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CASE REPORT
Coronary heart disease

A unique case report of mitral valve endocarditis associated with coronary stent infection

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Background Despite increasing use of percutaneous coronary intervention and stenting, septic complications such as coronary stent infections are rare. We report a unique case of mitral valve infective endocarditis and associated coronary stent infection which emerged 6 months after index stent insertion.

Case summary A 56-year-old previously healthy man underwent percutaneous coronary intervention and stenting of left circumflex (LCx) coronary artery in the setting of non-ST-segment elevation myocardial infarction. Six months later, he represented with inferior ST-segment elevation myocardial infarction and was found to have a coronary pseudoaneurysm of stented segment of LCx. The pseudoaneurysm was treated with insertion of a covered stent, and immediately following that he developed sepsis with methicillin-sensitive *Staphylococcus aureus* bacteraemia. Comprehensive work-up resulted in the diagnosis of mitral valve endocarditis complicated by coronary stent infection and myocardial abscess formation. He was managed with initial prolonged systemic antibiotic treatment followed by mitral valve replacement. Post-operative course was uneventful with a short duration of oral antibiotics. At 6-year follow-up, the patient was well with the satisfactory echocardiographic result.

Discussion This is a very rare case of mitral valve endocarditis with extensive cardiac involvement including coronary stent infection and surrounding myocardial abscess. Stents can act as an ideal vector for bacterial adherence from which bacteria could spread to the arterial wall and adjacent myocardium. This case suggests a potential complication of delayed endothelialization and risk of infective complication due to bacterial seeding or embolization.

Keywords Myocardial abscess • Coronary pseudoaneurysm • Mitral valve endocarditis • Case report • Coronary stent infection

ESS Curriculum 2.1 Imaging modalities • 3.2 Acute coronary syndrome • 4.11 Endocarditis

Learning points

- This is a classic case of a rare infection with extensive cardiac involvement, which required a multi-disciplinary team approach to develop an appropriate treatment plan.
- Despite reported high mortality rate of coronary stent infection, our patient survived with a combination of prolonged systemic antibiotic treatment and surgical intervention.
- Diagnosis of coronary stent infection is challenging and requires high index of suspicion and the use of multi-modality imaging.

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Introduction

Despite increasing use of percutaneous coronary intervention (PCI) and stenting, septic complications are rare.[1,2] Coronary stent infection is hard to treat, and potentially catastrophic with a high mortality rate.[1,3] We report a unique case of mitral valve endocarditis associated with coronary stent infection and myocardial abscess.

Timeline

Day 0	Admitted with acute myocardial infarction and underwent left circumflex artery (LCx) stent insertion.
Day 162	Inferior ST-segment elevation myocardial infarction, treated with thrombolysis, and a covered stent proximally for coronary pseudoaneurysm and another overlapping stent distally for distal stent edge restenosis.
Day 163	Fever and sepsis with *Staphylococcus aureus* bacteraemia.
Day 164	Transoesophageal echocardiography (TOE) showed mitral valve endocarditis, echo-free circular region surrounding LCx stent, and inter-atrial septal thickening with echo-lucent density.
Day 164	Computed tomograpgy coronary angiography demonstrating increased density within the epicardial fat adjacent to the LCx stent in keeping with TOE abnormalities.
Day 165	Cardiac magnetic resonance imaging showed inflammatory changes surrounding LCx stent, within the inter-atrial septum, posterior to the non-coronary aortic sinus and flail anterior mitral valve leaflet, with likely vegetations.
Day 193	Clinically improving with systemic antibiotic therapy, and discharged from hospital.
Day 200	Outpatient visit, clinically stable, afebrile with New York Heart Association class 2 heart failure symptoms.
Day 212	Outpatient TOE showed significant improvement; near normal serum inflammatory markers.
Day 227	LCx stent thrombosis requiring primary percutaneous coronary intervention.
Day 273	Elective mechanical mitral valve replacement.
Day 281	Antibiotic therapy ceased.
Day 310	Outpatient visit; patient was clinically stable and transthoracic echocardiography (TTE) was satisfactory.
Day 490	Outpatient visit; patient was clinically stable and TTE was satisfactory.

Case presentation

A 56-year-old previously healthy man was admitted with an acute coronary syndrome. He did not have any significant past medical history and was not on any regular medications. He was a smoker and consumed three to four standard drinks of alcohol per day, with no known significant family history. Coronary angiography revealed a severe mid left circumflex (LCx) artery stenosis, which was stented with a drug-eluting stent 3.0 × 32 mm (*Figure 1A* and *B*). He was discharged uneventfully after 2 days with recommendation of 12-month dual antiplatelet therapy.

Just under 6 months later, the patient presented to a regional hospital with severe chest pain and associated inferior ST-segment elevation following 1 week of worsening exertional angina. He was successfully thrombolysed with Tenecteplase and transferred to our centre. On initial examination, he was afebrile and had no significant clinical examination, with no cardiac murmurs or signs of heart failure. Coronary angiography demonstrated an aneurysmal dilatation adjacent the proximal segment of LCx stent. There was also severe in-stent restenosis at the distal edge of the stent with no other new coronary lesions. Working on a presumption that the aneurysmal dilatation was a pseudoaneurysm resulting from a poorly sealed dissection, a covered stent (3.0 × 16 mm) was placed. In addition, the re-stenotic segment was stented with another *everolimus* eluting stent (3.0 × 28 mm) (*Figure 1C* and *D*; *Video 1*).

Immediately post-procedure, the patient developed fever and rigors with a rise in serum inflammatory markers [white cell count 12.9×10^9/L (normal range $4.5–11 \times 10^9$/L), C-reactive protein 105 mg/L (normal range <5 mg/L)]. Three sets of blood culture grew *Staphylococcus aureus* sensitive to methicillin. A new pansystolic murmur was noted, though no stigmata of infective endocarditis were evident. He did not have any risk factors for infective endocarditis, was not immunocompromised, and was HIV negative.

Transoesophageal echocardiography (TOE) (*Figure 2A*, *Video 2*) revealed a 12 mm × 12 mm circular echo-free region at the location of the circumflex stent consistent with an abscess cavity or aneurysm. There were multiple small mobile echo-densities on the anterior and posterior mitral valve leaflets (*Video 2*), consistent with vegetations associated with severe mitral regurgitation from a flail anterior leaflet likely secondary to chordal rupture (*Video 3*). There were also thickening of the inter-atrial septum and inter-valvular fibrosa suggesting extension of infection (*Figure 2B*). On computed tomography (CT) and magnetic resonance imaging (MRI) scan (*Figure 3A* and *B*; Supplementary material online, *Video S1*), changes consistent with inflammation involving the LCx stent and inter-atrial septum were noted. Indium white blood cell scan also demonstrated activity at the region of coronary LCx stent consistent with an inflammatory collection (*Figure 3C* and *D*).

Treatment was instituted immediately with intravenous flucloxacillin, gentamicin (for 10 days), and oral rifampicin. Due to the scope of cardiac involvement, the cardiothoracic surgeons felt it unwise to attempt an extensive debridement and suggested attempt at medical sterilization with anti-microbials for 2 months. The patient improved on the antibiotic regime, with resolution of fever after 10 days and improvement in inflammatory markers after 2 weeks. Repeat TOE after 2 months revealed a reduction in abscess size (12 mm × 8 mm), less vegetations on the mitral valve leaflets with residual flail anterior mitral valve leaflet and severe mitral regurgitation, and almost normalization of the size of the inter-atrial septum and inter-valvular fibrosa. Elective progress angiography at the time showed widely patent stents and coronary intravascular ultrasound unable to visualize the cavity behind the stents due to the presence of the covered stent.

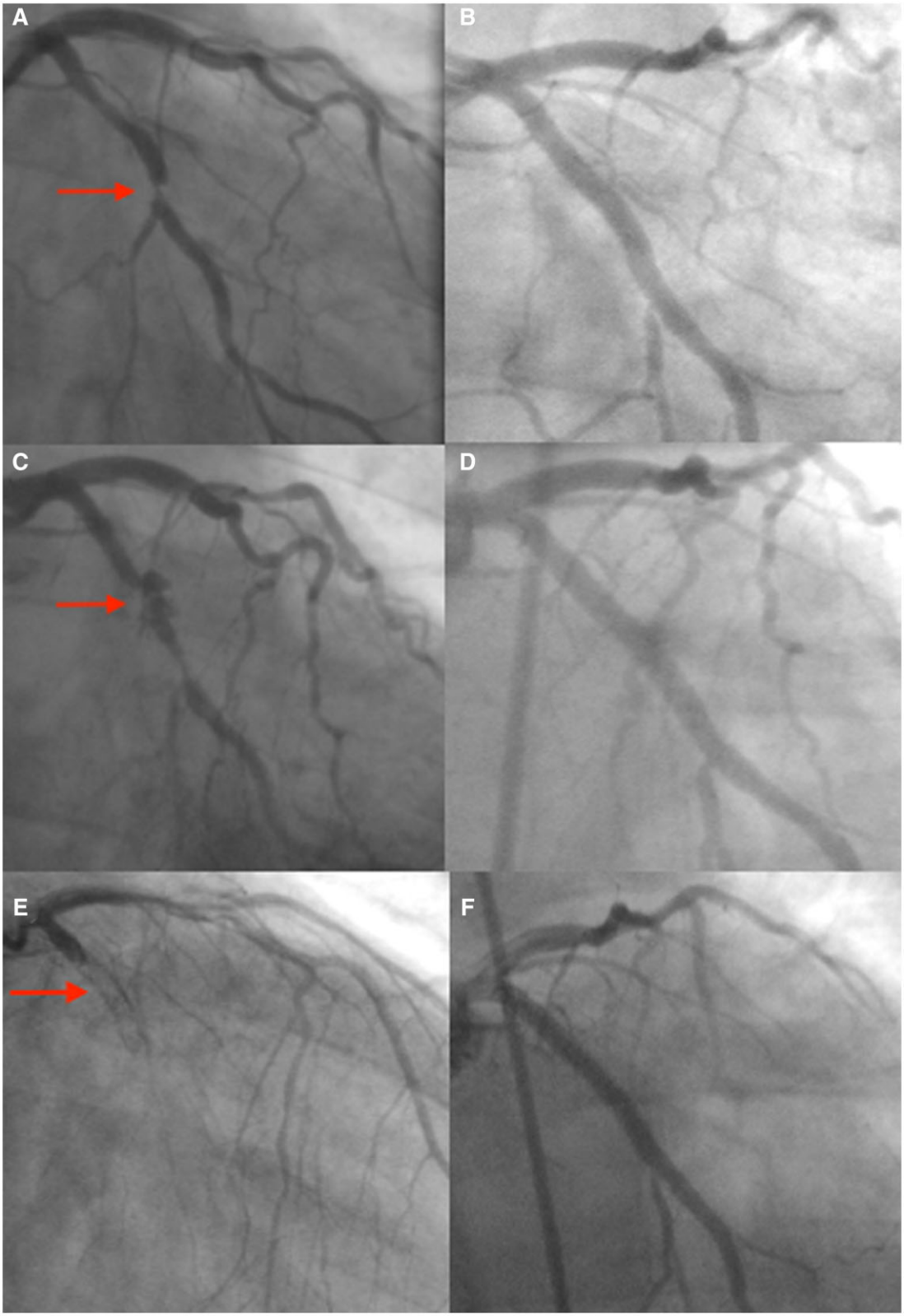
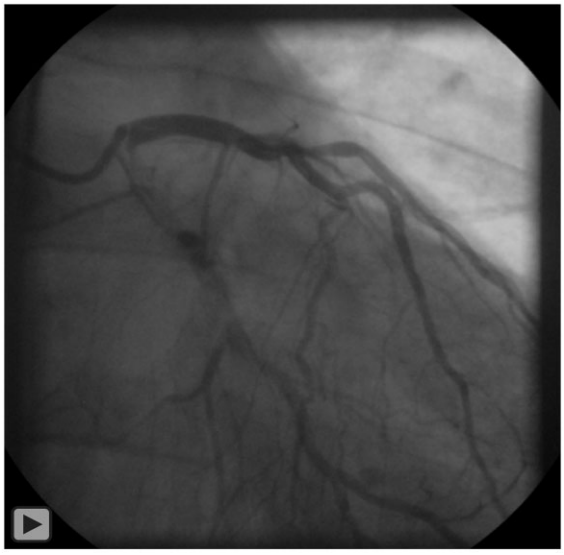


Figure 1 (*A*, *B*) First admission pre- (red arrow showing coronary stenosis) and post-percutaneous coronary intervention coronary angiogram of left circumflex. (*C*) Second admission coronary angiography showing mid left circumflex aneurysmal formation (red arrow), which was treated with a covered stent implantation (*D*). (*E*, *F*) Final admission with left circumflex stent thrombosis (red arrow) and primary percutaneous coronary intervention balloon angioplasty.

As the patient gradually developed signs of heart failure, it was decided to proceed with an elective mitral valve replacement, and surgical team recommended cessation of clopidogrel 1 week before surgery. This unfortunately resulted in the LCx stent thrombosis requiring re-intervention with balloon angioplasty (*Figure 1E* and *F*). He recovered but surgery was postponed for another 6 weeks.

Mitral valve replacement was performed 4 months after the initial presentation of mitral valve endocarditis and stent infection diagnosis. The LCx stent and abscess were not removed due to dense adhesions hampering access. The patient was maintained on intravenous flucloxacillin and oral rifampicin throughout these 4 months. Antibiotic therapy was continued for a further week and then ceased. Six months later, the patient remained well with no recurrence of sepsis and the inflammatory markers remained normal. He was well with the satisfactory mitral valve function in annual follow-ups for the subsequent 6 years.

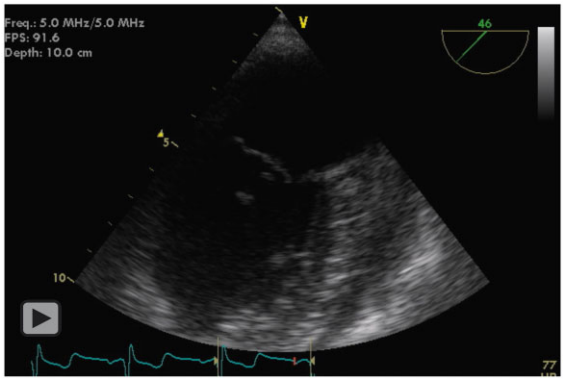


Video 1 Second admission coronary angiography showing mid left circumflex aneurysmal formation at the proximal segment of left circumflex stent.

Discussion

A rare but devastating complication of percutaneous coronary intervention

Despite the exceedingly high number of percutaneous coronary interventions performed annually, stent infection and myocardial



Video 2 Transoesophageal echocardiography demonstrating left circumflex stent with surrounding cavity suggestive of aneurysm or abscess, and mitral valve vegetation.

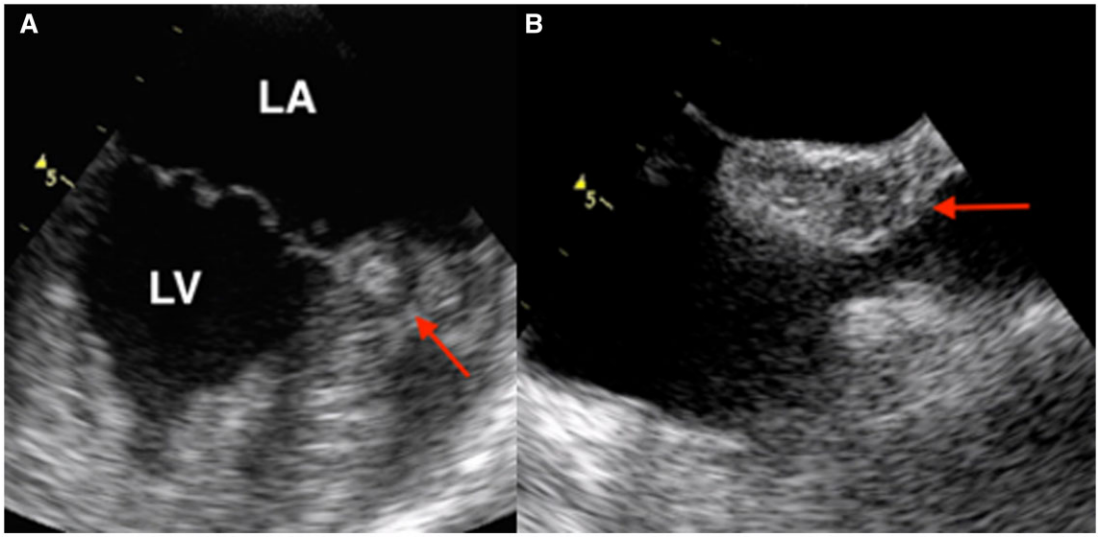
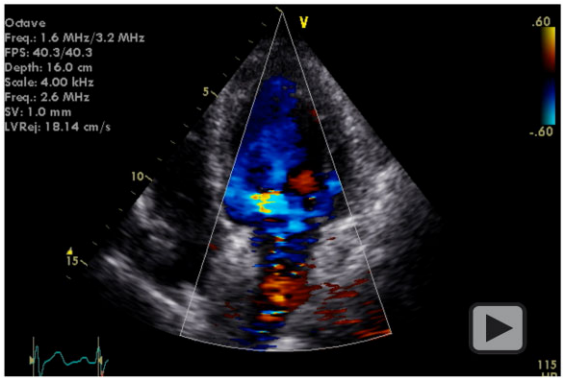


Figure 2 Transoesophageal echocardiography showing (*A*) myocardial cavity surrounding the left circumflex stent (red arrow) and (*B*) transcaval view showing interatrial septum echo lucent area suggestive of inflammation/abscess (red arrow). LA, left atrium; LV, left ventricle.



Video 3 Transthoracic echocardiography demonstrating mitral valve regurgitation.

abscess has remained a very rare complication. Elieson *et al.*[1] reported a total of 17 cases of coronary stent infection from 1987 to 2012. However, the mortality rate has been reported to be between 38% and 47%.[1,3] Majority of coronary stent infections occur within 4 weeks of stent implantation (88%)[1] and *S. aureus* is the most common organism involved (86–88%).[1,3]

Early coronary stent infection is believed to be due to equipment contamination or concomitant distant source of infection at the time of stent implantation.[3] It is postulated that stent implantation causes arterial intimal injury, and exposure of arterial medial layer to stent struts. Stent acts as an ideal vector for bacterial adherence, from which bacteria could spread to the arterial wall, initiate inflammatory response leading to necrosis.[4,5] It has been suggested that endothelialization of stent struts may be important in the prevention of stent infections.[2] However, it is unclear whether infection prevents stent incorporation or malapposition leads to infection.[4] Drug-eluting

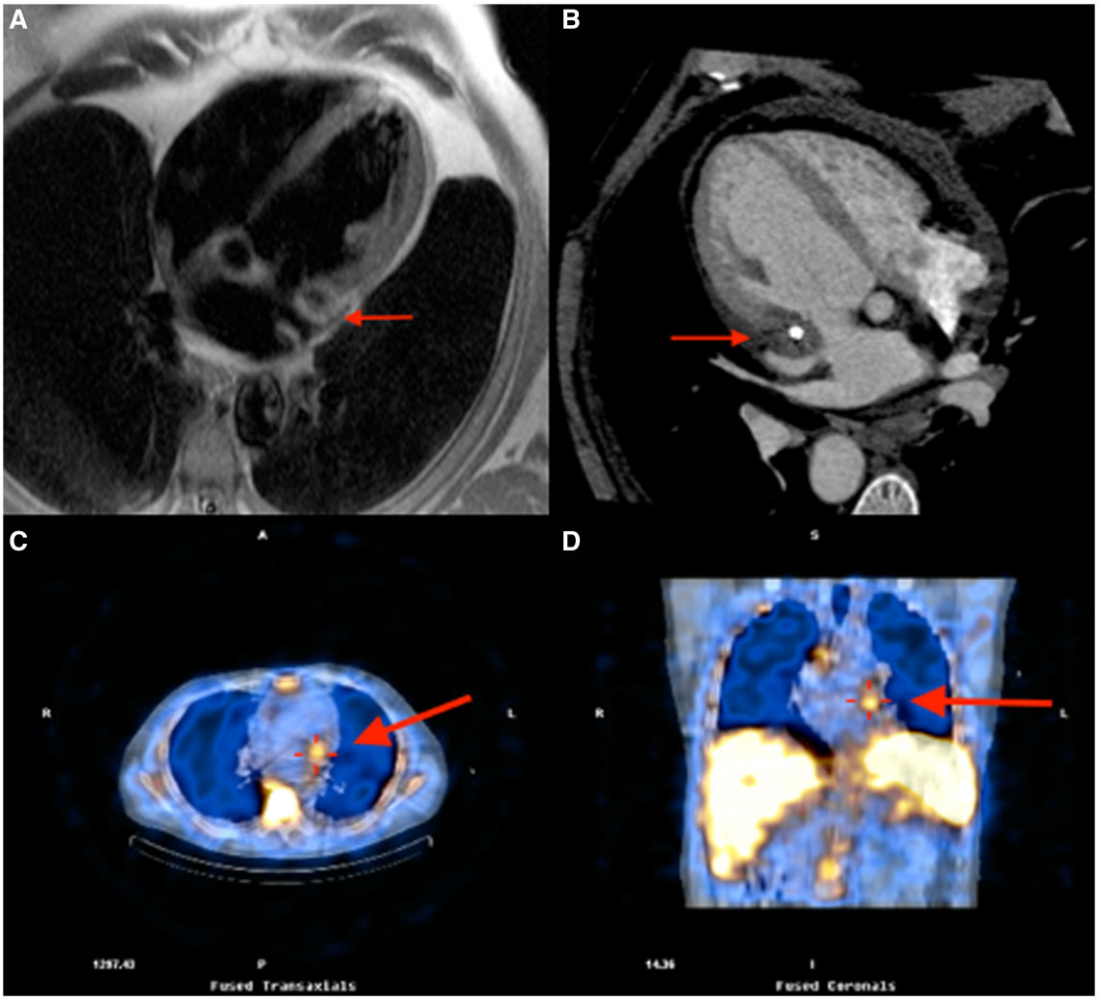


Figure 3 (*A*) Cardiac magnetic resonance imaging inflammatory changes within the interatrial septum, posterior to the non-coronary aortic sinus and adjacent to left circumflex stents (red arrow). (*B*) Cardiac computed tomography scan showing infiltration of epicardial fat surrounding stents with fluid and soft tissue density, including a 10 mm × 5 mm pocket of fluid density consistent with small abscess and reduced attenuation of LV myocardium adjacent to stent suggestive of myocarditis (red arrow). (*C*, *D*) Indium white cell study demonstrating activity at region of coronary circumflex stent consistent with inflammatory collection.

Table 1 Overview of clinical presentation, diagnosis, and management of coronary stent infection

Clinical suspicion	*Patient features:* • Fever and/or raised inflammatory markers with no other cause • Bacteraemia • Recent placement of coronary stent • Percutaneous coronary intervention coincident with another source of infection/sepsis *Procedural features:* • Multiple repeat procedures performed through the same arterial sheath • Prolonged arterial sheath retention • Long/complex coronary intervention • Vascular complications such as haematoma
Diagnosis	*Firstline imaging modalities:* • Coronary angiography • Echocardiography (transthoracic ± transoesophageal) *Additional/supplementary imaging tools:* • Cardiac CT scan • Cardiac MRI • PET scan • WBC scan
Management	*Early-onset/focal coronary stent infection:* • Initial medical/conservative therapy with systemic antibiotics • Consider surgery/debridement if no response to medical therapy or significant complications *Late-onset/extensive infection:* • Hybrid approach including systemic antibiotic and surgical treatment

CT, computed tomography; MRI, magnetic resonance imaging; PCI, percutaneous intervention; PET, positron emission tomography; WBC, white blood cell.

stents are known to delay endothelialization longer than 6 months and this may result in uncovered endoluminal stent struts forming a nidus for bacterial adherence.

Our case suggests a potential complication of delayed endothelialization which could be due to either possible stent under-expansion or pseudoaneurysm formation adjacent to the stent which exposes the stent struts to blood stream infections, seeding or infective embolization from a distant source (i.e. endocarditis in this case). Coronary stents were post-dilated and optimized based on the standard practice at the time. Unfortunately, intracoronary imaging was not performed before the covered stent insertion to evaluate the cause of stent failure and pseudoaneurysm formation.

Valvular endocarditis is more common than coronary stent infection and it is possible that coronary stent infection occurred as a result of bacterial seeding and/or embolization or local extension of mitral valve endocarditis in surrounding structures including LCx stent. This case of *S. aureus* infection 6 months after the index procedure is unique compared to existing cases in several aspects: (i) it coincided with mitral valve endocarditis and there was extensive cardiac involvement, (ii) the long latency from the initial stenting procedure, (iii) the initial presentation was an acute coronary syndrome instead of fever as in most cases, and (iv) finally, we were successful in sterilizing the extensive infection with prolonged intravenous antibiotics. Given the extensive cardiac involvement, it is unclear whether the primary event was a stent infection, which spread to involve the mitral valve or an invasive mitral valve infective endocarditis spreading to adjacent structures including the LCx stent.

Diagnosis

Dieter[6] proposed the criteria for the diagnosis of coronary stent infection with definitive diagnosis 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 bacteraemia, significant fever, or leucocytosis with no other cause; acute coronary syndrome; or positive cardiac imaging.

Diagnosing stent infection and myocardial abscess months after stent implantation is challenging and requires high index of clinical suspicion especially if patients present with a combination of cardiac symptoms and systemic inflammatory response. Various imaging modalities have been used to detect this ominous condition and in majority of cases coronary angiography and echocardiography are sufficient for diagnosis. Infrequently, other imaging modalities such as cardiac MRI, cardiac CT scan, and positron emission tomography could be helpful if diagnosis is uncertain. In our case, coronary angiography was significantly abnormal showing a new pseudoaneurysm formation of the stented LCx segment and TOE confirmed the diagnosis.

Management

Coronary stent infection poses a serious management problem as both conservative and surgical interventions carry a high morbidity and mortality risk. While success with parental antibiotics has been reported, mortality may be as high as 50% with parenteral antibiotics alone. On the other hand, surgical intervention with resection of infected stent and surrounding material is preferred but this group will also face a mortality rate of up to 38%.[3] Elieson *et al.*[1] suggested that surgery should be considered early in cases of late-onset infections and medical therapy alone in early-onset infections unless major complications are apparent on imaging or if medical therapy appears to be failing. Parenteral antibiotic therapy should continue for at least 4 weeks or possibly longer. Overview of coronary stent infection clinical assessment, diagnosis, and treatment is summarised in *Table 1*.

Conclusion

In summary, we presented a case of *S. aureus* mitral valve endocarditis involving a drug-eluting coronary stent 6 months after the index percutaneous coronary intervention. It is possible that delayed

endothelialisation due to under-expanded stent or malapposition as a result of pseudoaneurysm formation might have contributed to bacterial adherence and late coronary stent infection. There was extensive cardiac involvement including coronary aneurysm, myocardial abscess formation, and concomitant inter-atrial septal abscess. However, the infection was controlled with prolonged antibiotics therapy and mitral valve replacement.

Lead author biography

Ata Doost was awarded Fellowship of Royal Australian College of Physicians in adult cardiology in November 2019. He worked as the coronary interventional fellow at Fiona Stanley Hospital, Perth, Western Australia from July 2020 to August 2021 and is now Interventional and TAVI Fellow at King's College Hospital, London, UK.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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