

Review Article:**FLAIL CHEST MANAGEMENT IN ARDS****Heru Koesbijanto**

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ABSTRAK

Flail chest mempersulit sekitar 10% sampai 20% pasien dengan trauma tumpul dada dan berhubungan dengan angka kematian berkisar antara 10% sampai 35%. Pengelolaan flail chest berat secara bertahap berubah dalam beberapa tahun, sebagai konsekuensi dari teknik ventilasi dan pemahaman yang lebih baik tentang patofisiologi dari sindrom kegagalan pernafasan kompleks traumatis akut. Saat ini, luas diyakini bahwa gangguan pernafasan pada pasien flail chest hanya sebagian karena ventilasi tidak efisien terkait dengan gerakan paradoksial dinding dada, tetapi sangat dipengaruhi oleh cedera toraks terkait lainnya pada memar paru tertentu dan atelektasis. Dalam dekade terakhir, andalan pengobatan flail chest berat telah bergeser dari pengobatan segmen flail dengan pengelolaan cedera toraks yang terkait dengan perhatian khusus pada memar paru. Pernafasan sindrom gangguan akut (ARDS) adalah reaksi serius terhadap berbagai bentuk cedera pada paru-paru. Ini adalah gangguan yang paling penting sehingga peningkatan permeabilitas paru edema ARDS adalah penyakit paru-paru berat yang disebabkan berbagai langsung dan tidak langsung. Hal ini ditandai dengan peradangan pada parenkim paru yang menyebabkan pertukaran gas disertai rilis sistemik mediator inflamasi yang menyebabkan peradangan, hipoksemia dan sering mengakibatkan kegagalan organ multiple. Kondisi ini mengancam kehidupan dan sering mematikan, biasanya membutuhkan ventilasi mekanis dan dirawat di unit perawatan intensif. Bentuk yang lebih ringan disebut acute lung injury (ALI).

ABSTRACT

Flail chest complicates about 10% to 20% of patients with blunt chest trauma and is associated with a mortality rate ranging from 10% to 35%. The management of severe flail chest has gradually changed over years, as a consequence of improved ventilatory techniques and better understanding of the pathophysiology of the complex traumatic acute respiratory failure syndrome. At present, it is widely accepted that respiratory impairment in flail chest patients is only partially due to inefficient ventilation related to the paradoxical movement of the chest wall, but is significantly influenced by other associated thoracic injuries, in particular pulmonary contusion and atelectasis. In the last decade, the mainstay of treatment of severe flail chest has shifted from the treatment of the flail segment to the management of associated thoracic injuries with particular attention to pulmonary contusion. Acute respiratory distress syndrome (ARDS) is a serious reaction to various forms of injuries to the lung. This is the most important disorder resulting in increased permeability pulmonary oedema ARDS is a severe lung disease caused by a variety of direct and indirect insults. It is characterized by inflammation of the lung parenchyma leading to impaired gas exchange with concomitant systemic release of inflammatory mediators causing inflammation, hypoxemia and frequently resulting in multiple organ failure. This condition is life threatening and often lethal, usually requiring mechanical ventilation and admission to an intensive care unit. A less severe form is called acute lung injury (ALI).

Keywords: flail chest, trauma, ARDS, lung injury**Correspondence:** Heru Koesbijanto, Department Thoracic Cardiovascular Surgery, Faculty of Medicine, Airlangga University, Surabaya**INTRODUCTION**

Severe blunt thoracic trauma due to traffic accidents are still common and often cause serious complications and even the death of the victim. Flail chest is a very serious complication and often causes problems for the doctors in managing complications resulting from such trauma. Multidisciplinary care modalities in the treatment of flail chest complications is improving from year to year as a consequence of the development of ventilation techniques as well as advanced research on the pathophysiology of respiratory failure due to trauma,

implants for bone fixation and the advance of molecular biology.

Complication that often makes the mortality of impaired lung function due to severe trauma to the thoracic wall, which cause flail chest is pulmonary contusions which often develop to become a syndrome known as Acute Respiratory Distress Syndrome (ARDS). The cause of the syndrome to date is only partially known, many other things is still a puzzle requiring more in-depth research. Experts have suggested many theories about the syndrome but a definite mechanism as the cause of

the syndrome is still unknown until now. The combination of flail chest and the incidence of complications would result in increasingly complex management of ARDS patients, involving a variety of multidisciplinary medical field and the results so far have not been satisfactory, while maternal mortality and morbidity is still high.

History

Flail chest is observed and reported since a few years ago on the books and records in case of literature on medical field. In 1958 NATO war Emergency Handbook has described flail chest, said that cases of flail chest caused by complications are a potential cause mortality due to the failure of resuscitation (Bjerke 2006). Subsequently in 1988, Emergency War Surgery NATO Handbook mentioned once again that flail chest is a very morbid cases in patients, with no mention of the therapy and treatment recommendations that flail the sternum (Bjerke 2006).

In addition to the old textbook there were photos on the handling of flail chest using a clamp (clamp doek) for stabilization by lifting broken ribs segment. Then in the next year, along with advances in ventilation technology, internal stabilization in flail chest could be done, that is by doing orotracheal intubation and ventilation with positive pressure (Athanasadi and Gerazounis 2004).

ARDS was first published in 1821. A scientist Laennec described the gross pathology of heart and lung specimen, one of anasarca idiopathic pulmonary, pulmonary edema without heart failure. In 1961, the clinician Asbaugh described about something called Acute Respiratory Distress in Adult, which is a collection of pathophysiological abnormalities in a patient who experienced severe shortness of breath. In 1971, Asbaugh and Petty called the abnormality as Adult Respiratory Distress Syndrome, rather than "acute". And in the next year the word 'Adult' changed to 'Acute' again due to the discovery that this syndrome also occurs in infants and children (Ware & Matthay, 2000, Miller & Croce 2001).

Incident

Flail chest occurs in 10-20% of blunt thoracic trauma with a mortality rate of about 10-35%. Report of the Major Trauma Outcome Study revealed 50.000 trauma patients, 75 patients were with flail chest. Roughly stated, there were averagely of 1-2 cases of acquired flail chest per month in the U.S. (Bjerke 2006).

While the incidence of ARDS is somewhat difficult to determine because the term for ARDS varies, such as Stiff lung, shock lung, wet lung, and so on, there are averagely 150000-200000 cases per year in the United States. The majority of these cases occurred after the patient was in hospital. This incidence is higher in adults than in children. Again, pulmonary ARDS is not a specific disease, but more about pulmonary dysfunction caused by lung disease, such as sepsis, trauma, or pneumonia. Incidence and mortality of ARDS is still high enough. It was reported a few years ago that four of 10 patients with ARDS could be saved, currently 7 of 10 patients survive ARDS (Ware & Matthay, 2000, Meduri & Tolley 2002).

Respiratory physiology

It can be briefly described as follows: contraction chest muscles and diaphragm will expand the chest wall, diaphragm will move towards the thoracic cavity of the abdomen so that the volume will increase, this will result in pleural cavity pressure will decrease, smaller than the outside air pressure, the air will go to fill the lung alveoli, leading to inspiration. This process is an active process.

Expiration occurs as a passive process, which will return the chest wall. Diaphragm also will back up toward the chest, thoracic cavity volume will shrink, the pressure in the thoracic cavity will increase, larger than the outside air pressure and the air will be expelled from the alveoli.

If there is interference with the respiratory component of the organizing process, such as injury to the respiratory muscles, a broken sternum, injury to the lung parenchymal alveoli or diaphragm, there will be interference with the process of gas exchange at the alveoli level (Malhotra 2007, Cohn 1997).

Pathophysiology of Flail chest

The occurrence of a traumatic flail chest requires a large kinetic energy in the case of blunt thoracic trauma is on wide area in the thorax, such as that in traffic accidents, falls from heights and so on. The occurrence of flail chest is an indicator that the patient has trauma to the kinetic energy is large enough, except in certain cases such as osteoporosis, and multiple myeloma. In young children, a large kinetic energy are often not the cause of flail chest, because the ribs are elastic, but a large force will be forwarded to intra-thoracic organs, like lungs, heart, or existing structures in the mediastinum, which would cause complications in these organs.

Per definition, flail chest occurs when 2-3 or more ribs are fractured in two places or more, so that the ribs as one of the segments that make up the chest wall is fixed to the posterior thoracic vertebrae and sternum section in the anterior part of the original stable in the following movements respiratory will 'release' in flail chest. Costa, a flail segment, will 'float'.

What happened next is the movement of the chest wall segment floating opposite to the movement of the chest wall during normal breathing process. At the time of the respiratory muscles to contract, the thoracic cavity expands, the volume increases, the air pressure inside the chest cavity decreases, not just the air is sucked out, but the flail segment/float will also be 'sucked' towards the inside/medial, opposite to the movement of the intact chest wall. It is similar to that in the expiration. This movement is called 'paradoxical'.

This would create a paradoxical movement of lung function, such as ventilation, distribution, perfusion that will be disrupted. At the time of inspiration, the chest wall expands, the flail segment of costal pushed/sucked into the lungs will press, so the inspiration of air entering the lungs would leak into the intact walls, because the lung wall flail segment will be depressed by a fracture. When the smaller expiratory chest wall, rib segment that will expand flail, because intra-pleural pressure increases, the air should come out at the expiration, some of it will flow to the wall flail pulmonary (inhalation), because the fracture segment movement will cause the chest cavity more because the movement was paradoxical.

Furthermore, while inspiring more air in the lungs flail thrust into the lungs only, expiration process will be repeated as before. As a result of the case, the patient will experience a fairly severe hypoxia, because most of the air inside the lungs do not participate in the process of inspiration/expiration (Bjerke, 2006, Gabram et al., 1995, Tanaka et al., 2002).

Pathophysiology of ARDS

In patients with blunt chest trauma with flail chest, a large kinetic energy can break apart ribs, and the thorax will be forwarded to cavity nearby intrathoracic organ. Frequently affected organ is the lung in flail chest. The kinetic energy of the impact on the lungs may vary from mild to severe. The most common is a known complication with pulmonary contusions. Pulmonary contusions occur in blunt trauma 20% of thoracic and the recorded mortality is as much as 10-25% and 40-60% of patients requiring mechanical ventilation due to pulmonary contusions.

There are three components of the pulmonary contusions of the lungs and alveolar parenchyma: the presence of alveolar edema, hemorrhage in the lung and the presence of parenchymal atelectasis. With the three components above, there will be interference with diffusion/exchange of O₂ and CO₂ gas in the alveoli, which will result in the occurrence of hypoxia in these patients. Pulmonary contusions usually occur 24 hours after trauma, beginning with the interference of diffusion/gas exchange, increased pulmonary vascular resistance and decreased lung compliance. In addition, due to the trauma of the components of the lung, there will be an inflammatory process, in which it will lead to further expense and components of anti-inflammatory cytokines. The component will develop systematically, which may certainly affect various organs, such as the lung. Only 50-60% of patients with contusions Pulmonary will evolve into ARDS (Cohn 1997, Miller & Croce 2001).

ARDS or acute respiratory distress syndrome is a severe pulmonary function abnormalities, beginning with the onset of the inflammatory process in lung parenchyma systemically and at the same time the body will release inflammatory mediators that would lead to the onset of the inflammation, hypoxemia and further processing will lead to multiple organ failure. In general, ARDS is caused by two mechanisms: the first is the direct cause of the lung, such as aspiration of gastric acid, toxic gas inhalation and blunt thoracic trauma. Second, due to systemic. Factors such as sepsis, multiple trauma/severe, transfusion reactions, pancreatitis, or the use of heart lung machine is old. All this will lead to the spending of a lot of inflammatory mediators (interleukins/cytokines) that will cause lung damage in ARDS.

Acute phase is characterized by alveolar wall edema due to increased capillary permeability in the alveoli and result from the reaction of inflammatory cytokines (IL-8, IL-1, IL-10 and TNF). The acute phase is often referred to as exudative phase. Pulmonary edema will occur here and will be detected when the patient is examined by X-ray radiograph. Clinically the patient would appear cramped, due to progressive respiratory failure, accompanied by hypoxemia refractory to oxygen administration. CT scan showed alveolar filling, consolidation and sometimes accompanied by atelectasis. Although some patients can recover from the acute phase, most patients will experience further progression of the collar to next phase, the fibroproliferative phase. This phase occurs in the alveoli fibrosis (fibrosing alveolitis) with persistent hypoxemia, increased alveolar dead space, and will continue to decrease in lung compliance. In this phase, fibrosing alveolitis on chest X-ray image obtained with the

opaque linear line shows a profile of fibrosis (Ware and Matthay 2000).

Briefly, ARDS is characterized by: acute, bilateral infiltrate on chest X-ray images, pulmonary artery wedge pressure <18 mmHg, and $\text{PaO}_2:\text{FiO}_2 <200$ mmHg. Clinically, patients with ARDS would appear weak, rapid shallow breathing, cyanosis, and on examination of blood gas analysis, oxygen levels will be low. If the condition of patients with ARDS as described above coupled with a flail chest, the situation will rapidly deteriorate and will lead to mortality. One attempt to overcome such problem that can be directly performed by surgeons is surgical intervention for flail chest and ARDS, carried out by a multi-disciplinary experts who deal directly with the disorder, such as Intensivist, pulmonologist and fisioterapist (Ware and Matthay 2000).

Management of flail chest with pulmonary contusion

Respiratory failure in flail chest is caused by an inefficient ventilation due to paradoxical chest wall motion as well as the occurrence of complications resulting from flail chest, pulmonary contusions, and atelectasis (Davignon & Kwo 2004, Gabram et al. 1995). At the end of this decade, the management of severe flail chest switching from a flail chest wall therapy towards the intensive management of the pulmonary contusions resulting from flail chest (Gabram et al., 1995).

Internal pneumatic stabilization with the use of mechanical ventilation is a standard therapy in patients with flail chest accompanied by respiratory failure, or if flail chest trauma is accompanied by other organs such as head trauma, abdominal trauma, and others who require mechanical ventilation to help improving the condition of the patient.

However, although the techniques of mechanical ventilation with low tidal volume may reduce the mortality of patients with severe flail chest, a long-term ventilator use can often lead to serious complications, the incidence of pneumonia and ARDS. Regarding the flail chest surgery, though is still controversial, many experts believe that surgical stabilization, especially in cases of severe flail chest and in cases under certain circumstances is an indication to do. The time is right and effective for surgical stabilization of flail chest in patients with ventilator, depending on the degree of parenchymal lung damage (Voggenreiter et al., 1998, Richardson et al., 1982). The presence of extensive pulmonary contusions is a contraindication of surgical stabilization performed on the chest wall. Such patient requires lengthy ventilator and early surgical

stabilization will not be beneficial (Voggenreiter et al., 1998, Richardson et al., 1982). The surgery has to be done if the patient is stable and the ventilator is weaned, meaning that there has been improvement of lung contusions.

In patients with blunt thoracic trauma and severe flail chest without pulmonary contusion, the use of the old ventilator for internal stabilization is not necessary, given the complications of infection from the use of a ventilator that will happen. This means that the use of a ventilator is not always effective in stabilizing the chest wall to correct respiratory failure in flail chest (Tzelepis et al., 1989-Fleming & Bowen 1972).

In patients with mild pulmonary contusions or without pulmonary contusions, early surgical stabilization can be done (only 1-2 days prior to the ventilator) to accelerate out of the ICU patients and prevent further complications.

Another indication for surgical stabilization is the case of extensive flail chest, including antero lateral area, in younger patients without severe respiratory failure. The main objective of these measures is to prevent the occurrence of chest wall movement restrictions. The next indication for surgical stabilization is essential in patients with flail chest when thoracotomy is performed due to possible complications in thoracic organ from blunt thoracic trauma.

According to research some of the experts, (Voggenreiter et al., 1998) indications for surgical stabilization in flail chest is as follows: 1. Patients who require thoracotomy because of other injuries/follow-up on the organ intrathoracic, 2. Patients with a ventilator, with decreased lung function without a clear profile of pulmonary contusions, a lot of secretions and severe pain with strong analgesics (Gattinoni et al., 2001). Patients with a ventilator, which previously suffered from pulmonary contusions and head injuries, who failed weaning (Meduri & Tolley 2002). Patients with extensive antero-lateral flail chest and the presence of severe fracture dislocation are prevented from late chest wall deformity, which may inhibit chest movement during breathing.

Patients with flail chest in general requires good management, such as the provision of strong enough analgesics, pulmonary toilet, and intubation and mechanical ventilation for the provision of internal stabilization of a fractured rib segments, especially if accompanied by respiratory disorders.

With the use of ventilation, the patient will relatively stay longer in the ICU and other complications often

arise such as pneumonia, septicemia, ARDS or barotrauma resulting from the use of mechanical ventilation. Deaths due to prolonged intubation is the most common in sepsis, pneumonia and ARDS.

With surgical stabilization performed in flail chest, a lot of benefits are reported by the surgeons from a variety of centers. These benefits include the duration of the use of mechanical ventilation, a short time in the ICU, length of hospital stay could be shortened, and respiratory dysfunction and deformity of the chest wall can be repaired.

Controversy regarding surgical stabilization for fractures of the ribs, especially flail chest is still much debated by scholars from many centers in the world, including in Indonesia. Multiple rib fractures conservative therapy is likely to lead to some things that will happen as the progressive displacement during fracture healing process that will lead to deformity, which reduced the volume of the chest cavity, atelectasis, and prolonged pain/chronic (Casali & Fontana 2006). Similarly, in flail chest, conservative therapy, in addition to the complications that arise as above, there are also problems such as stress fractures placed on the chest, chest pain in all parts exposed to trauma and sometimes dyspnea.

The problem of indications and the right time to do surgical stabilization is still controversial, but in principle the experts and researchers agree that if the person is in an unstable state due to respiratory dysfunction due to damage of the lung parenchyma, the stabilization effort should be suspended first. If the situation began to improve, and there was resolution of parenchyma lung damage and respiratory dysfunction, the surgical stabilization can be done. Several methods of surgical stabilization of rib fractures can be applied here with some of the advantages and disadvantages, such as the use of plate and screws, intramedullary devices, bridging and wiring (Engel et al. 2005ff)

As mentioned earlier, that the most common complication of flail chest is the incidence of pulmonary contusions. There are three important components that occur in pulmonary contusions, namely: the existence of alveolar edema, hemorrhage in parenchyma lung, and atelectasis. This disorder usually occurs 24 hours after blunt thoracic trauma/flail chest, beginning with the disruption of gas diffusion in the alveoli, increased pulmonary vascular resistance and decreased incidence of lung compliance. Pulmonary contusions occur 20% of blunt thoracic trauma, with a mortality of 10-25% and 40-60% require mechanical ventilation. Further complications of pulmonary contusions that occur

include the onset of ARDS (50%) (Ware & Matthay, 2000, Miller & Croce 2001).

Specific diagnosis of lung contusions sometimes difficult to determine from the clinical examination. The presence of blunt thoracic trauma with rib fractures or flail chest strongly supports the diagnosis of pulmonary contusions. By examination of the thoracic images, we can detect pulmonary contusions, which usually occur 24-48 hours after trauma. A gloom profile in the chest area exposed to trauma, such as pneumonitis or hemothorax profile with sharp phrenico-costalis angle, supports the diagnosis of contusions. With a CT scan we would be able to distinguish normal lung tissue, atelectasis, alveolar hemorrhage and edema (Voggenreiter et al., 1998). General management of pulmonary contusion is supportive therapy. The majority of pulmonary contusions do not require specific treatment. Only in pulmonary contusions that occur severe enough, there will be a disruption of gas diffusion, and the patient will experience hypoxia, especially in case of flail chest. The patient should be more intensively managed. These should be administration of oxygen, with ventilation and intubation and mechanical ventilation if necessary, the provision of adequate analgesics if there are rib fractures or flailchest.

For fluid therapy, classical fluid management, such as fluid restriction, is still controversial. From various studies and experiments, excessive fluid administration will result in pulmonary edema which would interfere with breathing, but otherwise fluid restriction will result in hypovolemia, especially in patients with multiple trauma, which would be detrimental and will lead to inflammatory reactions and lung trauma, acute lung injury (ALI) will be ARDS and Multiple Organ Failure (MOF). Therefore, in the case of pulmonary contusions, proper fluid administration is Euvolaemia (Miller & Croce 2001).

Clinically, patients with ARDS may show symptoms such as shallow breathing pattern is , rapid breathing, and symptoms that correspond to the basic disease, such as shock. To ensure ARDS, instead of other causes such as cardiogenic pulmonary edema, on the pulmonary artery catheter should be placed to measure pulmonary artery wedge pressure. Examination by CT scan and chest X-ray images will show a profile bilateral infiltrate. By laboratory examination, it occurs when the ratio of ARDS partial arterial pressure of oxygen (PaO₂). compared to the fraction of inspired oxygen (FiO₂) is below 200 mmHg. Normal pulmonary capillary wedge pressure (less than 18 mmHg). If the PaO₂/FiO₂ is less than 300 mmHg, with a photo illustration of bilateral infiltrate, it indicates the

existence of Acute Lung Injury (ALI), although the numbers differ with ARDS, ALI is a precursor of ARDS. Briefly, ARDS is characterized by (Ware & Matthay, 2000, Cohn 1997). an acute onset, presence of bilateral infiltrate on chest X-ray images, pulmonary artery wedge pressure <18 mmHg, PaO₂: FiO₂ <300 mmHg (acute lung injury), and PaO₂: FiO₂ <200 mmHg (ARDS).

Pathophysiologically, ARDS is characterized by a diffuse inflammatory process in both lungs parenchyma. This inflammatory process will lead to further expenditure of cytokines and inflammatory mediators released by local epithelial and endothelial cells in lung parenchyma. Further components, such as neutrophils, T-lymphocytes will spread parenchyma stricken with lung inflammation. Processes and reactions would further aggravate the situation of parenchyma lung permeability barrier between the capillaries and alveoli will increase so that they will be inundated with protein-rich fluid and pulmonary edema as seen on chest X-ray. Thus pulmonary edema due to endothelial injury and increased vascular permeability, is not caused by cardiogenic factors such as that patients with heart failure (Richardson et al., 1982).

The management of ARDS.

Actually there is no specific therapy to cease the inflammatory lesions in the lung due to ARDS. Management of ARDS in general is focused on the prevention of iatrogenic lesions of the lung, maintaining optimal tissue oxygenation, and reduces fluid in the lungs. Supportive therapies for ARDS are:

Therapy causes of ARDS

The cause of ARDS have two types: direct and indirect, as discussed earlier. Possible causes should be traced and identified carefully and eliminated, in this great blunt trauma to the thorax with the onset of complications of rib fractures/flail chest and pulmonary contusions. Antibiotics are given immediately after the blood culture results are known. More than 60% of ARDS patients with nosocomial infections are contracted both before and after lung injury.

Mechanical ventilation

Ventilation done via oro-intubation or tracheostomy tracheal event of prolonged ventilation for more than 2 weeks. The main purpose of the use of mechanical ventilation is to maintain adequate gas exchange and minimize the occurrence of complications. In general, the use of conventional mechanical ventilation with tidal volume is 12-15 ml/kg bw. Recent research

suggests that high tidal volume, when given to patients with ARDS will lead to over-distension of alveoli and can lead to volutrauma. Research on ARDS cases showed a decrease in mortality and morbidity in the use of mechanical ventilation with a tidal volume of 6 ml/kg tidal volume compared with 12 ml/kg which would result in hypercapnea and atelectasis (Malhotra 2007). Further positive end expiratory pressure (PEEP) was conducted on the use of mechanical ventilation in order to open the collapsed alveoli due to pulmonary edema.

Prone position/the tummy

In some studies, the distribution of pulmonary infiltrates of ARDS are not always symmetrical. Downside position of the patient will increase oxygenation because of atelectasis alveoli will open again and will improve perfusion in the alveoli. In this condition intrapleural pressure in the stomach will decrease, postural drainage will be improved so that atelectasis, especially the dorsal part, can be reduced (Gattinoni et al., 2001).

Fluid therapy

The management of a classical fluid in patients with lesions in the lung is fluid restriction, because various studies and experiments showed that excessive fluid administration will result in pulmonary edema, which would certainly interfere with breathing, but otherwise will result in patients with fluid restriction to hypovolemia, especially in patients with multiple trauma and bleeding, which is certainly detrimental, because it will interfere with perfusion and tissue oxygenation. This situation will lead to inflammatory reactions and lung will further aggravate the situation, There will be acute lung injury (ALI) or worse, and then ARDS. If it is not handled properly will lead to Multiple Organ Failure (MOF).

Thus, in the case of pulmonary contusion, the appropriate fluid that should be given is Euvolaemia (Miller & Croce 2001). However, some researchers still believe that pulmonary function would improve if the patient loses weight due to discharge or low wedge pressure resulting from diuretics or fluid restriction.

Corticosteroids

There is no advantage at all from high-dose corticosteroids in patients with ARDS. The study by Meduri demonstrated that administration of corticosteroids in moderate dose will show better results. It was possible that this study took place at fibroproliferative phase, there was an emphasis on the inflammatory process that occurs in this phase. Initial dose 2 mg/kg methylprednisolone per day for 3-5 days,

then the dose is tapered off the dose of 0.5-1.0 mg per day for 1-2 weeks (Meduri & Tolley 2002).

Nitric oxide

Provision of NO by inhalation can create pulmonary vascular vasodilation without causing systemic vasodilation. NO is a selective pulmonary vasodilator. Rapid arrest of NO by hemoglobin may prevent systemic vasodilator effect. Although in a clinical trial inhaled NO administration can have a positive effect, but in other experiments it turns out that the provision of NO did not reduce mortality and duration of mechanical ventilation performed. NO is possibly useful in patients with pulmonary hypertension and ARDS with severe hypoxemia (Ware and Matthay 2000). However, surfactant provision did not reduce mortality and morbidity in patients with ARDS (Malhotra 2007).

CONCLUSION

Blunt trauma of the thorax with great kinetic energy with pulmonary complications and mortality remains a major threat, especially if it is accompanied with a flail chest and ARDS. Less careful management of complications will result in difficulties. The use of mechanical ventilation of time will cause pneumonia, surgical stabilization with less precise timing would create post-operative morbidity and mortality and pulmonary contusions with inadequate management of 50% will lead to ARDS. By knowing the mode of flail chest injury, pathophysiology and complications such as pulmonary contusion, flail chest ARDS management is expected to be provided to overcome respiratory disorders due to the onset of pulmonary contusions. ARDS may also be more focused so that morbidity and mortality can be reduced to a minimum.

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