dioplegia, the aorta was opened and the septal avulsion was excised through the aortic valve. It was firmly attached and pink (Fig 1F). The VSD was closed through the tricuspid valve with autologous fixed pericardium. Large bites were taken through the rim of the friable VSD. Echo revealed no residual VSD. The patient was extubated on the table and discharged in 5 days. Later, a small VSD patch leak was noted and resolved on follow-up.

Comment

Cardiac injury occurs in 20% of high-speed MVAs with blunt chest trauma [2]. Though most are cardiac contusions, traumatic VSDs present in 2% to 10% of blunt chest trauma cases from MVAs [2]. They are more common in children and adolescents, probably from the pliability of the immature chest wall [3]. They result from compression of the heart between the chest wall and the spine, usually from rapid deceleration. In the course of the cardiac cycle, full ventricles with closed atrioventricular valves make the heart most susceptible to increases in intraventricular pressure that can rupture the septum [1, 3, 4]. Apical muscular VSDs are most common, and subpulmonic and infundibular defects less so [2].

Although most traumatic VSDs occur immediately, delayed onset from hours to weeks after trauma can occur, and are thought to result from myocardial injury followed by microvascular disruption followed by liquefaction necrosis and VSD formation [1, 4]. Appearance of a murmur is correspondingly delayed [5]. Symptoms vary with size of the VSD but most commonly include dyspnea, a new systolic murmur, cardiomegaly, and congestive heart failure [6]. If the VSD is large, hemodynamic decompensation and death can occur rapidly. Echocardiography is the diagnostic mainstay and is reliable, noninvasive, and easily repeated for changes in status, a new murmur, or before discharge. The location, size, and hemodynamic impact of the VSD should be assessed.

While traumatic VSD is well described, to our knowledge this is the first reported case of ventricular septal avulsion. The management is usually guided by the size of the VSD. Patients with small shunts (pulmonary-to-systemic flow ratio [Qp/Qs] < 1.5:1) like this child can usually be managed medically. Device closure may have been possible here had it not been for the hypermobile flap of avulsed septal muscle prolapsing ominously through the aortic valve. Any rush to repair was tempered by the patient’s head trauma (loss of consciousness), and pulmonary contusion with active hemoptysis. Because the attachment of the flap seemed thick at its base, and we deduced it to be muscle, we were somewhat reassured to allow the hemoptysis to clear preoperatively. Our suspicions were confirmed at surgery and the patient recovered quickly.

References


Stab to the Chest Causing Severe Great Vessel Injury

Eshan L. Senanayake, MRCS, Janan Jeyatheesan, MBChB, Venessa Rogers, MRCS, Ian C. Wilson, FRCS, Tim R Graham, FRCS

Department of Cardiac Surgery, University Hospital Birmingham NHS Foundation Trust, Birmingham, United Kingdom

Penetrating trauma has increased in developed and urban environments. Pulmonary artery injury is rare, but can be associated with significant morbidity. We report a case of delayed cardiac arrest following a stab injury to the chest. The patient had active great vessel bleeding and required extensive surgical intervention. Clinicians should have a high index of suspicion for life-threatening thoracic injuries following a stab injury to the chest, despite initial clinical stability or negative baseline radiological findings.


A ny penetrating injury to the thorax is a potentially life-threatening injury, particularly when the area of penetration is within the “thoracic box” bordered by the thoracic outlet, xiphisternum, and midclavicular lines. Blunt injury to the Pulmonary Artery (PA) is considered to be extremely rare, and one would expect penetrating injuries to the PA to be more common. However, there are limited reports in the literature of such injuries, with the majority of penetrating thoracic vascular trauma associated with injuries to the aorta or its branches [1].

A 54-year-old man weighing 110 kg presented to the emergency department within 1 h of the injury, with a 2-cm stab wound to the left side of his chest, 1 cm lateral to the sternum in the second intercostal space. The instrument was not found. He had pain at the injury site,

Accepted for publication March 23, 2012.

Address correspondence to Mr Graham, University Hospital Birmingham NHS Foundation Trust, Queen Elizabeth Hospital, Mindelson Way, Edgbaston, Birmingham B15 2WB, United Kingdom; e-mail: tim.graham@uhb.nhs.uk.

© 2012 by The Society of Thoracic Surgeons
Published by Elsevier Inc

http://dx.doi.org/10.1016/j.athoracsur.2012.03.104
but was alert and oriented to time and place. Physical examination revealed equal bilateral vesicular breath sounds and normal heart sounds. Pulse oximetry was 100% on 10 L of O₂ via a face mask, heart rate was 100 beats/min, and blood pressure was 78/36 mm Hg. Initial arterial blood gas showed a metabolic acidosis (Base excess, –9.2; lactate, 7.1). Following 1.5 L of colloid resuscitation, his heart rate was 80 beats/min and blood pressure was 110/59 mm Hg. Results of a secondary survey were unremarkable. Serial arterial blood gasses revealed an improving metabolic acidosis.

An anteroposterior chest radiograph suggested some widening of the mediastinum (Fig 1). Transthoracic-echocardiogram (TTE) showed no pericardial effusion and confirmed good left ventricular function with normal flow across the aortic arch. A computed tomography (CT) scan showed some anterior mediastinal hematoma with no clear evidence of vascular injury.

As a result of a period of hemodynamic stability, he was transferred to the intensive care unit for close monitoring and a repeated CT of the thorax. The repeated CT revealed an increasing collection retrosternally and a 12-mm defect along the left under surface of the aortic arch, communicating with the mediastinal hematoma; this implied active extravasation and aortic injury (Fig 2). He became hemodynamically unstable and sustained a pulseless electrical activity arrest. Cardiopulmonary resuscitation was performed and return of spontaneous circulation was established within 1 minute. He experienced another arrest approximately 5 minutes later lasting 1.5 minutes before return of spontaneous circulation. Judicious resuscitation with colloids and blood was required to manage a persistent hypotension. Transfer to surgery was expedited for an emergency sternotomy and exploration with full cardiac surgery theater and perfusion support.

At median sternotomy, he was found to have a large anterior mediastinal hematoma with a large left hemothorax. There was a 2-cm tear in the inferomedial aspect of the arch of the aorta; this required initial control with a Foley catheter, but satisfactory control could not be achieved to accomplish a satisfactory aortic arch repair. Urgent cardiopulmonary bypass (CPB) was instituted; arterial return into the ascending aorta and two-stage venous cannula into the right atrium and inferior vena cava. The patient was cooled to 17°C, and hypothermic circulatory arrest (HCA) achieved. The aortic tear was repaired during HCA. During this period of HCA the main pulmonary artery was inspected and no defect was identified. On attempting to discontinue from CPB, there was excessive bleeding from the anterior aspect of the right PA, which was difficult to access. Therefore, CPB was reinstituted, the patient’s core body temperature was cooled to 26°C, and the heart arrested with intermittent antegrade cold blood cardioplegia. The ascending aorta was transected to provide access, which revealed a laceration in the anterior wall of the right PA. This anterior tear was extended to visualize the posterior wall, which showed a tear in the posterior wall of the right PA. These tears were subsequently repaired, and CPB was successfully discontinued with considerable vasoconstrictor and inotropic requirements. During this exploration, it was not possible to exclude an esophageal injury posterior to the right PA.

Perioperatively and early postoperatively, he required a massive blood transfusion and treatment for a coagulopathy. His postoperative course was unremarkable, and he was discharged home on the ninth postoperative day. The results of a water-soluble contrast study to assess esophageal integrity were unremarkable. Follow-up at 6 weeks postoperatively showed good recovery and return to normal activities.

Comment
Penetrating trauma to the thorax following a stab wound is more common than gunshot injuries and has the potential to damage intrathoracic great vessels [1]. A stab

---

**Fig 1. Chest radiograph showing a widened mediastinum.**

**Fig 2.** Computer tomography of the thorax. The yellow arrow shows opacification after intravenous contrast showing active bleeding, enveloped by a large mediastinal hematoma and stranding. Red arrow shows the likely position of the aortic tear.
wound to the chest can have a wide range of clinical presentations, from no intrathoracic injury to extensive life-threatening great vessel damage, as outlined in this case. Injury to the pulmonary artery alone can be complex, albeit amenable to successful surgical repair [2]. In this case, there was additional injury to the inferomedial aspect of the aortic arch, which in itself could be fatal [3].

TTE to exclude a pericardial effusion can be a useful tool; however, it was a false-negative finding in this case. Negative findings, although initially reassuring, must not be misleading particularly in a case with penetrating trauma to the thoracic box. CT scans can aid operative planning and provide information on the location of the injury [4]. In this case, the CT scans provided an accurate location of the aortic arch injury, but were only suggestive of a pulmonary artery injury. This patient had no features of hemothysis, which could be an indication of PA injury [2]. There have been reports of delayed cardiac tamponade following initial negative investigations [5]; therefore, a high index of suspicion for injury must be maintained in any patient with a penetrating injury to the thorax.

The potential for a posterior right PA injury was recognized perioperatively by extending the anterior right PA injury. This could only be repaired by extension of the anterior tear. Transection of the aorta provided necessary access, to visualize and repair both the anterior and posterior walls of the right PA. The use of CPB facilitated this repair, and transection of the aorta should be considered when faced with extensive injuries to the pulmonary artery.

This case illustrates the rapid decline in hemodynamic stability, resulting in cardiac arrest, associated with such an injury. This patient’s reasonable clinical status following initial resuscitation was falsely reassuring. In retrospect, the nature of the injury, initial hypotensive presentation, metabolic acidosis, chest radiographic findings, and initial CT thorax scan provided sufficient cause for earlier surgical exploration.

Median sternotomy allowed establishment of CPB, HCA, and cardiopulmonary arrest and provided the best access for repair of the aortic arch and right PA. If this patient had remained cardiovascularly unstable and then arrested, a more utilized approach, of an urgent emergency room anterior bilateral thoracotomy may have been indicated [6]. This approach, although useful in most circumstances of penetrating thoracic injury, would not have allowed control of the bleeding, institution of central CPB, and surgical repair, because of the nature of these injuries. Correct interpretation of the CT scan facilitated the correct operative strategy and environment for this patient.

A single stab injury to the thorax can cause extensive intrathoracic damage to the great vessels and is associated with morbid consequences. A high index of suspicion for intrathoracic injury should remain despite initial cardiovascular stability and early negative investigative findings. This suspicion should prompt further investigations and early surgical exploration as required.

References


Transaortic Valve-in-Valve Implantation After Previous Aortic Root Homograft

Leo Ihlberg, MD, PhD, Antero Sahlman, MD, Juha Sinisalo, MD, Janne Rapola, MD, PhD, and Mika Laine, MD, PhD

Departments of Cardiothoracic Surgery and Cardiology, Helsinki University Hospital, Helsinki, Finland

Catheter-based valve implantation techniques are becoming a viable option in various clinical situations to replace difficult redo open heart surgical procedures. This is a report of a first, to our knowledge, successful valve-in-valve (VinV) transcatheter aortic valve implantation (TAVI) into a homograft through the transaortic (T Ao) access route using an Edwards SAPIEN valve prosthesis (Edwards Lifesciences, LLC, Irvine, CA) in a patient with poor left ventricular function and generalized severe atherosclerosis.

© 2012 by The Society of Thoracic Surgeons

Repeated aortic root operations for a failed homograft can be a technically challenging undertaking, especially if the whole aortic root needs to be replaced. Associated risks could even make a conventional open surgical approach prohibitive. Transcatheter aortic valve implantation (TAVI) is a new treatment modality that could potentially be used for this patient group. Herein we report the first, to our knowledge, valve-in-valve (VinV) TAVI into a homograft through the transaortic approach followed by a homograft explantation.

© 2012 by The Society of Thoracic Surgeons

Accepted for publication March 29, 2012.

Address correspondence to Dr Ihlberg, Department of Cardiothoracic Surgery, Helsinki University Hospital, PO Box 340, FI-00029 Helsinki, Finland; e-mail: leo.ihlberg@hus.fi.

Dr Ihlberg discloses that he has a financial relationship with Edwards Lifesciences.