Thoracic Trauma

Injuries, Evaluation, and Treatment

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KEYWORDS

- Thoracic trauma • Penetrating • Blunt • Primary survey
- Emergency department thoracotomy

KEY POINTS

- A focused primary survey is essential in the diagnosis and rapid treatment of life-threatening injuries in thoracic trauma.
- Thoracic trauma can uniquely benefit from bedside procedures (eg, tube thoracostomy, emergency airway creation, emergency department thoracotomy) to alleviate immediate threat of mortality and allow further definitive treatment.
- Patient stabilization is necessary before definitive surgical intervention is attempted.
- A thorough secondary survey in combination with diagnostic laboratory and radiologic studies will uncover most traumatic injuries.

INTRODUCTION

Thoracic injury is a common and potentially devastating component of acute trauma care. The incidence of such injury is 14% in blunt trauma and 12% in penetrating. Yet, thoracic injuries account for up to a quarter of early trauma-related mortality, second only to head and neck insults.[1,2] Despite the often serious nature of thoracic trauma, many of these injuries are can be quickly diagnosed, and at times mitigated, in the trauma bay.[3]

PRIMARY SURVEY

The first priority in management of thoracic trauma is evaluation and stabilization of airways, breathing, and circulation (ABC).[4] This initial primary survey encompasses urgent assessment of the airway, quality of respiration, and stability of circulatory status. When any of these factors is insufficient, urgent intervention must be performed and the primary survey reassessed. Although many trauma victims can benefit from basic

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interventions, emergent bedside surgical intervention is uniquely beneficial in thoracic trauma if performed promptly and appropriately (**Table 1**).

Airway

Assessment and stabilization of the airway is always the first priority in the management of all trauma patients. A great deal of assessment can be based on basic patient appearance and verbalization. However, in the presence of severe injury or diminished mental status, evaluation can be more difficult and should be focused on a combined assessment of oxygenation, ventilation, and airway protection.[4] Because the mortality of direct airway injury is so high, most of these patients do not reach the emergency room. As such, loss of airway in hospitalized trauma patients is generally due to secondary failure. Diminished mental status, head injury, cervical spine injury, soft tissue neck trauma, facial wounds, and clavicle fractures can all indirectly lead to loss of adequate airway protection.[2] Initial treatment must include cervical stabilization and proper head positioning with eventual definitive control by endotracheal intubation. If direct airway injury is suspected on primary survey, laryngoscopy can be considered; however, only with the understanding that it is time intensive. If the airway is truly compromised in the setting of suspected injury, an emergency airway is required. The standard method is via cricothyroidotomy due to its speed and ease. The technique can be used in a variety of settings with as little as a scalpel, clamp or bougie, and endotracheal tube. The primary contraindication to this procedure is direct neck injury at the position of the cricothyroid membrane. In this setting, tracheostomy is preferred because its distal location avoids an incision through traumatized soft tissue and reduces the risk of worsening a tracheal disruption.[3]

Breathing

Following, or paralleling, airway evaluation, the patient's respiratory status must be assessed. In the setting of traumatic thoracic injury, this step is of particular importance because significant compromise may be present and emergent bedside intervention may be indicated. Basic evaluations of respiratory rate, chest wall motion, oxygenation, and breath sounds are performed, as in any trauma. The physician must have a suspicion for pneumothorax in the setting of thoracic injury; in particular, there is concern for a life-threatening tension pneumothorax.

Table 1
Primary survey: life-threatening injuries and emergent treatments

Injury	Intervention
Airway obstruction or rupture	Intubation if possible and safe, consideration of cricothyroidotomy vs tracheostomy
Tension pneumothorax	Initial needle decompression followed by definitive tube thoracostomy
Open pneumothorax	Initial 3-sided occlusive dressing followed by wound closure and tube thoracostomy
Massive hemothorax	Volume resuscitation, tube thoracostomy, consideration of emergency thoracotomy
Pericardial tamponade	Pericardiocentesis if the patient is stable, consideration of emergency thoracotomy

A list of the common life-threatening thoracic traumatic injuries that can be diagnosed during the primary survey. If identified or even strongly suspected, the corresponding emergent intervention must be strongly considered.

Tension pneumothorax is the progressive accumulation of gas within the thoracic cavity to a point that positive pressure is exerted on the mediastinal and intrathoracic structures.[5] This circumstance can be secondary to chest wall trauma, a large bronchial or parenchymal injury, or a smaller lung injury exacerbated by positive pressure ventilation. Regardless of cause, the end result is the development of a 1-way valve; air enters the thoracic cavity without egress. By Boyle's law, the increased volume of air results in increased thoracic pressure, causing ipsilateral lung collapse, contralateral lung compression, mediastinal shift, and decreased venous return to the heart.[2] Tension pneumothorax can progress rapidly and diagnosis should not be delayed for imaging but rather made by clinical examination. As with all pneumothoraces, primary survey may reveal absent breath sounds, unequal chest rise, and hypoxia. However, signs unique to the tension variety include tracheal deviation (away from injury), jugular venous distension, and hypotension.[3] Many of the signs of tension pneumothorax are vague and nonspecific in the presence of other injuries. However, there is substantial morbidity if the pathologic condition is missed, so any suggestion of tension pneumothorax necessitates intervention during the primary survey. Initially, a partially occlusive dressing can be placed (open chest wall wounds) and needle decompression should be performed. This maneuver will allow time for reassessment of the ABCs; however, definitive treatment requires tube thoracostomy once the primary survey is complete.

Circulation

The final element of the primary survey is assessment of circulatory status. Again, with thoracic trauma, circulatory compromise can be profound and rapid. If any trauma patient presents with hypotension, the general assumption is that massive hemorrhage is the cause. However, in thoracic trauma, obstructive shock must also be considered. The deadly circulatory pathologic conditions that are associated with the most immediate morbidity and, therefore, must be dealt with promptly, are massive hemothorax and pericardial tamponade. Analysis of vital signs, pulse pressure, neck veins, and breath sounds may help identify life-threatening injuries. Increased suspicion should be present with near-midline trauma (between the nipples or, posteriorly, the medial scapular borders).[2]

Similar to the abdomen, pelvis, and lower extremities, the thoracic cavity provides a potential space large enough for patient exsanguination. Furthermore, massive hemothorax into this space is not only worrisome for potential large volume blood loss but also for possible tension effects on central venous return. Initial treatment of hemothorax is chest tube placement. This intervention is rapidly decompressive and allows for direct evaluation of hemorrhage volume. General teaching is that if the immediate chest tube output exceeds 1500 mL, or 200 mL per hour for 2 to 4 successive hours, the patient should proceed to the operating room for thoracotomy.[3] A rare but deadly complication associated with hemothorax is bronchovenous air embolism. When direct penetrating pulmonary trauma occurs, injury to both parenchyma and vasculature can result. The already low pulmonary venous pressures exacerbated by hemorrhage allows potential air uptake from injured parenchyma and subsequent air embolism into the left heart. Risk of this phenomenon increases as airway pressures increase. Although air embolism is rare, it should be suspected when rapid circulatory collapse occurs following the initiation of positive pressure mechanical ventilation.[6]

Pericardial tamponade may also produce rapid circulatory collapse following thoracic trauma. For hemothorax to cause significant compromise, substantial blood loss is required. In contrast, tamponade can occur with very little blood in the pericardial space. As this closed space progressively fills and applies pressure on the

pericardial contents, preload is initially compromised. If allowed to progress, end diastolic volume is impaired by means of ventricular compression. Signs of pericardial tamponade characteristically present in 3 phases. First observed is tachycardia and increased systemic vascular resistance, followed by decreased cardiac output, and finally complete circulatory collapse.[7] In the setting of severe injury, pericardial filling can be rapid and, therefore, collapse can occur without warning. Along with the previously described changes in vital signs, examination findings of muffled heart sounds, jugular venous distension, and pulsus paradoxus (large decrease in systolic blood pressure during inspiration) may be present in the setting of tamponade. However, though considered standard symptoms, all 3 are only present concurrently 15% of the time and individually are too vague to be accurately predictive.[8] Similarly, elevated intracranial pressure or pulseless electrical activity may be foretelling but are nonspecific. The increased use of ultrasound (US) in the emergency room has helped to alleviate some of the ambiguity in diagnosis, with a sensitivity of 96% and a specificity of 98% in detection. Though, even at bedside, the study requires valuable time and expertise.[9] It is critical, therefore, to have a high suspicion for pericardial tamponade any time thoracic trauma is present, particularly penetrating, in the setting of circulatory collapse.

If pericardial tamponade is recognized early enough (in the first 2 phases), pericardiocentesis can be performed as temporary decompression before definitive repair. If, however, the patient is already in obstructive shock, the patient requires an emergency department thoracotomy (EDT).

Whether for tamponade or hemorrhage, EDT consists of a left anterolateral thoracotomy in approximately the 5th intercostal space with or without extension across the sternum. Once the chest is open, the pericardium should be opened, hemothorax evacuated, lung distracted, and descending aorta clamped if appropriate (**Box 1**). The primary objectives are to release tamponade, control hemorrhage, evacuate air embolization, perform cardiac massage, and allow adequate resuscitation. Hemorrhage from proximal pulmonary vascular injury can be temporized by twisting the lung on its hilum until an appropriate clamp is placed. EDT is potentially life-saving but not without risk, so should only be performed for very specific indications. EDT only demonstrates significant benefit for those patients with a reasonable chance of neurologic

Box 1
Emergency department thoracotomy: important steps

- Fifth intercostal space incision from sternum to bed (when the patient is supine)
- Placement and opening of a rib spreader, dial, or crank on the posterior blade
- Evacuation of the thorax and identification or control of the hemorrhagic source
- Craniocaudal (parallel to bed) incision in pericardium with pericardial evacuation and delivery of the heart
  - Control of hemorrhage if appropriate
- Distraction of the lung with hilar torsion
- Incision of the pulmonary ligament, aortic isolation via blunt dissection, clamping of the aorta at the level of the diaphragm
- Internal cardiac massage, if appropriate
- Consideration of extension of the thoracotomy across the sternum (with the Lebsche knife) if concerned for right-sided injury.

recovery; therefore, it is used for patients who become pulseless in the trauma bay or those who continue to demonstrate pupillary response, spontaneous movement, organized electrocardiograph patterns, or cardiac activity on US despite pulselessness.[2] Furthermore, EDT is of highest benefit in penetrating trauma with an immediate survival rate of 35% for cardiac injury and 15% for all-comer penetrating injury. Conversely, patient outcomes are poor with EDT for blunt trauma, with only 2% survival for those in shock and 1% for those with loss of vital signs.[10,11] Regardless of the scenario, for an EDT to be effective it must be used as a damage control procedure with rapid pericardial decompression or hemorrhage control followed by expedited transfer to the operating room. Even after stability is obtained, patients with a high injury severity score will often benefit from simple packing, vacuum therapy, and delayed closure.[12]

SECONDARY SURVEY

Once immediately life-threatening injuries have been managed and the patient's ABCs have stabilized, a thorough head-to-toe examination must be performed. A full set of hematologic and metabolic laboratory studies should be sent. This secondary survey not only allows for a more complete picture of the patient's condition but also for the discovery of other nonemergent but possibly life-threatening injuries (**Box 2**).

Imaging

In addition to a thorough but expedited head-to-toe examination, a key component of the secondary survey is imaging. In most trauma situations, especially thoracic trauma, a portable chest radiography (CXR) is recommended. This rapid test can evaluate chest wall injuries, mediastinal trauma, pneumothorax, hemothorax, pulmonary contusion, and pleural effusions, as well as guide further management without the need to transport a potentially unstable patient. For similar reasons, US has become ever more prevalent in the trauma bay. The effectiveness of focused assessment with sonography for trauma (FAST) has been well-documented.[13] The emergence of extended-FAST (eFAST) makes US all the more useful in thoracic trauma. This bedside imaging study can diagnose pericardial effusions and aortic injuries in addition to the previously mentioned pathologic conditions. Studies have suggested that

Box 2
Secondary survey

Components

- Evaluation of glasgow coma scale
- Head-to-toe examination
- Basic laboratory studies
- CXR
- Consideration of eFAST
- Further imaging based on clinical suspicion

Injuries

- Potential life-threatening injuries: simple pneumothorax, hemothorax, rib fractures, sternal fracture, cardiac injury, aortic or major vessel trauma, pulmonary injury, tracheobronchial tree disruption, esophageal trauma, diaphragm rupture.

the effectiveness of US rivals computed tomography (CT) with certain injuries, sensitivity, and specificity nearing 100%. However, there are certainly disadvantages to this modality. First, US depends on the identification of fluid, or the effects of an injury, rather than the injury itself. Therefore, its guidance in treatment is not always clear. Furthermore, though some studies extol the predictive value of US, others cite sensitivities and specificities of less than 50%. This discrepancy is likely due to several factors associated with the modality, including variations in provider technique, imaging analysis not done by a radiologist, and no standardization of anatomic landmarks. Additionally, air in the thoracic or subcutaneous spaces can significantly limit effectiveness.[13]

The definitive imaging modality in chest trauma is CT. It does not offer the cost-effectiveness or speed of radiography or US but provides far superior resolution and scope, allowing the diagnosis of injuries that would otherwise fail to be recognized. With the use of intravenous (IV) or by mouth contrast agents, CT can further identify luminal trauma that formerly required angiography or endoscopy. The drawbacks of CT include the cost, radiation exposure, examination duration, and need for patient travel. If used only when injuries are suspected and imaging is likely to affect management, the first 3 disadvantages are negligible. The latter concern, however, can be significant and careful consideration of the patient's stability must be made before their removal from the trauma bay.

INJURIES AND MANAGEMENT

Pneumothorax

One of the most common pathologic conditions encountered with thoracic trauma is the nontension pneumothorax, with an estimated incidence of 20%.[14] Simple pneumothoraces occur as air slowly leaks from the lung parenchyma. Such leaks often occur as the result of direct penetration by missiles or fractured ribs. Pneumothorax can also occur secondary to alveolar rupture during rapid intrathoracic pressure changes. Findings of tachycardia, hypoxia, decreased breath sounds, or crepitus arise with pneumothorax. If the patient is symptomatic, tube thoracostomy is indicated. Minor pneumothoraces are often asymptomatic and often diagnosed on initial radiography or US. Given the 20% to 35% false-negative rate of supine CXR, small pneumothoraces are frequently only discovered after thoracic CT scan (**Fig. 1**).[2] The

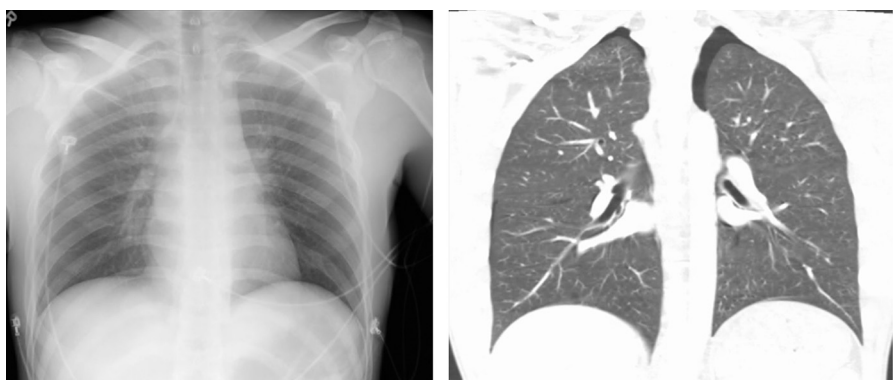

Fig. 1. Demonstration of a seemingly normal portable CXR (*left*) after motor vehicle collision but with evidence of occult bilateral apical pneumothoraces discovered on chest CT (*right*).

treatment of occult pneumothoraces, which occur in 2% to 8% of blunt traumas, is controversial.[15] Some studies conclude that tube thoracostomy is required for pneumothoraces with volumes greater than 5 times 80 mm, those involving more than 2 rib fractures, or in patients requiring positive pressure ventilation.[16,17] However, other studies argue that there is no correlation between these factors and the need for procedural intervention.[18] Aspiration has been described for the treatment of spontaneous pneumothorax and is being adopted in the trauma setting, whereas other physicians use oxygen therapy to aid in intrathoracic air reabsorption.[19] Although this latter treatment has been used for decades, it has been suggested that high oxygen tension therapy (>60%) is no more beneficial than standard low-flow nasal cannula.[20] It is unclear, therefore, what the optimal treatment regimen for small pneumothoraces should be. No matter the intervention, the most important step in management is close clinical monitoring for pneumothorax expansion.

Hemothorax

Intrathoracic hemorrhage often occurs in concurrence with pneumothorax. Nonmassive hemothorax occurs by injury to lung parenchyma, intercostal vessels, or other chest wall vessels. Hemorrhage is rarely large enough to cause immediate patient instability; however, failure to treat early can lead to interval development of empyema or fibrothorax. Small amounts of bleeding (<200–300 mL) are rarely detectable on physical examination or portable supine CXR. If there is concern for hemorrhage, CT allows for the identification of source, estimation of hemothorax volume, and evaluation for associated injuries.[21] Because of the complications associated with retained hemothorax, treatment requires drainage with tube thoracostomy. Although even early chest tube placement fails to evacuate the thorax in 5% of cases, this failure rate increases dramatically when treatment is delayed more than 24 hours after injury; prompt intervention is crucial.[22] For retained hemothoraces failing chest tube drainage, instillation of tissue plasminogen activator and deoxyribonuclease has been found 65% to 90% successful. This treatment does take multiple days, however, and is associated with reported fevers and pleuritic pain. The ultimate treatment of undrained hemothoraces is surgical decortication, whether by thoracotomy or video-assisted thoracoscopic surgery (VATS). Under direct visualization, surgical intervention allows for not only hematoma drainage but also thorough removal of organized collections and loculation debridement. VATS decortication has largely replaced traditional thoracotomy due to decreased long-term cost, length of stay, and chest tube days. However, these benefits are only reliable with careful patient selection. For patients with previous empyema, those who have undergone previous ipsilateral thoracic surgery, those who are hemodynamically unstable, those who cannot tolerate single-lung ventilation, or those who have an organized fibrothorax, open thoracotomy is still recommended.[23]

Rib Fractures

One of the most common injuries from blunt thoracic trauma is rib fracture. Though often considered minor and rarely requiring surgical intervention, these fractures can cause direct trauma to lung parenchyma or intra-abdominal organs. Rib fractures can also cause respiratory complications because significant pain impairs adequate pulmonary toilet. The presence of fractures is an independent risk factor for pneumonia and death in trauma patients.[24] Traditional treatment of rib fractures is supportive, consisting of respiratory therapy and multimodal analgesia, including epidural or paravertebral blocks. Rib plating or stabilization is controversial for most fracture patterns but has demonstrated some benefit when performed in the management of flail

chest. A flail segment occurs by disassociation of a portion of the chest from the surrounding chest wall. Diagnosis requires the fracture of 2 or more adjacent ribs in 2 or more locations. This disassociation leads to pain and disrupted respiratory mechanics but also indicates a high-energy traumatic mechanism that should raise suspicion for other injuries, particularly pulmonary contusions (**Fig. 2**).[3] The suggested treatment of flail chest has fluctuated throughout the years. Initially, external stabilization was recommended but high rates of complications pushed treatment toward internal fixation. As positive pressure mechanical ventilation improved and was able to provide effective internal splinting, surgical fixation fell out of favor.[2] However, over the past decade, several studies have demonstrated that, although surgical stabilization of flail chest does not improve overall mortality, there may be benefit for patients with refractory pain or respiratory compromise. Data show reduced length of stay, intensive care unit days, and time on mechanical ventilation.[25,26] Furthermore, longitudinal studies have suggested that patients have improved quality of life, pulmonary function, and pain control over the first year after traumatic flail chest if rib fixation was performed.[27]

Pulmonary Contusion

When it comes to primary respiratory compromise, though pneumothorax may produce the most immediately profound symptoms following thoracic trauma, pulmonary contusion may be the most destructive injury. Contusions are associated with 5% to 30% mortality and are directly causative in many of these deaths.[28] The diffuse energy dissipation of trauma across the pulmonary parenchyma and chest wall causes a combination of intraparenchymal hemorrhage, atelectasis, and consolidation. This form of injury is indolent, not noticeable on initial CXR or examination. Contusion begins to progress at 4 to 6 hours after trauma and develops maximal effect within 24 to 48 hours.[29] Radiography and CT imaging may help with initial diagnosis and allow for monitoring but they rarely change outcomes or allow prediction of eventual severity. Assessment of the patient's pressure of arterial oxygen to fractional inspired oxygen concentration ratio (Pao_2/Fio_2) is helpful in demonstrating the clinical severity of injury and guiding the need for mechanical ventilation but is not necessarily predictive.[2] Management of pulmonary contusion is supportive and consists of oxygen therapy, aggressive pulmonary toilet, fluid restriction, careful pain management, and appropriate diuresis. Early intubation and antibiosis have been studied but at this time are

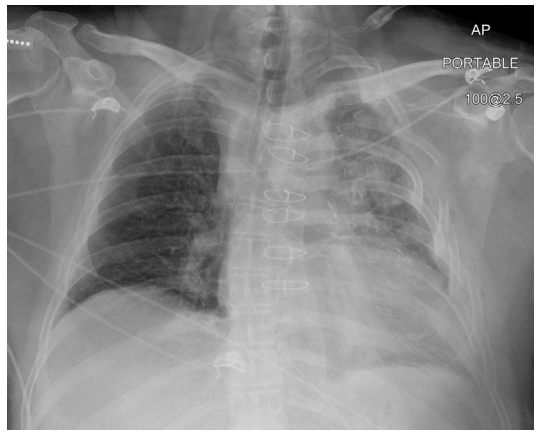

Fig. 2. Portable CXR after motorcycle crash displaying multiple left-sided rib fractures, including a flail segment, as well as the commonly underlying pulmonary contusions.

not recommended unless otherwise indicated. Unlike with penetrating trauma, surgical intervention for blunt pulmonary contusion is rarely indicated. In circumstances when parenchymal contusion is severe enough to produce large regions of necrosis, lobectomy can be performed to prevent infection or improve shunt; however, even after intervention, mortality is as high as 50%.[11]

Blunt Cardiac Injury, Pulmonary Vessel Injury, and Sternal Fractures

Blunt cardiac injuries are generally caused by direct sternal impact or rapid thoracic deceleration. Sheer forces, direct compression, and abrupt alterations in thoracic pressure all contribute to cardiac damage.[30] In its most severe form, free cardiac or, even less commonly, aortic valve rupture may occur. Though most patients with uncontained cardiac rupture die before reaching the hospital, it is important to be aware of the pathologic condition and its signs.[31] Because rupture most commonly occurs in the right heart, hemorrhage correlates to preload. Therefore, in the setting of hypotension, massive hemorrhage or tamponade may not be obvious but will present rapidly with initial trauma resuscitation. A similar presentation can occur when the neighboring proximal pulmonary vessels are injured. Trauma to this low-pressure vasculature may lead to either rapid hemorrhage into the pleural or pericardial space or, contrastingly, may produce few to no symptoms if contained. In severe cases, in which the patient is hemodynamically unstable, EDT and subsequent surgical repair is required; whereas, for stable ruptures and pseudoaneurysms, nonoperative management may be appropriate. Though pulmonary vessel injury can be devastating, it is relatively rare and there is, therefore, little consensus about its ideal treatment. Though complete cardiac and/or pulmonary vessel disruption can occur, most blunt cardiac trauma is milder and more insidious, only being discovered because of subtle secondary signs, such as new arrhythmia, murmur, or abnormal pulse pressure. The most common traumatic cardiac injury is cardiac contusion, a vague pathologic state describing focal myocardial hematoma or edema. Echocardiography may be helpful in diagnosis; however, electrocardiography is more sensitive, and able to detect associated arrhythmias or ST changes. Seventy-six percent of cardiac contusions are associated with sternal fractures. Because of this high correlation, if a fracture is present, the patient should be treated as if cardiac injury is also present.[32] Unless there is an associated effusion that would benefit from pericardiocentesis, treatment is supportive and primarily involves 24 to 48 hours of telemetry monitoring.[3] Cardiac biomarkers are of little value in the initial assessment of a cardiac injury; however, in trending, they may uncover coronary damage. Whether or not cardiac injury is present, sternal fractures are associated with high patient morbidity because of the force required to create such an injury. The degree of sternal displacement is correlated with the risk for further thoracic injury.[33] As with that of rib fractures, management is primarily supportive. However, if the fracture is unstable, pain is uncontrolled, or sternal infection is present, surgical intervention is required. Infected bone necessitates debridement and sternal closure can be achieved by sternal wires, plating, or both. If a significant defect remains, soft tissue flap coverage may be required.[2]

Blunt Aortic Injury

Although penetrating aortic trauma is notably deadly, blunt traumatic aortic injury (BTAI) can be similarly devastating. Second only to head trauma, aortic injury carries 1 of the highest associated mortality rates in all of blunt traumas.[34] Furthermore, incidence is not low; it is estimated that anyone who suffers more than a 10 foot fall or motor vehicle crash (MVC) at more than 40 mph is at risk for aortic trauma.[2] Eighty percent of aortic trauma occurs due to MVCs and can range from small intimal tears

to total aortic transection, from which 80% of patients suffer immediate death.[35] Disruption of the aorta in blunt trauma is generally due to rapid patient deceleration. Because the aortic arch is relatively free-floating in contrast to the tethered descending aorta, deceleration imparts significant sheer force at the site of transition, the ligamentum arteriosum. Hence, most blunt aortic injury occurs just distal to the left subclavian artery (**Fig. 3**).[36] Unless the injury is severe, signs and symptoms are often vague and aortic disruption is instead suspected based on the mechanism of trauma or the associated injury pattern, and is confirmed by radiographic studies. Chest CXR may suggest aortic disease through findings of hemorrhage such as hemothorax or mediastinal widening. Thoracic CT scan with IV contrast remains the gold standard, preferably using a trauma or angiogram protocol. In the unstable patient, transesophageal echocardiography (TEE) is also an option and further provides the opportunity for cardiac and valvular evaluation.

If aortic injury is confirmed, the initial goals of treatment are stabilization and prevention of further propagation. Anti-impulse therapy is the first line of treatment and is achieved using such agents as beta and calcium channel blockers. The functional effect is a decreased change in aortic pressure over time (dP/dT). Goals of therapy are based on maximal heart rate reduction while maintaining systemic perfusion.[37] Adequate anti-impulse therapy not only serves as definitive treatment in select patients but also decreases operative mortality should they subsequently require intervention for the aortic injury.[38] This subsequent intervention remains controversial. Traditionally, expeditious surgical repair was the standard of care. Surgical intervention remains the appropriate immediate treatment of an unstable trauma patient with aortic injury but is associated with up to 31% mortality and 9% incidence of paralysis.[39,40] Timing of intervention in the hemodynamically stable patient is less clear.[41] Decision-making is largely based on the risk of aortic free rupture (**Table 2**). Although this risk has been correlated with the size of intramural hematoma or contained rupture, injury propagation remains unpredictable (**Box 3**).[42] Regardless, recent analysis has demonstrated that delayed operative intervention is not only safe but is associated with a decreased mortality. Over the past decade, the average delay to intervention has gone from 15 hours to 55 hours with a 65% decrease in 30-day mortality.[43] The Eastern Association for the Surgery of Trauma (EAST) has supported these findings by formally endorsing delayed aortic intervention.[44]

In the past, a second controversy in the management of BTAI has been open versus endovascular surgical intervention. Currently, thoracic endovascular aortic repair

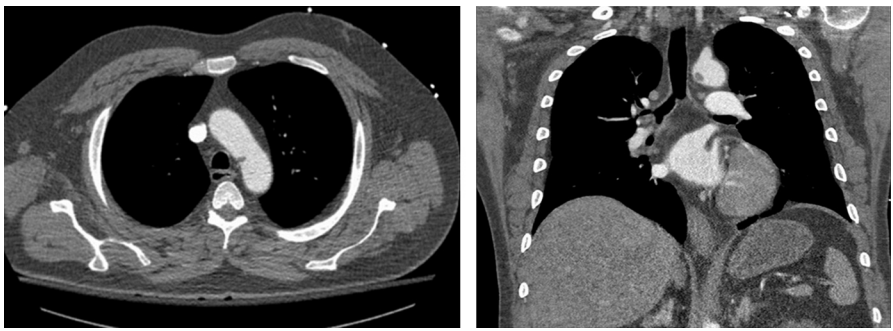

Fig. 3. Axial (*left*) and coronal (*right*) slices of a thoracic CT scan demonstrating a small thoracic aortic lesion after patient fall. Note that this lesion is in the classic location, adjacent to the left subclavian takeoff (seen best in the coronal slice).

Table 2
Classification of traumatic aortic injury

Grade	Description
I	Intimal tear
II	Intramural hematoma
III	Pseudoaneurysm
IV	Rupture

Graded classification of traumatic aortic injury severity. Treatment based on these grades continues to evolve. However, in general, low-grade injuries may be treated medically, moderate grade with delayed endovascular intervention, and high-grade with more urgent surgery.

(TEVAR) is the recommended treatment if anatomically feasible.[34] TEVAR demonstrates decreased rates of paraplegia, heparin-associated hemorrhage, and immediate mortality compared with open aortic repair (3.1% vs 17.8%).[45] Endovascular intervention, however, is not without its own risks. Despite a low early mortality, studies have confirmed late deaths with TEVAR, no decrease in the need for operative reintervention, closer required follow-up, and an overall complication rate nearing 20%. These complications are primarily stent infolding due to oversizing, endoleaks, and cerebrovascular accidents (CVAs).[35] Some of these complications are due to the 50% need for left subclavian coverage during stent placement. There are conflicting data on the risk of subclavian occlusion and, similarly, inconsistent opinions on management.[46] If staged intervention is feasible, pre-TEVAR carotid to subclavian bypass or subclavian transposition is possible. However, their benefit is questionable without clear improvement in post-TEVAR CVA or spinal cord ischemia. Furthermore, most analyses of subclavian coverage addresses nontraumatic injuries and so the applicability with aortic trauma is unclear.[47] With the increasing utility of TEVAR, enthusiasm about the possible use of resuscitative endovascular balloon occlusion of the aorta (REBOA) in BTAI has emerged. REBOA refers to the insertion of a balloon catheter into the aorta via the femoral artery. The balloon is inflated proximal to the aortic injury, halting further distal blood flow and hemorrhage. This new technology is promising; however, indications and criteria for REBOA still need to be better defined.[48]

Penetrating Cardiovascular Trauma

Penetrating cardiac, aortic, and other large vessel trauma can be devastating with rapid hemodynamic compromise. When not immediately fatal, severe injuries often

Box 3
Aortic rupture: risk score criteria

- Lactate >4 mM/L
- Mediastinal hematoma greater than 10 mm
- Lesion to normal aortic diameter ratio greater than 1.4

High risk for aortic rupture after initial traumatic nonrupture injury when more than 1 factor is present.

From Harris DG, Rabin J, Kufer JA, et al. A new aortic injury score predicts early rupture more accurately than clinical assessment. J Vasc Surg 2015;61:532–3; with permission.

result in massive hemothorax or tamponade and require EDT followed by prompt transfer to the operating room. However, in a minority of patients, penetrating trauma causes occult injuries that are only identified during diagnostic workup. Pericardial effusion or hemothorax on radiography may suggest cardiovascular injury but are nonspecific. FAST, TEE, and multidetector CT, on the other hand, are able to identify even stable or contained injuries.[49] Although the management of injury to thin-walled vasculature, such as the inferior vena cava and pulmonary vessels, is controversial, treatment of nonexpanding aortic injuries in the stable patient can be managed nonsurgically. These insults can treated similarly to small blunt cardiovascular injuries, with hemodynamic stabilization, anti-impulse therapy, close monitoring, and nonemergent endovascular intervention.

Tracheobronchial Tree Disruption

Major tracheobronchial injury in trauma is rare but when present is associated with an 80% mortality in the field. Penetrating tracheal injury is often cervical in location and easily detected. In contrast, most tracheobronchial injury suffered from blunt trauma is near the carina and can be more difficult to diagnose.[50,51] Patients may demonstrate dyspnea, dysphonia, pneumomediastinum, pneumothorax, or subcutaneous emphysema (**Fig. 4**). Although these signs can also be attributed to other pathologic conditions, together these findings should raise suspicion for tracheobronchial tree injury.[52] Initial diagnostic studies should include CT scan to evaluate for mediastinal hematoma, pneumomediastinum, or tracheal deviation; however, a negative study does not obviate bronchoscopy if tracheobronchial trauma is suspected.[53] If injury is discovered, treatment depends on the severity. Nonoperative management is appropriate for injuries less than 4 cm, with viable airway tissue and an absence of associated esophageal damage or respiratory compromise.[54] For those injuries that do not meet nonoperative criteria, surgical intervention depends on location and extent of disruption. The proximal two-thirds of the tracheobronchial tree can be approached via a low collar incision, which can be extended through the manubrium for improved

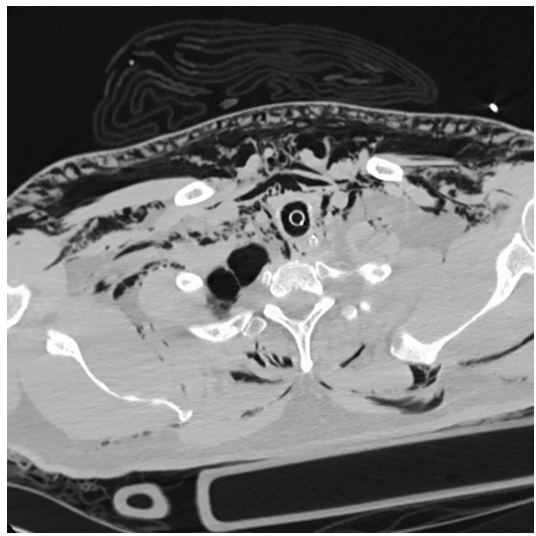

Fig. 4. CT chest of a trauma victim with evident subcutaneous emphysema and mediastinal air tracking superiorly into the anterior chest wall and neck.

distal access. The distal third of the trachea and the intramediastinal main stem bronchi, on the other hand, are more easily exposed by right posterolateral thoracotomy.[55] Once adequate exposure is obtained and nonviable tissue is debrided, simple lacerations can be repaired directly with interrupted absorbable sutures. Larger defects, however, may require pedicle flap closure from the pericardium, parietal pleura, intercostal muscles, or infrahyoid muscles. To further protect the newly repaired tracheobronchial tree, some surgeons use a guardian stitch, or stitch from the chin to the chest, to ensure neck flexion and, therefore, decreased tracheal tension for the first postoperative week.[2] Despite all efforts, complications after tracheobronchial surgery are not infrequent with stenosis and/or dehiscence occurring in 3% to 6% of cases.[56,57] Symptoms of these complications are often subtle, consisting of dyspnea, wheezing, and stridor, and are regularly not discovered until 2 to 4 weeks postoperatively. If symptoms necessitate urgent intervention, bronchoscopic dilation can relieve stenoses but definitive surgical reconstruction may be required in a more elective manner.[58]

Esophageal Trauma

Esophageal injury is rare, occurring in 2% of penetrating and well less than 1% of blunt traumas; however, it is associated with a mortality rate of 20% to 40%.[59] Management follows that of more common nontraumatic causes; however, location of injury is typically more proximal and diagnosis is often delayed. Symptoms can be subtle and often overlap with other cervical injuries. Signs of abscess and mediastinitis are more specific but often slow to present.[60] Similarly, initial CXR and CT scan may demonstrate subcutaneous emphysema or pneumomediastinum but are not sensitive modalities for diagnosing esophageal injury. The gold standard diagnostic modality is the complementary use of esophagram (fluoroscopic or CT) and esophagoscopy, each alone missing 15% of injuries but achieving near 100% sensitivity in combination. Traditionally, surgical intervention was recommended if the injury was diagnosed within 24 hours, whereas medical therapy was recommended for delayed diagnoses. This protocol is changing, however, and early intervention with source control and debridement is recommended.[2] Furthermore, primary repair should be attempted not based on timing but rather tissue viability. Pleural, pericardial, diaphragmatic, or muscle flap reinforcement can be used to aid in tissue protection as can endoscopically placed negative pressure therapy.[61] When the esophageal injury is truly too large to repair, resection and diverting esophagostomy may be required until definitive interposition or conduit creation can be performed. Endoscopically placed esophageal stent therapy is also emerging as a viable treatment modality. Stents have demonstrated 90% effectiveness with iatrogenic esophageal injuries; however, it is unclear if these data are applicable to traumatic disruptions.[62]

Diaphragmatic Injury

Diaphragmatic injury is among the most difficult diagnoses to make in a traumatic setting. Incidence is unclear but is estimated to be anywhere from 0.6% to 24%.[2] Diaphragmatic trauma can be direct from a rib or weapon but can also be due to rapid pressure changes in the abdomen and thorax. Occasionally, diagnosis is aided when bowel is discovered in the thorax on diagnostic imaging. However, the liver and spleen commonly prevent acute herniation of the hollow viscus. Because of this, diagnostic imaging is considered inaccurate, exemplified by sensitivities of CT scan of 78% and 50% with left and right diaphragmatic injuries, respectively.[63] Additionally, symptoms are often vague, relating to injuries of neighboring structures, such as the lungs, spleen, and liver. Often the diagnosis of diaphragmatic trauma is made

intraoperatively when intervening for another cause. When an injury is discovered, primary repair can usually be accomplished, and can be performed either from an abdominal or a thoracic approach. If the defect is too large to close primarily, placement of mesh, or cadaveric or bovine graft, is appropriate.

SUMMARY

Thoracic injury is common in both high-energy and low-energy trauma and is associated with significant morbidity and mortality. As with the management of all traumas, thoracic trauma evaluation requires a systematic approach, first prioritizing airway, respiration, and circulation, followed by a focused secondary survey. Unlike other forms of trauma, chest injuries have the potential to progress rapidly and require prompt procedural intervention in the emergency room. Because of this urgency, physicians must have a high level of situational awareness and pay close attention to physical examination findings. Once the primary survey is complete and stable, the secondary survey is crucial in uncovering most thoracic injuries and guiding effective care. The specifics of this care have undergone evolution throughout the recent decades, but an overarching emphasis remains on patient stabilization before definitive surgical repair.

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