CLINICAL REPORT

Acute ascending aortic intramural hematoma as a complication of the endovascular repair of a Type B aortic dissection

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Abstract Endovascular aortic graft repair (EVAR) for patients with Type B aortic dissection is a less invasive surgical procedure (compared to traditional open surgical repair) that is associated with less morbidity and shortened recovery times. However, there are notable complications for the patients undergoing EVAR. We report a patient who was brought to our hospital with a Type B dissection and underwent a thoracic EVAR but suffered iatrogenic aortic injury resulting in cardiac tamponade. This case study highlights the importance of intraoperative transesophageal echocardiography to facilitate early detection of possible EVAR complications.

Keywords Aortic dissection · Cardiac tamponade · Endovascular surgery · Transesophageal echocardiography

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Introduction

Endovascular aortic graft repair (EVAR) of descending aortic aneurysm dissection (Type B) is a promising alternative treatment modality to open graft placement [1-3]. However, EVAR is also associated with potentially fatal complications such as ascending aortic aneurysm dissections (Type A), with an incidence rate of 1.3-6.8% [3-5]. The overwhelming majority of these emergent Type A dissections have been diagnosed using computed tomographic (CT) scans or angiograms [4, 5]. The utility of transesophageal echocardiography (TEE) in EVAR procedures is well documented [6, 7], and a recent article exploring intraoperative TEE use suggests that it can be utilized to guide and evaluate stent placement and function, supplement angiographic information, detect endoleaks, and monitor cardiac function [8]. However, there is no published information on the intraoperative use of TEE to provide surveillance for intramural hematoma and dissections of the ascending aorta for complications resulting from EVAR of Type B dissections. This case report represents an acute ascending aortic intramural hematoma with a resultant cardiac tamponade, diagnosed intraoperatively using TEE, which arose as a complication of an EVAR for a Type B aortic dissection. The expanding intramural hematoma led to cardiac tamponade and resulted in cardiac arrest as well as the patient's demise prior to open surgical intervention.

Case report

An 82-year-old man was transferred to our hospital with complaints of abdominal and right leg pain, with a loss of leg motor function. A CT scan revealed a Type B dissection 2 cm from the left subclavian artery to the infrarenal abdominal aorta with complete aortic occlusion distally. Consent for the patient's enrollment in the Medtronic Valiant Captivia (Minneapolis, MN, USA) device evaluation in the treatment of acute thoracic dissection was obtained from the patient's son by the vascular surgery service.

After preoxygenation, induction was performed with etomidate 14 mg, fentanyl 100 μ g, and rocuronium 50 mg. Subsequently, atraumatic oral tracheal intubation (8.0 mm outer diameter [OD] tube) was initiated during loss of consciousness with stable hemodynamics throughout. Anesthesia maintenance was achieved with 2% sevoflurane in 60% oxygenation. TEE provided initial survey of heart and thoracic aorta. TEE confirmed a Type B dissection with normal ascending aorta and aortic valve. The entry tear was visualized to be isolated to the false lumen with pulsatile blood flow in the true lumen. Additionally, mild mitral and tricuspid valvular regurgitation, dyskinesia of the inferoseptal wall, and a 40% left ventricular ejection fraction were present.

Fluoroscopy and intravascular ultrasound (IVUS) provided information for endograft stent "landing zone" and size selection, respectively. Following endograft IVUS examination, the guide wire was exchanged from a malleable wire to a stiff wire in preparation for stent-graft device positioning. During guide wire exchange, intraoperative ventricular ectopy developed secondary to an inadvertent IVUS probe advancement into the left ventricle confirmed by fluoroscopy. Once the IVUS probe was retrieved, the ectopy was resolved.

A 36-mm endoprosthesis [219 mm total length (207 mm covered length)], with proximal free flow and distal closed web configuration, was enclosed in the Captivia delivery system and deployed with the open stent positioned across the left common carotid artery origin, while the cloth portion of the device covered the left subclavian artery origin. Post-procedure TEE evaluation showed no presence of endoleak. However, upon further surveillance of the entire thoracic aorta it was noted that a crescent-shaped intramural hematoma at the sinotubular junction, extending at least 2-3 cm cephalad in the midesophageal (ME) ascending aorta short-axis view (Fig. 1), was a new finding that had not been present at the baseline TEE examination. The most distal portion of the hematoma was not visualized secondary to hypoechoic dropout of the distal ascending aorta. The hematoma expanded from 0.7 to 1.1 cm in its short axis within the ensuing 20 min (Fig. 1). Color Doppler examination revealed no flow within the expanding hematoma (supplementary Fig. 1 [S1]). An orthogonal view (ME ascending aorta long-axis view) was also obtained to rule out the presence of echo artifact. The cardiothoracic surgeon was informed of the expanding hematoma and a new development of pericardial fluid noted on TEE. As we do



Fig. 1 Midesophageal (ME) aortic valve short-axis view demonstrating the expanding intramural hematoma



Fig. 2 Transgastric (TG) mid-papillary short-axis view demonstrating pericardial fluid

not have a convertible endovascular suite or percutaneous cardio-pulmonary support equipment, preparations were made to transport the patient to the cardiac operating room. During this time, the patient acutely decompensated and went into cardiopulmonary arrest. TEE examination demonstrated progression into acute cardiac tamponade (Fig. 2/ supplementary Fig. 2 [S2]). Advanced cardiac life support was initiated. A subxiphoid angiocatheter evacuation of pericardial blood was performed. The patient expired despite resuscitative efforts.

Discussion

Endovascular aortic graft repair (EVAR), while associated with lower morbidity and mortality than open surgical repair, is associated with significant complications including stroke, rupture of the aneurysm, endoleak, spinal cord ischemia, paraplegia, retroperitoneal bleeding, and iatrogenic retrograde Type A dissection [9, 10, 11]. The

majority of ascending aorta complications are diagnosed utilizing CT or angiography [4, 5]. A recent publication demonstrated that A-View TEE (which uses a water-filled catheter to eliminate the distal ascending aorta and threedimensional [3D] TEE anatomical "blind spot") is useful for pre-operative aortic evaluation [12], and another article exploring intraoperative TEE use suggests that it can be utilized to guide and evaluate stent placement and function. supplement angiographic information, detect endoleaks, and monitor cardiac function [8]. To the best of our knowledge, ascending aorta intramural hematoma and hemopericardium, as a complication of EVAR, has not previously been reported. Moreover, thoracic EVAR-associated hemopericardium, resulting in cardiac tamponade, has been previously reported in only one case [13]. However, the inciting event in that case was the direct wire perforation of the left ventricle. Our case represented pericardial fluid originating from the intrapericardial portion of the ascending aorta.

A postmortem autopsy showed a perforation in the ascending aorta at the same level as the hematoma visualized on TEE. Iatrogenic injury along the greater curvature (as represented in our case) is more probable to occur than the lesser curvature due to the recoil of a stiff wire around the aortic arch. Hence, when wire-related injury is suspected it is important to pay close attention to the aortic wall along the greater curvature. The rigid wire plays an important role in the mechanism. In the present case, the rigid wire accumulated tension as it advanced through the tortuous descending aorta. As this tension increased, the IVUS, acting as a sheath, was allowed to shoot forward as the wire tension was relieved by the frictional forces of the IVUS probe. This caused the wire to recoil out to its natural straightened position and the IVUS probe then sprang forward beyond the wire tip, resulting in an aortic wall perforation.

While aortic perforation was not visualized on TEE, it can be surmised that an intramural hematoma is a manifestation of aortic wall injury. Notably, the aforementioned portion of the ascending aorta, at the sinotubular junction, contains maximal wall stress. Increased intraluminal pressure with turbulent blood flow and high shearing force caused retrograde hematoma extension into the pericardium. Rapid fluid accumulation led to acute cardiac tamponade. The echocardiographic features of tamponade were visualized immediately prior to the physiological manifestations. In summary, the use of TEE for intraoperative surveillance in EVAR can result in the earlier detection of complications such as dissection or hematoma formation. The dilemma underscores the advances in technology which improve diagnostic detection, yet the ability to truly "save" a patient is limited by the facilities and resources that are readily available (e.g., in our case, no

percutaneous cardiopulmonary support and no convertible endovascular suite). Nevertheless, intraoperative TEE use by trained anesthesiologists can direct surgical management and serve as an early detection modality for thoracic aortic pathologies.

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