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An Unusual ED Case: Scrotal Edema from a Thoracic Aortic Aneurysm in a 58-Year-Old Male

Yevgeniy Polunin
Rowan University

Emily Nguyen
Rowan University

Bhumi Shah
Rowan University

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Yevgeniy Polunin DO, Emily Nguyen MD, Bhumi Shah DO

Emergency Medicine Residency and Department of Emergency Medicine, Rowan University, SOM

Abstract:

Scrotal edema is not an uncommon presentation in emergency medicine. We describe the case of a 58-year-old male with no prior medical history who presented to the emergency department (ED) due to progressive scrotal edema. The patient noted increased painless testicular enlargement, dry cough, and dyspnea on exertion over the course of several weeks. Scrotal edema was evaluated by an Ultrasound (US), showing bilateral testicular edema with concern for congestive heart failure (CHF). Computed tomography pulmonary angiography (CTPE) scan showed a large 6.51 cm thoracic aortic aneurysm (TAA), cardiomegaly with dilated left sided chambers, and a small pericardial effusion. Subsequently, the patient had a transthoracic echocardiogram (TTE) and was found to have left ventricular ejection fraction of 35% with severe aortic regurgitation, causing heart failure. Patient was transferred to the operating room (OR) for an aortic valve, aortic root, and ascending aorta replacement. Patient was deemed medically stable for discharge to home 2 weeks after initial TAA findings. TAA is a silent disease and an important cause of mortality in adults. Early recognition by astute clinicians can significantly reduce morbidity and mortality.

Introduction:

Painless scrotal edema in adults has many causes including congestive heart failure, fluid retention, epididymitis, orchitis, hernia, hydrocele, varicocele, injury, testicular torsion, and testicular cancer [1]. Scrotal edema can be secondary to an underlying pathology outside of the scrotum itself. Here we describe an unusual case of a painless scrotal edema that was provoked as a result of a large TAA causing severe aortic regurgitation leading to acute CHF.

Case Presentation:

A 58-year-old male presented to the ED with a complaint of scrotal edema which had been ongoing for the past 8 weeks and was associated with intermittent dyspnea on exertion, dry cough, and weight gain. He denied chest pain, shortness of breath at rest, abdominal pain, recent sexual activity, testicular pain, trauma, penile discharge, urinary output changes, or dysuria. He denied any medical conditions or any prior surgeries. He did not take any medications daily. His last physician evaluation occurred 20 years prior to this presentation. Social history was negative for tobacco, alcohol, and drug use.

On presentation, his vital signs were blood pressure of 140/85 mm Hg, heart rate of 117 beats per minute (bpm), respiratory rate of 18 breaths per minute, temperature 98.7 degrees Fahrenheit orally, and a pulse oximetry of 95% on room air. His Body Mass Index was 26.5 kg/M2. Physical examination revealed non-toxic appearing, well appearing male. On auscultation, he had a 4/6 diastolic ejection murmur with loud decrescendo at the left lower sternal border. There was jugular venous distension and crackles at the bases of the lungs with decreased breath sounds at the right base. He had bilateral (b/l) scrotal edema without erythema, ecchymosis, pain, or lesions. Lower extremities had non-pitting edema without calf tenderness, capillary refill of less than 2 seconds, with palpable b/l dorsalis pedis and posterior tibial pulses. Otherwise, the examination was unremarkable.

A chest X-ray (CXR) performed showed trace right pleural effusion, cardiomegaly, and mild central vascular congestion. An electrocardiogram showed atrial fibrillation with rapid ventricular response at 128 bpm, left ventricular hypertrophy, without acute ST segment or T wave changes. Laboratory studies completed revealed B-type natriuretic peptide of 1715 and a high sensitivity troponin of 58. A complete blood count and basic metabolic panel were unremarkable. Urinalysis was noted to have 1+ protein, otherwise negative. Ultrasound of scrotum and testicles revealed diffuse scrotal wall edema with small b/l hydroceles, without evidence of intratesticular mass or torsion. Ultrasound of b/l lower extremities showed no evidence of deep vein thrombosis. Patient appeared clinically in acute CHF, but with new onset atrial fibrillation, lower extremity edema, dyspnea on exertion, and oxygen saturation of 95%, CTPE study was ordered to rule out a pulmonary embolism. CTPE showed a large 6.51 cm TAA, cardiomegaly with dilated left sided chambers, and a small pericardial effusion (Figure 1). After consultation with cardiothoracic surgery, the patient was started on heparin, and urgently taken to the OR for an aortic valve, aortic root, and ascending aorta replacement. Post-operative course was complicated by an episode of ventricular fibrillation relieved by a brief period of cardiopulmonary resuscitation. After 14 days in the hospital, the patient was deemed medically stable for discharge to home on Eliquis and Metoprolol.

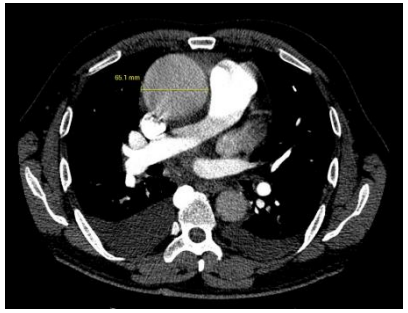

Figure 1: CTPE showing a TAA measuring 6.51 cm

Discussion:

TAA and its associated complications rank in the top 20 leading causes of mortality in the United States and the 15th leading cause of death in people over the age of 65. As Clouse et al. pointed out, the mortality rates of patients presenting with TAA has remained high even if they are treated before complications occur [2].

The incidence of TAA has been reported to be 6-13 cases per 100,000 per year, however mortality is extremely high, with rates of 57% without surgery and 17% to 25% with surgery [3]. Many acquired, congenital, and hereditary etiologies of TAA have been implicated but will not be reviewed in this case report.

Dilatation of the ascending aorta is a very indolent process as it takes many years to develop. Typically, patients are asymptomatic until either a catastrophic event occurs or it is incidentally found on cardiovascular imaging. TAA can be complicated by aortic dissection or rupture, cardiac tamponade, pericardial hemorrhage, and occlusion of aortic branches [4]. There are many imaging modalities for evaluation of TAA including CXR, Transthoracic Echocardiography (TTE), Computed Tomography (CT), and Magnetic Resonance Imaging (MRI). Typical CXR findings include widening of the mediastinum, however, CXRs have been found to be normal 15-20% of the time. TTE is a simple non-invasive technique that can be used to evaluate the aortic root, proximal ascending aorta, aortic valve, left ventricular morphology and function. TTE is the recommended imaging modality for screening of patients with suspected TAA and for follow-up [5]. CT angiography is the preferred imaging modality due to ease of accessibility and rapid results. It allows for visualization of aortic anatomy and its branching vessels with sensitivity and specificity reaching up to 100%. Cardiac MRI, allows for better assessment of aortic root anatomy, ventricular and aortic valve function, though it is less accessible and more time consuming then other modalities [6].

Clinical guidelines suggest that the size of the ascending aorta and root should be evaluated every 6 to 12 months. Imaging studies are recommended more frequently, every 3 to 6 months, when the aorta exceeds 4.5 cm or the growth rate is greater than 0.5 cm per year [7]. Elective surgical repair remains the mainstay for the management of symptomatic aneurysm or asymptomatic aneurysm of which the diameter is greater than 5.5 cm [8]. Secondary management of TAA includes lifestyle modification, serial imaging, and medical treatment with beta blockers, maintaining blood pressure under 130/80 mm Hg in those with diabetes or chronic renal disease and 140/90 mm Hg in those without [9].

Conclusions:

TAA is a lethal disease. Complications of TAA can be disastrous even if diagnosed promptly and properly managed. Here we describe a case of a 58-year-old male with scrotal edema as a result of heart failure from a large TAA. Early diagnosis of TAA and arrangement for proper follow-up, medical management, and surgical treatment on a non-emergent basis leads to improved clinical outcomes and can prevent vascular catastrophes for individual patients.

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