



Pleural Effusion: Pathophysiology, Diagnostic Approaches, and Evidence-Based Management Strategies for Healthcare Professionals



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Abstract

Background: Pleural effusion, the abnormal accumulation of fluid in the pleural space, is a common clinical manifestation of various systemic and local diseases. It represents a significant healthcare burden, with approximately 1.5 million cases annually in the United States. The condition arises from an imbalance between pleural fluid formation and absorption, driven by disturbances in hydrostatic pressure, oncotic pressure, capillary permeability, or lymphatic drainage.

Aim: This article aims to provide a comprehensive review of the pathophysiology, diagnostic evaluation, and evidence-based management strategies for pleural effusion, serving as a resource for healthcare professionals to improve patient outcomes.

Methods: The review synthesizes current medical literature and guidelines. It details the anatomical and physiological basis of pleural fluid dynamics, the application of diagnostic tools (including chest radiography, ultrasound, and computed tomography), and the systematic analysis of pleural fluid using Light's criteria and other laboratory tests to differentiate transudates from exudates.

Results: Accurate diagnosis hinges on integrating clinical presentation with imaging and pleural fluid analysis. Management is twofold: treating the underlying cause (e.g., heart failure, infection, malignancy) and providing symptomatic relief through fluid drainage. Therapeutic options range from thoracentesis and chest tube drainage for acute management to indwelling pleural catheters or pleurodesis for recurrent malignant effusions. Ultrasound guidance is critical for procedural safety and efficacy.

Conclusion: A structured, evidence-based approach is essential for the effective management of pleural effusion. This involves a thorough diagnostic workup to identify the etiology, followed by tailored interventions that address both the fluid accumulation and its root cause, ultimately aiming to relieve symptoms, prevent recurrence, and manage complications.

Keywords: Pleural Effusion, Pathophysiology, Light's Criteria, Thoracentesis, Ultrasound, Exudate, Transudate, Management.

1. Introduction

A pleural effusion occurs when fluid collects within the pleural space. Normally a thin film of fluid lines the pleural cavity. That film lubricates the lung surfaces and allows the lungs to glide during breathing. Production and absorption of that fluid remain in balance under healthy conditions. Pathology can upset that balance and cause excess fluid to build up. In the United States about 1.5 million patients develop pleural effusions each year. [1][2] In several patient groups the presence of pleural fluid signals higher risk of complications and death. [3] Clinicians make the diagnosis through clinical assessment supported by imaging and laboratory data. Chest radiography and ultrasound identify fluid and guide procedures. Computed tomography adds detail when complex anatomy or loculation is suspected. Analysis of pleural fluid after thoracentesis yields diagnostic clues. Thoracentesis serves both diagnostic and therapeutic roles. It provides samples for cell counts, chemistry, microbiology and cytology and it relieves symptoms in patients with large effusions [1][2][3].

Treatment targets the cause that drives fluid accumulation. When heart failure or liver disease produces the effusion treating the primary disorder reduces recurrence. When infection or malignancy causes the collection targeted therapy is required. Procedural options range from single therapeutic thoracentesis to indwelling catheters or pleurodesis for recurrent symptomatic effusions. Management also addresses complications such as infected pleural collections and pleural fibrosis that can impair lung expansion. The pleural space sits between the visceral and parietal pleura. The visceral pleura adheres to the lung surface and follows lobar anatomy and fissures. It consists of a single layer of mesothelial cells over connective tissue. That layer creates a smooth surface for lung motion. The parietal pleura lines the thoracic wall and divides into mediastinal diaphragmatic costal and cervical regions. The parietal layer is thicker and contains more blood vessels and sensory nerves than the visceral layer. Those nerve fibers mediate pain from pleural disease [1][2][3].

The pleural cavity normally contains minimal fluid. That fluid reduces friction between pleural layers during breathing. Fluid volume in the cavity reflects the balance of hydrostatic and oncotic pressures, the effect of gravity and the

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mechanical forces of ventilation. These forces determine net fluid movement across the pleural surfaces. [4] Disease of the pleura disrupts anatomy and physiology and produces symptoms that prompt evaluation. Pleural inflammation increases vascular permeability and alters fluid resorption. Cardiac failure raises hydrostatic pressure and pushes plasma into the pleural space. Hypoalbuminemia lowers oncotic pressure and favors transudation. Malignancy can obstruct lymphatic drainage or increase local production of fluid. Each mechanism produces fluid that differs in composition and clinical implications. A practical grasp of pleural structure and the forces that govern pleural fluid is essential for accurate diagnosis and effective treatment. Integrating clinical findings with imaging and pleural fluid analysis guides the choice of interventions. Drainage procedures reduce breathlessness. Etiologic therapy reduces recurrence. Attention to complications preserves lung function. These principles shape an evidence based approach to care for patients with pleural effusion [4].

Etiology

Pleural effusion arises from multiple disease processes and is commonly categorized by the biochemical character of the pleural fluid into transudates and exudates. Distinguishing the two types guides the search for the underlying mechanism and dictates the subsequent diagnostic algorithm. Light's criteria remain the widely used standard for this classification. According to Light's criteria an effusion meets the definition of exudate when at least one of the following is present: the pleural fluid to serum protein ratio exceeds 0.5 the pleural fluid to serum lactate dehydrogenase LDH ratio exceeds 0.6 or the pleural fluid LDH concentration is greater than two thirds of the upper limit of normal for serum LDH. If none of these thresholds are met the fluid is classified as transudative. [5][6] These cutoffs reflect differences in capillary permeability protein leakage and lymphatic clearance that underpin the two categories.

Heffner and colleagues proposed alternative thresholds that simplify or complement Light's criteria. Under Heffner's approach an exudate is suggested that pleural fluid protein exceeds 2.9 g per dL when pleural fluid cholesterol is greater than 45 mg per dL or when pleural fluid LDH exceeds two thirds of the upper limit of normal for serum LDH. Heffner's criteria can help resolve borderline cases and may improve specificity in some clinical contexts. [7] Both sets of criteria remain tools rather than absolute rules and must be interpreted in the clinical context; prior diuretic therapy for example can concentrate pleural fluid and lead to misclassification of a transudate as an exudate. Transudative effusions result from systemic forces that alter hydrostatic or oncotic balance across the pleural microvasculature or from mechanical factors that impede fluid removal. Common examples include left ventricular failure where raised hydrostatic pressure forces plasma ultrafiltrate into the pleural space nephrotic syndrome and hypoalbuminemia where reduced plasma oncotic pressure favors fluid egress and hepatic cirrhosis with ascites where movement of ascitic fluid through diaphragmatic defects produces hepatic hydrothorax. Peritoneal dialysis can likewise permit fluid passage into the thorax. In these settings the pleural fluid is typically low in protein and LDH and the diagnostic and therapeutic focus is correction of the underlying systemic disorder [7].

Exudative effusions reflect local pleural processes that increase vascular permeability or impair lymphatic drainage. Infectious causes are frequent; bacterial pneumonia frequently produces a parapneumonic effusion that may progress to empyema when organisms invade the pleural space. Tuberculosis remains an important cause of exudative effusion in many regions. Malignancy produces exudates by direct pleural involvement, increased vascular permeability and lymphatic obstruction. Inflammatory diseases such as acute pancreatitis connective tissue disorders including systemic lupus erythematosus and rheumatoid arthritis can trigger pleural inflammation and exudation. Postcardiac injury syndromes and direct pleural injury after thoracic surgery including post coronary artery bypass grafting may generate exudative collections. Chylothorax and hemothorax represent specific etiologies in which the character of the fluid is diagnostic because of elevated triglyceride content or frank blood respectively. Benign asbestos related pleural effusion constitutes another recognized exudative entity. Management priorities in exudative effusions include targeted therapy for infection or malignancy and procedural drainage when respiratory compromise or diagnostic uncertainty exists.

Less common causes merit attention because they may present atypically or defy initial classification. Pulmonary embolism can produce either transudative or exudative effusions depending on the degree of infarction associated with inflammation and right heart strain. Drug induced pleural disease is an important iatrogenic cause with agents such as methotrexate amiodarone phenytoin and dasatinib implicated in case series and reports; manifestations are typically exudative and may resolve with drug withdrawal. Radiation to the thorax can damage pleural microvasculature generating exudative fluid. Esophageal rupture produces a chemical and often infects exudative effusion. Rare systemic syndromes such as ovarian hyperstimulation can also produce pleural collections through mechanisms that remain incompletely defined. [8][9] Accurate etiologic classification depends on integration of pleural fluid biochemistry with clinical history imaging and additional laboratory tests. Recognition of the principal mechanisms hydrostatic pressure oncotic pressure capillary permeability and lymphatic drainage directs focused evaluation. Treating the root cause reduces recurrence. When etiology remains unclear targeted diagnostic procedures including pleural biopsy or advanced imaging may be required to obtain a definitive diagnosis [8][9].

Epidemiology

Pleural effusion represents the most frequently encountered pleural disorder and is responsible for considerable clinical and economic burden. In the United States alone, approximately 1.5 million patients are affected each year, generating significant healthcare utilization and costs. [10] Despite this high prevalence, the epidemiology of pleural effusion remains incompletely characterized, as few large-scale population-based studies exist to define its distribution, risk factors, and changing patterns across regions. This gap in epidemiological knowledge limits a comprehensive understanding of disease dynamics and complicates healthcare planning. The distribution of pleural effusion varies by geography, reflecting differences in underlying causes, healthcare access, and disease prevalence. In developed nations, transudative effusions are most often linked to congestive heart failure, which remains the leading cause. Malignant pleural effusions, typically associated with lung cancer, breast cancer, and lymphomas, represent another major contributor, accounting for a substantial proportion of cases referred to specialist care. Parapneumonic effusions and empyema continue to be important, particularly in older adults and in

patients with comorbidities that predispose pulmonary infection. Together, these three categories—heart failure, malignancy, and parapneumonic processes—constitute the majority of annual cases worldwide [8][9].

In contrast, in low- and middle-income countries, tuberculosis accounts for a much larger share of pleural effusions. The higher prevalence of TB-associated effusion in these regions highlights the role of socioeconomic, environmental, and public health factors in shaping epidemiological patterns. Additionally, chronic liver disease leading to hepatic hydrothorax, nephrotic syndrome, and connective tissue disorders make meaningful contributions in certain populations, though precise global estimates remain limited. Overall, pleural effusion is a common endpoint for diverse systemic and local diseases. Its epidemiology reflects the prevalence of cardiovascular, infectious, and neoplastic conditions within a population. Continued surveillance and population-based studies are needed to refine incidence data, identify trends, and guide allocation of healthcare resources for early detection and management [8][9].

Pathophysiology

The pleural space normally contains a very small amount of fluid, estimated at 0.1 to 0.3 mL per kilogram of body weight. This thin layer of liquid plays an essential role in hydromechanical coupling between the lung and chest wall, ensuring smooth gliding during respiration. The origin of pleural fluid lies in the systemic vessels of the parietal pleura, where hydrostatic pressure drives filtration into the pleural cavity. Clearance occurs primarily through lymphatic channels concentrated in the dependent areas of the pleura, especially in the parietal layer, where stomata lead directly to lymphatic vessels. This finely tuned balance between formation and absorption maintains normal pleural fluid homeostasis. Disturbance of this equilibrium results in pleural effusion, which can occur through either excessive production of pleural fluid or impaired clearance. Several mechanisms explain the pathophysiology. One of the most important is elevation of pulmonary capillary pressure, commonly seen in left-sided heart failure or renal failure with fluid overload. In these conditions, increased hydrostatic forces push plasma ultrafiltrate across capillary membranes into the pleural cavity, producing transudative effusions. Another central mechanism is increased pulmonary capillary permeability, as occurs in pneumonia, tuberculosis, or systemic inflammatory processes. In these settings, inflammatory mediators damage endothelial junctions and allow proteins, cells, and large molecules to enter the pleural space, producing exudates. Decreased intrapleural pressure also contributes to fluid accumulation. In atelectasis, negative intrapleural pressures generated by lung collapse favor fluid transudation into the pleural cavity. Similarly, reduction in plasma oncotic pressure reduces the force that normally retains fluid within vascular compartments. Hypoalbuminemia from nephrotic syndrome, advanced liver cirrhosis, or severe malnutrition lead to low oncotic pressure and facilitates pleural fluid accumulation [10][11].

Another mechanism is increased pleural membrane permeability due to infection, trauma, or inflammation. This is frequently observed in parapneumonic effusions and empyema. Direct injury to the pleura disrupts the mesothelial barrier, promoting local exudation. Malignancy represents a unique process in which tumor infiltration obstructs lymphatic drainage, especially through the parietal pleural lymphatics, impairing clearance of pleural fluid. Malignant pleural effusions often combine increased permeability with lymphatic obstruction, explaining their tendency to recur despite drainage. Migration of fluid from extrathoracic sites further expands the pathophysiological spectrum. Hepatic hydrothorax exemplifies this process, where ascitic fluid from cirrhosis traverses small diaphragmatic defects into the pleural cavity, usually on the right side. Similarly, peritoneal dialysis may allow dialysate fluid to track into the thorax. Retroperitoneal processes such as urinorhorrax, where urine from urinary tract rupture enters the pleural space, also fall into this category. Rupture of vascular or lymphatic structures can produce hemorrhagic or lipid-rich effusions. Haemothorax arises from disruption of blood vessels following trauma, surgery, or vascular malformations. Chylothorax results from thoracic duct injury or obstruction, allowing leakage of chyle rich in triglycerides into the pleural space. Both are classified as exudates due to their protein and lipid content [11][12].

Drug-induced pleural effusion represents another mechanism. Medications such as amiodarone, methotrexate, and certain tyrosine kinase inhibitors provoke effusions through direct toxicity, immune-mediated injury, or disruption of pleural clearance mechanisms. Radiation therapy may act similarly, increasing vascular permeability within the pleura. [11][12] Overall, pleural effusion develops from a limited set of physiological disturbances: increased hydrostatic pressure, reduced oncotic pressure, increased capillary or pleural permeability, impaired lymphatic drainage, direct leakage from vessels or ducts, or transdiaphragmatic migration of fluid. While the mechanisms differ, the clinical result is the same: accumulation of fluid within the pleural space that interferes with normal respiratory mechanics. Recognition of these pathophysiological processes is essential for accurate diagnosis, classification into transudates or exudates, and effective management tailored to the underlying cause [11][12].

History and Physical

The clinical manifestations of pleural effusion vary widely, ranging from asymptomatic cases discovered incidentally on imaging to patients presenting with disabling dyspnea. The degree of symptomatology does not correlate linearly with the volume of effusion. Instead, the mechanical restriction imposed on thoracic expansion appears to be the primary determinant of breathlessness. [13] In some patients, small effusions may produce significant symptoms if lung compliance is already compromised by parenchymal disease, whereas others may tolerate large effusions with minimal complaints. Oxygen exchange impairment, reduced lung expansion, and coexisting pulmonary pathology such as pulmonary edema in heart failure contribute further to the symptom profile. The most common symptom is exertional dyspnea, which gradually worsens as effusion size increases. Cough is frequent, often nonproductive, and can result from compression or irritation of the airways. Fever, night sweats, and systemic malaise may accompany infectious or inflammatory causes such as pneumonia or tuberculosis. Chest pain, especially when pleural inflammation is present, is another important complaint. Pleuritic pain is typically sharp, localized, and characteristically worsens with inspiration or coughing, following a crescendo-decrescendo pattern. This pain frequently diminishes as fluid accumulates and separates inflamed pleural surfaces. By contrast, constant unremitting chest pain raises suspicion of malignancy, particularly mesothelioma or metastatic disease. On

rare occasions, massive effusions can exert sufficient intrathoracic pressure to impair venous return, leading to hemodynamic compromise that clinically mimics cardiac tamponade [13].

Physical examination provides valuable clues but is often influenced by the volume of fluid present. In large effusions, inspection may reveal asymmetric chest expansion with fullness of intercostal spaces and lag of the affected side during inspiration. Palpation often demonstrates reduced tactile fremitus over the effusion due to dampened transmission of vibrations through fluid. Percussion characteristically yields dullness in dependent zones where fluid accumulates, contrasting with resonant sounds over aerated lung fields. On auscultation, breath sounds are typically diminished or absent over the area of fluid, although bronchial breathing and egophony may be heard just above the fluid meniscus, where compressed lung transmits sound with altered tonal quality. Egophony, a nasal bleating quality to the patient's vocalization, is a classic sign that localizes to the superior margin of an effusion. During active pleural inflammation without significant fluid, a pleural friction rub may be heard. This low-pitched, grating sound can be confused with coarse crackles but is distinguished by its persistence throughout the respiratory cycle and its close association with pleuritic pain [13].

A detailed history is critical for determining etiology. Inquiry should cover comorbid illnesses such as congestive heart failure, chronic kidney disease, liver cirrhosis, autoimmune conditions, or malignancy. Medication history is also important, as several agents including amiodarone, methotrexate, and certain chemotherapeutics have been associated with pleural effusion. A history of tuberculosis exposure, recent pneumonia, or systemic infection provides diagnostic clues in patients presenting fever and respiratory symptoms. Occupational history may reveal exposure to asbestos, suggesting a risk for mesothelioma or benign asbestos-related effusion. The physical examination should extend beyond the chest to assess systemic signs of disease. Findings such as jugular venous distension, an S3 gallop, and peripheral edema strongly suggest congestive heart failure as the underlying cause. Stigmata of chronic liver disease, including ascites, spider angiomas, and caput medusae, may indicate hepatic hydrothorax as the source. In patients with nephrotic syndrome, generalized edema and frothy urine provide additional context. Cachexia, lymphadenopathy, or unexplained weight loss may direct suspicion toward malignancy. Ultimately, the history and physical examination offer indispensable insights into the pathogenesis and cause of pleural effusion. While imaging and diagnostic procedures confirm the diagnosis, bedside clinical evaluation remains the cornerstone of initial assessment, guiding further workup and shaping the clinician's index of suspicion for specific underlying conditions [13].

Evaluation

Chest radiography remains the initial imaging modality for suspected pleural effusion. An upright posteroanterior radiograph can show blunting of the costophrenic angle with small effusions. A meniscus sign forms when fluid collects to a volume that typically exceeds two hundred milliliters. Lateral decubitus radiographs increase sensitivity and can detect as little as fifty milliliters of pleural fluid. These plain radiographs guide the need for further imaging and procedural planning. Ultrasound offers higher sensitivity than radiography. It is portable and can be performed at the bedside. Thoracic ultrasound confirms effusion and helps characterize fluid. It also guides safe thoracentesis and reduces complication rates. Computed tomography provides anatomic detail. CT clarifies complex anatomy and identifies loculations, pleural thickening, nodularity, and pulmonary parenchymal disease that may coexist with effusion. CT is valuable when malignancy, empyema, or complex parapneumonic disease is suspected. Ultrasound and CT complement each other in diagnostic workflows. [15][7][8][14]

Thoracentesis is the cornerstone of diagnostic evaluation for new pleural effusions except when heart failure is strongly suspected and a trial of diuresis is planned. Thoracentesis yields fluid for laboratory analysis and produces symptom relief. Integration of thoracic ultrasound into the decision to perform thoracentesis is recommended. Rapid availability of ultrasound often reduces the need for CT and informs whether the effusion is simple or complex. Ultrasound can also distinguish effusion from other radiopaque processes such as consolidation or atelectasis. On ultrasound, simple transudative or uncomplicated exudative fluid usually appears anechoic or hypoechoic. Complex effusions show internal echoes, septations, or loculations. The presence of septations or echogenic debris suggests complicated parapneumonic effusion or empyema and often mandates chest tube placement. Hemothorax may demonstrate the hematocrit sign with swirling, smoke-like echoes. These sonographic features help determine the urgency and type of drainage required. [15][7][8][14]

Laboratory testing should be systematic. Blood tests include complete blood count with differential, serum electrolytes, renal and liver panels, albumin, lipase, and cardiac biomarkers when indicated. Pleural fluid analysis should include pH, glucose, total protein, lactate dehydrogenase LDH, cell count with differential, Gram stain, and culture. Cytology should be sent when malignancy is a concern. Other targeted tests include adenosine deaminase ADA or *Mycobacterium tuberculosis* studies for suspected tuberculous effusion, pleural fluid amylase for pancreatitis related effusion, and pleural fluid triglycerides when chylothorax is suspected. A milky appearance and triglyceride concentration above 110 mg per dL indicate chylothorax. Hemothorax is diagnosed when pleural fluid hematocrit is greater than 50 percent of the serum hematocrit. These condition specific assays expedite etiologic diagnosis and direct therapy. Light's criteria remain central to fluid classification. Use pleural fluid and serum protein ratios and LDH ratios to identify exudative effusions. Interpret Light's criteria in the clinical context. Prior diuretics can concentrate pleural fluid and misclassify transudates as exudates. When heart failure is suspected but Light's criteria indicate exudate, measure pleural fluid B type natriuretic peptide or calculate protein or albumin gradients. A serum to pleural fluid albumin gradient greater than 1.2 g per dL or a serum to pleural fluid total protein gradient greater than 2.5 g per dL supports a transudative process in suspected heart failure. Use these adjuncts to avoid unnecessary invasive testing. [16]

Cellular analysis of pleural fluid refines the differential. Neutrophil predominance points to acute processes such as parapneumonic effusion or pulmonary embolism. Lymphocyte predominance narrows the differential toward tuberculosis, lymphoma, chronic inflammatory disorders, post cardiac surgery effusions, and malignancy. Eosinophilic effusions are uncommon. They suggest pneumothorax, hemothorax, parasitic infection, or drug induced disease. Extremely elevated pleural LDH values, for example above one thousand units per liter, raise concern for empyema, tuberculosis, or certain malignancies such as lymphoma. A pleural fluid pH below 7.20 in a parapneumonic effusion indicates a complicated effusion and typically

requires chest tube drainage and intrapleural therapies. Low pH in pleural fluid also occurs with esophageal rupture and rheumatoid pleuritis. Use cell counts, pH, glucose, and LDH together to determine the need for immediate drainage and antimicrobial therapy [16].



Figure 1: X-Ray of Pleural Effusion.

Cytological evaluation has moderate sensitivity on the first thoracentesis. The approximate diagnostic yield for malignant cells on the first sample is around sixty percent. Yield increases with repeated sampling and reaches higher values when three separate samples are obtained on different days. Persistent cytology negative results in the face of high clinical suspicion warrant tissue diagnosis. Medical thoracoscopy or video assisted thoracoscopic surgery VATS with pleural biopsy offers the highest diagnostic yield for malignancy and tuberculous pleuritis. If imaging shows pleural nodularity, thickening, or masses, proceed to pleural biopsy sooner rather than later. Thoracoscopy allows direct visualization, targeted biopsies, and therapeutic interventions in the same session. [17] When initial noninvasive testing is inconclusive, percutaneous image guided pleural biopsy can be effective. Closed needle biopsy retains a role where thoracoscopy is not available. The choice among biopsy techniques depends on suspected diagnosis, local expertise, and patient fitness. In suspected tuberculosis, pleural biopsy increases diagnostic sensitivity when pleural fluid studies are non-diagnostic [16][17].

Consider fewer common causes when routine tests do not yield a cause. Check pleural and serum creatinine when urinothorax is possible. Measure pleural fluid amylase when pancreatitis or esophageal rupture is in the differential. Evaluate pleural elastance and lung reexpansion on drainage when trapped lung is suspected. Trapped lung and lung entrapment require different management strategies and may prompt early referral to thoracic surgery or pleural specialists. Finally, synthesize data from history, examination, imaging, and fluid analysis to direct targeted therapy. Distinguish transudative processes that demand correction of systemic disease from exudative processes that often need local drainage and specific therapy. Use ultrasound guidance for all invasive pleural procedures. Reserve CT and thoracoscopy for complex or uncertain cases. Incorporate condition specific markers to reduce diagnostic delay. Escalate to surgical or thoracoscopic approaches when cytology and percutaneous biopsy fail to provide a definitive diagnosis. This structured evaluation optimizes diagnostic yield and minimizes procedural risk while guiding effective patient centered care [16][17].

Treatment / Management

The management of pleural effusion centers on addressing the underlying cause while simultaneously alleviating symptoms. In most cases, treatment involves both diagnostic and therapeutic procedures, with decisions tailored to the patient's clinical status, the etiology of the effusion, and the presence or absence of symptoms. Symptomatic patients generally benefit from drainage of pleural fluid, which can provide rapid relief from dyspnea and discomfort. For asymptomatic individuals, fluid drainage is usually reserved for diagnostic purposes, unless infection or hemorrhage is suspected. In cases of heart failure, thoracentesis is typically not performed unless diuretics fail or the patient experiences

significant distress. Chylous effusions are initially approached with conservative strategies such as dietary modification and somatostatin analogs, though surgical interventions may be required in persistent cases [18][19].

Thoracentesis remains the cornerstone diagnostic and therapeutic tool. Bedside ultrasound guidance has become a standard practice, significantly improving the accuracy of the procedure and reducing complications such as pneumothorax. Ultrasound can also identify fluid loculations, which may necessitate a different approach to drainage. Every sample of pleural fluid should be sent for biochemical, cytological, and microbiological analysis to establish the nature of the effusion. Light's criteria help differentiate between transudates and exudates, though the clinical context must always guide interpretation. A pleural fluid pH measurement is essential, especially in suspected parapneumonic effusions or empyema, since a pH below 7.2 usually indicates the need for chest tube insertion. Complex parapneumonic effusions and empyema require chest tube drainage combined with antibiotic therapy. Small-bore chest tubes (10–14 gauge) are now preferred because they are equally effective compared to large-bore tubes, easier to insert, and associated with less discomfort. For patients with loculated effusions that do not drain adequately, intrapleural fibrinolytic therapy combined with DNase has shown to improve fluid clearance and reduce the need for surgical intervention. When conservative measures fail, thoroscopic decortication may be necessary to remove thickened pleural peel and restore lung expansion [18][19].

Management of malignant pleural effusions requires a palliative approach, as these often recur and rarely resolve spontaneously. Repeated thoracenteses may be offered for symptom control, but frequent aspirations are generally discouraged due to patient burden and risks of infection. Pleurodesis using talc or other sclerosing agents is a widely accepted option, promoting adherence of the pleural layers to prevent fluid reaccumulation. Alternatively, tunneled pleural catheters can be placed for patients with recurrent symptomatic effusions, allowing controlled outpatient drainage and minimizing hospital stays. Importantly, large-volume thoracentesis should be performed cautiously, with a maximum recommended removal of about 1500 mL in a single session to reduce the risk of reexpansion pulmonary edema. Supportive care such as supplemental oxygen, analgesia, and optimization of comorbidities (for example, managing heart failure with diuretics or addressing liver disease) is essential to complement procedural interventions. Overall, treatment of pleural effusion is multifaceted, requiring accurate diagnosis, careful procedural technique, and individualized long-term strategies to manage recurrent or complex cases effectively [20][21].

Table 1: Management Strategies Based on Effusion Type and Context.

Effusion Type / Context	Primary Goal	First-Line Management	Secondary / Advanced Options
Suspected Heart Failure	Treat underlying cause.	Diuretic therapy.	Therapeutic thoracentesis if diuresis fails or for symptom relief.
Uncomplicated Parapneumonic	Resolve with antibiotics.	Antibiotics are targeted to pathogen.	Monitor with ultrasound; drainage if no improvement.
Complicated Parapneumonic/Empyema	Drain infected space.	Chest tube drainage + antibiotics.	Intrapleural fibrinolytic therapy (tPA/DNase); VATS decortication.
Symptomatic Malignant Effusion	Palliate symptoms prevent recurrence.	Therapeutic thoracentesis.	Talc Pleurodesis or Indwelling Pleural Catheter (IPC).
Chylothorax	Reduce chyle flow.	Conservative (NPO, TPN, Somatostatin).	Thoracic duct ligation or embolization.
Recurrent Benign Effusion	Prevent recurrence.	Treat underlying cause (e.g., optimize CHF meds).	Pleurodesis or (rarely) surgical pleurectomy.

Differential Diagnosis

The differential diagnosis for pleural effusion spans systemic disorders and localized pleural or pulmonary processes. Classifying effusions as transudative or exudative frames the diagnostic pathway. Transudative collections arise from alterations in hydrostatic or oncotic pressures or from mechanical transfer of fluid across diaphragmatic defects. The common cardiovascular cause is congestive heart failure where elevated pulmonary venous pressure forces plasma ultrafiltrate

into the pleural space. Pulmonary embolism may present with a transudate or with an exudate when infarction or inflammation accompanies the embolic event. Infradiaphragmatic conditions produce transudative effusions by permitting abdominal fluid to move into the thorax. Cirrhosis with hepatic hydrothorax occurs when ascitic fluid traverses small diaphragmatic defects. Peritoneal dialysis can allow dialysate to migrate into the pleural cavity. Disorders that reduce plasma oncotic pressure such as nephrotic syndrome or severe hypoalbuminemia favor transudation of fluid. Advanced renal failure and marked malnutrition produce similar biochemical milieus that promote pleural fluid accumulation [20][21].

Exudative effusions reflect local pleural inflammation, increased vascular permeability or impaired lymphatic drainage. Infectious causes are among the most frequent. Bacterial pneumonia commonly produces parapneumonic effusions that may progress to empyema if the pleural space becomes infected. Tuberculous pleuritis remains a major cause of exudative effusion in many low and middle income regions and typically yields a lymphocyte predominant fluid. Viral infections of the respiratory or systemic compartments can produce modest exudative collections. Fungal and parasitic agents cause pleural disease less often but are important in endemic areas and in immunocompromised hosts. Neoplastic processes rank high among exudative etiologies. Primary pulmonary malignancies frequently involve the pleura either by direct invasion or by obstructing lymphatic clearance, producing recurrent effusions. Metastatic disease from breast ovary colon and other visceral primaries commonly seeds the pleural surfaces and prompts exudation. Mesothelioma is a critical diagnostic consideration in patients with a history of asbestos exposure because it frequently presents unilateral recurrent pleural fluid and pleural thickening [20][21].

Infradiaphragmatic inflammatory and obstructive processes can lead to exudative effusions by direct extension of inflammatory mediators or by transdiaphragmatic passage of fluid. Acute pancreatitis often results in pleural fluid with elevated amylase due to transmigration of pancreatic enzymes. Peritonitis intraabdominal abscess and biliary leaks producing bilothorax represent additional mechanisms that link abdominal pathology to thoracic fluid collections. Meigs syndrome combines an ovarian neoplasm with ascites and a right sided pleural effusion and must be considered in women with this constellation of findings. Autoimmune disorders cause pleural inflammation and exudation. Rheumatoid arthritis systemic lupus erythematosus and other connective tissue diseases can produce recurrent pleural effusions that are inflammatory in nature and that may resist simple drainage. Drug induced pleural disease is an iatrogenic cause of exudative fluid. A range of medications have been implicated through immune mediated injury direct toxicity or disruption of pleural clearance mechanisms [20][21].

Postoperative and traumatic states generate exudative collections by direct injury to the pleura or thoracic duct. Cardiac and thoracic surgery may yield hemothorax chylothorax or inflammatory effusions. Blunt or penetrating trauma can perforate vessels or lymphatics producing hemorrhagic or chylous effusions. Less common but clinically significant causes include urinorhthorax from urinary tract disruption amyloidosis with pleural deposition esophageal rupture producing a chemical and often infected effusion and benign asbestos related pleural effusion. Each of these entities carries diagnostic features that can be elicited through targeted history and investigations. Differentiating among these causes requires integration of clinical presentation imaging and pleural fluid analysis. Light's criteria separate transudates from exudates but may misclassify effusions in patients who have received diuretics or who have mixed mechanisms. Imaging with chest radiography ultrasound and computed tomography refines the differential by revealing loculations pleural thickening nodularity or underlying pulmonary disease. Specific laboratory tests such as pleural fluid amylase adenosine deaminase triglycerides and microbiologic studies narrow the possibilities. Clinical clues such as congestive signs suggest cardiac origin whereas fever pleuritic pain and leukocytosis indicate infection. Weight loss lymphadenopathy or known malignancy increase the likelihood of neoplastic effusion [20][21].

Prognosis

Data on prognostic factors for patients with pleural effusion remain limited. Malignant pleural effusion carries a poor prognosis and correlates with reduced survival across tumor types. [22][23][22] By contrast, outcomes for nonmalignant effusions have not been defined with the same precision. Few large prospective cohorts have quantified long term mortality for these patients, and existing series use heterogeneous inclusion criteria and variable follow up, which complicates direct comparison across studies. Several prospective investigations, however, link the presence of pleural fluid to an increased risk of death independent of the primary diagnosis. [3][24] These studies indicate that pleural effusion often marks advanced disease or systemic derangement and that its occurrence should prompt clinicians to reassess overall prognosis.

Prognostic assessment should integrate the etiology of the effusion, the patient's comorbid conditions, performance status, and response to initial therapy. Malignancy, persistent empyema, and bilateral large effusions most often predict worse outcomes. The physiologic impact of effusion on gas exchange and ventilatory mechanics contributes to acute risk, particularly in patients with underlying cardiopulmonary disease. In addition, recurrent effusions that require repeated interventions signal an ongoing pathologic process that may shorten survival and increase morbidity. Biomarkers and pleural fluid characteristics provide limited prognostic information beyond establishing cause. Cytologic positivity for malignant cells implies invasive disease and associates with shortened survival when compared with cytology negative malignant effusions, but the magnitude of this effect varies by tumor histology and available oncologic treatment. Clinical use of prognostic information should focus on guiding goals of care, tailoring intervention intensity, and prioritizing symptom relief. For patients with malignant effusion and limited life expectancy, emphasis often shifts to outpatient management options that control symptoms while minimizing hospital time. For nonmalignant effusions, identifying reversible drivers such as heart failure or hepatic hydrothorax and optimizing medical therapy can improve outcomes. Future research should aim to define standardized prognostic models that combine clinical, radiologic, and pleural fluid variables to predict survival and inform shared decision making [23][24].

Complications

Pleural effusion, while frequently treatable, carries the risk of significant complications that can impair respiratory health and overall prognosis. Among the most serious is empyema, which arises when the pleural space becomes infected, leading to the accumulation of purulent material. Empyema is associated with systemic manifestations such as fever, leukocytosis, and in advanced cases, sepsis. The infected fluid can compromise pulmonary mechanics by restricting lung expansion and worsening dyspnea. Management requires prompt initiation of broad-spectrum antibiotics, with adjustments guided by culture results. In many cases, invasive procedures are necessary to achieve adequate drainage. Thoracentesis is often used initially, but in more severe or loculated cases, the placement of a chest tube thoracostomy (CTT) is indicated to facilitate continuous drainage and reduce the risk of ongoing infection. Failure to manage empyema effectively can lead to persistent infection, chronic fibrothorax, and high morbidity [23].

Another notable complication is pleural thickening, which occurs due to fibrous adhesions forming within the pleural layers. This condition frequently develops after recurrent or unresolved effusions, or as a sequela of chronic inflammatory processes such as tuberculosis or recurrent parapneumonic effusion. Pleural thickening limits lung compliance and can result in restrictive ventilatory defects, with patients experiencing exertional dyspnea and reduced pulmonary function on spirometry. The progression of fibrotic changes can also cause trapped lung, where mechanical restriction prevents adequate re-expansion despite fluid removal. Management strategies vary depending on severity. Mild cases may be addressed with pulmonary rehabilitation, corticosteroid therapy, or supportive measures aimed at preserving functional capacity. However, in advanced cases where adhesions markedly compromise lung mechanics, surgical options are often considered. Procedures such as video-assisted thoracoscopic surgery (VATS) decortication or open thoracotomy may be required to excise fibrous tissue and restore lung expansion [23]. The chronic impact of these complications extends beyond respiratory function, as both empyema and pleural thickening are linked to prolonged hospitalizations, increased healthcare costs, and reduced quality of life. Therefore, timely identification of complications, combined with evidence-based therapeutic interventions, is crucial to improving patient outcomes and preventing progression to irreversible disease states.

Patient Education

Patient education plays a central role in the prevention, early detection, and management of pleural effusion. Empowering patients with knowledge about lifestyle modifications, risk factors, and clinical warning signs is crucial for reducing the incidence and recurrence of this condition. Preventive education can be categorized into primary and secondary strategies, both of which have a significant impact on health outcomes. Primary prevention focuses on reducing the likelihood of developing diseases that predispose individuals to pleural effusion. Lifestyle interventions are foundational in this regard. Regular physical activity not only improves cardiovascular and pulmonary health but also helps maintain an optimal body weight, reducing the risk of conditions such as heart failure that frequently contribute to effusion. A balanced diet rich in fruits, vegetables, and lean proteins supports immune function and reduces systemic inflammation, while limiting alcohol consumption helps protect cardiac and hepatic function, thereby lowering the risk of cirrhosis and congestive heart failure—two common causes of pleural effusion. Smoking cessation remains one of the most critical preventive measures, as tobacco use is strongly associated with chronic obstructive pulmonary disease (COPD), lung cancer, and recurrent respiratory infections, all of which are significant risk factors for effusion [22][23][24].

Preventive healthcare also extends to immunization. Vaccination against respiratory pathogens such as influenza and *Streptococcus pneumoniae* can reduce the incidence of pneumonia, one of the leading causes of parapneumonic effusion. Patient education should emphasize adherence to vaccination schedules, particularly among older adults, individuals with chronic illnesses, and those who are immunocompromised. Occupational health and safety are additional aspects of prevention. Patients employed in industries with potential exposure to asbestos, silica, or other hazardous inhalants should be advised on the consistent use of protective respiratory equipment. Education on workplace safety policies and regular occupational health assessments can reduce the likelihood of pleural effusion related to occupational lung disease. Secondary prevention emphasizes the importance of early detection and prompt intervention to prevent complications and disease progression. Patient education should encourage regular medical check-ups, especially for individuals with known risk factors such as chronic heart disease, tuberculosis, or malignancy. Patients must be informed about the early signs of pleural effusion, including persistent shortness of breath, chest pain, dry cough, or unexplained fatigue, so they can seek medical attention without delay. Diagnostic tools such as chest radiography, ultrasound, or computed tomography may be necessary for individuals presenting with suggestive symptoms. Early imaging can detect effusion at a stage when it is more easily managed and before complications develop [23][24].

Timely treatment of underlying conditions is also critical to secondary prevention. For example, optimal management of heart failure with diuretics and lifestyle modifications can prevent recurrent effusions. Similarly, completing antibiotic courses for pneumonia and maintaining adherence to tuberculosis therapy can reduce the risk of pleural involvement. Patients with a prior history of pleural effusion should be educated about the possibility of recurrence and the importance of follow-up care. They should also be counseled on adherence to treatment plans that may include thoracentesis, indwelling pleural catheters, or pleurodesis when indicated. An essential component of patient education is highlighting the importance of self-care and active participation in disease management. Patients should be encouraged to monitor symptoms, keep a record of respiratory difficulties, and promptly report any changes to healthcare providers. This proactive approach allows for early interventions, reducing hospitalizations and improving long-term quality of life. Overall, effective patient education integrates lifestyle guidance, preventive healthcare measures, and practical instructions for monitoring and managing risk factors. By addressing both primary and secondary prevention, patients gain the knowledge and tools needed to reduce the incidence of pleural effusion, recognize it at an early stage, and prevent complications through timely medical care. This dual approach not only improves patient outcomes but also reduces the burden of disease on healthcare systems by decreasing hospital admissions and long-term complications [23][24].

Enhancing Healthcare Team Outcomes

The management of pleural effusion requires coordinated efforts among multiple healthcare professionals, as the condition often arises from diverse etiologies and demands both acute interventions and long-term care. Pulmonologists occupy a pivotal role by evaluating respiratory symptoms, confirming diagnoses through imaging and pleural fluid analysis, and determining whether interventions such as thoracentesis or chest tube placement are indicated. Their leadership in guiding care plans sets the foundation for effective management. However, complex cases often extend beyond pulmonology, making collaboration with other specialists essential for improving patient outcomes. Cardiothoracic surgeons contribute significantly when surgical interventions are necessary. For example, they may perform video-assisted thoracoscopic surgery (VATS) for decortication in cases of empyema or pleurodesis for recurrent malignant effusions. Their involvement ensures that patients with refractory or severe disease have access to advanced procedural options. Radiologists, in turn, play an indispensable role by providing accurate imaging assessments, from chest x-rays to CT scans and ultrasound, which inform both diagnosis and procedural planning. Interventional radiologists extend this contribution by guiding catheter placements and assisting in minimally invasive procedures, reducing risks associated with traditional surgical approaches [25][26].

Pathologists bring clarity by analyzing pleural fluid or tissue samples to identify infectious, malignant, or inflammatory etiologies. Their findings direct therapeutic strategies, especially in patients with suspected cancer, tuberculosis, or autoimmune conditions. In many cases, oncologists, cardiologists, hepatologists, and infectious disease experts are engaged to treat underlying causes such as malignancy, heart failure, cirrhosis, or complex infections. This multidisciplinary diagnostic and therapeutic input is vital to reducing morbidity and preventing recurrence. Nurses act as frontline providers who monitor patients during and after procedures, recognize early signs of complications, and educate patients on self-care strategies. Their role in care coordination ensures seamless communication across specialties, reducing delays in treatment. Respiratory therapists support symptom control by administering oxygen therapy, guiding patients in breathing exercises, and optimizing ventilatory support when necessary. Physical therapists further contribute by designing rehabilitation programs that improve lung expansion, restore mobility, and enhance overall functional status after prolonged illness or surgical procedures [25][26].

Patient-centered care in pleural effusion also hinges on education and psychosocial support, areas where nursing staff and allied health professionals are particularly effective. They help patients and families understand diagnostic procedures, treatment options, and preventive strategies, fostering adherence to care plans. Social workers may also be involved in addressing barriers to care, especially for patients requiring long-term catheter management or palliative interventions. The success of this interprofessional team depends on effective communication, structured care pathways, and clearly defined responsibilities. Multidisciplinary meetings and case discussions provide opportunities to align treatment goals and ensure continuity of care. Evidence suggests that integrated team-based care not only reduces hospital stays but also improves patient satisfaction and functional recovery in conditions like pleural effusion [25][26]. Ultimately, the collective expertise of pulmonologists, surgeons, radiologists, pathologists, nurses, and rehabilitation specialists enhances outcomes by delivering comprehensive and coordinated care tailored to the unique needs of each patient.

Conclusion:

Pleural effusion is not a disease in itself but a significant clinical sign of underlying pathology, ranging from common conditions like congestive heart failure to complex issues such as malignancy or empyema. This review underscores the critical importance of a systematic and evidence-based approach to its management, which is fundamental to improving patient outcomes. The journey begins with a meticulous clinical assessment and is greatly enhanced by the strategic use of diagnostic imaging, primarily chest radiography and thoracic ultrasound, the latter being indispensable for both confirming the presence of fluid and safely guiding invasive procedures. The cornerstone of the diagnostic process remains the analysis of pleural fluid obtained via thoracentesis. The application of Light's criteria provides a robust framework for classifying effusions as transudates or exudates, thereby effectively narrowing the differential diagnosis and directing subsequent investigation. However, this classification must be interpreted within the full clinical context, as factors like diuretic use can confound results. Further fluid analysis, including cell counts, pH, glucose, and targeted biomarkers, refines the diagnosis and dictates management urgency, such as the need for chest tube drainage in complicated parapneumonic effusions. Management is inherently dualistic, requiring simultaneous attention to symptomatic relief and the treatment of the underlying cause. While therapeutic thoracentesis offers immediate relief from dyspnea, long-term success depends on addressing the primary driver, be it optimizing diuresis for heart failure, administering antibiotics for infection, or initiating oncologic therapy for malignancy. For recurrent symptomatic effusions, particularly those of malignant origin, strategies like talc pleurodesis or indwelling pleural catheters provide effective palliative options, enhancing quality of life. Ultimately, the successful management of pleural effusion relies on an interprofessional team model. Collaboration among pulmonologists, radiologists, pathologists, oncologists, cardiologists, specialized nurses, and respiratory therapists ensures comprehensive care. By integrating clinical expertise with diagnostic precision and patient-centered therapeutic strategies, healthcare professionals can effectively alleviate suffering, reduce recurrence, manage complications, and improve the overall prognosis for patients presenting with this common yet complex condition.

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