Review Article

Penetrating trauma

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Abstract: Pneumothorax occurs when air enters the pleural space. Currently there is increasing incidence of road traffic accidents, increasing awareness of healthcare leading to more advanced diagnostic procedures, and increasing number of admissions in intensive care units are responsible for traumatic (non iatrogenic and iatrogenic) pneumothorax. Pneumothorax has a clinical spectrum from asymptomatic patient to life-threatening situations. Diagnosis is usually made by clinical examination and imaging techniques. In our current work we focus on the treatment of penetrating trauma.

Keywords: Chest tube; pneumothorax; penetrating trauma

Submitted Aug 25, 2014. Accepted for publication Aug 26, 2014.
doi: 10.3978/j.issn.2072-1439.2014.08.51
View this article at: http://dx.doi.org/10.3978/j.issn.2072-1439.2014.08.51

Introduction

Thoracic injuries account for 20-25% of deaths due to trauma and contribute to 25-50% of remaining deaths. The increased prevalence of penetrating chest injury and improved prehospital and perioperative care have resulted in an increasing number of critically injured, but potentially salvageable patients presenting to trauma centers (1).

Penetrating injury is usually the result of the abrupt, direct application of a mechanical force to a focal area. A knife or projectile, produces tissue damage by stretching and crushing, and injury is usually confined to tissues in the path of penetration. The severity of the internal injury depends on the organ penetrated and on how vital the organ is.

Gun shot wounds

The degree of injury also depends on the bio mechanics of the penetrating projectile and includes the efficiency with which energy is transferred from the object to the body tissues. Other factors that dictate the severity of injury include the physical characteristics of the weapon, such as its velocity, size of impact face, deformability and the density of the body tissues penetrated. The velocity of the penetrating projectile is the single most important factor that determines the severity of the wound.

The amount of tissue damage is directly related to the amount of energy exchange between the penetrating object and the body part. The density of the tissue involved and the frontal area of the penetrating object are the important factors determining the rate of energy loss.

The mechanism of injury may be categorized as low, medium, or high velocity. Knives usually produce limited injury because they are classified as low-velocity projectiles. Medium-velocity injuries include bullet wound from most

types of handguns and air-powered pellet guns, and are characterized by much less primary tissue destruction than wound caused by high-velocity forces. High-velocity injuries include bullet wound caused by rifles and wounds from military weapons.

Bullets causes injuries of similar severity to knife wounds and tissue damage in the path of penetrating bullet. However, bullets also produce injury in structures adjacent to the bullet path. They produce tissue cavitation and by producing shock waves, extend the area of tissue damage. This cavity collapses because of the resultant vacuum effect, then reforms and collapses several times with diminishing amplitude until all motion ceases. These missiles results in exit wounds that are substantially larger than their corresponding entrance wounds (2).

Dense organs, such as bone and liver, absorb more energy resulting in more injury. Lungs, which have a much lower density, absorb less energy and fortunately suffer less of an injury (3). This explains the low frequency of operative intervention in penetrating chest trauma.

The cardinal rule of management is to leave the impaling object in situ while the patients rapidly transported to an operating theater, because it can have a tamponade-like effect on damaged vascular structures. The object should be removed only in controlled surgical environment.

As always in trauma, initial management begins with establishing ABCs. Indications for emergency endotracheal intubation include apnea, profound shock and inadequate ventilation. Chest radiography is not indicated in patients with clinical signs of a tension pneumothorax and immediate chest decompression is accomplished with either a large-bore needle at the second intercostal space or, more definitively, with a tube thoracostomy.

Volume replenishment is the cornerstone of treating hemorrhagic shock, but can also cause significant compromise of other organs systems, such as acute respiratory distress syndrome (ARDS) or a tremendous increase in lung water (soggy lungs) and cardiac compromise (4).

A large chest wall defect can result in a sucking chest wound or large open pneumothorax. This occurs when the injury consists of a large chest wall defect in addition to a sizable visceral pleural injury. A tension pneumothorax usually does not occur because there is a large chest wall defect which allow egress of air. Hypoxia and respiratory acidosis caused by hypoventilation and often asphyxiation can result if this condition is not treated. A patient's inability to ventilate can be temporarily corrected by covering the wound with a plastic sheet that is taped shut with the exception of a small area of a few inches left unsealed to act as a one-way valve to permit the egress of air from the hemithorax during the phase of exhalation. Immediate placement of a chest tube to prevent tension pneumothorax is required and to facilitate more normal pulmonary ventilation (5).

In hospital, management of a large open chest defects requires operative debridement with removal of devitalized tissue and foreign bodies, such as shotgun wadding materials and bone fragments and closure of the wound. Sometimes, this can be accomplished by mobilizing the surrounding tissues. However, large soft tissue defects may require rotational or free musculocutaneous flaps. The pectoralis muscle, latissimus dorsi or rectus abdominis flaps can be used. The use of synthetic materials such as Marlex, Gore-Tex or methylmethacrylate may be appropriate for elective chest wall reconstruction, but their usage is not recommended after the trauma, due to the risk of infection from contaminationed injury (6).

Stab wounds

Stab wounds of the chest can be made by the different sharp objects such as knifes, daggers, pieces of glass or other metals. The shape of this wounds is not typical, due to the skin elasticity which usually shrink the entrance to the wound.

The severity of stabbing depends on the point of entry to the chest (wound below the nipples in front and the inferior scapular angle at back should be considered as thoraco-abdominal wounds) which organ has been injured (chest wall vessels, lung, heart, great thoracic vessels, visceral pleura, oesophagus, diaphragm), shape and sharpness of penetrating object and finally is penetrating object still in the chest wound or has been taken out. In most cases, stabbing object penetrate through the chest wall hurting intercostal blood vessels and with its tip visceral pleura, contributing developing of pneumothorax or hematopneumothorax (7).

Clinical manifestation of stabbing wound is pain at the point of stabbing, irritation cough and short breath. After taking out the stabbing object from the wound, profuse bleeding is usually present and sometimes wheezing from the suction wound. Blood clot and transected tissue from the chest wall can cover the stabbing wound converting open pneumothorax to tension pneumothorax.

Bleeding from the wound is usually from the intercostal blood vessels, since they are part of systemic circulation,

comparing to blood vessels from lungs which are the part of pulmonary circulation with lower pressure.

Open pneumothorax

Open pneumothorax can be recognized by drifting the air through the wound, synchronously with breathing and may be visibly bubbling. During inspiration, when a negative intra-thoracic pressure is generated, air is entrained into the chest cavity not through the trachea but through the hole in the chest wall. This is because the chest wall defect is much shorter than trachea, and hence provides less resistance to flow. Once the size of the hole is more than 0.75 times the size of the trachea, air preferentially enters through the thoracic cavity (8).

This results in hypoxia and respiratory acidosis caused by hypoventilation and often asphyxiation can result if left untreated. The open pneumothorax can become tension if a flap has been created that allows air into the pleural cavity, but not out.

Clinically, breathing is rapid, shallow and laboured. There is reduced expansion of the hemithorax, accompanied by reduced breath sounds and an increased percussion note (9).

The definitive management of the open pneumothorax is to place an occlusive dressing over the wound, converting the open pneumothorax into the closed and immediately placing the intercostal chest tube. Rarely, if a chest tube is not available and the patient is far from a definitive care facility, a bandage may be applied over the wound and taped on three sides. This acts as a flap-valve to allow air to escape from the pleural cavity during expiration, but not to enter during inspiration (10).

Tension pneumothorax

Tension pneumothorax develops when a disruption involves visceral, parietal pleura or the tracheobronchial tree. Tension pneumothorax can occur as traumatic, spontaneus or iatrogenic etiology. The disruption occurs when a one-way valve forms, allowing air inflow into the pleural space and prohibiting air outflow. The volume of this nonabsorbable intrapleural air increases with each inspiration. As a result, pressure rises within the affected hemithorax, causes the mediastinum shift toward the contralateral side and compresses the other lung and right atrium of the heart (superior and inferior vena cava) compromised returing the venous blood to the heart-reducing pre load to the heart. Hypoxia leads to increased pulmonary vascular resistance via vasoconstriction. If untreated, the hypoxemia, metabolic acidosis and decreased cardiac output lead to cardiac arrest and death (11,12).

Physical examination in patient with a tension pneumothorax usually reveals severe respiratory distress, distended neck veins, a hyper-expanded chest, a deviated trachea and absent breath sounds on the affected side.

The immediate release of a tension pneumothorax is best accomplished by placing a needle into the pleural space to allow pressure in the pleura to equilibrate with the outside air. A large-bore, sterile hypodermic needle is introduced into the second intercostal space in midclavicular line. This relieves the compression of the underlying lung as well as the distortion of vital mediastinal structures. Release of the pressure decreases compression on the superior and inferior vena cavae and allows better venous return to the heart and immediate equilibration between the pleural space and the ambient air.

Tension pneumothorax may develop insidiously, especially in patients with positive pressure ventilation.

Penetrating pulmonary injuries are managed with tube thoracostomy alone in most patients (approximately 75%) (13). Of those who require operative intervention, 24% have been shown to require repair of pulmonary hilar or major parenchymal injuries (14). Pulmonary resections in this setting have been shown to carry a mortality rate of 30-60% (15). Death typically ensues from exsanguinating hemorrhage or massive air embolism (16). Air embolism occurs in the setting of a fistulous connection between a bronchus and a pulmonary vein. With spontaneous respiration, the pressure differential favors a gradient from the vein to the bronchus resulting in hemoptysis in 22% of these patients. With positive pressure ventilation or with Valsalva-type respiration, the gradient is reversed (17-30) and results in systemic air embolism (28,31-44).

Acknowledgements

Disclosure: The authors declare no conflict of interest.

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Cite this article as: Kuhajda I, Zarogoulidis K, Kougioumtzi I, Huang H, Li Q, Dryllis G, Kioumis I, Pitsiou G, Machairiotis N, Katsikogiannis N, Papaiwannou A, Lampaki S, Zaric B, Branislav P, Dervelegas K, Porpodis K, Zarogoulidis P. Penetrating trauma. J Thorac Dis 2014;6(S4):S461-S465. doi: 10.3978/j.issn.2072-1439.2014.08.51

