Review Article

# Pulmonary contusion

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**Abstract:** Lung contusion resulting from chest trauma may be present various clinical pictures. It quite often remains unrecognized and is only suspected later when severe complications have developed. Lung contusion may present in association with chest trauma but may also occur alone. It has to be emphasized, that lung contusion as a clinical identity does not necessarily require a blunt or penetrating chest to be in the background. Nowadays, as a result of traffic accidents, following high energy deceleration, lung contusion may present without an actual tissue damage in the chest wall as a condition initiating an independent, life-threatening generalised process. Although lung contusion shows similarities to blast injury of the lung with respect to clinical consequences, other factors play a role in its aetiology and pathology. Its description and recognition as an independent pathology is not simple. Several approaches exist: thoracic trauma, pulmonary contusion, pulmonary laceration, lung contusion; although these may show similar clinical signs, manifest in different pathologies. Pathologies with similar meaning and possibly similar clinical course cannot, actually, be differentiated; they may accompany other injuries to the trunk, skull or extremities, which, alone, are associated with high morbidity and mortality. Generally, it can be declared that besides high energy, blunt injuries affecting the trunk, lung contusion, has been an often neglected additional radiological finding attached to the main report, despite the fact, that its late consequences crucially determine the prospects of the injured.

**Keywords:** Blunt thoracic trauma; pulmonary contusion; acute respiratory distress syndrome (ARDS)

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## Introduction

Pulmonary contusion is a protean clinical entity. Depending on the nature and severity of the trauma and the condition/reserves of the patient who suffers the injury the outcome can range from a transient discomfort to a sudden death. Changes in the mechanism of accidents induced by the development of the car industry have manifested in injuries affecting organs of drivers/passengers.

As a result of improved active and passive protection systems of vehicles, injuries causing significant tissue damage have become less common, while injuries leading to subcapsullary damage or contusion of parenchymal organs, caused by deceleration, have become more frequent.

Asymmetric warfare and bombing terror attacks highlight consequences of blast injuries resulting in lung contusion. Lung contusion presenting as monotrauma may in itself turn into a life-threatening pathology, lung contusion in a polytraumatized patient worsens chances of survival manifold. Although it may seem logical, it is not only primary impairment of tissue oxygenation that is responsible for resulting morbidity and mortality. Several

factors of Systemic Inflammatory Response Syndrome (SIRS) caused by lung contusion are still unknown (1,2). Recent years have seen several studies published in the literature investigating the connection between severity of lung contusion and inflammatory markers responsible for multiple organ failure (3,4).

Blunt chest trauma usually results from a high-energy collision affecting the chest wall. Patients, most often, are involved in a traffic accident or sustain an injury due to high pressure affecting the chest. The severity of organ damage depends on several factors, e.g., location and direction of the force, velocity, weight and size of the impulse, flexibility of the chest wall, general condition of thoracic organs, the body position and whether the body is in motion or fixed at the moment the accident happens. Although high energy, blunt chest injuries usually cause fractures to the ribs, acutely developing, potentially life-threatening pneumothorax (PTX) and haemothorax (HTX) do not necessarily go together with fractured ribs.

The mechanism of lung contusion is not exactly known. Based on principles of physics, it is obvious that the acceleration or deceleration of the human body can cause damage to the lung tissue without any considerable degree of a collision, similar to compression or a sudden hit on the chest wall. Some studies, involving animal and cadaver experiments, found the speed of the impulse and the resulting compression to be the most important factors causing tissue damage (5). Naturally, the viscoelasticity of visceral organs and their ability to slide on top of one another do also influence the extent of tissue damage (6).

The fact that direct injury to the lung tissue does not only lead to haematomas in the alveolar structures, i.e. the breathing surface and consequently to atelectasis, but also to the parenchyma of the lung is not emphasized enough. This complex injury, the alveolar, vascular and parenchymal damage of lung is lung contusion that can be the cause of acute respiratory distress syndrome (ARDS) (7).

In the background of the mortality of acute lung injury (ALI), higher than 40% according to some studies, there are clinical signs that are not exclusively specific to this pathology. In a progressive, severe case, hypoxia, reduced lung compliance, capillary leakage and non-cardiogenic pulmonary oedema may turn into respiratory failure, the effective treatment of which requires intensive therapy and ventilation (8).

This chapter does not discuss damage to the bony and soft tissue structures of the chest wall, i.e., penetrating injuries and resulting changes in the lung, large vessels in the chest and the heart or injuries to the main airways. The complex symptomatology, diagnostics and therapeutic possibilities of chest trauma are not included in this chapter either. The aim, exclusively, is the examination of lung contusion. Although lung contusion is only regarded as an individual pathology in theory, its consequences crucially determine the survival of traffic accident victims.

## Historical background

According to some sources, lung contusion as a pathology was first described in 1761 by the Italian anatomist, Morgagni (9). The term ‘pulmonary contusion’ was coined by the French military surgeon, Dupuytren, in the nineteenth century (10). It was Smith, in 1840, who first described damage to the parenchyma of the lung as the cause of death in one of his patients (11). An increase in the number of pulmonary contusion cases can be linked to the more widespread use of explosives beginning mainly at the time of the First World War, when soldiers fell victim to pulmonary bleeding, resulting from blast injury, without external signs on the chest. Hooker was the first to report the marked clinical importance of lung injuries caused by explosion (12). During the Second World War, as a consequence of air raids, the number of lung contusion cases increased, consequently, lung contusion gained more attention. Investigations were carried out using animal lung tissues placed at various distances from an explosion site. It was shown that, by wearing protective gear, the severity of injury could be reduced, or injury could even be prevented (9). The Vietnam War served as another opportunity for studying war injuries. This was the time when the use of X-rays as a diagnostic tool became more widespread and when ’wet lung’ was defined as a separate condition. It was during the 1960s as well, when advantages of positive pressure ventilation in this particular form of lung injury were recognized (9). During subsequent decades, as a result of investigations into lung contusion, the pathological background of the injury itself was clarified and the increasingly efficient early diagnostics and therapy significantly improved the prognosis (12,13).

Later, it became apparent, that lung contusion injuries and blast lung injuries have different underlying pathomechanisms, the combination of which may be fatal. Studies of mass injuries resulting from wars and terror attacks of the recent decades have led to further questions. In modern wars, it is blunt chest injuries and explosion injuries that cause the majority of combat related thoracic

 

trauma.

Advanced, high-tech, individualised body armours have changed the character and clinical manifestations of chest trauma. Under field circumstances, the incidence of blunt chest trauma is four times higher than under civilian circumstances, in certain situations, pulmonary contusion occurs in nearly 50% of blunt chest trauma cases. Although modern body armours cover a larger body surface and provide an all-around protection and thus, have resulted in a decrease in the number of perforation injuries, the consequences of injuries caused by blunt trauma often fail to get adequate attention (14).

In case of long contusion, the injured, initially, manifest minimal respiratory distress which nevertheless, may progress into a rapidly manifesting ARDS and pneumonia. Long-term respiratory failure may have a 10–25% mortality (1,15).

## Definition

Lung contusion is when, as a result of chest trauma, there is direct or indirect damage of the parenchyma of the lung that leads to oedema or alveolar haematoma and loss of physiological structure and function of the lung (16). This type of injury leads to reduced gas exchange, increased pulmonary vascular resistance and decreased pulmonary compliance within 24 hours. In case of severely injured patients, the inflammatory response results in ARDS (7).

The mechanism of pulmonary contusion is most probably the consequence of alveolar damage and alveolar strain accompanied by the alveoli being torn away from the bronchi and dislocated leading to a reduction of breathing surface (17).

Lung contusion is an injury to the lung tissue without actual structural damage. Consequently, blood and other fluids accumulate within lung tissues. Excess fluid causes a decrease of breathing surface leading to hypoxia. The pathophysiology of lung contusion includes ventilation and perfusion changes, increased intrapulmonary shunt, increased lung water, segmental lung damage and reduced compliance (12).

The Murray Lung Injury Score (LIS) is used for the assessment of the severity of ALI. Four parameters are examined and scored according to severity from 0 to 4. These are:

- Atelectasis;
- Hypoxemia score ($PaO_2/FiO_2$);
- PEEP score;
- Lung compliance.

The determination of the above parameters requires a chest X-ray, blood gas analysis and other data displayed on the respirator.

The result is the sum of examined parameters divided by the number of parameters examined. Upon a result of 0: there is no lung injury. Scores 0.1–2.5 indicate mild or average injury. If the score is above 2.5 ARDS is present (18).

The first clinical definition of ARDS dates back to 1967. The Berlin definition that has recently been accepted defines it as follows: Acute diffuse, inflammatory lung injury, leading to increased pulmonary vascular permeability, increased lung weight, and loss of aerated lung tissue with hypoxemia and bilateral radiographic opacities, associated with increased venous admixture, increased physiological dead space and decreased lung compliance (19).

A study investigating 4,397 trauma patients, revealed, that 4.5% of blunt chest trauma cases develop ARDS. In the pathomechanism of post-trauma ARDS, penetrating and blunt chest trauma showed a nearly equal incidence (9,20,21).

## Aetiology

According to some data, 7–10% of trauma patients develop ARDS irrespective of whether they have had primary lung contusion or not. Rubenfeld *et al.*, found 38.5% mortality in ALI, and 41% mortality in ARDS, in a study examining an adult population (22).

Based on the mechanism of the accident, lung contusion can develop as a result of a blunt or penetrating injury as well, or due to a combination of both.

Blunt chest trauma accounts for more than 17% of emergency admissions. Lung contusion developing during the first 24 hours appears in about 20–22% of the injured, resulting in damage to the parenchyma of the lung, oedema, alveolar haematoma and loss of the physiological structure and function of the lungs.

The underlying cause is usually the sudden deceleration of the body, while the chest collides with a fixed, stationary object. In daily life, this typically happens during traffic accidents in approximately 70% of lung contusion patients. Additionally, falls from great heights or more rarely, explosions or sport injuries may also cause lung contusion (1). A recent study investigated the incidence of chest trauma in an urban environment. Out of 1,490 trauma survivors, most of whom suffered a blunt injury, 55% showed signs of soft tissue damage, 35% sustained

 

a rib fracture, out of which 18% developed subcutaneous emphysema and 3% flail chest, and 18% showed signs of haemothorax and/or pneumothorax (23).

Another study investigating 127 cases of blunt chest injury without rib fracture found that 16% of patients had cardiac contusion, 8% had diaphragm rupture, 7% aorta rupture, 3% cardiac rupture, 2% tracheobronchial, 1% large vessel and 2% pulmonary laceration (24). Only 20–30% of the patients required a thoracic drain and less than 10% needed surgery (25).

It is a frequent phenomenon in clinical practice that injury to an identical size of lung tissue may manifest in conditions of varying severity. This is suspected to result from the immunological status and molecular mechanisms responsible for the inflammatory response.

Concomitant injuries require surgery in most cases, nevertheless, as these patients are often unstable surgery is not always possible. Therefore, determining the most appropriate time for the surgical intervention may pose a problem for clinicians. At present, there are no laboratory values or examination methods available that could provide guidance.

## Mechanism and pathophysiology

Blunt chest injury by the resulting direct and indirect consequences may cause respiratory failure. Respiratory failure can develop immediately after the trauma, acutely or it may even take a week. After a longer period of time, however, it is more difficult to reveal the primary, underlying cause. Consequently, mechanical factors and pathophysiology of the injury shall be discussed together.

Tear or penetration of the visceral pleura, that, by the resulting haemo- or pneumothorax leads to a life-threatening condition, is a frequent consequence of direct injury caused by blunt or penetrating trauma. The force of injury transferred onto the parenchyma of the lung results in atelectasis subsequently leading to respiratory failure.

Raghavendran and colleagues in 2008, investigated factors aggravating lung contusion with an animal experiment. They observed in several cases, that the extent of lung injury did not correlate with the severity of subsequent hypoxia. In a considerable number of models, hypoxia caused by the isolated lung contusion settled within 48 hours.

One possible factor that could have contributed to the development of a contusion with severe respiratory failure and consequent ARDS was the aspiration of gastric acid. Thus, the authors concluded, that concomitant aspiration of gastric acid together with increased permeability in the lung and enhanced inflammatory processes may contribute to the development of more serious lung contusion (26,27).

Lung contusion develops as a result of damage to the compressed lung tissue caused by an external force. Three further mechanisms are suggested to be in the background: inertial effect, spalling effect and implosion effect.

- The inertial effect leads to a similar axonal damage. The lighter alveolar tissue is injured as a result of the shearing force of hilar structures. This is a consequence of the fact that tissues of different density decelerate and accelerate differently (28).
- Spalling: a tear develops in the lung tissue where the pressure wave encounters different bordering surfaces, such as e.g the alveolar wall (29).
- Implosion: it occurs upon sudden increase in airway pressure. Shock waves compress the gas within tissues containing air bubbles, thereafter, the gas expands to manifold its original volume causing micro explosions within the aerated tissue (30).

Contusion usually develops at the site of the impact. Rarely, similar to brain trauma patients, via a contrecoup mechanism, it may manifest in the lung tissue contralateral to the direct trauma (31). It is the flexibility of the chest wall that is responsible for the extent of the energy impulse affecting the lung tissue. The flexible rib cage of children is able to transfer more energy without rib fractures. The more rigid rib cage of adults, however, is capable of absorbing more energy. Patients with fractures accompanied by lung contusion are children in 62% and adults in 80% of the cases. Elderly patients are more likely to suffer a fracture than a contusion (30).

As a result of the contusion, alveoli and capillaries are torn, blood and interstitial fluid leak into the alveoli and tissues. First, a haematoma appears in the area of the injury, then, several hours later, oedema develops in and around the affected area (9). Ventilation further impaired by the developing inflammatory response and the proteins appearing in the alveoli (32). There is a decrease in the amount of surfactant and the alveoli ultimately, collapse. The pathological fluid appearing in the alveoli becomes increasingly thicker, the lung tissue loses its elasticity. Upon injury to only one side of the chest, the resulting inflammatory reaction may initiate oedema and an inflammatory reaction in the other, intact side as well.

In severe cases, the injury can lead to the development of ARDS (33).

Ventilation of the contused areas gradually worsens as oxygenated air cannot enter the alveoli during inspiration, perfusion is also impaired as the part of the lung affected by hypoventilation is excluded from the circulation by reflex vasoconstriction. This is the process of the resulting systemic hypoxemia and hypercapnia (18,34,35). Several studies have revealed that, subsequent to pulmonary contusion, the activation of the immune system creates an acutely developing inflammation within the lung. Consequently, in the lung of patients having sustained a chest contusion injury, immune system activation causes the local accumulation of large amounts of various cytokines and chemokines followed by the infiltration of neutrophyl granulocytes. Altered cytokine production and apoptotic processes within immune-competent cells have been observed in the lung and in distant organs as well. Deficient bacterial clearance and an increased susceptibility for sepsis have also been reported (2).

## Clinical presentation

Clinical signs vary on a large scale. Mild contusion may remain asymptomatic. In case of severe lung contusion, disturbed alveolar gas exchange decreases arterial oxygen concentration. The resulting tissue hypoxia leads to dyspnoea, tachypnoea and consequently tachycardia. Clinical signs manifesting are not typical and often develop slowly. Auscultation may reveal reduced breathing sounds above the area affected by contusion, difficulty breathing and cough may also be observed. Excessive bronchorrhea and haemoptysis are not characteristic, may present only in severe contusion. Pain, rib fractures, haematoma and subcutaneous emphysema may occur in the affected area of the chest, next to tachycardia and hypotension (17).

Age above 60, falls from heights over 6 meters, deceleration from a speed of >65 km/h, chest pain, abnormal mental status, painful chest wall on the physical examination, painful injury, intoxication, are influencing factors and signs of chest trauma (36).

The symptoms and signs need time to develop. A severe injury manifests in symptoms sooner, within hours and may even lead to death, whereas milder injuries cause gradual deterioration in the patient's status, the characteristic clinical picture appears 24–48 hours after sustaining the injury (35,37,38).

In order to arrive at an exact diagnosis, assessment of the patient's general status and the mechanism of the injury, consideration of the physical examination results and analysis of imaging test results and targeted laboratory tests are necessary.

Respiratory parameters observed during mechanical ventilation, if needed, may add further information and thereby assist in setting up a definitive diagnosis. Depending on the aetiology of the accident, lung contusion may be accompanied by soft tissue injury, bone fractures or polytrauma. Thus, trauma to the skull, heart contusion, spleen rupture and damage to the bowels are not infrequent. The most severe consequences of high impact blunt chest trauma include lung rupture, tracheobronchial rupture and laceration.

A common and immediate consequence of the injury is the developing haemo- and pneumothorax. In young patients, high impact trauma to the more flexible chest may result in severe diffuse contusion without an actual injury to the bones of the chest. Rib fracture, however, is a very common finding in older patients with more rigid chest structures after having sustained blunt chest trauma (16). ARDS manifests in 17% of patients that only have lung contusion and 78% of patients with polytrauma. ARDS develops in 82% of patients if more than 20% of the pulmonary volume is affected, in cases where it is below 20%, it is only 22% (18). Pneumonia develops in 20% of patients. In respirated patients, this number may actually be higher. There is no difference with respect to the development of ARDS or pneumonia between children and adults (39).

It is very difficult to investigate lung contusion in a human patient population and also to decide whether severe complications result from a multi- or polytrauma, or an isolated lung injury as they can also lead to similar pathologies. Apparently, we needed animal models that can imitate isolated lung contusion injuries.

Several animal models have been mentioned in the literature for the investigation of chest trauma. John R. Border and his team were already conducting experiments with dogs in 1968, investigating the effect of chest trauma on the lung. Pathological examinations clearly indicated injury to the lung tissue and atelectasis (40). Nichols *et al.* modified Border's model by adding a metal plate that they attached to the chest of the dogs. They observed that the hypoxaemic peak developed over 24 hours (41). Wang and colleagues invesigated a combination of lung and myocardiac contusion with the help of a pendulum on mice. The advantage of their method was that they could apply a well-defined energy to cause trauma, however, the resulting myocardial injury undoubtedly contributed to the hypoxia

 

and often killed the mice (42). Raghavendran *et al.* were the first to create a model that could produce a bilateral lung contusion. Previous models had only been able to recreate monolateral trauma. They found that frequently, there was no correlation between the extent of lung injury observed and the severity of subsequent hypoxemia. There were patients with extended contusion that had no hypoxia and others with serious oxygen deficit alongside a lung injury hardly detectable by computed axial tomography.

As pulmonary contusion in itself is an independent risk factor of pneumonia, ALI and ARDS, it was important to create a model that could help investigate the isolated pathophysiology of bilateral lung injury without causing severe injury to large vessels of the heart and chest. The various models were not only able to imitate injuries sustained during involvement in motorcycle and car accidents but can also demonstrate injuries resulting from falls from heights. Tissue samples taken from the lung verified lung contusion; haematoma, monocytic, lymphocytic, neutrophyl granulocytic infiltrations were observable in the alveolar space, oedema was present in the interstitial space. The results led to the conclusion that there was a reverse relation between the size of damaged lung tissue and partial oxygen pressure. The bigger the area injured, the bigger was the decrease in oxygen pressure. According to several animal experiments investigating immune response, there exists a connection among the extent of the force causing the trauma, the size of surface injured, resulting tissue damage and the immune response triggered. Acute phase reaction is a comprehensive physiological change, arising at an early stage of inflammation due to infection or tissue damage caused by trauma. It is characterised by fever, vascular permeability and changes in the biosynthetic, metabolic and catabolic processes in different organs. Acute phase reaction is triggered and coordinated by a large number of various inflammatory mediators—cytokines, anaphylatoxins, glycocorticoids. Results of measurements are in line with the observation from clinical practice that the acute phase reaction occurs during the first 2–3 days (27,43).

## Diagnosis

The diagnostic algorhythm of penetrating, high-energy injuries that massively destroy chest tissue and have manifest clinical signs, is well-known and can provide valuable guidance. Knowledge of the mechanism of the accident, the manifestation or absence of the characteristic clinical signs, do not provide enough evidence to be able to exclude or suspect the presence of lung contusion with certainty (44).

The availability of clinical diagnostic procedures inevitably depend on how advanced the given health care system is. Traditional X-ray and chest ultrasound may be sufficient in the lack of a CT to diagnose a PTX or HTX study involving 13,564 injured patients compared traditional radiological and focused CT results with whole body CT. The authors concluded that the routine use of whole body CT in trauma care changed diagnostic accuracy in patients with severe chest trauma (45). On the contrary, however, the diagnostic accuracy in the case of minor chest injuries increased significantly. The decreasing number of injuries of the lung parenchyma and an increase in the number of lung contusion injuries result from the enhanced sensitivity and spatial resolution of examination methods. Nonetheless, no improvement was found in chances of survival (46).

Results of radiological examinations of blunt chest injuries have diagnostic and prognostic value. It is important to emphasize, that blunt chest injuries do not 'respect' anatomical boundaries of the lung, thus, the interpretation of traditional X-ray pictures may pose difficulties. More detailed, layered CT images of the chest may, however, prove more informative for the examination of the pulmonary interstitium and vasculature, and may help predict the consolidation of injured lung areas.

The differential diagnosis of lung contusion from other pulmonary diseases, or pneumonia may also prove problematic. Pulmonary contusion is, apparently, a result of a direct trauma. Nevertheless, with respect to pathomechanism, the appearance of injuries resulting from indirect shearing forces should also be kept in mind. The flexibility of the lung parenchyma is capable of absorbing and transferring energy impulses, it is not infrequent therefore, that symptoms only manifest 48–72 hours after the injury. Chest X-ray performed after the lung injury is not sensitive enough to show signs of lung contusion, further CT examination is required to arrive at a more exact diagnosis (47). The sensitivity of chest CT as compared to that of X-ray provides 38–81% more precise diagnostic accuracy. In the USA, civilian trauma accounts for 25% of deaths. Among patients with chest injuries as a result of involvement in traffic accidents or industrial accidents, rib fractures and pneumothorax are the most common, the pulmonary contusions numbers are lower (47).

In the modern military environment, injury to the lung parenchyma is the most common, occurring in nearly half of thoracic injuries. Third and fourth generation body

 

armours transform the kinetic energy of the bullet in a way that even if the expanding pressure wave damages the skeletal structure of the chest, causing fractures, the impact causes no actual alveolar tissue destruction, only alveolar haematoma (47). Paradoxically, pulmonary tissue damage is the greatest if there is no damage to the chest wall itself, as in this case, kinetic energy is transferred to the lung. Under non-military circumstances, such injuries occur in traffic accidents and falls from heights.

Manifest clinical hypoxia may have in reduced breathing surface due to blood-filled alveoli, increased shunt fraction and direct injury to the pulmonary vessels in the background. Interestingly, the arising inflammatory response reactions in the intact contralateral lung may also result in parenchymal damage, thus clinical-stage ARDS may soon manifest (47).

The question may, at this point, arise whether it is possible for a clinically relevant, high-mortality and morbidity respiratory failure to develop without actual tissue damage subsequent to a relevant impact.

On the X-ray picture, acute injury presents as a patchy consolidation. The edges of the contused area are not well defined and are non-segmental, they do not respect' structural, anatomical boundaries. CT is better than radiography for the diagnosis and evaluation of pulmonary contusion, however, the patchy coverage, intra-bronchial bleeding and features of a lobar infarction may not only characterise pulmonary contusion. In trauma cases, the aspiration of gastric acid is a common complication of loss of consciousness, the radiological presentation of which may further complicate the interpretation of radiology findings, posing a great challenge in terms of differential diagnosis. It is important to emphasize that the aspiration of gastric acid and pulmonary contusion injury may further worsen mortality and morbidity statistics.

The radiological presentation of pulmonary contusion is not necessarily immediately manifest, in about 47% of patients it is delayed. Markedly detectable radiological within the first 24 hours indicate negative prognosis. Early radiological sign are non-specific and may refer to pneumonia, lipid embolism and aspiration as well (28,29,48).

Pulmonary laceration cannot be identified on chest X-ray pictures at first, due to the fact that the elastic shrinkage of the normal lung parenchyma surrounds the laceration. Initial radiological signs appear after the first 48–72 hours. For the exact diagnosis, a chest CT is the next mandatory step. The presentation of parenchymal damage is less characteristic in the lung as compared to other organs, its reason being that the lung, containing air, may suffer injuries of unpredictable directions and dimensions. As a consequence of air being compressible, and the lung tissue being elastic, injuries to tissues, haematomas, and the radiological presentations of air-filled cavities are not specific and often not relevant to the clinical signs.

## Therapy

As with every disease, it is also true for lung contusion, that prevention plays a pivotal role. Developments of modern car industry, protective gear designed for free-time and professional sports, and body armours used by the military and law enforcement all focus on protecting the trunk in order to reduce the risk of injury to visceral organs. It has to be emphasized, however, that despite high-tech protective gear, subsequent to high-energy collisions, coup-contrecoup injuries to the lung cannot be fully avoided or prevented and these may remain hidden upon initial examinations.

The varied pathology of lung contusion does not make our task easier either. The implied diagnostic challenges and difficulties, and non-specific clinical presentations may result in tendencies to over- or under-triage in early stages of hospital treatment. This is explained by the fact that modern imaging techniques are able to detect even a mild contusion, resulting in cases being diagnosed which are irrelevant from the therapeutic standpoint.

On the other hand, based on our clinical experience, it seems that lung contusion may disappear without complication in 3–7 days.

It is a fundamental rule of early therapy that the injured is to be seated in a reclining position. The prior aim is to avoid respiratory failure and to ensure adequate tissue oxygenation. In cases that do not require mechanical ventilation, by using non-invasive ventilation, the first five post-trauma days are spared until the expected development of frank lung contusion. This, however, requires a proper patient monitoring system, arterial blood gas analysis, and the availability of radiological back up, if needed. General and special laboratory diagnostic results used in everyday clinical practice may only indirectly refer to the rapid progression or silent elimination of the condition. There is no treatment currently available that could facilitate recovery. The aims of supportive therapy are to avoid respiratory insufficiency, to eliminate pain, to manage the cleaning of airways and to prevent the development of complications.

The goal of oxygen administration and the use of positive

 

pressure ventilation is to achieve adequate oxygenation of the blood (33). The second cornerstone of adequate therapy is intravenous fluid replacement. It is pivotal to manage hypovolemia. Fluid overload, on the other hand, should be avoided as hypervolemia increases pulmonary oedema and consequently aggravates gas exchange. Regarding the recommended volume for fluid resuscitation, the literature suggests the monitoring of pulmonary artery pressure, the normal range of which is 25–30/9–10 mmHg (13). By using the venodilator effect of furosemid, we can decrease pulmonary vascular resistance and intra-capillary pressure in the lung and it can also be administered as a diuretic in case of hypervolemia (13). Adequate pain management is crucial. Chest pain aggravating upon inspiration leads to hypoventilation, other types of pain cause hyperventilation, both affect respiratory function negatively.

There is no specific therapy for post-traumatic respiratory failure that could significantly decrease the risk of developing ARDS. Targeted or ex juvantibus antibiotic therapy may improve the outcome of pneumonia. Otherwise, respiratory failure and potential multiple organ failure should be treated according to updated intensive therapy guidelines and professional standards (49). Some studies have suggested the use of inhalation agents, the effects of which, upon various levels of post-traumatic alveolar pathology, may increase the number of ventilator-free days, although, their straightforward positive effects are still to be verified (50,51). Similarly, several studies are underway investigating the effect of statins in improving endothelial function, and the role of aspirin in decreasing the number of micro-thrombi in the lung, which thereby, contribute to reducing the incidence of ARDS. The results of these studies do not, at present, provide straightforward evidence in support of their clinical relevance (52,53).

Arising complications and additional injuries e.g., haemo-, pneumothorax do also have to be treated. In the case of small-scale pneumothorax, observation may suffice as it may get absorbed spontaneously. However, if it causes a disturbance of gas exchange, or if positive pressure ventilation is being applied thoracic drainage is absolutely indicated. The prior goal is to improve gas exchange by choosing the most ideal method of invasive, or non-invasive oxygen support. Besides observation of the patient's status, non-invasive ventilation should be prolonged, the success of which could be facilitated by supportive pain management, positional drainage and respiratory physiotherapy.

Non-invasive positive pressure ventilation (NIPPV) may reduce the need for intubation in haemodynamically stable post-trauma patients without progressing respiratory failure. A crucial issue with respect to respiration is adjusting the optimal positive end expiratory pressure (PEEP), prevention of atelectotrauma and the maintenance of physiological ventilation/perfusion parameters. Currently, there is no straightforward consensus about the optimal PEEP, which, by providing the best oxygenation, could help avoid pulmonary hyperinflation. In patient care, we should aim at reaching and maintaining the optimal PEEP for the individual patient (54). Even so, the risk of ARDS may still persist and it may happen that even after having applied NIV intubation invasive ventilation will still be necessary (55).

Consequences of inadequate ventilation may include non-resolving hypoxia and hypercapnia. Over-ventilation, on the other hand, may lead to hyperoxia and hypocapnia. Due to its haemodynamic effects it may aggravate respiratory insufficiency. The use of excessively high pressures may cause barotrauma, that of excessively high volumes results in volume-trauma. Early invasive ventilation of trauma patients may further deteriorate lung contusion status, thus it is a potential morbidity and mortality risk factor.

The essence of respiration strategy in post-trauma patients is to provide a pulmo-protective respiration. It means a low-tidal-volume respiration calculated as 6–8 mL/kg ideal body weight. This way, we can reduce the over-distention and structural damage of alveoli (56).

By titrating the ideal respiratory parameters it is possible to avoid volume-trauma, barotrauma and atelecto-trauma, and to reduce the risk of ARDS, the number of ventilation days and hospitalisation time as well. The respiration mode for mechanical ventilation currently held to be ideal is the pressure or volume-targeted synchronised intermittent mandatory ventilation with pressure support ventilation SIMV + PSV). Modern respirators allow for several combinations of setting with the aim to provide the best patient comfort and the most optimal gas exchange to achieve the best possible outcome. Alveolar recruitment manoeuvers have been proven to improve pulmonary compliance, oxygenation, and to decrease atelectasis and pro-inflammatory cytokine levels (57).

Respirated patients with severe ARDS, lying in a prone position, have been reported to show significant decrease in mortality. Nevertheless, we would question the viability of this method in patients having sustained isolated, structural thoracic laesions. The use of extra corporeal membrane oxygenation and extra corporeal $CO_2$ removal undoubtedly hold promises for the future (58,59). Clinical results with

 

these two methods are promising, but there is no sufficient patient information that could support them having straightforward benefits (58,59).

## Prognosis

Looking at lung contusion as a separate pathology, its prognosis is largely determined by the severity of the contusion and previous pulmonary disease in the history (31). In most cases, it heals spontaneously within 5–7 days (32). Decreased respiratory function may develop as a complication. X-ray abnormalities usually disappear within 10 days, if they persist, it indicates the development of pneumonia (29). In more than 90% of patients affected, laboured breathing occurs in the first six months after the accident. In some cases this laboured breathing persists throughout the life of the patient. Chronic pulmonary disease may also develop as a consequence of severe contusion. Four years after the injury, the functional residual capacity of the lung decreases in the majority of contused patients. Pulmonary fibrosis develops 6 years after the injury (29,48). The appearance of early or late complications, however, is not specific to lung contusion injury, these may occur irrespective of earlier lung contusion.

## Summary

Lung contusion in trauma patients increases morbidity and mortality. It can be suspected on the basis of information in connection with the mechanism of the accident, as in the early stage of hospitalisation, the patient's symptoms and physical examination findings may not directly indicate an underlying lung contusion. Imaging test results may be misleading, radiological signs are often more serious than the actual clinical signs, our therapeutic steps are often uncertain and non-specific. The recognition of lung contusion is a cornerstone in the treatment of a polytraumatized patient, especially, since a direct force affecting the lung is not only important because of the immediate reduction in breathing surface and resulting hypoxic tissue damage, but also because lung parenchyma injuries may directly trigger inflammatory responses. Consequent pathomechanisms may, in interaction, lead to fatal multi-organ failure. The investigation of early inflammatory reactions is going to be a key issue for future research with the aim to identify an adequate biomarker that could assist in the timing of second-look surgeries in trauma patients.

Although the pathology of direct lung tissue damage and consequent function reduction, resulting from solitary lung injury is well-defined, further research is needed to identify the underlying biochemical processes and inflammatory response reactions. In order to further expand the array of our therapeutic options, it is inevitable to gain further knowledge relating to the underlying pathophysiological mechanisms. At present, our therapeutic capacities are only supportive, nonetheless, technological and methodological advances in respiration therapy have undoubtedly led to better morbidity and mortality statistics.

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## Footnote

*Conflict of Interest:* The authors have no conflicts of interest to declare.

## References

1. Ullman EA, Donley LP, Brady WJ. Pulmonary trauma emergency department evaluation and management. Emerg Med Clin North Am 2003;21:291-313.
2. Perl M, Gebhard F, Bruckner UB et al. Pulmonary contusion causes impairment of macrophage and lymphocyte immune functions and increases mortality associated with a subsequent septic challenge. Crit Care Med 2005;33:1351-8.
3. Deshmane SL, Kremlev S, Amini S, et al. Monocyte chemoattractant protein-1 (MCP-1): an overview. J Interferon Cytokine Res 2009;29:313-26.
4. Hoth JJ, Wells JD, Brownlee NA, et al. Toll-like receptor 4-dependent responses to lung injury in a murine model of pulmonary contusion. Shock 2009;31:376-81.
5. Viano DC, Lau IV. A viscous tolerance criterion for soft tissue injury assessment. J Biomech 1988;21:387-99.
6. Viano DC. Live fire testing: Assesing blunt impact and acceleration injury vulnerabilities. Mil Med 1991;156:589-95.
7. Bakowitz M, Bruns B, McCunn M. Acute lung injury and the acute respiratory distress syndrome in the injured patient. Scand J Trauma Resusc Emerg Med 2012;10;20:54.
8. Zhou D, Qiu J, Liang Y, et al. Epidemiological analysis of 9,596 patients with acute lung injury at Chinese Military

 

Hospitals. Exp Ther Med 2017;13:983-8.
9. Cohn SM. Pulmonary contusion: Review of the clinical entity, J Trauma 1997;42:973-9.
10. Karmy-Jones R, Jurkovich GJ. Blunt chest trauma. Curr Probl Surg 2004;41:211-380.
11. Balci AE, Balci TA, Eren S, et al. Unilateral post-traumatic pulmonary contusion: findings of a review. Surg Today 2005;35:205-10.
12. Simon B, Ebert J, Bokhari F, et al. EAST practice management workgroup for pulmonary contusion - flail chest. The Eastern Association for the Surgery of Trauma, 18 June 2008.
13. Simon B, Ebert J, Bokhari F, et al. Management of pulmonary contusion and flail chest: An Eastern Association for the Surgery of Trauma practice management guideline, Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg 2012;73:S351-61.
14. Lichtenberger JP, Kim AM, Fisher D, et al. Imaging of Combat-Related Thoracic Trauma - Blunt Trauma and Blast Lung Injury. Mil Med 2018;183:e89-e96.
15. Pfeifer R, Heussen N, Michalewicz E, et al. Incidence of adult respiratory distress syndrome in trauma patients: A systematic review and meta-analysis over a period of three decades. J Trauma Acute Care Surg 2017;83:496-506.
16. Zsolt C, László B. Intensive therapy and surgical treatment of bilateral diffuse lung contusion. Military Physician (Hun) 2007;59:59-63.
17. Ganie FA, Lone H, Lone GN, et al. Lung Contusion: A Clinico-Pathological Entity with Unpredictable Clinical Course. Bull Emerg Trauma 2013;1:7-16.
18. Nikischin W, Gerhardt T, Everett R, et al. A new method to analyze lung compliance when pressure-volume relationship is nonlinear, Am J Respir Crit Care Med 1998;158:1052-60.
19. Ranieri VM, Rubenfeld GD, Thompson BT, et al. Acute respiratory distress syndrome: the Berlin Definition. JAMA 2012;307:2526-33.
20. Cohn SM, Dubose JJ. Pulmonary contusion: an update on recent advances in clinical management. World J Surg 2010;34:1959-70.
21. Onat S, Ulku R, Avci A, et al. Urgent thoracotomy for penetrating chest trauma: analysis of 158 patients of a single center. Injury 2011;42:900-4.
22. Rubenfeld GD, Caldwell E, Peabody E, et al. Incidence and Outcomes of Acute Lung Injury. N Engl J Med 2005;353:1685-93.
23. Liman ST , Kuzucu A, Tastepe AI, et al. Chest injury due to blunt trauma. Eur J Cardiothorac Surg 2003;23:374-8.
24. de Lesquen H, Avaro JP, Gust L, et al. Surgical management for the first 48 h following blunt chest trauma: state of the art (excluding vascular injuries). Interact Cardiovasc Thorac Surg 2015;20:399-408.
25. Lerer LB, Knottenbelt JD. Preventable mortality following sharp penetrating chest trauma. J Trauma 1994;37:9-12.
26. Raghavendran K, Davidson BA, Huebschmann JC, et al. Superimposed gastric aspiration increases the severity of inflammation and permeability injury in a rat model of lung contusion. J Surg Res 2009;155:273-82.
27. Raghavendran K, Davidson BA, Helinski JD, et al. A rat model for isolated bilateral lung contusion from blunt chest trauma. Anesth Analg 2005;101:1482-9.
28. Boyd AD. Lung injuries. In: Hood RM, Boyd AD, Culliford AT. editors. Thoracic Trauma. Philadelphia: Saunders, 1989:153-55.
29. Allen GS, Coates NE. Pulmonary contusion: A collective review. Am Surg 1996;62:895-900.
30. Allen GS, Cox CS. Pulmonary contusion in children: Diagnosis and management, South Med J 1998;91:1099-106.
31. Sattler S, Maier RV. Pulmonary contusion. In: Karmy-Jones R, Nathens A, Stern EJ. editors. Thoracic Trauma and Critical Care. Berlin: Springer, 2002:159-60 and 235-43.
32. Livingston DH, Hauser CJ. Trauma to the chest wall and lung. McGraw-Hill Professional, 2003:532.
33. Sutyak JP, Wohltmann CD, Larson J. Pulmonary contusions and critical care management in thoracic trauma. Thorac Surg Clin 2007;17:11-23.
34. Prentice D, Ahrens T. Pulmonary complications of trauma. Crit Care Nurs Q 1994;17:24-33.
35. Miller DL, Mansour KA. Blunt traumatic lung injuries. Thorac Surg Clin 2007;17:57-61.
36. Rodriguez RM, Langdorf MI, Nishijima D, et al. Derivation and Validation of Two Decision Instruments for Selective Chest CT in Blunt Trauma: A Multicenter Prospective. Plos Med2015;12:e1001883.
37. Yamamoto L, Schroeder C, Morley D, et al. Thoracic trauma: The deadly dozen. Crit Care Nurs Q 2005;28:22-40.
38. Gavelli G, Canini R, Bertaccini P, et al. Traumatic injuries: Imaging of thoracic injuries, Eur Radiol 2002;12:1273-94.
39. Tovar JA. The lung and pediatric trauma. Semin Pediatr Surg 2008;17:53-9.
40. Border JR, Hopkinson BR, Schenk WG Jr. Mechanisms of pulmonary trauma: an experimental study. J Trauma 1968;8:47-62.

 

41. Nichols RT, Pearce HJ, Greenfield LJ. Effects of experimental pulmonary contusion on respiratory exchange and lung mechanics. Arch Surg 1968;96:723-30.
42. Wang ND, Stevens MH, Doty DB, et al. Blunt chest trauma: an experimental model for heart and lung contusion. J Trauma 2003;54:744-8.
43. Wang S, Ruan Z, Zhang J. A modified rat model of isolated bilateral pulmonary contusion, Exp Ther Med 2012;4:425-9.
44. Reade MC. Thoracic Trauma and Management of Ventilation in the Critically Injured Patient. Trauma and Combat Critical Care in Clinical Practice 2016:189-224.
45. Staub LJ, Biscaro RR, Kaszubowski E, et al. Chest ultrasonography for the emergency diagnosis of traumatic pneumothorax and haemothorax: A systematic review and meta-analysis. Injury 2018;49:457-66.
46. Lang P, Kulla M, Kerwagen F, et al. The role of whole-body computed tomography in the diagnosis of thoracic injuries in severely injured patients - a retrospective multi-centre study based on the trauma registry of the German trauma society (TraumaRegister DGU®). Scand J Trauma Resusc Emerg Med 2017;25:82.
47. Hoff SJ, Shotts SD, Eddy VA, et al. Outcome of isolated pulmonary contusion in blunt trauma patients. Am Surg 1994;60:138-42.
48. Wanek S, Mayberry JC. Blunt thoracic trauma: Flail chest, pulmonary contusion, and blast injury. Crit Care Clin 2004;20:71-81.
49. Boyle AJ, Mac Sweeney R, McAuley DF. Pharmacological treatments in ARDS; a state-of-the-art update. BMC Med 2013;11:166.
50. Gao Smith F, Perkins GD, Gates S. et al. Effect of intravenous β-2 agonist treatment on clinical outcomes in acute respiratory distress syndrome (BALTI-2): a multicentre, randomised controlled trial. Lancet 2012;379:229-35.
51. Miller AC, Elamin EM, Suffredini AF. Inhaled anticoagulation regimens for the treatment of smoke inhalation-associated acute lung injury: a systematic review. Crit Care Med 2014;42:413-9.
52. McAuley DF, Laffey JG, O'Kane CM, et al. Simvastatin to reduce pulmonary dysfunction in patients with acute respiratory distress syndrome: the HARP-2 RCT. Southampton (UK): NIHR Journals Library; 2018 Jan.
53. Truwit JD, Bernard GR, Steingrub J. Rosuvastatin for sepsis-associated acute respiratory distress syndrome. N Engl J Med 2014;370:2191-200.
54. Walkey AJ, Del Sorbo L, Hodgson CL et al. Higher PEEP versus Lower PEEP Strategies for Patients with Acute Respiratory Distress Syndrome. A Systematic Review and Meta-Analysis. Ann Am Thorac Soc 2017;14:S297-303.
55. Duggal A, Perez P, Golan E, et al. Safety and efficacy of noninvasive ventilation in patients with blunt chest trauma: a systematic review. Crit Care 2013;17:R142.
56. Curley G, Hayes M, Laffey JG. Can 'permissive' hypercapnia modulate the severity of sepsis-induced ALI/ARDS? Crit Care 2011;15:212.
57. Hashimoto S, Sanui M, Egi M, et al. The clinical practice guideline for the management of ARDS in Japan. J Intensive Care 2017;5:50.
58. Sud S, Friedrich JO, Neill KJ, et al. Effect of prone positioning during mechanical ventilation on mortality among patients with acute respiratory distress syndrome: a systematic review and meta-analysis. CMAJ 2014;186:E381-90.
59. Ried M, Bein T, Philipp A, et al. Extracorporeal lung support in trauma patients with severe chest injury and acute lung failure: a 10-year institutional experience. Crit Care 2013;17:R110.

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