Penetrating Aortic Ulcer of the Descending Aorta Thoracalis Concomitant with Coronary Artery Disease

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Abstract:

A male in his 50s with a history of hypertension, heavy smoking, and a strong family history of heart problems presented with different types of chest pain. He experienced tearing-like sensation chest pain recently, but for the past 8 years, he had complained of heavy chest pain that would subside with rest or sublingual ISDN. An electrocardiogram revealed sinus rhythm with QS pattern in V1-V2 and inverted T wave at III and AVF. Echocardiography revealed normal systolic function of the left ventricle, impaired diastolic function, and mid anteroseptal hypokinesis. A thorax CT scan confirmed the presence of a penetrating aortic ulcer (PAU) with a neck size of 20.7 mm and a depth of 16.0 mm after a chest x-ray raised suspicion of descending aorta abnormality. The patient underwent thoracic endovascular aortic repair (TEVAR) with the implantation of a single body stent graft. Additionally, diagnostic coronary angiography revealed three-vessel coronary artery disease.

Keywords: Penetrating Atherosclerotic Ulcer, Coronary Artery Disease, TEVAR

Backgrund:

Penetrating atherosclerotic ulcers (PAU) are characterized by atherosclerotic plaque ulceration that penetrates the aortic media. They account for 2.3-7.6% of acute aortic syndrome cases and are commonly associated with severe atherosclerotic disease. Hypertension and coronary artery disease (CAD) frequently coexist with PAU. Studies show that around 81.25% of hypertensive patients and 56.25% of CAD patients have PAU. Hypertension is identified as the primary risk factor in approximately 80.2% of PAU cases, while CAD patients with PAU account for 58.5%. [1]–[3]Patients with advanced atherosclerosis are more susceptible to developing penetrating aortic ulcers, and PAU can present with chest pain alongside CAD. PAU may manifest with a very common symptom (chest pain) and relatively concomitant with coronary artery disease. It is qualitatively severe and may often be separated from coronary ischemia by its sudden onset and peak intensity at the beginning.

Case Presentation

A male in his 50s was referred from rural hospital, he complained of different characteristic of chest pain, at that moment he felt tearing like sensation chest pain while doing mild to moderate activities with VAS 7-8/10 and relieved by rest around 10-15 minutes. Previously, since 8 years ago, he had complained of chest pain with heavy like sensation VAS 2-3/10 duration <10 minutes accompanied with cold sweating but the symptom was relieved by rest or ISDN sublingual. He routinely controlled to cardiologist and routinely consumed medication. He had history of hypertension, history of heavy smoker and strong family history. His physical examination showed no abnormality. 12-lead electrocardiography showed sinus rhythm with QS pattern in V1-V2 and Inverted T wave at III, AVF. Echocardiography showed systolic LV was normal (EF 55% biplane), Impaired diastolic LV, AR mild and hypokinetic mid anteroseptal segment. He underwent chest x-ray

examination and suspected aorta descendent abnormality. He was planned to do Thorax CT and obtained an image of the PAU with the largest neck size of 20.7 mm and a depth of 16.0 mm with a distance from proximal PAU to the left subclavian branch is 20mm. He has been done TEVAR with implantation 1 body stent graft in complicated penetrating atherosclerotic ulcer (PAU). At that time he was also performed DCA. From the DCA patient was diagnosed with CAD 3VD.

FIGURE/VIDEO CAPTIONS

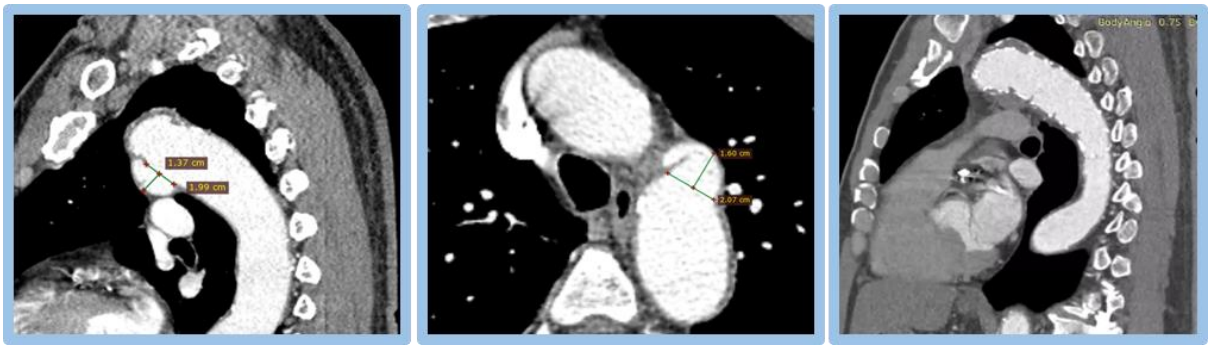

Figure 1. CT Angiography of this patient (a) Pre procedure, sagittal view, PAU showed, the **width was 19.9mm and the depth was 13.7mm (b) Pre procedure,** transaxial view, PAU showed, the **width was 20.7mm and the depth was 16.0mm (c) 3 month post TEVAR procedure,** sagittal view, graft still good without endoleak

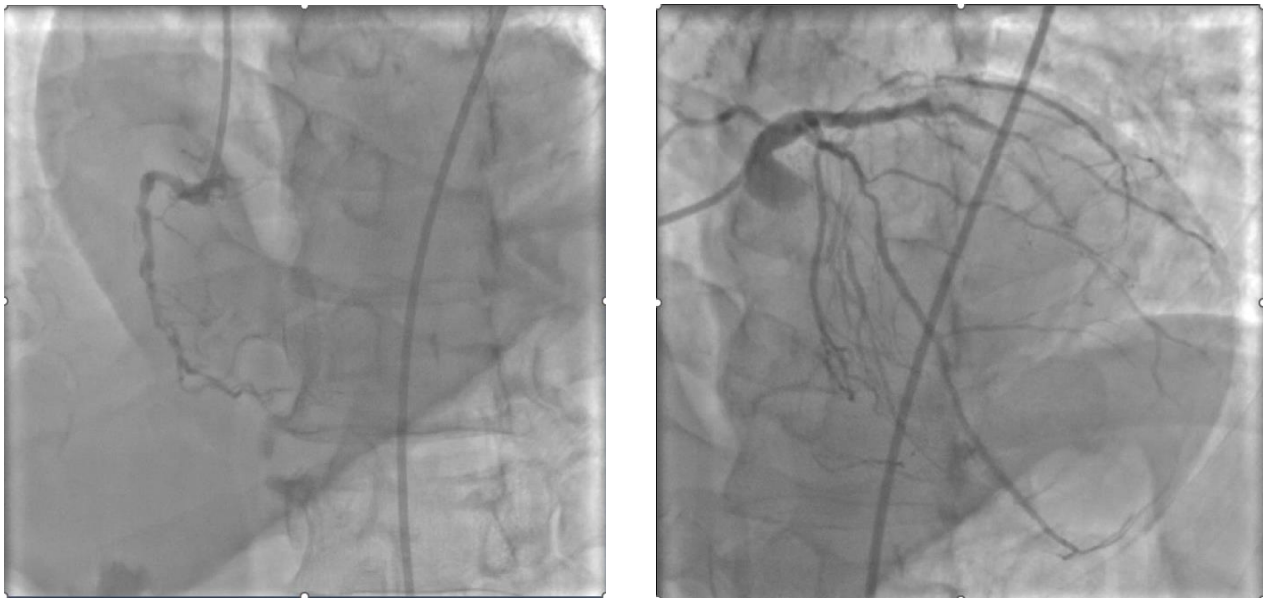

Figure 2. Diagnostic Coronary Angiography (a) LAO CRA view**,** Diffuse stenosis from prox-mid RCA. CTO after RV branch obtained collateral from bridging collateral from RV, septal branch, and distal LAD. Diffuse stenosis at RV branch **(b)** LAO CAU view**,** LAD artery : Diffuse stenosis from prox-distal LAD with maximal stenosis 90% at mid LAD. Diffuse stenosis at septal branch. Septal branch provides collateral to OM and RCA branch, LCx artery : High posisiton of OM1. CTO at distal LCx before OM2. OM2, OM3, and OM4 obtained collateral from branch septal and distal LAD

TREATMENT If relevant

Even though complicated type B dissections account for only 15% to 20% of cases, they have a 50% mortality rate. Patients with incidental PAU findings who are initially asymptomatic are monitored with further imaging. If pain at initial presentation improves with medicinal therapy and there is no radiological evidence of progression during short-term follow-up, individuals with involvement of the descending aorta (type B-PAU) may be handled conservatively with strict blood pressure management [4][5]. Medical therapy, such as pain management and blood pressure control, is advised in ESC guideline 2014 for all PAU patients [6].

In complicated Type B PAU, endovascular repair with TEVAR should be considered. It has been suggested asymptomatic PAU with a depth of more

than 10 mm and a diameter of more than 20 mm is more likely to progress and may be a candidate for early treatment [7]. This patient has the largest neck size of 20.7 mm and a depth of 16.0 mm with a distance from proximal PAU to the left subclavian branch is 20mm. These abnormalities, which are a component of the aortic dissection spectrum, may potentially raise the risk of an aortic rupture [8]. We underwent endovascular treatment by TEVAR for this patient, we have been TEVAR with implantation 1 body stent graft in complicated penetrating atherosclerotic ulcer (PAU) at descending aorta with good result. In comparison to surgical aortic reconstruction, endovascular treatment offers a better 30-day/in-hospital survival for acute difficult type B aortic dissection. A 30-day mortality rate for PAU patients who get TEVAR is 4.8%. When compared to medical therapy alone, TEVAR appears to have a better outcome for aortic remodeling and the aortic-specific survival rate [9]. TEVAR in addition to optimal medical treatment is associated with improved 5-year aorta-specific survival and TEVAR is associated with increased 5-year aorta-specific survival and postponed disease progression in addition to appropriate medical care. Preemptive TEVAR should be taken into consideration in stable type B dissections with adequate anatomy to improve late result [10][11]. Inspiring long-term outcomes were discovered by meta-analysis, with a survival rate extended to 10 years of 69.7% [12]. Since a PAU is a focal lesion, an adequate landing zone is almost present [13]. All commercially available thoracic stent grafts need a proximal seal zone that is 20 mm long. In order to prevent endoleak or device migration, it is essential to achieve adequate apposition across this segment. The distal landing zone must have a 20-mm seal zone with the currently available devices in order to prevent type Ib endoleak [14].

OUTCOME AND FOLLOW-UP

Following the procedure, the patient is currently asymptomatic, and able to engage in daily activities without any limitations. He maintains regular check-ups with his cardiologist and adheres to prescribed medications. Evaluations through CT scans were conducted at one month, three months, and six months post-procedure, revealing no signs of endoleaks or any other abnormalities.

Discussion ***Include a very brief review of similar published cases***

Atheromatous plaques that rupture and cause damage to the internal elastic lamina are the source of penetrating aortic ulcers [14]. It was an emergency condition that was uncommon but life-threatening. Inflamed atherosclerotic plaques that disrupt the normal structure of the aortic wall cause erosion of the internal elastic membrane, allowing luminal blood to leak past the media of the aorta and beyond. [15]. Aortic rupture or the development of a pseudoaneurysm can result from erosion into the medial layer, which can also cause an intramural hematoma or a dissection. It mostly affects the descending thoracic aorta and is infrequent in the infrarenal aorta [16](Quint et al, 2001). Up to 40% of patients who have PAU may develop (localized) intramural hematoma from the ulcer's arrosion of the aorta vasa vasorum, or they may develop (pseudo)aneurysms that lead to overt aortic dissection or rupture [14]. The middle and lower descending thoracic aorta are the most often affected areas by PAU (Type B PAU). While involvement of the ascending aorta is uncommon, PAUs are less frequently found in the aortic arch or abdominal aorta [6].

Patients with advanced atherosclerosis are more prone to developing penetrating aortic ulcers. [17]. PAU typically manifests in advanced age, as well as male gender, atherosclerotic disease, coronary artery disease, hypertension, hyperlipidemia, a history of heavy smoking, chronic obstructive pulmonary disease, concurrent abdominal aneurysm and renal disease [6], [17]. Our patient is a male with history of heavy smoking and hypertension for 7 years ago routinely controlled and consumed amlodipine and valsartan. We discovered diffuse stenosis from the proximal - distal LAD and the proximal - mid RCA, as well as CTO at the distal LCx and following the RV branch, from the patient's DCA. Hypertension and CAD most often coexist with PAU. Stanson et al. found that 81.25 percent of patients with hypertension have PAU and 56.25 percent of patients with coronary artery disease (CAD) have PAU. This is correlates with a study conducted by Charles et al, where hypertension is the main risk factor for patients with PAU, approximately 80.2%, while CAD patient which suffer from PAU is 58.5% [12][3][2].

Acute aortic dissection was stated to affect 30 per million people annually by some sources, while the incidence of acute dissection was 15 per 100,000 patient years [15]. PAU is the primary cause of 2.3–7.6% of acute aortic syndromes [18]. Only 4.6% of aortic dissections in an old postmortem study were found to be caused by penetrating aortic ulcers (Hirst 1958). De Carlo et al, showed 273 patients (0.46%) out of 58 800 patients who had CT angiography during the research period had an asymptomatic PAU. In contrast, just 94 individuals (0.16%) throughout that time period presented with a symptomatic PAU [2]

PAU may manifest with a very common symptom (chest pain) and relatively concomitant with coronary artery disease. It is qualitatively

severe and may often be separated from coronary ischemia by its sudden onset and peak intensity at the beginning. Midscapular or anterior chest discomfort is the most common initial symptom. Similar to aortic dissections, ascending aortic involvement is typical in patients with anterior chest pain, whereas descending aortic involvement is common in patients with back discomfort. Acute coronary syndrome, aortic aneurysm, aortic dissection, intramural hematoma, and pulmonary embolism are among the possible diagnoses with this common presentation [19]

Acute type B dissections can be categorized as uncomplicated and complicated. Complications of type B aortic dissection include malperfusion syndrome with ischemia of the kidney, viscera, or extremities, rupture or imminent rupture, uncontrolled hypertension, chronic discomfort in the chest or abdomen, or signs of fast enlargement on computed tomography (CT) imaging [9]. As we define our patient referred to complicated PAU because he has large single entry. DeBakey III or Stanford type B aortic dissection begins in the descending thoracic aorta and does not extend retrogradely into the ascending aorta [20][8]

LEARNING POINTS/TAKE HOME MESSAGES 3-5 *bullet points*

- PAU is one of the AAS with a high risk of complications and life threathening condition
- It was challenging to work up this diagnosis
- PAU may manifest with a chest pain that coexists with coronary artery disease
- Patients with PAU should undergo screening for other vascular diseases

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