DOI: https://doi.org/10.2298/SARH230415072C
UDC: 616.132.2-073: 616.12-008.315

CASE REPORT / ПРИКАЗ БОЛЕСНИКА

Cardiac arrest and repeated ST-segment elevation caused by initially unrecognized coronary vasospasm

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SUMMARY

Introduction Coronary artery spasm (CAS) is defined as a transient vasoconstriction of an epicardial coronary artery with a total or subtotal vessel occlusion, causing myocardial ischemia. Although the overall incidence of CAS during coronary angiography is estimated to be between 0.3% and 1%, CAS remains an underdiagnosed and undertreated cause of acute cardiac events.

Case outline We report a case of a 62-year-old male presenting with cardiac arrest and repeated ST-segment elevations during chest pain episodes, caused by initially unrecognized CAS. Although percutaneous coronary intervention (PCI) is generally not recommended for the treatment of CAS, due to clinical manifestations and the presence of underlying flow-limiting stenosis, we decided to perform PCI of the left anterior descending coronary artery. During the two-year-follow up period, the patient reported no chest pains nor exercise limitations at regular outpatient controls.

Conclusion CASs should be considered an unrecognized cause of refractory angina, acute coronary syndrome, malignant arrhythmia, and even cardiac arrest. Although medical therapy is the first option for CAS treatment, PCI could be a safe and effective approach in selected patients.

Keywords: coronary vasospasm; fractional flow reserve; myocardial infarction; optical coherence tomography; percutaneous coronary intervention

INTRODUCTION

Coronary artery spasm (CAS) is defined as a transient vasoconstriction of an epicardial coronary artery with a total or subtotal vessel occlusion, causing myocardial ischemia [1, 2, 3]. Depending on the site, duration, and severity of the spasm, the clinical presentation of CAS ranges from "silent ischemia" to transmural myocardial infarction, malignant arrhythmias, and even sudden cardiac death [2]. Although the exact prevalence of CAS remains unknown, it is believed that prevalence is decreasing, probably due to the wide usage of angiotensin-converting enzyme inhibitors, calcium channel blockers, and statins in cardiovascular medicine, as well as the decrease in smoking habits in developed countries [4, 5]. However, CAS remains an underdiagnosed and undertreated cause of acute cardiac events [2, 6]. In this article, we report a case of a 62-year-old male presenting with cardiac arrest and repeated ST-segment elevations during chest pain episodes, caused by initially unrecognized CAS.

Received • Примљено: April 15, 2023
Revised • Ревизија: July 12, 2023
Accepted • Прихваћено: July 13, 2023
Online first: July 21, 2023

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CASE REPORT

A 62-year-old male was admitted to the Emergency Department due to sharp chest pain that lasted for 15 minutes. Except for smoking and dyslipidemia, no other cardiovascular risk factors were identified. He was asymptomatic on admission, the electrocardiogram (ECG) showed no significant findings, troponin I was in the reference range, and the transthoracic echocardiography (TTE) was orderly. After 12 hours of observation, the patient was discharged for further home treatment, with a prescription for 100 mg of acetylsalicylic acid per day. Three days after, he performed an exercise stress test (Bruce protocol), which was interrupted before reaching a submaximal heart rate, due to extreme fatigue. ECG before and during the test was without ischemia-related changes. However, after four minutes of rest, the patient experienced cardiac arrest with a rhythm of ventricular fibrillation. Regular heart rhythm was restored after cardioversion, and ECG showed ST elevation in leads V1–V5. The patient was immediately transferred to the Emergency Department, where ECG showed 0.5 mm elevations of ST-segment in inferior leads, without any ST-segment denivelations in anterior leads. A coronary angiogram, performed after 30 minutes, visualized stenosis up to 80% in the proximal part and borderline stenosis above the ectatic segment in the medial right coronary artery (RCA). The finding on the left coronary artery (LCA) was orderly, except for borderline stenosis in the medial left anterior descending (LAD) artery (Figures 1A and 1B). We note that the patient reported no chest pain at the Catheterization laboratory, and ECG monitoring was without ST-segment denivelations. We decided to perform the percutaneous coronary intervention (PCI) of the RCA. After administering 180 mg of ticagrelor

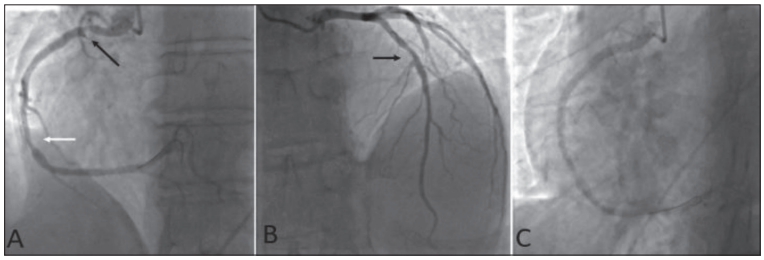

Figure 1. Coronary angiogram at admission;

A – coronary angiogram of the right coronary artery, showing stenosis up to 80% in the proximal part (black arrow) and borderline stenosis at the medial segment (white arrow); B – coronary angiogram showing borderline stenosis in the medial segment of the left anterior descending artery; C – coronary angiogram after performing percutaneous coronary intervention of the right coronary artery

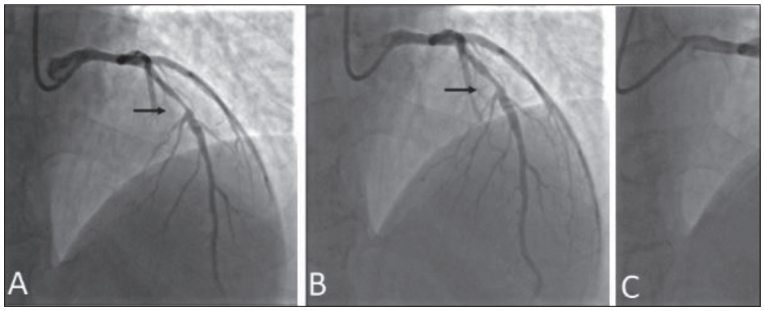

Figure 2. Repeated coronary angiogram and percutaneous coronary intervention of the left anterior descending coronary artery;

A – coronary angiogram showing stenosis up to 85% in the medial part of the left anterior descending coronary artery (black arrow); B – spasm reduction after nitroglycerin was administered intracoronary, with a persistence of a significant lumen reduction (black arrow); C – coronary angiogram after performing percutaneous coronary intervention of the left anterior descending artery

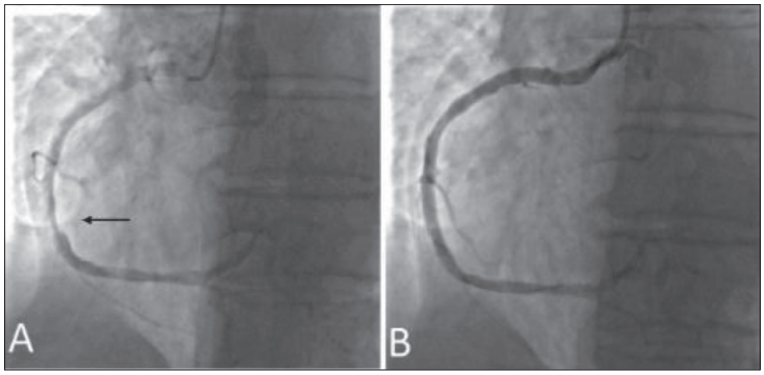

Figure 3. Repeated coronary angiogram and percutaneous coronary intervention of the right coronary artery;

A – coronary angiogram showing 70% lumen reduction in a distal segment of the right coronary artery (black arrow); B – coronary angiogram after performing percutaneous coronary intervention of the right coronary artery

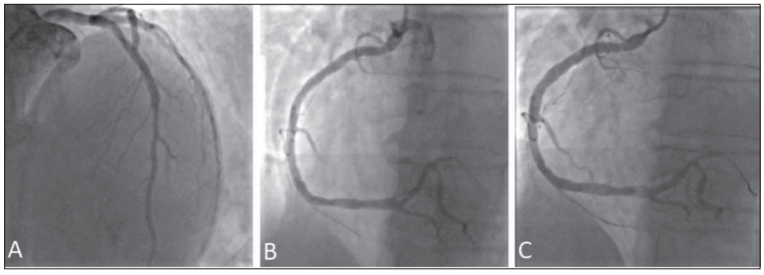

Figure 4. Repeated coronary angiogram, three months after the index hospitalization;

A – orderly coronary angiogram of the left coronary artery; B – angiogram of the right coronary artery, before optical coherence tomography and stent optimization; C – angiogram of the right coronary artery after the stent optimization

and lesion preparation, the 4.0 × 18 mm drug-eluting stent (DES) was implanted in the proximal RCA (Figure 1C). The patient continued the inpatient treatment at the Cardiology clinic for further observation. Control TTE findings after the procedure showed reduced global systolic function, with the left ventricle ejection fraction estimated at 45–50%, hypokinesia of the basal and medial segment of the inferior wall, as well as the basal segment of the septum.

The patient developed intense chest pains in the early morning of the fourth hospital day, accompanied by nausea and vomiting. The ECG recorded significant ST-segment elevations in precordial leads, with minimal ST-segment depression in the inferior leads. The patient was immediately transferred to the Catheterization Laboratory, where a coronary angiogram showed stenosis up to 85% in the medial part of the LAD (Figure 2A). The stenosis in the distal RCA was estimated at 70% (Figure 3A). After the intracoronary administration of nitroglycerin, the spasm in the LAD was reduced (Figure 2B). The fractional flow reserve (FFR) value distal to stenosis was 0.78. Due to the underlying flow-limiting stenosis, we decided to perform PCI of the LAD. After predilatation with a 3 × 15 mm semi-compliant balloon, the 4 × 15 mm DES was implanted. The stent was optimized using the 4.5 × 12 mm non-compliant (NC) balloon at 16 atmospheres. The final angiographic result was optimal with the "Thrombolysis In Myocardial Infarction" (TIMI) 3 flow along the LAD (Figure 2C). After that, we decided to perform PCI of the distal RCA. The 4 × 24 mm DES was implanted, with the optimal final angiographic result and the TIMI 3 flow after the NC balloon postdilatation (Figure 3B).

After nine days of hospital treatment, the patient was discharged from the Cardiology Clinic. In addition to acetylsalicylic acid 100 mg daily and ticagrelor 90 mg twice a day, we opted for a statin, selective beta blocker, and dihydropyridine calcium channel blocker.

Repeated coronary angiography with optical coherence tomography (OCT) of the RCA and LAD was performed three months after the index procedure. The angiogram of the LCA was orderly, and OCT showed adequate stent expansion and apposition, without any stent-related complications (Figures 4A and 5A). Although the angiogram of the RCA was orderly (Figure 4B), OCT revealed malposition of the proximally

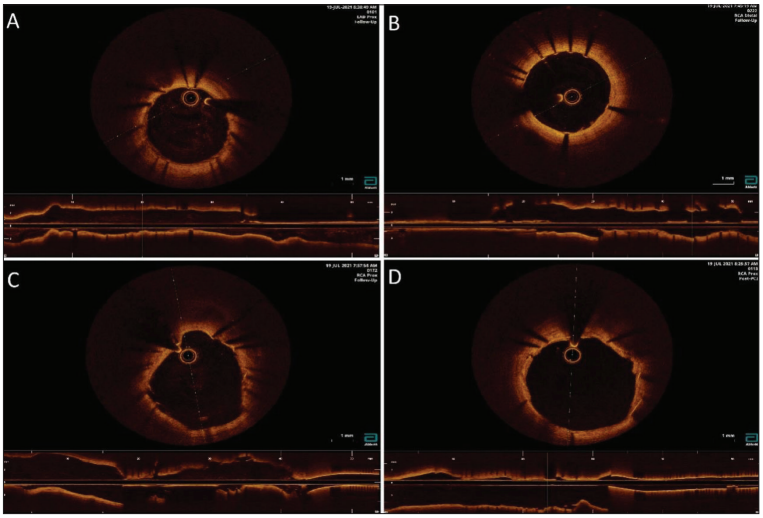

Figure 5. Optical coherence tomography (OCT) imaging after three months follow-up; A – OCT imaging of the proximal left anterior descending artery showed adequate stent expansion and apposition, without any stent-related complications; no signs of atherosclerotic disease were visualized outside the stented segment; B – OCT imaging of the distal right coronary artery showed adequate stent expansion and apposition, without any stent-related complications; C – OCT imaging of the proximal right coronary artery showed malposition of the implanted stent; D – control OCT imaging of the proximal right coronary artery showed adequate stent expansion and apposition

implanted stent, with adequate apposition and expansion of the second stent (Figures 5B and 5C). Therefore, stent optimization was conducted using the 5 mm NC balloon, with satisfactory control OCT run (Figures 4C and 5D).

During the two-year-follow up period, the patient reported no chest pains nor exercise limitations at regular outpatient controls.

We obtained verbal and signed consent of the patients to publish the case report. This article was planned in compliance with the Patient Rights Directive and ethical rules by considering the principles of the Declaration of Helsinki.

DISCUSSION

CAS is a phenomenon caused by smooth muscle cell hyperactivity and vascular wall hypertonicity, which results in a transient complete or partial closure of a coronary artery with subsequent myocardial ischemia [2]. The exact pathogenesis has not been fully elucidated; however, the current findings suggest a complex interaction of several important mechanisms such as primary hyperactivity of vascular smooth muscle cells, endothelial dysfunction, chronic low-grade inflammation, altered autonomic nervous system response, and others [1, 2, 3]. The common clinical presentation of CAS-induced myocardial ischemia are ST-segment changes and chest pain, similar to that in a stable angina and myocardial infarction [4, 5]. However, there are several distinctions that should be considered. First of all, "silent" ischemia is twice more prevalent in patients with CAS compared to symptomatic presentation. In those with symptomatic ischemia, chest pains are usually more severe, prolonged, and accompanied by cold sweating, nausea, and even syncope. ECG changes commonly consist of transient ST-segment elevation, but can also include ST-segment depression, T wave alterations, negative U wave, and even malignant arrhythmias, more often seen in multi-vessel CAS [2, 3, 5]. Importantly, due to its association with the circadian rhythm, coronary vasospasm occurs at rest, often between midnight and early morning. In contrast to effort angina, CAS is usually not associated with physical activity, however, it is shown that even mild activity in the morning can potentiate the spasm. Additionally, coronary vasoconstriction could be potentiated by several factors, including cigarette smoking, prolonged emotional stress, Valsalva maneuver, exposure to cold, and use of some medications, such as sympathomimetic agents, nonselective beta-blocker, and others [1, 5]. The sex differences also exist and latest meta-analysis in that regard demonstrate it [6], beyond case reports [7], while women with confirmed CAS tend to have more autoimmune rheumatic diseases in the Women's Ischemia Syndrome Evaluation – Coronary Vascular Dysfunction cohort [8] and long-term follow-up solutions for this growing patient group remains in the normalization of the concept of heart centers for women – both internationally and locally, in Serbia [9, 10].

An important obstacle in diagnosing is the difficult recognition of CAS in a coronary angiogram, since vasospasm, if even present, cannot be easily distinguished from atherosclerotic narrowing [11]. Definitive diagnosis is made after angiographic evidence of coronary vasoconstriction that reverses with the administration of intravenous or intra-arterial nitroglycerin [1, 12]. Findings from OCT studies suggest that a significant portion of CAS occurs at the site of the atherosclerotic segment, and unrecognized CAS increases the risk of overestimating the stenosis severity [13]. This is particularly important since PCI is generally not recommended for the treatment of CAS in the absence of severe atherosclerotic disease [6]. Recommended medical treatment options consist of pharmacological agents that prevent vasoconstriction and promote vasodilation in the coronary vasculature, such as calcium channel blockers and nitrates. Additionally, lifestyle changes such as smoking cessation, improving diet and exercise habits, and managing stress can also help prevent vasospasm from occurring [1]. However, despite medical therapy, some patients experience refractory angina and other CAS-related complications. The potential reason could be the residual, flow-limiting atherosclerotic stenosis, even after complete spasm resolution [14]. Findings suggest that CAS induces local thrombus formation as well as the production of inflammatory mediators, therefore increasing the risk of atherosclerotic plaque progression and ischemic events in patients with CAS [14]. In addition, studies have shown that patients

with coexisting CAS and obstructive flow-limiting stenosis have a greater risk of major adverse cardiac events and worse outcomes [15, 16]. Therefore, PCI management of CAS associated with significant atherosclerotic stenosis could be considered in selected patients [2, 6, 11]. In adequate decision-making, adjuvant methods such as functional coronary assessment and intracoronary imaging can be helpful. OCT imaging can be useful in both diagnosing and understanding the pathophysiology of CAS [17]. In addition, OCT is recognized as a helpful tool for accessing procedural complications and improving stent apposition and expansion, therefore decreasing the risk of stent thrombosis and the incidence of stent-related complications [17]. In our case, OCT was not used in the index procedure by the operator's choice, due to already confirmed CAS in the setting of flow-limiting atherosclerotic plaque. OCT was used in the follow-up, to access the quality and the potential complications of the stent implanted in the segment affected by CAS. In our case, the OCT run showed adequate apposition and expansion, without any complications.

In conclusion, CAS should be considered an unrecognized cause of refractory angina, acute coronary syndrome, malignant arrhythmia, and even cardiac arrest. Although medical therapy is the first option for CAS treatment, PCI could be a safe and effective approach in selected patients. In adequate decision-making, adjuvant methods such as functional coronary assessment and intracoronary imaging should be considered.

ACKNOWLEDGMENTS

The authors would like to thank the entire cardiovascular team involved in the care of the patient presented in the case and their day-to-day dedication.

Conflict of interests: None declared.

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Срчани застој и понављане елевације *ST*-сегмента узроковане иницијално непрепознатим коронарним вазоспазмом

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САЖЕТАК

Увод Коронарни артеријски спазам се дефинише као транзиторна вазоконстрикција епикардне коронарне артерије са тоталном или суптоталном оклузијом крвног суда и последичном исхемијом миокарда. Иако је описан у 0,3–1% коронарних ангиографија, сматра се да коронарни вазоспазам у значајној мери остаје недијагностикован узрок акутних коронарних догађаја.

Приказ болесника У овом раду приказујемо 62-годишњег пацијената са срчаним застојем и понављаним елевацијама *ST*-сегмента током епизода бола у грудима, који су узроковани иницијално непрепознатим коронарним вазоспазмом. Иако перкутане коронарне интервенције генерално нису препоручен избор лечења коронарног вазоспазма, услед клиничких манифестација и присуства удружене значајне атеросклеротске болести, одлучили смо се за имплантацију стента на левој предњој десцедентној коронарној артерији. Током двогодишњег праћења на редовним амбулантним контролама, пацијент негира ангинозне болове и ограничења у физичкој активности.

Закључак Коронарни артеријски спазам би требало размотрити као непрепознат узрок рефракторне ангине, акутног коронарног синдрома, малигних аритмија и срчаног застоја. Иако је медикаментна терапија прва опција у лечењу коронарног вазоспазма, перкутана коронарна интервенција би могла бити безбедна и ефикасна метода лечења код одређених пацијената.

Кључне речи: инфаркт миокарда; коронарни вазоспазам; оптичка кохерентна томографија; перкутане коронарне интервенције; фракциона резерва протока