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Histologic analysis of a covered stent implanted for pseudoaneurysm in a coronary artery

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Abstract

Covered stents are the standard of care when coronary perforations complicate percutaneous coronary interventions (PCIs) and have also been utilized in the treatment of coronary aneurysms. We present the clinical and histologic features of a patient who developed a coronary perforation and pseudoaneurysm four years following deployment of intracoronary stents. Although the covered stent successfully sealed the perforation, subsequent thrombosis led to a fatal acute myocardial infarction.

Keywords

Percutaneous Coronary Interventions (PCI); Coronary perforation; Pseudoaneurysm; Covered Stent

Introduction

Covered stents are the standard of care when coronary perforations complicate percutaneous coronary interventions (PCIs)[1,2,3,4,5] and have also been utilized in the treatment of coronary aneurysms [6]. To our knowledge, only histologic evaluation of covered stents deployed in experimental animals have been reported [7, 8] but not following implantation in human coronary arteries. We present the clinical and histologic features of a patient who

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developed a coronary aneurysm and perforation 4 years following deployment of intracoronary stents.

Results

A 59 year-old white man with a family history of early coronary disease in his father, ulcerative colitis in his mother and daughter and Wegener's granulomatosis in his son, had a personal history of hypertension and coronary artery disease including a myocardial infarction 4 years prior. At the time of the myocardial infarction he had seven stents placed in the 3 major epicardial coronary arteries (2 in the right coronary artery, 2 in the left anterior descending, 1 in the diagonal artery and 2 in the left circumflex). He had been asymptomatic since that time with normal annual stress tests and medicated with carvedilol 50mg/day and ASA 81mg/day. Two months prior to this admission he developed flu-like symptoms including fever, myalgias, malaise and cough. These symptoms mostly subsided after a week but occasional low grade fever and non-productive cough persisted, associated with positional chest pain and some shortness of breath. He was hospitalized and a chest CT revealed a small-moderate sized pericardial effusion. Screening enzymes revealed a mild CK-MB elevation which trended down. Lisinopril 80mg/day was added. Coronary angiography revealed an approximately 3.0 mm diameter left circumflex (dominant) with a proximal perforation vs a pseudoaneurysm (Figure 1A). He was then transferred to our institution. Aerobic and anaerobic blood cultures did not show growth after 5 days. ECG on admission showed negative ST segment displacement and negative T waves in the inferior and lateral walls. Upon admission to our hospital, vasculitis was suspected but ANCA, anti-PR3 and anti-MPO were all negative. It was decided to seal the perforation percutaneously with a 3 × 12 mm Graftmaster covered stent (Figure 1B) followed by post-dilation with a 3.5 × 12mm Voyager NC balloon. Optical coherence tomography immediately following implantation confirmed adequate stent deployment with a 3.0 mm mean diameter. Post-procedure angiogram demonstrated TIMI 3 flow with no evidence of perforation, dissection or embolization (Figure 1C). He was placed on aspirin 81mg/ day and clopidogrel 75mg/ day.

Over the following days the patient developed hypotension, tachycardia, and fever and had to be intubated. ECG now showed a 1 mm ST segment elevation in the lateral wall. A blood culture was positive for streptococcal viridians and a transthoracic echocardiogram demonstrated severe mitral regurgitation with possible papillary muscle rupture. After insertion of an intraaortic balloon, he was taken to the OR 22 days following covered stent deployment. At surgery, 4/4 grade mitral regurgitation, rupture of the posteromedial papillary muscle head with no evidence of endocarditis were noted and the mitral valve was replaced with a 29 mm Medtronic Mosaic porcine valve prosthesis. The posterior valve apparatus was left intact. Histologic examination of the papillary muscle showed coagulation necrosis. The anterior leaflet showed no evidence of endocarditis. The patient subsequently developed pulseless electrical activity and expired 8 days following mitral valve replacement.

Post-mortem examination revealed cardiomegaly (780g), a remote anteroseptal subendocardial, a sub acute myocardial infarction involving the posteromedial papillary muscle, and an external rupture (Figure 2) with blood and blood clots in the pericardial space (pericardium was left open following the mitral valve surgery). On gross examination there was a 3.4 by 2 cm fusiform aneurysm/pseudoaneurysm containing a well formed thrombus overlying the perforation site in the circumflex vessel. This vessel contained the covered stent that could be seen sealing the perforation (Figure 3A). Histologic evaluation of the covered stent shows that the stent was slightly under-expanded in the segment of calcified artery (Figure 3B) at the site of perforation. The stent contained a sub-occlusive

thrombus (Figure 3B and 3C). At the perforation site there was very little inflammation within the vessel wall; however, there was a florid mononuclear inflammatory infiltrate (and some eosinophils) in the aneurysmal vessel wall adjacent to the rupture site (Figure 3D). Immunohistochemistry showed the vast majority of inflammatory cells to be CD68 and CD3 positive macrophages and T cells, respectively with almost complete absence of B cells. The myocardial histology did not show inflammation in non-infarcted areas. The remainder of the autopsy revealed small vessel bilateral pulmonary thromboemboli, moderately fatty liver (1,700 g), arteriolonephrosclerosis, and anoxic brain injury.

Discussion

Covered stents have been successfully employed to treat perforations that occur as a complication of PCI [1–5]. Although coronary aneurysms or pseudoaneurysms are known possible complications of stents, especially drug-eluting stents [9,10,11], this case is unusual because leaking occurred not at the site of the stent but 1.5 cm from its edge. The stent was placed 4 years prior and since no angiography was performed in the ensuing 4-year-span, it is impossible to know the age of the aneurysm. Likewise, the etiology of the aneurysm, inflammation and leak is unknown at this stage following the covered stent deployment. Based on the clinical history, the coronary leak and the pericardial effusion were probably associated with the flu-like symptoms that developed two months prior to this admission. The florid inflammation observed in the aneurysm wall may have been associated with the aneurysmal dilatation of the native stented artery and eventually led to development of the vascular leak which necessitated covered stent deployment. Although free of significant inflammation at the site of perforation (highest wall stress?), the vessel containing the covered stent did develop stent thrombosis which led to a large infarct, papillary muscle infarct with rupture, and free wall rupture.

In summary, this is an unusual case with aneurysm formation and perforation 4 years following stent deployment. The etiology of the aneurysm is unclear but it seems plausible that there was vessel wall inflammation associated with the stent placed four years previously and this caused the vessel leak, necessitating placement of the covered stent. The latter, although successful in sealing the perforation and achieving TIMI 3 flow, was followed a few days later by stent thrombosis and devastating mechanical complications.

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Figure 1.

Figure 1A. Left circumflex coronary artery perforation with an adjacent aneurysmal cavity (outlined by arrows).

Figure 1B. Graftmaster covered stent (arrows) is successfully deployed at the site of the perforation.

Figure 1C. Angiogram immediately following stent deployment shows sealing of perforation with TIMI 3 flow distal to covered stent.

Figure 2.

Cross section of the heart at the level of the base of the papillary muscles. The old anterior subendocardial infarct (arrowhead) and the acute posterolateral infarction can be seen with marked thinning of the myocardial wall near the rupture site (arrow).

Figure 3.

Figure 3A. Postmortem view of opened aneurysmal cavity (outlined by black arrows) reveals the perforation of the left circumflex coronary artery with adequate sealing of perforation by the Jostent Graftmaster stent (white arrow).

Figure 3B. Histology of plastic section taken at the level of the perforation confirms adequate sealing of perforation (arrows) but shows incomplete expansion in area adjacent to calcification (arrowheads) and subocclusive thrombus; only scant inflammation is seen in the vessel wall.

Figure 3C. Higher power view of the wall of the vessel at the site of the perforation (arrow). Calcium deposits are present in the wall of the vessel (C).

Figure 3D. Florid inflammation comprising primarily mononuclear cells with some eosinophils is present in the aneurysm cavity wall near the perforation site.