A 63-year-old man was referred to our outpatient clinic with atypical chest discomfort. The patient had had known rheumatoid arthritis for >30 years. Because of recurrent pleural effusions, the patient had right-sided pleural decortication performed 25 years ago. Twelve years ago, the patient presented with chest pain not related to exertional activity but relieved by sublingual nitroglycerin. The patient had no complaints of dyspnea or objective signs of heart failure. Coronary angiography was normal except for myocardial bridging of the mid left anterior descending coronary artery (LAD; Movie I in the online-only Data Supplement). The patient responded well to treatment with antianginal medication. However, because of nitroglycerin-resistant chest discomfort, the patient was referred again to our clinic. No dyspnea was present, and there were no signs of left- or right-sided heart failure. His heart rate was 75 bpm and blood pressure was 135/80 mm Hg. Pulsus paradoxus was absent, and heart sounds were normal. Laboratory investigations including tests for liver and renal function, calcium metabolism, hemoglobin, and C-reactive protein were normal. A normal interferon-\( \gamma \) level excluded previous tuberculosis. Transthoracic echocardiography revealed normal biventricular function, a grade 2 aortic regurgitation, normal pulmonary pressure, and normal movement of the interventricular septum. No pericardial effusion was present; however, image quality did not allow a detailed evaluation of the pericardium. Cardiac computed tomography showed a calcified pericardial band from the base of the heart along the interventricular and atrioventricular grooves encircling the heart (Figure 1A). A calcific sprout from the interventricular portion of the calcific band crossing the LAD embedded into the midportion of the vessel, leading to severe calcification, at which location stenosis could not be excluded (Figure 1B and 1C). In all other coronary segments, calcification was absent. Retrospective evaluation of the previous coronary angiography examination revealed the presence of a heavily calcified pericardial band (Movie 1 in the online-only Data Supplement). What was thought to be classic myocardial bridging was in fact an indentation in the LAD by the anterior calcific band, leading to systolic mid-LAD compression. A simultaneous right- and left-sided cardiac catheterization with pressure measurements was not indicative of pericardial constriction. A high-grade fixed stenosis of the mid-LAD was demonstrated on coronary angiography (Figure 2 and Movie II in the online-only Data Supplement). Fractional flow reserve measurement (after nitroglycerin injection and adenosine infusion) over the stenosis was positive (fractional flow reserve, 0.70). The mid-LAD lesion was treated with percutaneous coronary intervention using rotablation with a 1.25-mm drill and balloon angioplasty, followed by deployment of a drug-eluting stent (Movie III in the online-only Data Supplement). The treatment relieved the patient’s discomfort.

Discussion
Calcific pericarditis (CP) is most frequently associated with tuberculosis; hence, the condition is increasingly rare in developed countries. Other causes of calcific pericarditis include cardiac surgery, radiation, infection, sarcoidosis, malignancy, uremia, and rheumatoid disorders. In the present case, rheumatoid arthritis (with previous pleuritis and thoracic surgery) is the most likely cause of CP. CP is usually generalized, but the localized pattern with constriction has previously been described. In a previous report, a calcific pericardial ring led to both constriction and systolic mid-LAD obstruction, which was successfully treated with pericardectomy. In the present case of localized CP, we could for the first time demonstrate the evolution from a dynamic to a hemodynamically significant fixed mid-LAD stenosis caused by LAD entrapment by an anterior pericardial calcific band. Besides pericardial manifestations, rheumatoid disorders are associated with accelerated atherosclerosis and endothelium dysfunction, resulting in coronary artery disease. However, neither computed tomography nor coronary angiography revealed coronary disease beyond what was found in relation to the solitary LAD lesion. Moreover, the patient had no clinical signs of peripheral arterial or cerebral atherosclerotic disease. Bypass grafting to the distal LAD, percutaneous coronary intervention, and antianginal medication were discussed as potential treatment strategies. Local pericardectomy of the anterior calcific band was assumed to be associated with a high risk of LAD damage. On the
basis of the presence of myocardial ischemia as assessed by fractional flow reserve and the patient’s treatment preference, percutaneous coronary intervention with stenting was performed. Because the disease process of chronic CP may be ongoing, clinical and echocardiographic follow-up has been scheduled.

Conclusions
CP is a rare but important differential cause of coronary artery stenosis. In this case, a significant coronary stenosis caused by localized CP was successfully treated with percutaneous coronary intervention.

Disclosures
None.

References
Entrapment of the Left Anterior Descending Coronary Artery by Localized Calcific Pericarditis: From Dynamic to Fixed Coronary Stenosis
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**Movie Legend**

**Movie 1:** Coronary angiography (April 2000), right cranial view, showing systolic compression of the mid portion of the left anterior descending coronary artery (yellow arrow) caused by an anterior pericardial calcific band (red arrow). Best viewed with Windows Media Player.

**Movie 2:** Coronary angiography (December 2012), right cranial view, showing the fixed stenosis in the mid portion of the left anterior descending coronary artery (yellow arrow) caused by an anterior pericardial calcific band (red arrow). Best viewed with Windows Media Player.

**Movie 3:** Coronary angiography (March 2013) after stenting of the left anterior descending coronary artery. Best viewed with Windows Media Player.