

Case Report

Percutaneous coronary intervention in critical occlusion of the left main coronary artery after acute aortic dissection.**María Redondo-Orts¹, Manuel García-Delgado², Inés Navarrete-Sánchez³****Intensive Care Unit, Hospital Universitario Virgen de las Nieves, Avenida de las Fuerzas Armadas, 2. 18014 Granada, Spain. Phone: +34 958 020125 ; Fax: +34 958 020174****EMAILS:**María Redondo-Orts: maria.redondo.orts@gmail.com;Manuel Garcia-Delgado: mjgardel@hotmail.com;Inés Navarrete-Sánchez : galdurias@hotmail.com;**Received date:** 19-01-2016; **Accepted date:** 08-02-2016; **Published date:** 13-02-2016**CORRESPONDENCE AUTHOR:** Dr. María Redondo-Orts**ADDRESS:** Intensive Care Unit, Hospital Universitario Virgen de las Nieves. Avenida de las Fuerzas Armadas, 2. 18014 Granada, Spain. Phone: +34 679 397 222 ;**CONFLICTS OF INTEREST**

There are no conflicts of interest for any of the authors.

ABSTRACT:

We report the case of a 45-yr-old-male referred to our hospital with diagnosis of type A aortic dissection. During emergency surgery, the ascending aorta was replaced with a supracoronary tube, and a Y-shaped graft bypass was performed from aortic tube to brachiocephalic artery and left carotid artery due to the involvement of both carotids. Signs of myocardial ischemia appeared on the third week after surgery, with electrocardiographic changes, cardiac marker elevation and severe ventricular dysfunction. Coronary angiography showed critical stenosis of the left main coronary artery, due to extrinsic compression, and extensive dissection of the left coronary tree. The patient underwent percutaneous coronary intervention in which four intracoronary stents were placed with a satisfactory outcome.

KEY WORDS: Aortic rupture; drug-eluting stent; postoperative complication**INTRODUCTION**

Proximal extension of acute aortic dissection (AAD) involving the origin of coronary arteries produces a myocardial perfusion deficit and myocardial infarction and is a severe and frequently lethal complication of AAD¹. In most cases of myocardial ischemia from AAD, ischemia signs are usually observed before surgery and most frequently affect the right coronary tree². We report the case of an AAD in which coronary involvement appeared as a proximal flap progression in week 3 after aortic surgery, affecting the left coronary tree. We selected a percutaneous interventionist approach rather than conventional surgical management.

CASE REPORT

A 45-yr-old male with a history of colonic polyposis and ulcerous colitis under treatment with mesalazine attended the Emergency Department of another hospital with chest pain. Complementary

test results yielded a diagnosis of type B acute aortic dissection (AAD), and the patient was scheduled for endovascular treatment. After progression to retrograde type A AAD during this procedure, the patient was transferred to our center for emergency surgery.

Transesophageal echocardiography and computed tomography studies on admission confirmed the diagnosis of type A AAD with extension from aortic valvular plane to iliac arteries and severe involvement of supra-aortic and abdominal arteries. Emergency surgery was performed, replacing the ascending aorta with a supracoronary aortic tube after verifying the competence of the aortic valve. Because of the proximal dissection of the brachiocephalic artery and left carotid artery, it was also necessary to perform a Y-shaped graft bypass from the aortic tube to these vessels.

Post-surgical complications during the first week of intensive care unit (ICU) stay included: perioperative extensive acute right ischemic stroke, respiratory sepsis, and venous thrombosis of internal and common jugular veins and of both subclavian veins. The clinical course of the patient was favorable, with a tendency towards stabilization and control of the above complications. However, in week 3 of ICU stay, the electrocardiogram showed anterolateral changes and there was an increase in cardiac enzymes, with a peak troponin I level of 10 ng/ml (upper normal limit of 0.5 ng/ml). Transesophageal echocardiography revealed: segmental contractility alterations in anterior, lateral, and inferolateral segments; significant left ventricular dysfunction (estimated left ventricular ejection fraction, 30%); and progression of the dissection to the non-coronary sinus and left coronary sinus; the latter was distal to the ostium of the left main coronary artery (LMCA), which showed partial thrombosis and ostial involvement, indicating its possible dissection or thrombosis (figure 1).

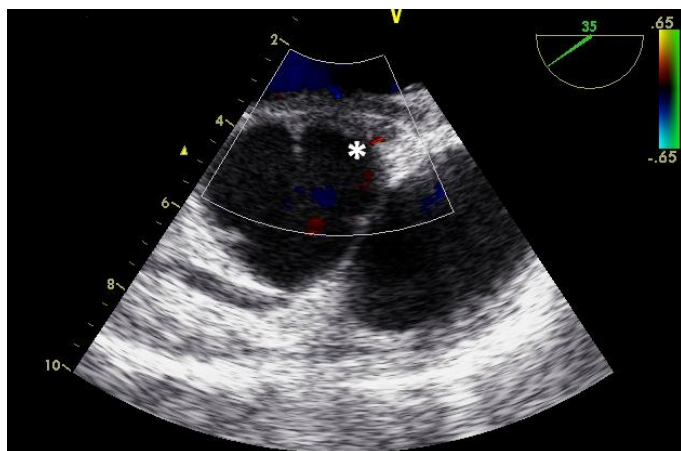


Figure 1: Transesophageal echocardiography image; cross-sectional slice very close to the aortic valve showing thickening of the aortic wall with decreased inner diameter and blood flow (*) of the left main artery, corresponding to aortic dissection with false lumen thrombosis.

This case of aortic dissection progression to the LMCA with myocardial ischemia and severe ventricular dysfunction was considered for surgery. However, given the high surgical risk due to the recent extensive acute stroke and the graft from the aortic tube to both carotids, a percutaneous coronary intervention (PCI) was ordered. Intra-procedure findings included critical stenosis of the LMCA ostium due to extrinsic compression (flap with thrombosis of the false lumen) and dissection of the anterior descending artery (DA) and circumflex artery down to medial portions (figure 2a). Four

pharmacocactive stents were placed during the PCI: two overlapping stents in the DA, one in the circumflex artery, and the fourth at the level of the LMCA and DA, with a satisfactory outcome (figure 2b). On day 6 post-PCI, a new catheterization showed that the coronary tree and stents had an excellent appearance, and the echocardiography study evidenced an improvement in the ventricular dysfunction. After 7 weeks of ICU stay, the patient was discharged to a normal hospital room in a good general state.

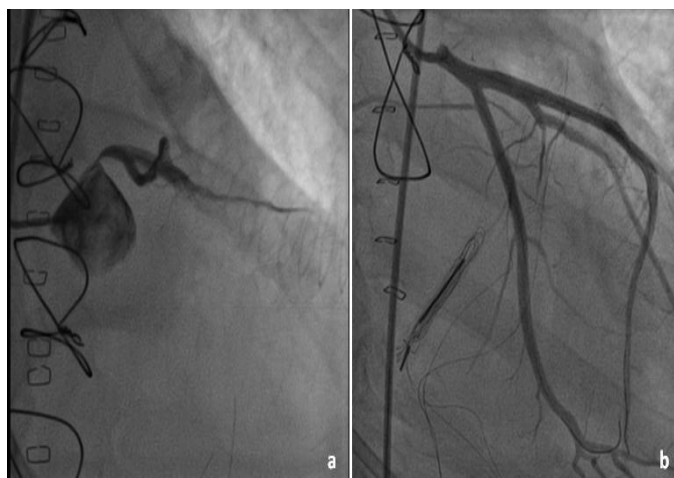


Figure 2: (a) Coronary angiogram evidencing subtotal stenosis of the left main coronary artery at proximal level. The flow of the anterior descending artery and circumflex artery is reduced due to the dissection in the two vessels; (b) Coronary angiogram after stent placement, showing the good flow of the coronary arteries and the post-procedure appearance of the left main artery.

DISCUSSION

AAD is an extremely severe disease with an annual incidence of 5-30 cases/million inhabitants³. It is caused by the formation of a false lumen in the medial layer of the aortic wall. Dissections are classified as a function of their retrograde or antegrade extension and the presence and localization of primary tears. AAD types A and B were defined by the Stanford group according to the involvement or non-involvement, respectively, of the ascending aorta, a critical issue for the therapeutic management of the patients. Known predisposing factors for AAD include advanced age, systemic arterial hypertension, congenital anomalies of the aortic valve, hereditary disorders of the connective system, and traumas, among others. AAD can also have an iatrogenic origin, with a reported incidence of 1.33-6.8% during endovascular treatment for type B AAD⁴.

Despite major diagnostic and therapeutic advances, the mortality rate for type A AAD remains elevated, with the most recent data for Spain (Spanish Acute Aortic Syndrome Register, 2009) evidencing a hospital mortality rate of 41.1% (33.4% of surgically treated patients and 71.2% of medically treated patients)⁵.

We highlight the delayed onset of myocardial ischemia (week 3 post-surgery) in the present patient, possibly attributable to proximal progression of the dissection or a residual dissection. Myocardial ischemia attributable to coronary artery involvement is observed in 5.7 - 11.3% of patients with type A AAD at the time of diagnosis, due to compression of the coronary ostium by a hematoma or its occlusion by an intimal flap⁶. The localization in our patient is also unusual, with few reports of AAD-related LMCA involvement in the literature, most frequently in patients who develop heart failure from left ventricular dysfunction or who do not survive^{7,8}. Spittell studied 236 AAD cases and found the dissection to be more frequent in the right versus left coronary artery, with a consequently greater involvement of the inferior myocardial wall⁹. The false lumen usually develops on the right anterior side of the ascending aorta, explaining the higher incidence of dissection in the right coronary tree.

Another noteworthy feature of the present case is the application of PCI to treat coronary artery dissection. Revascularization surgery has conventionally been applied for the challenging management of patients with AAD and coronary artery involvement. Neri et al² classified coronary ostial lesions into three types as a function of the extent of the proximal dissection: type A, ostial dissection; type B, dissection with a coronary false channel; and type C, circumferential detachment with an inner cylinder intussusception. The authors recommended revascularization surgery for the repair of all three types of lesion. However, this surgery is not always feasible, and the present results confirm that PCI is a viable alternative to conventional surgical treatment. Some data have been published on the utilization of PCI, angioplasty, and stent implantation to facilitate early coronary revascularization and reduce myocardial damage in cases of AAD with coronary dissection⁶. However, the evidence is limited to case reports, and the effectiveness of this approach has not been verified in a clinical trial. Moreover, PCI was usually performed before surgery in the previous studies, and very little information is available on the use of PCI after aortic repair surgery¹⁰.

CONCLUSIONS

A delayed myocardial ischemia in patients with type A AAD, as in the present patient, is uncommon but should be taken into consideration because of its severity and the need for rapid action. The present results indicate that PCI is a viable therapeutic option for this complication.

REFERENCES:

1. Kawahito K, Adachi H, Murata S, Yamaguchi A, Ino T. Coronary malperfusion due to type A aortic dissection: mechanism and surgical management. *Ann Thorac Surg* 2003; 76: 1471-1476.
<http://www.ncbi.nlm.nih.gov/pubmed/14602269>
2. Neri E, Toscano T, Papalia U, Frati G, Massetti M, Capannini G, et al. Proximal aortic dissection with coronary malperfusion: presentation, management, and outcome. *J Thorac Cardiovasc Surg* 2001; 121: 552-560.
<http://www.ncbi.nlm.nih.gov/pubmed/11241091>
3. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA* 2000; 283: 897-903.
jama.jamanetwork.com/article.aspx?articleid=192401
4. Lu S, Lai H, Wang C, Sun X, Hong T, Song K, et al. Surgical treatment for retrograde type A aortic dissection after endovascular stent graft placement for type B dissection. *Interact Cardiovasc Thorac Surg* 2012; 14: 538-542.
<http://www.ncbi.nlm.nih.gov/pubmed/20081764>
5. Evangelista A, Padilla F, Lopez-Ayerbe J, Calvo F, López-Pérez JM, Sánchez V, et al. Spanish Acute Aortic Syndrome Study (RESA). Better diagnosis is not reflected in reduced mortality. *Rev Esp Cardiol* 2009; 62: 255-262.
<http://www.ncbi.nlm.nih.gov/pubmed/19268069>
6. Imoto K, Uchida K, Karube N, Yasutsune T, Cho T, Kimura K, et al. Risk analysis and improvement of strategies in patients who have acute type A aortic dissection with coronary artery dissection. *Eur J Cardiothorac Surg* 2013; 44: 419-424.
<http://www.ncbi.nlm.nih.gov/pubmed/23504116>
7. DeSanctis RW, Doroghazi RM, Austen WG, Buckley MJ. Aortic dissection. *N Engl J Med* 1987 ; 317: 1060-1067.
8. Barabas M, Gosselin G, Crepeau J, Pefitlerc R, Cartier R, Thérroux P. Left main stenting-as a bridge to surgery-for acute type A aortic dissection and anterior myocardial infarction. *Cathet Cardiovasc Intervent* 2000; 51: 74-77.
9. Spittell PC, Spittell JA Jr, Joyce JW, Tajik AJ, Edwards WD, Schaff HV, et al. Clinical features and differential diagnosis of aortic dissection: experience with 236 cases (1980 through 1990). *Mayo Clin Proc* 1993; 68: 642-651.
10. Funatsu T, Fukuda H, Takeuchi M, Masai M, Kawano S, Abe K. Progression of left coronary artery dissection during and after aortic replacement in acute type A aortic dissection: a case report. *Ann Thorac Cardiovasc Surg* 2007; 13: 209-212.