A 64-YEAR-OLD, 86-kg, 183-cm man with prostate cancer was evaluated in the authors’ preoperative assessment clinic before a scheduled prostatectomy. He denied cardiac symptoms and described only mild shortness of breath with heavy exertion. His past medical history was notable for an acute type-A aortic dissection that occurred 14 years before the current evaluation. The medical records were obtained from a local community hospital where the patient had received care. According to the operative report, the intimal tear of the dissection was located in the mid-ascending thoracic aorta. The dissection extended retrograde into the sinuses of Valsalva, coronary ostia, and aortic valve annulus; proceeded antegrade into the iliac arteries; involved the innominate and right carotid arteries; and was associated with severe aortic valve insufficiency. During surgery, the native aortic valve was deemed to be unsalvageable and was excised; a 25-mm bileaflet mechanical prosthesis was implanted. The ascending thoracic aorta was replaced with a 28-mm woven Dacron graft (Hemashield, Maquet Cardiovascular, Wayne, NJ), and the coronary arteries were reimplanted. The aortic arch was not addressed. The patient made an uneventful recovery without neurologic or other end-organ sequelae. He was treated chronically with warfarin because of the aortic valve prosthesis.

Six years after the aortic dissection, the patient was involved in a motor vehicle accident. At that time, a computed tomography (CT) scan obtained as part of the diagnostic evaluation revealed a persistent false lumen from the original dissection involving the aortic arch, the descending thoracic aorta, and the abdominal aorta. A mediastinal hematoma also was present. The patient was managed medically and recovered without requiring intervention. The patient currently was treated for essential hypertension with metoprolol, lisinopril, and hydrochlorothiazide, but had no other active medical problems except for the prostate malignancy. The prothrombin time and the serum prostate specific antigen concentration were elevated, but the remainder of the laboratory analysis was noncontributory. A preoperative posterior-anterior chest radiograph was abnormal (Fig 1), and a CT scan with angiographic contrast was obtained (Fig 2). What is the diagnosis?

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DIAGNOSIS: LARGE AORTIC ARCH ANEURYSM COMPLICATING CHRONIC AORTIC DISSECTION

The chest radiograph showed a widened mediastinum and suggested the presence of a large mass involving the aortic arch (Fig 1). The CT scan showed a large aortic arch aneurysm complicating a chronic aortic dissection (Figs 2 and 3). The aneurysm had a maximum transverse dimension of 7.0 cm and extended into the proximal portion of the descending thoracic aorta. The false lumen of the dissection was located along the lesser curvature of the arch, measured 4.1 cm in its largest dimension, and contained a large amount of thrombus. The dissection also involved the innominate and right carotid arteries.

The patient subsequently was admitted to the hospital and taken to the operating room for repair of the aortic arch aneurysm. After anesthetic induction and endotracheal intubation, transesophageal echocardiography (TEE) confirmed and extended the CT findings (Figs 4 and 5; Video clips 1 and 2). In addition to thrombus, spontaneous echocardiographic contrast was noted within the false lumen of the chronic dissection consistent with residual flow. Indeed, several distal fenestrations were seen in the dissection flap through which blood flow from the true to the false lumen was observed (Fig 6; Video clip 3). The aortic valve prosthesis functioned normally. The patient was cooled to 21°C during cardiopulmonary bypass. The proximal innominate, left carotid, and left subclavian arteries were removed sequentially from the aortic arch aneurysm, and anastomoses were created between each of these vessels and a trifurcated woven Dacron graft. A fenestration also was made in the innominate artery dissection flap before the anastomosis was created. Antegrade cerebral perfusion was maintained through the right axillary artery as the innominate artery and left carotid artery anastomoses were created in sequence. Blood flow to the remainder of the thorax, abdomen, and lower extremities was maintained using a right femoral arterial cannula after the native left subclavian artery was removed from the arch for anastomosis to the trifurcated graft. The arch aneurysm then was excised, a large fenestration was created in the distal dissection flap to assure distal vital organ perfusion, and the arch was replaced with a 28-mm woven Dacron graft. Antegrade cerebral perfusion from the right axillary artery was continued during the arch repair through the trifurcated graft and its anastomoses with the great vessels. Finally, an end-to-side anastomosis between the trifurcated graft and the aortic arch graft was created. After rewarming and deairing, the patient separated from cardiopulmonary bypass with inotropic support (intravenous infusions of milrinone and norepinephrine). The patient was transferred to the surgical intensive care unit in stable condition.

Aneurysm formation distal to the site of a type-A aortic dissection repair is a well-known but relatively unusual complication. The results of several studies clearly demonstrated that the ten-year freedom from reoperation on the distal aorta or its major branches after primary dissection repair exceeds 70% and may approach 90% in many centers. When dilation of the distal aorta after dissection repair does occur, the aortic arch and the proximal descending thoracic aorta are most
likely to be affected, as was seen in the current patient. The mechanism responsible for aortic dilation and aneurysm development is thought to involve progressive expansion of the false lumen resulting from continued stress on the already damaged, effaced aortic wall. In addition to male gender, younger age, and a history of Marfan’s syndrome or another connective tissue disorder, poorly controlled essential hypertension and increased pulse pressure are significant risk factors for distal aortic dilation after dissection repair. The current patient’s acute dissection occurred when he was 50 years of age, and he required triple drug therapy to control his hypertension, but he did not have a connective tissue disease. The size and patency of the chronic dissection false lumen also suggest that subsequent aortic dilation may occur. In fact, Immer et al showed that a patent false lumen exceeding more than 70% of the total cross-sectional area of the aorta is an ominous predictor of subsequent distal aortic dilation and aneurysm formation. The false lumen size in the current patient was greater than this 70% value in the aortic arch and the proximal descending thoracic aorta. The use of postoperative anticoagulation also contributes to continued false lumen patency, and the risk of long-term aortic dilation; the authors’ patient received chronic warfarin therapy because a mechanical bileaflet aortic valve prosthesis was implanted during his original operation.

Interestingly, the presence of both thrombus and blood flow within the false lumen, known as “partial patency,” may be associated with a greater risk of aortic dilation, aneurysm development, reoperation, and mortality compared with a fully patent false lumen or one that is completely thrombosed. Such “partial patency” was clearly evident in the current patient, as large quantities of thrombus were present in the aortic arch concomitant with residual, sluggish blood flow in his chronic dissection’s false lumen. The rate of enlargement of the distal aorta after dissection repair is also greater when a patent false lumen is present, but the timing and rate of distal aortic expansion cannot be anticipated with any degree of accuracy, and some patients may develop aneurysmal dilation following many years of relative dimensional stability. These observations emphasize the need for consistent lifelong surveillance in patients with repaired type-A aortic dissection to assure long-term survival because of the unpredictable nature of subsequent aneurysm development and rupture. The current patient did not have routine radiologic follow-up during the 14-year period after his original dissection repair, although a CT scan obtained 6 years after his original surgery suggested the absence of aortic arch dilatation. Lastly, failure to surgically address the aortic arch during repair of acute type-A aortic dissection may be associated with a greater risk of subsequent false lumen patency, distal aortic dilation, reoperation, and mortality regardless of whether the arch itself contains the intimal tear. The ascending thoracic aorta, which contained entry site of the patient’s aortic dissection, was replaced during his first operation, but the aortic arch was left intact, and this factor also may have played a role in the patient’s subsequent clinical course. Nevertheless, a primary objective in the treatment of acute type-A aortic dissection remains resection of the intimal entry site, especially in a hemodynamically unstable patient who may otherwise not tolerate a more extensive repair of the entire aortic arch.

APPENDIX A. SUPPLEMENTARY DATA

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1053/j.jvca.2013.05.002.

REFERENCES