

## An Accident Waiting to Happen: Thoracic Aortic Aneurysm

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n 85-year-old man presented with dyspnea and mild left precordial pain. He was a heavy smoker with chronic obstructive pulmonary disease, diabetes, hypertension, and a 20-year history of thoracic aortic aneurysm (TAA) that he refused to have repaired. Over the past few weeks he became increasingly dyspneic, dysphonic, and developed solid-food dysphagia.

On admission, he was tachypneic (22/min) and hypoxemic (91%), and breath sounds over the left hemithorax were barely audible. Chest X-ray revealed a large TAA (Figure). Hemoglobin was 10.5 g/dL (normal renal, adrenal, thyroid function), erythrocyte sedimentation rate was 60 mm/h, serum sodium level was 124 mmol/L, urine sodium level was 43 mmol/L, and urine osmolality was 693 mOsm/kg.

Chest computed tomography was ominous (Supplemental Figure A and B, available online at http://www.mayoclinicproceedings.org). He died suddenly hours later.

Most TAAs are asymptomatic and discovered incidentally on imaging in patients older than 55 years.<sup>1</sup> Current guidelines recommend intervention for any symptomatic TAA (regardless of size), aortic diameter of more than 55 mm, or rapid expansion (>5 mm/y) to anticipate acute aortic events (dissection/ rupture).<sup>2</sup> However, most patients with type A dissections have smaller-diameter aneurysms,<sup>3</sup> and recent studies suggest a need to consider a lower threshold for repair.<sup>2</sup>

Medial degeneration is the principal pathology, accelerated by hypertension and atherosclerosis risk factors. Our patient's refusal to consider intervention led to a prolonged natural history of relentless expansion (faster the larger the diameter) and myriad symptoms due to compression of the trachea and lung (causing dyspnea), esophagus (dysphagia), and recurrent laryngeal nerve (hoarseness). Despite the aortic arch aneurysm (10% of TAAs), he had neither acute neurological symptoms nor thromboembolism.<sup>1</sup> His syndrome of inappropriate antidiuretic hormone secretion has not been previously reported in TAA to our knowledge.



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FIGURE. The patient's chest X-ray 2 years prior (left) showing moderate enlargement of the aortic arch, and on admission (right) showing huge aortic aneurysm filling the left upper hemithorax.

Lack of intervention led to the inevitable conclusion.<sup>4</sup> He died of probable TAA rupture, heralded by mild pain and the "bulge sign" (Supplemental Figure A and B, available online at http://www.mayoclinicproceedings. org) on the computed tomography scan.

## ACKNOWLEDGEMENTS

The expertise of Y. Drahy, MD, in the analysis of the imaging studies is gratefully acknowledged.

## SUPPLEMENTAL ONLINE MATERIAL

Supplemental material can be found online at http://www.mayoclinicproceedings.org. Supplemental material attached to journal articles

has not been edited, and the authors take responsibility for the accuracy of all data.

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