Traumatic Aortic Incompetence Associated With Transection of the Thoracic Aorta*

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A case of blunt chest trauma resulting in transection of the descending thoracic aorta and disruption of the aortic valve is presented. Successful treatment required graft repair of the aortic injury followed by aortic valve replacement.

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Key words: aortic valve incompetence; blunt chest trauma; thoracic aortic transection

Survival following cardiac and great vessel injury is rare. In this report, we describe a patient who suffered both aortic valve disruption and transection of the descending thoracic aorta as a result of blunt chest trauma. The patient survived the injuries and tolerated the corrective procedures well. This is an unusual combination of injuries in a surviving patient.

Case Report

An otherwise healthy 18-year-old man was admitted to the Emergency Department after being involved in a high-speed head-on motor vehicle accident. He was awake and lucid, mainly complaining of shortness of breath, pain on the left side of the chest, and lower leg pain. Physical examination revealed a blood pressure of 95/45 mm Hg, a pulse of 120, and a respiratory rate of 30 breaths/min. Other significant findings included decreased breath sounds over the left hemithorax, a grade II diastolic heart murmur, mild diffuse abdominal tenderness, and dislocation of both ankles. A chest radiograph demonstrated an apical cap on the left and a widened mediastinum (Fig. 1). Peritoneal lavage was grossly positive for hemoperitoneum. The patient underwent emergency laparotomy which revealed multiple grade I and II liver lacerations. He also had bilateral ankle fractures reduced and splinted. An arch aortogram was performed directly after surgery. This study revealed disruption of the descending aorta just distal to the origin of the left subclavian artery and aortic insufficiency. The patient had no known history of cardiac disease. At thoracotomy, the descending aorta was three-fourths transected 2 cm distal to the isthmus. Using the technique of simple aortic cross-clamping, a Dacron interposition graft was used for repair of the aorta. Aortic cross-clamping time was 30 min.

Postoperatively, the patient developed signs of pulmonary edema and congestive heart failure. He required daily doses of diuretics, digoxin, and hydralazine hydrochloride for control of heart failure and elevated systolic blood pressure (180 to 200 mm Hg). Echocardiography revealed moderate aortic valve regurgitation and left ventricle of normal size. After extubation and chest tube removal, cardiac catheterization was performed. This study demonstrated severe aortic regurgitation from the region of the left coronary cusp. Through a median sternotomy, the patient underwent aortic valve replacement with a 23-mm St. Jude prosthesis. The native valve was remarkable for a large disruption in the midportion of the left coronary cusp (Fig 2). There was no evidence of discontinuity between the valve and aortic annulus. The patient subsequently had an uneventful hospitalization and is currently well 3½ years postoperatively.

Discussion

Aortic valve injury as a result of blunt chest trauma is an unusual event. There have been fewer than 30 cases of aortic valve damage associated with blunt chest trauma that have required surgery. Parmley et al,1 in 1958, reported one case of isolated aortic valve injury and three cases of valve injury associated with cardiac rupture. Leonard et al,2 in 1955, described a 17-year-old boy who developed acute aortic insufficiency and congestive heart failure after being kicked by a horse. Beall and Shirkey3 described a patient who devel-

![Figure 1. Chest radiograph on admission.](image1)

![Figure 2. Aortic valve leaflets showing damage to the left coronary cusp.](image2)
opened a tear in the intima of the aorta at the attachment of the commissure between the left and noncoronary cusps of the aortic valve after blunt chest injury. Attempts were made to repair the valve, but replacement was eventually required. Devine and McKenzie,3 Schwartzberg and Khalil,5 and German et al6 have also reported cases of aortic valve injury after blunt trauma. In those cases, successful treatment required valve replacement.

The mechanism of valve injury is thought to be a sudden increase in intrathoracic pressure during diastole when the valve is closed. Tears across a cusp and avulsion of a cusp from the annulus have been described. In contrast to aortic valve injury from blunt trauma, rupture of the thoracic aorta is not unusual, accounting for 15 to 20% of deaths among victims of automobile accidents.7 It is commonly taught that 80% of patients who suffer this injury die at the scene and another 10 to 15% die in the hospital. Many of these patients have second injuries that would prove fatal even if the aortic rupture had not occurred.

The most common mechanism of aortic rupture is the force generated by rapid deceleration of the body. The greatest strain in the thoracic aorta is at the isthmus where the mobile thoracic aorta joins the more fixed aortic arch. It is this area where most aortic ruptures are identified.

Aortic valve disruption and rupture of the thoracic aorta share a common mechanism of injury. It is not unusual that these injuries could occur together. Most of these patients die at the scene of the accident from the thoracic and cardiac trauma or from associated fatal second injuries.

In the case presented, the patient had an abnormal chest radiograph suggestive of aortic transection in addition to gross hemoperitoneum by lavage. We believed that the abdominal injury should take precedence because he was actively bleeding from this site. An arch aortogram was performed directly after surgery for diagnosis of aortic transection. Transesophageal echocardiography performed during the exploratory laparotomy would have been the diagnostic modality of choice had the technology been available to us at the time. This would have easily demonstrated the aortic disruption as well as the valve insufficiency. We would then have proceeded to thoracotomy and repair of the aorta without delaying for an arteriogram.

Postoperatively, the patient developed congestive heart failure. This was not unexpected in the face of acute aortic regurgitation. Despite this, we felt it more prudent to manage his failure medically than to place a patient with multiple grade II liver lacerations on cardiopulmonary bypass immediately after exploratory laparotomy and thoracotomy. Aortic valve replacement was subsequently performed without complications.

The most unusual feature of this case is the favorable outcome for the patient. He had two routinely fatal injuries (cardiac and aortic) in addition to intra-abdominal and orthopedic trauma. Standard trauma protocol required emergency laparotomy to prevent exsanguination followed by diagnosis and repair of the thoracic aortic injury. The patient subsequently required aortic valve replacement for acute aortic insufficiency. Currently, at 3½ years postoperatively, the patient is leading a normal life.

References


Transmission of Invasive Aspergillosis From a Subclinically Infected Donor to Three Different Organ Transplant Recipients*

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Objective: To describe a cluster of donor-transmitted cases of invasive aspergillosis.

Design: Case series of epidemiologically linked cases of invasive aspergillosis.

Setting: Two tertiary care centers with solid-organ transplant programs.

Patients: Two kidney recipients, one heart recipient, and the single donor.

Measurements: Routine clinical, microbiological, and pathologic investigation as dictated for patient care. Epidemiologic analysis to establish linkage among cases.

Results: Three allografts (two kidneys and a heart) from a single donor transmitted invasive aspergillosis to the recipients. Three weeks after transplantation, the two kidney recipients had fever and urine cultures positive for Aspergillus fumigatus. The infected kidneys had multiple Aspergillus abscesses and had to be removed to cure the patients. The heart recipient had a negative workup when a diagnosis of aspergillosis was made for the kidney recipients but presented three months later with aspergillus endocarditis with

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