to the chest wall that needed to be taken down. Pulsion diverticula are more common at this location; however, we did note all layers of the esophagus in the diverticulum (although the muscularis was attenuated), making this a true diverticulum.

There might also be some abnormalities in esophageal peristalsis at the time of the initial leak secondary to the inflammation in the area. Akin to changes in small bowel motility after resection, esophageal peristalsis may have been altered after perforation. We have no literature that sheds light on how esophageal peristalsis is affected by stent placement, but there may be some changes that need to be studied further.

Esophageal stents have changed the management of patients with esophageal perforations. In selected cases, they help to avoid major surgical interventions and the accompanying morbidity and mortality. Little is known, however, regarding the long-term complications of treating esophageal perforations with these minimally invasive approaches. In light of our findings, further studies looking at the early, intermediate, and long-term complications of esophageal stenting for the management of spontaneous perforation are needed to clearly establish the risk for poststent esophageal diverticulum formation.

We thank John Hagen for his excellent illustrations of the surgical procedure.

References

Acute inferior wall myocardial infarction secondary to ruptured sinus of Valsalva aneurysm in a 22-year-old man
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Sinus of Valsalva aneurysm (SVA), a rare condition of thinning of the wall of the aortic sinus, usually enlarges with time and remains undetected until rupture. After rupture, SVAs may protrude into any heart chamber, more commonly the right atrium or ventricle, or rupture into the pulmonary artery or interventricular septum. Those that remain unruptured are typically asymptomatic hence easily ignored. We report a case highlighting the importance of recognition of atypical manifestations of the disease.
CLINICAL SUMMARY

A 22-year-old man without any significant medical, surgical, or family history of any cardiovascular disease, was driving a car when he suddenly felt chest discomfort (squeezing type radiating to inner part of left shoulder and upper arm), palpitations, profuse sweating, and transient loss of consciousness. He was brought to the emergency department, where on arrival his vital signs were stable. Results of general physical examination were normal except for a grade 2/6 diastolic murmur heard over the apex. Electrocardiography showed ST-segment elevation in leads II, III, and aVF. Echocardiography showed coronary artery disease manifestations. Laboratory findings showed a creatine kinase level of 67 IU/L, a creatine kinase isoenzyme MB level of 26.17 IU/L, and a troponin I level of 0.13 ng/mL. Acute inferior wall myocardial infarction (MI) was diagnosed, and the patient was transferred for percutaneous coronary intervention. Coronary angiography (Figure 1, A) showed occlusion in the distal right coronary artery (RCA). Percutaneous aspiration thrombectomy was done, and blood flow to the RCA was restored. Postprocedure coronary angiography showed successful revascularization without residual stenosis, and distal blood flow was evaluated at Thrombolysis In Myocardial Infarction level 3 (Figure 1, B), thus not necessitating coronary stent implantation. The patient’s chest pain was relieved after the procedure; however, a grade 2/6 diastolic murmur remained, warranting further evaluation. Echocardiography at this time showed aortic clover flap and ruptured right coronary sinus protruding into the right atrium (0.7 × 0.8 cm). Color Doppler Flow imaging detected continuous shunt of the sinus into the right atrium from left to right, mainly during diastole. The clinical impression was a ruptured SVA. The patient was then shifted to the cardiac surgery unit for emergency repair. The intraoperative finding was a ruptured right coronary sinus protruding into the right atrium (0.5 × 0.6 cm; Figure 2), which was repaired successfully.

DISCUSSION

SVA is a rare condition resulting from an absence of normal elastic and muscular tissue, leading to thinning of the wall of the aortic sinus. SVAs usually enlarge with time and remain undetected until they rupture. Once ruptured, they can protrude into any of the heart chambers, usually the right atrium (as happened in this case) or the right ventricle. Occasionally, SVAs rupture into the pulmonary artery or interventricular septum. Those that remain unruptured are typically asymptomatic and thus easily ignored.

FIGURE 1. A, Occlusion of the distal right coronary artery. B, The distal right coronary artery is recanalized after the procedure, with blood flow at Thrombolysis In Myocardial Infarction level 3.

FIGURE 2. Operative view of the ruptured aneurysm of the sinus of Valsalva (arrow).
Early structural valve deterioration of the Trifecta aortic valve biological prosthesis: A word of caution

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The St Jude Medical Inc (St Paul, Minn) Trifecta valve is a relatively new biological prosthesis that completed a multi-center US Food and Drug Administration Investigational Device Exemption study in 2011. During a median patient follow-up of 0.9 years of the 1014 valve implantations in that study, 1 patient underwent explant of the valve for structural valve deterioration.1 We report an additional case.

CLINICAL SUMMARY
A 67-year-old woman presented to us with New York Heart Association class IV dyspnea and severe Trifecta prosthetic aortic valve stenosis. She had multiple previous cardiac operations that included an aortic valve replacement plus coronary artery bypass graft surgery 18 years previously, redo coronary artery bypass graft surgery 13 years previously, and repeat aortic valve replacement with a 21-mm Trifecta valve 4 years previously.