CASE REPORT
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Giant left main coronary artery aneurysm

Ogromna aneurizma leve glavne koronarne arterije

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Abstract

Introduction. Coronary artery aneurysms (CAAs) are rare disorders, especially the left main CAA. In the literature, there are several reported cases with CAAs, various localization, size, clinical presentation, and way of treatment. There is no unique consensus about the most adequate treatment for these patients; however, surgery is still preferable, although there are some new experiences of percutaneous treatment. The decision is made for each patient individually. We report a case of giant left main CAA, with acute coronary syndrome and heart failure presentation, surgically treated. **Case report.** A 66-year-old female patient was admitted to the emergency department of our clinic due to chest pain and dyspnea. Acute non-ST-elevation segment myocardial infarction (STEMI) of anterolateral localization was diagnosed (creatine kinase max 1,111 U/L, troponin T 3.754 ng/mL), complicated with acute heart failure. Heart catheterization and coronary angiography revealed a giant saccular, 3.5 × 3.5 mm left main CAA full with thrombi, compressing the proximal segments of the left anterior descending and circumflex artery. **Conclusion**. Giant left main CAAs are rare pathologic findings, and there are no established principles for treatment. Although the percutaneous way of treatment is now available in selected cases, the surgical approach is still preferred for these patients.

Key words:
cardiac catheterization; coronary aneurysm; coronary angiography; cardiac surgical procedures; diagnosis; non-st elevated myocardial infarction.

Apstrakt

Uvod. Aneurizme koronarnih arterija (KA) su retki poremećaji, naročito aneurizme glavnog stabla leve KA. U literaturi je opisano više slučajeva aneurizmi KA, različite lokalizacije, veličine, kliničke slike i načina lečenja. Ne postoji jedinstven stav o najadekvatnijem načinu lečenja kod tih bolesnika. Međutim, i dalje je poželjniji operativni način lečenja, mada je moguć i perkutani način lečenja. Odluka se donosi za svakog bolesnika pojedinačno. Prikazan je redak slučaj bolesnika, lečenog hirurškim putem, sa velikom aneurizmom glavnog stabla leve KA, koja se prezentovala akutnim koronarnim sindromom i srčanom insuficijencijom. **Prikaz bolesnika.** Bolesnica stara 66 godina primljena je u urgentni centar naše klinike zbog bolova u grudima i dispneje. Dijagnostikovan je akutni infarkt miokarda bez elevacije ST segmenta anterolateralne lokalizacije (maksimalne vrednosti kreatin kinaze 1 111 U/L, troponina T 3,754 ng/mL), komplikovan akutnom srčanom insuficijencijom. Kateterizacijom srca i koronarografijom otkrivena je ogromna (3,5 × 3,5 mm) sakularna aneurizma glavnog stable leve KA, puna tromba, koja je pritiskala proksimalne segmente leve prednje silazne i cirkumfleksne arterije. **Zaključak.** Ogromne aneurizme leve glavne KA su redak patološki nalaz i ne postoje utvrđeni principi lečenja. Mada je perkutani način lečenja sada dostupan u odabranim slučajevima, za te bolesnike je i dalje poželjan hirurški pristup.

Ključne reči:
kateterizacija srca; aneurizma, koronarna; hirurgija, kardijalna, procedure; dijagnoza; angiografija koronarnih arterija; infarkt miokarda bez st-elevacije.

Introduction

There are two entities of coronary arteries enlargement – coronary artery ectasias (CAEs) and coronary artery aneurysms (CAAs). CAEs refer to diffuse dilatation up to 50% of the largest diameter, and CAAs are defined as localized dilatation which exceeds the largest vessel diameter by > 50%. CAAs are very rare, and the overall incidence is 0.3–5.3% [1].

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Left main CAAs are extremely rare, with an incidence of 0.1% found by Topaz et al. among 22,000 coronary angiograms [2]. The lowest incidence of 0.02% is shown for giant CAAs, while the highest incidence of 5.9% is reported for those associated with congenital artery fistula [3]. "Giant aneurysms" are those whose diameter exceeds 2 cm [1–3]. Owing to their rarity, there are no unique guidelines for their treatment. Therefore, case reports are important for discussing individual strategies. We present a patient with symptomatic giant left main CAA presented as acute myocardial infarction and severe left ventricle dysfunction.

Case report

A 66-year-old female patient was admitted to the emergency department of our clinic due to chest pain and dyspnea. Acute non-ST-elevation segment myocardial infarction (non-STEMI) of anterolateral localization was diagnosed [creatine kinase max 1,111 U/L, normal range (NR): 0–200 U/L; troponin T 3.754 ng/mL, NR: < 0.3 ng/mL], complicated with acute heart failure. She had never been treated for cardiovascular disorders before. According to her past medical history, she has been suffering from hypertension and hypothyreosis for a long time; both were under medication control. Physical examination revealed the following: inspiratory crackles; normal loudness of heart sounds with gallop rhythm of 120 bpm; a systolic regurgitation heart murmur was heard at the apex and parasternal area; blood pressure was 110/80 mmHg. She was monitored closely.

Assessment

Electrocardiography findings showed sinus tachycardia with ST depression in several leads: DI and aVL, V2–V6, and slight ST-elevation in aVR. On chest X-ray, an enlarged heart silhouette and pulmonary congestion were revealed. Echocardiography revealed signs of ischemic cardiomyopathy with hypo- to akinetic septum, lateral and anterior wall, reduced ejection fraction (EF) 25–30% (NR ≤ 50%), and significant secondary mitral regurgitation (3–4+). Heart catheterization and coronary angiography revealed a giant saccular, 3.5 × 3.5 mm left main CAA full with thrombi, compressing the proximal segments of the left anterior descending (LAD) and circumflex (Cx) artery (Figure 1). Prior to decision of the further treatment, a high-resolute computed tomography imaging study was performed. The giant saccular thrombotic left main CAA also completely compressed the origin of the LAD and Cx coronaries (Figures 2 and 3). We also performed immunology analyses to exclude vasculitis.

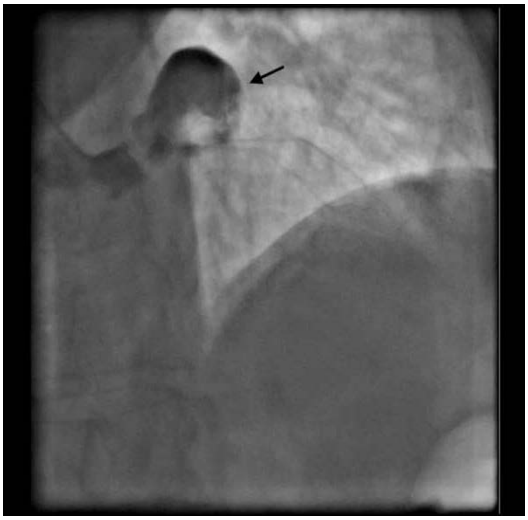


Fig. 1 – Coronary angiography shows a large left main coronary artery aneurysm (arrow), full with thrombus, compressing left anterior descending and circumflex coronary artery.

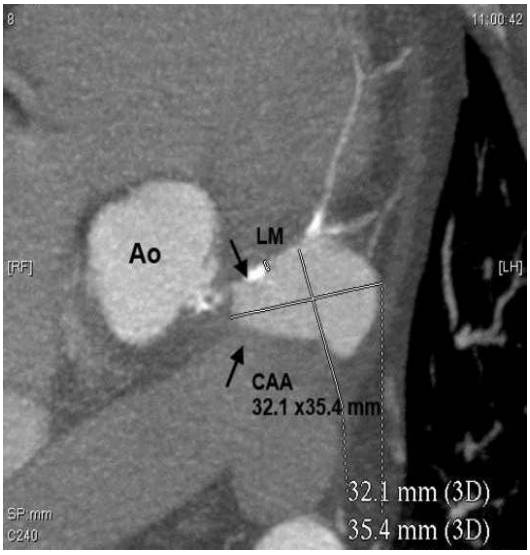


Fig. 2 – Multislice computed tomography imaging study confirmation of left main (LM) coronary artery aneurysm (CAA) (lower arrow). Ao – aorta.

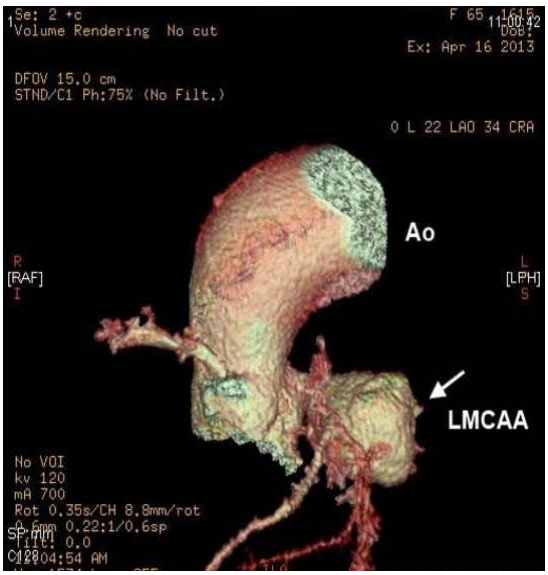


Fig. 3 – Volume rendered computed tomography imaging study of left main coronary artery aneurysm (LMCAA) (arrow). Ao – aorta.

Intervention

The patient was treated with anticoagulation therapy, beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, mineralocorticoid receptor antagonists antagonists, loop diuretics, and statins. When the stable condition was achieved with complete resolution of acute myocardial injury, cardiac surgery was performed with resection (Figure 4 A and B) and aneurysmorrhaphy and two by-passes: left internal mammary artery (LIMA) on LAD and saphenous vein graft – on first obtuse marginal (OM) branch. The histopathology of the CAA wall suggested that the etiology was an atherosclerotic change.

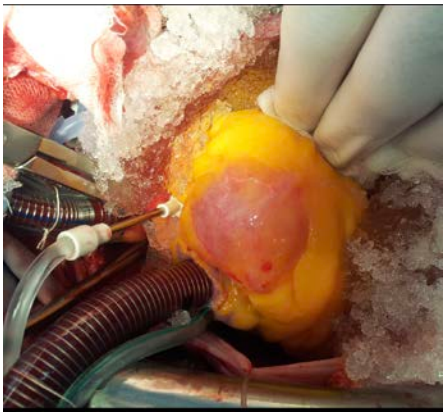
Outcomes

Control computed tomography imaging study was performed one month after the operation (Figure 5). It showed patency of LIMA graft and advanced atherosclerotic lesions on saphenous vein graft on the first OM branch, without significant stenosis. Clinically, the patient was relieved of symptoms despite the persistence of poor EF (ischemic dilated cardiomyopathy and heart insufficiency).

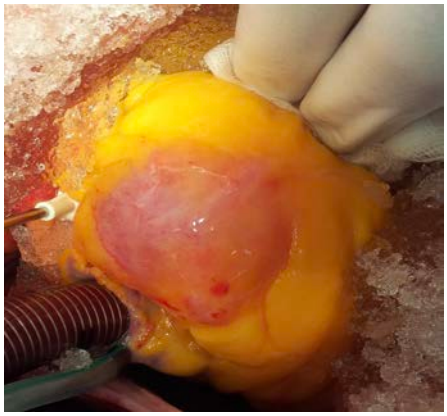
Discussion

CAAs are rare anomalies with various incidences regarding localization. The most common anomalies are the right CAAs (rCAAs) (40%), followed by the LAD artery (32%), the left Cx artery aneurysms (17%), and the left main CAAs (3.5%) as the rarest localization [4, 5]. CAAs can be divided into true and false (pseudoaneurism) depending on vessel wall composition. They can also be stratified as fusiform (longitudinal diameter is larger than transverse diameter) or saccular (transverse diameter is larger than longitudinal diameter) [5, 6].

The majority of them have atherosclerotic origin and account for > 90% of CAAs in adults [2]. Other causes are inflammatory vascular disorders (systemic lupus erythematosus, polyarteritis nodosa, Kawasaki disease, etc.) and connective tissue disorders (Ehlers-Danlos syndrome, Marfan's syndrome, scleroderma). Some infectious diseases, such as infective endocarditis, can also cause CAAs, as described in a few case studies [7]. Congenital etiology of aneurysms is usually accompanied by coronary artery fistulas [8]. In our case, we excluded systemic vascular etiology performed by immunology analysis, and pathohistology confirmed atherosclerotic origin.



A



B

Fig. 4 – Giant left main coronary artery aneurysm during surgery (A) and evacuation (B).

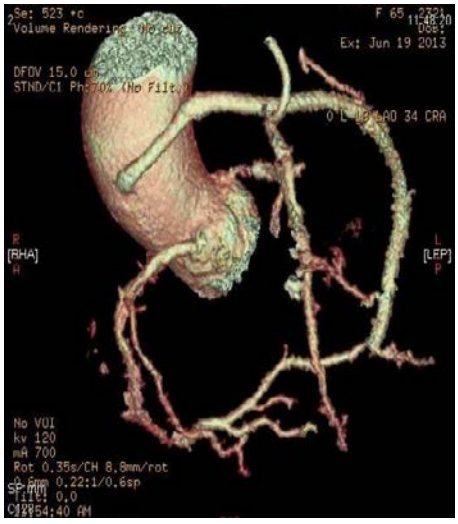


Fig. 5 – Volume rendered computed tomography imaging study after one month of surgical treatment.

Increasing the use of percutaneous interventional treatment in cardiology is also one of the common causes of CAA. Injury of the media of blood vessels due to the extension of the atherosclerotic process and the percutaneous procedure is the probable pathologic mechanism. It has been noticed that balloon dilatation with inadequate balloon size leads to CAA over time. Furthermore, patients with dissection after percutaneous coronary intervention (PCI) have a higher chance of developing CAA. It was also described that first-generation drug-eluting stents could precipitate coronary aneurysms. Over time after implantation, embedded drug stent (sirolimus or paclitaxel) can consequently cause hypersensitive and inflammatory reactions with wall weakness and dilatation [1, 2].

Clinical presentations can be various. Small CAAs are usually asymptomatic. Sometimes they disguise as an anterior mediastinal mass [9]. Myocardial ischemia could be developed due to spasms of the affected artery or distal embolization from thrombotic lesions in the CAA. If these are frequent, large infarction could develop, followed by signs and symptoms of heart failure, as described in our case. CAAs are also prone to progressive enlargement, possible rupture, and potential development of hematopericadium or cardiac tamponade [2].

Diagnostic tools could be invasive and noninvasive. Echocardiography is a noninvasive method for diagnosis of the left main coronary aneurysm, but it can visualize just the proximal segments of coronary arteries [4, 5]. Multidetector computed tomography of coronary arteries is one of the imaging methods for diagnosis, but the "gold standard" is invasive coronary angiography, especially along with the concomitant intravascular ultrasound use. They give us information about the size, shape, location, presence, and number of coexisting anomalies, as well as information about CAA wall structure. It can also suggest atherosclerosis as the underlying condition [4].

Currently, the optimal management strategy of CAAs remains challenging due to the lack of published outcome data. The evidence suggests that asymptomatic small CAAs may not require any treatment, just optimal medication therapy and regular follow-up. The final decision should be individualized depending on size, location, and clinical context. In symptomatic patients marked as unsuitable for PCI, surgical excision is the preferred option [10].

All patients should receive an aggressive modification of coronary risk factors whether or not obstructive coronary artery disease is present [10]. Medicament treatment alone encompasses observation, anticoagulation therapy, and antiplatelet therapy in case of atherosclerotic etiology. Yan et al. [11] suggest that novel oral anticoagulants may be suitable for CAA treatment. Some authors emphasize the important role of elevated levels of matrix metalloproteinase-3 in CAA's development; therefore, statin therapy is of crucial importance [12]. Avoiding nitrates in these patients due to possible steal syndrome and exacerbation of angina is also advocated. Gulec et al. [13] found that the polymorphism of the ACE gene is a potent risk factor for CAA. Thus, the use of ACE inhibitors in CAA's therapy to prevent dilatation progression is advocated; however, it has not been proven yet. Accordingly, our patient received a proper medication treatment with significant symptom improvement. Nevertheless, pharmacotherapy is the only prevention of possible complications of CAA, but definite treatment should be either percutaneous stent implantation or surgical treatment. Still, limited evidence is available on the effectiveness and safety of this interventional practice. Yet, there is limited evidence available on the effectiveness and safety of percutaneous interventional practice.

Treatment of CAAs with covered stents has been reported in several case reports; however, there is limited evidence available on the effectiveness and safety of this interventional practice [14]. Burzotta et al. [15] suggested the use of the self-expandable Symbiot PTFE-covered stent for percutaneous treatment of a large coronary aneurysm.

Win et al. [16] described a case of a large fusiform aneurysm, successfully treated with a technique of stent-assisted coil embolization. Dai et al. [17] suggest that multiple overlapping stents might be a promising therapeutic target for CAAs. Some authors also described interventional complications as prolapse of the covered stent into CAA due to significant LAD motion during the cardiac cycle [18]. However, surgery is the preferred way of treatment in patients with giant, symptomatic CAAs [19, 20].

Indications for surgery treatment are the following: severe coronary artery disease, CAAs near the bifurcation of large branches, evidence of emboli from the aneurysm to the distal coronary bed resulting in myocardial ischemia, progressive enlargement of a CAA documented by serial angiographic measurements during follow up, CAAs in the left main stem, complications such as fistula formation or compression of cardiac chambers, and giant CAAs [2]. In our case, surgical treatment was undertaken due to the giant size and specific localization of CAA with compression of proximal segments of LAD and Cx arteries and evidence of embolization as well.

There are various surgical strategies, such as resection, reconstruction, and exclusion with a bypass operation. Since there is no typical surgical approach for giant coronary aneurysms, the decision has still been made on an individual basis [20]. The prognosis of CAA depends on the size of the aneurysm. Small aneurysms have a favorable prognosis with a low risk of myocardial ischemic events and/or mortality and giant CAAs have a worse prognosis due to common complications. Baman et al. [21] predicted a 5-year mortality rate of 29.1% among patients with CAAs. They found no statistically significant difference between the survival curves of aneurysmal patients with and without obstructive coronary artery disease.

Conclusion

Giant left main CAAs are rare pathologic findings and there are no established principles for treatment. Although the percutaneous way of treatment is now available in selected cases, the surgical approach is still preferred for these patients.

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