Unsuspected chronic traumatic aortic pseudoaneurysm – what to do about it. Late post-traumatic aortic pseudoaneurysm

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CB Marcu, R Nijveldt, AC Van Rossum. Unsuspected chronic traumatic aortic pseudoaneurysm – what to do about it. Late post-traumatic aortic pseudoaneurysm. *Can J Cardiol* 2008;24(2):143-144.

A 59-year-old man with multiple risk factors for coronary artery disease who had been in a motor vehicle accident 30 years earlier presented with new-onset angina pectoris. During cardiac catheterization, an ill-defined dense area was noted in the mediastinum. Chest radiography showed an area of calcification around the proximal descending aorta. Cardiovascular magnetic resonance imaging demonstrated a pseudoaneurysm of the proximal descending thoracic aorta. Due to the typical location (aortic isthmus), the pseudoaneurysm was thought to be the result of deceleration injury sustained by the patient in the previous motor vehicle accident. The present manuscript discusses the natural history and management options of an uncommon consequence of traumatic aortic injury: chronic post-traumatic aortic pseudoaneurysm.

Key Words: Cardiovascular magnetic resonance; Traumatic aortic pseudoaneurysm

A 59-year-old man presented to his primary care physician with complaints of precordial discomfort, which occurred one week earlier while running to catch a bus. The discomfort, described as a “heavy weight on the chest”, lasted for 5 min, was unrelated to position or respiration, subsided with rest and did not recur. His prior history included hypercholesterolemia, systemic hypertension, cigarette smoking (one pack per day for 35 years) and involvement in a serious motor vehicle accident 30 years previously.

An electrocardiogram performed at rest demonstrated no abnormalities. The diagnosis of new-onset effort angina pectoris was made; atenolol 50 mg daily was added to the patient’s previous medication regimen, which included acetylsalicylic acid 80 mg, hydrochlorothiazide 25 mg and atorvastatin 80 mg daily. The patient was referred for cardiac catheterization. Coronary angiography demonstrated 50% diameter stenosis of the mid-left anterior descending artery and 75% diameter stenosis of a small, heavily calcified first obtuse marginal branch. An ill-defined dense area was seen during fluoroscopy in the upper mediastinum. Chest radiography was performed and showed an area of calcification in the proximal descending aortic region (Figures 1A and 1B).

The patient was referred for cardiovascular magnetic resonance imaging (CMR) for better assessment of the aortic findings. CMR demonstrated an oval-shaped structure measuring 2.5 cm × 3 cm, situated on the medial aspect of the proximal descending thoracic aorta (aortic isthmus) and connected to the aortic lumen through a narrow ‘neck’ – a finding consistent with an aortic pseudoaneurysm (Figures 2A, 2B and 2C). The pseudoaneurysm was situated in the aortic isthmus, opposite from the insertion point of the ligamentum arteriosus, a typical location for deceleration aortic injury (1).

The patient underwent adenosine technetium-99m myocardial perfusion imaging to assess the coronary stenosis functional significance

Pseudo-anévrisme aortique post traumatique chronique insoupçonné : Mesures à prendre – pseudo-anévrisme post-traumatique tardif

Un homme de 59 ans manifestant plusieurs facteurs de risque coronariens a consulté pour ce qui s’est révélé être de l’angine *de novo*; cet homme avait été impliqué dans un accident de la route 30 ans auparavant. Durant le cathétérisme cardiaque, une zone dense et diffuse a été observée au niveau du médiastin. La radiographie pulmonaire a révélé une zone de calcification autour de l’aorte descendante proximale. L’imagerie par résonance magnétique cardiovasculaire a pour sa part révélé un pseudo-anévrisme de l’aorte thoracique descendante proximale. En raison de la localisation typique (isthme aortique), le pseudo-anévrisme a été jugé consécutif à une lésion de décélération subie par le patient lors de son accident de la route. Le présent article aborde l’histoire naturelle et les options thérapeutiques pour cette résultante rare d’une atteinte traumatique de l’aorte : le pseudo-anévrisme aortique post-traumatique chronique.

Figure 1) Chest radiography. **A** Posteroanterior projection – ring-shaped calcification (arrow) superimposed on the aortic arch area. **B** Lateral-projection aortic pseudoaneurysm and oval-shaped calcification (arrow) in the region of the proximal descending aorta. L Left

while on beta-adrenoreceptor blocker therapy. Only a small area of mild, reversible defective activity in the inferolateral region (perfused by the obtuse marginal arteries) was demonstrated. Because the patient had no further anginal symptoms, no evidence of ischemia in the left anterior descending artery territory, and because the lesion of the obtuse marginal was not suitable for percutaneous revascularization, isosorbide dinitrate (30 mg orally once daily) was added to his previous medical regimen. In consultation with the cardiothoracic surgery service, a decision was made to treat the otherwise asymptomatic aortic pseudoaneurysm medically by controlling the patient’s blood pressure using beta-blockers (target systolic blood pressure of lower than 120 mmHg) and follow up with a CMR examination of the aorta in six to 12 months.

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Received for publication February 20, 2006. Accepted April 10, 2006

Figure 2 Cardiovascular magnetic resonance imaging – true fast imaging and steady precession ('bright blood') cardiovascular magnetic resonance imaging in sagittal (A) and transverse (B) planes showing aortic pseudoaneurysm (arrow) situated on the medial aspect of the proximal descending aorta, with areas of calcification appearing as signal loss (dark). C Maximum-intensity projection gadolinium-contrast enhanced thoracic aortic angiogram indicating pseudoaneurysm (arrow) distal to the left subclavian artery (LSA) in the aortic isthmus region. AO Aorta; DAo Descending thoracic aorta

DISCUSSION

Traumatic aortic transections (TATs) are the result of penetrating trauma (gunshot or stab wounds) in 85% of cases and blunt trauma (motor vehicle accidents or falls) in 15% of cases (1). The anatomical classification of TATs includes six groups: intimal hemorrhage, intimal hemorrhage with aortic laceration, medial aortic laceration, complete aortic laceration, false aneurysm formation and periaortic hemorrhage (2,3). Rupture of the thoracic aorta causes death at the site of injury in 90% of cases. The 11% of patients with acute TATs and less serious associated injuries who survive for more than 6 h after trauma owe their lives to perivascular hematomas and the periaortic tissues, which maintain blood within the aorta (1,2). Although the aortic isthmus (between the left subclavian and the third intercostal artery) is the injury site reported in 54% to 65% of acute TAT cases in autopsy series, it is the region involved in 84% to 100% of patients arriving at the hospital alive (1,3). This discrepancy is most likely explained by the protection conferred by the periaortic isthmus tissues against free rupture, which allows for transfer to a hospital (1). The classic involvement of the isthmus in blunt TATs is not yet fully understood. Two popular theories include the 'whiplash' theory and the 'osseous pinch' theory (1,3). The 'whiplash' hypothesis explains involvement of the isthmus by differences in mobility between the aortic arch (a relatively mobile structure) and the proximal descending thoracic aorta, which is fixed by the ligamentum arteriosum, the left stem bronchus and the intercostal arteries. In the 'osseous pinch' theory, it is hypothesized that the anterior chest bones (manubrium, first rib and clavicles) rotate posteroinferiorly and impact the vertebral bodies, thus 'pinching' the aorta in between.

Of patients with TATs that were not operated on or even detected, approximately 2% survive long enough to develop chronic

pseudoaneurysm (1,2,4). More than 90% of these false aneurysms involve the aortic isthmus – again a reflection of the protective effect conferred by periaortic tissue in that area (1,2,4). Blood in the pseudoaneurysm thromboses, while the adventitia and surrounding tissues organize into a fibrous wall. Subsequently, most pseudoaneurysms undergo extensive calcification (1,4). The reported long-term survival is good in self-selected, asymptomatic patients who develop pseudoaneurysms after undiagnosed TATs (1,4). In a review of 413 cases of chronic traumatic aortic pseudoaneurysms, Finkelmeier et al (5) found that 85% of patients underwent surgical repair and 15% were followed up without surgical intervention. Patients who were not operated on had five-year, 10-year and 20-year survival rates of 71%, 66% and 62%, respectively. In the same group, the probability of being alive and free of symptoms or signs related to aneurysm expansion 20 years after the initial trauma was 33%. Patients who underwent surgery had five-year and 10-year survival rates of 93% and 85%, respectively.

The diagnosis of chronic traumatic aortic pseudoaneurysm requires a high index of suspicion. Probably the most important step is questioning the patient about a history of prior trauma. Plain chest radiography may demonstrate the calcified pseudoaneurysm, while computed tomography or magnetic resonance imaging allows for precise lesion measurement and localization.

Compared with conventional thoracic aneurysms (for which certain management criteria that take into account the lesion diameter have been established), there are no specific guidelines for the surgical treatment of asymptomatic, chronic traumatic pseudoaneurysms. A uniform and thick layer of calcium in the pseudoaneurysm wall is believed to confer a certain level of protection against rupture (4). Thus, asymptomatic patients with calcified traumatic pseudoaneurysms detected more than two years after the initial event may be managed medically using beta-adrenoreceptor blockers for control of blood pressure and to decrease aortic wall tension. The pseudoaneurysm should be followed radiographically at six- to 12-month intervals for evidence of change in size (CMR, which does not use ionizing radiation, is an excellent option). The presence of symptoms, such as pain or signs suggesting compression of surrounding organs (hoarseness, dysphagia), or an increase in aortic diameter of 1 cm or more during 12 months, are indications for prompt intervention (surgical resection and patch repair or endovascular aortic stenting, depending on the particular case and local expertise) (4).

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