery seems to be the best surgical strategies. When a conservative approach is proposed, avoidance of vigorous exercise and prescribing beta-blockers are advised.

Nevertheless, further research is warranted to determine the optimal treatment strategy.

Acknowledgment

The authors thank to Kaoru Tanaka, MD, PhD, and Daniele Plein, MD, for delivering images.

References

[1] Maron BJ, Thompson PD, Puffer JC, et al. Cardiovascular preparticipation screening of competitive athletes: a statement for health professionals from the sudden death committee (clinical cardiology) and congenital cardiac defects committee (cardiovascular disease in the young), American Heart Association. Circulation 1996;94:850–6.
[2] Maron BJ, Epstein SE, Roberts WC. Causes of sudden death in competitive athlete. J Am Coll Cardiol 1986;7:204–14.
[3] Yamanaka O, Hobbs RE. Coronary artery anomalies in 126,595 patients undergoing coronary arteriography. Cathet Cardiovasc Diagn 1990;21:28–40.
[4] Angelini P, Velasco JA, Flamm S. Coronary anomalies: incidence, pathophysiology, and clinical relevance. Circulation 2002;105: 2449–54.
[5] Lee HJ, Hong YJ, Kim HY, et al. Anomalous origin of the right coronary artery from the left coronary sinus with an interarterial course: subtypes and clinical importance. Radiology 2012;262:101–8.
[6] Ayalp R, Mavi A, Serçelik A, et al. Frequency in the anomalous origin of the right coronary artery with angiography in a Turkish population. Int J Cardiol 2002;82:253–7.
[7] Angelini P. Coronary Artery Anomalies: A Comprehensive Approach. Philadelphia: Lippincott Williams & Wilkins, 1999; 4:50–53.
[8] Yuksel S, Meric M, Soylu K, et al. The primary anomalies of coronary artery origin and course: a coronary angiographic analysis of 16,573 patients. Exp Clin Cardiol 2013;18:121–3.
[9] Garg N, Tewari S, Kapoor A, et al. Primary congenital anomalies of the coronary arteries: a coronary angiographic study. Int J Cardiol 2000;74:39–46.
[10] Angelini P. Coronary artery anomalies—current clinical issues. Tex Heart Inst J 2002;29:271–8.
[11] Alexander RW, Griffith GC. Anomalies of the coronary arteries and their clinical significance. Circulation 1956;14:800–5.
[12] Maron BJ, Haas TS, Murphy CJ, et al. Incidence and causes of sudden death in U.S. college athletes. J Am Coll Cardiol 2014;63:1636–43.
[13] Finocchiaro G, Papadakis M, Robertus JL, et al. Etiology of sudden death in sports. Insights from a United Kingdom Regional Registry. JACC 2015;67:2108–15.
[14] Penalver JM, Mosca RS, Weitz D, et al. Anomalous aortic origin of coronary arteries from the opposite sinus: a critical appraisal of risk. BMC Cardiovasc Disord 2012;12:83.
[15] Wren C, O'Sullivan JJ, Wright C. Sudden death in children and adolescents. Heart 2000;83:410–3.
[16] Virmani R, Chun PK, Goldstein RE, et al. Acute takeoffs of the coronary arteries along the aortic wall and congenital coronary ostial valvelikeridges: association with sudden death. J Am Coll Cardiol 1984; 3:766–71.
[17] Zhang LJ, Wu SY, Huang W, et al. Anomalous origin of the right coronary artery originating from the left coronary sinus of Valsalva with interarterial course: diagnosis and dynamic evaluation using dual-source computed tomography. J Comput Assist Tomogr 2009;33:348–53.
[18] Angelini P. Coronary artery anomalies: an entity in search of an identity. Circulation 2007;115:1296–305.
[19] Topaz O, DeMarchena EJ, Perin E, et al. Anomalous coronary arteries: angiographic findings in 80 patients. Int J Cardiol 1992;34:129–38.
[20] Kragel AH, Robers WC. Anomalous origin of either the right or left main coronary artery from aorta with subsequent coursing between aorta and pulmonary trunk analysis of 32 necropsy cases. Am J Cardiol 1988;62:177–87.
[21] Taylor AJ, Byers JP, Cheitlin MD, et al. Anomalous right or left coronary artery from the contralateral coronary sinus: "high-risk" abnormalities in the initial coronary artery course and heterogeneous clinical outcomes. Am Heart J 1997;133:428–35.
[22] Lee BY, Song KS, Jung SE, et al. Anomalous right coronary artery originated from left coronary sinus with interarterial course: evaluation of the proximal segment on multidetector row computed tomography with clinical correlation. J Comput Assist Tomotgr 2009;33:755–62.
[23] Taylor AJ, Rogan KM, Virmani R. Sudden cardiac death associated with isolated congenital coronary artery anomalies. J Am Coll Cardiol 1992;20:640–7.
[24] Kaku B, Shimizu M, Yoshio H, et al. Clinical features of prognosis of Japanese patients with anomalous origin of the coronary artery. Jpn Circ J 1996;60:731–41.
[25] Basso C, Maron BJ, Corrado D, et al. Clinical profile of congenital coronary artery anomalies with origin from the wrong aortic sinus leading to sudden death in young competitive athletes. J Am Coll Cardiol 2000;35:1493–501.
[26] Juredidini SB, Marino CJ, Singh GK, et al. Aberrant coronary artery: a reliable echocardiographic screening method. J Am Soc Echocardiogr 2003;16:756–63.
[27] Sahin ST, Uurdakul S, Cengiz B, et al. Subclinical left ventricular systolic dysfunction in patients with coronary artery anomaly: a speckle tracking and velocity vector imaging-based study. JACC 2013;62:C208.
[28] Von Ziegler F, Pilla M, McMullan L, et al. Visualization of anomalous origin and course of coronary arteries in 748 consecutive symptomatic patients by 64-slice computed tomography angiography. BMC Cardiovasc Disord 2009;9:54.
[29] Lee HJ, Kim YJ, Hur J, et al. Coronary artery anomalies: detection on coronary artery calcium scoring scan. AJR Am J Roentgenol 2010;194: W382–7.
[30] Taylor AM, Thorne SA, Rubens MB, et al. Coronary artery imaging in grown up congenital heart disease: complementary role of magnetic resonance and X-ray coronary angiography. Circulation 2000;101: 1670–8.
[31] Mavrogeni S, Spargias K, Karagiannis S, et al. Anomalous origin of right coronary artery: magnetic resonance angiography and viability study. Int J Cardiol 2006;109:195–200.
[32] Kim SY, Seo JB, Do KH, et al. Coronary artery anomalies: classification and ECG-gated multi-detector row CT findings with angiographic correlation. Radiographics 2006;26:317–33.
[33] De Oliveira DM, Gomes V, Caramori P. Intravascular ultrasound and pharmacological stress test to evaluate the anomalous origin of the right coronary artery. J Invasive Cardiol 2012;24:E131–4.
[34] Uebleis C, Groebner M, von Ziegler F, et al. Combined anatomical and functional imaging using coronary CT angiography and myocardial perfusion SPECT in symptomatic adults with abnormal origin of a coronary artery. Int J Cardiovasc Imaging 2012;28:1763–74.
[35] Warnes CA, Williams RG, Bashore TM, et al. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines on the Management of Adults With Congential Heart Disease). Developed in Collaboration With the American Society of Echocardiography, Heart Rhythm Society, International Society for Adult Congenital Heart Disease, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. J Am Coll Cardiol 2008;52:e143–263.
[36] Ouali S, Neffeti E, Sendid K, et al. Congenital anomalous aortic origins of the coronary arteries in adults: a Tunisian coronary arteriography study. Arch Cardiovasc Dis 2009;102:201–8.
[37] Brothers JA, Gaynor JW, Jacobs JP, et al. The registry of anomalous aortic origin of the coronary artery of the Congenital Heart Surgeons's Society. Cardiol Young 2010;20:50–8.
[38] Doorey AJ, Pasquale MJ, Lally JF, et al. Six-month success of intracoronary stenting for anomalous coronary arteries associated with myocardial ischemia. Am J Cardiol 2001;86:580–2.
[39] Fedoruk LM, Kern JA, Peeler BB, et al. Anomalous origin of the right coronary artery: right internal thoracic artery to right coronary artery bypass is not the answer. J Thorac Cardiovasc Surg 2007;133:456–60.
[40] Reul RM, Cooley DA, Hallman GL, et al. Surgical treatment of coronary artery anomalies: report of a 37½-year experience at the Texas Heart Institute. Tex Heart Inst J 2002;29:229–307.