INFECTIONS IN THE HEART
ENDOCARDITIS AND BEYOND

Very Late Coronary Stent Infection and Abscess following Staphylococcus aureus Bacteremia

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INTRODUCTION

Despite widespread use of percutaneous coronary intervention (PCI) and stenting, infective complications are rare.1-2 Coronary stent infection is difficult to treat and is potentially disastrous, with a high mortality rate.1,3 We present a case of very late coronary stent infection with a large abscess 5 years after coronary stent implantation.

CASE PRESENTATION

A 50-year-old woman was admitted to our institution with dyspnea and pleuritic left-sided chest pain with a background of coryzal symptoms for 1 week. She had a very complex medical history including type 2 diabetes mellitus, diabetic nephropathy and end-stage renal failure on hemodialysis, hypertension, and paroxysmal atrial fibrillation. She also had known ischemic heart disease with a left anterior descending artery (LAD) stent implantation (Xience Prime, Gurnee, IL) 5 years prior.

Investigations at the time showed sinus tachycardia on electrocardiography with subtle T-wave inversion in lateral leads (1 and aVL), raised serum inflammatory markers (white cell count $13.76 \times 10^6$, C-reactive protein 590), and high troponin levels (3,130 ng/L). Computed tomography pulmonary angiography was negative for pulmonary embolus but showed small pericardial effusion and pulmonary consolidation. Her transthoracic echocardiography (TTE) showed mild global left ventricular systolic dysfunction (left ventricular ejection fraction of 45%-50% by biplane Simpson’s method) and abscess also grew proximal to the stented segment. Microbiological culture of the stent revealed $S. aureus$ sensitive to dicloxacillin, and treatment was instituted immediately with intravenous fluoroquinolone and a renally adjusted dose of vancomycin.

Repeat coronary angiography was performed, which demonstrated a large aneurysmal dilatation arising from the stented segment of LAD (Figure 1B, Video 4). There was no significant in-stent stenosis and no other new coronary lesions. We decided not to proceed with intracoronary imaging due to the potential risk of perforation or rupture of the aneurysmal segment with coronary instrumentation. Cardiovascular magnetic resonance imaging with gadolinium contrast was contraindicated, and she was treated as myopericarditis and type 2 myocardial infarction.4 Outpatient follow-up TTE in 1 week showed reduction in pericardial effusion and improved serum inflammatory markers, and the patient reported no symptoms.

Twenty days later, the patient was transferred from a regional hospital with chest and left shoulder pain, reduced exercise tolerance, and fever of 39°C. On initial examination she was afebrile and had no cardiac murmurs. She had a raised serum C-reactive protein level of 340 and troponin level of 11,200 ng/L, and her electrocardiography showed deep T-wave inversion in anterolateral leads. Repeat TTE showed new severe hypokinesia of the basal to mid anterior and inferior septum (Videos 2 and 3). Three sets of blood cultures grew $S. aureus$ sensitive to dicloxacillin, and treatment was instituted immediately with intravenous fluoroquinolone and a renally adjusted dose of vancomycin.

Due to the scope of cardiac involvement, size of coronary aneurysmal dilation, and control source of infection, the cardiothoracic surgeon undertook an urgent thoracotomy with a left internal mammary graft to the mid LAD, drainage of abscess and excision of part of the LAD stent, resection of mycotic aneurysm, and ligation of the LAD proximal to the stented segment. Microbiological culture of the stent and abscess also grew $S. aureus$, but unfortunately histopathology was not requested on the specimen. This is a limitation as it is not clear whether the aneurysmal dilation was a true aneurysm, pseudoaneurysm (Figure 2, Video 5). The likely source of staphylococcal bacteremia was believed to be from home hemodialysis and an unsterile technique. The patient did not have any other identifiable source of infection, recent surgeries, or dental procedures. Another potential source of infection could be from intravenous cannula insertion during her initial admission at which she was treated with intravenous antibiotics for pneumonia. The patient...
was maintained on intravenous flucloxacillin for 6 weeks. This admission was complicated by post-operative tamponade requiring pericardial washout, thrombosed arteriovenous fistula requiring revision, and skin pressure sores that were managed with wound care. At 6- and 12-month follow-up, the patient remained well with no recurrence of sepsis, and the inflammatory markers normalized.

DISCUSSION

Coronary Artery Aneurysms

Coronary artery aneurysms are rare, with an overall incidence of 0.3%-5.3%. The pathophysiology of coronary aneurysm remains controversial, and it is generally attributed to vessel wall weakening and subsequent dilatation. The most common cause of coronary aneurysm is atherosclerosis, with connective tissue disorders and vasculitis (such as Kawasaki disease) being rare causes of coronary aneurysms. Infections rarely lead to coronary aneurysm as a result of direct invasion of pathogens from surrounding structures, septic emboli, or immune complex deposition. Percutaneous coronary intervention and drug-eluting stent (DES) insertion also were reported to contribute to formation of coronary aneurysm due to vessel wall trauma, intimal dissection, and delayed endothelialization as a result of stent malapposition or the antiproliferative nature of DES coating.

An Uncommon but Catastrophic Complication of PCI

An increasing number of PCIs are performed annually; however, coronary stent infection and myocardial abscess are exceedingly rare. In a literature review from 1987 to 2012, a total of 17 cases of coronary stent infection were reported, but the mortality rate has been reported to be as high as 38%-47%. Coronary stent infections most commonly occur within 4 weeks of index procedure (88%), and Staphylococcus aureus is the most common organism involved (86%-88%). It is believed that early coronary stent infection is a result of equipment contamination or another concurrent distant source of infection at the time of stent insertion. Bacteria can adhere to the stent and spread to the arterial wall, initiating an inflammatory response leading to necrosis. Endothelialization of stent struts may play an important role in the prevention of stent infections. However, it is uncertain whether infection prevents stent endothelialization or poor endothelialization leads to bacterial adherence and stent infection. Drug-eluting stents delay endothelialization more than 6 months, and this may lead to uncovered endoluminal struts, creating an ideal focus for bacterial adherence. This suggests another possible complication of stent underexpansion and its resultant delayed endothelialization.

This case of Staphylococcal aureus coronary stent infection years after PCI is unique because it was the longest interval from the index coronary procedure to the manifestation of symptoms, it was promptly diagnosed based on abnormal coronary angiography and echocardiography, and, importantly, we successfully treated the

**Figure 1** (A) Coronary angiogram posteroanterior/cranial view showing a widely patent stent in LAD (yellow arrow). (B) Coronary angiography posteroanterior/cranial view after 1 month showing aneurysm formation at the stented segment of LAD (red arrow).
extensive infection with prolonged intravenous antibiotics and surgical debridement. We believe the likely cause of coronary stent infection in this case was a distant source of infection and bacteremia along with either septic emboli or seeding of bacteria on incompletely endothelialized stent struts. However, it is not possible to prove this hypothesis without intracoronary imaging.

**Diagnosis**

Diagnosis of coronary stent infection is definitive if the diagnosis is made by autopsy or by examination of surgical material. Possible diagnosis is made if three of the following criteria are present: placement of a coronary stent within the previous 4 weeks; multiple repeat procedures performed through the same arterial sheath; the presence of bacteremia, significant fever, or leukocytosis with no other cause; acute coronary syndrome; or abnormal cardiac imaging findings.

Diagnosing coronary stent infection and abscess years after stent implantation is formidable and depends on a high index of clinical suspicion, especially if patients present with a combination of cardiac and systemic inflammatory symptoms. A wide range of imaging tools has been employed to diagnose this dangerous condition, and in most cases coronary angiography and echocardiography are sufficient for diagnosis. Occasionally, other imaging modalities such as cardiac magnetic resonance imaging, white blood cell scan, cardiac computed tomography, or positron emission tomography could be beneficial if the diagnosis is undetermined. In this case, coronary angiography was significantly abnormal for a new aneurysmal dilatation surrounding the LAD stent segment, and transesophageal echocardiography supported the diagnosis.

**Treatment**

Management of coronary stent infection is problematic as both conservative and surgical treatments pose a high morbidity and mortality risk. Successful treatment of stent infection with intravenous antibiotics has been reported, but a lethal outcome is unavoidable in up to 50% of patients. Surgical intervention and excision of the infected stent and surrounding material is preferred, but this therapy can carry
a high mortality rate of 38%. It has been suggested that surgery should be considered early in cases of late-onset infections and medical therapy alone in early-onset infections unless significant complications are manifest on imaging or if medical therapy appears to be unsuccessful. Parenteral antibiotic therapy should be maintained for about 4 weeks or possibly longer. Figure 3 provides a summary of infected coronary stent clinical presentation, diagnosis, and treatment.

In summary, we report a rare case of *Staphylococcal aureus* infection involving a DES stent 5 years after the index procedure, which was complicated with coronary mycotic aneurysm and myocardial abscess formation. Delayed endothelialization might have contributed to this very late presentation of stent infection. The infection was successfully treated with excision of coronary aneurysm/stent and prolonged antibiotics therapy.

**SUPPLEMENTARY DATA**

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2021.09.001.

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