POSTER PRESENTATIONS

[PP-5]

Right Coronary Artery Post-Stenotic Giant Saccular Aneurysm that is Managed by Graft Stenting after Revascularization of Aneurysmatic LAD Lesion for Acute Anterior STEMI

Emine Büşra Kıtlık, Sabiye Yılmaz, Hasan Çağlayan Kandemir

University of Health Sciences Türkiye, Kocaeli City Hospital, Kocaeli

Coronary artery aneurysms (1.5 times dilation the reference-vessel) are uncommon and have been diagnosed with increasing frequency with coronary angiography. The frequency of CAA varies widely, from 0.3% to 5%, and complications include thrombosis, embolic phenomena, arteriovenous fistulisation, spasm, rupture with haemopericardium and sudden death. We present a case giant saccular RCA aneurysm that requiring urgent revascularization by graft stenting without surgicalü intervention after successful culprit LAD revascularization in 65 year old patient for anterior STEMI.

Sixty-five-year old woman with hypothyroidism with in her mother's medical history involving cranial berry aneurysm had been presented with chest pain and had been admitted to our clinic by emergency transportation after diagnosis of anterior STEMI, ECG showing widespread v1-v6 st segment elevation. Coronary angiography was showing proximal lad containing 100% stenosis after successful stenting for culprit vessel patient was followed in our coronary intensive care unit. Post MI heart failure developed and echocardiographic imaging showed ejection fraction 35% anterior and apical segments had aneurysmatic dilatation. Patient had been evaluated by cardiac surgery department for RCA giant aneurysmal post-stenotic saccular aneurysm but before evaluation patient rhythm showed ventricular fibrillation that followed by cardiac arrest and resuscitation. Because of high-risk and clinical deterioration patient had been evaluated for high-risk PCI for rca giant saccular aneurysm after extubation. Informed consent was taken from her and first degree relatives for high-risk procedure. Patient was taken to cath lab by femoral access percutaneus coronary intervention was performed succesfully. RCA proximal 95% stenosis and poststenotic saccular aneurysm, RCA distal had retrograde flow from LAD. Predilatation by 1.5x20 mm and 2.75x25 mm SC ballons were applied.Without occlusion of conal branch and rv branch of rca 3.5x35 firehawk stent implanted to RCA lesion after that covering rca saccular aneurysm bentley graft stent 3.5x24 mm was implanted. Control angiography showed small leakage proximal to graft stent. Another graft stent 3.5x16 mm was implanted to proximal to first graft stent. Post dilatation performed by 4.5x15 mm NC balloon. Control angiography didn't show any leakage and procedure was successfully ended.

Coronary aneurysms are observed in 0.15-4.9% of patients undergoing coronary angiography. We present a case of anterior myocardial infarction with post MI heart failure. CAAS are classified mainly based on the shape and two different types have been proposed: a saccular aneurysm and fusiform aneurysm. The first has a spherical-shaped distension, characterized by a transverse diameter > longitudinal diameter; in the second type, the longitudinal diameter is larger than the transverse diameter, describing a spindle shape. The risk factors for developing a CAA are atherosclerosis, inflammation, and non-inflammation causes. Atherosclerosis is the most frequent risk factors in adults, reaching a frequency of 50% in all CAAs, independent of the presence of coronary artery plaque. Hormonal and genetic causes are reported in literature. Phaeochromocytoma is related to coronary artery aneurysm. Von Hippel Lindau and MEN syndromes that are related to phaeochromocytoma present coronaryand intracranial aneurysm of arteries. CAAs can present in other syndromes, such as connective tissue disease (Marfan syndrome, Ehlers-Danlos syndrome,fibromuscular dysplasia, neurofibromatosis), vasculitis (lupus, rheumatoid arthritis, ankylosing spondylitis, scleroderma), infections (human immunodeficiency virus, bacterial, mycobacterial, syphilis, Lyme disease, mycotic aneurysm, septic emboli), drug use (cocaine, amphetamine, protease inhibitors), neoplastic lesions, and cardiac lymphoma. In addition to these risk factorsthere is also risk of developing coronary artery aneurysm after PCI with coronary artery drug eluted stenting and drug eluted ballons. Treatment options are cardiac surgery, PCI and medical treatment. Medical treatment with DAPT showed lower ratemortality in coronary artery aneurysm registry study, which conducted an average follow-up of 37.2 months in approximately 1,500 patients. CABG is suitable for the aneurysms involving especially involving large collateral branches; left main aneurysm; giant or multiple aneurysms; mechanical complications, compression of structures, or fistula formation;signs of rupture; Kawasaki disease or infected aneurysms; symptomatic venous graft aneurysms; or those causing significant down stream flow reduction. Aneurysm and other coronary stenoses can be treated by either a PTFE-covered stent, alongside other drug-eluting stent implantations in tracts outside the aneurysm, or the so-called "stent-assisted" embolization, which involves percutaneous treatment using membrane-covered stents and coil embolization, with a strong limitation in patients with large or multiple CAAs. Another described off-label technique is the use of carotid stent implantation for the treatment of CAAs but data is limited.

Keywords: Giant coronary saccular aneurysm, graft stenting, coronary aneurysm

Figure 1. RCA saccular aneurysm

Figure 2. RCA saccular aneurysm graft stenting leakage closure after stenting

[PP-6]

Spontaneous Coronary Artery Dissection Complicated by Massive Thrombosis in a Patient with Concomitant Systemic Lupus Erythematosus, Sjögren's Syndrome and Secondary Antiphospholipid Syndrome: a Case Report

Mahsa Khanmohammadi[1], Hasan Burak İşleyen[1], Ahmet Bacaksiz[1], Nusret Açıkgöz[1], Oğuzhan Abanoz[1], Esra Danışman[1], Kamran Kerimzade[1], Lina Boukhemis[1], Sezgin Uzunoğlan[2], Behice Hande Şişman Uzunoğlan[2]

[1]*Department of Internal Medicine, Bezmiâlem Vakıf University Faculty of Medicine, İstanbul*

[2]*Clinic of Internal Medicine, Kırklareli Training and Research Hospital, Kırklareli*

Aim: Spontaneous coronary artery dissection (SCAD) in the setting of systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS) can be a rare but severe mechanism of acute coronary syndrome (ACS). This article presents a case of SCAD complicated by massive thrombosis in a patient with SLE and Sjögren's syndrome.

Case Report: A 51-year-old female presented with sudden-onset chest pain; she was on maintenance methylprednisolone 16 mg/day. Electrocardiography revealed acute inferolateral ischemic changes. High-sensitivity cardiac troponin I levels increased from 1,240 ng/L to 18,500 ng/L (reference <14 ng/L). C-reactive protein was 54 mg/L, and erythrocyte sedimentation rate was 72 mm/hour. Complement C3 and C4 levels were 1.8 g/L and 0.5 g/L, respectively. Lupus anticoagulant (LA) was positive, and anticardiolipin IgG/IgM and anti-beta-2 glycoprotein I (anti-β2GPI) IgG/IgM were found at high titers (>40 GPL/MPL; >40 SGU/SMU), consistent with APS (re-confirmation planned after 12 weeks). Emergent coronary angiography revealed Saw Type 1 SCAD in the right coronary artery with an initial thrombolysis in myocardial infarction flow grade of 1; no significant atherosclerosis was observed in other vessels (were not utilized). During the procedure, rapid development of a superimposed massive intraluminal thrombus on the dissected segment and flow restriction were observed. Due to these complications, 3.0×48 mm and 3.0×12 mm drug-eluting stents were implanted. Tirofiban (intracoronary bolus+24-hour IV infusion) and unfractionated heparin 10 U/kg IV bolus were administered. Echocardiography demonstrated: LVEF: 40%, inferior and inferolateral walls are severe hypokinetic, LA: 30 mm, AoS: 30 mm, LVED: 40 mm, LVES: 32 mm, IVS: 8 mm, mild MR. Although a conservative approach is generally preferred in SCAD, percutaneous intervention became mandatory due to ongoing ischemia/flow restriction and rapid thrombus propagation secondary to APS. Regarding immunomodulation following myocardial infarction, abrupt cessation of steroids was avoided considering the risk of an SLE flare; the lowest effective dose and a steroid-sparing approach were planned in consultation with rheumatology. Warfarin was preferred at discharge due to the high thrombotic risk; early de-escalation to dual therapy (warfarin+clopidogrel) was planned after one week of triple therapy (warfarin+aspirin+clopidogrel) by discontinuing aspirin.

Conclusion: In the presence of concomitant autoimmune disease and APS, SCAD can be complicated by massive thrombosis, necessitating aggressive revascularization and antithrombotic strategies; the management approach must be multidisciplinary and individualized.

Keywords: Spontaneous coronary artery dissection, antiphospholipid syndrome, systemic lupus erythematosus, Sjögren's syndrome, thrombosis

Table 1. Key diagnostic parameters

Parameter	Value	Reference / Note
hs-cTnI (peak)	18,500 ng/L	Reference <14 ng/L
CRP	54 mg/L	Elevated
ESR	72 mm/hour	Elevated
Complement C3	1.8 g/L	Low-normal
Complement C4	0.5 g/L	Low-normal
Lupus Anticoagulant	Positive	-
aCL IgG/IgM	>40 GPL/MPL	High titer, APS criteria
anti-β2GPI IgG/IgM	>40 SGU/SMU	High titer, APS criteria
LVEF (Echo)	40%	Reduced
Wall Motion	Inferior/Inferolateral hypokinesis	Severe
Initial TIMI flow (RCA)	Grade 1	Severely reduced
Post-stent TIMI flow	Grade 3	Restored

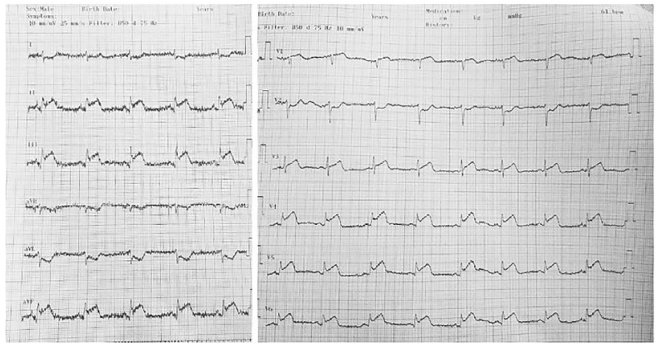

Figure 1. Admission 12-lead electrocardiogram demonstrating acute inferolateral ischemic changes

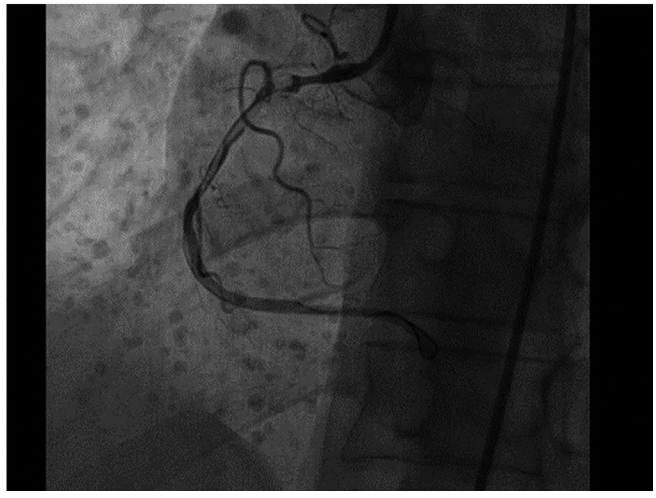

Figure 2. Baseline coronary angiography revealing. Saw type 1 spontaneous coronary artery dissection in the right coronary artery with an initial TIMI 1 flow

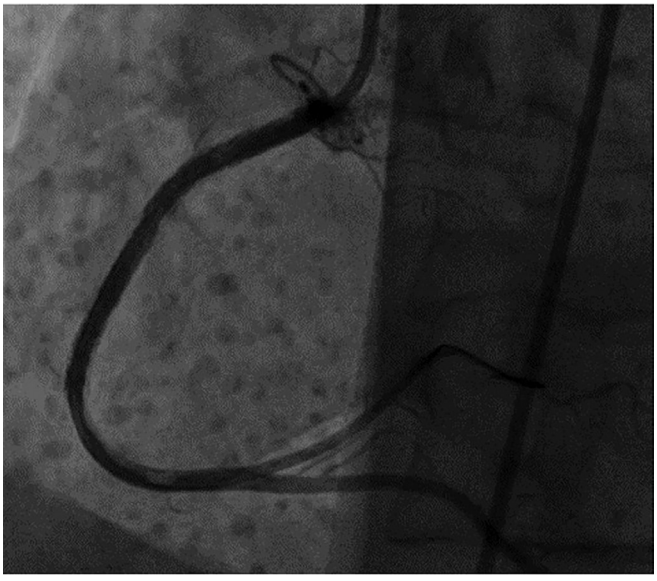

Figure 3. Intra-procedural coronary angiography demonstrating the rapid development of a superimposed massive intraluminal thrombus at the dissection site

Figure 4. Final coronary angiography showing successful implantation of drug-eluting stents and restoration of TIMI 3 epicardial blood flow following intracoronary tirofiban administration

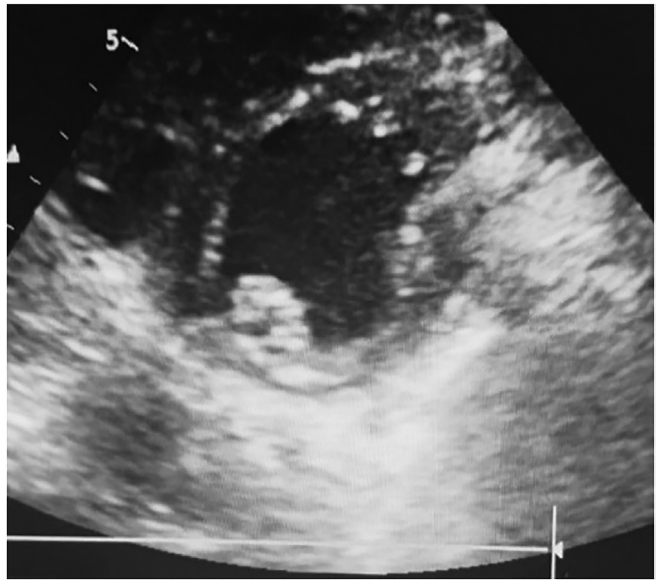

Figure 5. Transthoracic echocardiography demonstrating reduced left ventricular ejection fraction wall motion abnormality