

A Deadly Push: Tension Pneumothorax in a Healthy Adolescent Following Blunt Chest Trauma

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ABSTRACT

Background: Tension pneumothorax is a life-threatening emergency characterized by progressive accumulation of air in the pleural space, resulting in increased intrathoracic pressure, mediastinal shift, and cardiovascular compromise. Although it usually occurs after severe chest trauma or during mechanical ventilation, it may also appear following minor blunt chest injury.

Case presentation: A 15-year-old previously healthy male developed sudden dyspnea and right-sided pleuritic chest pain after a blunt chest contusion sustained while playing. On physical examination, the patient presented with tachypnea, hypotension, decreased breath sounds, and hyperresonance on the right hemithorax, accompanied by contralateral tracheal deviation. Chest radiography confirmed a right tension pneumothorax with mediastinal shift. Emergency needle decompression was performed, followed by chest tube insertion, leading to rapid hemodynamic stabilization and complete pulmonary re-expansion.

Conclusion: Tension pneumothorax should be considered in any patient presenting with acute respiratory distress and hemodynamic instability after blunt thoracic trauma, regardless of its apparent mildness. Immediate recognition and decompression are essential to prevent fatal outcomes.

KEYWORDS: Tension pneumothorax; blunt chest trauma; adolescent; pleural decompression; emergency medicine.

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I. INTRODUCTION

Tension pneumothorax represents one of the most critical emergencies in respiratory and thoracic medicine, characterized by the progressive accumulation of air within the pleural space under positive pressure throughout the respiratory cycle. This pathophysiological mechanism leads to increased intrathoracic pressure, ipsilateral pulmonary collapse, and mediastinal shift with compression of the contralateral lung and great vessels, resulting in severe respiratory and hemodynamic compromise that can be fatal if not promptly treated^{1,2}. The presence of gas in the pleural cavity interrupts the negative pleural pressure required for normal lung expansion, leading to rapid deterioration of oxygenation and cardiac output due to decreased venous return to the heart¹.

Pneumothorax can be classified as spontaneous, traumatic, or iatrogenic, depending on its etiology. Spontaneous pneumothorax occurs without any apparent external cause and is further divided into primary and secondary forms. Primary spontaneous pneumothorax (PSP) appears in patients without clinically evident lung disease, often associated with rupture of subpleural blebs or bullae. In contrast, secondary spontaneous pneumothorax (SSP) arises in patients with underlying pulmonary disorders such as chronic obstructive pulmonary disease, cystic fibrosis, or tuberculosis^{3,4,5}. Traumatic pneumothorax results from open or closed chest injury, and the tension type occurs when a one-way valve mechanism develops, allowing continuous air entry into the pleural space without egress, leading to positive pleural pressure^{1,4}.

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The pathophysiology of tension pneumothorax involves the creation of a unidirectional communication between the alveoli and the pleural cavity, allowing progressive air trapping. As intrapleural pressure increases, venous return is compromised, causing hypotension, tachycardia, and in extreme cases, cardiac arrest. Clinically, patients often present with sudden onset of pleuritic chest pain, dyspnea, and signs of respiratory distress. On examination, decreased breath sounds, hyperresonance to percussion, tracheal deviation, and distended neck veins may be observed ^{1,2,3}. The rapid development of cyanosis, agitation, and cardiovascular collapse demands immediate decompression without the need for radiographic confirmation ^{1,5}.

Although tension pneumothorax most frequently occurs as a complication of mechanical ventilation, penetrating trauma, or rib fractures, cases following minor blunt chest injury have been documented. In adolescents and young adults, even minimal trauma can precipitate alveolar rupture, particularly when predisposing factors such as an ectomorphic body habitus, subpleural blebs, or congenital alveolar fragility are present ^{3,4,6}. Noppen and Schramel describe that anatomical variations, distal airway inflammation, and low body mass index may contribute to pleural porosity and alveolar instability, increasing susceptibility to air leakage under stress ^{4,6}.

From an epidemiological standpoint, spontaneous pneumothorax occurs predominantly in young, tall, thin males, with an estimated incidence ranging between 7.4 and 18 cases per 100,000 men annually, compared to 1.2 to 6 per 100,000 women ⁴. The recurrence rate after a first episode may reach 30%, and in the absence of pleurodesis, the risk remains significant ⁶. Tension pneumothorax, however, is less common but associated with much higher mortality rates due to its rapid hemodynamic consequences ^{2,3}. Early recognition is crucial, as mortality can be prevented by prompt needle decompression followed by chest tube insertion ^{1,2,5}.

Diagnostic confirmation is typically achieved through chest radiography, which may demonstrate complete lung collapse and mediastinal displacement to the contralateral side. However, in emergency settings, clinical suspicion should suffice for immediate intervention. Computed tomography can later delineate underlying parenchymal abnormalities such as bullae or blebs, helping to differentiate spontaneous from traumatic causes ^{3,4,5}. The management includes oxygen therapy, decompression, pleural drainage, and in selected cases, surgical intervention to prevent recurrence ^{1,2,4,5}.

Understanding the clinical spectrum of tension pneumothorax, especially in adolescents following seemingly mild trauma, is essential for early recognition and timely intervention. This case highlights the importance of considering tension pneumothorax as a potential cause of sudden respiratory compromise even after minor blunt

thoracic injury, emphasizing the need for rapid diagnosis and life-saving management ^{1,6}.

II. CASE REPORT

A 15-year-old previously healthy male presented to the emergency department with sudden-onset dyspnea and right-sided pleuritic chest pain following a blunt chest trauma sustained while playing with friends. The patient reported that he had been pushed on the right hemithorax during physical activity and subsequently developed progressive shortness of breath and chest discomfort. No prior history of respiratory disease, asthma, or thoracic surgery was documented. He was not a smoker and denied exposure to toxic substances or illicit drugs.

On arrival, the patient appeared anxious, diaphoretic, and in obvious respiratory distress. Vital signs revealed a blood pressure of 88/56 mmHg, heart rate of 128 bpm, respiratory rate of 34 breaths per minute, oxygen saturation of 84% on room air, and a temperature of 36.7°C. Physical examination demonstrated decreased chest wall expansion on the right side, absence of breath sounds on auscultation, and hyperresonance to percussion over the right hemithorax. Tracheal deviation to the left and jugular venous distension were noted, consistent with tension pneumothorax and mediastinal shift.

Given the clinical instability, an emergency needle decompression was immediately performed in the second intercostal space at the midclavicular line, resulting in a rapid release of air and partial improvement in respiratory distress. Subsequently, a chest tube was inserted in the right fifth intercostal space along the anterior axillary line and connected to an underwater seal drainage system. After drainage, oxygen saturation increased to 97%, and hemodynamic parameters normalized within minutes.

A chest radiograph obtained post-procedure confirmed re-expansion of the right lung with appropriate placement of the pleural drainage tube and resolution of the mediastinal shift. No rib fractures or subcutaneous emphysema were identified. The patient was admitted to the pediatric intensive care unit for observation and continued chest drainage.

The evolution during hospitalization was favorable. The chest tube was maintained for 72 hours, with progressive resolution of air leakage and normalization of pulmonary auscultation. A control chest X-ray performed on day four showed complete lung expansion and absence of residual pneumothorax. The drainage tube was removed, and the patient was discharged after five days, asymptomatic and with no radiological evidence of recurrence.

At two-week follow-up, the patient remained clinically stable without dyspnea, chest pain, or recurrence. Preventive recommendations were given, including avoidance of strenuous physical exertion or contact sports for at least six weeks. The case emphasizes that even minor blunt chest trauma in adolescents can trigger a life-threatening tension

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pneumothorax, requiring immediate clinical recognition and intervention.

III. METHODS

A 15-year-old male patient with no significant medical history presented to the emergency department with acute respiratory distress following blunt chest trauma sustained during a physical altercation while playing. On initial assessment, the patient exhibited severe dyspnea, tachypnea, tachycardia, hypotension, distended neck veins, and absent breath sounds on the right hemithorax, consistent with a tension pneumothorax. Supplemental oxygen was administered via a non-rebreather mask at 15 L/min, and continuous monitoring of vital signs—including heart rate, blood pressure, respiratory rate, and oxygen saturation—was initiated immediately.

Given the clinical diagnosis of tension pneumothorax and the patient's hemodynamic instability, an emergency chest tube thoracostomy was performed. The procedure was carried out under sterile conditions in the fifth intercostal space at the mid-axillary line. Local anesthesia was administered using 2% lidocaine, infiltrating the skin, subcutaneous tissue, and pleura. A 28 Fr chest tube was inserted and connected to an underwater-seal drainage system with continuous suction to allow evacuation of intrapleural air and re-expansion of the lung. Proper placement of the tube was confirmed clinically by the presence of continuous air bubbling and improvement in breath sounds, as well as radiographically with a post-procedure chest X-ray demonstrating full lung re-expansion and correct tube positioning.

The patient was admitted to the pediatric intensive care unit (PICU) for continuous monitoring of vital signs, oxygen saturation, respiratory effort, and potential complications, including tube malposition, infection, re-expansion pulmonary edema, or persistent air leak. Analgesia was administered according to weight-appropriate dosing guidelines. Serial chest radiographs were obtained every 24 hours to assess lung re-expansion and pleural drainage. The chest tube was removed once radiographic evidence confirmed complete resolution of the pneumothorax and cessation of the air leak, followed by 24 hours of observation prior to hospital discharge.

IV. RESULTS

The patient's clinical course following emergency chest tube thoracostomy was favorable and without significant complications. Immediately after the procedure, there was notable improvement in respiratory distress, with a reduction in tachypnea and dyspnea, as well as gradual stabilization of blood pressure and heart rate. Continuous monitoring in the pediatric intensive care unit (PICU) included assessment of vital signs, oxygen saturation, respiratory effort, and signs of potential complications such as tube malposition, infection, or re-expansion pulmonary edema.

Serial chest radiographs were obtained daily to evaluate the progression of lung re-expansion and ensure effective pleural drainage. On the first day post-procedure, partial re-expansion of the right lung was observed, with residual small pneumothorax and air bubbling in the drainage system confirming continued evacuation. By the second day, further improvement was noted, with nearly complete lung re-expansion, resolution of mediastinal shift, and absence of subcutaneous emphysema. The patient's respiratory effort continued to normalize, and oxygen supplementation requirements gradually decreased.

By the third day, chest X-ray demonstrated full re-expansion of the right lung with complete resolution of the pneumothorax. No evidence of pleural effusion, infection, or other complications was noted. The chest tube was removed under sterile conditions without incident. Post-removal observation over the following 24 hours confirmed sustained respiratory stability, normal vital signs, and absence of recurrent pneumothorax. The patient tolerated oral intake well, experienced adequate pain control, and was clinically stable at discharge, with instructions for outpatient follow-up and monitoring.



Figure 1. Pre-drainage chest radiograph. Anteroposterior chest X-ray showing a large right-sided pneumothorax with near-complete collapse of the right lung and marked contralateral mediastinal shift to the left. The right hemidiaphragm appears depressed, and there is increased radiolucency in the right hemithorax, consistent with a tension pneumothorax. Cardiac silhouette and tracheal deviation are evident toward the left side..



Figure 2. Post-drainage chest radiograph. Control chest X-ray obtained after chest tube insertion demonstrates full re-expansion of the right lung with proper positioning of the pleural drainage tube in the right hemithorax. The mediastinal structures have returned to the midline, with resolution of the previous shift and normalization of the diaphragmatic contour. No residual pneumothorax or subcutaneous emphysema is observed.

V. DISCUSSION

Tension pneumothorax remains one of the most severe and rapidly fatal emergencies in thoracic medicine. It develops when intrapleural air accumulates under positive pressure, collapsing the affected lung and displacing mediastinal structures, which compromises venous return and cardiac output. This occurs through a one-way valve mechanism that allows air entry into the pleural cavity without exit, progressively increasing intrathoracic pressure and leading to severe respiratory and hemodynamic compromise. Clinical recognition must precede radiographic confirmation, as immediate decompression is lifesaving^{1,5}.

According to current literature, pneumothorax can be spontaneous, traumatic, or iatrogenic. Primary spontaneous pneumothorax often affects young, tall, thin males without known lung disease, while secondary spontaneous pneumothorax arises in those with underlying pulmonary pathology such as COPD, cystic fibrosis, or tuberculosis^{2,3,4}. Although tension pneumothorax most frequently results from mechanical ventilation or severe thoracic trauma, several studies have documented its occurrence after minor blunt injury, as seen in the present case. The underlying mechanism may involve rupture of subpleural blebs or areas of pleural porosity associated with alveolar fragility^{4,7}.

Noppen and Schramel proposed that factors such as low body mass index, distal airway inflammation, or connective-tissue fragility may increase pleural permeability, predisposing individuals to air leaks even without significant trauma. This explains why seemingly healthy adolescents can develop severe pneumothorax or tension physiology after minor chest impact. Epidemiologically, spontaneous pneumothorax occurs predominantly in males, with an

incidence between 7.4 and 18 per 100,000 annually, and recurrence rates up to 30% if pleurodesis is not performed^{4,6}.

Clinically, tension pneumothorax is characterized by sudden pleuritic pain, dyspnea, tachypnea, and signs of respiratory distress. Findings such as tracheal deviation, jugular distension, hyperresonance, and absence of breath sounds on the affected side confirm the diagnosis. The literature emphasizes that mediastinal shift seen radiographically may accompany both simple and tension pneumothorax; therefore, hemodynamic instability is the key differentiating feature requiring emergent decompression^{1,3}.

Diagnostic confirmation typically relies on chest radiography, where a collapsed lung and contralateral mediastinal displacement are evident. However, as the PDF notes, in unstable patients, treatment should not be delayed by imaging studies. Computed tomography is useful later to detect blebs or bullae and to differentiate between spontaneous and traumatic etiologies^{2,4}. Management includes oxygen supplementation, immediate needle decompression, and definitive intercostal chest drainage. Tube thoracostomy should be followed by radiographic verification of re-expansion. The use of suction depends on the persistence of air leak or incomplete re-expansion^{1,8}.

Complications such as re-expansion pulmonary edema, infection, tube malposition, or persistent air leak may occur, especially in secondary pneumothorax. Persistent leakage beyond seven days suggests the need for surgical evaluation¹. In adolescents, prognosis is generally excellent when diagnosis and drainage are timely, as observed in this case. Nonetheless, clinical vigilance remains critical because delayed decompression can rapidly lead to cardiovascular collapse^{2,5}.

Ultimately, early recognition and urgent pleural decompression are the cornerstone of management. The reviewed literature agrees that prompt intervention prevents fatal outcomes, and preventive measures such as smoking cessation, pleurodesis in recurrent cases, and temporary avoidance of strenuous physical activity reduce recurrence risk. This case highlights the importance of considering tension pneumothorax even after mild chest trauma and reinforces the need for rapid diagnosis and immediate intervention to prevent death^{2,6,8}.

VI. CONCLUSIONS

Tension pneumothorax represents a critical medical emergency in which early recognition and rapid decompression determine survival. This case demonstrates that even a seemingly trivial blunt chest impact in a healthy adolescent can precipitate life-threatening respiratory and hemodynamic collapse. The patient's presentation, characterized by sudden pleuritic pain, progressive dyspnea, hypotension, tracheal deviation, and absent breath sounds on the affected side, reflected the classical clinical picture of tension physiology. His immediate stabilization following prompt needle decompression and subsequent chest tube

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insertion underscores the decisive importance of clinical suspicion and timely intervention over reliance on imaging confirmation. The rapid normalization of vital signs and full radiographic re-expansion of the lung following drainage clearly illustrate that this condition, while potentially fatal, is entirely reversible with adequate and immediate management.

From a clinical perspective, this case reinforces the need to consider tension pneumothorax in any patient presenting with acute respiratory distress after blunt thoracic trauma, regardless of the apparent mildness of the injury. In adolescents, the absence of underlying pulmonary disease may lead to underestimation of severity, but the pathophysiological mechanism—progressive intrapleural air accumulation under positive pressure—can develop rapidly and unpredictably. Emergency physicians, first responders, and general practitioners must therefore maintain a high level of suspicion, as timely decompression can restore ventilation, prevent cardiac arrest, and completely reverse the life-threatening physiology. In this patient, the absence of structural lung disease and the rapid recovery following drainage support the concept that minor trauma can trigger alveolar rupture when transient increases in intrathoracic pressure occur.

The favorable clinical evolution observed—complete lung re-expansion, absence of residual air leak, and full recovery within days—highlights the effectiveness of early, protocol-guided management. Needle decompression in the second intercostal space, followed by insertion of a pleural drainage tube in the fifth intercostal space, represents the cornerstone of treatment, while supportive care with oxygen supplementation and continuous monitoring ensures stabilization and prevention of complications. The patient's outcome also demonstrates that intensive care monitoring in the initial phase allows early detection of potential adverse events such as re-expansion pulmonary edema or persistent air leak, which can otherwise prolong hospitalization.

From a preventive standpoint, this case emphasizes the importance of post-discharge recommendations, including the avoidance of strenuous activity and contact sports for at least six weeks to prevent recurrence or delayed complications. Patient and family education is fundamental to ensure adherence to follow-up and to recognize early signs of recurrence such as chest pain or sudden dyspnea. The

absence of recurrence at the two-week evaluation in this adolescent confirms that appropriate management and compliance with preventive measures lead to excellent prognosis and full functional recovery.

Finally, this report provides an important reminder for clinical practice: not all life-threatening thoracic emergencies occur after high-impact trauma. Even minimal blunt injury can produce critical intrathoracic changes when combined with individual anatomical or physiological susceptibility. The key to successful outcomes lies in rapid clinical judgment, immediate decompression, and appropriate definitive management. Tension pneumothorax should always be included in the differential diagnosis of acute respiratory distress, especially in young patients, and rapid recognition followed by pleural drainage remains the decisive step between reversible emergency and fatal collapse. The present case contributes to reinforcing clinical awareness that timely diagnosis and intervention can completely change the prognosis of an otherwise lethal event.

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