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CASO CLÍNICO

PENETRATING AORTIC ULCER TYPE B AND MOBILE AORTIC THROMBUS

Autores

Gonzalo Pullas-Tapia, MD Ph. (1), Carla López-Pérez, MD (2), Jorge Jarrín-Menéndez, MD (1).

RESUMEN

El síndrome aórtico agudo (SAA) engloba tres patologías potencialmente mortales, con manifestaciones clínicas similares pero con diferente fisiopatología, la cual puede deberse a una lesión en la capa íntima de la pared de la arteria o a un rotura de la vasa vasorum que originará una hemorragia en la capa media de la pared de la arteria. Como consecuencia de estos procesos fisiopatológicos tendremos el desarrollo de tres patología las cuales son: hematoma intramural (HIM), ulcera aortica penetrante (UAP), o disección aortica aguda (DA). Este artículo presenta el caso de un paciente de 78 años de edad que presentó disección aórtica, úlceras penetrantes agudas tipo B y trombo aórtico móvil, que comenzó con tratamiento clínico; sin embargo, debido al alto riesgo de embolización de la placa ateromatosa, se realizó el manejo endovascular, con excelentes resultados, reduciendo la morbi-mortalidad de la paciente.

Palabras clave:
Acute Aortic Syndrome, Type B Dissection, Penetrating Aortic Ulcer, Mobile Aortic Thrombus, Endovascular Treatment.

ABSTRACT

Acute aortic syndrome (AAS) encompasses three life-threatening pathologies, with similar clinical manifestations but with different pathophysiology, which may be due to a lesion in the intima of the wall of the artery or a rupture of the vasa vasorum that will cause a hemorrhage in the middle layer of the artery wall. As a consequence of these physopathological processes we will have the development of three pathologies which are: intramural hematoma (IMH), penetrating aortic ulcer (UAP), or acute aortic dissection (AD). This article presents the case of a 78-year-old patient who presented aortic dissection, acute penetrating ulcers type B and mobile aortic thrombus, which began with clinical treatment; However, due to the high risk of embolization of the atheromatous plaque, endovascular management was performed, with excellent results, reducing the morbidity and mortality of the patient.

Keywords
Acute Aortic Syndrome, Type B Dissection, Penetrating Aortic Ulcer, Mobile Aortic Thrombus, Endovascular Treatment.

(1) Postgraduate Lecturer of Vascular and Endovascular Surgery at Pontificia Universidad Católica del Ecuador
(2) General physician

Autor Correspondencia: *Gonzalo Pullas Tapia*
Direccion: Calle Pedro Fermin Cevallos y del Amor
Teléfono: 593– 98443873
Correo electrónico: gpullas@yahoo.com

INTRODUCTION

The aorta is the largest artery in the human body. The ascending aorta has an intrapericardial segment and an extrapericardial segment, it has a straight trajectory of approximately 3 centimeters, located before the brachiocephalic artery. At this level, the first branches of the aorta emerge, the coronary arteries, which supply the heart. The aortic arch is the portion that continues to the ascending aorta and is located in the superior mediastinum, begins in the second right sternocostal joint and ends in the second left sternocostal joint. The most frequent intimate rupture is at the level of the ascending aorta, at 3-5 cm from the right sinus of Valsalva. The descending aorta begins in the isthmus, this zone is located between the origin of the left subclavian artery and the ductus arteriosus. At the thoracic level it descends in the left paraspinal region, its distal segment crosses the diaphragmatic aortic hiatus from this area is called abdominal aorta. [1]

The normal diameter of the descending aorta varies from 24 to 29 mm in men and 24 to 26 mm in women, at diaphragm its diameter is 24 to 27 mm in men and 23 to 24 mm in women. The aortic diameter varies according to age and body mass index. [1]

Histologically, the aortic wall is composed of three layers: the intima, media and adventitia. The intima is the inner layer formed by the endothelium and an internal elastic lamina. Its function is to prevent thrombosis and atherosclerosis. This function is reduced in front of certain factors such as: smoking, hypertension, hyperlipidemia, diabetes and direct traumatisms. The media consists of concentric layers of collagen, elastin and smooth muscle cells. The adventitia is the outer layer of the Aorta and is formed by collagen fibers, external elastic lamina and small vessels. [1,3,4]

Acute aortic syndrome (AAS) encompasses several life-threatening pathologies whose clinical manifestation is similar but with different pathological, demographic and survival differences. Controversy exists as to whether there are three different pathologies or a pathology with several spectra that evolve or coexist with each other, the pathologies that make up this syndrome are: aortic dissection (AD), intramural hematoma (IMH) and penetrating aortic ulcer (PAU). [3]

Statistically, the most common pathology is aortic dissection (62-88%), followed by intramural hematoma (10-30%) and finally penetrating aortic ulcer (2-8%). [2] There are no specific epidemiological data regarding mobile aortic thrombus and penetrating aortic ulcer, we have found a report of 130 cases of mobile aortic thrombus in relation to peripheral embolism. [6]

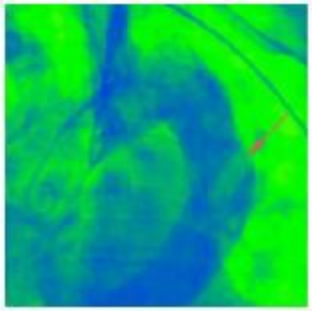
CASE REPORT

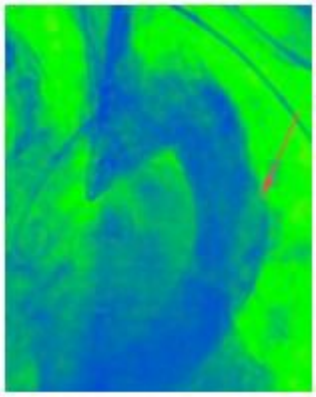
A 78-year-old female patient, mestiza, housewife, with a history of high blood pressure under treatment with Losartan, presented edema of the limbs. That was associated with dyspnea of small efforts in the last 7 days, physical examination, blood pressure: 176 / 92, oxygen saturation: 87% in ambient air, jugular engorgement, lower limbs with bilateral edema +++ / +++, erythema and local increase in temperature. She was admitted with a diagnosis of decompensated heart failure NYHA functional class III. Upon admission, tests were performed that reported: PROBNP: 2057.00, EKG: sinus rhythm, HR: 66, right bundle branch block, chest radiography: cardiomegaly grade III, without pleural effusion.

The following day, a venous ultrasound in the lower limbs was performed, ruling out deep venous thromboembolism. Echocardiogram: LVEF 66.2%. 1. Severe pulmonary hypertension with dilation of the right cavities and dysfunction of the right ventricle. Normal pulmonary capillary pressure. 2. Type IA diastolic dysfunction with left ventricular filling pressures normal at rest (according to age). 3. Mitral and aortic valvular apparatus with slight sclerosis and competent. 4. There are no signs of ischemia and / or resting infarction.

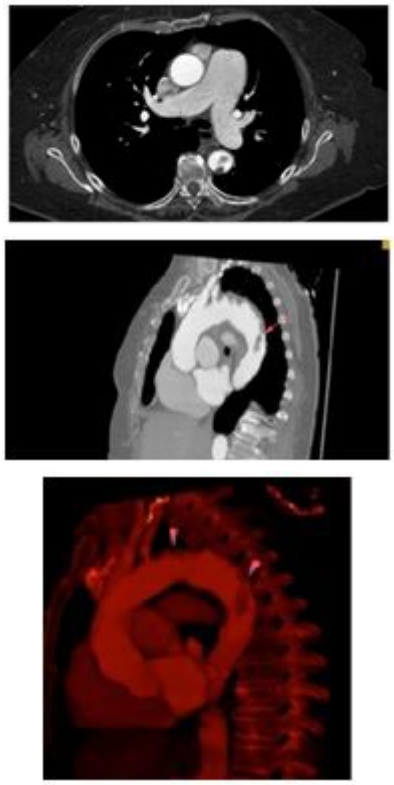
Seven days after admission, the pacient monitoring was performed: Transesophageal echocardiogram that reopened: tricuspid valve: dilation of the annulus and great dilation of right cavities. Presence of aneurysm of the interatrial septum that occupies the entire septum without event-proof derivation after echo with contrast and maneuvers. Descending aorta presence of type II atheroma plaques, cryptics with intracripta thrombi. There is a 22 x 11 mm dehiscent plate very mobile below the left subclavian artery, reminiscent of the aorta with signs of poor prognosis for penetrating ulceration or other acute aortic syndrome. Thinning due to vascular surgery, an angiotomography was performed in which multiple aortic ulcers were observed in the descending aorta and type B dissection flap.

Angiography images**: **dehiscent**, **mobile plate is





Angiotomography images:



The patient was treated for 15 days with clinical treatment based on acetylsalicylic acid, low molecular weight heparins at doses of isocoagulation and beta-blockers. In other Doppler echocardiography study that reported: tricuspid valve: normal, with mild functional regurgitation, before it was severe. Gradient right ventricle-right atrium: 62.3 mmHg, 88.6 mmHg above. Pulmonary valve: normal, mild regurgitation and pulmonary-artery gradient right ventricle 21 mmHg, above 25.5 m MHG. Pulmonary hemodynamics: pulmonary pressures severely increased at rest. Lung flow type I.

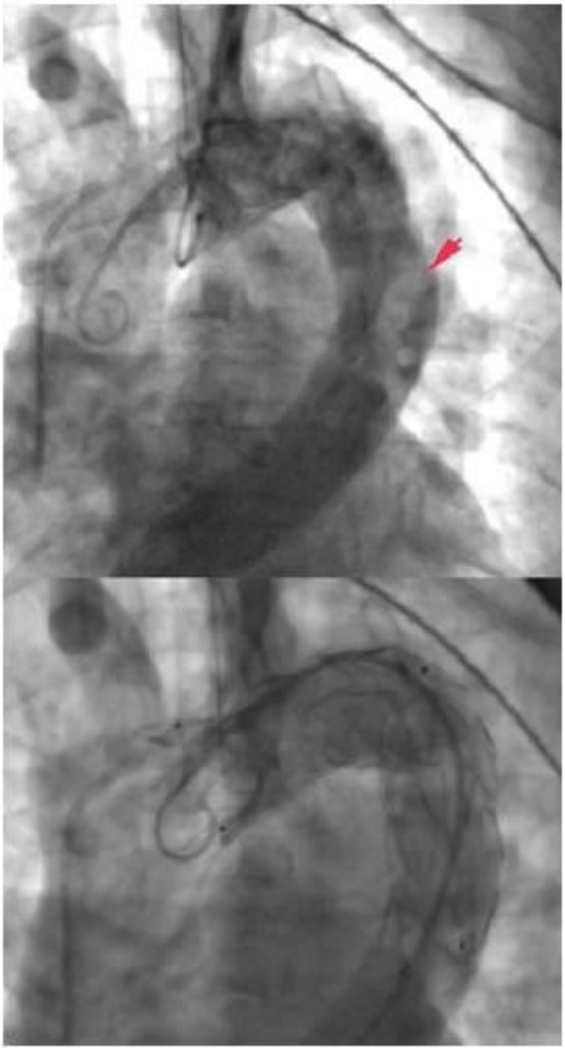
After of cardiac stabilization and with the aforementioned findings, a kind of endovascular surgical treatment was proposed due to the high risk of plaque rupture and embolization. Previous nephrological protection against the use of contrast, stent placement was performed on the posterior thoracic aorta at the emergence of the left subclavian artery to correct flap type B dissection and aortic ulcers. Surgical findings: 1. Aortic ulcers prior to artery emergence left subclavian over aortic arch, 2. Type B dissection flap over aortic arch, 3. Pedunculated thrombus in descending aorta, 4. Bovine thoracic aorta.

In the immediate postoperative period, the patient was admitted to Intensive Care Unit for hemodynamic control.

Presented hypotension refractory to fluid resuscitation and tachycardia. For which, the patient required treatment with sympathomimetics and catecholamines for 5 days until stabilizing hemodynamic function.

The patient also remained on mechanical ventilation for 4 days due to the presence of respiratory acidosis, receives antibiotic treatment with piperacillin tazobactam + amikacin for possible infection of pulmonary focus, with good response to it. The patient had stayed for 27 days, with favorable evolution. At the time of high haemodynamic, ventilation and metabolic confection. Up to the time of this report, the patient is asymptomatic vascular, with cardiology treatment for heart failure, arterial hypertension, severe pulmonary hypertension and cor pulmonale.

Angiography images: Pre endovascular treatment and Post endovascular treatment.



DISCUSSION

Acute Aortic Syndrome has three same components that share similar anatomical, physiopathological and clinical characteristics that can coexist together. The three pathological processes that are included in Acute Aortic Syndrome anatomically affect the middle layer of the aortic wall, originate through a lesion in the intimate layer or a spontaneous bleeding in the middle layer that will divide the intima layer of the aortic wall. Media causing a formation of a true lumen and a false lumen. Clinically, the three pathological processes involved share clinical risks for their presentation and progression of these. The most important is hypertension. At the beginning these pathologies can be asymptomatic and only present manifestations when they are complicated. The symptomatology characteristic of these is intense pain, Heartbreaking, sudden onset located in anterior thoracic region. The Stanford classification has been generalized for the three pathologies of acute aortic syndrome, which divides pathologies in type A to those affecting the ascending aorta and type B those that exclude the aorta ascending. In this way, the management have also been established, depending on the classification in which they are pigeonholed, the recommendation for those classified as type A is endovascular or open surgical treatment. Depending on the clinical and anatomical characteristics of each case. For type B the treatment of election is the conservative based on medical treatment and adequate control of blood pressure mainly however, it should be mentioned that these are recommendations, each patient should be assessed individually and take the therapeutic decision that best suits each case. In the case that we present, our patient presented type B acute aortic dissection associated with penetrating aortic ulcers. Although the patient did not present clinical criteria of complicated aortic dissection or complicated aortic ulcer, surgical management was decided. Although the recommendations indicate that the treatment of choice in these cases is the doctor. However, it should be mentioned that the patient in our case had a mobile aortic thrombus, pedicled, with high risk of fragmentation and embolization.

It is also important to consider the statistical study reported by Fattori R. et al. Survival After Endovascular Therapy in Patients With Type B Aortic Dissection. A Report from the International Registry of Acute Aortic Dissection (IRAD) in which they compare the efficacy of endovascular surgical treatment versus medical treatment alone. Although the report at two years of follow-up indicates that medical treatment is superior to surgical treatment in terms of morbidity and mortality. At 5 years of follow-up, these statistics change completely revealing that endovascular surgical treatment in patients with Type B dissection does not complicated, presents lower morbi-mortality versus medical treatment alone, whose patients usually present clinical complications and require subsequent surgical treatment. [5]

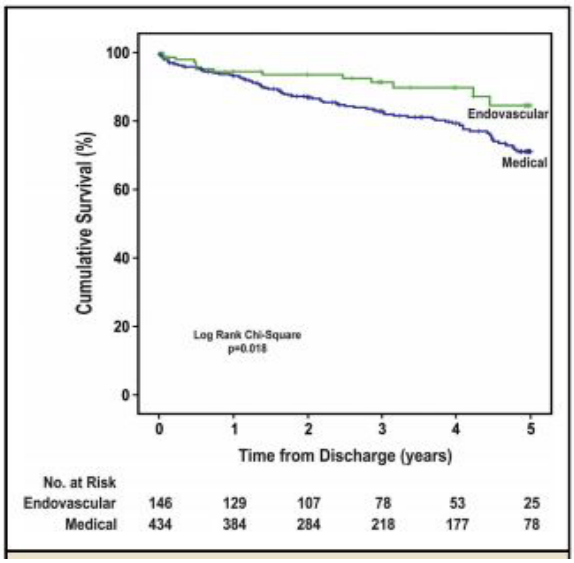


Figure 4 Long-term cumulative survival of patients with uncomplicated TBAD is improved after endovascular intervention compared with medical treatment alone. (From Fattori R, Montgomery D, Lovato L, et al. Survival after endovascular therapy in patients with type B aortic dissection: a report from the International Registry of Acute Aortic Dissection (IRAD). JACC Cardiovasc Interv 2013;6(8):879).

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