Blunt Traumatic Aortic Injury of Right Aortic Arch in a Patient with an Aberrant Left Subclavian Artery

INTRODUCTION

Right aortic arch (RAA) is an uncommon anatomical variant that occurs in about 0.1 percent of the population [1]. Two main types are commonly seen: mirror-image branching (Type I) (Figure 1) and aberrant left subclavian artery (LSA) (Type II) (Figure 2). Although extremely rare, there is also a third type, which involves an isolation of the LSA [3]. Type I RAA is commonly associated with congenital cyanotic heart disease, while Type II RAA is often accompanied by a Kommerell’s diverticulum, an aneurismal diverticulum that develops at the origin of the LSA and the proximal aspect of descending aorta [4].

Patients with Type II RAA are often asymptomatic and only diagnosed incidentally in adulthood or when complications arise from compression of the mediastinal structures, caused by a growing Kommerell’s diverticulum [5]. Current literature regarding RAA focuses on the development of this anatomical variant and the surgical techniques available to manage aortic aneurysmal disease in such patients.

In this paper, we report a patient with a Type II RAA who suffered a blunt thoracic aortic injury (BTAI) caused by a road traffic accident (RTA). Although BTAI is found in only 2 percent of patients who suffer a blunt trauma to the thorax [6], it is ranked the second leading cause of death in individuals aged 4 to 34 [7], with 81 percent of cases caused by automobile collisions [8].

CASE PRESENTATION

A 20-year-old man was admitted to the hospital after being involved in an RTA. On initial examination, the patient was drowsy and only responded to voice. He com-
plained of severe pain arising from the center of his chest that radiated to his back. His respiratory rate was 12 and oxygen saturation was 98 percent on 60 percent oxygen with a non-rebreather mask. His blood pressure was 106/60 mmHg, and his heart rate was regular at 78 beats per min. An urgent whole-body, contrast-enhanced computed tomography (CT) scan was arranged. Based on the patient’s presentation, the emergency physicians suspected aortic injury, and hence, a contrast-enhanced CT scan was chosen over non-contrast CT.

The scan revealed a RAA that descends along the right side of the spine before turning left to enter the aortic hiatus at the normal position. An aberrant LSA was also seen arising at the junction of the aortic arch and the descending aorta (Figure 3). The branches of the RAA — proximally to distally — are as follows: left common carotid (LCCA), right common carotid (RCCA), right subclavian artery (RSA), and aberrant LSA (Figure 4).

At the origin of the aberrant LSA, there was a dilatation of the arch of the aorta known as Kommerell’s diverticulum. Facing anteriorly, a pseudo-aneurysm arose between the origins of the right and left subclavian artery (Figures 3 and 4). It measured 31.5mm and was compressing the trachea. As the pseudo-aneurysm was anatomically separate from the Kommerell’s diverticulum, we believe it likely was caused by the RTA.

An endovascular repair of the pseudo-aneurysm using a covered stent graft was deemed unsuitable due to the proximity of its location to the subclavian arteries, as it may have compromised blood supply to the upper limbs.

Instead, a method known as the frozen elephant trunk technique was employed.

A sternotomy was carried out to expose the heart, the ascending aorta, and the aortic arch. A cardiopulmonary bypass (CPB) was established after cannulation of the distal ascending aorta and right atrium. A left ventricular vent was inserted via the right superior pulmonary vein to maintain a dry surgical field and prevent ventricular distension following reperfusion and rewarming — a critical period especially if ventricular function does not return immediately after the release of the aortic cross clamp. A retrograde cardioplegic cannula was inserted through the right atrium to maintain selective antegrade cerebral perfusion. The RSA was anastomosed to a 10mm Hemashield graft, where perfusion of cold blood continued.

Replacement of the damaged aortic arch and re-anastomosis with its tributaries was performed using the frozen elephant trunk technique. A 26mm Vascutek Thoraflex Hybrid stent graft was deployed into the ascending aorta, and its distal endovascular stent was deployed into the proximal descending aorta using a guide wire. A transesophageal echocardiogram (TOE) was used to verify its position. Distal body circulation recommenced with one of the side arms of the stent graft along with systemic re-warming, while cerebral perfusion remained cool.

During the re-warming period, the LCCA and RCCA were anastomosed to their respective branches of the graft.
along its arch, while the RSA was anastomosed to its most proximal side-branch. The aberrant LSA remained untouched. Re-warming of cerebral perfusion was initiated. After reaching a core temperature of 35°C, the patient was weaned off CPB with no complications.

Post-operative TOE showed a well-expanded stent graft with no extravasations (Figure 5). Perfusion to all branches of the aortic arch was also present. The patient was discharged 2 days later.

Discussion

In this reported case, the frozen elephant trunk technique was employed to repair the damage caused by the blunt traumatic injury. This is a variation of the original elephant trunk technique, which was developed in particular to treat extensive aortic disease of the aortic arch and descending aorta in two stages. First, the diseased aortic arch is removed, with the great vessels preserved as an island graft for later anastomosis. A conventional tube graft is invaginated into the proximal descending aorta. The folded end of the graft is sutured to the transected edge of the proximal descending aorta. The proximal end of the graft is then retracted and anastomosed to the transected edge of the proximal aortic arch. The great vessel island graft is then separately sutured to the prosthetic aortic arch. The distal end of the graft remains hanging freely within the descending thoracic aorta until it is utilized in a second surgery to repair the descending aorta [9]. Though initially pioneered in 1983 by Borst et al. [10] to lower the risk of lung, blood vessel, and nerve injury as well as the duration of the aortic cross-clamp times associated with thoracic aortic repair, the elephant trunk technique has undergone decades of modification, refinement, and optimization into various adaptations.

The frozen elephant trunk technique condenses the classical technique into a single step. It is performed with a “hybrid” vascular graft, which is a combination of a conventional tube graft proximally and an endovascular stented graft distally. The distal end, containing a Dacron collar, allows for adaptation of the graft to the aorta — the diameter of which may vary in aneurysmal disease. The Dacron collar, along with the endovascular stenting of the proximal descending aorta, achieves a hemodynamic seal, thereby completing the repair of the descending aorta. The aims of a traditional elephant trunk are thus achieved in a single step without the need for a secondary endovascular or surgical procedure [11].
different origins [18,19,20]. The propagation of a water-hammer effect caused by a blunt injury is another proposed mechanism of BTAI. This occurs when pressure waves created from an abrupt obstruction of blood flow reflect backward along the blood vessel, thereby temporarily increasing stress on the vessel wall. It has been established that an increase in curvature of the aorta can intensify the force of this pressure wave. In RAA patients, there is a more acute curvature of the arch, which intensifies the water-hammer effect, thus potentially causing more damage on the aortic wall [25].

A recent proposed theory also argues that a BTAI is caused by an “osseous pinch” in which entrapment of the aorta between the vertebral column and anterior bony structures, such as the sternum, first ribs and medial clavicles, result in lacerations of the vessel wall. However, it is likely that instead of only one mechanism, it is a collection of the previously mentioned mechanisms that result in a BTAI [26].
These theories suggest that it is an increase in the stress experienced by the aortic wall that ultimately leads to rupture and that this is further exacerbated in a RAA. As we proposed earlier, the aortic isthmus in a patient with Type II RAA could possibly be an anatomical point of weakness based on its embryological origins, as well as by the presence of Kommerell’s diverticulum.

In conclusion, based on this information, we propose that patients with Type II RAA may be at an increased risk of BTAI. Unfortunately, due to the rarity of cases for BTAI seen in Type II RAA, it will be difficult to conduct a robust observational study. However, to explore this relationship further, we would like to suggest that investigational studies be conducted, which may be able to link histopathological characteristics of RAA with the biomechanical features of BTAI. An example would be a histological study of RAA in rare patients who do not survive BTAI, as autopsies would be able to reveal the structural characteristics of the aortic wall in such patients, and how the fibrinogen and elastin fibers are positioned at aortic isthmus. This would allow for biomechanical simulation of the stress-strain distribution during traumatic rupture and reveal if the aortic isthmus is truly at the highest risk for BTAI.

REFERENCES
1. Shuford WH, Sybers RG, Gordon JJ, Baron MG, Carson GC. Circumflex retroesophageal right aortic arch simulating mediastinal tumor or dissecting aneurysm. AJR Am J Roentgenol. 1986;146:491-6.
2. Hsu KC, Tsung-Che Hsieh C, Chen M, Tsai HD. Right aortic arch with aberrant left subclavian artery – prenatal diagnosis and evaluation of postnatal outcomes: report of three cases. Taiwan J Obstet Gynecol. 2011;50(3):353-8.
3. Türkvatan A, Büyükbayraktar G, Ölçer T, Cumhur T. Congenital anomalies of the aortic arch: Evaluation with the use of multidetector computer tomography. Korean J Radiol. 2009;10(2):176-84.
4. Edwards JE. Anomalies of the derivatives of the aortic arch system. Med Clin North Am. 1948;32:925-48.
5. Hastreiter AR, D’Cruz IA, Cantez T, Namin EP, Licata R. Right-sided aorta. I. Occurrence of right aortic arch in various types of congenital heart disease. II. Right aortic arch, right descending aorta, and associated anomalies. Br Heart J. 1966;28:722-39.
6. Ungar TC, Wolf SJ, Haukoos JS, et al. Derivation of a clinical decision rule to exclude thoracic aortic imaging in patients with blunt chest trauma after motor vehicle collisions. J Trauma. 2006;61(5):1150-5.
7. Schulman CI, Carvajal D, Lopez PP, Soffer D, Habib F, Augenstein J. Incidence and crash mechanisms of aortic injury during the past decade. J Trauma. 2007;62:664-7.
8. Fabian TC, Richardson JD, Croce MA, et al. Prospective study of blunt aortic injury: Multicenter Trial of the American Association for the Surgery of Trauma. J Trauma. 1997;42(3):374-80.
9. Johnson PT, Corl FM, Black JH, Fishman EK. The elephant trunk procedure for aortic aneurysm repair: an illustrated guide to surgical technique with CT correlation. AJR Am J Roentgenol. 2011;197(6):W1052-9.
10. Borst HG, Walterbusch G, Schaps D. Extensive aortic replacement using “elephant trunk” prosthesis. Thorac Cardiovasc Surg. 1983;31(1):37-40.
11. Pichlmairer MA, Töebken OE, Baraki J, Havérich A. The frozen elephant trunk technique. Multimed Man Cardiothorac Surg. 2007;(329:mmcts.2006.001990.
12. Ciniá CS, Aithani H, Pasenau J, Abouzahr L. Kommerell’s diverticulum and right-sided aortic arch: a cohort study and review of the literature. J Vasc Surg. 2004;39(1):131-9.
13. Naoum JJ, Parenti JL, LeMaire SA, Coselli JS. Endovascular repair of a right-sided descending thoracic aortic aneurysm with a right-sided aortic arch and aberrant left subclavian artery. Ann Thorac Surg. 2008;85(3):1074-6.
14. Tsukui H, Aomi S, Yamazaki K. Surgical strategy for Kommerell’s diverticulum: Total arch replacement. J Thorac Cardiovasc Surg. 2014;148(4):1423-7.
15. Matsumoto M, Tanemoto K, Inagaki E, Hamanaka S, Masaki H, Nakata M, et al. Traumatic rupture of a right aortic arch in a patient with an aberrant left subclavian artery. J Thorac Cardiovasc Surg. 2006;131(2):464-5.
16. Majesky MW, Dong XR, Regan JN, Hoglund VJ. Vascular smooth muscle progenitor cells. Building and repairing blood vessels. Circ Res. 2011;108:365-77.
17. Jiang X, Rowitch DH, Soriano P, McMahon AP, Sucov HM. Fate of the mammalian cardiac neural crest. Development. 2000;127:1607-16.
18. Cheung C, Bernardo AS, Trotter MWB, Pedersen RA, Sinha S. Generation of human vascular smooth muscle subtypes provides insight into embryological origin-dependent disease susceptibility. Nat Biotechnol. 2012;30:165-73.
19. Raffetto JD, Khalil RA. Matrix metalloproteinases and their inhibitors in vascular remodeling and vascular disease. Biochem Pharmacol. 2008;75:346-59.
20. El-Hamamsy I, Yacoub MH. Cellular and molecular mechanisms of TAAs. Nat Rev Cardiol. 2009;6:771-86.
21. Elefteriades JA, Farkas EA. Thoracic aortic aneurysm clinically pertinent controversies and uncertainties. J Am Coll Cardiol. 2010;55:841-57.
22. Sevitt S. The mechanisms of traumatic rupture of the thoracic aorta. Br J Surg. 1977;64:166-73.
23. Moar JJ. Traumatic rupture of the thoracic aorta. An autopsy and histopathological study. S Afr Med J. 1985;67(10):383-5.
24. Lundewall J. The mechanics of traumatic rupture of the aorta. Acta Pathol Microbiol Scand. 1964;62:34-6.
25. Bodine JA, D’Souza VJ, Formanek AG. An unusual type of dissecting thoracic aneurysm in association with right aortic arch. Vasa. 1982;11:223-8.
26. Ben-Menachem Y, Handel SF. The mechanism of injury. Angiography in trauma. A work atlas. London: W.B Saunders; 1981.