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Blunt thoracic trauma: flail chest, pulmonary contusion, and blast injury

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Thoracic trauma is common and causes a variety of injuries, ranging from simple abrasions and contusions to life-threatening insults to the thoracic viscera. Thoracic trauma also is associated with a high morbidity. Twenty percent of all trauma deaths involve chest injury, making it second only to head and spinal cord injuries [1]. Fortunately, most thoracic injuries do not require major surgical intervention. Most chest wall and intrathoracic injuries can be managed with simple tube thoracostomy, mechanical ventilation, aggressive pain control, and other supportive care. The elderly and other patients with diminished pulmonary reserve are the most vulnerable for pulmonary deterioration and will require critical care observation at the least. Because critical care physicians will encounter patients with pulmonary and chest wall injuries frequently, an in-depth knowledge of the pathophysiology and treatment of thoracic trauma is necessary.

Mechanisms of injury

The overwhelming etiology of blunt chest trauma in the United States is the endemic motor vehicle crash (MVC). Although motor vehicle travel has become safer with each successive decade, 44% of the approximately 98,000 unintentional injuries in the United States in 2001 were caused by MVC [2]. There is a disabling injury from a MVC every 14 seconds. Additionally, there is an estimated 7% risk of a serious thoracic injury with any MVC, and in the United States, at least 1500 patients a day present with a life-threatening thoracic injury from MVC alone [2,3]. Falls from height, work or recreational-related crush injuries, and assaults are less common but substantial additional causes [4].

The three types of blunt force that lead to thoracic injury are compression, shearing, and blast. Thoracic compression injuries such as rib fractures occur when

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the applied force exceeds the strength of the thoracic cage. The area of maximal chest wall weakness is found at a 60° rotation from the sternum, where the ribs are flatter and less well-supported [5]. Frequently, however, ribs subjected to lateral or anteroposterior (AP) compression will fracture in two places: once at approximately 60° and again posteriorly [6]. AP compression also can create a costochondral disruption, resulting in a sternal flail.

Shearing forces cause soft tissue and vascular injury. In response to a rapid acceleration or deceleration, soft tissue and vascular organ movement is restricted at anatomic and developmental attachments. Ultimately, if the tensile strength of the attached tissue is exceeded, tearing or rupture will occur. This inertial effect is responsible for one of the most lethal thoracic injuries: aortic transection. Because the aorta is tethered by the ligamentum arteriosum and by the vertebrae below, the junction of the more mobile aortic arch and the stationary descending aorta is the most common site of disruption. Both full thickness tears with free rupture and

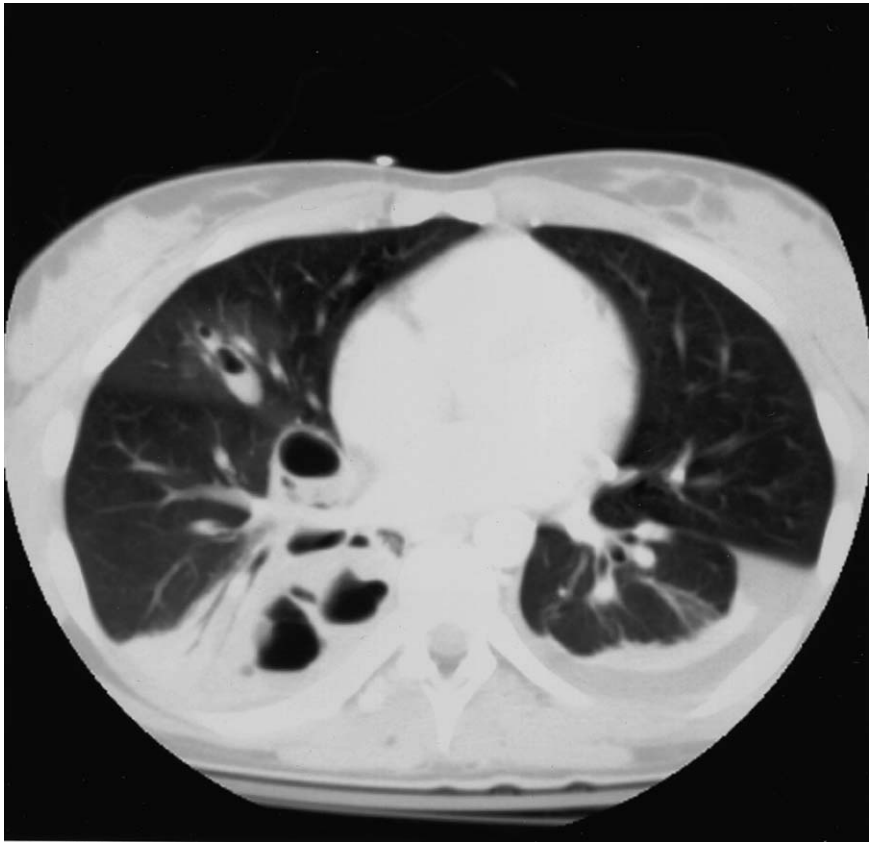

Fig. 1. Posterior right traumatic pneumatocele formation following blunt thoracic trauma.

partial thickness injuries leading to pseudoaneurysm formation are possible results. Shearing within the pulmonary parenchyma can lead to laceration, hematoma, contusion, or pneumatocele (Fig. 1).

Explosions are especially deadly, not only because of the blast pressure wave, but also because the victim can be launched considerable distances, and surrounding debris become missiles. Primary pulmonary blast injury occurs when the pressure wave strikes the chest wall and creates a pressure differential at the air–tissue interface. The greater the pressure differential, the more force is transmitted to the lung. The severity of the pulmonary injury is related inversely to the victim's distance from the blast [7]. Explosions within an enclosed space are more severe, because the pressure waves are reflected back to the patient, intensifying the original insult. The characteristic pathology of pulmonary blast injury is a contusion with edema and alveolar hemorrhage [8,9]. Secondary blast injuries result from objects sent into motion by the explosion, impacting the patient. Tertiary injuries are caused by the individual being displaced. Associated injuries caused by thermal burns, inhalation agents, and crush secondary to structural collapse are also common.

Rib fractures

Although rib fractures are common, it is difficult to determine the true prevalence among seriously injured patients, because the anteroposterior radiograph is not exceptionally sensitive for rib fractures [4,10–15]. In addition, national surveys of injured patients usually only track the three most principle diagnoses per patient, and rib fractures, in spite of their clinical importance, may not be included in the principle diagnoses of multiply injured patients. For example, the Health Care Cost and Use Project's Nationwide Inpatient Sample indicates that only 140,000 patients with rib fractures were admitted to United States hospitals in 2000 [16]. The actual number of patients with rib fractures seen in United States hospitals and ICUs may be significantly higher.

Rib fractures are clinically important for three reasons: as a marker for serious intrathoracic and abdominal injury, as a source of significant pain, and as a predictor for pulmonary deterioration, particularly in the elderly. Case series of rib fracture patients presenting to trauma centers reveal that 84% to 94% of patients will have significant associated injuries [4,10]. The most common associated thoracic injuries are pneumothorax, hemothorax, and pulmonary contusion [4,10,15]. The most common abdominal organs injured are the liver and the spleen. Patients with right-sided rib fractures, including the eighth rib and below, have a 19% to 56% probability of liver injury, while left-sided fractures have a 22% to 28% probability of splenic injury [12,17].

Contrary to historical beliefs, rib fractures, including those of the first and second ribs, are not associated statistically with aortic injury [18,19]. In fact, many trauma surgeons are recommending chest computed tomography (CT) angiography as a screening tool for occult intrathoracic injury in patients with significant

blunt chest trauma irrespective of chest radiograph findings [20,21]. Eight percent of patients brought to a trauma center following a high-speed MVC, a fall greater than 4.5 m, or having been struck by an automobile and thrown more than 3 m had aortic injury revealed by chest CT angiography [21]. Sixty-five percent of patients with significant blunt chest trauma who have an admission chest CT will have significant intrathoracic injuries that are missed by chest radiograph alone. [20]

The presence of rib fractures is especially ominous in children and the elderly [22,23]. The bones of children lack calcification; consequently, their chest walls are more compliant than adults'. Rib fractures in a child indicate a much higher absorption of energy than would be expected in an adult. Correspondingly, the absence of rib fractures in a child should not diminish concern for significant intrathoracic injury. In a study of 986 pediatric patients with blunt chest trauma, 2% had significant thoracic injuries without evidence of any chest wall trauma [15].

Thirty-eight percent of children with pulmonary contusion do not have radiographic evidence of rib fractures [14]. In contrast, minimal trauma rib fractures (ie, ground level falls) comprise 12% of all skeletal fractures in the elderly and are as common as humeral fractures [24,25]. Osteoporosis, loss of muscle mass, and comorbidities not only decrease the force required to cause rib fractures in the elderly, but they also decrease the physiologic reserves present to tolerate such injuries. Bulger et al compared patients who were at least 65 years old to a matched cohort of 18- to 64-year olds who sustained blunt chest trauma with rib fractures [13]. The elderly group had twice the mortality and thoracic morbidity. The risk of pneumonia increased by 27%, and mortality increased by 19% for each additional rib fracture in the elderly group.

Pain management is crucial to treating the patient with rib fractures [26]. Options include oral or intravenous (IV) nonsteroidal anti-inflammatory agents (NSAIDS), oral and IV narcotics, intercostal nerve blocks with local anesthetics, intrapleural catheters, and thoracic epidurals. Oral narcotics are rarely effective for pain control during the acute phase, and IV narcotics suffer from the adverse effect of respiratory depression. Although intercostals nerve blocks are effective, they are not an attractive option, because they require reinjection every 4 to 6 hours. Continuous thoracic paravertebral infusion of bupivacaine has been shown in an observational study to provide adequate pain relief and improved respiratory parameters compared with baseline measurements, and this benefit was sustained for 4 days [27]. Once the thoracic spine has been cleared radiographically, thoracic epidural catheter infusion of narcotics and local anesthetics acting synergistically is an excellent means to obtain pain control without sedation. Wisner reviewed 307 patients over the age of 60 and found the use of epidural analgesia was an independent predictor of decreased mortality and decreased incidence of pulmonary complications [28].

Flail chest

Flail chest is rare, but it is the most serious of the blunt chest wall injuries (Fig. 2). The prevalence of flail chest among patients with chest wall injury is estimated

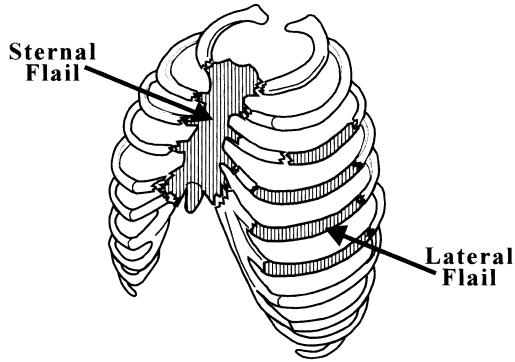

Fig. 2. Two types of flail chest: sternal and lateral. (*From* Mayberry JC, Trunkey DD. The fractured rib in chest wall trauma. Chest Surg Clin N Am 1997;7:239–61; with permission.)

between 5% and 13% [1]. The diagnosis of a flail chest is established most readily by observing the paradoxical motion of the affected segment in a spontaneously breathing patient. Upon inspiration, the flail segment is pulled inward by the negative intrathoracic pressure. With exhalation, the positive pressure forces the segment to protrude outward (Fig. 3). Muscular splinting of the chest early in the postinjury period, however, may mask the paradoxical motion until the flail becomes apparent hours later with the development of fatigue. In patients who are mechanically ventilated, a high degree of suspicion with palpation of the chest wall

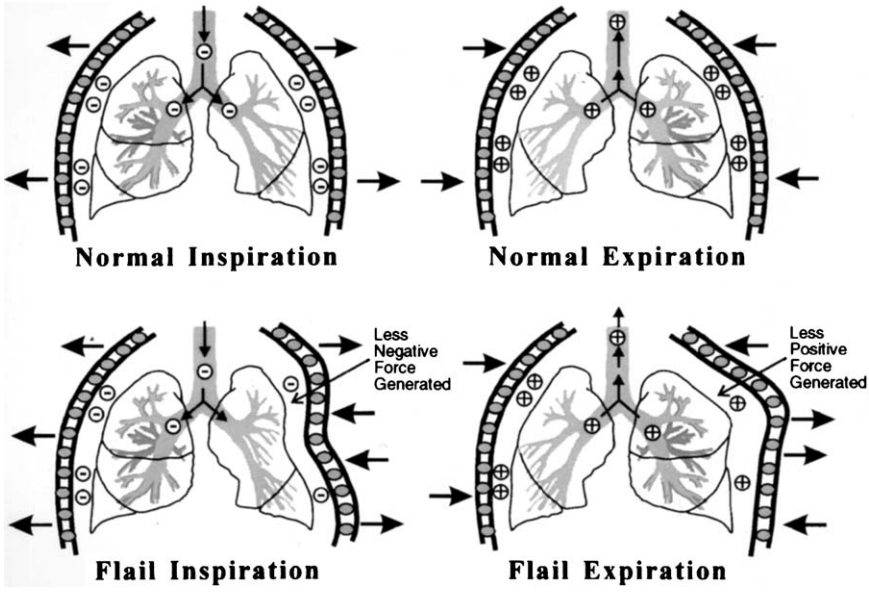

Fig. 3. Flail chest physiology. (*From* Mayberry JC, Trunkey DD. The fractured rib in chest wall trauma. Chest Surg Clin N Am 1997;7:239–61; with permission.)

for crepitance and fractures and review of chest radiograph or CT are necessary. Placement of the patient on a low level of pressure support (ie, 5 cm of water) may unmask the flail segment.

By definition, fractures of at least four consecutive ribs in two or more places are required to produce a clinically significant impairment of respiratory function; however, the patient's comorbidities and age greatly influence the clinical effect. Beyond the age of 55, the likelihood of death in cases of flail chest increases 132% for every 10-year increase in age and 30% for each unit increase in injury severity score [29]. In nonintubated patients, the disruption of chest wall mechanics will dramatically decrease tidal volume and effective coughing with a corresponding predisposition to sputum retention, atelectasis, and pneumonia. An associated pulmonary contusion further contributes to the development of bronchial obstruction and intrapulmonary shunting. A low threshold for intubation of patients with flail chest, especially those with comorbidities and the elderly, is warranted. Sankaran et al reported that early intubation in patients 30 years or older with moderate-to-severe injury resulted in 6% mortality, but withholding intubation for 24 hours until they developed clinical evidence of hypoxia or hypercapnia resulted in greater than 50% mortality [30].

Treatment

Fortunately, 68% of patients with flail chest requiring intubation are extubated by the third day [31]. Patients with more severe pulmonary dysfunction expected to require prolonged ventilatory support may be considered for tracheostomy. Postural therapy including rotational therapy and intermittent therapeutic bronchoscopy may be beneficial also. The ventilation mode may influence weaning in a patient with persistent chest wall instability. Tzelepis et al demonstrated that modes of ventilation, such as intermittent mandatory ventilation (IMV), which require the patient to have a significant number of unassisted breaths, increase the paradoxical chest wall displacement and the work of breathing more than modes that include a high-flow continuous positive airway pressure (CPAP) component [32]. CPAP apparently works to splint the paradoxical segment throughout the ventilatory cycle.

Selected patients with flail chest may be considered for operative stabilization. Pain control, restoration of hemithorax volume loss, and failure to wean from mechanical ventilation are the most common reasons cited [33–37]. A nonrandomized comparison of patients with flail segments undergoing operative stabilization and patients with flail segments without repair found that the duration of time on the ventilator could be diminished from a mean of 26.7 days to 6.5 days [33]. The same study, however, indicated that patients with an accompanying pulmonary contusion probably do not benefit. A similar series comparing internal fixation (Kirschner wires) with mechanical ventilation found that the former required 1.3 days of intubation and that the latter required 15 days of intubation [34]. Both of these studies found shorter ICU length of stay, rates of pneumonia, and mortality in patients treated with operative stabilization.

Pulmonary contusion

Pulmonary contusion should be anticipated in any patient who sustains significant, high-energy blunt chest impact. A history of the inciting event and physical findings of chest wall trauma, especially the presence of fractures or a flail segment, increase the odds of having an underlying lesion. The absence of rib fractures, however, does not eliminate the possibility of pulmonary contusion. Focal or diffuse homogeneous opacification on chest radiograph is the mainstay of diagnosis. Unlike aspiration pneumonitis, the opacification seen with pulmonary contusions is irregular and does not conform to segments or lobes within the lung. Pulmonary contusion is not always immediately apparent radiographically; one-third of patients fail to demonstrate a lesion consistent with this diagnosis on the initial chest radiograph [38,39]. Although the mean time to opacification is 6 hours, it may take up to 48 hours for pulmonary contusion to blossom [40]. Tyburski et al, in an attempt to quantify the volume of the pulmonary contusion and correlate this volume with outcome, compared the pulmonary contusion score (PCS) of the initial radiograph to a repeat film 24 hours later [39]. The mean increase in the PCS of 7.9 units was nearly equivalent to an entire hemi–lung volume.

Computed tomography scans have been advocated as a more accurate means of detecting and quantifying pulmonary contusion. Thirty-eight percent of anesthetized dogs sustaining blunt chest trauma showed evidence of a pulmonary contusion on plain radiograph, compared with 100% using CT scans [41]. Miller et al, in a series of pulmonary contusion detected by CT scan, found that a mean of 18% of the pulmonary parenchyma was contused, and that the contusion increased by 11% with a repeat CT scan at 24 hours [40]. Moreover, 82% of patients with a contusion of at least 20% developed acute respiratory distress syndrome (ARDS) versus only 22% of patients with a contusion less than 20%. There was also an increased trend in the development of pneumonia in cases of greater contusion. Wagner reported that all patients with pulmonary contusions greater than 28% of total volume required intubation, compared with no patients with less than 18% contusion [42]. Prospective studies, however, have failed to show significant changes in management and outcome when chest CT scans are obtained solely for the assessment of pulmonary contusion [43–45].

Treatment

As with flail chest, the primary treatment of pulmonary contusion is supportive, and further modalities are directed toward the respiratory dysfunction it produces [45]. Initial efforts should address associated injuries with placement of thoracostomy tubes to relieve hemopneumothorax and pain control for chest wall injuries. Supplemental oxygen to treat the hypoxia, aggressive pulmonary toilet with coughing, deep breathing and suctioning as needed, and postural changes have been shown to improve outcomes. Prophylactic intubation without signs of impending respiratory failure is not indicated.

Controversy has surrounded the issue of fluid management in relation to pulmonary contusion. Although the overzealous use of crystalloid has been blamed for exacerbation of the hypoxia of pulmonary contusion, prospective studies have failed to substantiate this claim [46,47]. Standard resuscitation with crystalloids and blood products as indicated to obtain euvolemia is probably the ideal. The use of steroids has been shown to be of no benefit and may impair bacterial clearance within the pulmonary tissue [46,48]. Furthermore, empiric use of antibiotics is not warranted, as this will only foster development of resistant organisms. Antibiotic use should be reserved for treatment of specific organisms with the diagnosis of a superimposed pneumonia.

Outcome

In a study of 144 blunt thoracic trauma patients, mortality was 16% when the patient had either a flail chest or a pulmonary contusion, but it increased to 42% if patients had both injuries [12]. Furthermore, 92% with a combined injury pattern had at least one other intrathoracic injury, compared with 67% who had either injury alone. The most common cause of death following significant blunt thoracic injury, however, is brain injury.

The long-term dysfunction associated with pulmonary contusion and flail chest is quite variable in duration and outcome. Most pulmonary contusions heal within 14 days without complications. Patients with larger contusions and those associated with more severe injuries express fairly consistent symptoms in the few studies completed. Most patients complain of dyspnea, decreased exercise tolerance, and chest pain on the side of the injury [35,49–52]. Landercasper et al reviewed 62 patients who sustained a flail chest, including 42 who had a concomitant pulmonary contusion [49]. Thirty-two patients were available for follow-up for a mean of 5 years. Twenty-five percent complained of chest tightness; 49% complained of thoracic pain, and 63% complained of dyspnea. On exam, 46% were unable to expand their chest circumference by greater than 5 cm, and 57% had abnormal spirometry. Similar data were presented by Beal and Oreskovich, showing 64% of patients sustaining a flail chest injury had long-term morbidity of persistent chest wall pain exacerbated by activity, chest wall deformity, and dyspnea on exertion [52]. Despite such morbidity, 50% to 86% of these patients were able to return to full-time employment [49,50].

This prompted Livingston and Richardson to study 28 patients surviving severe chest injuries (abbreviated injury scale 4 or 5) prospectively to assess the degree of pulmonary dysfunction and the duration of this disability [51]. They found severe pulmonary dysfunction with pulmonary function tests (PFTs) at 40% to 50% of predicted values within 2 weeks of hospital discharge. At 4 months, however, there was marked improvement, and this trend for improvement continued out to at least 18 months following discharge, with PFTs 65% to 90% of predicted. Only 5% of patients met criteria for pulmonary disability.

Kishikawa et al prospectively followed 18 patients with severe blunt chest trauma [52]. Patients without a pulmonary contusion had normal PFTs within 6 months of the injury, while patients with pulmonary contusion and a group of patients from 1 to 4 years earlier showed CT evidence of fibrosis, persistent decreases in functional residual capacity and PaO_2. Some patients experience dyspnea indefinitely following their injury.

Summary

Blunt thoracic trauma can result in significant morbidity in injured patients. Both chest wall and the intrathoracic visceral injuries can lead to life-threatening complications if not anticipated and treated. Pain control, aggressive pulmonary toilet, and mechanical ventilation when necessary are the mainstays of supportive treatment. The elderly with blunt chest trauma are especially at risk for pulmonary deterioration in the several days postinjury and should be monitored carefully regardless of their initial presentation. Blunt thoracic trauma is also a marker for associated injuries, including severe head and abdominal injuries.

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