Aneurysms of aortoconary saphenous vein bypass grafts are a rare complication of coronary artery bypass grafts. The authors report the incidental finding of four aneurysms in two aortoconary saphenous vein bypass grafts in a 77-year-old man with progressive generalized weakness, left facial drooping, and digitalis toxicity. A brief review of the literature on this rare condition is also provided.

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The most common symptoms of aortocoronary saphenous vein bypass graft (SVBG) aneurysms are retrosternal chest pain or discomfort associated with hemoptysis, hypotension, nausea and vomiting, and shortness of breath. Such aneurysms may occur in coronary artery bypass grafts (CABGs). We observed this rare occurrence in a 77-year-old white man with digitalis toxicity and possible transient ischemic attack on initial examination. The patient was brought to the emergency department because of generalized weakness and difficulty walking. He had not been able to stand and walk for 3 days.

Medical History
The patient had undergone two CABGs for severe multivessel coronary artery disease, 17 and 21 years previously. The medical record for his 1984 CABG was unavailable. The record of his 1988 procedure described a reverse saphenous vein graft that was anastomosed to the right coronary artery; another segment of the saphenous vein graft was anastomosed to the obtuse marginal artery; the same vein graft was anastomosed to the diagonal artery sequentially; and the left internal mammary artery was anastomosed to the left anterior descending coronary artery (LADCA).

One year prior to the current hospitalization, the patient underwent placement of a single-chamber implantable ventricular defibrillator and a single-chamber pacemaker with the minimum rate set at 40 beats/min. Cardiac catheterization at that time revealed severe disease in three native coronary arteries with a 70% or worse left main lesion and total occlusion of the LADCA, the circumflex artery, and the right coronary artery. The following grafts were identified: left internal mammary graft to the mid-LADCA, widely patent; LADCA collateralized to the distal right coronary artery; saphenous vein grafted to the circumflex marginal branch with extreme ectasia and multiple aneurysmal segments but surprisingly good flow into a large marginal branch; and saphenous vein graft to the right coronary artery occluded at its origin. Severe left ventricular dysfunction with ejection fraction about 15% to 20% and moderately severe mitral valve regurgitation were also noted.

The patient’s history also included atrial fibrillation, benign prostatic hypertrophy, chronic obstructive pulmonary disease, hypertension, myocardial infarction, type 2 diabetes mellitus, and urinary retention. In addition, he had abused alcohol for more than 30 years and smoked cigarettes for more than 60 years.

Clinical Evaluation
On initial examination, the patient reported that he had fallen and could not get up. He also reported symptoms of fatigue with generalized weakness that started 4 months previously and had progressively worsened until admission. He denied having chest pain and worsening of shortness of breath. He appeared awake and alert and had a mild left facial droop. He was afebrile, his blood pressure was 102 mm Hg systolic and 60 mm Hg diastolic; pulse rate, 44 beats/min; respiratory rate, 24/min; and oxygen saturation, 91% in room air. There was no jugular venous distention or bruit on neck examination. Decreased air entry at the right lung base was found, with no wheeze, rales, or rhonchi. Results of abdominal examination were unremarkable. A neurologic examination revealed normal muscle strength and tone. An osteopathic structural examination revealed a normal spinal curve for the patient’s age. Examination of the lower extremity showed venous stripping.

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No edema was present, and pulses were palpable bilaterally. The patient’s skin appeared dry.

A complete blood cell count showed a white blood cell count of 8.32 (reference range, 4.6–10.2). Hematocrit was 42% (reference range, 43.5%–53.7%); hemoglobin, 13 g/dL (reference range, 14.1–18.1 g/dL); and platelet count, 248 \times 10^3/\mu L (reference range, 142–424 \times 10^3/\mu L). Chemical analysis revealed the following: sodium, 140 mEq/L (reference range, 136–146 mEq/L); potassium, 3.9 mEq/L (reference range, 3.5–5.1 mEq/L); chloride, 101 mEq/L (reference range, 98–106 mEq/L); CO2, 32.2 mEq/L (reference range, 23–29 mEq/L); glucose, 143 mg/dL (reference range, 83–100 mg/dL); blood urea nitrogen, 36 mg/dL (reference range, 8.0–25.0 mg/dL); and creatinine, 1.2 mg/dL (reference range, 0.7–1.3 mg/dL).

Cardiac studies found B-type natriuretic peptide, 757 pg/mL (reference range, 0–100 pg/mL); first creatine kinase, 1444 U/L (reference range, 32–250 U/L); second creatine kinase, 713 U/L; first creatine kinase-MB fraction, 21.6 ng/mL (reference range, 0–10.4 ng/mL); second creatine kinase-MB fraction, 5.2 ng/mL; first troponin I, 0.33 mg/mL (reference range, 0–0.4 mg/mL); second troponin I, 0.16 mg/mL; and digoxin, 3.2 ng/mL (reference range, 0.8–2.1 ng/mL).

Results of a 12-lead resting electrocardiogram done prior to examination showed atrial fibrillation with a ventricular response rate of 63 beats/min and diffuse ST-T abnormalities.

An enhanced multidetector CT scan of the brain revealed an old left temporal occipital infarct. No abnormal enhancement was seen. A chest radiograph revealed changes consistent with cardiomegaly and an intact pacemaker defibrillator system (Figure 1) and showed a retrocardiac nodular density in the lateral view (Figure 2). This was a new finding compared with that obtained by chest radiographic examination done 4 months earlier and 1 year earlier.

Multidetector CT scan of the chest was done before and after intravenous contrast was injected. Findings were consistent with those seen on the chest radiograph in the anteroposterior (Figure 3) and lateral (Figure 4) views. The aorta showed some atheromatous change, and the aortic root measured 4 cm. No hilar or mediastinal lymphadenopathy was seen. There were what appeared to be aneurysms of the CABGs on the right and left sides. The right-sided graft revealed a proximal aneurysm with maximum dimension of 4 cm, with an intraluminal clot and a true lumen of 16 mm (Figure 5). Distally, there was a second aneurysm measuring almost 4.4 cm, with a clot and a true lumen of about 9 mm (Figure 6). The left CABG revealed a proximal aneurysm measuring 2.6 cm, with a true lumen of about 2 cm (Figure 7). A second aneurysm was found distal to the first, measuring 2.4 cm, with a small true lumen (Figure 8).

It was determined that the patient had a transient ischemic attack. Blood clots found in the patient’s bladder, most likely caused by long-term use of indwelling urinary catheters for chronic urinary retention and benign prostatic hypertrophy, were flushed out by a large catheter. His insulin therapy was reduced, owing to the controlled hospital diet, and he was also converted to oral furosemide from intravenous administration. The patient did very well after diuresis. His digoxin level decreased to within the therapeutic range. His strength improved with minimal intervention. His facial droop resolved. Serial cardiac markers...
knowledge, the literature has not reported four aneurysms occurring simultaneously. These aneurysms are usually discovered incidentally as a mediastinal or cardiac mass on chest radiograph\(^1\)\(^5\)\(^7\) in patients who have had CABG. Diagnosis is confirmed by CT scan\(^5\)\(^\) echocardiography\(^3\) and magnetic resonance imaging\(^1\)\(^4\). The diagnostic gold standard is coronary angiography\(^4\)\(^7\).

Two types of CABG aneurysms have been reported: true aneurysms and pseudoaneurysms. True aneurysms occur at the body of the graft\(^4\) and involve the entire vessel wall\(^5\). The

Comment

After CABG, aneurysms of aortocoronary SVBGs are reported from a few days to 21 years after surgical intervention.\(^4\) To our knowledge, the literature has not reported four aneurysms occurring simultaneously. These aneurysms are usually discovered incidentally as a mediastinal or cardiac mass on chest radiograph\(^1\)\(^5\)\(^7\) in patients who have had CABG. Diagnosis is confirmed by CT scan\(^5\)\(^\) echocardiography\(^3\) and magnetic resonance imaging\(^1\)\(^4\). The diagnostic gold standard is coronary angiography\(^4\)\(^7\).

Two types of CABG aneurysms have been reported: true aneurysms and pseudoaneurysms. True aneurysms occur at the body of the graft\(^4\) and involve the entire vessel wall.\(^5\) The
origin of true aneurysms is unknown, though risk factors may include atherosclerosis and vessel trauma during harvest. Pseudoaneurysms have been reported at the site of anastomosis, either proximally or distally. The etiologic process of pseudoaneurysms is similarly unknown. Iatrogenic causes such as suture defects and deficiency in the preparation of the saphenous vein have been implicated.

There is no consensus on clinical management of SVBG aneurysms. Intervention may be considered elective, urgent, or emergent depending on patient presentation. The treatment plan recommended by some experts is immediate surgical intervention regardless of aneurysm size to avoid potential complications of coronary embolization, fistula formation, or rupture. Other experts recommend treatment based on the size and growth rate of the aneurysm, comorbid conditions, and overall life expectancy. In this model, surgical intervention is offered to patients with low operative risk only. Medical management has not been predictive of positive outcomes, and an early aggressive surgical approach has also failed to demonstrate increased rates of survival.

In conclusion, aortocoronary SVBG aneurysms should be a part of the differential diagnosis for patients whose medical history includes CABG regardless of the time interval between surgical intervention and onset of the following symptoms: retrosternal chest pain, shortness of breath, nausea, vomiting, hemoptysis, and hypotension.

References