Coronary Artery Perforation Complicated by Pericardial Abscess Formation

A Clinical Dilemma

Laura J. Davidson, MD; Mark J. Ricciardi, MD

Coronary artery perforation is a rare complication of percutaneous coronary intervention, which occurs in 0.1% to 3% of cases.[1] An infrequent cause of coronary artery perforation is stent infection, 80% of which are because of *Staphylococcal aureus*.[2] This is a case of a *S. aureus* pericardial abscess discovered in the setting of a late left anterior descending (LAD) coronary artery perforation and the potential explanations for this rare presentation. Reports of sepsis after coronary stent implantation, leading to coronary artery aneurysm formation or perforation,[3,4] and of purulent pericarditis after percutaneous coronary intervention are exceedingly rare.[5–7] This is a rare first report of a pericardial abscess found in conjunction with a perforated coronary artery.

Case Presentation

A 75-year-old man with a history of hypertension and hyperlipidemia presented to another hospital with upper back pain and electrocardiographic evidence for an ST-segment–elevation myocardial infarction. Coronary angiography revealed a proximal 90% occluded calcified LAD lesion, which was treated with a 2.5×18 mm drug-eluting stent (Figure 1), aspirin, and ticagrelor. A transthoracic echocardiogram demonstrated an ejection fraction of 50% and anterior wall hypokinesis with no significant valvular lesions. Postprocedural shortness of breath developed and was attributed to ticagrelor, which was replaced with clopidogrel. Despite this, he continued to complain of shortness of breath. Nine days after initial presentation with an ST-segment–elevation myocardial infarction, the patient returned to the same hospital for evaluation of his shortness of breath, and a repeat echocardiogram was performed that demonstrated a large pericardial effusion. Pericardiocentesis produced 500 cc of bloody fluid. His shortness of breath improved, and he was discharged home.

Several days later, a rash on his upper and lower extremities developed and was attributed to a clopidogrel drug reaction. He was, therefore, switched back to ticagrelor. The rash did not resolve, and 2 weeks after his pericardiocentesis, he was readmitted with progressive shortness of breath.

At this time, he was transferred to our institution for further management. On arrival to our hospital, he complained primarily of shortness of breath. His blood pressure was 117/85, his pulse was 83 beats per minute, with an oxygen saturation of 98% on 2 L nasal cannula. Scattered petechiae and palpable purpura with central necrosis were evident on the dorsum of his hands and feet. He had decreased breath sounds at the left lung base. His electrocardiogram showed normal conduction and no ischemic changes. Laboratory data revealed a mild leukocytosis, a hemoglobin of 10 g/dL, normal renal function, and a mild transaminitis.

Discussion

Dr Davidson: The patient has just arrived at your institution, what initial work up would you pursue at this juncture?

Dr Ricciardi: My initial tests of choice are a repeat echocardiogram and chest x-ray, given his progressive shortness of breath and concern for another pericardial effusion. A pericardial effusion requiring drainage following stent placement is concerning for coronary artery perforation and therefore requires further, immediate investigation. However, on review of the angiogram performed at the time of the patient's ST-segment elevation myocardial infarction, the LAD stent does not appear oversized, making perforation less likely.

Dr Davidson: The echocardiogram was performed and demonstrated reaccumulation of pericardial fluid posteriorly (Figure 2), without evidence of hemodynamic compromise. There was also evidence of apical hypokinesis, which is a different distribution of wall motion abnormalities compared to the anterior hypokinesis seen immediately following his ST-segment elevation myocardial infarction presentation. A chest x-ray demonstrated a left-sided pleural effusion. What steps would you take at this point?

Dr Ricciardi: Given that there is no evidence of hemodynamic compromise on the echo or any clinical signs of cardiac tamponade, urgent drainage of the fluid is not necessary at this time. The left-sided pleural effusion is an interesting finding, which should be explored further, as the patient has no clinical signs of heart failure and the asymmetry of the effusion could indicate an infectious etiology, especially with his leukocytosis. Another consideration is extravasation of fluid from the pericardium into the pleural space. It is notable that the patient is anemic, which could

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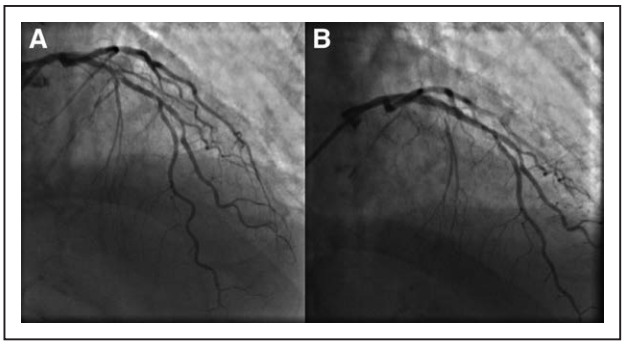

Figure 1. A, A 90% proximal, calcified left anterior descending lesion was seen on the initial coronary angiogram. **B**, The lesion was successfully treated with a 2.5×18 mm drug-eluting stent.

be due to bleeding into the pericardial and pleural space. I recommend further characterizing the effusion with a computed tomography (CT) scan of the chest, obtaining blood cultures, and evaluating the rash with a skin biopsy.

Dr Davidson: Pathologic examination of the biopsied skin lesions was consistent with leukocytoclastic vasculitis and not a drug reaction. What does this indicate to you?

Dr Ricciardi: Hypersensitivity reactions to clopidogrel are seen in up to 5% of patients, which most commonly manifest as a rash.[8] Therefore, switching the patient to an alternative antiplatelet agent in the setting of a rash is a reasonable first step in diagnosis and management. Additionally, shortness of breath can be observed in 10% to 20% of patients who take ticagrelor.[9] However, patients taking ticagrelor have known coronary artery disease and potentially other cardiac disease, and shortness of breath should be merely a diagnosis of exclusion after all other potential etiologies of a patient's shortness of breath have been explored. Another consideration in a patient with recent drug-eluting stent implantation is an adverse reaction to the stent polymer or drug.[10] In this patient, the suspicion for adverse drug reactions is less likely and the biopsy finding of leukocytoclastic vasculitis raises greater suspicion for an infectious etiology of his rash, especially in the setting of an abnormal chest x-ray. A chest CT scan is critical at this juncture to evaluate for potential infection.

Dr Davidson: A chest CT scan with contrast was ordered. Shortly prior to the CT scan, the patient developed a new left bundle branch block on telemetry but denied any symptoms of chest pain or worsening shortness of breath. The CT scan was negative for a pulmonary embolism but did show extensive ground glass opacities, a loculated pericardial effusion with mass effect, and high attenuation adjacent to the LAD extending into the pericardial effusion, concerning for active hemorrhage (Figure 3). Additionally, compressive atelectasis at the left lung base was noted with a left pleural effusion, which was communicating with the pericardium. His hemoglobin had remained stable since admission. What are your thoughts on the immediate management of this patient?

Dr Ricciardi: While the ground glass opacities certainly raise the suspicion for infection, the concern for active hemorrhage is of more immediate concern. Even though the patient is not showing any clinical signs of cardiac tamponade, it is necessary to evaluate this finding immediately. Additionally, it appears that the pericardial effusion is contiguous with the pleural effusion, raising concern for hemorrhage into the pleural space as well. In light of the left bundle branch block that recently developed, the apical wall motion abnormality, and the CT scan findings, I would recommend urgent coronary angiography to specifically evaluate the LAD and the coronary stent that was recently placed.

Dr Davidson: An emergent coronary angiography demonstrated free extravasation of contrast from the LAD consistent with perforation (Figure 4). How would you treat this life-threatening lesion?

Dr Ricciardi: On review of the coronary angiogram, it is clear that the distal vessel is not opacified on angiography. Therefore, successful placement of a covered stent may not be feasible. The perforation does appear to be at the location of

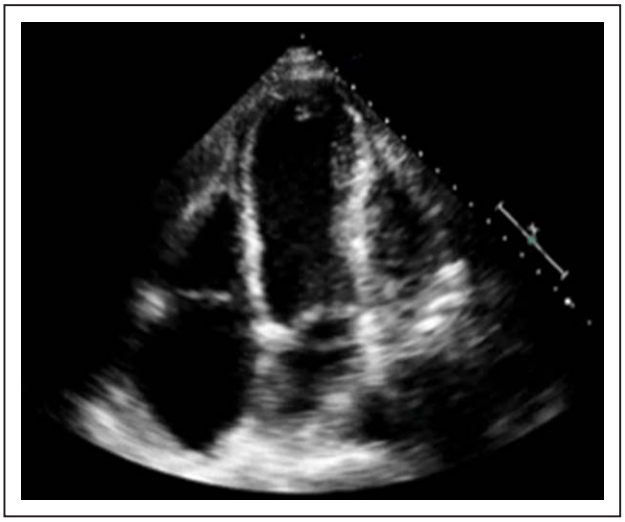

Figure 2. An echocardiogram revealed a pericardial fluid collection, measuring 3.2 cm and causing indentation of the left ventricle. Apical hypokinesis was also observed.

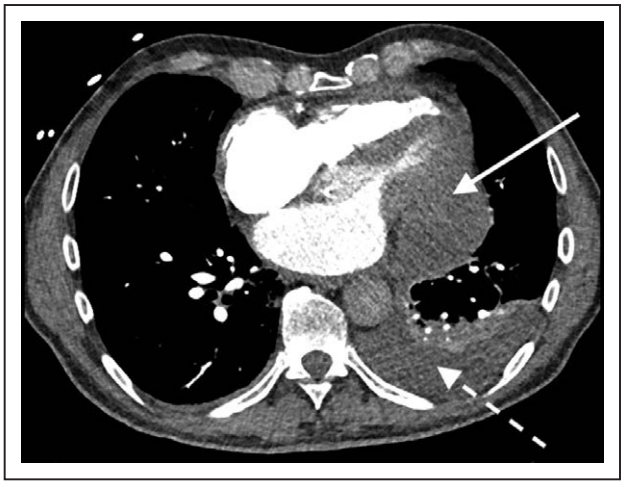

Figure 3. A computed tomographic scan demonstrated high attenuation adjacent to the left anterior descending extending into a loculated, posterior pericardial effusion (solid arrow), which is suspicious for active hemorrhage. The pericardial effusion exhibits mass effect on the left ventricle. Compressive atelectasis at the left lung base is also noted associated with a left pleural effusion (dotted arrow).

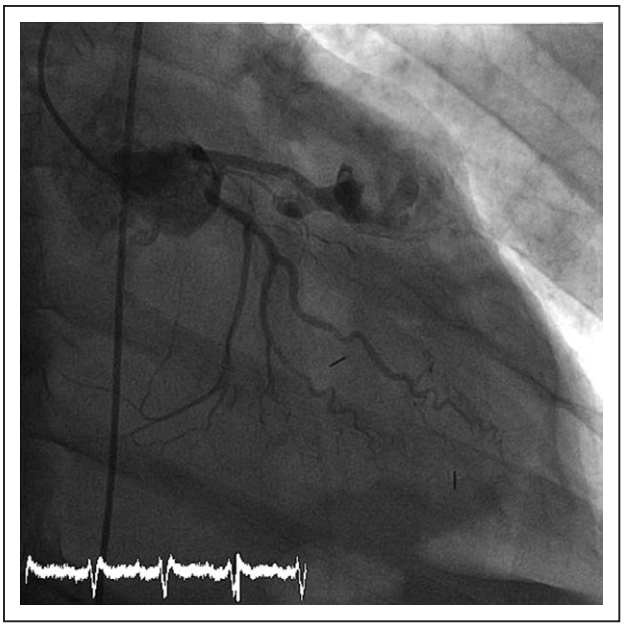

Figure 4. An emergent coronary angiogram revealed free extravasation of contrast from the left anterior descending, consistent with a perforation.

the previously placed LAD stent. Given signs of active hemorrhage, the only viable option at this point is to send the patient to the operating room for surgical correction of the perforated LAD.

Dr Davidson: In the operating room, exploration revealed a posterior pericardial abscess, a densely adherent, fibrotic pericardium, and a proximal LAD perforation. Blood from the LAD was freely extravasating into the pericardial space. While in the operating room, blood cultures drawn 1 day prior returned positive for Gram-positive cocci, which subsequently speciated as *S. aureus*. In spite of his bacteremia, the patient never developed a fever until after surgery. How can we account for the constellation of findings in this case, including a perforated LAD, pericardial effusion and abscess, *S. aureus* bacteremia, leukocytoclastic vasculitis, and ground glass opacities?

Dr Ricciardi: Although unusual, I believe that the most plausible explanation for this patient's unique presentation is a coronary stent infection. Reports of coronary stent infection leading to coronary mycotic aneurysm formation and rupture, as well as reports of purulent pericarditis following drug-eluting stent placement, have been described but rarely rupture and develop purulent pericarditis in the same patient.[11] In this case, where both coronary artery rupture and pericardial abscess formation occurred, I hypothesize that an infected stent caused abscess formation within the LAD, leading to rupture and a hemorrhagic pericardial effusion. I believe that the thick and adherent pericardium compressed the perforated vessel, thereby avoiding overt hemopericardium and tamponade. I speculate that the pericardium was subsequently seeded by the bacteria from the infected stent and a pericardial abscess formed. Staphylococcal bacteremia and septic emboli ensued and was manifested by cutaneous leukocytoclastic vasculitis and multifocal pneumonia. As with this patient, coronary stent infections are most often due to *S. aureus*, further supporting this diagnosis.[12] A pericardial effusion in the setting of recent stent placement is always concerning for a perforated coronary artery until proven otherwise.

Dr Davidson: Are there any alternative explanations for this constellation of clinical findings?

Dr Ricciardi: An alternative explanation for the patient's presentation is coronary perforation as a complication of stent placement resulting in hemorrhagic tamponade. Others have reported pseudoaneurysm formation at the site of stent implantation and subsequent cardiac tamponade.[13] While stent infection is a reported cause of coronary pseudoaneurysm, balloon overexpansion and dissection are much more common causes for this complication.[14–16] In addition, my review of the patient's initial angiogram showed no evidence of procedural dissection or perforation. This alternative explanation could, therefore, only be plausible if an unrecognized microdissection had occurred leading to the subsequent development of hemorrhagic pericardial effusion. In this scenario, I would presume that the pericardial abscess and systemic infection was the result of instrumentation during the pericardiocentesis.

Finally, it is plausible that the patient developed post-myocardial infarction pericarditis and effusion, which was contaminated with *S. aureus* during pericardiocentesis. Subsequent formation of an abscess cavity could have then eroded into the LAD causing perforation. This seems unlikely given the posterior location of the pericardial abscess, and it does not explain the hemorrhagic nature of the initial pericardial effusion.

Dr Davidson: What can we learn from this rare case and apply to other patients in the future?

Dr Ricciardi: Although it is a rare complication of DES placement, this case illustrates that diagnosing a pericardial abscess and effusion should raise suspicion for stent infection and possible coronary artery rupture. Likewise, the diagnosis of late coronary artery perforation should prompt suspicion for stent infection and close monitoring for signs of systemic infection.

Clinical Outcome

In the operating room, the abscess was drained, and a left internal mammary artery to distal LAD graft was placed. Antibiotics were started immediately, and the patient was brought to the intensive care unit. Despite recovering well from a cardiovascular perspective, the patient continued to experience the severe systemic *S. aureus* infection that affected the lungs, pericardium, skin, and blood. He died several days postoperatively because of septic shock.

Conclusions

Coronary artery stent infection caused by *S. auerus* is a rare but often fatal complication of stent implantation. The first case of *S. aureus* related stent infection was reported in 1993, and since then, at least 23 other cases have been reported (Table).[17] Patients typically present with a wide array of symptoms, including fever, constitutional symptoms, chest

Table. ***Staphylococcal Aureus*** **Sepsis Reports After Percutaneous Coronary Intervention**

Publication	Year	Presentation/Complication	Organism	Treatment	Survived?
Günther et al[17]	1993	Pericardial empyema	MRSA	Surgery	Yes
Grewe et al[18]	1999	Pancarditis	MSSA	Antibiotics only	No
Rensing et al[19]	2000	Abscess	*Staphylococcal Aureus*	Antibiotics only	Yes
Sankari et al[5]	2000	Purulent pericarditis	MSSA	Surgery	Yes
Liu et al[20]	2003	Mycotic psuedoaneurysm and abscess	MSSA	Surgery	Yes
Golubev et al[21]	2004	Abscess	MSSA	Surgery	No
Singh et al[22]	2005	Mycotic aneurysm	MSSA	Surgery	Yes
Hoffman et al[23]	2005	Abscess	MSSA	Antibiotics only	Yes
Alfonso et al[24]	2005	Purulent pericarditis	MSSA	Antibiotics only	No
Gonda et al[3]	2007	Abscess	MSSA	Surgery	Yes
Salinas et al[25]	2007	Coronary artery aneurysm	MRSA	Surgery	No
Kishida et al[26]	2007	Mycotic aneurysm	*S. aureus*	Surgery	Yes
Jang et al[27]	2007	Abscess and pseudoaneurysm	*S. aureus*	Surgery	Yes
Schoenkerman and Lundstrom[11]	2009	Mycotic aneurysm and coronary rupture	*S. aureus*	Antibiotics only	No
Schoenkerman and Lundstrom[11]	2009	Mycotic aneurysm and coronary rupture	*S. aureus*	Surgery	Yes
Schoenkerman and Lundstrom[11]	2009	Purulent pericardial effusion	MRSA	Antibiotics only	No
Wu et al[2]	2010	Ruptured coronary artery and pseudoaneurysm	MRSA	Stent graft	Yes
Lim et al[28]	2011	Mycotic pseudoanerysm	MRSA	Surgery	No
Elieson et al[12]	2012	Myocardial rupture	MSSA	Antibiotics only	No
Del Trigo et al[29]	2012	Mycotic pseudoanuerysm	*S. aureus*	Surgery	No
Roubelakis et al[30]	2015	Coronary artery aneurysm and rupture	MSSA	Surgery	Yes
Shafer et al[31]	2017	Coronary fistula	MRSA	Surgery	No
Takahashi et al[32]	2017	Coronary aneurysm	*S. aureus*	Covered stents	Yes

MRSA indicates methicillin-resistant *Staphylococcal aureus*; and MSSA, methicillin-sensitive *Staphylococcal aureus*.

discomfort, and myocardial infarction. On clinical evaluation, at least half of the patients who present with *S. aureus* associated stent infection are found to have a mycotic aneurysm or pseudoaneurysm (n=12). Clinical management of the patients with aneurysms and pseudoanuerysms varies and ranges from surgical resection of the aneurysm and stent, covered stent placement, and conservative management with aggressive antibiotic therapy and is individualized based on the clinical stability of patients and their ability to tolerate surgical intervention. However, there are other patients who have presented with different presentations of infection, including pericardial disease and abscess formation. Overall, on reviewing the previous cases of *S. aureus* sepsis reported post percutaneous coronary intervention, mortality is ≈45%, indicative of a poor prognosis in spite of the best surgical, procedural, and medical attempts made to intervene on this condition. As observed in the case presented, pericardial effusion postpercutaneous coronary intervention should prompt investigation into possible stent infection with associated coronary artery perforation.

Disclosures

None.

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