

RUPTURED AORTIC ANEURYSM PRESENTING AS A STRIDOR

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SUMMARY

Stridor is an abnormal, high-pitched, whining breathing sound caused by a blockage in the throat or larynx that is usually heard in children. We describe an unusual case of an 81-year-old man brought to our emergency department with sudden onset of dyspnea and shortness of breath. Stridor could be heard without a stethoscope. We found a huge mass over the left upper chest on chest radiography, suggesting an aortic aneurysm. We believed that these symptoms were caused by a huge thoracic aortic aneurysm with trachea/bronchi compression. Chest computed tomography confirmed the diagnosis. [International Journal of Gerontology 2010; 4(2): 96–98]

Key Words: aortic aneurysm, stridor

Introduction

Shortness of breath is one of the common symptoms in the emergency department. Patients with asthma or chronic obstructive pulmonary disease may show dyspnea or increasing respiratory effort in some situations such as acute exacerbation or secondary infection.

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a history of asthma, chronic obstructive pulmonary disease or congestive heart failure, sudden onset of shortness of breath raises concern about cardiogenic asthma or a cardiovascular lesion-related problem, if there is no foreign body in the airway.

Case Report

An 81-year-old man presented at our emergency department with sudden onset of shortness of breath and dyspnea 1 hour previously. He stated that he had not choked before arrival at our emergency department, did not have a sore throat, cough, hemoptysis, fever, body weight loss, history of asthma or recent chest trauma, and did not smoke. The patient had not experienced an allergic reaction and had not had similar symptoms before. He had a past medical history of benign prostatic hyperplasia, chronic kidney disease and hypertension, which was controlled with regular attendance at a local clinic.

On examination, the patient was afebrile, with a heart rate of 96 beats/min, a blood pressure of 164/89 mmHg, a respiratory rate of 26 breaths/min, and an oxygen saturation of 84% in room air, which improved to 97% after supplementation with oxygen by a face mask (10 L/min). He had labored respiration and was



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Accepted: June 23, 2009

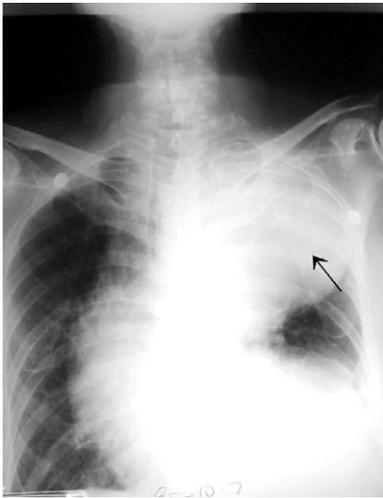


Figure 1. A huge mass lesion (arrow) over the left upper chest region with left side pleural effusion.

unable to speak in full sentences. He had no jugular vein distention, and there were no neck masses. However, marked stridor was heard even without a stethoscope, and his lung fields had bilateral crackling. His heart rate and rhythm were regular. No murmurs, rubs or gallops were heard. His abdomen was soft, without palpable masses. The patient had grade 1+ pitting edema of the lower extremities bilaterally.

The arterial blood gas was determined with a face mask (10 L/min), revealing a pH of 7.40, partial oxygen pressure of 99.5 mmHg, and partial carbon dioxide pressure of 34.8 mmHg. Blood test results were as follows: blood urea nitrogen, 34 mg/dL; creatinine, 1.7 mg/dL; and complete blood count, coagulation profile, D-Dimer and serum electrolyte panel were all within normal limits. A chest radiograph was performed and revealed a huge mass over the left upper chest region with left pleural effusion (Figure 1). We suspected a TAA with tracheal/bronchial compression leading to stridor. For further evaluation, an emergency chest computed tomography was performed and showed marked aneurysmal dilatation of the aortic arch, thus confirming the diagnosis (Figure 2).

The patient was admitted to the intensive care unit for further management and observation. After oxygen supplementation, the patient gradually stabilized. Aortic aneurysm repair was an option, but he was unwilling to undergo surgery because of his advanced age and the possible high complication rate from the operation. After 4 days of conservative treatment, he was discharged, and attendance at the outpatient department of cardiovascular surgery was arranged for him.

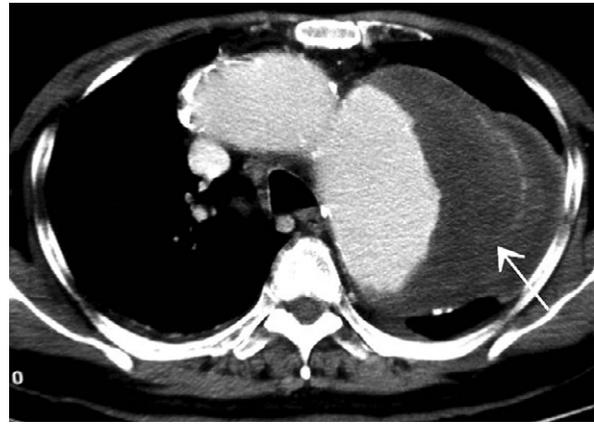


Figure 2. Marked aneurysmal dilatation of the aortic arch with cap-shaped bulging (arrow), indicating a ruptured aortic aneurysm.

Discussion

Stridor is an abnormal, high-pitched, whining breathing sound caused by a blockage in the throat or larynx. It is usually heard on inspiration. It is usually seen in children; viral laryngotracheobronchitis (croup) is the most common cause and usually has a self-limited course with occasional relapses in early childhood. The causes of stridor include foreign bodies, tumor formation, infections, subglottic stenosis, airway edema, laryngomalacia, subglottic hemangioma, abnormalities of vocal cord function, congenital anomalies of the airway, and vascular rings compressing the trachea^{1,2}.

The incidence of TAA is increasing with improvements in screening, as well as advances in imaging. TAAs are more frequently seen in men and typically occur in the sixth and seventh decades of life. The majority of TAAs (60%) involve the aortic root and ascending aorta, 40% involve the descending aorta, 10% involve the arch, and 10% involve the thoracoabdominal aorta. Most patients with TAAs are asymptomatic at the time of diagnosis, because the aneurysms are typically discovered accidentally on imaging studies. When TAAs are large, patients may suffer a local mass effect, such as compression of the trachea or main stem of the bronchus (causing cough, dyspnea, wheezing, or recurrent pneumonitis), compression of the esophagus (causing dysphagia), or compression of the recurrent laryngeal nerve (causing hoarseness). The dangerous consequence of a TAA is aortic dissection or rupture, which is potentially lethal^{3,4}.

In 1996, Nakano et al.⁵ reported a 77-year-old woman with positional dyspnea caused by compression of the tracheobronchial tree by an extensive TAA. The patient did not receive surgery, and she died from rupture of the TAA. Kameyama et al.⁶ described a 63-year-old man with dyspnea and inspiratory stridor caused by compression of the upper airway by an aortic arch aneurysm. The patient underwent surgery and died of severe pneumonia and septicemia on the sixth postoperative day.

In our case, the patient had a huge arch TAA with initial clinical manifestations of shortness of breath, dyspnea, and stridor, but not rupture, leakage or dissection. After he was advised of his condition, he chose conservative treatment rather than further investigations or surgery. He was a happy old man satisfied with the existing state of affairs, and we all respected his decision. Perhaps this was the best treatment for him.

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