I. Introduction

Nowadays, percutaneous coronary intervention (PCI) is an established procedure, and annually more than 500,000 coronary stents are implanted in the United States\(^1\). However, most physicians hardly recognize that a coronary stent can be a potential source of infection and that the risk may last for several years. Stent infection is extremely rare and difficult to identify. Delay in definite diagnosis often leads to death. We describe a case of stent infection that occurred 8 years after implantation. A 66-year-old woman was admitted to our hospital with high-grade fever. She underwent placement of a bare-metal stent to the right coronary artery at 59 years of age. She also underwent kidney transplantation at 58 years of age and had been taking multiple immunosuppressants. Although whole-body computed tomography (CT) scan at the time of admission found no source of bacterial infection, blood cultures grew *Staphylococcus aureus*. Brain magnetic resonance imaging revealed multiple cerebral infarctions. Infective endocarditis (IE) was suspected but transthoracic and transesophageal echocardiogram found no evidence of IE. The patient became afebrile after administration of intravenous antibiotics and intravenous immunoglobulin, and blood cultures were negative. However, echocardiogram revealed a decline in left ventricle function, and thereafter, the patient developed acute inferior wall myocardial infarction. Urgent coronary angiography exhibited a large coronary artery aneurysm at the origin of the right coronary artery where a previous coronary stent was implanted, and repeat CT also confirmed a very rapidly developing coronary aneurysm. We performed emergent removal of the mycotic aneurysm along with the infected stent. However, the right heart had been severely damaged prior to surgery. She underwent four days of veno-arterial extracorporeal membrane oxygenation but developed bacterial pneumonia and expired on postoperative day 15. This case highlights the long-term risk of coronary stent infection several years after implantation.

KEY WORDS: coronary aneurysm, percutaneous coronary intervention, stent
revealed multiple cerebral embolisms. Although these findings led to a suspicion of infective endocarditis (IE), the transthoracic and transesophageal echocardiogram on hospital day 3 revealed no evidence of vegetation on heart valves or any other source of emboli (Fig. 2a). The patient was started on antibiotic therapy with intravenous immunoglobulin and tazobactam/piperacillin, which were switched to ceftriaxone after the causative organism was proved to be MSSA.

On hospital day 7, she developed shortness of breath. Chest radiography revealed pulmonary edema. A contrast-enhanced CT scan was performed to investigate the focus of the infection and the cause of pulmonary edema, but did not indicate any infection focus. She was treated with diuretic therapy and improved immediately. On hospital day 13, repeat CT scan showed slight deformity of the sinus of Valsalva (Fig. 1b) with new pericardial effusion, which was not detected on the CT scan obtained on day 7. However, these findings were not thought to be associated with infection. Repeat echocardiography also exhibited presence of new pericardial effusion; however, no valvular regurgitation or vegetation was noted (Fig. 2b). On hospital day 24, gallium scintigraphy demonstrated the presence of inflammation at the right shoulder. However, synovial fluid culture was negative.

On hospital day 30, the electrocardiogram showed ST-segment elevation in leads III and aVF, and also showed negative T-wave in leads II, aVF, and V3 to V6. Transthoracic echocardiogram revealed decline in the left ventricular ejection fraction from 73% on admission to 38%. Therefore, immediate coronary angiography was performed, which showed a large aneurysm (6.5 × 5 cm) at the origin of the RCA (Fig. 3). This finding was also confirmed by plain (Fig. 1c) and contrast CT scans (Fig. 4). At this time, transthoracic echocardiogram did not reveal any evidence of vegetation on each heart valve, and thus, a diagnosis of IE was ruled out (Fig. 2c).

A bare-metal coronary stent, which was implanted 8 years prior, was determined to be the source of infection and presumed to be associated with the rapidly growing aneurysm and inferior wall myocardial infarction. Consequently, surgical treatment for
the infected coronary stent was scheduled. However, the next morning after the examinations, the patient went into cardiogenic shock. She underwent emergency resection of the aneurysm along with reconstruction of the right sinus of Valsalva using a prosthetic vascular graft and bypass grafting to the RCA using a saphenous vein graft. Intraoperative findings revealed purulent pericardial effusion and an infected pseudoaneurysm adjacent to the previously placed coronary stent at the origin of the RCA (Fig. 5). There was no obvious evidence of infection on the aortic valve. Transesophageal echocardiogram performed during the operation could not find vegetations on any other valve. Pathological examination confirmed a coronary pseudoaneurysm containing thrombus (Fig. 6). Granulation tissue and inflammatory cells comprising mostly neutrophils were found outside the arterial wall. A small amount of Gram-positive cocci were noted inside the thrombus. There was no growth on culture. The final diagnosis was mycotic coronary aneurysm.

The right heart had been severely damaged prior to surgery, and thus, the patient could not be weaned off cardiopulmonary bypass and needed veno-arterial extracorporeal membrane oxygenation (VA-ECMO). Although the patient was successfully weaned off VA-ECMO on postoperative day 4, she developed bacterial pneumonia on postoperative day 11 and expired on postoperative day 15.

III. Discussion

We describe the case of coronary stent infection that occurred 8 years after implantation in a 66-year-old woman.

It is well known that any foreign body, including coronary stents, carries a potential risk of infection once implanted in human body. In the present case, bacteria may have incidentally entered the blood stream from a minor infection on the necrotic toe, and settled down on the foreign material. Once bacteria are seeded on a foreign material, they create a biofilm, which acts as a strong barrier to antibiotics. Therefore, foreign body infection is not easily resolved by antibiotic therapy alone, and often requires surgical intervention. Endothelialization of the stent’s strut appears to be protective against hematogenous bacterial dissemination[8]. However, even 5 years after stent implantation, complete endothelialization was achieved only in 71% of patients who underwent coronary interventions with BMS, and the percentage was much lower (42%) in patients with DES[8]. Furthermore, surgeons often encounter coronary stents protruding...
into the aortic root without endothelialization when they are implanted in the ostial lesion. Persistently exposed stent struts may provide a nidus for infection during an episode of bacte-

mia.

In the present case, the patient was a typical immunocompromised host: diabetic, had prior history of renal transplantation, and took multiple immunosuppressants. In addition to the above background, she had a chronic foot ulcer due to peripheral vascular disease, which was treated by a dermatologist at an outpatient clinic for more than three months prior to the hospitalization, and this could have led to a spread of bacteria in the bloodstream. The patient had a history of multiple coronary interventions, and one of the coronary stents was implanted at the orifice of the RCA. Therefore, it can be speculated that bacteria spread to the unhealed stent strut, and consequently resulted in a large mycotic aneurysm even 8 years after stent implantation. It has been reported that most stent infections occur within 4 weeks, probably because complete endothelialization has not been achieved yet. However, physicians need to be aware that coronary stents may remain unhealed for years, and can pose a long-term risk of infection, although the incidence is very small.

Another issue is that stent infection is very difficult to diagnose unless it develops structural complications, such as coronary thrombosis, purulent pericardial effusion, and mycotic aneurysm. Delay in definitive diagnosis often results in severe or even fatal consequences. In the present case, the coexistence of a cerebral embolism, a fever of unknown origin and bacteremia was the typical presentation of IE. However, any evidence supporting a diagnosis of IE could not be found on repeated echocardiogram. Fig. 2 shows a temporal comparison of transthoracic echocardiography images performed on hospital day 3 (a), day 17 (b) and day 31 (c). No vegetation and regurgitation on the aortic and mitral valves were found. The images show the increase in pericardial effusion over time. The image on hospital day 31 demonstrates the destruction of the sinus of Valsalva that could not be identified on day 3 and day 17. Coronary angiography was not performed until the patient developed inferior wall myocardial infarction. Although angiography finally revealed coronary aneurysm at the origin of the RCA on hospital day 30, it should have been diagnosed earlier. In an endeavor to identify the source of infection, serial CT scans were performed during admission, and, in retrospect, CT images on day 13 already

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**Fig. 6** Pathological images of the surgically removed coronary aneurysm.

a: Elastica van Gieson staining shows rupture of the elastic fiber (arrows) indicating a pseudoaneurysm.
b, c: Hematoxylin and eosin (H&E) staining and close-up of the H&E staining image show remarkable neutrophilic infiltration outside the artery and hematoma formation with hemosiderin, indicating that the infection had occurred while the patient was alive.
c: Close-up of the H&E staining image.
d: Gram staining shows Gram-positive cocci (arrow) in the thrombus.
showed a growing aneurysm with new pericardial effusion (Fig. 1b). In fact, these were subtle structural changes, and to make matters worse, the lack of suspicion for a very late stent infection made us overlook the initial signs of stent infection. Retrospectively, brain MRI on admission demonstrated acute multiple cerebral infarctions. The infected stent was placed at the very origin of the RCA, and the multiple cerebral emboli may have been derived from the bacterial mass, which grew on the surface of the stent strut. Echocardiography also showed thick pericardial effusion after hospital day 17 (Fig. 2b) and could have been a clue for infection.

After a definitive diagnosis was established according to coronary angiography findings, close inspection of her whole body, involving reexamination of echocardiography, coronary CT angiography, and brain MRI scans, was planned. Such examinations delayed the operation and the patient developed cardiac shock on the very next morning. Alternatively, an emergent operation just after coronary angiography could have been performed.

IV. Conclusion

This case report is aimed at alerting physicians regarding the long-term risk of coronary stent infection even years after implantation, and also highlights the difficulty in diagnosing coronary stent infection until after serious complications, such as mycotic coronary aneurysm and myocardial infarction, have developed. If a patient with history of stent implantation has persistent bacteremia despite appropriate antibiotic therapy, physicians should highly suspect stent infection and check for any subtle changes in coronary artery structures on imaging data.

Conflicts of interest

The authors have no conflict of interest to disclose.

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