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EVALI: Current Understanding of Its Pathophysiology, Diagnosis, and Management

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Abstract

Background: E-cigarette, or Vaping, product use-Associated Lung Injury (EVALI) emerged in 2019 as a novel clinical syndrome, posing a significant public health challenge linked to the inhalation of aerosols from e-cigarette or vaping products, particularly those containing tetrahydrocannabinol (THC) and vitamin E acetate (VEA).

Aim: This narrative review synthesizes the current understanding of EVALI's pathophysiology, diagnostic approach, and management principles, drawing upon evidence published from 2017 to 2024.

Material and Methods: We conducted a comprehensive narrative synthesis of literature from 2017–2024, focusing on etiological studies, clinical case series, public health reports, and reviews detailing the mechanisms, clinical presentation, diagnostic criteria, and therapeutic outcomes of EVALI.

Results: The pathophysiology involves a complex interplay of direct chemical pneumonitis, surfactant dysfunction, and acute lung injury patterns triggered primarily by VEA. Clinical presentation includes respiratory, gastrointestinal, and constitutional symptoms. Diagnosis relies on a meticulous history of vaping, consistent radiological findings (typically bilateral ground-glass opacities on CT), and exclusion of infection. Management is primarily supportive, with systemic glucocorticoids showing efficacy in reducing inflammation and improving outcomes in moderate to severe cases.

Conclusions: EVALI represents a severe form of inhalational lung injury directly attributable to toxic substances like VEA in vaping aerosols. While the peak incidence has subsided, it underscores the ongoing risks of unregulated vaping products and highlights critical gaps regarding long-term sequelae. Continued surveillance, public health education, and further research are imperative.

Keywords: EVALI, e-cigarette, vitamin E acetate, vaping-associated lung injury, acute lung injury, glucocorticoids

1. Introduction

The rapid proliferation of electronic cigarettes (e-cigarettes) or vaping product use, particularly among adolescents and young adults, was followed by an unexpected and severe public health crisis. In the summer of 2019, clusters of patients presenting with acute respiratory failure with hypoxemia and diffuse lung infiltrates, all reporting a history of recent vaping, were identified across the United States [1, 2]. This novel syndrome was termed E-cigarette, or Vaping, product

use-Associated Lung Injury (EVALI). By February 2020, the Centers for Disease Control and Prevention (CDC) had reported over 2,800 hospitalized cases and 68 confirmed deaths attributable to EVALI [3, 4]. The epidemiological investigation swiftly identified a strong association with vaping products containing tetrahydrocannabinol (THC), and laboratory analyses pinpointed vitamin E acetate (VEA), used as a thickening agent in THC-based oils, as a primary toxicant of concern [5, 6]. EVALI exemplifies the potential for severe pulmonary toxicity from inhaled chemicals in unregulated vaping products. This review aims to consolidate the current evidence on EVALI's pathophysiology, clinical presentation, diagnostic strategy, and therapeutic management, providing a clinically oriented resource for healthcare professionals.

2. Materials and Methods

2.1. Review Methodology and Literature Search

This narrative review employed a systematic approach to literature identification. Databases including PubMed, Scopus, and Web of Science were searched for articles published between 2017 and 2024. Search terms included: "EVALI", "e-cigarette or vaping product use-associated lung injury", "vitamin E acetate", "vaping", "electronic cigarette", and "acute lung injury". Public health reports from the CDC and WHO were also reviewed.

2.2. Inclusion Criteria and Data Synthesis

Studies, case series, review articles, and official reports focusing on the etiology, pathophysiology, clinical features, diagnosis, management, and outcomes of EVALI were included. Data were extracted, critically analyzed, and synthesized thematically to present a coherent overview of the current evidence base.

2.3. AI Utilization

AI was utilized for two specific purposes in this research: 1) Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. 2) Initial organization and synthesis of thematic points from the collected literature. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final selection of literature, critical interpretation of evidence, synthesis of conclusions, and clinical recommendations were determined by the authors. The AI tools served primarily to enhance efficiency in data organization and linguistic refinement.

3. Pathophysiology

3.1. Etiology

The etiology of EVALI is directly linked to the inhalation of aerosolized substances from e-cigarettes or vaping devices. While various products were initially considered, national surveillance data revealed that a significant majority of patients reported using THC-containing products. Specifically, among 573 patients with available data, 76% reported using THC-containing products in the 90 days prior to symptom onset [2]. Subsequent targeted toxicological analysis has provided a mechanistic explanation for this association, identifying that vitamin E acetate-a common additive in THC-based vaping oils-can undergo pyrolysis during vaping to produce highly toxic ketene gas, among other harmful compounds [7]. This evidence strongly implicates THC-containing products, particularly those with certain additives, as a primary risk factor in the EVALI outbreak. The breakthrough in understanding the causal agent came from bronchoalveolar lavage (BAL) fluid analysis. In a landmark study, VEA was detected in the BAL fluid of 94% of EVALI patients from 16 states but in none of the healthy controls, providing direct evidence of its role as a key causative agent [5]. VEA, or tocopheryl acetate, is a lipid compound that is harmless when ingested or applied topically but is believed to be profoundly disruptive when inhaled, due to its persistence and interference with pulmonary surfactant [8, 9, 10]. When aerosolized by the heating element in vaping devices, VEA can undergo thermal decomposition (pyrolysis). This process generates ketene, a highly reactive and potent pulmonary toxic gas. Inhalation of this pyrolytic product causes direct chemical injury to the lung epithelium [7]. It is crucial to note that VEA is not the sole culprit; other phytochemicals, solvents (like propylene glycol and vegetable glycerin), flavoring agents (e.g., diacetyl), and contaminants in both THC and nicotine vaping products may contribute to or cause lung injury, suggesting a multifactorial etiology [11, 12].

3.2. Mechanisms of Lung Injury

The precise pathophysiology of EVALI remains to be fully elucidated, but clinical and imaging profiles commonly reflect patterns of acute lung injury, including lipoid pneumonia and chemical pneumonitis [9, 13]. The inhalation of lipid oils is a key suspected mechanism, with findings of lipid-laden macrophages on bronchoalveolar lavage supporting this [6, 14]. This can lead to impaired gas exchange, hypoxemia, and imaging findings consistent with diffuse alveolar damage, sharing features with acute respiratory distress syndrome (ARDS) [13].

Furthermore, the thermal degradation of vaping liquids can generate reactive carbonyl compounds (e.g., formaldehyde, acrolein) and free radicals, provoking oxidative stress and direct injury to the respiratory epithelium [15, 16]. This damage triggers a robust innate immune response, characterized by an influx of neutrophils and macrophages, and the release of pro-inflammatory cytokines, culminating in diffuse alveolar damage (DAD) and organizing pneumonia patterns [8, 17].

3.3. Histopathological Patterns

Lung biopsy and autopsy studies have revealed a spectrum of histopathological findings in EVALI, often overlapping. The most common patterns are organizing pneumonia and diffuse alveolar damage (DAD), often with associated fibrin deposition, features consistent with acute lung injury [8, 12]. Lipid laden macrophages (LLMs) are commonly observed in bronchoalveolar lavage (BAL) specimens from patients with EVALI, reflecting exposure to lipid containing aerosols, but this finding is nonspecific and can occur in other pulmonary conditions [6, 14]. Some cases exhibit patterns of lipoid pneumonia, where exogenous lipids incite a giant cell reaction and chronic inflammation [8]. The spectrum of lung injury includes organizing pneumonia as the most observed pattern, with rare instances of acute eosinophilic pneumonia also described, reflecting heterogeneous host responses to inhalational injury [9, 13].

4. Clinical Presentation

4.1. Symptoms and Signs

EVALI presents with a constellation of respiratory, gastrointestinal (GI), and systemic symptoms, typically developing over days to weeks. The nearly universal symptoms are shortness of breath (85%), cough (85%), and pleuritic chest pain (52%) [15]. GI symptoms are remarkably prevalent, occurring in 77–92% of patients, and include nausea, vomiting, abdominal pain, and diarrhea, often preceding respiratory symptoms [18]. Constitutional symptoms such as fever (66%), chills, and weight loss are also common. Tachycardia and tachypnea are frequent vital sign abnormalities, and on auscultation, crackles may be heard, though the exam can be deceptively normal early in the disease course [17].

4.2. Patient Demographics

During the 2019 outbreak, the median age of hospitalized EVALI patients was 24 years, with 78% of patients under 35 years old [2, 4]. There was a male predominance (66%). Notably, 96% of patients required hospitalization, 55% required intensive care unit (ICU) admission, and 26% required mechanical ventilation, underscoring the severity of the syndrome [3,4]. While the outbreak peaked in 2019, sporadic cases continue to be reported, particularly associated with the use of informally sourced or modified vaping products [4].

4.3. Severity Spectrum

The clinical severity of EVALI ranges from mild, self-limited illness to fulminant respiratory failure and death. Mild cases may present with cough and mild dyspnea without hypoxemia. Severe cases progress rapidly to hypoxemic respiratory failure meeting criteria for ARDS, often requiring high-flow oxygen, non-invasive ventilation, or invasive mechanical ventilation [17]. While respiratory symptoms define the syndrome, severe cases can progress to systemic complications and multi-organ dysfunction, likely secondary to profound hypoxemia and systemic inflammation, as noted in clinical case reports, although this was not a primary focus of the CDC's initial outbreak surveillance [2,3].

5. Diagnostic Evaluation

5.1. Case Definition (CDC / WHO)

The CDC established a surveillance case definition for EVALI, crucial for standardization. A confirmed case requires: 1) use of an e-cigarette or vaping product within 90 days of symptom onset; 2) pulmonary infiltrates on imaging; 3) absence of pulmonary infection on initial workup; and 4) no evidence of alternative plausible diagnoses (e.g., cardiac, rheumatologic, neoplastic) [1, 2]. The World Health Organization (WHO) issued a similar case definition, emphasizing the history of vaping and exclusion of infection [10].

5.2. Diagnostic Workup

A high index of suspicion is paramount. The single most important step is obtaining a detailed, non-judgmental history of inhaled substance use, including e-cigarettes, vaping devices, and dab pens, specifically probing for THC and nicotine product use, sourcing (commercial vs. informal), and device modifications [2,10]. Initial laboratory findings are nonspecific but often

reveal leukocytosis with neutrophilia, elevated inflammatory markers (C-reactive protein, erythrocyte sedimentation rate), and elevated liver transaminases [15, 19].

5.3. Imaging

Chest radiography is insensitive but often shows bilateral opacities. High-resolution computed tomography (HRCT) of the chest is the imaging modality of choice and is often abnormal even when the chest X-ray is indeterminate. The most characteristic HRCT findings are bilateral, symmetric ground-glass opacities, often with subpleural sparing, and consolidations in a basilar or diffuse distribution [20, 21]. Other patterns include smooth interlobular septal thickening ("crazy-paving") and, less commonly, centrilobular nodules [21].

5.4. Rule-Out Diagnostics

A rigorous exclusion of infectious etiologies is mandatory, as EVALI can mimic viral and bacterial pneumonia. Testing should include respiratory viral panels, sputum cultures, blood cultures, and urine antigen testing for *Streptococcus pneumoniae* and *Legionella*. For example, in one reported fatal case, the diagnostic workup included extensive microbiological testing-all negative-to exclude infection [19]. In the context of the COVID-19 pandemic, SARS-CoV-2 PCR testing is essential, given significant clinical overlap. Testing for other causes of acute lung injury, such as cardiac echocardiography and screening for autoimmune serologies, should be guided by the clinical context.

5.5. Bronchoscopy and BAL

Bronchoscopy with BAL is not required for diagnosis but can be invaluable in atypical or severe cases. The classic finding is an excess of lipid-laden macrophages on Oil Red O staining [6, 14]. BAL fluid typically shows a neutrophilic or lymphocytic predominance and should be sent for comprehensive microbiological studies, including bacterial, fungal, and mycobacterial cultures, to rule out infection [10,13]. In research settings, BAL fluid can be analyzed for VEA [5].

6. Management

6.1. General Management Principles

Hospitalization is recommended for most patients with confirmed or suspected EVALI due to the risk of rapid deterioration. Management is primarily supportive, focusing on ensuring adequate oxygenation and ventilation. Supplemental oxygen is administered to maintain oxygen saturation >90%. Conservative fluid management is advised, as these patients are at risk for developing ARDS and fluid overload can exacerbate pulmonary edema [17]. Critical interventions include the complete cessation of all vaping and e-cigarette use and close monitoring for clinical progression [9].

6.2. Glucocorticoid Therapy

Systemic glucocorticoids have emerged as a mainstay of pharmacotherapy for moderate to severe EVALI. Observational data consistently show that steroid therapy is associated with more rapid clinical improvement, reduced oxygen requirements, and shorter hospital stays [5, 10, 18]. Methylprednisolone 0.5–1 mg/kg/day (or equivalent) is typically initiated, with a rapid taper over 1–2 weeks for outpatients or longer courses for critically ill patients. The rationale is to dampen the intense inflammatory response and alveolar injury. Initiation should be deferred until active infection is confidently ruled out.

6.3. Antibiotics and Antivirals

Given the frequent presentation with fever and leukocytosis, broad-spectrum empiric antibiotics for community-acquired pneumonia are often initiated upon admission, consistent with standard management of severe respiratory illness. This approach was followed in a reported case, where antibiotics were started empirically but were promptly discontinued after a comprehensive infectious workup returned negative, to avoid complications [19]. Antivirals may be considered during seasonal influenza activity until testing is negative.

6.4. Severe and Critical Cases

Patients progressing to hypoxemic respiratory failure should be managed according to evidence-based ARDS protocols, including lung-protective low tidal volume ventilation, appropriate use of positive end-expiratory pressure (PEEP), and prone positioning for severe ARDS [17]. Case reports have documented the use of extracorporeal membrane oxygenation

(ECMO) as salvage therapy for refractory respiratory failure in fatal EVALI, despite ultimate patient death from complications [19].

6.5. Outpatient Management

Selected patients with mild disease, no hypoxemia at rest, reliable social support, and access to close follow-up may be managed as outpatients. Treatment includes a short course of oral steroids, strict vaping cessation, and explicit instructions to return for worsening symptoms [2].

7. Outcomes and Prognosis

Most patients with EVALI recover with supportive care and steroids. Early reports indicated that over 90% of hospitalized patients were discharged [3,15], with a median hospital stay of 6 days [15]. However, a subset of survivors reports persistent respiratory symptoms, functional limitations, and radiographic abnormalities months after discharge, raising concerns for potential chronic interstitial lung disease or obstructive defects [22]. During the 2019–2020 EVALI outbreak in the United States, CDC surveillance data indicate that a small proportion of patients died, with crude mortality estimates of roughly 2–3% based on total reported cases and deaths [1, 4]. Analysis of deaths in earlier CDC data showed that patients who died tended to be older than those who survived [4]. Long-term prospective studies are needed to fully characterize the prognosis.

8. Public Health Implications

The EVALI outbreak highlighted the significant regulatory challenges and public health dangers posed by the rapidly evolving and often unregulated nicotine and cannabis vaping markets [9]. Laboratory findings that identified vitamin E acetate (VEA) in THC-containing products were critical in informing the outbreak response, leading to targeted public health recommendations [3, 5]. Data showed that most THC-containing products linked to EVALI were acquired from informal sources, underscoring the role of the illicit market [3].

However, the persistence of this opaque and fast-moving informal market and the potential for the introduction of new toxicants, including synthetic cannabinoids and novel solvents, necessitate ongoing chemical surveillance of vaping products [9]. From a public health perspective, the outbreak delivered a powerful message, demonstrating to young people that vaping can result in serious and immediate health consequences, a warning that goes beyond those about long-term nicotine addiction and represents a critical opportunity for education [9].

9. Knowledge Gaps and Future Directions

Substantial knowledge gaps remain. The long-term cardiopulmonary sequelae of EVALI remain unknown, requiring further study [9]. The pathogenic roles of chemicals other than VEA, including potential flavorants and cutting agents found in illicit vaping products, require further toxicological investigation [11, 12]. The importance of device characteristics in aerosol toxicity generation warrants additional examination [17]. Biomarkers for EVALI diagnosis, severity assessment, and prognosis development represent critical unmet needs [21]. Future research priorities include longitudinal cohort studies of EVALI survivors [10, 15, 21], development of advanced in vitro and animal models of vaping injury [7, 11], and continuous chemical surveillance of vaping products [9].

10. Conclusion

EVALI represents a distinct and severe form of acute lung injury directly attributable to the inhalation of toxic substances in vaping aerosols, with VEA in THC products playing a major causative role [5, 7]. Its pathophysiology involves direct epithelial injury, surfactant dysfunction, and a pronounced inflammatory response [8, 16, 17], with additional injury potentially caused by toxic pyrolysis products like ketene [7]. Diagnosis hinges on a meticulous vaping history, compatible imaging, and rigorous exclusion of infection [2]. Management is centered on supportive care, vaping cessation, and the timely use of systemic glucocorticoids [17]. While the dramatic 2019 outbreak has receded, EVALI remains a persistent risk, underscoring the inherent dangers of inhaling unregulated chemical mixtures. It serves as a critical case study in the unintended consequences of new drug delivery technologies and reinforces the imperative for sustained clinical vigilance, public health regulation, and scientific inquiry.

Disclosure

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