



Case Report

Anesthetic Management in Complex Arch Surgery: Debranching of Innominate and Left Common Carotid Arteries in Extensive Aortic Dissection without Cardiopulmonary Bypass

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Abstract

Aortic dissection begins with the formation of a tear in the aortic intima, and it directly exposes an underlying diseased medial layer to the driving force of the intraluminal blood. This blood penetrates the diseased medial layer and cleaves the media longitudinally, thereby dissecting the aortic wall. Herein, we report the case of a 38-year-old woman, who presented with chest pain and dyspnea. After physical examination, laboratory evaluation, echocardiography, and CT-angiography, extensive aortic dissection was diagnosed involving the innominate and left common carotid arteries. Accordingly, the debranching of the aortic arch arteries was performed. During the procedure, the patient was monitored with bilateral regional cerebral tissue oximetry. The patient did not show any signs of complications either in the postoperative period or at postoperative three-month weekly follow-up or at subsequent monthly follow-up for the past year.

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Introduction

Acute aortic dissection is defined as the rapid development of a false, blood-filled channel within the tunica media of the aorta. It has an estimated incidence of 3 per 100000 persons per year.

Three acute aortic dissections are ultimately diagnosed out of every 1000 emergency department patients presenting

with acute back, chest, or abdominal pain. Mortality in untreated acute aortic dissection is estimated at more than 1% per hour after the onset of symptoms, whereas 30-day survival for appropriately treated patients is greater than 80%. Therefore, the timely diagnosis and rapid management of acute aortic dissection are of paramount importance to the emergency physician.¹ Diagnosis is delayed more than 24 hours after the initial presentation in almost half of all cases,

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highlighting the need for emergency physicians to maintain appropriate clinical suspicion for acute aortic dissection in patients with chest, back, or abdominal pain.

Failure to diagnose acute aortic dissection carries a significant risk for poor outcomes because of the consequences of progressive disease (e.g., aortic rupture) and the possibility of treating a falsely diagnosed myocardial infarction (MI) or pulmonary embolism (PE) with anticoagulation, a potentially catastrophic error.

Surgery on the aortic arch requires cardiopulmonary bypass, profound hypothermia, and a period of circulatory arrest. Focal and diffuse neurologic deficits are the major complications associated with the resection of the aortic arch, occurring in 3% to 18% of patients.

Case Report

A 38-year-old woman was admitted to the emergency room with chest pain and dyspnea. The patient's past surgical history was the Bentall operation, performed 7 years earlier, and the stenting of the descending aorta 6 months previously due to aortic dissection. The patient's medical history was significant for hyperthyroidism and asthma, and her physical examination revealed blood pressure of 160/90 mmHg, heart rate of 90 beats per minute, metallic sound on heart auscultation, reduced breathing sounds in the left lung on lung auscultation, soft abdomen without tenderness, and symmetric and present peripheral pulses in the extremities. Additionally, the electrocardiogram (ECG) showed normal sinus rhythm and 1-mm ST depression in leads aVL, V5, and V6, and chest X-ray demonstrated a white left lung. (Figure 1)

Echocardiography demonstrated normal left ventricular chamber size, no left ventricular hypertrophy, moderate reduction in systolic function, global wall motion abnormality, ejection fraction of 35%, smoky pattern in the left ventricle, normal left atrium, normal right ventricle size and no right ventricular hypertrophy, normal mitral valve and no mitral stenosis or mitral regurgitation, normal hemodynamic of the aortic valve prosthesis (aortic valve replacement was performed 7 years earlier), normal prosthetic ascending aorta, aneurismal descending aorta, normal flow pattern of the descending thoracic aorta, and a large aneurysm (8 × 5 cm) filled with clot at the proximal portion of the descending aorta with reduced blood flow in the true lumen and deviation of the flow in the abdominal aorta - suggestive of aortic dissection. Echocardiography also revealed normal pulmonary valve with no pulmonary stenosis or pulmonary insufficiency, normal tricuspid valve with trivial tricuspid regurgitation but no tricuspid stenosis, normal drainage of the pulmonary vein, greater systolic pressure than diastolic pressure, no ventricular septal defect, no atrial septal defect, normal inferior vena cava with 50% respiratory variation,

and no pericardial effusion.

CT angiography of the aorta with contrasted media revealed remarkable dilatation of the descending thoracic aorta (100-mm total transverse diameter) due to aortic aneurysm without active dye flow - containing thrombosis, a stent containing flow inserted in the aorta (Figure 2), normal aorta at the root and the ascending portion, mild pleural reaction in the left pleural space, evidence of dissection with flow in the true and false lumens of the abdominal aorta, flow in the renal arteries, enlarged left ventricular chamber, and normal main pulmonary artery and pulmonary artery branches.

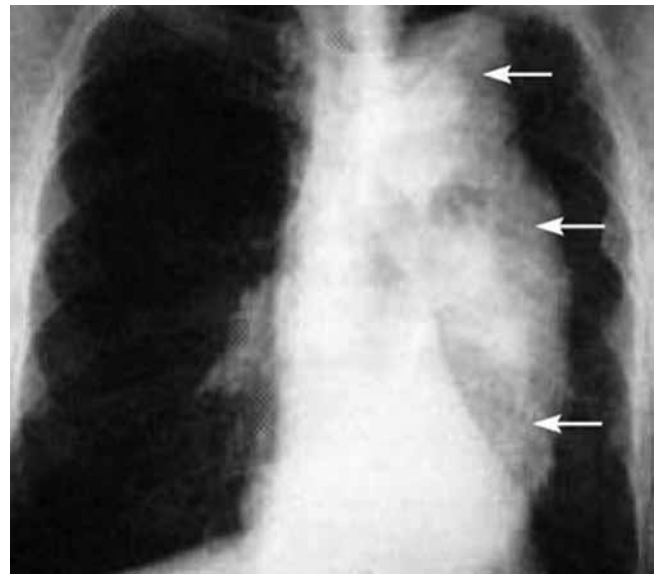


Figure 1. Chest x-ray showing extreme dissection of the aorta (arrows)



Figure 2. Coronal reformatting image, demonstrating the dissection of the aortic arch, involving the innominate and left common carotid arteries. (Complete arrows show false lumens and head arrows show true lumens)



Aortography showed a significant endoleak (type I) from the proximal part of the graft into the false lumen.

The patient had sinus tachycardia (140-150/min), which was treated with Metoral (100 mg/ TDS). The endocrinology consultant recommended Methimazole (5 mg/day). Pulmonary function test revealed a moderate restrictive lung pattern.

In the operating room, the patient was reevaluated carefully under ECG and pulse oxygen saturation monitoring. Two arterial lines were inserted in the right radial and right femoral arteries to monitor the blood pressure above and below the aortic arch. The central venous pressure line was inserted via the left femoral vein. The involvement of the aortic and carotid arteries, given the fact that the patient had dissection and aneurysm as well as tachycardia and high blood pressure, rendered perianesthetic cerebral oxygenation extremely crucial. It is absolutely vital that all the above-mentioned factors be meticulously controlled in order to prevent the aggravation of the patient's conditions. Overall, our anesthetic plan - including techniques, drugs, and monitoring for this complicated case - was individualized to enhance the outcome of the procedure. Indeed, a rationale existed for choosing the right radial and right femoral artery for invasive pressure monitoring. Furthermore, right radial arterial pressure monitoring was important because the procedure required clamping of the left subclavian artery and femoral arterial pressure monitoring allowed us the assessment of distal aortic perfusion.

Anesthesia was induced by using Sufentanil in incremental and titrated doses up to 20 cc, Propofol in incremental and titrated doses up to 20 mg, and Cisatracurium (0.2 mg/kg). Additionally, neuromuscular block was monitored with a nerve stimulator with train of forth (T.O.F) mode to ensure adequate neuromuscular block. While in deep anesthetic state and deep neuromuscular block, the patient was intubated without any bucking and hemodynamic changes. For hemodynamic management during instrumentation, she was treated with Trinitroglycerin (TNG), Esmolol, and Sodium Nitroprusside. Anesthesia maintenance was achieved using the balanced technique, utilizing Propofol infusion (75-100 mcg/kg/min), narcotic (Sufentanil [1-2 mcg/kg/h]), and Cisatracurium (2 mcg/kg/min). Hemodynamic management was accomplished by maintaining the central venous pressure between 12 and 16 cmH₂O in tandem with the infusion of TNG (10-50 mcg/min) and Sodium Nitroprusside at an infusion rate of 1.5-5 mcg/min. Bolus doses of Esmolol (0.5-1 mg/kg) were delivered via the central venous pressure line to control unexpected tachycardia.

In addition to invasive blood pressure, central venous pressure, temperature, heart rate, ECG, and end tidal CO₂ monitoring, the patient was monitored during the entire time of anesthesia and procedure with bilateral regional cerebral tissue oximetry (rSO₂) with the INVOS[®] in which regional saturation system measures changes in oxygen saturation

levels beneath the sensor, allowing doctors and nurses to measure site-specific oxygen saturation levels. Cerebral oximetry (Somanetics Corp., Troy MI) ensures adequate bilateral hemispherical oxygenation. During anesthesia and procedure, especially during carotid clamping, rSO₂ was above 60%, which is higher than the acceptable lower limit of rSO₂ (55%)^{2,3} (67% in the right side and 66% in the left side).

Right clamp time was 25 minutes and left common carotid clamp time was 20 minutes. Basal activated clotting time (ACT), measured for coagulation monitoring, was 100 seconds. Heparin (100 mg) was administered, and second ACT was measured to be 345 seconds. Arterial blood gas was monitored every 30 minutes and was at an acceptable level.

After mid-sternotomy, the innominate and left common carotid arteries were found dissected; accordingly, proximal and distal control was done. First, a 16 gel-sealed tube was anastomosed to the side of the ascending graft and then an end-to-side anastomosis to the innominate artery was performed. For the left common carotid artery, a 12 gel-sealed tube was anastomosed end to side to the ascending aorta graft and thereafter end to end to the left common carotid artery without cardiopulmonary bypass (Figure 3). The patient was monitored in the Intensive Care Unit and at home. She had only a moderate rise in creatinine (up to 3.8-4 mg/dl), which later returned to the normal value of 1.6-1.9 mg/dl during hospitalization. She was discharged without any other complications. She was followed up for 4 weeks and then monthly for one year, during which time no problems were detected.

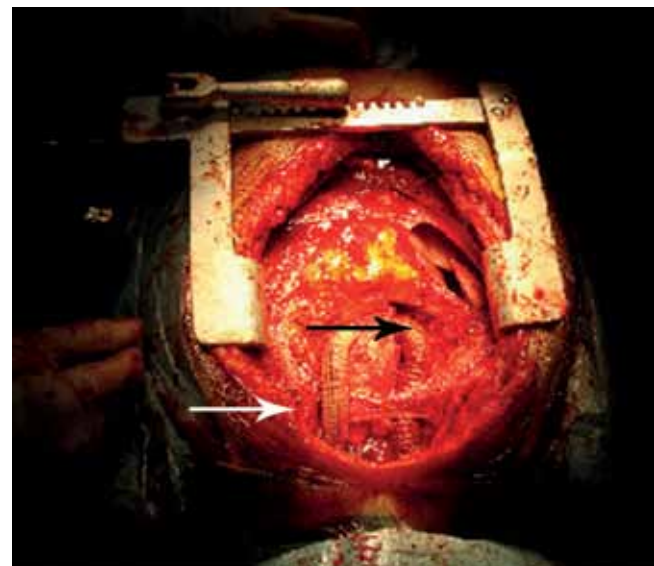


Figure 3. End-to-side anastomosis of the innominate (black arrow) and left common carotid arteries (white arrow) to the ascending aorta

Discussion

In aortic arch dissection, monitoring of vulnerable organs and careful management are crucial. In the case of the involvement of arteries which supply the brain blood flow, monitoring of end-organ oxygenation is extremely important. The theory underlying noninvasive INVOS® cerebral oximetry is conceptually simple. Near-infrared photons are injected into the skin over the forehead. After being scattered about inside the scalp, skull, and brain, some fractions of the injected photons survive to return and exit the skin (reflectance). By measuring the quantity of the returning photons and their wavelength, one can infer the spectral absorption of the underlying tissue and make some conclusion about its average oxygenation. To reduce the interference of extra-cerebral oxygenation, INVOS® utilizes two source detectors in spacing: a “near” and a “far” source detector. Theoretically, subtraction of some of the near signals from the far signals should leave a signal origination, predominantly in the brain cortex. The potential for neurological injury is the Achilles heel of aortic and heart surgery, but the technological advancement and innovations in anesthetic techniques have allowed us to offer surgical treatment to patients at the highest risk for neurological injury.⁴ Near-infrared spectroscopy devices demonstrate a significant correlation with cerebral venous oxygen saturation monitoring (SjO₂) and mixed venous oxygen saturation.⁵

Yamamoto-k et al.⁶ reported that near-infrared spectroscopy is suitable for monitoring immediate change in oxygenation during carotid surgery. Monitoring the cerebral rSO₂ in coronary artery bypass graft surgery patients prevents profound cerebral desaturation and it is associated with significantly lower incidence rates of major organ dysfunction.⁷

As was mentioned in the previous paragraphs, in the case of the dissection of the aortic arch and its branches, we can handle the aortic arch branches without cardiopulmonary bypass but by using rSO₂, which prevents cerebral ischemia. In this complicated case study, patient had previously undergone aortic valve replacement and had a large number of coexisting medical conditions such as replacement of the ascending aorta and intra-arterial proximal descending aortic stenting. Therefore, debranching of the aortic arch branches was performed via meticulous control of hemodynamic and brain oxygenation as well as rSO₂ instead of cardiopulmonary bypass or deep hypothermic circulatory arrest. To our knowledge, this technique has not been previously reported in literature and may extend to a larger subset of patients who might otherwise have been excluded.

In addition, regional oxygen saturation correlates well with the mean flow velocity during carotid clamping. It appears that cerebral oximetry is a satisfactory and possibly superior

device for monitoring the adequacy of cerebral perfusion and oxygenation during carotid endarterectomy (CEA) in comparison with transcranial Doppler (TCD).⁸

The validity of rSO₂ also has been assessed by comparison with direct microprobe measurement of brain tissue oxygen partial pressure and reasonable agreement between these two measures has been found.⁷ The normative preprocedural value rSO₂ is 67.2 ± 10.3 scale units. The pre-induction baseline brain rSO₂ for this case was right 80% and left 70%. Previous studies have stated that a brain rSO₂ ≤ 50 scale units appears to represent an increased risk for hypoxic injury and that a drop by 20% below baseline is clinically important.^{9, 10} In the case of our patient - at any given time during anesthesia and procedure, the rSO₂ values were not below 50% scale units or 20% below baseline.

Conclusion

This case presented a rare challenge of handling aortic dissection without cardiopulmonary bypass, only by using rSO₂. This method prevents cerebral ischemia without any profound neurologic complications due to the use of cardiopulmonary bypass and deep hypothermic cardiac arrest. Further debranching of the aortic arch, especially vertebral arteries debranching with this method, requires further investigations.

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