



Overview of Blunt Chest Injury with Multiple Rib Fractures

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Authors' contributions

This work was carried out in collaboration between both authors. Author EEE designed the study, wrote the protocol and wrote the first draft of the manuscript and also managed the literature searches. Both authors read and approved the final manuscript.

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ABSTRACT

Introduction: Thoracic trauma leading to multiple fractured ribs (MFR) remains common. The significant chest pain of multiple rib fractures can be difficult to manage and can lead to decreased pulmonary function, increased hospital stay, and increased health care expenditures.

Aims: To evaluate the treatment options available for pain control in blunt chest injury with multiple rib fractures.

Study Design: Internet research.

Methodology: Literature review on pain management of blunt chest injury associated with multiple rib fractures was done from 1970 to 2014 using manual library search, journal publications on the subject, and Medline.

Results: Various modalities have been in use including systemic modalities, regional modalities, transcutaneous modalities and cutaneous modalities.

Conclusion: The current research has shown differs modalities available for control of chest pain

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in blunt chest injury and multiple rib fractures in the ranges of systemic therapy, regional therapy and trans-cutaneous therapy. Summation of evidences favours regional therapy over others.

Keywords: Blunt chest injury; multiple rib fractures; pain management.

1. INTRODUCTION

Thoracic trauma leading to multiple fractured ribs (MFR) remains common [1]. While the chest pain associated with a single rib fracture is relatively easy to control, the significant chest pain of multiple rib fractures can be difficult to manage and can lead to decreased pulmonary function, increased hospital stay, and increased health care expenditures [2]. Patients with traumatic rib fractures often present with varying degrees of chest pain, which in turn leads to impairment of pulmonary mechanics, retention of trachea-bronchial secretions, and atelectasis [3]. Multiple rib fractures cause severe chest pain that can seriously compromise respiratory mechanics and exacerbate underlying lung injury and pre-existing respiratory disease, predisposing to respiratory failure [4]. Good analgesia may help to improve the patient's respiratory mechanics, avoid intubation of the trachea for ventilatory support and therefore may dramatically alter the course of recovery [1]. The cornerstone of chest pain management is early institution of effective pain relief [4].

Analgesia could be provided using systemic opioids, transcutaneous electrical nerve stimulation or non steroidal anti-inflammatory drugs. Alternatively, regional analgesic techniques such as intercostal nerve block, epidural analgesia, intrathecal opioids, intrapleural analgesia and thoracic paravertebral block have been used effectively. Although invasive, in general, regional blocks tend to be more effective than systemic opioids, and produce less systemic side effects [4]. Pain relief in chest trauma patients with rib fractures includes several modalities, such as administration of analgesics, rib belts, intercostal nerve block with bupivacaine hydrochloride (Marcaine, Astra), continuous intercostal nerve block, intercostal nerve block with morphine chloride, and subpleural block with 0.5% bupivacaine [3]. Studies have shown that injection into one intercostal groove blocks not only the intercostal nerve of that groove, but at least the one above and below it because of subpleural tracking [3]. Immediate pain relief after intercostal nerve block and improvement in pulmonary mechanics have been demonstrated

in several reports [3]. Surgical stabilization of rib fractures in form of open reduction and internal fixation is an alternative treatment for pain management of multiple rib fractures [5].

Thoracic epidural, thoracic paravertebral, and intercostal blocks are the top choices for patients with MFR and they are of equivalent efficacy [1].

Considering the numerous benefits of good analgesic in ameliorating the respiratory morbidity and mortality of traumatic multiple rib fractures, we set to review the various analgesic modalities for blunt chest injury and multiple rib fractures with a view to advising on the best modalities.

2. METHODOLOGY

A literature review on pain management of blunt chest injury associated with multiple rib fractures was done from 1970 to 2014 using manual library search, journal publications on the subject, and Medline. Full texts of the materials, including those of relevant references, were collected and studied. Information relating to the approaches to pain management of patients with blunt chest injury associated with multiple rib fractures including observed complications was extracted from these materials.

3. RESULTS

Pain management techniques for control of chest pain in blunt chest injury with multiple rib fractures have featured prominently in the literatures. Various modalities have been in use including systemic modalities, regional modalities, transcutaneous modalities and cutaneous modalities. The regional modalities are noted to show more measurable evidences over the other modalities.

4. DISCUSSION

4.1 Anatomy of the Chest Wall

The chest is truncated, dynamic structure with upper and lower boundaries that vary with respiration and patient's position [6]. The general surgeon considers the thoracic inlet to be zone

one of the neck. Some articles define both the subclavian and axillary vessels to be part of the thoracic inlet, and others describe these vessels as belonging to the upper extremity. The cupola of the lungs may rise into supra-clavicular spaces of the neck. With respiration especially in the supine position, the dome of the diaphragm may rise as high as the fourth interspace. When the patient is standing and lungs are fully expanded, the dome of the diaphragm may be as low as the 10th intercostal space. These variable anatomic considerations are important when determining associated neck and abdominal injuries in a patient with a major thoracic injury [6].

The chest wall consists of the bony structures (ribs, sternum and the spine), the related muscles and the diaphragm. Together they function as the respiratory bellows. The carefully regulated rigidity and mobility of the chest wall normally permit a balance between the supportive and ventilatory functions. The ventilatory function is however impaired by chest wall trauma with corresponding impairment of breathing.

The intercostal neuro-vascular bundle runs in the subcostal groove arranged craniocaudally as vein, artery and nerve, which makes the intercostal nerve easily accessible with injection needle.

The diaphragm is the principal respiratory muscle and accounts for 60% of the total amount of ventilated air [7].

4.2 Epidemiology of Chest Trauma

There are approximately 12 thoracic injuries per day per 1 million populations. Of these 12, four will require hospitalization, with one being severe [6]. Although commoner in young adult males, no age or sex is immune to chest trauma. Trauma to the chest and its structures may be due to: [6].

1. Blunt injury, including blast injuries
2. Penetrating injury
3. Pulmonary injury secondary to aspiration or infection
4. Pulmonary, vascular and cardiac alterations secondary to hormonal, immunologic and cell mediator responses, and
5. Iatrogenic injuries.

Of patients dying from trauma, 25% die as a direct result of the chest trauma. In an additional

25%, the chest traumas contribute to their demise [6]. In the United State, of 50,000 injuries in the major outcome study, 15,000 patients had chest traumas. Of these, 70% were secondary to blunt trauma and 30% to penetrating trauma. From 1962 to 1970, 60% of chest traumas were from motor vehicles accidents, 14% occurring at work, 10% at home, with 18% resulting in death. Of these, 71% involved the chest wall, 41% produced a pneumothorax or hemothorax, 7% involved the heart, and 7% involved the diaphragm. About 4% of patients will have injury to a great vessel, with less than 5% having injury to the tracheobronchial tree or oesophagus. Blunt and penetrating chest trauma may involve the liver, kidney and spleen. The many organs involved and problems produced by penetrating chest trauma influence morbidity and mortality [6].

In civilian practice, automobile accidents are the commonest causes of chest traumas (over 70%) [7]. Other causes of chest traumas include stab wounds, fall from heights, gun shots, falling objects and industrial accidents. In war, chest traumas account for about 10% of all wounds.

Penetrating wounds may be produced by a knife, piece of glass, a portion of an automobile, or a gunshot wound. Blunt trauma most commonly produced by motor vehicle accidents causes deceleration injuries. Blunt injuries include those produced by blasts, falls and other impacts. Iatrogenic injuries may be secondary to both diagnostic and therapeutic interventions resulting in injury to the oesophagus or trachea-bronchial tree during endoscopy, to the lungs or heart during catheterization and percutaneous biopsies, to the chest wall, the heart or even the great vessels during cardiopulmonary resuscitation [6].

4.3 Biomechanics of Blunt Chest Trauma

The three types of blunt force that lead to thoracic injury are compression, shearing, and blast [8]. Thoracic compression injuries such as rib fractures occur when 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. Frequently, however, ribs subjected to lateral or anteroposterior (AP) compression will fracture in two places: one at approximately 60° and again posteriorly. AP compression also can create a costochondral disruption, resulting in a sternal or anterior flail [8,9].

Shearing forces cause soft tissue and vascular injury [8]. 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 mediastinal pleura to the vertebrae below, the junction of the more mobile aortic arch and the stationary descending aorta is the most common site of disruption [8]. Both full thickness tears with free rupture and partial thickness injuries leading to pseudo-aneurysm formation are possible results. Shearing within the pulmonary parenchyma can lead to laceration, hematoma, contusion, or pneumatocele [8].

Blasts or 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 [8]. The severity of the pulmonary injury is related inversely to the victim's distance from the blast. Explosions within an enclosed space are more severe, because the pressure waves are reflected back to the patient, intensifying the original insult [8]. The characteristic pathology of pulmonary blast injury is a contusion with oedema and alveolar haemorrhage.[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 [8].

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 [8]. 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 [8]. 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 intra-thoracic and abdominal injury, as a source of significant chest pain, and as a predictor for pulmonary deterioration, particularly in the elderly. Case series of rib fracture patients presenting to trauma centres reveal that 84% to 94% of patients will have associated injuries.[8] The most common associated thoracic injuries are pneumothorax, haemothorax, and pulmonary contusion. 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 [8]. Contrary to historical beliefs, rib fractures, including those of the first and second ribs, are not associated statistically with aortic injury [10]. 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 [11-13]. Eight percent of patients brought to a trauma centre following a high-speed motor vehicular crash (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 [11]. 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 [11]. The presence of rib fractures is especially ominous in children and the elderly. The bones of children lack calcification; consequently, their chest walls are more compliant than adults' [14]. 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 paediatric patients with blunt chest trauma, 2% had significant thoracic injuries without evidence of any chest wall trauma [14]. Thirty-eight percent of children with pulmonary contusion do not have radiographic evidence of rib fractures. 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 [14]. 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 [2]. 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 [2]. 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 [2].

Flail chest is rare, but it is the most serious of the blunt chest wall injuries. The prevalence of flail chest among patients with chest wall injury is estimated between 5% and 13% [15]. 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. Muscular splinting of the chest early in the immediate post-injury period, however, may mask the paradoxical motion until the flail becomes apparent hours later with the development of chest wall muscle fatigue. In patients who are mechanically ventilated, a high degree of suspicion with palpation of the chest wall for crepitation 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 [12].

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 [15]. In non-intubated 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 co-morbidities and the elderly, is warranted [15].

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 [16]. 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 [16]. 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 [16]. Although the mean time to opacification is 6 hours, it may take up to 48 hours for pulmonary contusion to blossom. Tyburski et al. [16] 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. 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 [16]. Miller et al. [17] in a series of pulmonary contusion detected by CTscan, 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. 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 [18]. 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 [18].

4.4 Pathophysiology of Blunt Chest Trauma

Chest injuries adversely affect pulmonary function by three separate mechanisms: altered mechanics of breathing, ventilation/perfusion imbalance, and impairment of gas transfer [19].

4.4.1 Altered mechanics of breathing [19]

The great majority of blunt injuries to the thoracic cage and those penetrating injuries that cause haemothorax or pneumothorax impair ventilation. Even relatively minor trauma with fractured ribs but no underlying pathology may cause pain sufficient to lead to hypoventilation, atelectasis, failure to clear secretions, pneumonia, septicaemia, respiratory failure, and even death in an elderly or bronchitic patient. More serious problems which cause severe impairment of the mechanics of breathing include pneumothorax (particularly tension pneumothorax), haemothorax, ruptured diaphragm, multiple rib fractures with unstable segments, and injuries to the major airways. The most extensive disruption of the chest wall tends to occur with crush injuries where multiple bilateral rib fractures, ruptured diaphragm, or fractures of the spine or sternum coexist. Multiple fractures may cause substantial blood loss into the chest wall and pleural cavity; this may be increased by laceration of the underlying lung by sharp edges. There are two main types of chest wall derangement, a functionally important traumatic defect (sucking chest wound) or a flail segment. The latter may be unilateral with double fractures of three or more ribs, or anterior with fractures of three or more ribs on both sides of the sternum. Some fractures may occur through the costochondral junctions, and are therefore invisible on plain chest radiographs. The unstable segment moves inwards on inspiration (paradoxical movement) and consequently compromises ventilation by reducing tidal volume. Following diaphragmatic rupture, the abdominal contents are similarly sucked into the chest on inspiration. If the pleural cavity is filled with air or blood, ventilation of partially collapsed lung is similarly compromised. Injuries to the major airways or inhalation of foreign material including teeth, windscreen glass, or stomach contents may physically occlude the large or small airways, thereby obstructing air entry.

4.4.2 Ventilation/perfusion imbalance [19]

Effective oxygenation of the blood and elimination of carbon dioxide (CO₂) depend on a balance between ventilation of the lung and its blood supply. Thoracic injuries compromise ventilation/perfusion/balance by a number of different mechanisms. Mechanical obstruction of the airway is an obvious cause of impaired ventilation, but in practice other mechanisms predominate. Distribution of ventilation in the

lung is influenced by regional variations in airways resistance and compliance. The latter is governed by gravity-dependent intrapleural pressure gradients, and gas distribution in the lung is therefore uneven during normal resting tidal ventilation. In the lower or more dependent pleural space the pressure is closest to atmospheric (least negative). It becomes increasingly negative towards the apex or non-dependent region of the lung. When a normal inspiration is taken from end-expiration (functional residual capacity) expansion of the initially smaller dependent alveoli is governed by the steep portion of the compliance curve. Consequently, they expand more for each unit of pressure change than do those at the apices which are influenced by the upper, flatter portion of the curve. These differences result in preferential distribution of inspired gases to the areas of greater expansion in the dependent portions of the lungs. However, if terminal air spaces have collapsed due to haemopneumothorax or adult respiratory distress syndrome, inspiration results in preferential distribution to already expanded areas because of the influence of the compliance curve. The maldistribution of ventilation is worsened by airflow obstruction in terminal airways due to external compression, elevated intrapleural pressure, or interstitial oedema fluid. When a whole lobe or lung eventually collapses, there is perfusion of non-ventilated lung and a serious veno-arterial shunt effect. Ineffective oxygenation of the venous blood is reflected by widening of the alveolar-arterial oxygen tension difference and systemic hypoxia [19].

Movement of blood through the lungs is also influenced by gravity and the pressure gradient between the pulmonary arteries and left atrium. Blood flow is normally directed preferentially to the dependent parts of normal lung where ventilation is also most efficient. However, perfusion is often impaired by thrombosis of vessels in contused lung or widespread pulmonary microembolism by fat from bone marrow or platelet / neutrophil microemboli in patients with disseminated intravascular coagulation or adult respiratory distress syndrome.

In many patients after even minor thoracic trauma, the effects of ventilation/perfusion mismatch may lead to unsuspectedly severe hypoxia which is seldom recognized without blood gas analysis. Pulmonary contusion, intrapulmonary haemorrhage, and haemothorax

or pneumothorax are invariably associated with serious deterioration in pulmonary function.

4.4.3 Impairment of gas transfer [19]

Passive diffusion of gas across the alveolar capillary barrier is dependent on the surface area available, the width of the membrane, certain plasma and erythrocyte enzymatic factors, and the partial pressure gradient between the alveolar and vascular spaces. Following thoracic trauma a number of factors, including injury to the pulmonary parenchyma by contusion, damage to the alveolar capillary barrier by inhalation of gastric contents or smoke, impaired cardiac output, and interstitial pulmonary oedema due to over-transfusion of crystalloid, colloid, or blood (elevated left atrial pressure), may adversely affect gas exchange. However, the most sinister process involved is that which begins with the pathophysiological effects of shock and, if uninterrupted by prompt resuscitation, may progress to acute respiratory distress syndrome. The humoral and cytological changes that culminate in this syndrome are probably triggered by activation and interaction of the complement, coagulation, kallikrein, and plasminogen cascades, and result in trapping of 'activated' neutrophils in the pulmonary microvasculature. Here they release protease enzymes and generate oxygen free radicals with the potential to damage the alveolar capillary membrane. When full-blown acute respiratory distress syndrome occurs in a patient with thoracic trauma particularly following multiple injuries, the chances of survival decrease markedly. Gas exchange is impaired by extension of the diffusion pathway by the presence of hyaline membrane and oedema fluid in the alveoli; accumulation of interstitial fluid within the septum and of proliferated type II cells along its alveolar border; reduction in the surface for diffusion because of terminal air space collapse and closure of capillary channels; and the detrimental influence of consequent hypoxaemia, hypercapnia, and acid-base shifts in erythrocyte enzyme kinetics. Some of the consequences of this process precipitate further deterioration in pulmonary dysfunction. Pulmonary arterial hypertension develops because of hypoxic arteriolar vasoconstriction. Higher flow resistance in small vessels can be made worse by increased interstitial fluid pressure. Intravascular coagulation may exacerbate these problems and contribute to ventilation/perfusion mismatch.

Hypoxaemia is the first objective sign of the onset of acute respiratory distress syndrome, and is the cardinal index of its progressive severity. At a later stage CO₂ retention develops, with its consequent disturbances of acid-base balance. Unless blood gases are monitored continuously in patients with thoracic trauma the primary effects in the lung may be misinterpreted. The manifestations of respiratory insufficiency are reflected principally in deterioration of cardiovascular and central nervous system dysfunction.

In conclusion, injuries to the chest often produce derangement in respiration function resulting in pulmonary hypoventilation, inadequate oxygenation and respiratory acidosis. There may also be associated shock from massive bleeding or cardiac dysfunction [7]. Therefore the pathophysiology of chest trauma includes three factors: hypoxia, hypercapnia, and acidosis. Hypoxia can be caused by airway obstruction, changes in intra-thoracic pressure, ventilation-perfusion mismatches, intra-pulmonary shunting and hypovolemia. Hypercarbia is caused by inadequate ventilation resulting from the presence of a collapsed lung associated head injuries with altered mental status, or exogenous intoxication (drugs and alcohol). Acidosis is caused mainly by hypoperfusion from blood loss [20].

4.5 Rib Fractures

A rib fractures at the point of impact or at its weakest point which is the angle [7]. Rib fractures are the most common injuries after blunt chest injuries. Ribs 4 through 10 are usually fractured. One or two fractures without pleural or lung involvement are usually treated on an outpatient basis [20]. However, in the elderly, because of their decreased bone density, reduced chest wall compliance and increased incidence of underlying parenchymal disease, rib fractures may lead to decreased ability to cough, reduced vital capacity, and infectious complications [20]. Pain on inspiration is usually the primary clinical manifestation after rib fractures. Other clinical signs associated with rib fractures include tenderness to palpation and bony crepitus [20].

Simple fractures involve only the ribs and they are painful but not serious except in the elderly and patients with a low pulmonary reserve in whom impairment of ventilation and coughing may precipitate pneumonia [7]. The main feature

is pain with tenderness at the site of the fracture, worst on breathing or compression of the sternum. The fractured ends of the rib may puncture the underlying lung and cause pneumo- or haemothorax or surgical emphysema [7].

Multiple rib fractures are the hallmark of a severe trauma caused by the high – energy transfer.[20] Patients with multiple rib fractures should have intra-thoracic and abdominal injuries excluded by appropriate investigations. Fractures of the lower ribs (ninth to twelfth) are associated with an increased incidence of hepatic and splenic injuries, and fractures of the upper ribs (first to third), clavicle, or scapula are associated with major vascular injury [20].

4.6 Diagnosis of Blunt Chest Trauma

Diagnosis of blunt chest trauma associated with rib fracture(s) can usually be made by clinical evaluation of the patient with relevant history, physical examination and radiological examination. Pain on inspiration is usually the primary clinical manifestation after rib fractures. Other clinical signs associated with rib fractures include tenderness to palpation and bony crepitus [20].

Rib fractures are confirmed by chest x-ray (CXR) in only 50% of cases [12]. Screening CXRs miss rib fractures more than 50% of the time [12]. Radiology reports are often not sufficiently descriptive or are incomplete with respect to the number and location of fracture and reliance on these data will lead to erroneous conclusions. Using computerized tomographic (CT) scanning only, the finding of rib fractures in multiple locations was associated with increased incidence of respiratory failure. In contrast, the presence of any parenchymal injury or visible rib fracture on the screening CXR significantly increases the risk for subsequent pulmonary morbidity (odds ratio, 3.8; CI₉₅, 2.2-6.6). Although transthoracic CT (TCT) scanning markedly improved the diagnosis and delineation of rib fractures, the screening CXR was a better predictor of subsequent pulmonary morbidity and mortality [12]. TCT is highly sensitive in detecting thoracic injuries after blunt chest trauma and is superior to routine CXR in visualizing lung contusions, pneumothorax, and haemothorax. Early TCT influences therapeutic management in a significant number of patients [13].

4.7 Treatment of Blunt Chest Trauma

4.7.1 Historical perspective

The treatment of blunt chest trauma has undergone dramatic evolution over the twentieth century. In the first half of the century, the primary emphasis was on mechanical stabilization of the bony injury [21]. This was first done by such external devices as sandbags or traction systems and later by various surgical methods such as wires or screws. After 1950, the concept of “internal pneumatic stabilization” with positive-pressure mechanical ventilation was developed [21]. This became more prevalent and obligatory mechanical ventilation became the standard for blunt chest wall trauma [21].

The management of severe, blunt chest trauma evolved into the modern era with the publication of two studies in 1975. In a small series, Trinkle demonstrated that optimal pain control, chest physiotherapy, and noninvasive positive-pressure ventilation could avert the need for intubation and mechanical ventilation [22]. Also in 1975, Dittman published the first in a series of three articles on pain management in blunt chest trauma [23,24]. In the first study, 19 patients with multiple rib fractures and flail segments were treated with continuous epidural analgesia and intubation and mechanical ventilation were withheld. Using objective clinical criteria to monitor progress (e.g., vital capacity, respiratory rate, and tidal volume), 17 patients were successfully managed without positive-pressure ventilation. Dittman subsequently showed that 46 of 49 (94%) spontaneously breathing patients maintained a vital capacity greater than 13 mL/kg and avoided positive-pressure ventilation through the use of morphine analgesia by means of a thoracic epidural catheter [23,24].

Thus, the management of blunt chest trauma today focuses on both the underlying lung injury and on optimization of mechanics through chest physiotherapy and optimal analgesia. The critical importance of measuring ventilatory function tests as an objective means of monitoring adequacy of this analgesia was emphasized by the authors of the early studies [21]. Subsequent studies of pain management in blunt chest trauma patients would use the same methodology and additionally focus on comparisons between modalities and on objective outcome parameters.

4.7.2 Treatment of rib fractures

Rib fractures are the commonest of all chest injuries and are identified in 10% of patients after trauma [21]. The overall incidence is probably higher because not all rib fractures are seen on chest radiographs or otherwise detected [12,13]. Multiple fractured ribs cause severe pain, which may be more debilitating and harmful than the injury itself [21]. Pain limits one's ability to cough and breathe deeply, resulting in sputum retention, atelectasis, and a reduction in functional residual capacity (FRC). These factors in turn result in decreased lung compliance, ventilation-perfusion mismatch, hypoxemia, and respiratory distress. Failure to control pain, compounded by the presence of pulmonary contusion, flail segment, and other insults, can result in serious respiratory complications and death [21].

The treatment for injuries of the bony thorax has varied over the years, ranging from various forms of mechanical stabilization to obligatory ventilatory support [21]. It is now generally recognized that pain control, chest physiotherapy, and mobilization are the preferred mode of management for blunt chest trauma [4,21,25]. Failure of this regimen and ensuing mechanical ventilation sets the stage for progressive respiratory morbidity and mortality [21]. Consequently, several different strategies of pain control have been used, including intravenous narcotics, local rib blocks, pleural infusion catheters, paravertebral blocks, and epidural analgesia. Each of these modalities has its own unique advantages and disadvantages, and on the overall most efficacious method has not previously been clearly identified [21]. Subsequently, analgesic practices vary widely in this crucial setting. In one recent review, the majority of blunt chest trauma patients were still managed with intravenous or oral narcotics [21]. Other authors noted that epidural catheters were offered in only 22% of elderly blunt chest trauma patients and 15% of a younger cohort [2]. Poor pain control significantly contributes to complications such as atelectasis and pneumonia [26].

4.7.3 Modalities of analgesia

4.7.3.1 Intravenous narcotic

Intravenous narcotics have historically been the initial and most prevalent modality for relief of surgical and traumatic pain of all types [21]. They

are administered either by intermittent injection when pain is noted by the patient or continuous infusion. Most recently intravenous patient-controlled analgesia (PCA) has been developed to exploit the benefits of both methods. In this modality, a baseline intravenous infusion of morphine is provided and the patient may elicit an additional bolus for breakthrough pain [21].

The obvious advantages of intravenous narcotics are ease of administration and monitoring by nursing without the risks of an invasive procedure or need for specialized personnel. The efficacy of this modality for blunt chest wall trauma is controversial [21]. Intravenous narcotics have been shown to improve pain scores and vital capacity, yet some clinicians consider them inadequate in this setting. The disadvantages of systemic narcotics are the tendency to cause sedation, cough suppression, respiratory depression, and hypoxemia [21].

4.7.3.2 Epidural narcotics/anaesthetics

Epidural analgesia is a method whereby narcotics, anaesthetic agents, or combinations thereof are introduced into the spinal epidural space at the thoracic or lumbar level to provide regional analgesia [21]. This is accomplished by introduction of a polyvinyl catheter into the epidural space and delivery of agents by either a bolus, continuous infusion or, more recently, a demand system [21].

The major advantage of epidural analgesia is its apparent effectiveness in the absence of sedation. Epidural analgesia has been shown to result in an increased functional residual capacity, lung compliance, and vital capacity; a decreased airway resistance; and increased PO₂. Tidal volume is increased and chest wall paradox in flail segments is reduced [21]. Patients with epidural analgesia generally remain awake and can cooperate with pulmonary toilet and chest physiotherapy [21].

There are numerous real and theoretical disadvantages to epidural analgesia. Insertion may be technically demanding. Epidural anaesthetics can cause hypotension, particularly in the face of hypovolemia, and occasional epidural infection [21]. Epidural hematoma, accidental entry into the spinal canal, and spinal cord trauma can also occur [21]. Inadvertent "high block" may lead to respiratory insufficiency [21]. By combining an epidural narcotic with the anaesthetic agent, the dose of anaesthetic can

be decreased and these effects mitigated. However, the narcotic can cause nausea, vomiting, urinary retention, pruritus, and occasionally respiratory depression [21]. The contraindications to epidural analgesia may prove problematic in the trauma patient. These include fever, coagulation abnormalities of even minor degrees, and altered mental status [21]. There is some anecdotal concern that the bilateral analgesia effect may mask the symptoms of intra-abdominal injury. Finally, nursing intensity in monitoring for the effects of sympathetic block is somewhat more demanding than that for intravenous analgesia [21].

4.7.3.3 Intercostal nerve block

Intercostal analgesia or "intercostal nerve block" traditionally involves individual injections of local anaesthetic into the posterior component of the intercostal space [21]. Because of segmental overlap of intercostal nerves, it is necessary to induce block above and below any given fractured rib. Blocks of adequate scope have been shown to relieve pain with multiple rib fractures and improve peak expiratory flow rate and volume [21]. However, the effect lasts only approximately 6-24 hours depending on local anaesthetic agent used and whether adrenaline has been added to the local anaesthetic agent [21].

As a unilateral block, hypotension is rare, and bladder and lower extremity sensation are preserved [21]. The disadvantages of intercostals nerve block include the need to palpate the fractured ribs for injection, and the need for multiple and repeated injections [21]. Local anaesthetic toxicity may theoretically occur because of the higher doses needed, and the incidence of pneumothorax increases with the number of ribs blocked [21]. Also, inducing block for upper rib fractures is technically difficult because of the proximity of the scapula [21]. Intercostal catheterization and continuous infusion has been successfully used and mitigates the need for multiple injections. However, the anatomic endpoint of catheter placement, piercing of the "posterior intercostal membrane," is often unclear, raising the possibility of misplacement [21]. The full anatomic limits of the spread of intercostal drugs are unclear [21].

4.7.3.4 Intra-pleural analgesia

Intra-pleural analgesia involves placement of a local anaesthetic agent into the pleural space by

means of an indwelling catheter [21]. This produces a unilateral intercostal nerve block across multiple dermatomes by gravity-dependent retrograde diffusion of agent across the parietal pleura [21]. As a unilateral modality, it has advantages similar to intercostal block regarding hypotension and bladder and lower extremity sensation. Successful use of this modality has been reported in blunt chest trauma patients [21].

In terms of disadvantages, a significant amount of anaesthetic may be lost if a tube thoracostomy is in place, which is often the case with blunt chest trauma patients [21]. This can be mitigated by temporary "clamping" of the thoracostomy, which in turn evokes concerns of tension pneumothorax. Conversely, in the absence of a tube thoracostomy, intrapleural catheter placement may cause a pneumothorax [21]. The presence of haemothorax, also common in blunt chest trauma patients, may theoretically impair diffusion of anaesthetic [21]. Because distribution of agent is gravity-dependent, effectiveness also varies with patient position, catheter position, and location of fractured ribs. Diffusion is most widespread in the supine position, which is not optimal position for pulmonary function in the blunt chest trauma patients [21]. Conversely, the semi-upright position commonly adopted by chest trauma patients may allow disproportionate diffusion inferiorly and adversely affect diaphragmatic function [21].

4.7.3.5 Thoracic paravertebral block

Thoracic paravertebral block involves the administration of a local anaesthetic agent in close proximity to the thoracic vertebrae [21]. This can be achieved by intermittent injection, bolus by means of a catheter, or continuous infusion, and produces a unilateral somatic and sympathetic block that extended over multiple dermatomes [21].

Despite the fact that little recent investigation has been performed with this modality, its theoretical advantages are numerous [21]. It does not require painful palpation of ribs, is not in conflict with the scapula, and is felt by some to be technically easier than epidural anaesthesia. Because there is no risk of spinal cord injury as with epidural analgesia, this modality can be instituted on sedated or anesthetized patients [21]. It has few contraindications and requires no special nursing management. The most common complications are vascular puncture, pleural

puncture, and pneumothorax [21]. The unilateral nature of the block makes hypotension rare, preserves bladder sensation, and allows monitoring of the lower extremity neurologic examination when necessary [21].

4.7.3.6 Transcutaneous electrical nerve stimulation

Transcutaneous electrical nerve stimulation (TENS) produces pain relief by releasing endorphins in the spinal cord [4]. Sloan et al. [27] used it in patients with multiple rib fractures and found it provided better subjective pain relief, with improvement in peak expiratory flow rates and arterial blood gases when compared with a group of patients receiving nonsteroidal anti-inflammatory drugs (NSAIDs). The paucity of data on this mode of analgesia in patients with multiple rib fractures suggests that it is rarely used in this group of patients [4].

4.7.3.7 Oral analgesic drugs

Oral analgesic drugs such as nonsteroidal anti-inflammatory drugs (e.g., diclofenac, indomethacin) and acetaminophen do not depress the central nervous system or the cardiovascular system and are useful for mild to moderate pain. As with transcutaneous electrical nerve stimulation, there is a paucity of data on the use of oral analgesics in patients with multiple rib fractures [4]. However, there may be a place for the use of NSAIDs as adjuncts to other methods of pain relief in patients with multiple rib fractures [4]. Non-steroidal anti-inflammatory drugs can cause gastrointestinal upset and platelet and renal dysfunction. The latter would contraindicate the use of NSAIDs in patients who are not adequately resuscitated [4].

4.7.3.8 Rib fixation operations with titanium plates

The fixation of rib fractures is indicated in case of flail chest, concomitant lung lesion, serious alteration of the chest shape and persistent and chronic pain that affects normal life. Titanium devices (clips and bars; screws and plates) are effective and safe for repair of rib fractures and large chest wall defect reconstruction with minimal complications and good long-term results. In the experience of Bille, et al, there were 12 males, and the median age was 61 years. There were no postoperative deaths. The only postoperative complication observed was a pleural effusion requiring drainage in one patient who had titanium clips for the fixation of multiple

traumatic rib fractures. Median length of stay of the drain and median length of hospital stay were 3 days (range 1–6) and 4 days (range 2–42 days), respectively. The average follow-up period of operatively managed patients was 6 months, (range 2–14 months). Two cases of hardware failure occurred in two patients treated for a lung hernia with large chest wall defects involving the anterior costal margin with either devices [28].

4.8 Pain Pathway

Pain is described as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage indicating some problem, and often causing further physiological effect(s) in the body of the sufferer [29]. The physiology of normal pain transmission involves some basic concepts that are necessary to understand the pathophysiology of abnormal or non-physiologic pain. These include the concept of transduction of the first-order afferent neuron nociceptors. The nociceptor neurons have specific receptors that respond to specific stimuli if a specific degree of amplitude of the stimulus is applied to the receptor in the periphery. If sufficient stimulation of the receptor occurs, then there is a depolarization of the nociceptor neuron. The nociceptive axon carries this impulse from the periphery into the dorsal horn of the spinal cord to make connections directly, and indirectly, through spinal interneurons, with second-order afferent neurons in the spinal cord. The second-order neurons can transmit these impulses from the spinal cord to the brain. Second-order neurons ascend mostly via the spinothalamic tract up the spinal cord and terminate in higher neural structures, including the thalamus of the brain. Third-order neurons originate from the thalamus and transmit their signals to the cerebral cortex. Evidence exists that numerous supraspinal control areas—including the reticular formation, midbrain, thalamus, hypothalamus, the limbic system of the amygdala and the cingulate cortex, basal ganglia, and cerebral cortex—modulate pain. Neurons originating from these cerebral areas synapse with the neuronal cells of the descending spinal pathways, which terminate in the dorsal horn of the spinal cord [29].

4.9 Methods of Pain Measurement

Pain assessment helps the clinician to imagine the intensity of pain the patient is experiencing. If this assessment is done before, during and after a course of pain treatment, it helps the clinician

to know to what extent the treatment has helped his patient and therefore the efficacy of the drug. So as pain is, most of the methods of pain assessment are also subjective, and probably none is completely satisfactory [29]. The methods of pain assessment in cognitively intact persons include the 4-point verbal rating scales of Zubroid, the numerical rating scales, and the visual analogue scales [29]. These are the most widely used pain rating scales in both clinical practice and research. They are simple, minimally intrusive, effective and easy to administer and score [29].

4.9.1 Verbal pain rating scales of Zubroid

This stratifies pain intensity according to levels of severity. It employs different word descriptions to rate the patient's pain, e.g no pain (0), mild pain (1), moderate pain (2), and severe pain (3). These words can be translated to any language. Numbers are assigned to each of these words for recording and comparison purposes.

4.9.2 Numerical rating scales

This asks the patient to rate their pain from "no pain" (0) to "worst pain possible" (10). It is assumed that each number represents a proportional increase in pain severity.

4.9.3 Visual analogue scales

This employs a 10 cm line rated from "no pain" at the left to "worst pain" possible on the right. It requires the patients to mark their pain on the continuum. The VAS "score" is the distance from the "no pain" point to the patient's estimate. There should be no markings, numbers or words along the line, as these tend to influence the results.

4.10 Comparative Analysis of Different Analgesic Techniques in Patients with Blunt Chest Trauma and Multiple Rib Fractures

There are relatively few clinical trials comparing the efficacy of the various analgesia techniques in patients with blunt chest trauma. This may in part be because each technique has unique strengths, weaknesses, and contraindications, and pain management is individualized on the basis of the clinical condition and extent of injury. This makes randomized, controlled comparisons difficult or hard to justify [4].

Gabram et al. [30] prospectively compared intrapleural bupivacaine with systemic narcotics (morphine, meperidine, or hydromorphone) for the management of 48 patients with rib fractures. The patients with the block statistically had more compromised pulmonary function as measured by forced vital capacity (FVC) at admission; however, they tended toward a greater objective improvement of FVC at discharge, although the difference did not reach statistical significance [29]. When analyzing a cohort of severely impaired patients (initial FVC < 20% predicted), half of the systemic medication patients compared with only 10% of the block group failed and required another mode of therapy. Catheter complications were minor and did not contribute to overall morbidity.

Luchette et al. [31] prospectively evaluated analgesia for 72 hours in 19 blunt trauma patients with unilateral rib fractures. They found that thoracic epidural bupivacaine 0.125%, 8 to 10 mL/h continuous infusion, as compared with intrapleural bupivacaine 0.5% intermittent boluses of 20 mL every 8 hours, resulted in significantly lower visual analog scale pain scores, less use of "rescue" narcotics, and greater tidal volume and negative inspiratory force. Vital capacity, Fio₂, minute ventilation, and respiratory rate were not affected. Mild hypotension was a common complication with epidural blocks only. Shinohara et al. [32] had more favourable results with interpleural block. They studied 17 patients with unilateral multiple rib fractures and hemopneumothorax in a randomized, crossover, before/after trial on the first and second hospital days. An interpleural catheter was inserted along with a chest tube, and an upper thoracic epidural catheter was also established in the same patient. They administered 10 mL of 1% lidocaine for both blocks. The range of thermohypesthesia was unilateral and shorter with the interpleural block, whereas it was bilateral and wider with the epidural block. The effects of pain relief were almost the same. Respiratory rate decreased and Pao₂ tended to elevate similarly. Unlike epidural block, the systemic blood pressure with interpleural block changed only minimally. Serum levels of lidocaine were similar and in the safe range.

Mackersie et al. compared epidural and intravenous fentanyl for pain control and restoration of ventilatory function after multiple rib fractures in a prospective, randomized trial involving 32 patients. Pre-fentanyl and post-

fentanyl parameters were compared in both groups. Both methods significantly improved visual analog pain scores. The epidural method produced improvement in both maximum inspiratory pressure and vital capacity, whereas intravenous analgesia produced improvement in only vital capacity. Intravenous fentanyl produced increases in PaCO₂ and decreases in PaO₂, whereas no significant changes in arterial blood gases were observed with epidural fentanyl administration. Side effects were similar between the groups, with pruritus being more pronounced with epidural fentanyl [33].

Sloan et al. compared two groups of patients with MFRs who were randomized to receive either TENS or naproxen sodium 250 mg every 8 hours and also a mixture of paracetamol 1 g and dihydrocodeine tartrate 20 mg on an as-required basis. The peak expiratory flow rate change at 24 hours after the commencement of treatment, the PaO₂, and the pain relief were all significantly better in the TENS group [27].

Various modalities of analgesia for control of chest pain in patients with blunt chest injury and multiple rib fractures have been practised in our cardiothoracic unit with varying efficacy [34-37]. However we have not started using TENS in our unit.

5. CONCLUSION

The current research has shown differs modalities available for control of chest pain in blunt chest injury and multiple rib fractures in the ranges of systemic therapy, regional therapy and trans-cutaneous therapy. However summation of evidences favours regional therapy over others.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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