

Coronary Artery Stent Infection

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Summary: This paper aimed to examine the literature for cases of coronary artery stent infection in order to provide comprehensive data to clinicians regarding its prevalence, clinical presentations, and possible treatments. Coronary artery stenting was initially reported in 1987. Stenting of the coronary arteries is now used in 40–60% of all interventional coronary artery procedures. The understanding of the pathophysiology of coronary artery disease is evolving. It has been suggested that atherosclerosis may be a complication of an infectious etiology. By using a stent to treat coronary artery disease, a foreign body is directly juxtaposed with an area of inflammation. The first reported case of an infected coronary artery stent was in 1993. Although this is an exceedingly rare event, the associated mortality is alarmingly high. Analysis of the literature reveals a total of four reported cases of coronary artery stent infection. Symptoms of stent infection present days to weeks after the initial coronary intervention. All four patients developed fevers and at least two patients developed postintervention angina. In patients who have had a coronary artery stented, the presence of angina and fevers should make the clinician suspicious for a stent-related infection. Two of the patients had infection with *Pseudomonas aeruginosa*, which seems to be an unusual organism for a catheter-related infection. Surgical removal of the infected stent and artery complex was performed on nearly all cases. Despite aggressive measures, the majority of patients died. Few data are available on the long-term risk for coronary artery stent infection. In a patient who has undergone coronary artery stent placement, the clinician must be very sensitive to fever, return of angina, and bacteremia. The complication rate at the present time does not warrant the use of prophylactic antibiotics prior to high-risk procedures (e.g., dental procedures). Furthermore, the low infection rate of coronary artery stents may be a result of the in-

flammatory nature of atherosclerosis, which may provide a protective benefit against bacterial infection of the stent.

Key words: stent, infection, coronary artery, complication

Introduction

Coronary artery stenting was initially reported in 1987.¹ The original indications for stenting of coronary arteries were to treat abrupt closure of the artery and restenosis following percutaneous transluminal coronary angioplasty (PTCA).² Stenting of the coronary arteries is now used yearly in 40–60% of all interventional coronary artery procedures in over 700,000 patients.^{2,3}

The understanding of the pathophysiology of coronary artery disease (CAD) is evolving. It has been suggested that atherosclerosis may be a complication of an infectious etiology.⁴ By using a stent to treat CAD, a foreign body is directly juxtaposed with an area of inflammation. Although the precise pathophysiology has not been elucidated, the first case of an infected coronary artery stent was reported in 1993.⁵ Although this is an exceedingly rare event, the associated mortality is alarmingly high. This article is designed to describe the reported cases of stent infection in order to call attention to a problem that may be underrecognized and more common than appreciated.

Case Analysis

Analysis of the four cases in Table I shows that symptoms of stent infection present days to weeks after the initial coronary intervention. All four patients developed fevers and at least two patients developed postintervention angina. Two patients had infection with *Pseudomonas aeruginosa*, which seems to be an unusual organism for a catheter-related infection. Perhaps the femoral access site predisposes to pseudomonas infections. Surgical removal of the infected stent and artery complex was performed on nearly all cases. Despite aggressive measures, the majority of patients died. In patients who have had a coronary artery stented, the presence of angina and fevers should make the clinician suspicious for a stent-related infection.

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Received: November 16, 1999

Accepted with revision: January 6, 2000

TABLE I Summary of cases

Age /sex	Presenting symptoms	Vessel	Stent type	Organism	Hospital course and treatment	Outcome	Source
49/M	MI 6 years prior to admission; unstable angina and fevers 1 week after intervention	90% proximal LAD	Palmaz-Schatz	<i>Pseudomonas aeruginosa</i>	Empiric antibiotics on and off for several weeks. Anterior mitral valve prolapse and chordal rupture of anterior papillary muscle. Surgical repair.	Death	Leroy <i>et al.</i> (5)
38/M	Diabetes mellitus; unstable angina s/p stent with return of angina 4 days later accompanied by fevers and chills	Circumflex	Palmaz-Schatz	<i>Pseudomonas aeruginosa</i>	CT of chest revealed increased contrast uptake around the stent and moderate pericardial effusion. Surgical repair and antibiotics × 4 weeks	No immediate complications	Bouchart <i>et al.</i> (10)
54/M	Angina; 4 days after the stent placed, the patient suffered an anterior MI with subsequent fevers and leukocytosis	90% stenosis of LAD	Microstents (AVE, Inc.) ×2	<i>Staphylococcus aureus</i>	Progressive heart failure and death. Postmortem showed complete destruction of LAD near the distal stent	Death	Grewe <i>et al.</i> (17)
66/F	4 Weeks after stent placement, the patient developed fevers and leukocytosis	RCA	Palmaz-Schatz	<i>Staphylococcus aureus</i>	Deterioration of the patient's status; surgical resection of the stent and infected artery	Death	Gunther <i>et al.</i> (18)

Abbreviations: MI = myocardial infarction, LAD = left anterior descending artery, CT = computed tomography, M = male, F = female.

Other investigators have reported cardiovascular complications of stent placement or PTCA. McCready *et al.* reported a patient who developed aortic valve endocarditis and subsequently died.⁶ Timsit *et al.* described an aortic root abscess and *Pseudomonas aeruginosa* bacteremia after PTCA.⁷ Dieter *et al.* report a case of an infected aortic arch pseudoaneurysm 1 month after coronary stent deployment in an 82-year-old man (methicillin resistant *Staph. aureus*). The patient died 2 days postoperatively after repair of the aortic arch.⁸

Few data are available on the long-term risk for coronary artery stent infection. James *et al.*⁹ retrospectively examined the risk of bacteremia associated with stent deployment between January 1992 and February 1996. In all, 210 patients underwent coronary artery stenting. Seven of these patients had bacteremia (3—*S. aureus*, 2—*Enterobacter cloacae*, 1—*Proteus mirabilis*, 1—*Serratia marcescens*). Six of the seven bacteremic patients were accessed via the right common femoral artery and the seventh patient via the brachial artery. The arterial sheaths were left in for up to 2 days.⁹ James *et al.* noted the following factors associated with bacteremia: local complications at the site of arterial puncture in 6 of the 7 bacteremic patients and only in 10 of the nonbacteremic patients; however, none of the stents appeared infected.⁹ Bouchart *et al.* suggest that multiple repeat procedures through the same arterial sheath may increase the risk for bacterial infection.¹⁰

In an analysis of coronary angiography and PTCA, Shea *et al.*¹¹ prospectively followed 164 patients for bacteremia. Blood cultures of the patients were examined at the end of

each procedure and again 30 min later via the arterial sheath. In this study, approximately 21% of the patients were diabetic and 31% had sustained a myocardial infarction within 14 days of the study. A total of 286 blood cultures was drawn from the 164 patients; 23 (8%) of these were positive (*S. epidermidis*—14, polymicrobial—3, *Corynebacterium sp*—2, *S. aureus*—1, *Strep viridans* group—1, *S. morbillorum*—1, *Bacillus sp.*—1). Only one of the positive blood cultures (*S. aureus*) was thought to be related to the procedure; the others were thought to be contaminants, making the overall incidence 0.61%. There were no late complications in the 5 months following the study. Positive blood cultures were not associated with the length of the procedure nor use of the ipsilateral artery for repeat catheterizations.¹¹ In a study by Sande *et al.*, there were no bacteremic cases in 106 patients undergoing cardiac catheterization when the cultures were drawn from a peripheral site.¹² Malanoski *et al.* calculated the risk of *S. aureus*-associated bacteremia to be 0.25% after PTCA in 1,944 patients over 25 months.¹³

In a related study, Hearn *et al.*¹⁴ studied the risk for infection of an intravascular metallic stent with delayed bacterial challenge in a swine model. In this study, Palmaz metallic stents were placed in the iliac artery of 14 swine. Four weeks later, bacterial challenge with 5 ml of *S. aureus* (3×10^6 organisms/ml) via the ear was injected in the swine; 72 h later, the swine were euthanized. Seven of the 14 stent/artery complexes were culture positive for *S. aureus*, whereas 1 of the 14 contralateral iliac arteries (angioplastied but not stented) was positive for

TABLE II Proposed criteria for diagnosis of coronary artery stent infection

Definitive diagnosis
Autopsy or surgical specimen demonstrating an infected coronary artery/coronary artery stent complex
Possible diagnosis
Any three of the following should prompt the clinician to entertain the possible diagnosis of an infected coronary artery stent:
Coronary artery stent placement in preceding 4 weeks
Multiple repeat procedures through the same arterial sheath or complications at the site of arterial puncture
Bacteremia
Significant fevers (>101.5°F) in the absence of a known bacterial infection
Leukocytosis in the absence of a known bacterial infection or acute coronary syndrome
Acute coronary syndrome
Cardiac imaging (CT, MRI, echocardiogram, etc.) consistent with persistent inflammation

Abbreviations: CT = computed tomography, MRI = magnetic resonance imaging.

S. aureus ($p = 0.03$). The infected stented arteries showed periarterial inflammatory reaction and lymphadenopathy. The pathophysiology behind the stent infection is unknown; perhaps the stents or endothelial injury served as a nidus for bacterial adherence.¹⁴ This study emphasizes the potential of stents to become infected. However, the relation to coronary artery stent infections is less clear, since a relatively large inoculate of bacteria was used and the clinical implications of the infected stents were not studied prior to the swine being euthanized.

Furthermore, the antiplatelet medication ticlopidine and, to a lesser extent, clopidogrel can cause neutropenia which may predispose to increased risk for bacteremia following stent placement. The risk of neutropenia from ticlopidine is approximately 1% and usually develops 2–3 weeks to months after treatment is initiated.^{2, 15, 16}

Conclusion

Although the incidence of coronary artery stent infection is thought to be extremely low, based on the few reported cases it is possible to propose criteria for a definitive diagnosis of coronary artery stent infection and for a possible diagnosis of coronary artery stent infection (Table II). Since these criteria are based upon a small number of cases, it is difficult to judge their overall sensitivity and specificity. However, with such a poor prognosis for infected coronary artery stents, it is perhaps best to err initially on the side of sensitivity. It is hoped that these criteria will be revised in the future to enhance the diagnosis of coronary artery stent infection.

In a patient who has undergone coronary artery stent placement, the clinician must be very sensitive to fever, return of

angina, and bacteremia. If evidence demonstrates that the stent is infected, appropriate antibiotics should be instituted, the stent should be removed, and the infected tissue should be debrided.^{5, 10} The complication rate at the present time does not warrant the use of prophylactic antibiotics prior to potentially high-risk procedures (e.g., dental procedures). Furthermore, the low infection rate of coronary artery stents may be a result of the inflammatory nature of atherosclerosis which may provide a protective benefit against bacterial infection of the stent.

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