A Case of Stentocarditis, Rare Complication of Percutaneous Coronary Intervention

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INTRODUCTION

Fever occurs sometimes, usually transiently after coronary catheterization. We report a very rare case that suggests persistent high fever and systemic inflammation caused by stentocarditis induced by percutaneous coronary catheterization in a chronic total occlusion of the right coronary artery. The most important point to remember is that timely detection requires a high index of suspicion and early appropriate treatment is very important in preventing mortality.

PRESENTATION OF CASE

A 48-year-old male presented with unstable angina 6 months back, ECG at presentation showed sinus rhythm, poor R wave progression in lead V1-V3, Chest X-ray showed cardiomegaly, and subsequent evaluation showed positive treadmill test in stage 2 (Bruce protocol). Echocardiography showed LVIDd 54 mm, LVIDs 43 mm, EF 40 % and hypokinesia of basal, mid anterior wall segments and entire interventricular septum. Coronary angiography revealed a normal left main coronary artery, two discrete tandem lesions producing 90 % luminal occlusion in the proximal left anterior descending artery before the first diagonal, and no flow-limiting lesions in the left circumflex artery, which was non-dominant. The right coronary artery was dominant and showed a mid chronic total occlusion with retrograde filling of distal RCA.

Proceeded with angioplasty, left coronary artery engaged with 6F Judkins left 3.5 guide catheter, LAD wired with whisper extra support coronary wire, lesion predilated with 2.5 x 12 mm semi-compliant balloon at 12 atm pressures, stented with 3 x 36 drug-eluting stent and post dilated with 3x15 noncompliant balloon at 18 atm pressures, with final TIMI3 flow.

RCA engaged with 7FR guide catheter, CTO crossed with 0.014” progress 120 coronary wire, through microcatheter (0.014”). Serially dilated with 1.25 x 10 mm followed by 2 x 15 mm semi-compliant balloon at 12 atm pressures. Distal RCA stented with 2.5 x 40 mm DES deployed at 10 atm pressures. Proximal RCA stented with 3 x 44 mm DES deployed at 14 atm pressures, with TIMI3 flow post-procedure.

The post-procedure period was uneventful. As it was an uncomplicated angioplasty, he was discharged on the next day, with aspirin (75 mg), ticagrelor (90 mg BD), atorvastatin (40 mg), and oral hypoglycaemic and oral diuretics. Blood pressure and heart rate were optimum. Post-procedure haemoglobin, blood sugars and biochemical parameters and troponins were normal. No features of right femoral artery puncture site infection.

He had short febrile illness on the 6th post-procedure day and was admitted to medical ward, his baseline blood counts and viral markers were normal, urine microscopy normal, Covid RTPCR repeatedly negative. He again presented with a high fever (102 F) with chills and rigours, nausea & vomiting and started on oral amoxicillin-clavulanic acid and was admitted for evaluation.

On readmission, ECG showed old changes, echocardiography showed hypokinesia in the anterior wall segments as before; no pericardial effusion, no vegetations over native valves. He continued to have spikes of fever and elevated acute phase reactants.
On the 4th day of admission, his echocardiography showed mild pericardial effusion (9 mm anterior to the right ventricle and 12 mm posterior to left ventricle with fibrin strands), no features of tamponade and mild left pleural effusion, on admission, the chest X-ray was normal.

Pleural fluid study showed straw-coloured fluid with 1218 cells, P27 L73. Albumin 1.8 (normal), protein 3.8 (normal) LDH 99 (serum LDH 551), pleural fluid was negative for malignant cells, not much contributory.

He had no rashes, arthritis or blue toes in the extremities.

Ultrasound abdomen showed hepatomegaly otherwise normal.

After admission, his acute phase reactants were on the rising trend (ESR 84 mm/1st hour, CRP 20 mg/dL) which was monitored during the hospital stay.

Blood counts were normal with reduced Hb (8.7 gm %) and normal PCV. Biochemical parameters remained normal.

Blood culture and urine culture were sterile, viral markers were negative, periodic echocardiography showed no vegetation, pericardial effusion gradually increased, but no features of tamponade, hence not aspirated. No intracardiac shunts. Modified apical 4 chamber view showed hugely dilated RCA with echodense shadows suggestive of possible RCA pseudoaneurysm with stent shadow (figure 1). The same affected region with the large aneurysm is clearly shown in the TEE image in figure 2.

Clinical Diagnosis and Management
Considering all the above factors, and after excluding most of the common infections by the necessary investigations, the diagnosis of stentocarditis was highly likely. Hence after taking blood and urine samples for culture, he was started on injection meropenem and linezolid, he improved symptomatically, and became afebrile by the 10th day of antibiotics.

Major differential diagnoses considered were chronic infections with acute presentations like tuberculosis and underlying malignancies, which were excluded with necessary investigations.

CTCAG was done on the 10th day of admission which confirmed the diagnosis of stentocarditis.

The patient clinically improved with treatment. After 1 month of antibiotics, he was taken up for diagnostic coronary angiography, which showed a patent stent in the left anterior descending artery, normal left system and large pseudo aneurysm (41x16 mm) involving the proximal and mid-right coronary artery (figure 3). PDA and PLB were normal. As he had a large pseudoaneurysm, we sought cardiovascular surgery consultation and planned to proceed with surgery.

Surgery was done by median sternotomy. Dense adhesions were noted between the heart and pericardium. 6 x 5 x 5 cm aneurysm was noted in relation to the proximal and mid-right coronary artery, with a stent freely floating inside (figure 4). There was diffuse oozing from raw areas. The procedure was done under cardiopulmonary bypass. Aneurysm opened, thrombus and nonviable tissue inside excised, both stents in the right coronary artery retrieved fully, thorough saline lavage was given, and both proximal and distal ends of the RCA sutured. Aneurysm scar excised and edges closed with continuous sutures. Saphenous vein graft was done to the distal RCA which was a 1.5 mm vessel with good flow. Came off CPB in stages. Hemodynamics were achieved, layers closed routinely, and the patient was stable at the end of the procedure.

At one month follow up, the patient was monitored for clinical symptoms, biochemical parameters and echocardiography. He was fever-free, acute phase reactants had come down and routine blood investigations were within the normal limits. Transthoracic echocardiography showed a thin rim of pericardial effusion posteriorly, hypokinetic RCA territory in addition to anterior wall segments and satisfactory LV systolic function. He is on regular medications and follow-up.

Legends

![Figure 1: 2D Echocardiography Image Showing a Hugely Dilated Segment of RCA, and Echodense Stent Strut](image1)

![Figure 2: TEE Image of the Affected Region Tilted 4 Chamber View Showing the Large Pseudo Aneurysm](image2)

![Figure 3: Angiographic LAO View of RCA Showing Large Aneurysm Measuring, 41x16 mm](image3)
Fever sometimes occurs usually transiently after coronary catheterization. The aetiologies of fever are thought to be local and systemic infection, allergic reactions against contrast material, inflammatory reactions against hematoma, and embolic reactions such as blue toe syndrome. We thought that the persistent high fever and systemic inflammation of this patient might be caused by an inflammatory response in the arterial wall, after percutaneous coronary stenting which was confirmed by further investigations. The persistent high fever decreased, after the institution of appropriate antibiotic therapy, the increased CRP and cytokines recovered to the normal range.

Various data in the literature on coronary artery stent infections suggest that early-onset infections (<10 days after stent implantation) are potentially amenable to medical therapy alone, but late-onset infections (≥10 days after implantation) or major complications necessitate combined surgical and medical therapy.

The evaluation of a patient with a potential stent infection has several goals. These include making a clinical, radiographic, and microbiologic diagnosis; detecting complications and determining optimal therapy. The clinical presentation of patients with stent infections is generally consistent. In particular, fever, chest pain, or both occurs in most of the patients.

There is a criterion proposed by Dieter to determine the diagnosis of coronary stent infection.[3] A definitive diagnosis was made by autopsy or by examination of surgical material. For possible diagnosis, 3 of the following criteria must have been present: placement of a coronary stent within the previous 4 weeks; multiple repeat procedures performed through the same arterial sheath; the presence of bacteraemia, significant fever, or leukocytosis with no other cause; acute coronary syndrome or positive cardiac imaging.

A definitive diagnosis of a stent infection is based on the presence of an abscess or inflammatory mass, or an aneurysm or pseudoaneurysm.

Expected findings in patients with coronary stents, which we refer to as vessel pathology, included local abscesses and simple pericarditis. These findings are rarely clinically significant. In contrast, major complications are unexpected, potentially catastrophic events, which include pericardial empyema, purulent pericarditis, vessel perforation, destroyed vessel, and myocardial rupture.

On reviewing the various works of literature, TEE was diagnostic in most of the cases.[2] The angiographic study should be considered as the procedure of choice, in conjunction with TEE, in making the primary diagnosis and detecting catastrophic complications.[3]

Various reviews indicate that the development of a major complication, the time of onset of infection, and the type of therapy-medical therapy alone or combined with surgery can dramatically affect the outcome.[4] Generally, early-onset infections appeared to be amenable to medical therapy alone. Medical therapy consists of broad-spectrum antibiotics. Surgical intervention includes stent removal if possible, and abscess drainage or perforation repair when indicated.

In our patient, we detected the stent infection and instituted timely appropriate antibiotics early in the course of the disease. As the aneurysm was large, we proceeded with resection of the aneurysm and removal of the infected stents. The patient improved and did well at the end of one month.

### Final Diagnosis

Stentocarditis; Coronary artery disease; double vessel disease; Post PTCA to LAD and RCA Moderate Left ventricular systolic dysfunction; Sinus Rhythm NYHA class II.

### References

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