CASE REPORT

UDC: 616.12-07:616.132.2
https://doi.org/10.2298/10.2298/VSP180314109B

Primary percutaneous intervention of the right coronary artery in a setting of anomalous origination of left coronary artery from the opposite sinus of Valsalva

Primarna perkutana intervencija desne koronarne arterije u okolnostima anomalnog porekla leve koronarne arterije iz suprotnog sinusa Valsalva

Rade Babić*†, Goran Grujić*, Dejan Kojić*, Jelena Kostić†‡, Zoran Trifunović‡, Saša Borović*†

***Dedinje Cardiovascular Institute, Belgrade, Serbia; University of Belgrade, †Faculty of Medicine, Belgrade, Serbia; ‡Military Medical Academy, Belgrade, Serbia**

Abstract

Introduction. Coronary artery anomalies are infrequent but anticipated findings during percutaneous coronary interventions (PCI). Compared to consistent reporting in angiographic series, they seem to be underreported in interventional studies, and particularly in the setting of primary PCI, where their prompt recognition is of the utmost importance. **Case report.** We present a 50 years old male with inferior ST-elevation of myocardial infarction (STEMI) and right ventricular involvement with solitary ostium for all three coronary arteries in the right aortic sinus of Valsalva. The patient had an extremely rare variant of coronary artery origination belonging to the type A4b2c2 of Angelini's classification. Correspondingly, it belongs to the left Anomalous origination of a Coronary Artery from the Opposite Sinus of Valsalva (ACAOS) class with the intraseptal course of left anterior descending artery. We managed successfully to implant a drug eluting stent in the proximal right coronary artery in a lengthy and stormy procedure, with the need for guiding catheter exchange, temporary pacing and dealing with no-reflow phenomenon. **Conclusion.** We summarize diagnostic hints for angiographic recognition of dominant variants of the left ACAOS and practical aspects of performing PCI in such patients. Also, we debate on the functional significance of coronary anomalies and its further implications from the prognostic and therapeutic aspects. We propose adoption of the novel classification of coronary anomalies of Angelini's group in the routine clinical practice. Finally, we call for the inclusion of specific training in coronary artery anomalies into the interventional cardiology fellowship curriculum.

Key words:
percutaneous coronary intervention; coronary vessel anomalies; no-reflow phenomenon; st-elevation myocardial infarction.

Apstrakt

Uvod. Anomalije koronarnih arterija nisu čest, ali su očekivani nalaz tokom pekutanih koronarnih intervencija (PCI). Za razliku od relativno redovnog prijavljivanja koronarnih anomalija pri dijagnostičkim koronarografijama, one se nedovoljno često prikazuju u izveštajima sa interventnih procedura, posebno u sklopu primarne PCI, kada je upravo njihovo promptno otkrivanje od najveće važnosti. **Prikaz bolesnika.** Prikazan je muškarac, star 50 godina, sa akutanim infarktom miokarda sa ST elevacijom (STEMI) donjeg zida koji je zahvatio i desnu komoru, kod koga je nađen zajednički ostijum za sve tri koronarne arterije u desnom aortnom sinusu Valsalve. Ova anomalija je ekstremno retka varijanta ishoda koronarnih arterija koja odgovara tipu A4b2c2 Angelinijeve klasifikacije. Istovremeno, ona pripada i kategoriji abnormalnog ishoda koronarnih arterija iz kontralateralnog sinusa Valsalve (ACAOS) sa intraseptalnim kursom proksimalnog dela prednje descendentne arterije. Kod bolesnika je uspešno ugrađen jedan stent obložen lekom, tokom produžene i turbulentne procedure, uz potrebu izmene katetera vodiča, upotrebu privremenog *pacemaker*-a i pojavu fenomena usporenog koronarnog protoka po otvaranju arterije. **Zaključak.** Detaljno su prikazani dijagnostički postupci za angiografsko prepoznavanje dominantnih varijanti abnormalnog ishoda koronarnih arterija iz kontralateralnog sinusa Valsalve, kao i praktični aspekti izvođenja perkutanih intervencija kod ovakvih bolesnika. Analizirani su funkcionalni značaj koronarnih anomalija i njihove implikacije na dalju prognozu i terapijski pristup. Predlaženo je usvajanje nove klasifikacije koronanih anomalija, predložene od Angelinijeve grupe, u rutinskoj kliničkoj praksi. Na kraju, potrebno je uključivanje specifične obuke o svim relevantnim aspektima koronarnih anomalija u redovni program obuke interventnih kardiologa.

Ključne reči:
perkutana koronarna intervencija; koronarni krvni sud, anomalije; fenomen odsustva ponovnog protoka; infarkt miokarda sa st-elevacijom.

Correspondence to: Saša Borović, Dedinje Cardiovascular Institute, MilanaTepića 1, 11 040 Belgrade, Serbia.
E-mail: sborovic2001@yahoo.com

Introduction

Anatomic variants of coronary artery origin and course occur infrequently, but with the consistent prevalence in different series of patients. Misdiagnosing of unsuspected aberrant origin of coronary arteries is a potential problem for busy operator leading to erroneous conclusion that the artery is occluded, been responsible for the prolongation or failure of the procedure. Particularly, it is important to have this entity in mind when dealing with acute coronary syndromes since the culprit lesion may be missed.

Case report

Clinical presentation

Fifty years old male was self-referred to the local emergency department because of the acute severe chest pain of prolonged duration followed by a brief syncopal episode. His risk factors were: hypertension, hypercholesterolemia, heavy smoking and positive family history (his father died of heart attack at the age of 50). On admission, 45 minutes from pain onset, electrocardiogram (ECG) showed inferolateral ST elevation myocardial infarction (STEMI) with intermittent nodal rhythm (Figure 1, A and B), and right precordial leads revealed the right ventricular involvement (Figure 1, C). Patient received loading antiplatelet therapy (ASA 500 mg and ticagrelor 180 mg) and was immediately transferred by the cardiology ambulance to our facilities for primary percutaneous coronary intervention (PCI) within 90 minutes of the first medical contact. During the transfer, the patient was bradycardic and hypotensive despite 1.5 liter of volume expansion; chest pain and ST elevation were persistent. On admission his blood pressure (BP) was 90/55 mm Hg, heart rate (HR) 48 bpm, ST elevation persisted in the same leads with transient nodal rhythm. Echocardiographic examination revealed inferolateral hypokinesia with the global left ventricular ejection fraction of 45%, and a trace of mitral regurgitation. On the catheterization table, immediately before arterial puncture, ECG showed ST elevation resolution followed by pain relief.

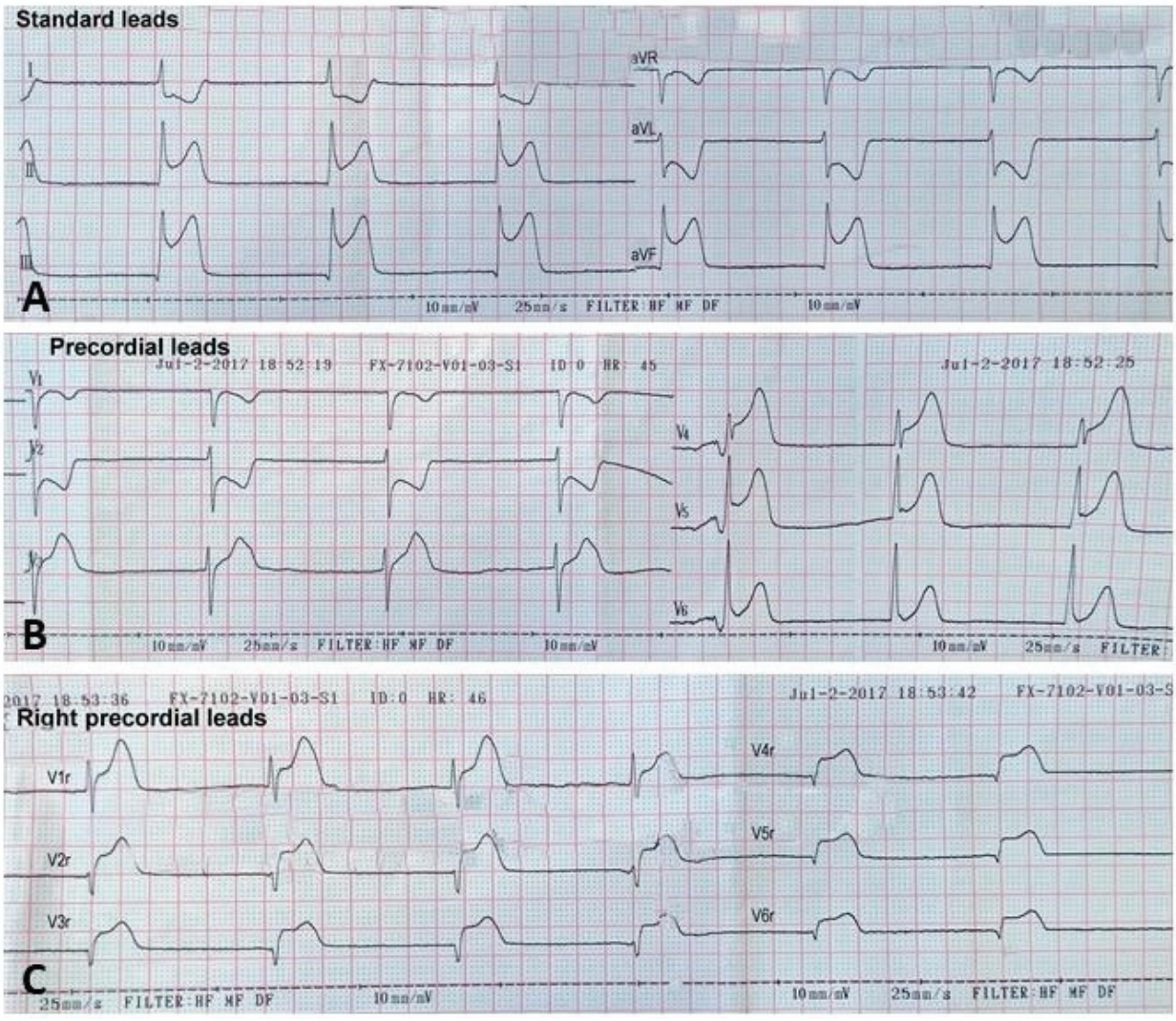

Fig. 1 – Electrocardiography (ECG) at presentation: acute inferolateral myocardial infarction (A and B), with right ventricular infarction (C), nodal rhythm.

Interventional procedure

Coronary procedure was done by the radial approach. Patient was heparinized with 100 IU/kg of unfractionated heparin (UFH). Because of bradycardia and intermittent nodal rhythm, a temporary pacemaker electrode was inserted by the femoral vein and left in a standby position. Accelerated volume substitution was continued by the i.v. line. Judkins Left 5F diagnostic catheter failed to cannulate left coronary artery in the left aortic sinus of Valsalva; non-selective contrast injection confirmed the absence of coronary ostium and the originating artery (Figure 2A).

Hockey Stick 6F SH (side holes) guiding catheter visualized solitary coronary ostium in the right sinus of Valsalva with the anomalous origin of all three main coronary vessels (Figure 2B and C).

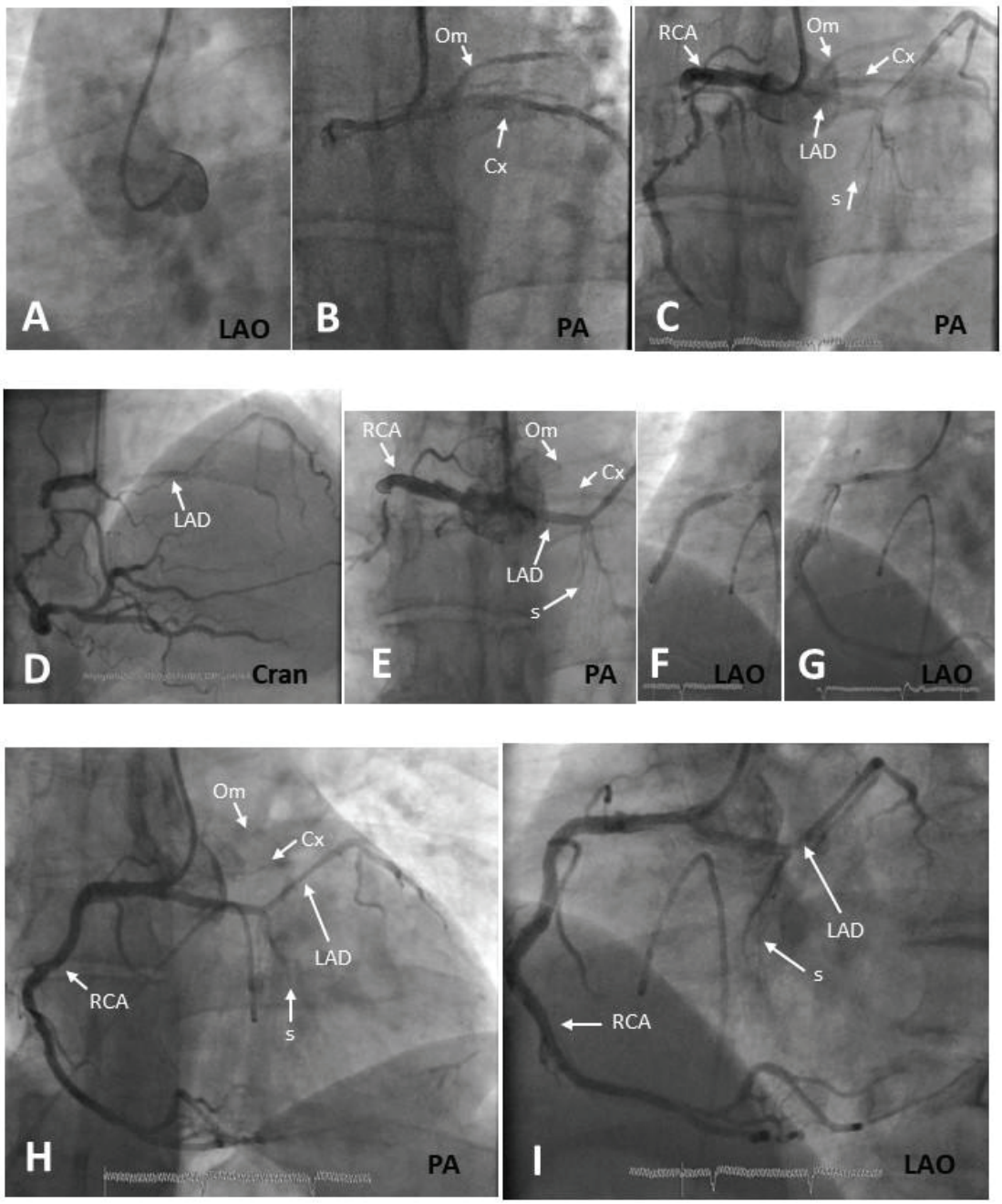

Fig. 2 – Angiographic images of the procedure: A) absence of coronary ostium in left sinus of Valsalva; B) sub-selective cannulation showing anomalous circumflex with caudal-posterior loop; C) non-selective cannulation showing all three coronary arteries originating from right sinus of Valsalva. Note the typical "eye" configuration between left anterior descending (LAD) (inferior) and circumflex (Cx) artery (superior) and proximal septal branches (S), LAD is showing caudal-anterior loop; same phenomenon can be appreciated at images E and H; D) subocclusive stenosis of right coronary artery (RCA) with substantial thrombotic burden; E) wire negotiating at subocclusive stenosis; F) coronary stenting; G) no-reflow phenomenon; H) and I) final result. Note the "hammock" appearance of the proximal LAD (also seen at images B, C, D and E) and proximal septal branches indicating septal trajectory.

Guiding catheter was then sub-selectively positioned into the dominant right coronary artery (RCA) revealing subocclusive, eccentric and heavily calcified stenosis at the proximal, bending part of RCA (Figure 2D). Distal flow in RCA was thrombolysis in myocardial infarction (TIMI) 2 grade, with spotty intraluminal filling defect distally to stenosis suggestive of acute thrombotic burden. Crossing of stenosis was initially attempted by Whisper® coronary guidewire, failing to negotiate the angulated course at the entry of stenosis (Figure 2, E). The same applied for the next attempt with Runthrough® NS coronary guidewire (the former wire was anchored in the conal branch for better support). Whole system was then replaced by AmplatzAL 2, 6F SH guiding catheter providing more selective cannulation (Figure 2F to I) and better backup enabling easy passage of ASAHI Sion Blue® coronary guidewire and its positioning in the distal RCA branch. Passage of the wire completely reoccluded the artery followed by the abrupt ST re-elevation and hemodynamic collapse (BP 50/30 mmHg with the development of complete AV block initiating ventricular pacing) necessitating rapid actions. Prompt lesion predilatation was done with noncompliant 2.5/20 mm balloon at 16 atmospheres followed by implantation of 3.5/25 mm Orsiro® stent at 14 atmospheres (Figure 2F). Immediate coronary angiography revealed no-reflow phenomenon in the targeted RCA (Figure 2G). Hemodynamic status improved marginally but remained unstable. Intracoronary bolus with 120 mcg of adenosine was injected distally to the implanted stent through the Finecross® MG microcatheter. That was followed by the normalization of coronary flow and gradual hemodynamic recovery (Figure 2H and I). At that point, an intracoronary high-dose bolus of tirofiban (Aggrastat®) was administered over 3 minutes (25 mcg/kg), followed by the continuous tirofiban infusion (0.15 mcg/kg/min) for the following 12 hours because of the substantial preexisting thrombotic burden.

Overall, the door-to-balloon time was less than 40 minutes; whole procedure was done utilizing 140 mL of non-ionic contrast media, with 21 minutes of fluoroscopy time and total radiation exposure dose of 1,950 milligroy (mGy). Patient remained stable following transfer to the coronary care unit where temporary pacemaker lead was removed.

During the remaining hospital stay, plateaued enzyme release and accelerated washout was observed, followed by the improvement of baseline wall motion abnormalities on control echocardiographic examination. Patient was discharged on standard post myocardial infarction therapy with particular attention to the continuation of beta blockers. He had an uneventful clinical course over the 6-month follow-up, avoiding extreme exertions, but refusing additional stress testing or further invasive reevaluation.

Discussion

Prevalence and classification of coronary artery abnormalities

The prevalence of coronary artery anomalies ranges from 0.5% in a multicenter pediatric autopsy study from 1994 [1] to 1.3% in the study reported from the Cleveland Clinic in 1990 among 126,595 patients undergoing coronary arteriography in which anomalies of origin and distribution occurred in 87% and coronary artery fistulae in 13% [2]. The similar prevalence of 1.34% was reported in a coronary arteriography study on a Central European population [3]. In a series of 12,457 adult coronary angiographies from Turkish population Yildiz et al. [4] reported 0.9% of prevalence of coronary artery anomalies, with 89.3% being anomalies of origin and distribution and 10.7% coronary fistulae.

Up to the present, several classifications of coronary artery anomalies have been proposed. The traditional classification of abnormal origin and distribution of coronary originates from Lipton et al. [5] in 1979. Being practical, but incomplete, this classification was further improved by Yamanaka and Hobbs [2] in 1990. In 1993, Shirani and Roberts [6] proposed atomically based classification of congenital coronary artery abnormalities, depending on the originating aortic sinus and the pattern of distribution of coronary arteries [7]. The latest classification came from Angelini's group in 2007 [8]. It is based on their long-lasting experience starting in 1999 by the analysis of 1,950 consecutive angiograms with adoption of strict criteria for assessing coronary normality versus abnormality [9]. This approach was refined and elaborated in subsequent publications dealing with pathophysiology and clinical relevance of coronary anomalies [10, 11], as well as with novel diagnostic aspects in adults [12–14]. Coronary artery abnormalities are classified into 4 categories: A) anomalies of origination and course; B) anomalies of intrinsic coronary arterial anatomy; C) anomalies of coronary termination; and D) anomalous anastomotic vessels. This classification starts with definition of normal coronary anatomy, making distinction between relevant arteries and smaller ones (such a conal branch), and defining that any form observed in > 1% of unselected general population is considered as normal. Also, it incorporates the concept of anomalous origination of a coronary artery from the opposite sinus of Valsalva anomalous coronary artery from the opposite sims (ACAOS), being lately in the focus because of its clinical and prognostic consequences.

Ectopic left coronary artery (or LAD) arising from the right coronary sinus may have 4 possible paths: 1) anterior to pulmonary outflow (free-wall), 2) interarterial, (between aorta and pulmonary artery), 3) intraseptal (intramural, or subpulmonary), 4) retro-aortic (or posterior). Ectopic circumflex arising from the right aortic sinus may have the following courses: 1) posterior atrioventricular groove or 2) retro-aortic.

Overall, our patient had a rare variant of abnormal origin of coronary arteries corresponding to Angelini classification type A4b2c2. Also, it fits into Shirani and Roberts II D3 class, Yamanaka R-II S class or LiptonR III class. Anatomically it belongs to the category of left ACAOS with intraseptal course of left anterior descending artery.

Current diagnostics of coronary artery anomalies

Selective coronary arteriography remains the gold standard for diagnosis of coronary artery anomalies, but occa-

sionally it may fail to identify the proximal course of coronary arteries, where multislice computed tomography (MSCT) gives more detailed information on the proximal tract of coronary arteries and their relationship with the surrounding structures [15]. It was shown recently that the prevalence of coronary anomalies is substantially higher when diagnosed by MSCT than with coronary angiography, even after exclusion of myocardial bridging [16].

However, radiation exposure and invasiveness are favoring nowadays the application of cardiovascular magnetic resonance (CMR) imaging for diagnosing coronary artery anomalies, particularly in children and young adults. Coronary artery anomalies were reported in 0.3% of 59,844 CMR scans performed over 15-year period in a multicenter study with valuable prognostic information over 4.3 years median follow-up [17].

Since most of coronary artery anomalies are diagnosed incidentally during coronary angiography, it is important to recognize them and to make correct angiographic identification, however that requires specific training of the operator [12]. Unfortunately, it may happen – mainly during primary angioplasty when operators are focused on opening an occluded artery and respecting the need for rational usage of contrast media – that acquired angiographic data do not allow correct identification and the precise reconstruction of the proximal course of anomalous vessels. However, a long time ago, precise instructions how to obtain angiographic identification of coronary artery anomalies have already been proposed. In 1985, Ishikawa and Brandt [18] presented a practical "cookbook" for diagnosing all four dominant types of anomalous origin of the left coronary artery from the right aortic sinus of Valsalva based on two orthogonal angiographic projections: right anterior oblique (RAO) and left anterior oblique (LAO), focusing on the orientation of the proximal loop in the frontal and sagittal planes. This concept was further simplified by Serota et al. [19] in 1990 providing the same identification of anomalous origin and proximal course of anomalous coronary arteries from a single RAO projection. Serota's method provides helpful diagnostic hints for facilitating this process using evocative "dot and eye" signs to distinguish easily between four main types of anomalous course. In addition, Chaitman et al. [20] proposed an useful diagnostic tip for distinguishing intraseptal course of left coronary artery originating from the right sinus of Valsalva (considered as benign variant) from the interarterial course: the presence of septal perforators in the proximal part of the vessel, preferably in the lateral and left anterior oblique projections. More demanding angiographic model, utilizing additional contrast, has been proposed by Wang et al. [21] in 1997. It is using simultaneous biplane coronary and pulmonary arteriography (RAO 30 and LAO 60) to define the course of anomalous left coronary artery originating from the right sinus of Valsalva. Levophase imaging of the ascending aorta additionally increases its diagnostic utility. Digital subtraction can further improve this procedure by enhancing levophase images of coronary artery-ascending aorta relationship [21]. An useful diagnostic hint by placing a catheter into the pulmonary artery to help in determining the course of anomalous coronary artery in relation to the pulmonary artery was proposed by Chu and Cheitlin [22] in 1993 allowing confident delineation of the interarterial variant from the other, benign forms. Also, if using pacing lead during the percutaneous procedure, like in our case, it may be positioned into the right ventricular outflow tract and utilized as a reference marker to aid in determining the proximal course of anomalous coronaries.

In our patient, shortly after failing to cannulate left coronary artery, we confirmed the absence of its ostium in left aortic sinus of Valsalva by sub-selective had injection of radiographic contrast (Figure 2A). When discovered ACAOS, we used orthogonal projections to determine the proximal course of anomalous arteries, following recommendations of Ishikawa and Brandt [18], and diagnostic hints by Serota et al. [19]. In addition, we confirmed our finding of the intraseptal course of left anterior descending (LAD) artery by the presence of septal perforators according to the findings of Chaitman et al. [20]. It is important to note the "hammock" appearance of the proximal course of LAD [2] at the Figure 1 images B, C, D, E, H and I confirming the septal course with caudal-anterior loop. On the other hand, circumflex (Cx) artery is taking the course behind the aorta and anterior to the atria to the left, making caudal-posterior loop. Similarly, image C at the Figure 2 is showing typical "eye" configuration between LAD (inferior) and Cx (superior) confirming, together with prominent septal (s) branches, the septal course of proximal portion of LAD [19, 20]. Schematic presentation of the origin and proximal course of main coronary arteries in our case is provided on the Figure 3.

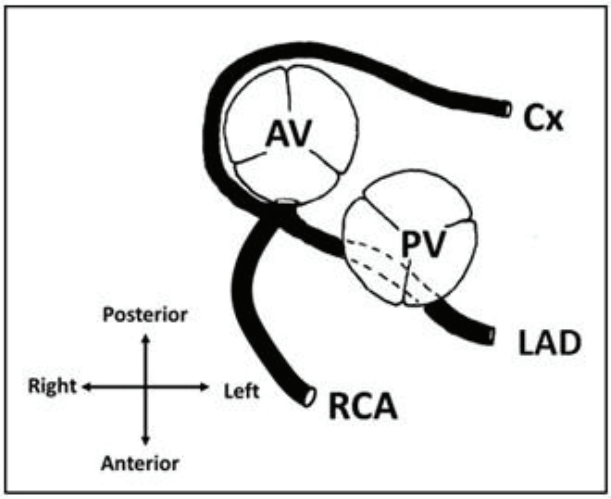

Fig. 3 – Schematic drawing: origin and proximal course of coronary arteries in the presented case. AV – aortic valve; PV – pulmonary valve; LAD – left anterior descending artery; RCA – right coronary artery; Cx – circumflex artery.

Clinical and functional significance

Precise definition of the origin and course of anomalous coronary arteries is important when planning coronary revascularization, either by means of surgery or coronary angio-

plasty. In a case of primary angioplasty where the "time is muscle", there is an even greater need for accurate and timely diagnostics.

Presence of certain types of anomalous coronary arteries has important prognostic implications. It has been shown that coronary artery anomalies may cause clinically disabling symptoms, especially in young adults, including dyspnea, angina pectoris and syncope [14]. Furthermore, such conditions are associated with increased frequency of myocardial ischemia and sudden cardiac death in young persons, especially during or immediately after strenuous exertion [14]. Maron et al. [23] followed a large registry, assembled over a 27-year period, of sudden deaths among US competitive athletes reporting constantly that after hypertrophic cardiomyopathy, anomalous origin of coronary artery from the opposite sinus of Valsalva is the second most common cause of sudden death in young competitive athletes in the USA [24]. Of 1,866 athletes who died suddenly (or survived cardiac arrest) over that period, hypertrophic cardiomyopathy was a cause in 36% of cases and congenital coronary artery anomalies in 17% [23]. Eckart et al. [25] was following military recruits in USA aged 18 to 35 years, finding 126 nontraumatic deaths, 64 of them were attributed to cardiac causes. An anomalous coronary artery accounted for one third of cardiac cases (21 of 64 cases), and in each of them left coronary artery originating from the right sinus of Valsalva was taking interarterial course between the pulmonary artery and aorta [25]. In that study, ACAOS was a more-common cause of cardiac death (61%) than cardiomyopathies (36%). Similarly, studying the sudden unexpected death in persons less than 40 years of age in Israel, where autopsy is obligatory in such cases, Drory et al. [26] in a series of 162 consecutive cases found that incidence of sudden death related to anomalies of coronary arteries was 0.6%.

Although anomalous origin of the right coronary artery is four times as common as anomalous origin of left, sudden death occurs more frequently in patients with anomalous origin of left coronary artery [27]. Interarterial course of anomalous artery originating from the contralateral sinus between pulmonary and aortic roots is held responsible in most cases, although exact mechanism is unclear. Traditionally, "scissor-like" mechanism was proposed, where the expansion of great vessels during vigorous exercising is compressing anomalous artery along its interarterial course and provoking ischemia. Also, it was proposed that expansion of aorta and pulmonary artery during exercise may provoke spasm, or kinking and torsion, of anomalous artery along its aberrant interarterial course. Presence of coronary ostial ridge which may function as valve, restricting flow during exertion has been proposed, as well. Less supported by evidences is the suggestion that anomalous origin may be associated with accelerated atherosclerotic disease, or that the congenitally small left coronary artery is responsible for ischemia. Novel insights in coronary anomalies with origination from the contralateral sinus of Valsalva (ACAOS) by intravascular imaging [10–14, 28, 29] are discovering that the proximal portion of aberrant vessels runs within the aortic wall (intramural, or intraparietalcourse) [30] being often hypoplastic and exposed to lateral compression forming a slit-like formation that compromises the flow. During vigorous activity this proximal part is susceptible for further collapsing or closuring provoked by theincrease of cardiac stroke volume and arterial hypertension.

Reports of primary PCI in anomalous coronary anatomy

There is a limited number of publications dealing with coronary interventions in patients with anomalous origin of coronary arteries, most of them being case reports. Data on primary PCI in this setting are even more scarce. However, most of authors report prolonged procedures, with higher than usual complication rates and occasionally with adverse acute outcome, particularly in the case of primary procedures.

In our patient, the cannulation or the coronary ostium was challenging, requiring interchange of guiding catheters for proper alignment and better support, as well as multiple wire manipulations to pass initial sharp bends. For that purpose, there are several reports of successful utilization of "mother and child" sub-catheter technique for selective cannulation of coronary ostium in the percutaneous treatment of coronary anomalies. Also, in order to avoid proximal dislodgment of thrombotic material and eventual massive embolization into adjacent arteries originated from the same ostium, we did not perform thrombo-aspiration.

Overall, in our case the primary PCI had a stormy course with favorable outcome, however with higher fluoroscopy time, contrast utilization and radiation exposure compared with our median values. During the index procedure, we managed angiographically to assess the course of proximal segments of all coronary arteries confirming the intraseptal trajectory of LAD consider asprognostically favorable anatomic variant. However, based on our findings, as well as on the history of sudden cardiac death of his father in his middle age, we have suggested more watchful clinical follow-up by means of repeated exercise stress testing and ECG Holter monitoring along with the avoidance of extreme physical activities.

Conclusion

Altered coronary anatomy mandates prompt recognition imposing higher procedural risk and requesting technically more demanding approach during percutaneous procedures. It is important to select appropriate guiding catheter providing sufficient support without pressure dumping and to prevent coronary dissection, being particularly threatening complication in this scenario. When performing PCI in the setting of anomalous coronary origin, operator may expect sharp bends or acute take-offs prolonging the procedure and requiring specific skills. It is important to perform meticulous measures to avoid thromboembolism or proximal thrombotic propagation into the adjacent coronary arteries originating from the same ostium. Likewise, such cases may need more aggressive antiplatelet therapy balanced by the individualized hemorrhagic risk. Operator should anticipate a higher than usual chance of hemodynamic instability, reperfusion disorders, as well as rhythm and conduction distur-

bances. For that reason, each coronary anomaly should be evaluated individually weighting its potential functional and prognostic significance and making decision about the need for specific therapy.

When facing with anomalous origin of coronary arteries, operators should do their best to define the precise anatomy and their exact trajectory. That requires specific awareness to coronary anomalies, so that we call for the inclusion of specific training in coronary artery anomalies into the interventional cardiology fellowship curriculum. Based on current experience, from the diagnostic and prognostic standpoint, we propose adoption and adherence in reporting with the latest classification of coronary artery anomalies from Angelini's group.

REFERENCES

1. *Lipsett J, Cohle SD, Berry PJ, Russell G, Byard RW.* Anomalous coronary arteries: a multicenter pediatric autopsy study. Pediatr Pathol 1994; 14(2): 287–300.
2. *Yamanaka O, Hobbs RE.* Coronary artery anomalies in 126,595 patients undergoing coronary arteriography. Cathet Cardiovasc Diagn 1990; 21(1): 28–40.
3. *Kardos A, Babai L, Rudas L, Gaal T, Horvath T, Talosi L,* et al. Epidemiology of congenital coronary artery anomalies: a coronary arteriography study on a central European population. Cathet Cardiovasc Diagn 1997; 42(3): 270–5.
4. *Yildiz A, Okcun B, Peker T, Arslan C, Olcay A, Bulent Vatan M.* Prevalence of coronary artery anomalies in 12,457 adult patients who underwent coronary angiography. Clin Cardiol 2010; 33(12): E60–4.
5. *Lipton MJ, Barry WH, Obrez I, Silverman JF, Wexler L.* Isolated single coronary artery: diagnosis, angiographic classification, and clinical significance. Radiology 1979; 130(1): 39–47.
6. *Shirani J, Roberts WC.* Solitary coronary ostium in the aorta in the absence of other major congenital cardiovascular anomalies. J Am Coll Cardiol 1993; 21(1): 137–43.
7. *Roberts WC, Shirani J.* The four subtypes of anomalous origin of the left main coronary artery from the right aortic sinus (or from the right coronary artery). Am J Cardiol 1992; 70(1): 119–21.
8. *Angelini P.* Coronary artery anomalies: an entity in search of an identity. Circulation 2007; 115(10): 1296–305.
9. *Angelini P, Villason S, Chan A V, Diez G.* Normal and Anomalous Coronary Arteries in Humans. In: *Angelini P,* editor. Coronary Artery Anomalies: A Comprehensive Approach. Philadelphia: Lippincott Williams & Wilkins; 1999. p. 27–150.
10. *Angelini P, Velasco JA, Flamm S.* Coronary anomalies: incidence, pathophysiology, and clinical relevance. Circulation 2002; 105(20): 2449–54.
11. *Angelini P, Walmsley RP, Libreros A, Ott DA.* Symptomatic anomalous origination of the left coronary artery from the opposite sinus of valsalva. Clinical presentations, diagnosis, and surgical repair. Texas Hear Inst J 2006; 33(2): 171–9.
12. *Angelini P, Flamm SD.* Newer concepts for imaging anomalous aortic origin of the coronary arteries in adults. Catheter Cardiovasc Interv 2007; 69(7): 942–54.
13. *Angelini P.* ACAOS is better revealed by intravascular ultrasonography than by computed tomographic angiography. Tex Heart Inst J 2015; 42(3): 246–7.
14. *Angelini P.* Novel imaging of coronary artery anomalies to assess their prevalence, the causes of clinical symptoms, and the risk of sudden cardiac death. Circ Cardiovasc Imaging 2014; 7(4): 747–54.
15. *Deibler AR, Kuzo RS, Vohringer M, Page EE, Safford RE, Patron JN,* et al. Imaging of congenital coronary anomalies with multislice computed tomography. Mayo Clin Proc 2004; 79(8): 1017–23.
16. *Ghadri JR, Kazakauskaite E, Braunschweig S, Burger IA, Frank M, Fiechter M,* et al. Congenital coronary anomalies detected by coronary computed tomography compared to invasive coronary angiography. BMC Cardiovasc Disord 2014; 14(81): 1–10.
17. *Ripley DP, Saha A, Teis A, Uddin A, Bijsterveld P, Kidambi A,* et al. The distribution and prognosis of anomalous coronary arteries identified by cardiovascular magnetic resonance: 15 year experience from two tertiary centres. J Cardiovasc Magn Reson 2014; 16: 34.
18. *Ishikawa T, Brandt PW.* Anomalous origin of the left main coronary artery from the right anterior aortic sinus: angiographic definition of anomalous course. Am J Cardiol 1985; 55(6): 770–6.
19. *Serota H, Barth CW 3rd, Seuc CA, Vandormael M, Aguirre F, Kern MJ.* Rapid identification of the course of anomalous coronary arteries in adults: the "dot and eye" method. Am J Cardiol 1990; 65(13): 891–8.
20. *Chaitman BR, Lesperance J, Saltiel J, Bourassa MG.* Clinical, angiographic, and hemodynamic findings in patients with anomalous origin of the coronary arteries. Circulation 1976; 53(1): 122–31.
21. *Wang A, Pulsipher MW, Jaggers J, Peterson GE, O'Laughlin MP, Bashore TM,* et al. Simultaneous biplane coronary and pulmonary arteriography: a novel technique for defining the course of an anomalous left main coronary artery originating from the right sinus of Valsalva. Cathet Cardiovasc Diagn 1997; 42(1): 73–8.
22. *Chu E, Cheitlin MD.* Diagnostic considerations in patients with suspected coronary artery anomalies. Am Heart J 1993; 126(6): 1427–38.
23. *Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO.* Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation 2009; 119(8): 1085–92.
24. *Basso C, Maron BJ, Corrado D, Thiene G.* 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(6): 1493–501.
25. *Eckart RE, Scoville SL, Campbell CL, Shry EA, Stajduhar KC, Potter RN,* et al. Sudden death in young adults: a 25-year review of autopsies in military recruits. Ann Intern Med 2004; 141(11): 829–34.
26. *Drory Y, Turetz Y, Hiss Y, Lev B, Fisman EZ, Pines A,* et al. Sudden unexpected death in persons less than 40 years of age. Am J Cardiol 1991; 68(13): 1388–92.
27. *Hata Y, Kinoshita K, Kudo K, Ikeda N, Nishida N.* Anomalous origin of the right coronary artery from the left coronary sinus with an intramural course: Comparison between sudden-death and non-sudden-death cases. Cardiovasc Pathol 2015; 24(3): 154–9.
28. *Angelini P.* Coronary artery anomalies--current clinical issues: definitions, classification, incidence, clinical relevance, and treatment guidelines. Texas Hear Inst J 2002; 29(4): 271–8.
29. *Angelini P.* Anomalous origin of the left coronary artery from the opposite sinus of valsalva: typical and atypical features Tex Heart Inst J 2009; 36(4): 313–5.
30. *Angelini P, Velasco JA, Ott D, Khoshnevis GR.* Anomalous coronary artery arising from the opposite sinus: descriptive features and pathophysiologic mechanisms, as documented by intravascular ultrasonography. J Invasive Cardiol 2003; 15(9): 507–14.

Received on March 14, 2018.
Revised on May 14, 2018.
Accepted on June 1, 2018.
Online First June, 2018