Chronic Cough as a Presenting Symptom of a Giant Thoracic Aortic Aneurysm: A Case Report

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Abstract

Chronic cough has a broad differential, and thoracic aortic aneurysm (TAA) is a rare but potentially life-threatening etiology. We present a giant arch TAA in a non-dyspneic, Pacific Islander man with significant tobacco-use history who presented with chronic cough with no acute pulmonary process noted on imaging. Given the high mortality rates associated with thoracic aortic aneurysms, the purpose of this report is to highlight the importance of keeping TAA as a rare differential for chronic cough, particularly when caring for patients with elevated risk. Recognition of patients with thoracic aortic disease who have a class I indication for surgical intervention (meaning there is evidence or general agreement that surgery will be beneficial, useful, and effective) as well as prompt evaluation of their anatomical landmarks in the perioperative period is critical. Imaging and, in particular, computed tomography remain the optimal modalities to screen for thoracic aortic disease.

Keywords

Thoracic Aortic Disease, Aneurysm, Cardiology, Cardiothoracic Surgery, Internal Medicine

Abbreviations and Acronyms

CABG = coronary artery bypass graft
COPD = chronic obstructive pulmonary disease
CT = computed tomography
ER = emergency room
PAH = pulmonary arterial hypertension
TAA = thoracic aortic aneurysm
TAD = thoracic aortic disease

Introduction

Thoracic aortic disease (TAD) is primarily managed with surgery, but optimal management greatly depends on appropriate workup.[1] For individuals older than 65, thoracic aortic aneurysms are considered the 17th most common cause of death.[2] Broadly, the Centers for Disease Control and Prevention has ranked complications secondary to aortic aneurysms such as dissections and ruptures as high as the 19th leading cause of death in the United States, and they are estimated to cause 43 000–47 000 deaths annually.[3] In the absence of surgical repair, unexpected rupture of aortic aneurysms is almost uniformly fatal.[4] The data for outcomes of non-operative management of descending thoracic aortic aneurysm (TAA) is dismal.[4] The majority of TAA are fusiform, but up to 20% of aortic aneurysms may be saccular.[4] In comparison to fusiform aneurysms, which are thought to arise secondary to wall degeneration from atherosclerosis, saccular aneurysms have a more varied etiology, including aortic infection vasculitides, trauma, atherosclerosis, and previous aortic surgery.[5,6,7] Connective tissue disorders, such as Marfan syndrome, are also associated with TAAs. Notably, saccular aneurysms were more frequently observed in the thoracic aorta than in the abdominal aorta.[4] Tobacco cessation, hypertension management, and lipid-lowering therapy are important risk modifiers. Repair is indicated when the aneurysm expands > 5.5 cm or undergoes rapid expansion ≥ 5 mm per year. Fluoroquinolone use is associated with an increased risk of aortic aneurysm dissection or rupture and should therefore be avoided in patients known to have aortic aneurysms.[8] For the descending aorta, an aneurysm > 6 cm is associated with a cumulative 15.6% risk of rupture, dissection, or death. In comparison, the mortality rate for patients who have undergone elective surgical repair of TAA is 4% to 21%, on average 13.9%.[9]

Case Report

An 81-year-old Pacific Islander man with a past medical history significant for a 30 pack-year smoking history (quit using tobacco in 2000), well-controlled type II diabetes mellitus (hemoglobin A1c: 7.3), and essential hypertension presented to his primary care physician reporting several months of a productive cough with non-bloody sputum. His other past medical history included hyperlipidemia, allergic rhinitis, gastroesophageal reflux disease, chronic kidney disease stage II (baseline serum creatinine 1.39 with a glomerular filtration rate of 74), erectile dysfunction, and benign prostatic hyperplasia. Notably, he did not have a personal nor family history of connective tissue disease, including Marfan syndrome, nor did he have a history of chronic obstructive pulmonary disease (COPD).

His blood pressure in the primary care physician's office was 160/85 mm Hg with a heart rate of 91. He was afebrile (temperature: 97.8°F), with a respiratory rate of 18 and an oxygen saturation of 98% on room air. Outpatient chest x-ray was concerning for a large thoracic aneurysm (Figure 1). Subsequently, he was sent to the emergency room (ER) for further workup, where a non-contrast chest computed tomography (CT) scan confirmed a large saccular aortic arch aneurysm (8.3 cm) without any leaks (Figure 2). The aneurysm was noted to extend anteriorly and inferiorly. The origin of the aneurysm was just distal to the takeoff of the left subclavian artery. The neck of the aneurysm measured 6.7 cm x 4.0 cm, and the overall area measured 8.3 cm x 7.2 cm x 2.3 cm. Gated CT of the aorta with contrast further confirmed the size and location of the aneurysm (Figure 3). Three-dimensional reconstruction of the aneurysm

from the Gated CT of the aorta is shown in Figure 4. There was no evidence of aortic dissection nor aortic wall rupture. Furthermore, in 2011, a screening abdominal aortic ultrasound was conducted, which ruled out an abdominal aortic aneurysm but noted a slightly ectatic (dilated) aorta measuring 2.5 cm.

His outpatient medications included alogliptin 12.5 mg once daily, metformin 1000 mg once daily, amlodipine 5 mg once daily, chlorthalidone 25 mg once daily, lisinopril 20 mg once daily, omeprazole 20 mg once daily, rosuvastatin 5 mg once daily, sildenafil 100 mg as needed, tamsulosin 0.4 mg once daily, finasteride 5 mg once daily, cyanocobalamin 500 mcg once daily, and folic acid 1 mg once daily.

In the ER, the patient's blood pressure was 202/107 mm Hg, and he was admitted to the hospital for hypertensive urgency and further evaluation and workup of the TAA. The lungs were clear bilaterally, and his heart showed regular rate and rhythm without murmurs, rubs, or gallops. Upper and lower extremities had 2+ pulses bilaterally. Transthoracic echocardiogram noted grade II diastolic function, aortic root measuring 4.3 cm, trileaftlet aortic valve, aortic sclerosis without stenosis, and normal systolic function with an estimated ejection fraction of 50% to 55%. Given that the CT scan noted atherosclerotic cardiovascular disease, pre-operative cardiac catheterization was obtained to evaluate the need for simultaneous intraoperative coronary artery bypass graft (CABG). Catheterization revealed moderate non-obstructive coronary artery disease with 50% to 70% stenosis in the mid-left anterior descending artery, 50% stenosis in the proximal left circumflex artery, and less than 25% stenosis within the right coronary artery. Therefore, CABG was not recommended as part of the operative plan. Following a detailed discussion with the patient and family members about the risk versus benefit of surgery, and given that the thoracic aortic aneurysm > 5.5 cm, aggressive blood pressure control was warranted and achieved with a goal of no greater than 130/80 mm Hg through esmolol drip. It was deemed that the patient had a class I indication for surgical intervention. He was determined to be a poor candidate for total endovascular aortic repair alone and required an expanded debranching aortic surgery. After an extensive discussion about the risks and benefits of surgery, the patient and family opted for definitive surgical management.

Post-operatively, the patient was transferred to the intensive care unit for recovery and neurovascular monitoring. He was extubated on postoperative day 1, and postoperative CT scans demonstrated an unchanged large saccular aortic arch aneurysm without any significant pulmonary pathology. In the ensuing days, the chest tube output remained minimal, but the patient continued to experience the same non-bloody, productive cough which had resulted in his initial presentation. Upon further investigation, flexible fiberoptic nasopharyngeal endoscopy revealed normal vocal cords. He was treated for gastroesophageal reflux disease with a proton pump inhibitor, which was also ineffective in resolving his cough. On postoperative day 10, the patient syncopized and died after an extended coughing fit.

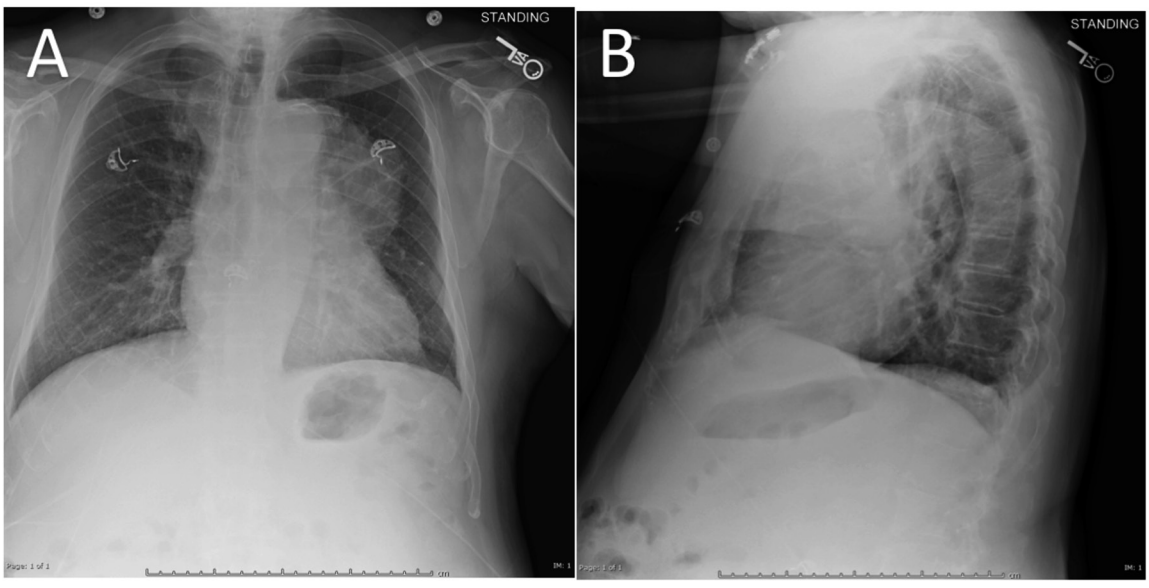

Figure 1. Outpatient Chest-X ray (Posterior-Anterior and Lateral) Demonstrating A Widened Mediastinum With A Probable Thoracic Aortic Aneurysm.

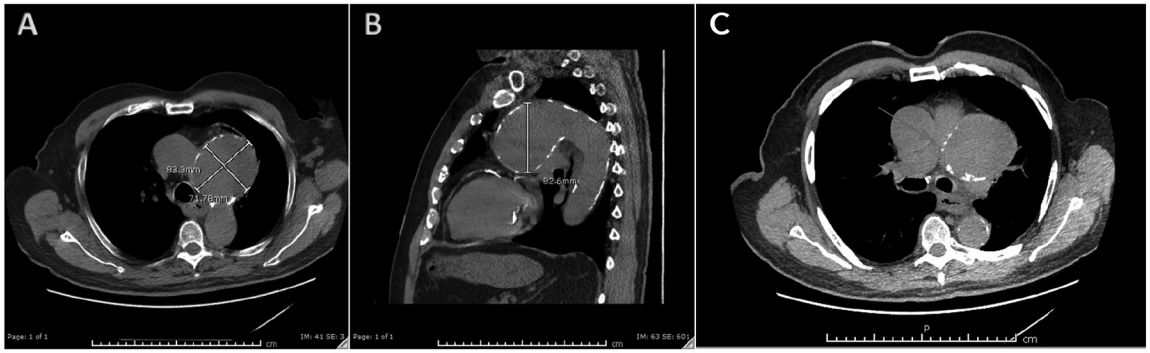

Figure 2. Non-contrast Computed Tomography Scan of the Chest. The axial view (A) and the sagittal view (B) show the 8.3-cm-diameter saccular aneurysm extending from the lateral anterior aspect of the aortic arch. Another axial view (C) demonstrates pulmonary vascular compression.

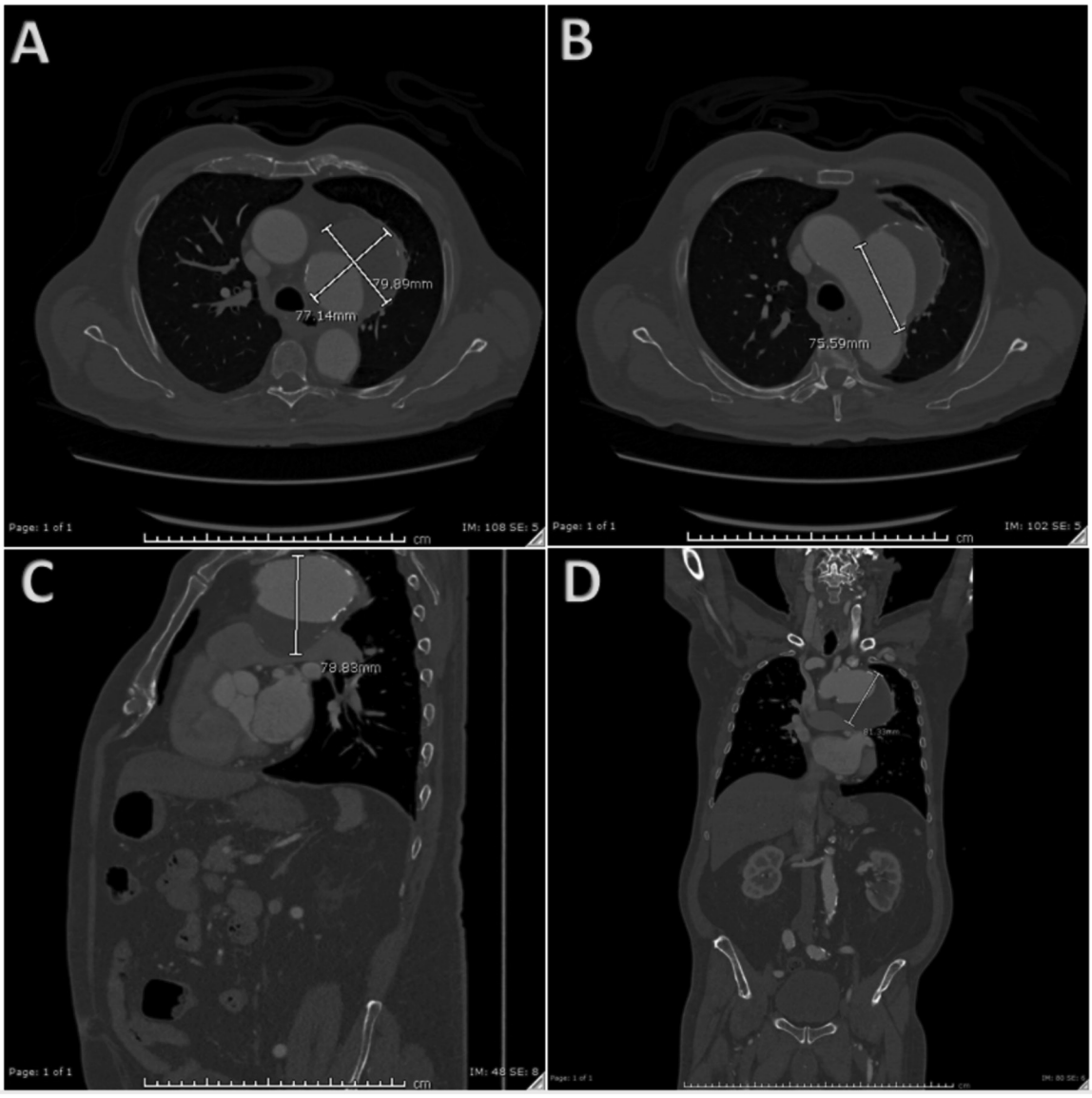

Figure 3. Gated Computed Tomography Scan of the Aorta. Scan with contrast shown in axial view (A and B), sagittal view (C), and coronal view (D) showing a large, saccular aneurysm involving the proximal aspect of the descending aorta just distal to the takeoff of the left subclavian artery, which measures approximately 7.7 cm x 7.9 cm x 8.1 cm in size (see labels).

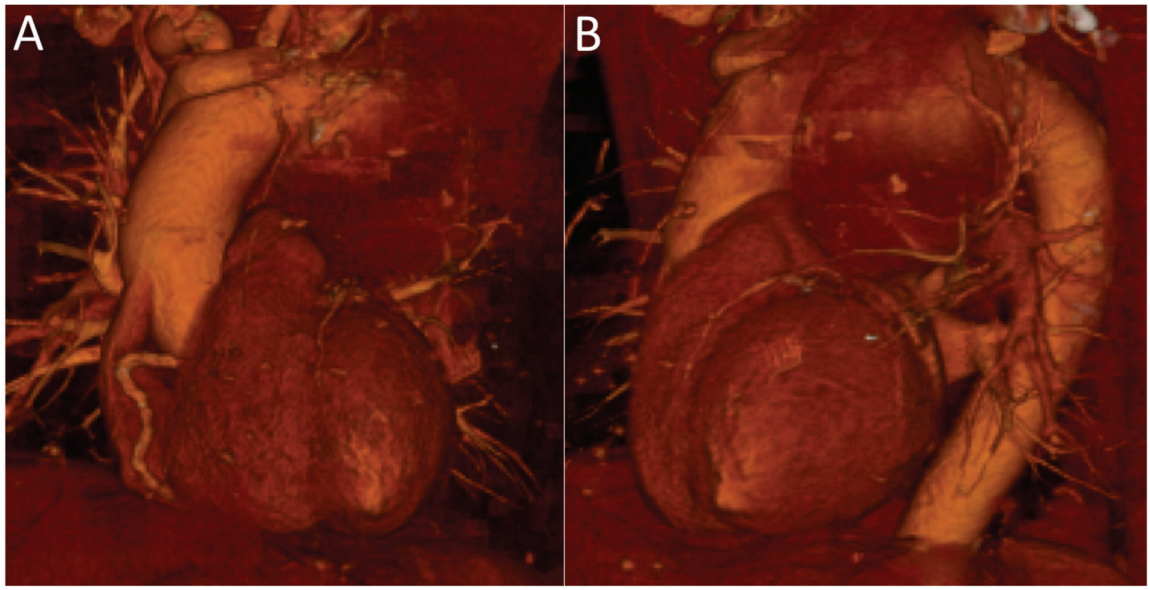

Figure 4. Three-Dimensional Computed Tomography Reconstruction of the Saccular Aortic Aneurysm. Coronal view (A) and sagittal view (B).

Discussion

Given that the patient's cough persisted in the postoperative period, it is reasonable to consider that the persistently elevated intrathoracic pressure contributed to a hemodynamic catastrophe, ultimately leading to death. Differentials worth considering include endoleak versus dissection versus rupture. Symptomatic management of the patient's cough, which included evaluation of vocal cord dysfunction and treatment for post-nasal drip and gastroesophageal reflux disease, was ineffective. The patient did not have a history of COPD, was never prescribed any medications for COPD, and his chest imaging was not consistent with a diagnosis of COPD. However, the patient also never underwent pulmonary function testing. The thoracic CT scan did not reveal any pulmonary nodules nor significant mediastinal lymphadenopathy. Ultimately it was thought that his cough possibly resulted from mass effect or compression of pulmonary vasculature by the giant TAA (Figure 2C). The mechanism of cough was thought to be similar to that of pulmonary arterial hypertension (PAH) because arterial blood flow to the lungs was traversing a narrower lumen due to mass effect. This narrowing would conceivably lead to elevated pulmonary arterial pressure. Although the precise pathophysiology of cough in PAH is unclear, Achouh et al. have suggested extrinsic small airway compression in PAH, leading to cough. Several studies also point towards small airways disease in PAH.[10] Other etiologies considered included compression of the mainstem bronchi versus the vagus nerve or a combination thereof.

Previously, there have been a few case reports of chronic cough as the presenting symptom of TAAs.[11–13] Of particular note is 1 case, where a saccular aortic aneurysm with a mural thrombus was noted to compress the left pulmonary artery and left mainstem bronchus, which was ultimately thought to be the etiology of persistent cough and hoarseness in a 71-year-old man with long-standing systemic hypertension.[11] Another case report noted the evaluation of chronic cough in a 65-year-old man with a history of hypertension and prior tobacco use, which revealed a giant thoraco-abdominal aortic aneurysm, which compressed the left main bronchus and its inferior branch.[12] Finally, 1 report noted cough as a presenting symptom of a sizeable thoracic aortic aneurysm in a 61-year-old man with a history of smoking, but that report did not comment on adjacent mass effect from the aneurysm.

Although it is unclear if our patient's cough was secondary to mass effect (Figure 2C) of his giant thoracic aortic aneurysm, this case adds to few other cases noted in the literature, which report cough as a presenting symptom of giant thoracic aortic aneurysm. Thoracic aortic disease (TAD) is typically asymptomatic but warrants a high degree of suspicion when evaluating cough in patients at an elevated risk of developing TAD. Such patients include those with a significant tobacco use history, history of hypertension, and those with genetic predispositions (Marfan syndrome, Loeys-Dietz syndrome, Takayasu arteritis, Bechet disease, and Giant cell arteritis).[13]

This case exemplifies high-risk surgery with higher than average morbidity and mortality in a patient older than 65 years, 15.6% mortality without surgery, and 13.9% mortality with elective repair in the average patient. Nonetheless, surgery was a class I indication because it was the only intervention with the pos-

sibility of efficacy. Here, shared-decision making between the patient and provider was invaluable for deciding on such an intervention. After extensive discussion with the patient and family describing the risks and benefits of this surgery, including intra-operative complications, postoperative complications, the possibility of prolonged life-support, and death, the patient and family opted for surgery.

Alternatively, the consequences of not proceeding with surgery would have been catastrophic, as evident by the esmolol drip requirement in the perioperative setting as well as the dismal data on non-operative management of thoracic aortic disease.[4] A large study about older adult patients and procedures, "Knowing the Risk" study, showed that patients > 80 years had 13 times the mortality rates of those younger than 65 years and 4.6 times those 65–80 years. However, the survival rate of those older than 80 years remained a respectable 93.6%.[14] Therefore, offering surgery to the patient was thought to be both medically and ethically indicated.

Conflict of Interest

None of the authors identify a conflict of interest.

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