Bypass Graft Stent Fracture Leading to Saphenous Vein Graft Pseudoaneurysm

W. Kyle Stribling, MD; Andreas W. Prinz, MD; On Topaz, MD; Rehan Khan, MD; Angela Gentili, MD; Ion S. Jovin, MD, ScD

An 83-year-old man, with a history of coronary artery bypass graft surgery 19 years before presentation and subsequent percutaneous coronary intervention to saphenous vein graft with placement of a paclitaxel drug-eluting stent 16 years later, presented with a 2-week history of fevers, productive cough, and pleuritic chest pain. He was found to have leukocytosis and chest x-ray evidence of pneumonia with a widened mediastinum. He was admitted for treatment of protracted pneumonia. He had a borderline elevated troponin I, which, in the absence of anginal pain, was thought to be secondary to demand ischemia. A chest radiograph obtained at admission showed bilateral pleural effusions, a right lower lobe infiltrate, and a widened mediastinum (Figure 1A and 1B). An ECG was initially unremarkable (Figure 1C). Blood cultures were positive for methicillin-sensitive *Staphylococcus aureus*, and despite antibiotic therapy, the patient became progressively hypotensive. A repeat ECG showed anterolateral T-wave inversions (Figure 1D). Chest computed tomography (CT) (Figure 2A and 2B) revealed a fractured stent and a giant aortocoronary saphenous vein graft pseudoaneurysm. Transthoracic echocardiography then revealed a mass that was compressing the right ventricular outflow tract, suggesting that the cause of the patient's hypotension was low cardiac output due to right ventricular outflow tract obstruction (Figure 2C and Movies I and II in the online-only Data Supplement). The patient was believed to be too unstable for surgery, and therefore he was taken urgently to the cardiac catheterization laboratory for placement of a covered stent across the apparent graft perforation (Figure 2D), but the attempt was unsuccessful. The patient became progressively hypotensive and later expired in the coronary care unit. Although autopsy was declined by the family, the cause of death was thought to be heart failure due to a giant aortocoronary saphenous vein graft pseudoaneurysm caused by stent fracture.

Stent fracture is a rare but increasingly recognized complication of percutaneous coronary intervention, usually occurring after placement of drug-eluting stents and stents placed in the right coronary artery.[1] Little is known about the clinical presentation of stent fracture, but the most common presentation appears to be acute coronary syndrome, with unstable angina or non–ST-segment elevation myocardial infarction being reported the most frequently.[2] In many cases, the long-term prognosis is benign.[1] Sixty-four–slice CT coronary angiography has been shown to be more sensitive and specific for detection of stent fractures than conventional coronary cine angiography and coronary intravascular ultrasound.[3] This case represents an unusual complication of stent fracture in which the diagnosis was apparent by CT imaging of the chest and was refined by echocardiography and coronary angiography.

Disclosures

None.

References

1. Kim HS, Kim YH, Lee SW, Park DW, Lee CW, Hong MK, Park SW, Ko JK, Park JH, Lee JH, Choi SW, Seong IW, Cho YH, Lee NH, Kim JH, Chun KJ, Park SJ, for the Long-DES-II Study Investigators. Incidence and predictors of drug-eluting stent fractures in long coronary disease. *Int J Cardiol*. 2009;133:354–358.
2. Chhatriwalla AK, Cam A, Unzek S, Bhatt DL, Raymond RE, Lincoff AM, Whitlow PL, Ellis SG, Tuzcu EM, Kapadia SR. Drug-eluting stent fracture and acute coronary syndrome. *Cardiovasc Revasc Med*. 2009; 10:166–171.
3. Pang JH, Kim D, Beohar N, Meyers SN, Lloyd-Jones D, Yaghmai D. Detection of stent fractures: a comparison of 64-slice CT, conventional cine-angiography, and intravascular ultrasonography. *Acad Radiol*. 2009; 16:412–417.

From the Department of Medicine, Virginia Commonwealth University, Richmond (W.K.S., A.W.P., O.T., A.G., I.S.J.), and Departments of Medicine (W.K.S., A.W.P., O.T., A.G., I.S.J.) and Radiology (R.K.), McGuire Veterans Affairs Medical Center, Richmond, Va.

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Correspondence to W. Kyle Stribling, MD, Department of Medicine, Virginia Commonwealth University, PO Box 980051, Richmond, VA 23298-0051. E-mail wkstrib@yahoo.com

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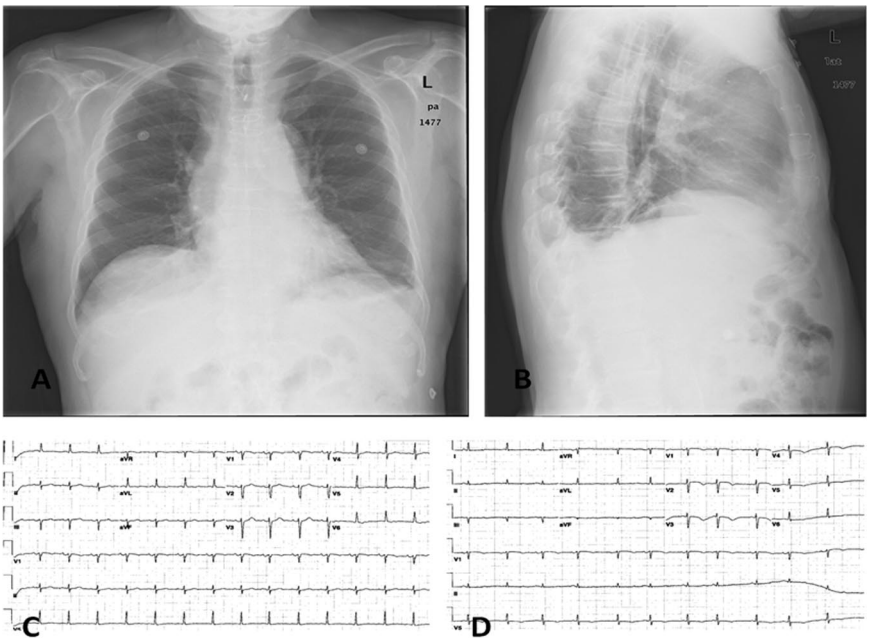

Figure 1. Chest radiograph in anteroposterior and lateral views (A and B) showing bilateral small pleural effusions, a right lower lobe infiltrate, and a widened mediastinum. ECG on admission (C) showed sinus rhythm without ischemic changes. Repeat ECG (D) showed diffuse anterolateral T-wave changes.

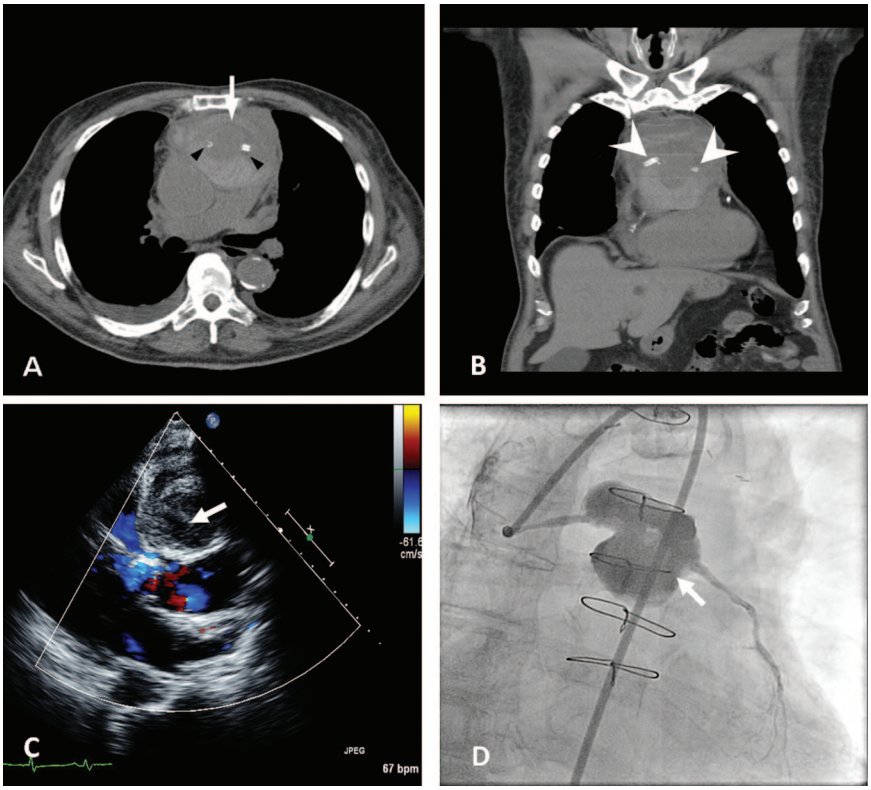

Figure 2. Chest CT (A and B) shows a fractured stent (arrowheads) and a giant aortocoronary saphenous vein graft pseudoaneurysm (arrow). Transthoracic echocardiography shows a fluid-filled mass (arrow) that was compressing the right ventricular outflow tract (C). Coronary angiogram shows contrast extravasation in the area of the fractured stent and the giant pseudoaneurysm visualized by CT and echocardiography (D; arrow). The attempt to deploy a covered stent across the perforation was unsuccessful.