Arterial and Venous Trauma and Great Vessel Injuries

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KEY POINTS

- Thirty percent of patients with great vessel injury (GVI) die within 6 hours of hospital arrival.
- Thirty percent to 50% of patients with blunt aortic injury have no signs of trauma.
- A normal chest radiograph does not exclude GVI.
- Computed tomographic angiography is the diagnostic test of choice to rule out traumatic aortic injury in hemodynamically stable patients.
- For patients too large for a computed tomography scanner, transesophageal echocardiographic evaluation of the aorta should be considered.
- Medical management of GVI is typically used as a bridge to more definitive operative care.
- β-Adrenergic blockade is instituted before nitroprusside in the medical management of GVI to avoid possible reflex tachycardia.

EPIDEMIOLOGY

Few traumatic injuries are more devastating than great vessel injury (GVI). With an average circulating volume of 5 L and a flow rate of up to 4.8 L/min in the circulatory system, it is easy to see why GVI can result in catastrophic outcomes quickly. The true incidence of traumatic aortic injury may never be known; however, according to the National Trauma Data Bank, blunt thoracic aortic injury occurred in 0.3% of trauma patients admitted to the hospital during a 5-year period. When patients survive an initial injury to their great vessels, rapid diagnosis and treatment are imperative to prevent subsequent exsanguination within the next minutes to hours. This highlights the ever-emphasized “golden hour” of trauma resuscitation.

Several contributing factors are important when evaluating potential GVI (Fig. 90.1). Although the mechanism and specific vessel injured are the most important of these factors, significant attention must be paid to the role of concomitant injuries and comorbid conditions on patients’ morbidity and mortality. Unfortunately, on initial evaluation the emergency physician is often lucky to be privy to one, let alone all, of these factors.

The most important branch point for both the likelihood and the type of GVI is a penetrating versus blunt mechanism. Penetrating mechanisms are associated with greater than 90% of great vessel trauma, and any thoracic vascular structure is at risk. Patients who survive to arrive at the emergency department, particularly if they are not in hemorrhagic shock, have a survival rate that approaches 50%.

In contrast, blunt traumatic injuries to the great vessels most often affect the aorta, although the innominate artery, pulmonary hilar vessels, and vena cava are also susceptible. Blunt aortic rupture carries an immediate mortality rate of greater than 80% and is responsible for 10% to 15% of motor vehicle accident fatalities. Because of the high association of blunt ascending aortic injury with fatal cardiac injury, the vast majority of those who survive to hospital evaluation have descending injuries. Of patients who survive until medical evaluation, 30% die within 6 hours and 40% within 24 hours. Because most of these injuries occur in young healthy males, the overall survival rate is much better than expected given the severity of injury.

Though incompletely understood, it is proposed that blunt aortic injury can result from any combination of shearing forces, rotational forces, increased intraluminal aortic pressure, or a pinching mechanism between the sternum and vertebral column. Given these forces, it is not a surprise that motor vehicle collisions cause the majority of blunt aortic injuries. This association increases with the speed of the accident. Shearing forces were originally thought to be the highest in frontal-impact accidents, where deceleration forces are the greatest. More recent studies, however, have shown that side-impact accidents are associated with a higher risk for blunt aortic injury. A review of 119 cases of known blunt aortic injury as a result of car accidents in the United Kingdom found that lateral impact direction to the same side was highly associated with aortic injury. A review of accident data from the United Kingdom and United States in 2004 mirrored these results and found that side impact involving the patient’s side of the vehicle carried a significantly higher risk for aortic injury than did frontal impact. Although motor vehicle accidents account for the majority of blunt GVI, falls from a height and crushing forces have also been known to cause the disease process.

In part because of difficulty isolating the hilum, injuries to the pulmonary arteries, veins, and thoracic vena cava are associated with mortality rates greater than 60%, regardless of whether they are caused by blunt or penetrating force, although the latter is much more common. Concomitant injuries clearly play a role in the epidemiology, morbidity, and mortality of GVI. One study on blunt thoracic trauma showed that patient with traumatic aortic injury carried a mean injury severity score (ISS) of 40
whereas patients without vascular injury had a mean ISS of just 16. Another showed that closed head injury was diagnosed in more than half of patients with GVI, with one quarter having intracranial hemorrhage.

Comorbid conditions such as underlying vascular disease, cardiopulmonary disease, and renal insufficiency contribute to the morbidity and mortality associated with GVI. Many disease processes affect a patient’s ability to tolerate the initial and delayed physiologic insults accompanying severe GVI.

**PATHOPHYSIOLOGY**

Knowledge of the vascular anatomy of the great vessels and the particular branch points of the more distal vasculature is important in identifying and potentially preventing morbidity and mortality in the setting of injury. This anatomy can be broken down into arterial, venous, and pulmonary components.

**ARTERIAL SYSTEM**

The systemic arterial great vessels include the ascending aorta, arch, and descending thoracic aorta. The innominate artery is the first branch of the aortic arch and gives rise to the right subclavian and common carotid arteries. The left carotid and then the left subclavian artery are the next two branches. These structures course in close approximation to the clavicle, the first and second ribs, and the brachial plexus. Just distal to the left subclavian takeoff, the descending aorta becomes a more fixed structure in comparison with the arch. The ligamentum arteriosum, a remnant of the ductus arteriosus, and the intercostal arteries tether it to other thoracic structures. This junction, often called the isthmus region, proves to be the most susceptible site for blunt aortic injury as the arch moves in relation to the relatively fixed descending aorta. The spinal arteries branching off the descending aorta are of particular importance because they supply the spinal column. Compromised flow to these small branches as a result of direct injury or vascular clamping plays a significant role in patients’ risk for paraplegia.

The microanatomy of the artery wall, with its intimal, medial, and adventitial layers, is integral in the spectrum of disease. Injuries range from isolated thrombogenic intimal flaps to full-thickness tears with free hemorrhage (Fig. 90.2).

**VENOUS SYSTEM**

The venous components of the great vessel system include the confluence of the subclavian and internal jugular veins, which ultimately combine to form the superior vena cava. The inferior vena cava receives blood from the portal system through the hepatic vein in the retrohepatic region. As a whole, this system is characterized by low pressure and resistance and high flow and compliance, unless tamponade or heart failure is present, which results in increased right-sided pressure. These factors make control of hemorrhage difficult.

**PULMONARY CIRCULATION**

The final component of the great vessel system is the pulmonary circulation. As mentioned earlier, the structures of this system reside in the hilum and are deep in the thorax, thus making them difficult to access. The pulmonary arteries and veins possess mediastinal and intrathoracic portions, which can result in different clinical findings, including mediastinal hematoma or hemothorax.

**PRESENTING SIGNS AND SYMPTOMS (Box 90.1)**

Traditionally, aortic dissection as a result of nontraumatic causes is believed to be manifested as tearing pain radiating
through the chest to the interscapular region of the back. This pain can be accompanied by various degrees of associated symptoms, including shortness of breath or vagal complaints. In patients with traumatic injury, this symptom pattern is seen less than 25% of the time; these patients most frequently either have vague chest-related complaints or no complaints at all because of distracting injuries. A remarkable 30% to 50% of patients with a blunt aortic injury may have no external signs of trauma.10

The signs and symptoms of GVI often result from its effect on blood flow, which can be secondary to direct vessel injury, traumatic thrombus formation, or vascular compression from surrounding hematoma. Of great concern are clinical signs of hemorrhagic shock such as hypotension, tachycardia, altered mental status, pallor, or diaphoresis. Frequently, hypertension occurs as a result of increased stimulation of sympathetic nerve fibers in close proximity to the aortic arch.11 Additionally, many of the other signs can be subtle. Dyspnea may result for any number of reasons, such as associated pulmonary injury, hemothorax, hypovolemia with poor tissue oxygenation, or tamponade. Neurologic symptoms can be found in patients with arterial injury involving the carotids or spinal arteries.

Extremity findings of altered transmission or diminished intensity of the pulse pressure wave suggest intravascular volume depletion or, if asymmetric, direct vascular injury. Femoral pulses are important to note, particularly with respect to upper extremity pulses, because change can indicate vascular injury in the descending aorta resulting in a pseudo-coarctation syndrome. Vascular bruits, which result from turbulent blood flow in the arterial system, either over the precordium or in the interscapular region, are heard in up to 30% of patients with aortic injury.

Unfortunately, none of the aforementioned signs and symptoms are sensitive or specific for making the diagnosis of GVI.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

GVI should be considered in the differential diagnosis of all patients with thoracoabdominal trauma and an appropriate mechanism of injury. Care must be taken to not exclude the diagnosis solely on the basis of identification of other injuries that might also be contributing to the patient’s clinical findings.

GVI is easily misdiagnosed in hemodynamically stable patients, particularly those without external signs of trauma, because of the nonspecific nature of the initial signs and symptoms of GVI. Penetrating injury in proximity to any of the great vessels mandates consideration of GVI. However, a diagnosis of blunt injury requires assessment of the severity of the causative mechanism (e.g., speed, forces), in combination with the patient’s complaints and findings on physical examination. This pretest probability will ultimately be used by the clinician to guide further diagnostic evaluation for GVI (Box 90.2).

GVI is often associated with either significant multisystem trauma or distinct penetrating injury. In the first situation, the signs and symptoms of GVI are frequently obscured by other distracting injuries, altered mental status, or intubation, thereby necessitating a high level of suspicion and a low threshold for diagnostic testing. In the second situation, diagnostic testing is driven mainly by the pretest probability of GVI (i.e., the location and mechanism of the injury). Figure 90.3 depicts a diagnostic management algorithm for suspected traumatic aortic injury.

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**BOX 90.1 Signs and Symptoms of Great Vessel Injury**

<table>
<thead>
<tr>
<th>Signs</th>
<th>Symptoms</th>
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<tbody>
<tr>
<td>Hemorrhagic shock</td>
<td>Tearing pain</td>
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<tr>
<td>• Hypotension</td>
<td>Pain radiating to the back</td>
</tr>
<tr>
<td>• Tachycardia</td>
<td>Difficulty breathing</td>
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<tr>
<td>• Altered mental status</td>
<td>Vaginal complaints</td>
</tr>
<tr>
<td>• Pallor</td>
<td>Vague chest-related complaints</td>
</tr>
<tr>
<td>• Diaphoresis</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Neurologic complaints</td>
</tr>
<tr>
<td>Dyspnea</td>
<td></td>
</tr>
<tr>
<td>Asymmetric pulse pressure</td>
<td></td>
</tr>
<tr>
<td>Vascular bruits</td>
<td></td>
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<tr>
<td>Focal neurologic findings</td>
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</tbody>
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**Fig. 90.2 Continuum of aortic vessel injury.**

A. Normal aortic cross-section.

B. Isolated intimal injury.

C. Intimal injury with contained hemorrhage.

D. Aortic wall rupture.

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**Fig. 90.3** Depicts a diagnostic management algorithm for suspected traumatic aortic injury.
### BOX 90.2 Pros and Cons of Imaging Modalities for Great Vessel Injury

#### Chest Radiography
**Pros**
- Inexpensive
- Performed at the bedside
- Easy to interpret

**Cons**
- Nonspecific
- False-negative rate of 7% to 10% for traumatic aortic injury

#### Computed Tomographic Angiography
**Pros**
- Identifies mediastinal hematoma and differentiates its causes
- Identifies aortic injury, including intimal tears
- Sensitivity and specificity approaching 100% for traumatic aortic injury

**Cons**
- Poor delineation of nonaortic vascular injuries
- Requires relative hemodynamic stability to obtain

#### Aortography
**Pros**
- Traditional “gold standard”
- Beneficial in the diagnosis of branch vessel injuries
- Delineates equivocal computed tomographic angiographic findings

**Cons**
- Difficult to obtain on an emergency basis
- Requires relative hemodynamic stability

#### Transesophageal Echocardiography
**Pros**
- May be performed at the bedside
- Not limited by body habitus

**Cons**
- Poor availability on an emergency basis
- Contraindicated in patients with an unstable cervical spine or suspected esophageal trauma

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The chest radiograph is the initial diagnostic screening tool in patients with history of chest trauma. An upright, posteroanterior view provides the best evaluation of the mediastinum. In patients with concern for spinal injury, a portable supine, anteroposterior view is commonly performed. Note that in the recumbent position the mediastinum may appear artificially widened and hemothorax can be obscured.

Chest radiography is not diagnostic of GVI; rather, it is used to identify any of the multiple findings suggestive of aortic injury. **Box 90.3** reviews the classic findings on chest radiographs.
radiography associated with GVI; a widened superior mediastinum (50% to 92%), increased mediastinal width (67% to 85%), and indistinct aortic knob (21% to 24%) occur with the greatest frequencies. An additional benefit is its ability to identify concomitant or alternative injuries. Limitations of CTA of the chest for GVI include poor delineation of nonaortic vascular injuries and the need for relative hemodynamic stability in the patient.

Because of the ease and accuracy of CTA, use of the traditional “gold standard” for traumatic aortic injury, aortography, has markedly diminished over the past decade. In fact, newer studies are questioning the role of aortography even in the setting of equivocal CT findings. Several studies have shown that catheter angiography following equivocal findings on CTA is unlikely to reveal GVI, which has led some authors to suggest that patients be monitored clinically with or without repeated cross-sectional imaging, thus obviating the need for aortography.

Transesophageal echocardiography (TEE) is another modality that can be used to evaluate aortic injury. Availability reduces its usefulness in the emergency setting, but it should be considered for patients who are too unstable to leave the department or when body habitus prohibits the use of a CT scanner. Its use is contraindicated in patients with unstable cervical spine injuries or suspected esophageal trauma. TEE has similar sensitivity as CTA of the chest in the evaluation of traumatic aortic injury.

**TREATMENT**

## INITIAL INTERVENTIONS AND PROCEDURES

When great vessel trauma is suspected, adequate vascular access should be obtained immediately. Although two large-bore peripheral intravenous lines are frequently cited as being sufficient for trauma patients, the benefits of central venous pressure monitoring and rapid large-volume resuscitation may necessitate central access. Ideally, the suspected vascular injury should not be affected by the site chosen for central venipuncture. For example, with penetrating injury involving the descending aorta, concomitant vena cava injury is possible; solely femoral or lower extremity access would therefore not be optimal. Unfortunately, in the setting of undifferentiated thoracic trauma, the options may be limited.

Many of the initial emergency resuscitative efforts needed in patients with great vessel trauma are dictated by hemodynamic instability. Patients with traumatic cardiac arrest or extreme hemodynamic compromise unresponsive to crystalloid and packed red blood cell transfusions are candidates for emergency department resuscitative thoracotomy. This procedure is usually performed without knowledge of the exact injury. Consequently, it must be executed methodically such that all potential lifesaving interventions are performed. Injuries to the ascending aorta or aortic arch mandate manual pressure for control of hemorrhage, whereas injuries to the descending aorta require cross-clamping above the site of injury until stability and repair can be achieved. Injuries to aortic arch branch vessels may be tempered by packing the apex of the injured hemithorax. Suspected injury to the right hemithorax requires extension of the thoracotomy into the right side of the chest. Right-sided thoracotomy could be

### BOX 90.3 Radiographic Findings Associated with Great Vessel Injury

- Superior mediastinal widening
- Indistinct aortic knob
- Left pleural effusion or hemothorax
- Left apical cap
- Deviation of the trachea
- Deviation of the nasogastric tube to the right
- Depressed left mainstem bronchus
- Narrowing of the carinal angle
- Opacification of the aortopulmonary window
- Widening of the left or right paraspinal stripe
- Sternal or rib fractures

Data from references 3, 5, and 12.

![Fig. 90.4 Chest radiograph demonstrating the three most common findings of traumatic aortic injury: widened superior mediastinum (A), increased mediastinal width (B), and obscured aortic knob (C).](image)
considered in the rare incidence of an isolated transthoracic right-sided penetrating injury.

Identification of pulmonary hilar injury or excessive bleeding from deep in the thorax, despite aortic cross-clamping, suggests the need to clamp the affected pulmonary hilum. This is achieved either through manual compression or with a vascular clamp. The goal of emergency department thoracotomy is to achieve enough clinical stabilization to allow definitive operative repair.

DEFINITIVE TREATMENT

Definitive treatment of GVI is usually surgical or endovascular repair. With mortality rates associated with these injuries increasing at an estimated rate of 1% per hour over the 48 hours after arrival at the hospital, expediting time to intervention is imperative. Surgical techniques usually include clamping and aortic reconstruction with or without vascular bypass. In part because of the need for vascular clamping, paraplegia can be a complication of repair in up to 4% to 20% of patients. Although cross-clamp times are not thought to directly correlate with the incidence of paraplegia, keeping times under 30 minutes is believed to be beneficial.

More recently, endovascular stenting has become an alternative to open surgical repair. Potential benefits of endovascular repair include a less invasive approach and avoidance of aortic cross-clamping (which may lead to lower rates of paraplegia), cardiopulmonary bypass, and systemic heparinization. Endovascular stenting can be performed in less time and in patients too unstable for operative intervention. A meta-analysis of endovascular versus open repair published in 2008 showed lower mortality rates and lower risk for paraplegia with endovascular repair. It is thought that the mortality benefit relates to the less invasive approach and lack of systemic heparinization given that the majority of these patients have concomitant intraabdominal, intracranial, or intrapulmonary injuries at high risk for bleeding complications.

Before surgical or endovascular intervention, medical management is critical and should include pharmacologic reduction of wall tension and shearing forces to prevent propagation of intimal tears and minimize the risk for subsequent catastrophic rupture of a contained hemorrhage. One study demonstrated that by starting pharmacologic management as soon as possible, even before a confirmative diagnostic test when suspicion is high enough, morbidity and mortality were reduced significantly.

Beta-blockers are the first-line medication for reducing wall stress and controlling the heart rate. Esmolol is ideal because of its rapid onset of action and short half-life, which makes it easy to titrate in a continuous infusion. When further blood pressure control is desired, vasodilators such as nitroprusside can be added after beta-blockade has been established. Because of the potential for reflex tachycardia, which increases shearing forces, caution should be exercised when using nitroprusside alone.

Table 90.1 lists drug dosages for esmolol and nitroprusside.

A second but controversial intervention is permissive hypovolemia, in which blood pressure is controlled by limiting fluid administration. Lower systolic pressures of 60 to 90 mm Hg are thought to decrease the risk for clot rupture and minimize the shear force on traumatized vessels. Patients often have associated pulmonary contusions, which strengthens the rationale for limiting fluid administration before operative intervention. Animal studies using permissive hypovolemia have shown consistent benefit, whereas human trials have been few and the results conflicting.

Two other interventions that are often used during global resuscitation require special consideration in the setting of GVI. Central line access provides important information during resuscitative efforts; however, caution must be taken to avoid further vessel damage by choosing a site farthest from the suspected vessel injury. Similar caution must be undertaken when considering chest tube placement to resolve hemothorax. This action may disrupt the containment of a great vessel hemorrhage with the catastrophic result of exsanguination of the patient.

Ideally, immediate surgical or endovascular repair of GVI has been recommended. However, recent studies have looked at delayed repair, particularly in patients with significant associated injuries or hemodynamic instability. Repair of great vessels has been delayed as long as 6 to 8 months. One prospective multicenter study in 2009 examined delayed repair of blunt traumatic aortic injury in stable patients and found significant survival benefits in all patient groups, particularly striking in those with associated injuries. Delayed repair is not routinely performed yet but may become more prevalent as further studies focus on this approach.

If a patient survives the first 24 hours without rupture, a stable pseudoaneurysm may develop. In such cases nonoperative management, including close monitoring and pharmacologic control of blood pressure, may be considered.

Table 90.1 Medical Management of Great Vessel Injury

<table>
<thead>
<tr>
<th>DRUG</th>
<th>DOSE</th>
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<tbody>
<tr>
<td>Esmolol*</td>
<td>Bolus 0.5 mg/kg over 1-min period</td>
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<tr>
<td></td>
<td>Continuous infusion 50-200 mcg/kg/min</td>
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<tr>
<td>Nitroprusside</td>
<td>0.3-10 mcg/kg/min</td>
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</table>

*A beta-blocker is first-line treatment; nitroprusside should be added if blood pressure control is not achieved. The goal is systolic blood pressure between 100 and 120 mm Hg.

SUGGESTED READINGS


REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES