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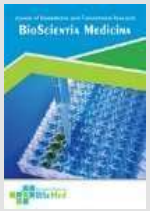

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# Hydropneumothorax Secondary to Community-Acquired Pneumonia with Broncholith: A Case Report

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**A B S T R A C T**

**Background:** Community-acquired pneumonia (CAP) is a common respiratory infection that can lead to serious complications, including hydropneumothorax, a condition characterized by the presence of both air and fluid in the pleural cavity. **Case presentation:** We present the case of a 76-year-old male who presented to the emergency department with a one-week history of shortness of breath, dry cough, and low-grade fever. He had no prior surgeries, history of tuberculosis, comorbid conditions, or significant pulmonary diseases. Physical examination revealed crackles, decreased breath sounds on auscultation, and hyperresonance on percussion of the right hemithorax. Laboratory investigations demonstrated leukocytosis and neutrophilia. Chest radiography showed consolidation consistent with pneumonia, and subsequent computed tomography (CT) confirmed the diagnosis of right hydropneumothorax with pleural effusion and a broncholith in the right basal lobar bronchus. The patient was treated with intravenous antibiotics and underwent water seal drainage (WSD). He made a full recovery and was discharged home. **Conclusion:** This case highlights the importance of considering hydropneumothorax as a potential complication of CAP, even in patients without significant comorbidities. Prompt diagnosis and management, including drainage and antibiotics, are crucial for a favorable outcome.

## 1. Introduction

Community-acquired pneumonia (CAP) remains a significant global health concern, associated with substantial morbidity, mortality, and healthcare costs. It is an acute infection of the lung parenchyma acquired outside of the hospital or healthcare setting. The incidence of CAP varies widely depending on age, comorbidities, and other risk factors, but it continues to pose a major public health challenge. The spectrum of CAP severity ranges from mild cases that can be managed in the outpatient setting to severe cases requiring hospitalization and intensive care. The severity of CAP is influenced by various factors, including the causative pathogen, the patient's age and underlying health status, and the presence of complications.[1-3]

Hydropneumothorax, a condition characterized by the simultaneous presence of both air and fluid in the pleural cavity, is a relatively rare but potentially life-threatening complication of CAP. It occurs when air and fluid accumulate between the visceral and parietal pleura, leading to lung collapse and impaired respiratory function. The pathogenesis of hydropneumothorax in the setting of CAP is often complex and multifactorial, involving various mechanisms such as the rupture of infected lung parenchyma, the formation of bronchopleural fistulas, and the development of parapneumonic effusions. Broncholiths, which are calcified or ossified masses within the bronchi, can also contribute to the development of hydropneumothorax. These bronchial stones can cause airway obstruction, leading to distal atelectasis and infection, which can subsequently

result in the formation of hydropneumothorax. The diagnosis of hydropneumothorax is typically made through a combination of clinical findings, imaging studies, and laboratory investigations. Chest radiography is often the initial imaging modality used to evaluate patients with suspected hydropneumothorax. The presence of an air-fluid level in the pleural space is a characteristic radiographic finding. Computed tomography (CT) scanning can provide more detailed information about the extent of the hydropneumothorax, the presence of underlying lung pathology, and the presence of broncholiths.[4-7]

The management of hydropneumothorax secondary to CAP typically involves a combination of medical and surgical interventions. Medical management includes the administration of appropriate antibiotics to treat the underlying pneumonia and supportive care to maintain adequate oxygenation and ventilation. Surgical intervention, such as chest tube insertion or thoracotomy, may be necessary to drain the pleural fluid and air and to re-expand the collapsed lung.[8-10] In this case report, we describe the presentation, diagnosis, and management of hydropneumothorax secondary to CAP in a 76-year-old male with a broncholith. This case highlights the importance of considering hydropneumothorax as a potential complication of CAP, even in patients without significant comorbidities. Prompt diagnosis and management, including drainage and antibiotics, are crucial for a favorable outcome.

**2. Case Presentation**

A 76-year-old male presented to the emergency department with a primary complaint of shortness of breath that had persisted for one week. The shortness of breath was accompanied by a dry cough and a low-grade fever, with the highest recorded temperature being 37.8°C. The patient denied experiencing any chest pain or weight loss. He had a significant smoking history, but no prior surgeries, history of tuberculosis, known comorbid conditions, or significant pulmonary diseases. Upon arrival at the emergency department, the patient was conscious and responsive, with a patent airway. However, his oxygen saturation was measured at 88%, indicating hypoxemia. His respiratory rate was elevated at 30 breaths per minute, and his heart rate was 112 beats per minute. His blood pressure was within the normal range at 122/56 mmHg. Physical examination revealed the presence of crackles in the right lung field upon auscultation. A chest X-ray was immediately ordered, which revealed semi-opaque lesions (consolidation) in the perihilar and right pericardia regions (Figure 2). The bronchovascular pattern was increased, with an air bronchogram pattern, both of which are indicative of pneumonia. Laboratory tests were conducted to further assess the patient's condition. The results showed leukocytosis, an elevated white blood cell count, and neutrophilia, an increased number of neutrophils, suggesting an ongoing inflammatory response. A rapid molecular test for tuberculosis was performed to rule out active tuberculosis infection, and the result was negative.

Based on the initial assessment, the patient was diagnosed with pneumonia and admitted to the hospital for further management. The initial treatment plan included oxygen supplementation through a nasal cannula at a rate of 4-5 liters per minute to address the hypoxemia. Intravenous fluids were administered to maintain hydration, and the patient was started on a course of intravenous antibiotics, including levofloxacin 750mg every 24 hours. Acetylcysteine 1200 mg every 24 hours was also prescribed, likely as a mucolytic agent to help thin the mucus and improve airway clearance. In addition to the above, the patient received nebulized ipratropium bromide + salbutamol sulfate and budesonide every 8 hours. This combination of medications was aimed at bronchodilation, reducing airway inflammation, and improving breathing. Vitamin D 1000 IU and zinc 20 mg were also included in the treatment regimen, likely to support the immune system and aid in recovery (Table 1).

On the third day of hospitalization, the patient's condition showed some improvement. His heart rate

had decreased to 112 beats per minute, his respiratory rate had reduced to 24 breaths per minute, and his oxygen saturation had improved to 93%. His temperature had also normalized to 36.9°C. However, physical examination of the chest still revealed crackles and decreased breath sounds upon auscultation of the right chest, along with hyperresonance on percussion. Given the persistent respiratory symptoms and physical examination findings, a computed tomography (CT) scan of the chest was ordered to obtain a more detailed evaluation of the lungs and surrounding structures. The CT scan revealed the presence of pneumonia, right hydropneumothorax with pleural effusion, and a broncholith in the basal lobar bronchus (Figure 3).

The discovery of hydropneumothorax, a condition where both air and fluid are present in the pleural space, prompted immediate intervention. The presence of a broncholith, a calcified mass in the bronchus, was also noted and considered a potential contributing factor to the development of hydropneumothorax. The treatment plan was adjusted to address the hydropneumothorax. Water seal drainage (WSD) was deemed necessary to remove the fluid and air from the pleural space and allow the lung to re-expand. The patient was taken to the operating room where thoracentesis, a procedure to drain fluid from the pleural space, was performed under local anesthesia. A WSD tube was then inserted into the pleural space to facilitate continuous drainage.

During the procedure, approximately 1.6 liters of pleural fluid were removed. A chest X-ray was performed after the procedure to confirm the correct placement of the WSD tube and assess the lung expansion. The intravenous antibiotic regimen was continued to address the underlying pneumonia. The fluid output from the WSD was closely monitored and measured every 6 hours. On the first day after the procedure, the WSD drained 480 cc of fluid. The drainage gradually decreased over the following days, with 250 cc, 300 cc, 250 cc, 100 cc, and 100 cc of fluid being drained on subsequent days (Figure 1).

After one week of WSD insertion, the patient's condition showed significant improvement. His shortness of breath had subsided, and the crackles in his right lung had decreased, with improved breath sounds upon auscultation. A follow-up chest X-ray indicated resolution of the hydropneumothorax. The WSD tube was then removed. The patient continued to recover well and was eventually discharged home with a full recovery. This case highlights the potential for serious complications, such as hydropneumothorax, to arise from pneumonia, even in patients without significant comorbidities or prior pulmonary diseases. It also emphasizes the importance of prompt diagnosis and appropriate management, including drainage and antibiotics, in ensuring a favorable outcome.

**3. Discussion**

Hydropneumothorax is a relatively uncommon condition characterized by the simultaneous presence of both air and fluid in the pleural cavity. It typically arises due to a disruption in the integrity of the pleural space, allowing air and fluid to accumulate and leading to partial or complete lung collapse. While various factors can contribute to hydropneumothorax, including trauma, iatrogenic causes, and underlying lung diseases, its occurrence as a complication of CAP is particularly rare. Before delving into the specifics of hydropneumothorax, it's essential to understand the normal anatomy and physiology of the pleural space. The pleural space is a thin, fluid-filled cavity that lies between the visceral pleura, which covers the lungs, and the parietal pleura, which lines the chest wall. This space normally contains a small amount of pleural fluid, which acts as a lubricant, allowing the lungs to move smoothly within the chest cavity during breathing. The pleural space is maintained at a negative pressure relative to the atmospheric pressure. This negative pressure is crucial for lung inflation and is generated by the opposing forces of the chest wall, which tends to expand outwards, and the lungs, which tend to recoil inwards.

Table 1. Timeline of disease.

| Day | Clinical findings | Treatment |
|---|---|---|
| 1 | Shortness of breath, dry cough, low-grade fever (37.8°C). No chest pain or weight loss. Smoker. No prior surgeries, history of tuberculosis, comorbid conditions, or significant pulmonary diseases. | Oxygen supplementation (4-5 liters/minute), intravenous antibiotics (Levofloxacin 750mg/24 hours), acetylcysteine 1200 mg/24 hours, nebulized Ipratropium Bromide+Salbutamol Sulvate and Budesonide/8 hours, Vitamin D 1000 IU, Zinc 20 mg. |
| 1 | Physical examination: Conscious, patent airway, oxygen saturation 88%, respiratory rate 30 breaths/min, temperature 37.8°C, blood pressure 122/56 mmHg. Crackles in the right chest. | |
| 1 | Chest X-ray: Semi-opaque lesions (consolidation) in the perihilar and right pericardia, increased bronchovascular pattern with an air bronchogram pattern indicating pneumonia. | |
| 1 | Laboratory tests: Leukocytosis and neutrophilia. Rapid Molecular Test for tuberculosis negative. | |
| 3 | Improved condition, heart rate 112 beats/min, respiratory rate 24 breaths/min, temperature 36.9°C, oxygen saturation 93%. | |
| 3 | Crackles and decreased breath sounds on auscultation, hyperresonant on percussion of the right chest. | |
| 3 | CT scan: Pneumonia, right hydropneumothorax with pleural effusion, a broncholith in the basal lobar bronchus. | |
| 3 - 4 | Water seal drainage (WSD) performed under local anesthesia, 1600 L of pleural fluid removed. | |
| 4 | Chest X-ray taken after the WSD procedure. | |
| 4 - 10 | Intravenous antibiotic regimen continued. Fluid output from WSD measured every 6 hours. | |
| 10 | Shortness of breath improved, decreased crackles, better breath sounds on auscultation. | |
| 10 | Chest X-ray indicated resolution of hydropneumothorax. | WSD removed. |
| 10 | Discharged home after full recovery. | |

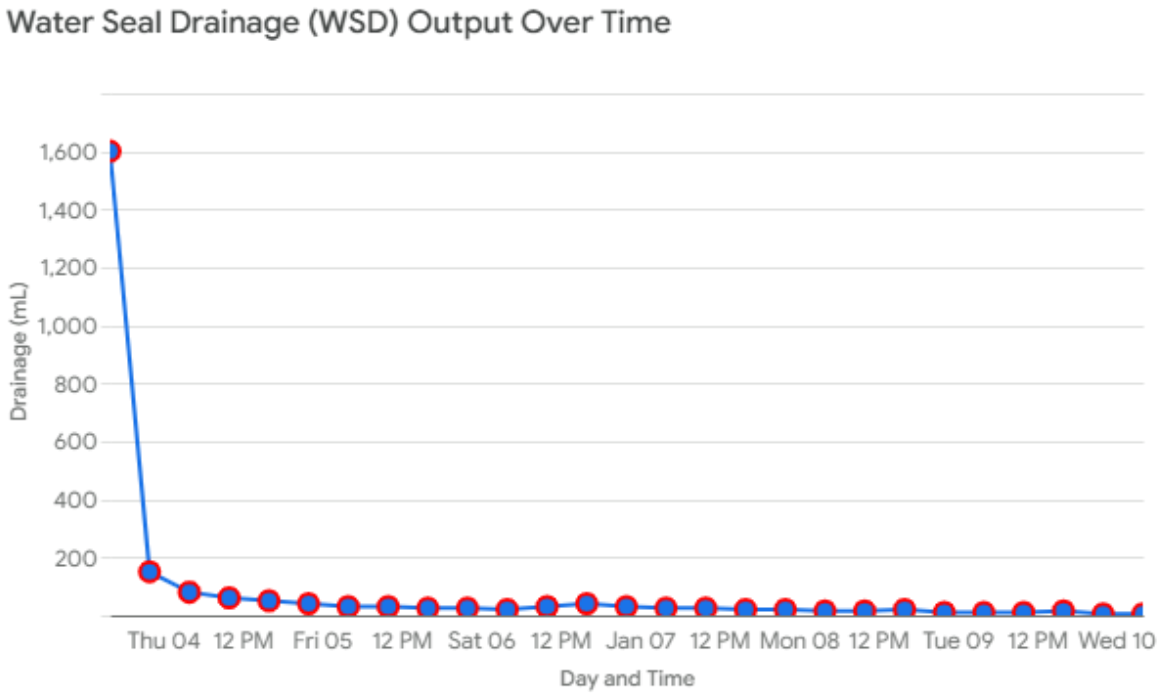


Figure 1. Water seal drainage (WSD) output over time.

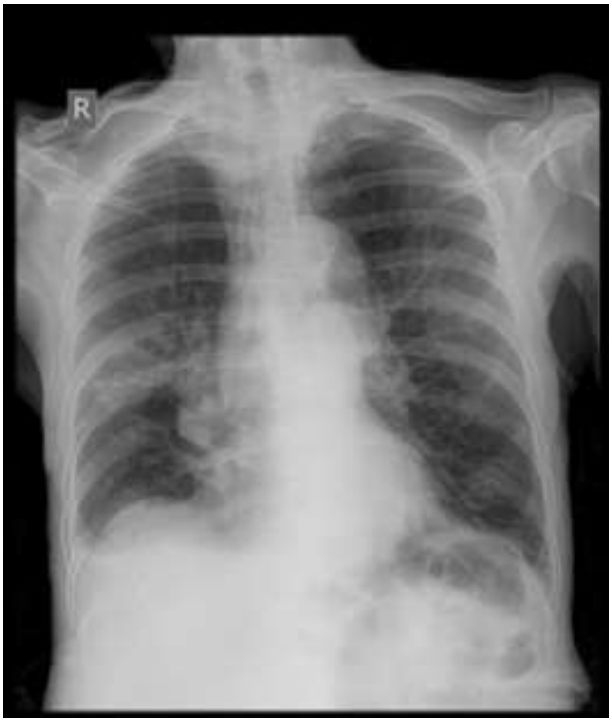


Figure 2. Chest radiograph showing right lower lobe consolidation and hydropneumothorax.

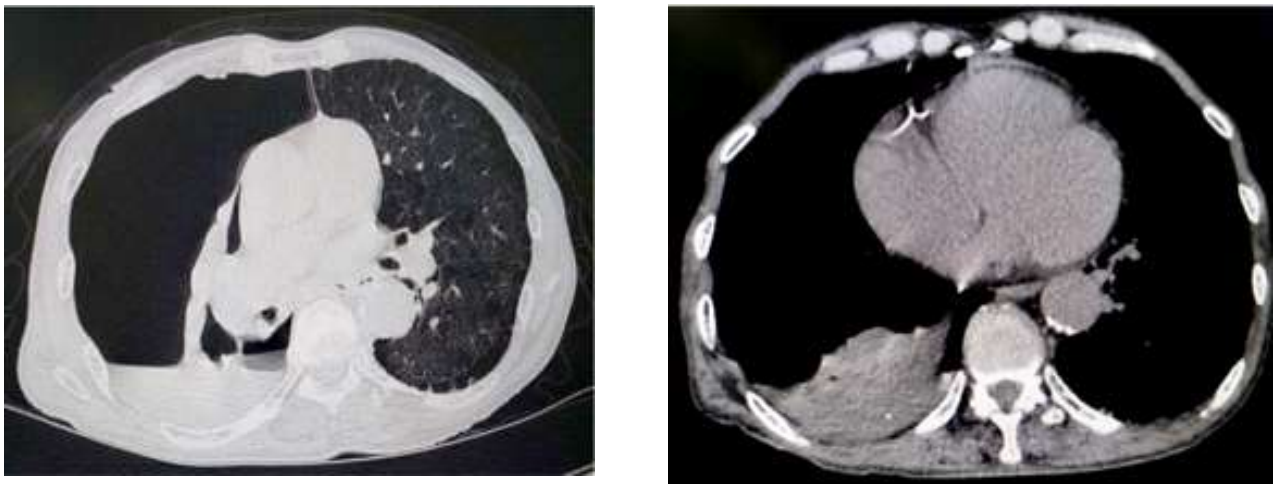

Figure 3. Chest CT scan. CT scan showed right hydropneumothorax with pleural effusion, a broncholith in the basal lobar bronchus.

The negative pressure in the pleural space creates a suction effect that keeps the lungs inflated against the chest wall. The visceral pleura is a thin, delicate membrane that adheres closely to the surface of the lungs, covering each lobe and fissure. It is composed of a single layer of mesothelial cells, supported by a thin layer of connective tissue. The visceral pleura is highly vascularized and innervated, allowing for efficient gas exchange and sensation. Parietal Pleura is thicker, more robust membrane lines the inner surface of the chest wall, diaphragm, and mediastinum. It is also composed of mesothelial cells and connective tissue, but it contains a more extensive network of lymphatic vessels. The parietal pleura is less sensitive to pain than the visceral pleura. Pleural Cavity is the potential space between the visceral and parietal pleura that is normally filled with a small amount of pleural fluid. The pleural fluid acts as a lubricant, reducing friction between the two pleural layers during breathing. It also helps to maintain the negative pressure within the pleural space. The pleural fluid acts as a lubricant, allowing the lungs to move smoothly within the chest cavity during breathing. This reduces friction and prevents damage to the delicate lung tissue. The opposing forces of the chest wall, which tends to expand outwards, and the lungs, which tend to recoil inwards, generate a negative pressure within the pleural space. This negative pressure is essential for lung inflation. The lymphatic vessels in the parietal pleura play a crucial role in draining excess pleural fluid and removing any debris or pathogens that may enter the pleural space. The close proximity of the visceral pleura to the lung parenchyma allows for efficient gas exchange between the alveoli and the capillaries in the visceral pleura. The pleural space acts as a barrier, protecting the lungs from infections and injuries. The pleural fluid is constantly being produced and reabsorbed, maintaining a delicate balance within the pleural space. The production of pleural fluid is primarily driven by hydrostatic and oncotic pressure gradients across the capillaries in the parietal pleura. The reabsorption of pleural fluid is mainly carried out by the lymphatic vessels in the parietal pleura. The normal volume of pleural fluid in the pleural space is very small, estimated to be around 10-20 mL. This small volume is crucial for maintaining the negative pressure within the pleural space and allowing for smooth lung movement during breathing. Lung compliance refers to the ease with which the lungs can expand. Diseases that reduce lung compliance, such as pulmonary fibrosis, make it more difficult for the lungs to inflate, leading to a more negative pleural pressure. Chest wall compliance refers to the ease with which the chest wall can expand. Conditions that restrict chest wall movement, such as kyphoscoliosis

or obesity, can reduce chest wall compliance and affect pleural pressure. Airway resistance refers to the resistance to airflow in the airways. Increased airway resistance, as seen in asthma or COPD, can lead to a more negative pleural pressure during inspiration. Increased intra-abdominal pressure, as seen in ascites or pregnancy, can push the diaphragm upwards, reducing the volume of the thoracic cavity and affecting pleural pressure. The pleural space is essential for normal lung function and plays a crucial role in maintaining respiratory health. Disruptions in the integrity of the pleural space, such as in hydropneumothorax, can lead to significant respiratory compromise. Understanding the anatomy, physiology, and fluid dynamics of the pleural space is crucial for healthcare professionals involved in the diagnosis and management of respiratory diseases. This knowledge allows for a better understanding of the pathophysiology of pleural diseases and the rationale behind various treatment strategies. Hydropneumothorax occurs when the integrity of the pleural space is compromised, allowing air and fluid to enter and accumulate. This disruption can occur due to various reasons, including trauma, iatrogenic causes, and underlying lung diseases. Penetrating chest injuries, such as stab wounds or gunshot wounds, can directly breach the pleural space, creating an opening for air and fluid to enter. The severity of the hydropneumothorax depends on the size and location of the injury, as well as the extent of damage to the underlying lung tissue. For instance, a small puncture wound to the chest may cause a limited hydropneumothorax, while a larger laceration can lead to a more extensive accumulation of air and fluid, potentially causing complete lung collapse. Blunt chest trauma, such as that sustained in a motor vehicle accident or a fall, can also disrupt the pleural space. This can occur through direct injury to the lung parenchyma, causing a tear or laceration that allows air and fluid to escape into the pleural space. Additionally, blunt chest trauma can cause rib fractures, and the sharp edges of the fractured ribs can lacerate the pleura, again leading to hydropneumothorax. The extent of the hydropneumothorax in blunt chest trauma depends on the force of the impact, the location of the injury, and the presence of any underlying lung conditions. Thoracentesis is a medical procedure that involves inserting a needle into the pleural space to drain fluid. While generally safe, this procedure carries a risk of inadvertently introducing air into the pleural space, leading to hydropneumothorax. This can occur if the needle is not properly positioned or if there is a sudden change in intrathoracic pressure during the procedure. The risk of hydropneumothorax following thoracentesis is relatively low but can be higher in patients with underlying lung disease or those who require repeated thoracentesis. Chest tube insertion is another medical procedure that can potentially disrupt the pleural space. A chest tube is a hollow tube that is inserted into the pleural space to drain air or fluid. While chest tubes are often used to treat pneumothorax or pleural effusion, improper insertion or management of the chest tube can lead to the introduction of air into the pleural space, causing hydropneumothorax. This can occur if the chest tube is not properly secured or if there is a leak in the drainage system. Mechanical ventilation, a life-saving intervention for patients with respiratory failure, can also increase the risk of hydropneumothorax. The positive pressure ventilation used in mechanical ventilation can cause alveolar overdistension and rupture, leading to air leakage into the pleural space. This risk is higher in patients with underlying lung disease or those who require high ventilator pressures. Central venous catheterization involves inserting a catheter into a large vein, typically in the neck or chest, to administer medications or fluids. While generally safe, this procedure carries a risk of puncturing the pleura and causing hydropneumothorax. This risk is higher if the catheter is not inserted correctly or if the patient has anatomical variations in the chest. Pneumonia, an infection of the lung parenchyma, can weaken the lung tissue and make it more susceptible to rupture. This can occur due to the inflammatory process

associated with pneumonia, which can cause tissue necrosis and breakdown. If a rupture occurs, air and inflammatory fluid can leak into the pleural space, leading to hydropneumothorax. Tuberculosis, a chronic bacterial infection that primarily affects the lungs, can also lead to hydropneumothorax. The infection can cause the formation of cavities within the lung tissue, which can rupture and allow air and fluid to enter the pleural space. Additionally, tuberculosis can lead to the formation of bronchopleural fistulas, abnormal connections between the bronchi and the pleural cavity, further increasing the risk of hydropneumothorax. Lung cancer, a malignant tumor that originates in the lungs, can directly invade and weaken the pleura, making it more prone to rupture. Additionally, lung cancer can cause obstruction of the airways, leading to distal atelectasis and infection, which can subsequently result in hydropneumothorax. Chronic Obstructive Pulmonary Disease (COPD), a group of lung diseases characterized by airflow obstruction, can also increase the risk of hydropneumothorax. The chronic inflammation and air trapping associated with COPD can weaken the lung tissue and make it more susceptible to rupture. Additionally, COPD can lead to the formation of bullae, large air spaces within the lung parenchyma, which can rupture and cause hydropneumothorax. Other lung diseases, such as cystic fibrosis, interstitial lung disease, and pulmonary embolism, can also contribute to the development of hydropneumothorax. These diseases can weaken the lung parenchyma or pleura, making them more prone to rupture and leakage of air and fluid into the pleural space. The occurrence of hydropneumothorax as a complication of community-acquired pneumonia (CAP) is a rare but serious event. It signifies a complex interplay of pathological processes stemming from the infection and host response. Understanding the mechanisms that contribute to hydropneumothorax in CAP patients is crucial for prompt diagnosis and effective management. In CAP, the lung parenchyma, the functional tissue involved in gas exchange, becomes the battleground for the body's fight against invading pathogens. The inflammatory response, while essential for containing the infection, can also lead to collateral damage. Inflammatory mediators, released by immune cells, cause increased vascular permeability, leading to fluid accumulation in the alveoli (the tiny air sacs in the lungs) and the surrounding interstitial tissue. This fluid, along with cellular debris and inflammatory cells, consolidates within the lung parenchyma, compromising its structural integrity. As the infection progresses, some areas of the lung parenchyma may undergo necrosis, or tissue death, due to inadequate blood supply and overwhelming inflammation. This necrotic tissue can break down, forming cavities within the lung, known as abscesses. These abscesses are filled with pus, a mixture of dead cells, bacteria, and inflammatory debris. The weakened lung tissue, riddled with inflammation and potential abscesses, becomes susceptible to rupture. A rupture can occur due to various factors, including coughing, straining, or even normal breathing. When a rupture occurs, air from the airways escapes into the pleural space, the normally closed cavity between the lung and chest wall. Along with air, infected fluid from the consolidated lung parenchyma or abscesses can also leak into the pleural space, leading to hydropneumothorax. Bronchopleural fistulas are abnormal connections between the bronchi, the air passages leading to the lungs, and the pleural cavity. In CAP, these fistulas can form due to the erosion of infected lung tissue into the bronchi. The ongoing inflammation and tissue destruction can weaken the walls of the bronchi, making them prone to rupture. Once a bronchopleural fistula forms, air from the bronchi can directly enter the pleural space, leading to pneumothorax, or air in the pleural cavity. Additionally, the fistula provides a pathway for the spread of infection from the lungs to the pleural space, further contributing to the accumulation of infected fluid and the development of hydropneumothorax. Parapneumonic effusions are the accumulation of fluid in the pleural space in response to pneumonia. The inflammation in the lung

parenchyma triggers increased permeability of the pleural capillaries, allowing fluid to leak into the pleural space. This fluid is initially a sterile exudate, containing inflammatory mediators and immune cells. In some cases, parapneumonic effusions can become infected, leading to the development of empyema, a collection of pus in the pleural space. Empyema formation is more likely to occur in patients with severe pneumonia, delayed antibiotic treatment, or underlying lung disease. An empyema can exert pressure on the surrounding lung tissue, further compromising its integrity. If the empyema ruptures into the lung, it can lead to the formation of a bronchopleural fistula, allowing air to enter the pleural space and contributing to hydropneumothorax. Hydropneumothorax, a condition characterized by the presence of both air and fluid in the pleural cavity, presents a unique diagnostic challenge due to its varied clinical presentation and the need to differentiate it from other respiratory conditions. The clinical presentation of hydropneumothorax can be quite diverse, depending on the underlying cause, the volume of air and fluid accumulated, and the overall health status of the individual. In some cases, hydropneumothorax may be asymptomatic, especially if it is small and develops gradually. Shortness of breath is a common symptom, particularly with larger hydropneumothoraces, as the air and fluid in the pleural cavity compress the lung and reduce its ability to expand fully. This can lead to feelings of breathlessness, rapid breathing (tachypnea), and increased work of breathing. In severe cases, it can progress to respiratory failure, requiring mechanical ventilation. Chest pain is another frequent complaint, often described as a sharp, stabbing pain that worsens with deep breathing or coughing. This pain arises from irritation of the parietal pleura, which is richly innervated with pain fibers. The location of the pain can vary depending on the location of the hydropneumothorax. Cough can be a symptom of both the underlying condition causing the hydropneumothorax, such as pneumonia, and the hydropneumothorax itself. The cough may be dry or productive, depending on the presence of associated infection or inflammation in the airways. On physical examination, decreased or absent breath sounds may be noted on the affected side. This is due to the reduced air entry into the collapsed lung. Percussion of the chest may also reveal hyperresonance, a drum-like sound, over the area of pneumothorax. In addition to the above, patients may experience other nonspecific symptoms, such as fatigue, weakness, anxiety, and a rapid heart rate (tachycardia). If the hydropneumothorax is associated with an infection, such as empyema, the patient may also present with fever, chills, and other systemic signs of infection. Given the variability in clinical presentation and the need to differentiate hydropneumothorax from other respiratory conditions, a comprehensive diagnostic approach is essential. A detailed medical history, including any history of trauma, recent medical procedures, or underlying lung disease, can provide valuable clues. Physical examination, including auscultation of the chest and percussion, can help identify signs suggestive of hydropneumothorax. Imaging studies play a central role in confirming the diagnosis of hydropneumothorax and assessing its extent. Chest X-ray is often the initial imaging modality used. It can reveal the presence of air and fluid in the pleural cavity, typically seen as an air-fluid level with a clear demarcation between the air and fluid components. However, chest X-ray may not always be definitive, especially in cases of small or loculated hydropneumothoraces. CT scanning is a more sensitive and specific imaging modality for evaluating hydropneumothorax. It provides detailed cross-sectional images of the chest, allowing for better visualization of the air and fluid in the pleural cavity. CT scanning can also help identify underlying lung pathology, such as pneumonia, bullae, or bronchopleural fistulas, that may be contributing to the hydropneumothorax. In addition to imaging, other investigations may be performed to assess the patient's overall condition and identify any underlying causes or complications. Arterial blood gas analysis

tests measure the levels of oxygen and carbon dioxide in the blood and can help assess the severity of respiratory compromise. Complete blood count test can help identify signs of infection or inflammation. Pleural fluid analysis, if pleural fluid is drained, it can be sent for analysis to determine its nature (exudate vs. transudate), identify the presence of infection, and guide antibiotic therapy. The differential diagnosis for hydropneumothorax includes other conditions that can cause similar symptoms or radiographic findings. Pneumothorax is the presence of air in the pleural cavity without fluid. It can cause similar symptoms to hydropneumothorax, such as shortness of breath and chest pain. However, on imaging, pneumothorax appears as a collection of air without a fluid level. Pleural effusion is the accumulation of fluid in the pleural cavity without air. It can also cause shortness of breath and chest pain, but on imaging, it appears as a homogenous opacity without an air-fluid level. Hemothorax is the accumulation of blood in the pleural cavity. It can occur due to trauma or chest surgery and can cause similar symptoms to hydropneumothorax. However, on imaging, hemothorax typically appears as a denser opacity compared to pleural effusion. Chylothorax is the accumulation of lymphatic fluid in the pleural cavity. It can occur due to trauma or thoracic duct obstruction and can cause shortness of breath and chest pain. However, on imaging, chylothorax typically appears as a milky fluid accumulation. The management of hydropneumothorax secondary to community-acquired pneumonia (CAP) necessitates a multifaceted approach, encompassing both medical and surgical interventions. The primary goals are to resolve the underlying infection, evacuate air and fluid from the pleural space, re-expand the collapsed lung, and restore optimal respiratory function. The cornerstone of medical management is prompt and appropriate antibiotic therapy to address the underlying CAP. The choice of antibiotics depends on the suspected or identified pathogen, local antibiotic resistance patterns, and the patient's overall health status. Empiric antibiotic therapy, targeting the most common pathogens associated with CAP, is often initiated while awaiting culture results. Intravenous antibiotics are typically preferred for patients with hydropneumothorax, as they provide rapid and effective drug delivery to the infected lung parenchyma and pleural space. Supportive care measures are essential to maintain adequate oxygenation and ventilation. Oxygen therapy is often administered to correct hypoxemia, and in severe cases, mechanical ventilation may be required to support breathing. Close monitoring of respiratory parameters, such as oxygen saturation, respiratory rate, and arterial blood gases, is crucial to guide respiratory support. Careful fluid management is essential to maintain fluid balance and prevent fluid overload, which can worsen respiratory distress. Intravenous fluids may be necessary to maintain hydration, but excessive fluid administration should be avoided. Diuretics may be used to promote fluid excretion if fluid overload occurs. Chest pain associated with hydropneumothorax can be significant and may require analgesics for adequate pain relief. Nonsteroidal anti-inflammatory drugs (NSAIDs) or opioids may be used, depending on the severity of pain. Chest tube insertion is the most common surgical procedure performed for hydropneumothorax. It involves inserting a hollow tube into the pleural space to drain the accumulated air and fluid. The chest tube is typically connected to a water seal drainage system, which allows for the evacuation of air and fluid while preventing air from re-entering the pleural space. The size and position of the chest tube depend on the volume and location of the air and fluid collection. Water Seal Drainage (WSD) is a critical component of chest tube management. The water seal system acts as a one-way valve, allowing air and fluid to escape from the pleural space but preventing air from re-entering. The system typically consists of a drainage chamber, a water seal chamber, and a suction control chamber. The drainage chamber collects the drained fluid, the water seal chamber prevents air from entering the pleural space, and the suction control chamber regulates the amount of

suction applied to the system. In some cases, thoracotomy, a surgical incision into the chest cavity, may be required to manage hydropneumothorax. This may be necessary to address the underlying cause of the hydropneumothorax, such as a bronchopleural fistula or a lung abscess. Thoracotomy may also be performed to remove loculated fluid or adhesions that are preventing adequate drainage with a chest tube. The presence of underlying lung disease, such as COPD or bronchiectasis, can complicate the management of hydropneumothorax. These patients may require more aggressive respiratory support and may be at higher risk for complications. Comorbid conditions, such as heart failure or diabetes, can also influence the management and prognosis of hydropneumothorax. These conditions may increase the risk of complications and may require additional medical management. Patient education is an essential component of management. Patients should be informed about their condition, the treatment plan, and potential complications. They should also be instructed on how to care for their chest tube and drainage system, if applicable. The prognosis for patients with hydropneumothorax secondary to CAP is generally good with prompt diagnosis and appropriate management. The majority of patients recover fully with appropriate antibiotics and drainage of the pleural space. However, the presence of underlying lung disease, comorbidities, or complications can worsen the prognosis.[11-14]

Broncholiths, also known as broncholithiasis, are calcified or ossified masses that form within the bronchi, the airways that carry air to and from the lungs. These "stones" in the airways can have a significant impact on respiratory health and, in certain cases, contribute to the development of complications such as hydropneumothorax. Broncholiths typically arise from the calcification of various materials that become lodged within the bronchi. Small foreign objects, such as food particles, beads, or small toys, that are accidentally inhaled can become trapped in the bronchi. Over time, these objects can become a nidus for calcification, leading to the formation of a broncholith. Lymph nodes, small bean-shaped structures that filter lymphatic fluid, are located throughout the body, including in the chest near the bronchi. In some cases, lymph nodes can become inflamed or infected, leading to calcification and the formation of a broncholith. This is particularly common in individuals with a history of granulomatous diseases, such as tuberculosis or histoplasmosis. Inflammatory exudates, the fluid and cellular debris that accumulate in response to inflammation or infection, can also contribute to broncholith formation. In chronic inflammatory conditions of the airways, such as bronchiectasis, the ongoing inflammation can lead to the deposition of calcium salts within the bronchi, eventually forming a broncholith. The broncholith itself can physically obstruct the airway lumen, partially or completely blocking the flow of air. The degree of obstruction depends on the size and location of the broncholith, as well as the diameter of the affected bronchus. The presence of a broncholith can irritate the bronchial mucosa, the lining of the airways, leading to inflammation and swelling. This inflammation can further narrow the airway lumen, exacerbating the obstruction. The broncholith can disrupt the normal function of the mucociliary escalator, the mechanism responsible for clearing mucus and debris from the airways. The broncholith can damage the cilia, the hair-like projections that propel mucus, or it can impair the production and composition of mucus, making it thicker and more difficult to clear. Atelectasis is the collapse or closure of lung tissue, preventing the exchange of oxygen and carbon dioxide. When a broncholith obstructs an airway, it prevents air from reaching the alveoli, the tiny air sacs in the lungs where gas exchange occurs. This can lead to the collapse of the affected lung segment or lobe, known as distal atelectasis. The obstruction caused by a broncholith can also create an environment conducive to infection. The impaired drainage of secretions and the accumulation of mucus behind the broncholith provide a breeding ground for bacteria. This can lead to localized pneumonia or bronchitis, further

compromising respiratory function. In chronic cases, the ongoing inflammation and infection associated with broncholith obstruction can lead to bronchiectasis, a condition characterized by permanent dilation and damage to the bronchi. Bronchiectasis can further impair mucociliary clearance and increase the risk of recurrent infections. The presence of a broncholith can significantly increase the risk of developing hydropneumothorax, particularly in the setting of CAP. The localized inflammation and infection caused by broncholith obstruction can weaken the lung parenchyma, making it more susceptible to rupture. If a rupture occurs, air and infected fluid can leak into the pleural space, leading to hydropneumothorax. The erosion of infected lung tissue into the bronchi, facilitated by the presence of a broncholith, can lead to the formation of bronchopleural fistulas. These fistulas provide a direct pathway for air to enter the pleural space, contributing to hydropneumothorax. The inflammation and infection associated with broncholith obstruction can exacerbate parapneumonic effusions, the accumulation of fluid in the pleural space in response to pneumonia. These effusions can become infected, leading to empyema, which can rupture into the lung and cause hydropneumothorax.[15-17]

Diagnosing hydropneumothorax presents a unique set of challenges due to its variable clinical presentation and the need to differentiate it from other respiratory conditions with overlapping symptoms. Imaging studies play a pivotal role in overcoming these challenges and confirming the diagnosis. The symptoms of hydropneumothorax, such as shortness of breath, chest pain, and cough, are not specific to this condition and can be seen in a wide range of respiratory diseases, including pneumonia, pleural effusion, pneumothorax, and even pulmonary embolism. This overlap in symptoms can make it difficult to pinpoint hydropneumothorax as the underlying cause based on clinical presentation alone. The clinical presentation of hydropneumothorax can vary significantly depending on several factors, including the volume of air and fluid accumulated, the underlying cause, and the patient's overall health status. In some cases, hydropneumothorax may be asymptomatic, particularly if it is small and develops gradually. This can further complicate the diagnosis, as patients may not seek medical attention until the condition becomes more severe. The presence of underlying lung pathology, such as pneumonia or COPD, can further complicate the diagnosis of hydropneumothorax. The symptoms of the underlying lung disease may mask or mimic the symptoms of hydropneumothorax, making it difficult to differentiate between the two. While physical examination can provide valuable clues, it may not always be definitive in diagnosing hydropneumothorax. Decreased breath sounds and hyperresonance on percussion can be suggestive of hydropneumothorax, but these findings can also be present in other conditions, such as pneumothorax or pleural effusion. Imaging studies are essential for confirming the diagnosis of hydropneumothorax and assessing its extent. Chest X-ray is often the initial imaging study performed in patients with suspected hydropneumothorax. It is readily available, relatively inexpensive, and can provide valuable information about the presence of air and fluid in the pleural cavity. The characteristic radiographic finding of hydropneumothorax is an air-fluid level, seen as a clear demarcation between the air (radiolucent) and fluid (radiopaque) components within the pleural space. Chest X-rays may not be sensitive enough to detect small hydropneumothoraces, particularly those located in the anterior or posterior costophrenic angles. The air-fluid level seen on chest X-ray can also be seen in other conditions, such as a loculated pleural effusion or a hydropneumothorax within a bulla (a large air space in the lung). The presence of underlying lung pathology, such as pneumonia or pleural thickening, can obscure the air-fluid level on chest X-ray, making it difficult to diagnose hydropneumothorax. CT scanning is a more sensitive and specific imaging modality for evaluating hydropneumothorax. It provides detailed cross-sectional images of the chest, allowing for better

visualization of the air and fluid in the pleural cavity, even in cases where the air-fluid level is small or obscured by other lung pathology. CT scanning can detect even small amounts of air and fluid in the pleural space, making it more sensitive than chest X-ray for diagnosing hydropneumothorax. CT scanning can differentiate hydropneumothorax from other conditions that may mimic its appearance on chest X-ray, such as loculated pleural effusion or hydropneumothorax within a bulla. CT scanning provides detailed anatomical information about the lungs and pleural space, allowing for the identification of underlying lung pathologies, such as pneumonia, bronchiectasis, or bronchopleural fistulas, that may be contributing to the hydropneumothorax. CT images can be reconstructed in three dimensions, providing a more comprehensive view of the hydropneumothorax and its relationship to surrounding structures. While chest radiography and CT scanning are the primary imaging modalities used in the diagnosis of hydropneumothorax. Ultrasound can be used to assess pleural fluid and identify the presence of air in the pleural space. It is particularly useful in bedside assessments and can help guide thoracentesis or chest tube insertion. MRI is generally not the first-line imaging modality for hydropneumothorax, but it may be useful in certain cases, such as when evaluating the chest wall or mediastinum for involvement.[18-20]

## 4. Conclusion

This case report presents a rare instance of hydropneumothorax occurring as a complication of community-acquired pneumonia (CAP) in a 76-year-old male patient with a broncholith. The broncholith likely played a significant role in the development of hydropneumothorax by causing localized inflammation, infection, and weakening of the lung parenchyma. The patient's condition was successfully managed with prompt antibiotic therapy and water seal drainage (WSD) to evacuate air and fluid from the pleural space. This case underscores the importance of considering hydropneumothorax as a potential complication of CAP, even in patients without significant comorbidities. Early diagnosis through imaging studies, such as chest radiography and computed tomography (CT) scanning, is crucial for prompt and appropriate management. This case also highlights the effectiveness of WSD in draining the pleural space and facilitating lung re-expansion. The patient's recovery was further aided by the continuation of intravenous antibiotics to address the underlying pneumonia.

## 5. References


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