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Lipoid pneumonia: a narrative review of recent developments

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Abstract
Lipoid pneumonia is an uncommon pulmonary condition resulting from the accumulation of lipids within the alveolar spaces. It is classified as exogenous when caused by aspiration or inhalation of lipid-containing substances or endogenous when lipids accumulate from cellular breakdown within the body. Despite increasing recognition, lipoid pneumonia remains diagnostically challenging due to nonspecific clinical and radiological presentations that often mimic malignancy or infectious processes. This narrative review synthesizes recent developments in the understanding of lipoid pneumonia, with particular emphasis on epidemiology, pathophysiology, diagnostic approaches, treatment strategies, and emerging etiologies, including vaping-related lung injury. Critically, the evidence base for lipoid pneumonia consists predominantly of case reports, case series, and small retrospective studies, with no randomized controlled trials or prospective cohort studies available to guide clinical decision-making. The natural history remains poorly characterized, and optimal therapeutic interventions beyond supportive care and elimination of exposure have not been rigorously evaluated. This review highlights key knowledge gaps and proposes directions for future research to improve the diagnosis and management of this challenging condition.

Introduction

Lipoid pneumonia represents a rare but clinically significant form of pulmonary disease characterized by the accumulation of lipid material within the lung parenchyma.[1,2] The condition was first described in the early 20th century and has since been recognized in two distinct forms: exogenous lipoid pneumonia (ELP), resulting from aspiration or inhalation of lipid-containing substances, and endogenous lipoid pneumonia, arising from accumulation of lipid-rich cellular debris.[3,4]

Autopsy series have reported an incidence of 1.0-2.5% in adults and 8.8% in children, though these figures likely underestimate the true prevalence given the diagnostic challenges and frequent asymptomatic presentation.[2] The condition has gained renewed attention in recent years due to emerging etiologies, particularly vaping-associated lung injury, and improved recognition through advanced imaging techniques.[5,6]

Despite increasing clinical awareness, lipoid pneumonia remains diagnostically challenging. The presentation is highly variable, ranging from asymptomatic radiographic findings to life-threatening acute respiratory distress syndrome (ARDS).[7,8] Radiological features are often nonspecific and may mimic malignancy, leading to unnecessary invasive procedures, including lobectomy in some cases.[1,2] Furthermore, the evidence base for diagnosis and treatment remains severely limited, consisting primarily of case reports and small retrospective series rather than prospective trials or controlled studies.[7]

This narrative review aims to synthesize recent developments in the understanding of lipoid pneumonia, with particular focus on diagnostic approaches, treatment strategies, natural history, and emerging etiologies. We emphasize the critical gaps in the current evidence base and propose directions for future research.

Epidemiology and etiology

Exogenous lipoid pneumonia

ELP develops when lipid-containing substances enter the airways through aspiration or inhalation, inciting an inflammatory response.[1] The largest retrospective series to date, from the Mayo Clinic, identified only 34 patients over a 22-year period (1998-2020), with a mean age of 71 years and no sex predominance.[1] A French multicenter study reported 44 cases, representing the other major retrospective series available in the literature.[9]

Common causative agents include mineral oil (particularly when used as a laxative), petroleum-based products, lipid-containing nasal decongestants, and various occupational exposures.[9,10] In the French series, 30 of 40 non-occupational cases were related to aspiration of liquid paraffin used for constipation treatment.[9] Predisposing conditions that facilitate aspiration or inhalation were present in 77% of patients, most commonly gastroesophageal reflux disease (45%) and neurological or psychiatric illness (32%).[9]

Recent case reports have identified novel etiologies, including avocado/soybean unsaponifiables used for osteoarthritis, medicated vapor rubs applied intranasally, and various cosmetic agents.[10,11] Occupational exposures to cutting oils and industrial vapors have also been documented.[9]

Vaping-associated lipoid pneumonia

The emergence of e-cigarette or vaping product use-associated lung injury (EVALI) represents a significant recent development. The 2019 outbreak in the United States resulted in over 2800 cases and 68 deaths, predominantly in young adults (median age 24 years).[5,6] While initial reports suggested lipoid pneumonia as the underlying mechanism, subsequent pathological studies demonstrated findings more consistent with acute lung injury and chemical pneumonitis rather than classic lipoid pneumonia.[6,12] The majority of EVALI cases (>80%) were associated with tetrahydrocannabinol-containing products, with vitamin E acetate identified as a likely causative agent.[6,13]

However, sporadic cases of true ELP related to vaping of nicotine-containing and oil-based products have been documented both before and after the EVALI outbreak, representing a distinct clinical entity from EVALI.[14-16]

Endogenous lipoid pneumonia

Endogenous lipoid pneumonia results from accumulation of lipid-rich debris from destroyed alveolar cells, typically in the setting of bronchial obstruction, chronic inflammation, or inborn errors of metabolism such as Niemann-Pick disease.[3,4,17] This form is less common and often associated with underlying pulmonary or systemic disease.

Pathophysiology and histopathology

The pathophysiological response to lipid accumulation in the lungs depends on the chemical characteristics of the inhaled substance.[3,7] Animal fats cause an intense inflammatory reaction, while mineral or vegetable oils stimulate a foreign body reaction.[3]

Histologically, ELP is characterized by larger and more variably sized lipid droplets in the interstitium, often associated with histiocytic giant cells and foamy macrophages.[3] Endogenous lipoid pneumonia demonstrates macrophages with foamy cytoplasm filling alveoli, with smaller, more uniform lipid droplets.[3] Lipid-laden macrophages may be present in the interstitium in both forms but are more common in exogenous disease.[3]

Chronic inflammation may lead to interstitial fibrosis, and the pathological examination often reveals lesions of various ages within the same patient, suggesting ongoing or recurrent exposure.[18] The progression from acute inflammation to chronic fibrosis remains poorly understood, and the mechanisms underlying disease progression versus resolution have not been systematically studied.[3]

Clinical presentation

The clinical presentation of lipoid pneumonia is highly variable and nonspecific.[1,7] In the Mayo Clinic series, 50% of patients were completely asymptomatic at diagnosis, with findings discovered incidentally on imaging.[1] Among symptomatic patients, the most common manifestations include cough (64%), dyspnea (50%), fever (39%), weight loss (34%), and chest pain.

The disease typically follows a chronic, indolent course, though acute presentations can occur and may be life-threatening.[3,7,8] A recent case report described severe ARDS requiring mechanical ventilation following accidental aspiration of sewing machine oil in a previously healthy middle-aged man.[8] Acute presentations are more common with hydrocarbon aspiration, particularly fuels and industrial oils.[19]

Possible complications include superinfection by nontuberculous mycobacteria, pulmonary fibrosis, respiratory insufficiency, cor pulmonale, and hypercalcemia.[7] The association with mycobacterial infection is particularly important to recognize during diagnostic evaluation.[20]

Diagnostic approach

Clinical history

A thorough exposure history is essential for diagnosis.[7] Clinicians should specifically inquire about use of mineral oil laxatives, nasal application of petroleum-based products, occupational exposures, vaping habits, and use of lipid-containing supplements or medications.[8,10] Predisposing conditions for aspiration, including gastroesophageal reflux, neurological disorders, and swallowing dysfunction, should be assessed.[9]

Imaging findings

High-resolution computed tomography is the optimal imaging modality for diagnosis (Figure 1).[7] The most characteristic finding is the presence of fat attenuation (-30 to -150 Hounsfield units) within areas of consolidation or ground-glass opacity.[3,9] However, this finding is present in only 41-71% of cases, limiting its sensitivity.[1,9]

Common radiological patterns include consolidation (57%), ground-glass opacities (39%), irregular nodules or masses (23%), and the "crazy-paving" pattern characterized by ground-glass opacity with superimposed septal thickening.[3,9,18] The distribution is typically bilateral (79%), with predominance

in the posterior and lower zones (74%), often with subpleural sparing (52%).[9] Middle and lower lobe involvement is characteristic.[3]
Additional findings may include septal thickening, lymphadenopathy, thin-walled cysts, and pleural effusion in acute cases.[3] Importantly, lipoid pneumonia may demonstrate avidity on fluorodeoxyglucose positron emission tomography, mimicking malignancy.[2,21] The "positive angiogram sign" (visible pulmonary vessels within areas of low attenuation) may be helpful when density measurement is not possible.[9]

Bronchoscopy and bronchoalveolar lavage
Bronchoalveolar lavage (BAL) can support the diagnosis by demonstrating lipid-laden macrophages and a milky appearance of the lavage fluid.[7] Oil Red O staining confirms the presence of lipid within macrophages.[8,22] However, BAL findings are not specific for lipoid pneumonia, as lipid-laden macrophages can be seen in other conditions.[3]
In the Mayo Clinic series, BAL confirmed the diagnosis in only 5% of patients, with most diagnoses established by lung biopsy (71%) or CT imaging (24%).[1] The 2025 ERS/ATS guidelines note that BAL is not always required for diagnosis, and the optimal role of BAL in the diagnostic algorithm remains undefined.[3]
BAL cellularity may show lymphocytic alveolitis (23%), neutrophilic alveolitis (14%), or mixed patterns (31%).[9]

Histopathological examination
Lung biopsy is frequently performed, often because the diagnosis is not initially suspected and malignancy is a concern.[1,2] In the Mayo Clinic series, three patients underwent lobectomy for suspected malignancy before the diagnosis was established.[1]
Histological examination reveals foamy macrophages in alveoli, lipid vacuoles of variable size, and often multinucleated giant cells in exogenous disease.[3,22] Interstitial fibrosis may be present, particularly in chronic cases.[3] Special stains including Oil Red O or Sudan stains confirm lipid content.[22]

Diagnostic algorithm
Based on the available literature, we propose the following diagnostic approach (Table 1).

Treatment and Management
Evidence base for treatment
It is critical to emphasize that the evidence base for treatment of lipoid pneumonia is severely limited. There are currently no randomized controlled trials, prospective cohort studies, or validated treatment protocols available in the literature.[7,8] All treatment recommendations are based on case reports, small case series, expert consensus, and extrapolation from related conditions. The largest available studies are retrospective series of 34 and 44 patients, respectively.[1,9]

Elimination of exposure
There is consensus that the fundamental management principle is identifying and discontinuing exposure to the causative lipid substance.[7] However, even with complete cessation of exposure, the natural history is often unfavorable. In the Mayo Clinic series, over a median follow-up of 1.2 years, only 20% of patients with chronic respiratory symptoms improved, while 50% worsened.[1] Radiologically, CT abnormalities improved or resolved in only 33% of patients and progressed in 39%.[1] A recent 2026 case report similarly documented slow radiographic progression despite discontinuation of suspected lipid exposure and supportive care.[23]

Supportive care
For asymptomatic patients, treatment beyond elimination of exposure remains controversial.[7]

For symptomatic patients, supportive care is the mainstay of management, though its efficacy has not been systematically evaluated.[8,19]
In critically ill patients with acute presentations, intensive supportive measures are essential and may include mechanical ventilation, prone positioning, and extracorporeal membrane oxygenation (ECMO) as a bridge to recovery or transplantation.[8,19] In a series of 11 patients with fuel aspiration, one patient with irreversible lung injury required ECMO and lung transplantation.[19]

Corticosteroids
Systemic corticosteroids are commonly used in clinical practice, though their efficacy has not been rigorously evaluated in controlled studies.[7,8,19] The use of corticosteroids is based primarily on case reports and theoretical benefit in reducing inflammation.[20,24,25] A 2020 animal study demonstrated that dexamethasone reduced pulmonary inflammation in a rat model of acute ELP by inhibiting the NF-κB pathway, but human data remain limited to case reports.[26]
In the fuel aspiration series, antibiotics and steroids were the most commonly used treatments, though the independent contribution of steroids to outcomes could not be determined.[19]

Bronchoalveolar lavage and whole lung lavage
The role of therapeutic lavage remains controversial and inadequately studied.[3,7]
Bronchoalveolar lavage may be beneficial for removing lipid material, but significant risks exist, and the decision to employ BAL should be made selectively.[19]
Whole lung lavage has been reported in isolated case reports and small series, with some demonstrating clinical and radiological improvement.[7,27] A prospective study of 10 children with mineral oil aspiration showed significant improvement in CT findings, oxygen saturation, and clinical recovery following 4-10 therapeutic BAL procedures performed weekly.[28] However, this remains the only prospective study available, and it was limited to pediatric patients.
Successful treatment with whole lung lavage has also been reported in endogenous lipoid pneumonia due to Niemann-Pick Type B disease.[17] The 2025 ERS/ATS guidelines specifically identify the role of whole-lung lavage as a key research priority requiring better definition through comparative studies.[3]

Surgical interventions
Thoracoscopy with surgical debridement has been reported in patients with localized disease or paraffinomas.[7,20] In some cases, lobectomy or wedge resection has been performed when malignancy was suspected.[1,2]

Antibiotics
Antibiotics are frequently administered, particularly in acute presentations where superinfection cannot be excluded.[19] The association between lipoid pneumonia and nontuberculous mycobacterial infection necessitates consideration of mycobacterial coverage in appropriate clinical contexts.[7,20]

Natural history and prognosis
The natural history of lipoid pneumonia remains poorly characterized, representing a critical knowledge gap.[1] Available data suggest that the clinical course is often unfavorable even with appropriate management.
In the Mayo Clinic series, patients who deteriorated clinically and radiologically were older and had a higher prevalence of gastrointestinal disorders compared to those who remained stable or improved.[1] However, specific factors predicting progression versus resolution have not been systematically identified.[3]
Acute presentations, particularly those related to hydrocarbon aspiration, may have favorable outcomes with timely and efficient treatment, though severe cases can result in irreversible lung injury requiring transplantation.[19] Chronic cases are more persistent and difficult to treat.[20]

Long-term complications include progressive pulmonary fibrosis, respiratory insufficiency, and cor pulmonale.[7] The frequency with which lipoid pneumonia develops progressive fibrosis and the variables predicting this progression remain unknown.[3]

Knowledge gaps and future research directions

The current evidence base for lipoid pneumonia is severely limited, consisting predominantly of case reports, case series, and small retrospective studies. No randomized controlled trials or prospective cohort studies have been conducted to evaluate diagnostic approaches or therapeutic interventions. This represents a fundamental limitation in our ability to provide evidence-based care for patients with this condition.

The 2025 ERS/ATS guidelines on interstitial pneumonias specifically identify multiple critical research priorities for lipoid pneumonia:[3]

1. Diagnostic studies: the role of BAL in diagnosis requires better definition. While commonly performed, its sensitivity, specificity, and optimal place in the diagnostic algorithm remain unclear.
2. Therapeutic interventions: research into therapeutic interventions beyond supportive care is urgently needed. While corticosteroids are commonly used, their efficacy has not been rigorously evaluated in controlled studies. Similarly, the role of therapeutic lavage (both segmental BAL and whole lung lavage) requires better definition through comparative studies.
3. Natural history: prospective studies are needed to better characterize the natural history of the disease and identify factors predicting progression versus resolution. Current data suggest that many patients progress despite elimination of exposure, but the mechanisms underlying this progression are unknown.
4. Pathophysiology: improved understanding of the pathophysiological mechanisms underlying chronic inflammation and fibrosis in lipoid pneumonia may identify novel therapeutic targets. The molecular pathways leading from lipid accumulation to tissue injury and fibrosis have not been systematically investigated.
5. Outcomes research: factors associated with poor outcomes in lipoid pneumonia need to be identified through adequately powered studies. This would enable risk stratification and potentially guide intensity of treatment.
6. Endogenous lipoid pneumonia: the potential etiologies of endogenous lipoid pneumonia remain incompletely characterized, particularly in the context of immunological diseases and metabolic disorders.

Emerging considerations

Vaping-related lung injury

The EVALI outbreak has highlighted the potential for novel inhalational exposures to cause severe lung injury.[5,6,12,13] While most EVALI cases represent acute lung injury rather than classic lipoid pneumonia, sporadic cases of true ELP related to vaping continue to be reported.[14,16] Clinicians should maintain awareness of vaping as a potential etiology when evaluating patients with unexplained pulmonary infiltrates, particularly in younger populations.

Novel exposures

Case reports continue to identify previously unrecognized sources of lipid exposure, including dietary supplements, cosmetic products, and occupational exposures.[10,11] A high index of suspicion and detailed exposure history remain essential for diagnosis.

Diagnostic mimics

The propensity for lipoid pneumonia to mimic malignancy on both imaging and PET scanning represents a significant clinical challenge.[2,21] Awareness of this diagnostic pitfall may prevent unnecessary invasive procedures and surgical resections.

Conclusions

Lipoid pneumonia remains a diagnostically and therapeutically challenging condition. Recent developments have improved recognition through advanced imaging techniques and increased awareness of emerging etiologies including vaping-related exposures. However, the evidence base for diagnosis and treatment remains severely limited, consisting almost entirely of case reports and small retrospective series rather than prospective trials or controlled studies.

The natural history is often unfavorable, with many patients experiencing progression despite elimination of exposure and supportive care. Corticosteroids and therapeutic lavage are commonly employed but lack rigorous evaluation of efficacy. Critical knowledge gaps exist regarding optimal diagnostic approaches, therapeutic interventions, prognostic factors, and pathophysiological mechanisms.

Future research priorities include prospective studies to characterize natural history and identify predictors of progression, controlled trials of therapeutic interventions including corticosteroids and whole lung lavage, better definition of the role of BAL in diagnosis, and investigation of molecular mechanisms underlying chronic inflammation and fibrosis. Until such studies are conducted, management will continue to rely on expert consensus and extrapolation from limited case series, with uncertain outcomes for many patients.

This narrative review addresses the reviewer's suggestions by:

1. Explicitly emphasizing the limited evidence base: throughout the manuscript, particularly in the Treatment and Management section and the Knowledge Gaps section, the review clearly states that only case reports, case series, and small retrospective studies are available, with no randomized controlled trials or prospective cohort studies to guide clinical decision-making.[1,3,7,8,19]

2. Including a comprehensive diagnostic algorithm table: Table 1 provides a structured flow-chart approach to diagnosis, incorporating clinical history, imaging, bronchoscopy, histopathology, and integration of findings, along with the limitations of each diagnostic step.[1-3,7-9]

The review synthesizes evidence from the largest available studies (Mayo Clinic series of 34 patients and French multicenter study of 44 patients) while acknowledging that even these represent limited evidence.[1,9] The 2025 ERS/ATS guidelines are prominently featured, highlighting the specific research priorities identified by international expert consensus.[3] The review also incorporates recent developments including EVALI and novel etiologies while maintaining appropriate skepticism about treatment efficacy given the absence of controlled trials.[5,6,19,26,28]

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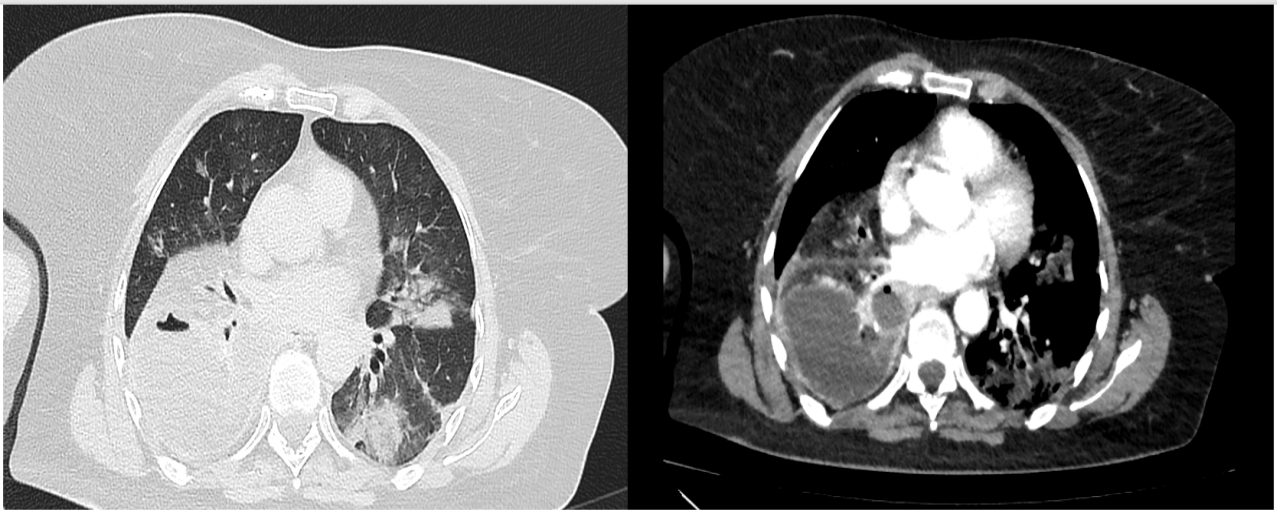

Figure 1. Lipidic pneumonia in the middle lobe of the lung.

Table 1. Diagnostic algorithm for lipoid pneumonia.

Diagnostic step	Key elements	Details	Limitations
Clinical suspicion	History of lipid exposure	Mineral oil use (laxatives), aspiration risk factors (gastroesophageal reflux, neurological disorders), occupational exposures, vaping oils, lipid-containing medications/supplements	Exposure history often not obtained until after diagnosis; patients may not recognize relevant exposures
Imaging	Chest CT with attenuation measurement	Consolidation or ground-glass opacities with fat attenuation (-30 to -150 HU); lower lobe predominance; "crazy paving" pattern; may show FDG-PET avidity mimicking malignancy	Fat attenuation present in only 41-71% of cases; findings nonspecific and may mimic malignancy, infection, or other interstitial lung diseases
Bronchoscopy	Bronchoalveolar lavage	Lipid-laden macrophages; milky appearance of BAL fluid; Oil Red O staining positive	Role in diagnosis not fully defined; not always required; findings not specific for lipoid pneumonia
Histopathology	Lung biopsy (if needed)	Foamy macrophages in alveoli; lipid vacuoles; variable-sized lipid droplets with giant cells (exogenous); may show interstitial fibrosis	Often performed to exclude malignancy when diagnosis not initially suspected; invasive procedure
Confirmation	Integration of findings	Combination of exposure history + characteristic imaging ± lipid-laden macrophages on BAL/biopsy in absence of endogenous causes	Remains a diagnosis of exclusion; no pathognomonic test available

CT, computed tomography; HU, Hounsfield units; BAL, bronchoalveolar lavage; FDG-PET, fluorodeoxyglucose positron emission tomography.