Spontaneous Pneumothorax: Clinical Assessment and Emergency Management for Paramedics and Emergency Healthcare Professionals

Mohammed Subayyil Al Harbi[1], Naif Mohammed Alsuhaymi[2], Talal Fares Awadh Almutairi[3], Abdullah Saleh Mohammed Alotaibi[4], Obaid Mohammed N. Alsubaie[5], Awad Mohammed Awad Alanazi[6], Saqer Owaid Awadh Alanazi[7], Saleh Maeish Fatini Oraibi[8], Mohammad Naseer Ali Alqahtani[9]

[1]National Guard Health Affairs, Al Qassim, Ministry of National Guard, Saudi Arabia
[2]National Guard Health Affairs, Ministry of National Guard, Saudi Arabia
[3]Ministry of National Guard, Saudi Arabia
[4]National Guard Health Affairs, Riyadh, Saudi Arabia
[5]Ministry of National Guard, Saudi Arabia
[6]Ministry of National Guard, Saudi Arabia
[7]Ministry of National Guard, Saudi Arabia
[8]Field Medical Command, Riyadh, Ministry of National Guard, Saudi Arabia
[9]National Guard Health Affairs, Ministry of National Guard, Saudi Arabia

Abstract

Background: Spontaneous pneumothorax is a common respiratory emergency characterized by the accumulation of air within the pleural cavity without preceding trauma or iatrogenic injury. It is classified into primary spontaneous pneumothorax (PSP), occurring in individuals without clinically evident lung disease, and secondary spontaneous pneumothorax (SSP), which develops in association with underlying pulmonary disorders.

Aim: This review aimed to provide a comprehensive overview of spontaneous pneumothorax, focusing on its etiology, epidemiology, pathophysiology, clinical presentation, diagnostic evaluation, contemporary management strategies, and prognosis among emergency healthcare professionals and paramedics.

Methods: A narrative literature review was conducted using current evidence and contemporary clinical guidelines addressing spontaneous pneumothorax. The review synthesized information regarding disease mechanisms, risk factors, diagnostic approaches, treatment recommendations, and long-term outcomes from published epidemiological studies and professional society guidelines.

Results: Spontaneous pneumothorax predominantly affects young men in PSP and older patients with underlying lung disease in SSP. Diagnosis relies on clinical assessment supported by chest radiography, ultrasonography, and computed tomography when indicated. Management ranges from conservative observation and oxygen therapy in stable patients to needle decompression, tube thoracostomy, chemical pleurodesis, and video-assisted thoracoscopic surgery in more severe or recurrent cases.

Conclusion: Spontaneous pneumothorax remains an important cause of acute respiratory distress requiring prompt recognition and individualized management. Advances in diagnostic imaging and evolving treatment strategies have improved patient outcomes. Early diagnosis, appropriate intervention, and recurrence prevention remain critical components of effective care, particularly in patients with secondary disease and high-risk clinical features.

Keywords: Spontaneous pneumothorax, primary spontaneous pneumothorax, secondary spontaneous pneumothorax.

Introduction

Spontaneous pneumothorax refers to the abnormal accumulation of air within the pleural cavity in the absence of traumatic injury or iatrogenic precipitating factors. This pathological condition disrupts the normal negative intrapleural pressure required for lung expansion, leading to partial or complete collapse of the affected lung. Spontaneous pneumothorax is conventionally categorized into two principal forms. Primary spontaneous pneumothorax (PSP) occurs in individuals who have no clinically apparent underlying pulmonary disease, whereas secondary spontaneous pneumothorax (SSP) develops in association with pre-existing respiratory disorders, including chronic obstructive pulmonary disease, cystic fibrosis, interstitial lung disease, and other forms of structural lung pathology. The distinction between these two entities is clinically significant because it influences disease severity, treatment decisions, recurrence risk, and overall patient prognosis. The clinical presentation of spontaneous pneumothorax is typically characterized by the abrupt onset of pleuritic chest pain accompanied by varying degrees of dyspnea. Patients may also exhibit tachycardia and other physiological manifestations resulting from compromised respiratory function. The severity of symptoms is influenced by several factors, including the extent of lung collapse, the patient's baseline pulmonary reserve, and the presence of underlying cardiopulmonary disease. Although many

cases remain hemodynamically stable, spontaneous pneumothorax has the potential to progress to tension pneumothorax, a critical and potentially fatal condition in which increasing intrapleural pressure causes significant respiratory compromise and cardiovascular instability. Such circumstances necessitate urgent recognition and immediate therapeutic intervention to prevent catastrophic outcomes [1][2].

The diagnosis of spontaneous pneumothorax is primarily guided by clinical assessment, with imaging studies serving a crucial confirmatory role. Chest radiography remains the most widely utilized diagnostic modality due to its accessibility and effectiveness in identifying pleural air and estimating the extent of lung collapse. In recent years, point-of-care ultrasonography has gained increasing acceptance as a rapid and highly sensitive bedside diagnostic tool, particularly in emergency and critical care settings where prompt diagnosis is essential. The integration of clinical findings with imaging results facilitates timely management and improves patient outcomes. Contemporary approaches to the management of spontaneous pneumothorax have undergone substantial evolution, reflecting a growing emphasis on individualized patient-centered care. Current treatment strategies are determined by several key factors, including the patient's clinical stability, the size of the pneumothorax, the presence of recurrent episodes, and whether the condition represents a primary or secondary form of disease [1][2]. Recent evidence has challenged the routine use of invasive interventions in all cases, particularly among clinically stable patients with small primary spontaneous pneumothoraces. Notably, the 2023 British Thoracic Society guidelines endorse a conservative management approach for selected patients, supported by clinical trials demonstrating spontaneous resolution rates approaching 94% within an eight-week period without the need for procedural intervention [3][4].

In contrast, invasive treatment remains an essential component of care for patients with recurrent, persistent, or complicated pneumothoraces. Surgical management, commonly performed through video-assisted thoracoscopic surgery or thoracotomy combined with pleurodesis, has emerged as the preferred strategy for reducing recurrence and achieving durable pleural symphysis. These recommendations are particularly relevant for patients with secondary spontaneous pneumothorax and those possessing high-risk clinical features, consistent with both British and European professional guidelines [3]. Consequently, the management of spontaneous pneumothorax increasingly reflects a balance between minimizing unnecessary procedures and ensuring definitive treatment when clinically indicated. Understanding the etiology, epidemiology, diagnostic principles, and evolving therapeutic approaches of this condition is essential for optimizing patient care and improving long-term clinical outcomes.

Etiology

Spontaneous pneumothorax is broadly classified into two major categories, namely primary spontaneous pneumothorax (PSP) and secondary spontaneous pneumothorax (SSP), based on the absence or presence of underlying pulmonary pathology. This classification is fundamental because it reflects distinct pathogenic mechanisms, epidemiological characteristics, clinical presentations, and therapeutic considerations. While both forms involve the accumulation of air within the pleural space resulting in varying degrees of lung collapse, the factors contributing to their development differ substantially. Primary spontaneous pneumothorax occurs in individuals who do not have clinically recognized lung disease. It is most frequently observed among young adults, particularly those between 15 and 40 years of age. Although these individuals are generally considered healthy, accumulating evidence suggests that subtle structural abnormalities may exist within the lungs despite the absence of overt respiratory disease. The most widely accepted mechanism involves the rupture of small subpleural blebs or bullae located near the lung apex, allowing air to escape into the pleural cavity. This condition demonstrates a strong demographic predilection for tall, thin males, a pattern that may be related to increased mechanical stress within the apical regions of the lungs. Additional risk factors such as cigarette smoking significantly increase susceptibility by promoting inflammatory changes and weakening alveolar structures. Genetic predisposition and familial clustering have also been reported, suggesting that hereditary factors may contribute to disease development in selected individuals. Epidemiological data indicate that the annual incidence of PSP ranges from 7.4 to 24 cases per 100,000 men and from 1.2 to 6 cases per 100,000 women, highlighting a substantially higher occurrence among males [5].

Secondary spontaneous pneumothorax, in contrast, develops in patients with established pulmonary disorders that compromise the structural integrity of the lung parenchyma. The presence of underlying lung disease increases the likelihood of alveolar rupture and subsequent leakage of air into the pleural space. Chronic obstructive pulmonary disease represents the most common underlying condition associated with SSP, particularly in older adults with advanced emphysematous changes. However, numerous other respiratory diseases have been implicated, including severe asthma, cystic fibrosis, necrotizing pneumonia, pulmonary abscesses, tuberculosis, and opportunistic infections such as Pneumocystis jirovecii pneumonia. Malignant pulmonary lesions and a wide spectrum of interstitial lung diseases, including idiopathic pulmonary fibrosis, sarcoidosis, and lymphangioleiomyomatosis, are also recognized causes of SSP. Beyond primary respiratory disorders, several systemic diseases and uncommon conditions may predispose individuals to secondary spontaneous pneumothorax. Connective tissue disorders such as Marfan syndrome, Ehlers-Danlos syndrome, and rheumatoid arthritis can weaken pulmonary connective tissue architecture, thereby increasing vulnerability to pleural air leakage. Additional causes include pulmonary infarction, foreign body aspiration, and rare hereditary syndromes such as Birt-Hogg-Dubé syndrome. Furthermore, catamenial pneumothorax, an uncommon but clinically important subtype associated with thoracic endometriosis, occurs predominantly in women of reproductive age and is characterized by recurrent episodes that coincide with the menstrual cycle. Compared with PSP, SSP generally affects older individuals and is associated with greater clinical severity because of

the reduced respiratory reserve resulting from pre-existing pulmonary dysfunction. Even relatively small pneumothoraces can produce significant respiratory distress and hemodynamic compromise in affected patients. Consequently, SSP is associated with higher rates of hospitalization, complications, and mortality. Epidemiological investigations have reported an annual incidence of approximately 6.3 cases per 100,000 men and 2.0 cases per 100,000 women, underscoring its substantial clinical burden despite its lower frequency relative to primary spontaneous pneumothorax [6]. Understanding the diverse etiological factors responsible for both PSP and SSP is essential for accurate diagnosis, risk stratification, preventive strategies, and the selection of appropriate management approaches.

Epidemiology

Spontaneous pneumothorax represents a significant respiratory condition with distinct epidemiological patterns influenced by age, sex, environmental exposures, and underlying pulmonary health. Although it can occur across all age groups, the condition is considerably more prevalent among adults than children and demonstrates a marked male predominance. The incidence of spontaneous pneumothorax varies according to whether the condition is classified as primary spontaneous pneumothorax (PSP) or secondary spontaneous pneumothorax (SSP), reflecting differences in underlying pathophysiological mechanisms and population characteristics. Primary spontaneous pneumothorax predominantly affects otherwise healthy individuals and is particularly common among adolescents and young adults. Epidemiological studies conducted in the United States have reported annual incidence rates ranging from 7.4 to 18 cases per 100,000 population among men, compared with 1.2 to 6.0 cases per 100,000 among women [7][8]. This pronounced gender disparity has been consistently observed across diverse populations and geographic regions. Several biological and anatomical factors have been proposed to explain the increased susceptibility among males, including differences in body habitus, pulmonary mechanics, and patterns of tobacco use. The condition is especially prevalent among tall, thin individuals, particularly young men during late adolescence and early adulthood, a period characterized by rapid growth and increased mechanical stress on the apical regions of the lungs [7]. Smoking remains one of the most significant modifiable risk factors associated with PSP. Numerous epidemiological investigations have demonstrated a strong dose-dependent relationship between tobacco exposure and the development of spontaneous pneumothorax. Cigarette smoking contributes to inflammatory and structural alterations within the respiratory system, increasing the likelihood of subpleural bleb formation and subsequent rupture. As a result, smokers experience a substantially higher risk of developing PSP compared with nonsmokers, regardless of age or sex [7]. This association highlights the importance of smoking cessation as a critical preventive strategy for reducing disease occurrence and recurrence.

Secondary spontaneous pneumothorax occurs in individuals with pre-existing pulmonary disease and generally affects older populations. The annual incidence of SSP has been estimated at approximately 6.3 cases per 100,000 men and 2.0 cases per 100,000 women. Although less common than PSP in certain age groups, SSP is associated with a greater burden of illness due to the presence of underlying respiratory pathology and diminished pulmonary reserve. Patients with chronic obstructive pulmonary disease, interstitial lung disorders, cystic fibrosis, pulmonary infections, and other structural lung abnormalities comprise the majority of affected individuals. Consequently, SSP frequently results in more severe clinical manifestations, prolonged hospitalization, and increased healthcare utilization. In the pediatric population, spontaneous pneumothorax is relatively uncommon. Epidemiological estimates indicate annual incidence rates of approximately 4.0 cases per 100,000 boys and 1.1 cases per 100,000 girls, demonstrating a similar male predominance to that observed in adults [8]. Despite its rarity, pediatric spontaneous pneumothorax remains clinically important because delayed diagnosis can lead to significant respiratory compromise. The broader healthcare implications of spontaneous pneumothorax are reflected in contemporary epidemiological trends. A large-scale study conducted in the United Kingdom between 2017 and 2023 documented approximately 72,000 hospital admissions attributable to spontaneous pneumothorax, emphasizing the considerable burden this condition imposes on healthcare systems [9]. These admissions contribute substantially to emergency department utilization, inpatient care requirements, procedural interventions, and healthcare expenditures. Recurrence constitutes one of the most important epidemiological and clinical challenges associated with spontaneous pneumothorax. In patients with PSP, recurrence rates approach 32%, with approximately 21% to 54% of recurrent episodes occurring within one to two years following the initial event [9]. This high recurrence frequency underscores the need for long-term follow-up and consideration of preventive therapeutic strategies in selected patients. Although recurrence rates in SSP are generally lower, ranging from 13% to 39%, the coexistence of significant pulmonary disease often complicates management and contributes to higher morbidity and poorer overall outcomes [9]. These epidemiological findings highlight the importance of risk assessment, early intervention, and recurrence prevention in reducing the clinical and healthcare burden of spontaneous pneumothorax.

Pathophysiology

The pathophysiological basis of spontaneous pneumothorax involves the abnormal accumulation of air within the pleural cavity, resulting in the loss of normal negative intrapleural pressure and subsequent partial or complete lung collapse. This process arises from a complex interplay of mechanical forces, structural abnormalities, inflammatory changes, and microscopic defects affecting the alveolar-pleural interface. Although the exact mechanisms may vary between primary spontaneous pneumothorax (PSP) and secondary spontaneous pneumothorax (SSP), both forms

ultimately result from the leakage of air from the pulmonary parenchyma into the pleural space. A fundamental event in the development of spontaneous pneumothorax is the disruption of the alveolar-pleural barrier. This disruption commonly occurs when transpulmonary pressure rises beyond the capacity of alveolar structures to withstand mechanical stress. Acute increases in intrathoracic pressure generated during activities such as forceful coughing, sneezing, straining, heavy exertion, or Valsalva maneuvers can create substantial pressure gradients across the alveolar walls. When these pressure changes exceed the tensile strength of the alveolar tissue, rupture may occur, allowing air to escape into the pulmonary interstitium and subsequently enter the pleural cavity [10]. The resulting loss of pleural integrity leads to progressive lung collapse and impairment of normal respiratory mechanics. Both PSP and SSP share the common mechanism of elevated alveolar pressure relative to surrounding pulmonary interstitial pressure. This pressure differential facilitates alveolar rupture and promotes the movement of air toward the pleural space. However, the susceptibility of alveoli to rupture is strongly influenced by pre-existing structural abnormalities within the lung and pleura. Weaknesses in the visceral pleura, including subpleural blebs, bullae, areas of localized necrosis, and connective tissue defects, significantly increase the risk of air leakage. Despite extensive research, the precise biological processes responsible for the formation and rupture of these lesions remain incompletely understood. Interestingly, spontaneous pneumothorax has been documented in association with intact bullae that lack obvious pleural defects, suggesting that additional microscopic mechanisms may contribute to disease development [11][12].
Emerging histopathological evidence has expanded understanding of these mechanisms by identifying alterations at the cellular level. Studies utilizing scanning electron microscopy have demonstrated desquamation and sloughing of pleural mesothelial cells, which may weaken the pleural surface and facilitate the passage of air into the pleural cavity [11][12]. These findings suggest that spontaneous pneumothorax is not solely the consequence of gross structural rupture but may also involve subtle microscopic defects that compromise pleural integrity. Such observations have shifted contemporary perspectives toward a more comprehensive understanding that incorporates both mechanical and cellular contributors to disease pathogenesis. In primary spontaneous pneumothorax, rupture most commonly occurs at sites of pre-existing weakness within the visceral pleura, particularly subpleural blebs and bullae. These abnormalities are identified in up to 77% of affected individuals through radiological and histopathological examination. The development of these lesions has been associated with alveolar overdistension, localized ischemic injury, inflammatory remodeling, and smoking-related pulmonary damage [10]. Tobacco exposure is believed to play a critical role by inducing chronic airway inflammation and promoting structural degeneration of alveolar tissues, thereby increasing vulnerability to rupture. Secondary spontaneous pneumothorax develops in the presence of established pulmonary disease and results from more extensive pathological disruption of lung architecture. Conditions such as chronic obstructive pulmonary disease, interstitial lung diseases, necrotizing pulmonary infections, and other destructive respiratory disorders cause alveolar wall thinning, fibrosis, cavitation, and focal tissue necrosis. These pathological alterations weaken the alveolar-pleural interface and markedly increase the likelihood of air leakage into the pleural space [6]. Because patients with SSP often have reduced pulmonary reserve, even small volumes of pleural air can produce substantial respiratory compromise.
Another important mechanism implicated in spontaneous pneumothorax is the formation of alveolar-pleural fistulae and bronchopleural fistulae. These abnormal communications establish direct pathways between the airways or alveoli and the pleural cavity, allowing continuous air leakage. Such fistulous tracts are particularly common in secondary spontaneous pneumothorax due to extensive pulmonary destruction but may also occur in severe or prolonged cases of PSP. Persistent air leaks resulting from these communications often complicate clinical management and may necessitate surgical intervention. Current understanding of spontaneous pneumothorax therefore extends beyond the traditional concept of pressure-induced alveolar rupture. Contemporary evidence highlights the combined influence of structural pleural abnormalities, airway remodeling, inflammatory injury, connective tissue defects, mesothelial cell dysfunction, and fistula formation in initiating and perpetuating the disease process. These multifaceted mechanisms collectively contribute not only to the onset of spontaneous pneumothorax but also to its recurrence and long-term clinical consequences.

History and Physical

The clinical presentation of spontaneous pneumothorax is typically characterized by an abrupt onset of symptoms that frequently occur in the absence of any identifiable precipitating event. Unlike traumatic pneumothorax, spontaneous pneumothorax often develops while the individual is at rest, with no preceding history of significant physical exertion or injury. The most common presenting complaint is the sudden appearance of sharp, unilateral chest pain that is pleuritic in nature, meaning that it intensifies during deep inspiration, coughing, or other respiratory movements. This chest discomfort is often accompanied by acute dyspnea, the severity of which varies according to the size of the pneumothorax, the degree of lung collapse, and the patient's baseline pulmonary function. Patients with primary spontaneous pneumothorax may experience relatively mild symptoms despite significant radiographic findings, particularly when pulmonary reserve is preserved. In contrast, individuals with secondary spontaneous pneumothorax frequently present with more severe respiratory distress because pre-existing lung disease limits compensatory mechanisms. Consequently, even a relatively small pneumothorax may result in marked shortness of breath, hypoxemia, and functional impairment in patients with underlying respiratory pathology [8][9].
Physical examination findings depend largely on the extent of pleural air accumulation. Tachycardia is among the most commonly observed clinical signs and may reflect both respiratory compromise and physiological stress. In cases

involving small pneumothoraces, particularly those occupying less than 15% of the hemithorax, physical examination may reveal few or no detectable abnormalities. As a result, clinicians must maintain a high index of suspicion when evaluating patients with characteristic symptoms despite an apparently normal examination. In larger pneumothoraces, more prominent clinical findings become evident. Inspection may demonstrate reduced chest wall expansion on the affected side due to impaired lung inflation. Auscultation often reveals diminished or absent breath sounds over the involved hemithorax, while percussion may produce a hyperresonant note resulting from the presence of excess intrapleural air. Palpation may identify decreased tactile fremitus, reflecting reduced transmission of vocal vibrations through the collapsed lung. Additional findings such as jugular venous distension and pulsus paradoxus may develop when intrathoracic pressure significantly impairs venous return and cardiopulmonary function. A particularly serious complication is tension pneumothorax, a medical emergency characterized by progressive accumulation of pleural air under pressure. This condition causes mediastinal displacement, compression of the contralateral lung, and impaired venous return to the heart. Clinically, patients may present with severe respiratory distress, profound hypoxemia, hypotension, tachycardia, and signs of obstructive shock. Tracheal deviation away from the affected side is considered a classic but often late finding. Prompt recognition of these manifestations is essential because delayed intervention can rapidly lead to cardiovascular collapse and death [13]. Consequently, careful assessment of both patient history and physical examination findings remains fundamental for the timely diagnosis and management of spontaneous pneumothorax.

Evaluation

The evaluation of spontaneous pneumothorax begins with a thorough clinical assessment that integrates patient history, symptom presentation, and physical examination findings. The diagnosis is frequently suspected in patients who present with the sudden onset of pleuritic chest pain and dyspnea, particularly when accompanied by characteristic physical findings such as diminished breath sounds and unilateral hyperresonance. However, because clinical manifestations may vary according to the size of the pneumothorax and the presence of underlying pulmonary disease, imaging studies play a crucial role in confirming the diagnosis and guiding subsequent management decisions. Chest radiography remains the primary and most widely utilized imaging modality for the evaluation of spontaneous pneumothorax. Characteristic radiographic findings include visualization of the visceral pleural line separated from the chest wall by a radiolucent area that lacks normal pulmonary vascular markings, indicating the presence of free air within the pleural space. Upright posteroanterior chest radiographs are generally preferred because they facilitate clearer identification of pleural air accumulation. Although expiratory radiographs were historically employed to enhance diagnostic accuracy, contemporary evidence suggests that expiratory imaging does not significantly improve the detection rate of spontaneous pneumothorax compared with standard inspiratory films. Consequently, routine use of expiratory radiography is no longer universally recommended. Point-of-care ultrasonography has emerged as a valuable diagnostic tool in both emergency and critical care settings. Ultrasound offers the advantages of rapid bedside assessment, absence of ionizing radiation, and high diagnostic sensitivity. Several studies have demonstrated that thoracic ultrasonography may be more sensitive than conventional chest radiography in detecting pneumothorax, particularly in acute care environments. Findings such as the absence of lung sliding, loss of comet-tail artifacts, and identification of the lung point sign can strongly support the diagnosis. Nevertheless, despite their effectiveness in detecting pleural air, both chest radiography and ultrasonography have limitations in accurately determining the precise size and extent of the pneumothorax [13][14].

Computed tomography (CT) of the chest represents the most sensitive and specific imaging modality for diagnosing spontaneous pneumothorax. Its use is particularly valuable when clinical suspicion remains high despite negative or inconclusive findings on initial imaging studies. CT scanning provides detailed visualization of pleural air collections and can identify underlying structural abnormalities such as blebs, bullae, cystic lesions, or parenchymal lung disease that may contribute to the development of primary or secondary spontaneous pneumothorax. Although CT is not routinely required in all patients, it serves an important role in complex diagnostic scenarios and preoperative assessment. Laboratory investigations are generally not required to establish the diagnosis of spontaneous pneumothorax. However, arterial blood gas analysis may provide useful information regarding the physiological impact of the condition, particularly in patients with severe respiratory compromise. Findings may include acute respiratory alkalosis resulting from hyperventilation and an increased alveolar-arterial oxygen gradient due to impaired gas exchange. These abnormalities are especially relevant in cases complicated by tension physiology, where progressive cardiopulmonary compromise necessitates urgent intervention [14]. Therefore, the evaluation of spontaneous pneumothorax relies on a combination of clinical judgment and appropriate imaging modalities to ensure accurate diagnosis, assessment of severity, and timely therapeutic management.

Treatment / Management

The management of spontaneous pneumothorax is directed toward two principal objectives: the elimination of air from the pleural cavity to facilitate lung re-expansion and the prevention of future recurrences. Contemporary treatment strategies are determined by several factors, including the size of the pneumothorax, the severity of symptoms, the patient's hemodynamic stability, the presence of underlying pulmonary disease, and whether the condition is classified as primary spontaneous pneumothorax (PSP) or secondary spontaneous pneumothorax (SSP). Current recommendations from the American College of Chest Physicians (ACCP) and the British Thoracic Society

provide evidence-based guidance for adult patients and emphasize individualized treatment approaches based on clinical presentation and risk stratification. Initial management begins with a thorough assessment of respiratory and hemodynamic stability. Supplemental oxygen administration plays a central role in the early treatment of spontaneous pneumothorax. In most patients, high-concentration oxygen delivered through a nonrebreather mask is recommended alongside continuous cardiopulmonary monitoring. Oxygen therapy accelerates the reabsorption of intrapleural air by increasing the diffusion gradient for nitrogen between the pleural space and pulmonary capillaries. Studies have demonstrated that oxygen supplementation can increase the rate of pleural air absorption by as much as fourfold compared with the spontaneous absorption rate of approximately 1% to 2% of pneumothorax volume per day in patients breathing room air. Consequently, oxygen administration remains a simple yet highly effective adjunctive measure in both conservative and interventional management strategies [15][16][17].

Patients who exhibit clinical instability require immediate intervention. Severe dyspnea, hypoxemia, hypotension, altered mental status, or signs suggestive of tension pneumothorax constitute medical emergencies that necessitate urgent decompression. Tension pneumothorax results from progressive accumulation of intrapleural air under pressure, leading to lung collapse, mediastinal displacement, impaired venous return, and cardiovascular compromise. In such situations, emergent needle decompression serves as a lifesaving procedure that rapidly relieves intrathoracic pressure. This intervention is typically performed as a temporary measure before definitive management through tube thoracostomy placement, which provides continuous evacuation of pleural air and facilitates sustained lung re-expansion [15][16][17]. Management of primary spontaneous pneumothorax depends largely on symptom severity and pneumothorax size. In clinically stable individuals experiencing a first episode of small PSP, conservative treatment is generally recommended. This approach consists of supplemental oxygen administration and careful observation, typically for a minimum period of six hours. During this observation interval, serial clinical assessments and repeat chest radiography are used to evaluate disease progression and ensure stability. If follow-up imaging confirms that the pneumothorax remains unchanged or demonstrates improvement, and if the patient has reliable access to medical care, discharge may be considered with detailed instructions regarding symptom monitoring and a scheduled reassessment within 24 hours. Recent evidence supporting conservative management has challenged the historical tendency toward routine intervention, particularly in stable patients with minimal symptoms. The British Thoracic Society has further expanded the role of observation by suggesting that selected asymptomatic patients with larger primary spontaneous pneumothoraces may also be suitable candidates for conservative treatment without immediate procedural intervention. This recommendation reflects growing evidence demonstrating that spontaneous resolution can occur in appropriately selected patients while avoiding the risks associated with invasive procedures. Nevertheless, careful patient selection and close follow-up remain essential components of this approach.

For patients presenting with large or symptomatic PSP, more active intervention is generally required. The ACCP recommends needle aspiration using a small-bore catheter measuring 14 French or less as an initial therapeutic option. Aspiration aims to remove pleural air and restore normal lung expansion while minimizing procedural morbidity. If aspiration fails to achieve adequate lung re-expansion or if symptoms persist, hospital admission with chest tube placement is recommended. Thoracostomy tubes ranging from 16 to 22 French are commonly employed to provide continuous drainage and facilitate complete resolution of the pneumothorax. Surgical intervention becomes increasingly important in cases involving recurrent episodes, persistent air leaks, bilateral pneumothoraces, or failure of conservative and catheter-based treatments. Video-assisted thoracoscopic surgery (VATS) has emerged as the preferred surgical approach because of its minimally invasive nature and favorable outcomes. During VATS, surgeons can perform bullectomy to remove identifiable blebs or bullae, pleurectomy to excise portions of the pleural lining, and mechanical pleurodesis through pleural abrasion. These procedures promote adhesion between the visceral and parietal pleura, thereby reducing the likelihood of future air accumulation. Compared with traditional thoracotomy, VATS is associated with reduced postoperative pain, shorter hospital stays, faster recovery, and excellent efficacy in preventing recurrence. Patients experiencing recurrent PSP are generally admitted for thoracostomy tube placement as an initial stabilizing measure while arrangements are made for definitive surgical management. However, not all individuals are suitable candidates for surgery. In patients who decline operative intervention, possess significant comorbidities that increase surgical risk, or receive care in facilities where thoracoscopic surgery is unavailable, chemical pleurodesis offers a valuable alternative. This procedure involves the instillation of sclerosing agents such as doxycycline, minocycline, or talc through the thoracostomy tube. These agents induce a controlled inflammatory response that promotes pleural fibrosis and adhesion formation, effectively obliterating the pleural space and decreasing recurrence risk. Chemical pleurodesis has demonstrated favorable outcomes in appropriately selected patients and remains an important component of nonoperative management [16][17].

The treatment approach for secondary spontaneous pneumothorax is generally more aggressive because affected patients often have significantly reduced pulmonary reserve and are at increased risk of adverse outcomes. Both the ACCP and the British Thoracic Society recommend hospital admission for all patients with SSP, regardless of pneumothorax size. Supplemental oxygen therapy and serial chest radiographic evaluations are integral components of management. Observation alone is generally discouraged because even small pneumothoraces can produce substantial respiratory compromise in individuals with underlying lung disease. Consequently, placement of a pleural catheter or thoracostomy tube is recommended when the pneumothorax is large, symptomatic, bilateral, or associated with significant physiological impairment. Because SSP frequently occurs in the context of complex pulmonary pathology, multidisciplinary management is often required. Consultation with thoracic surgery specialists is

recommended once the patient has been stabilized and adequate pleural drainage has been established. Specialist involvement facilitates evaluation for definitive interventions, including surgical repair, pleurodesis, or treatment of the underlying pulmonary disorder. Overall, contemporary management of spontaneous pneumothorax emphasizes prompt stabilization, individualized treatment selection, minimization of recurrence risk, and optimization of long-term respiratory outcomes through a combination of conservative, procedural, and surgical strategies [3].

Prognosis

The prognosis of spontaneous pneumothorax varies considerably according to whether the condition is classified as primary spontaneous pneumothorax (PSP) or secondary spontaneous pneumothorax (SSP), as well as the patient's underlying pulmonary status, treatment strategy, and risk of recurrence. Overall, patients with PSP generally experience favorable clinical outcomes, whereas those with SSP often face a more complicated disease course characterized by increased morbidity, prolonged hospitalization, and greater mortality risk. Primary spontaneous pneumothorax is typically associated with excellent survival outcomes because it occurs in individuals who usually possess normal baseline pulmonary function and adequate physiological reserve. Most patients recover completely following appropriate treatment, and mortality directly attributable to PSP is exceedingly uncommon. Despite these favorable survival outcomes, recurrence remains one of the most important long-term clinical challenges. Contemporary epidemiological investigations have demonstrated that recurrence can occur even after complete radiographic resolution of the initial episode. A large population-based study conducted in South Korea reported a five-year recurrence rate of approximately 20.3%, with recurrence occurring significantly more frequently among men than women. The reported recurrence rates were approximately 20.8% in males compared with 10.9% in females, reinforcing the recognized influence of sex-related risk factors on disease recurrence [18]. Interestingly, findings from this study suggested that patients managed conservatively experienced lower recurrence rates than those who underwent early surgical intervention, with recurrence observed in approximately 7.9% of conservatively treated patients compared with 23.7% among surgically managed individuals [18]. Although these findings differ from traditional assumptions regarding the protective role of surgery, they emphasize the importance of individualized treatment planning and careful patient selection when determining therapeutic strategies. Such observations also highlight the complexity of recurrence risk assessment and the need for continued investigation into factors influencing long-term outcomes.

Systematic reviews and meta-analyses have consistently reported overall recurrence rates approaching 32%, with the majority of recurrent episodes occurring during the first year following the initial pneumothorax [18]. Several factors have been identified as significant predictors of recurrence. Younger age, particularly individuals younger than 20 years, male sex, the presence of radiologically or surgically identified blebs and bullae, and management approaches that do not include definitive preventive interventions have all been associated with increased recurrence risk. Consequently, patients possessing these characteristics often require closer follow-up and consideration of strategies designed to minimize future episodes. Surgical management has demonstrated considerable effectiveness in reducing recurrence among patients with PSP. Procedures such as video-assisted thoracoscopic surgery (VATS) combined with pleurodesis and bullectomy provide durable long-term results by eliminating structural sources of air leakage and promoting pleural adhesion formation. These interventions frequently reduce recurrence rates to below 10%, making them valuable options for patients with recurrent disease, persistent air leaks, or other high-risk features. As a result, surgical therapy plays a critical role in improving long-term outcomes and reducing the burden of repeated hospital admissions. In contrast, the prognosis of secondary spontaneous pneumothorax is generally less favorable because it occurs in individuals with pre-existing pulmonary disease. The underlying respiratory pathology not only contributes to pneumothorax development but also limits the patient's ability to tolerate even modest reductions in lung function. Consequently, SSP is associated with higher rates of clinical deterioration, treatment complications, healthcare utilization, and mortality. A large retrospective cohort study reported an in-hospital mortality rate of approximately 2.3% and an overall recurrence rate of 9%, with both outcomes strongly influenced by factors such as poor functional capacity, significant comorbid illnesses, and larger pneumothorax size at presentation [19].

Complications are also considerably more common in SSP than in PSP. Evidence indicates that as many as 41% of patients with SSP experience adverse clinical events during their disease course. Among these complications, persistent air leaks represent a particularly significant challenge, occurring in approximately 13.5% of cases and often prolonging hospitalization while increasing the need for invasive interventions [19]. Furthermore, patients with SSP frequently experience slower recovery, greater symptom burden, and reduced quality of life compared with individuals affected by PSP. Surgical intervention remains an important therapeutic option in selected cases of SSP and has been associated with favorable long-term outcomes. Video-assisted thoracoscopic surgery can significantly reduce recurrence rates, which have been reported to range from 0% to 15.8% following definitive surgical management. However, operative risk must be carefully considered because mortality associated with VATS in patients with SSP is estimated to range between 2% and 4%. Outcomes are particularly influenced by the nature of the underlying pulmonary disease. Patients with interstitial lung disease exhibit substantially higher postoperative mortality rates, reaching as high as 15%, whereas individuals with chronic obstructive pulmonary disease generally experience more favorable surgical outcomes. Therefore, prognosis in spontaneous pneumothorax depends not only on the occurrence of pleural air accumulation itself but also on patient-specific characteristics, underlying respiratory health, recurrence risk, and the appropriateness of therapeutic interventions employed throughout the course of care [18][19].

Conclusion

Spontaneous pneumothorax is a clinically significant respiratory disorder that requires rapid recognition, accurate diagnosis, and evidence-based management to prevent complications and improve patient outcomes. The condition encompasses two distinct entities, primary spontaneous pneumothorax and secondary spontaneous pneumothorax, each with unique etiological factors, clinical characteristics, and prognostic implications. Although many patients with PSP experience favorable outcomes and complete recovery, recurrence remains a substantial concern that necessitates careful follow-up and individualized treatment planning. In contrast, SSP is associated with greater morbidity, prolonged hospitalization, increased healthcare utilization, and higher mortality because of underlying pulmonary disease. Advances in diagnostic imaging, particularly point-of-care ultrasonography and computed tomography, have enhanced diagnostic accuracy and facilitated timely intervention. Contemporary management strategies emphasize patient-centered care, balancing conservative observation with invasive procedures according to clinical stability, symptom severity, recurrence risk, and underlying lung pathology. Surgical interventions, especially video-assisted thoracoscopic surgery with pleurodesis, play a pivotal role in reducing recurrence and improving long-term outcomes. Continued adherence to current clinical guidelines and early multidisciplinary involvement remain essential for optimizing care and minimizing the burden of spontaneous pneumothorax on patients and healthcare systems.

References

1. Baig MA, Majeed MB, Attar BM, Khan Z, Demetria M, Gandhi SR. Efficacy and Safety of Indwelling Pleural Catheters in Management of Hepatic Hydrothorax: A Systematic Review of Literature. Cureus. 2018 Aug 06;10(8):e3110.
2. Ojeda Rodriguez JA, Hipskind JE. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Mar 9, 2024. Iatrogenic Pneumothorax.
3. Cheng HS, Wong C, Chiu PH, Tong CW, Miu PF. Management of spontaneous pneumothorax: a mini-review on its latest evidence. J Thorac Dis. 2024 Jul 30;16(7):4756-4763.
4. Shorthose M, Barton E, Walker S. The contemporary management of spontaneous pneumothorax in adults. Breathe (Sheff). 2023 Dec;19(4):230135.
5. Wang R, Chen X, Xu S, Jiang X, Liu J, Liu X, Ryu JH, Hu X. Prevalence and recurrence rates of spontaneous pneumothorax in patients with diffuse cystic lung diseases in China. Orphanet J Rare Dis. 2025 Feb 11;20(1):69.
6. Liao KM, Chiu CC, Lu HY. The risk of secondary spontaneous pneumothorax in patients with chronic obstructive pulmonary disease in Taiwan. Respir Med. 2024 Jul;228:107672.
7. Hallifax RJ, Goldacre R, Landray MJ, Rahman NM, Goldacre MJ. Trends in the Incidence and Recurrence of Inpatient-Treated Spontaneous Pneumothorax, 1968-2016. JAMA. 2018 Oct 09;320(14):1471-1480.
8. Savitsky E, Oh SS, Lee JM. The Evolving Epidemiology and Management of Spontaneous Pneumothorax. JAMA. 2018 Oct 09;320(14):1441-1443.
9. Zhong X, Goldacre R, Morris EJA, Hallifax RJ. Trends in incidence of pneumothorax in England before, during and after the COVID-19 pandemic (2017-2023): a population-based observational study. Lancet Reg Health Eur. 2024 Sep;44:100994.
10. Hallifax R. Aetiology of Primary Spontaneous Pneumothorax. J Clin Med. 2022 Jan 19;11(3)
11. Walker SP, Bibby AC, Halford P, Stadon L, White P, Maskell NA. Recurrence rates in primary spontaneous pneumothorax: a systematic review and meta-analysis. Eur Respir J. 2018 Sep;52(3)
12. Bertolaccini L, Congedo MT, Bertani A, Solli P, Nosotti M. A project to assess the quality of the published guidelines for managing primary spontaneous pneumothorax from the Italian Society of Thoracic Surgeons. Eur J Cardiothorac Surg. 2018 Nov 01;54(5):920-925.
13. June C, Viscusi C, DeLange B. Simultaneous Spontaneous Bilateral Tension Pneumothorax Post COVID-19 Infection: A Case Study. Cureus. 2022 May;14(5):e25107.
14. Aguinagalde B, Aranda JL, Busca P, Martínez I, Royo I, Zabaleta J., Grupo de trabajo de la GPC para el Manejo de Pacientes con Neumotórax espontáneo. SECT Clinical practice guideline on the management of patients with spontaneous pneumothorax. Cir Esp (Engl Ed). 2018 Jan;96(1):3-11.
15. Schnell J, Beer M, Eggeling S, Gesierich W, Gottlieb J, Herth FJF, Hofmann HS, Jany B, Kreuter M, Ley-Zaporozhan J, Scheubel R, Walles T, Wiesemann S, Worth H, Stoelben E. Management of Spontaneous Pneumothorax and Post-Interventional Pneumothorax: German S3 Guideline. Respiration. 2019;97(4):370-402.
16. Wong A, Galiabovitch E, Bhagwat K. Management of primary spontaneous pneumothorax: a review. ANZ J Surg. 2019 Apr;89(4):303-308.
17. Yi E, Park JE, Chung JH, Ahn CB, Chung E, Noh OK, Lee S. Trends in recurrence of primary spontaneous pneumothorax in young population after treatment for first episode based on a nationwide population data. Sci Rep. 2023 Aug 18;13(1):13478
18. Farag GAI, Zineldin MAI, Al Awady RSAA, Abd El Salam AB, Elkahely MA. Comparative Analysis of Demographic Characteristics, Management, and Outcomes in Primary Versus Secondary Spontaneous Pneumothorax. Cureus. 2024 Jul;16(7):e65216.
19. Değirmenci M. Morbidity, mortality, and surgical treatment of secondary spontaneous pneumothorax. Ulus Travma Acil Cerrahi Derg. 2023 Aug;29(8):909-919.