

Acute Respiratory Distress Syndrome Induced by E-Cigarette or Vaping Product Use Associated Lung Injury

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Article Info

Article history:

Received December 01, 2023

Revised December 27, 2023

Accepted December 30, 2023

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ABSTRACT

In 2019, a respiratory infection outbreak in individuals with a history of vaping was discovered, this condition was eventually termed e-cigarette or vaping product use-associated lung injury (EVALI) after it was linked to lung damage during imaging and histopathologic examination. The exact mechanism behind the lung injury seen in EVALI is unknown. Several studies show that EVALI has been associated with the incidence of acute respiratory distress syndrome. Nowadays, many people use Vapes/e-cigarettes, it is critical for clinicians to understand the hazards of using e-cigarettes, which can induce EVALI to ARDS. We used a number of research resources, including ScienceDirect, Google Scholar, PubMed, and Wiley, to search, select, and choose papers about the use of electronic cigarettes or vaping devices associated to lung injury trigger Acute Respiratory Distress syndrome. The literature search yielded 330 articles, of which 21 met the selection criteria based on the title and abstract. EVALI is still a clinical diagnosis that should be considered as an exclusion because the symptoms, physical examination, serologic, radiologic, and bronchoscopy results are not specific to the illness alone. ARDS and its sequelae may be more likely to occur in patients with EVALI who also have several chronic illnesses. For individuals who use excessively and consistently over weeks or months, there is strong correlative evidence suggesting a higher risk of VAPI and potential consequences from ARDS in the absence of prompt and aggressive therapy.

Keywords: Vape, Electronic Cigarette, Lung Injury, Acute Respiratory, Distress Syndrome"

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1. INTRODUCTION

About a decade ago, "modern" vaping with battery-powered electronic devices started, and it soon grew into a multibillion-dollar industry that serves an estimated 50 million users worldwide. Initially designed as an alternative to traditional cigarette smoking, vaping today appeals to a varied demographic, including a large number of young people who have never smoked. Vaping has evolved from humble beginnings as an alternative to traditional cigarette use into a complex, contentious, and intractable societal issue [1]. Vaping is a divisive topic, with strong arguments on both sides of the discussion [2]. Fundamentally, the problem stems from the rapid transformation of vaping into a recreational gadget for new users, particularly young ones, rather than an electronic nicotine delivery system for established smokers [3].

In 2019, a respiratory infection outbreak in individuals with a history of vaping was discovered by doctors, the U.S. Food and Drug Administration, the U.S. Centers for Disease Control and Prevention, or CDC, and state and local health authorities [4]. This condition was eventually termed e-cigarette or vaping product use-associated lung injury (EVALI) after it was linked to lung damage during imaging and histopathologic examination. Clinically speaking, EVALI typically presents as a rather acute sickness that resembles a viral infection. Although testing shows no harmful pathogen, these patients are frequently believed to have an infection due to their vague symptoms [5].

Several studies show that EVALI has been associated with the incidence of acute respiratory distress syndrome (ARDS). Most patients who die from EVALI are diagnosed with ARDS [6]. There have only been a few studies that look at the incidence of ARDS caused by EVALI [7]. Nowadays, many people use Vape/e-cigarettes, it is critical for clinicians to understand the hazards of using e-cigarettes, which can induce EVALI to ARDS [8]

2. METHODS

The author used a number of research resources, including ScienceDirect, Google Scholar, PubMed, and Wiley, to search, select, and choose papers about the use of electronic cigarettes or vaping devices associated to lung injury trigger Acute Respiratory Distress syndrome. The terms "Vape," "Electronic Cigarette," "Lung Injury," and "Acute Respiratory Distress Syndrome" were used to conduct research searches [9]. The literature search yielded 330 articles, of which 21 met the selection criteria based on the title and abstract. We prioritized research published within the last five years [10]. Writing begins with literature selection through title and abstract, followed by reviewing the contents of each piece of literature that meets the criteria and is followed by discussion between authors. Studies that are not fully accessible were excluded [11]. The final results will be obtained from the research that will be used in this literature observation [12]

3. RESULTS AND DISCUSSION

From 3 databases, 23,080 studies were identified. After adjusting the filter option on each database, 4,752 studies were chosen for further screening. After screening titles and abstracts, 35 studies were excluded. A number of studies were inaccessible, and the final number of studies included in this study were [13].

The respiratory effect of E-Cigarrates

Due to the fragile nature of the lungs, even slight inflammation might have negative effects. Lung homogenates, bronchoalveolar lavage analysis, and histopathological evaluation are three methods for determining lung inflammation. There have been reports of more severe bronchial injuries, erythematous and irritated airway mucosa in healthy e-cigarette users. Both bronchial epithelia and airway secretions exhibit higher amounts of MUC5AC mucin, nonetheless, it is noteworthy that a significant proportion of e-cigarette users were also former smokers. Mucins are a confirmed biomarker of damage because they are negatively correlated with the deterioration in lung function in people with Chronic Obstruction Pulmonary Disease (COPD) and are a biomarker of chronic bronchitis [12].

Setting aside more research to investigate potential subclinical alveolar injury from e-cigarettes would be crucial, since smokers are more likely to suffer from acute respiratory distress syndrome and potentially fatal alveolar injury. Damage to the integrity of the alveoli or macrophage activation sets off inflammatory reactions that can build up in the lung parenchyma in a matter of hours, resulting in significant harm and secondary sequelae that may be fatal [14]. The complex network of larger vessels and capillaries within the lung is adversely impacted in severe cases by the progressive loss of alveolar structure and density, cell death, tissue structural deterioration, and fluid accumulation [15]. This results in increased vascular pressure, pulmonary failure, and adverse remodeling. Numbers of case reports connects the use of e-cigarettes to severe inflammatory diseases that affect the alveoli and small airways, such as hypersensitivity pneumonitis, lipoid pneumonia, eosinophilic pneumonia, diffuse alveolar hemorrhage, organizing pneumonia, and respiratory bronchiolitis associated interstitial lung disease [16].

EVALI and ARDS

All cases of lung disease linked to vaping have been grouped together under the term vaping-associated pulmonary illness (VAPI). On the other hand, different pathomechanisms might be involved in the recent surge in relatively acute VAPI presentations. These cases are currently being referred to as e-cigarette, or vaping, product use-associated lung injury (EVALI). Vitamin E acetate has recently been found to be a chemical in EVALI that warrants concern [17].

An important identified risk factor for the development of EVALI is the use of e-cigarettes or similar devices. The use of e-cigarettes or similar devices is a significant risk factor for the development of EVALI that has been found. Nitrosamines, aldehydes, metals unique to tobacco, volatile organic compounds, phenolic compounds, hydrocarbons generated by polycyclics, tobacco alkaloids, flavorings, and medications are all possible ingredients in e-cigarettes and aerosol products. For example, there is strong evidence that certain e-cigarettes contain significant amounts of propylene glycol, vitamin E acetate, and metals including lead and arsenic. While oil is frequently used as a diluent in THC products, propylene glycol and glycerol are frequently employed as diluents in nicotine-containing e-cigarette goods. It has been demonstrated that inhaling e-cigarette vapor that contains glycerol and propylene glycol impairs immunological function and lipid homeostasis in mouse models [18].

In addition to serving as a fundamental component in e-liquids, propylene glycol is a typical chemical that is used to make polyester and as an antifreeze/de-icer. Acute toxicity to the kidneys and central nervous system can result from intravenous propylene glycol. It has been previously demonstrated that propylene glycol inhibits the activity of corneal Na⁺/K⁺ ATPase and renal glucose transport. When added to food in the recommended

proportions, propylene glycol and vegetable glycerin are categorized as "generally recognized as safe." Propylene glycol short-term occupational exposures caused discomfort and either minor or no objective effects on pulmonary function, these findings show that propylene glycol may operate as a sensory irritant. However, this label does not relate to inhalational safety. TRPV1 and TRPA1, two irritating receptors expressed in sensory nerves that innervate the airways, were activated by propylene glycol. In asthma models, these receptors exacerbate airway hyperreactivity and asthmatic inflammation. Chronic vapers' lungs had higher quantities of MUC5AC protein. After vaping in primary airway epithelia, propylene glycol/vegetable glycerin rather than nicotine increased the expression of mucin [19].

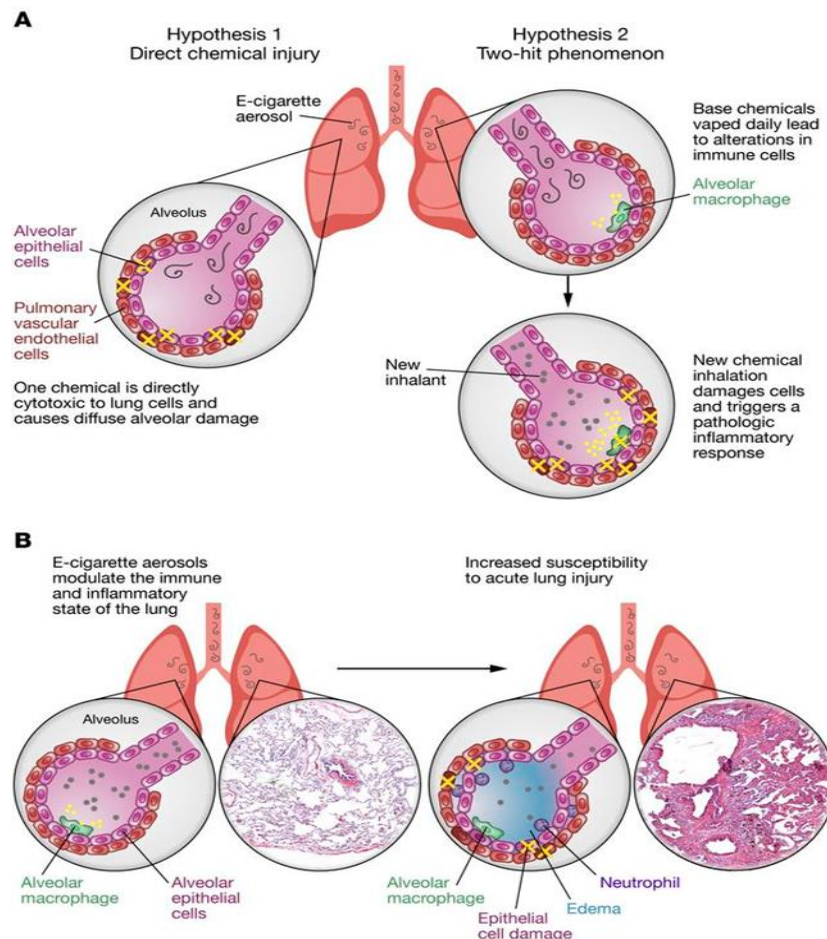


Figure 1. Mechanisms underlying lung injury in EVALI

The exact mechanism behind the lung injury seen in EVALI is unknown. There are several hypothesis explaining the mechanisms underlying lung injury in EVALI. According to the first hypothesis, direct chemical injury, a single chemical breathed from e-cigarette aerosols causes damage to lung cells. Necrosis of the endothelium and epithelium cells results from this cell injury [20]. As an alternative hypothesis, the lungs' inflammatory state is altered by inhaling common e-cigarette aerosols such as propylene glycol, glycerin, nicotine, or THC. This includes transforming alveolar macrophages into a proinflammatory phenotype through the release of inflammatory cytokines, which leads to a pathologic inflammatory response upon a second hit. Aerosols from e-cigarettes are known to cause long-term, everyday changes to the immunological status of the lungs. It is possible that vitamin E directly damages lung cells, causing necrosis, activation and recruitment of neutrophils, which causes collateral damage, and noncardiac pulmonary edema [21].

Since the symptoms, physical examination, serologic, radiologic, and bronchoscopy results are not unique to the disease, EVALI is still a clinical diagnosis that is best treated as an exclusion. Although the identification of EVALI patients requires clinical judgment on the part of healthcare personnel, this is made more challenging by the diverse appearances of EVALI. Furthermore, co-infection with bacteria or viruses might happen. It is especially crucial to recognize this during flu season since it can manifest similarly or even simultaneously with EVALI. The CDC has developed case criteria of "confirmed EVALI" cases to aid in the identification of the illness and aid the physician in diagnosing EVALI. The criteria used to define EVALI cases can be seen in table 1, which shows the diagnostic criteria for confirmed and probable EVALI patients [22].

Table 1. CDC Surveillance Case Definitions for E-Cigarette-Related Severe Pulmonary Disease

Case Classification	CDC Criteria	Additional investigations to consider:
Confirmed	Using an e-cigarette ("vaping") or dabbing ^b during the 90 days before symptom onset (and)	Consider toxicology to assess for THC or other inhalational agents
	Pulmonary infiltrate, such as opacities on plain film chest radiograph or ground-glass opacities on chest computed tomography (and)	Consider CT scan for increased sensitivity
	Absence of pulmonary infection on initial work-up: Minimum criteria include negative respiratory viral panel, influenza polymerase chain reaction or rapid test if local epidemiology supports testing. All other clinically indicated respiratory infectious disease testing (e.g., urine antigen for <i>Streptococcus pneumoniae</i> and <i>Legionella</i> , sputum culture if productive cough, bronchoalveolar lavage culture if done, blood culture, human immunodeficiency virus-related opportunistic respiratory infections if appropriate) must be negative (and)	HIV testing SARS-CoV-2 testing Procalcitonin CBC with differential
	No evidence in medical record of alternative plausible diagnoses (e.g., cardiac, rheumatologic, or neoplastic process).	Echocardiography Differential on CBC ANA RF ANCA ESR CRP
Probable	Using an e-cigarette ("vaping") or dabbing ^b in 90 days before symptom onset (and)	Consider toxicology to assess for THC or other inhalational agents
	Pulmonary infiltrate, such as opacities on plain film chest radiograph or ground-glass opacities on chest computed tomography (and)	Consider CT scan for increased sensitivity
	Infection identified via culture or polymerase chain reaction, but clinical team ^c believes this is not the sole cause of the underlying respiratory disease process OR minimum criteria to rule out pulmonary infection not met (testing not performed) and clinical team ^c believes this is not the sole cause of the underlying respiratory disease process (and)	HIV testing SARS-CoV-2 testing Procalcitonin CBC with differential
	No evidence in medical record of alternative plausible diagnoses (e.g., cardiac, rheumatologic, or neoplastic process).	Echocardiography Differential on CBC ANA RF ANCA ESR CRP

^aThese surveillance case definitions are meant for surveillance and not clinical diagnosis; they are subject to change and will be updated as additional information becomes available if needed

^bUsing an electronic device (e.g., electronic nicotine delivery system (ENDS), electronic cigarette (e-cigarette), vaporizer, vape(s), vape pen, dab pen, or other device) or dabbing to inhale substances (e.g., nicotine, marijuana, tetrahydrocannabinol, tetrahydrocannabinol concentrates, cannabinoids, synthetic cannabinoids, flavorings, or other substances)

^cClinical team caring for the patient

Complete blood count, comprehensive metabolic panel, C-reactive protein, erythrocyte sedimentation rate, and respiratory pathogen panel with influenza are all diagnostic tests to consider. Sputum Gram stain and culture, as well as testing for *Legionella*, endemic mycoses, and other opportunistic infections, may be performed as part of the infectious workup. Urine toxicology testing, including THC, should also be acquired [23]. Bilateral infiltrates or normal findings on a chest radiograph may be indicative of EVALI. Although chest computed tomography can detect bilateral ground-glass opacities, imaging results vary. If the chest radiograph is abnormal, computed tomography isn't needed to rule out EVALI.12 Bronchoscopy can also be used to collect pulmonary samples. However, depending on the patient's condition, it may not be possible right away, which could lead to additional deterioration and intubation. Bronchoscopy is likely to affect management in patients with aberrant radiological findings, such as cavitation or big nodules, as well as those who have recently been exposed to uncommon infections, are immunocompromised, or are already intubated [24].

ARDS is a potentially fatal illness that affects critically sick patients and is defined by acute onset, poor oxygenation, and pulmonary infiltrates. It is an acute, diffuse, inflammatory form of lung injury. The condition is linked to widespread alveolar damage and capillary endothelial injury at the microscopic level. When there is no sign of cardiogenic pulmonary edema, acute respiratory distress syndrome (ARDS) is characterized by bilateral lung infiltrates and severe progressive hypoxemia that begins within seven days of the inciting event. The ratio of the patient's arterial blood oxygen content (PaO₂) to the inspired air oxygen content (FiO₂) is what defines ARDS. Less than 300 is the PaO₂/FiO₂ ratio for these patients. Patients often have variable degrees of pulmonary artery vasoconstriction after developing ARDS, and they may go on to develop pulmonary hypertension as a result. There are few effective therapy approaches to treat ARDS, and the illness has a significant fatality rate [25].

ARDS can be caused by a number of pathogenic conditions, both acute and chronic. To name a few possibilities, ARDS can be caused by sepsis, severe pneumonia, COVID-19 infection, or inhalation of harmful chemicals. Fluid collection in the alveoli impairs gas exchange and reduces blood oxygenation. The predominant sign of ARDS is severe shortness of breath, and the consequences can be catastrophic, with the chance of mortality increasing with age and severity of sickness. ARDS survivors may endure long-term lung damage [26]. The 2019 EVALI epidemic demonstrated the rapid and sometimes lethal impact of acute inhalation injury and subsequent inflammatory damage, but it is important to note that EVALI does not occur as a normal result of commercially

purchased vaping juices, but rather from "street vapes" primarily containing THC and vitamin E acetate.^{1,16} In fact, it's generally accepted that using vape devices in a typical manner won't cause an EVALI-type crisis in the immediate hours to days following inhalation. But there is a chance that long term use over a period of weeks or months could cause a more subtle type of harm that leads to VAPI. There is large correlative evidence for individuals who use heavily and frequently for weeks or months, suggesting an increased risk of VAPI and the potential for ARDS complications in the absence of prompt and aggressive intervention [27]. Individuals who have been exposed to vaping fumes in the last 90 days and have rapidly progressive symptoms of cough, chest pain, weight loss, fatigue, or dyspnea or have resting oxygen saturation of 95% or level of 88% with exercise or are below their abnormal baseline level are considered hypoxemic patients with EVALI. Patients who are hypoxemic are at the greatest risk of developing respiratory failure and require inpatient oximetry monitoring and prevention of additional exposure for at least the first 48 hours to detect, prevent, and manage progressive hypoxemia, as well as urgent intervention if they develop respiratory failure [28].

With developments in radiology, the utility of imaging in the early detection of vaping-induced lung injury is now being used and is traditionally associated with four patterns of lung illness on CT. Acute eosinophilic pneumonia, diffuse alveolar damage, organizing pneumonia, and lipoid pneumonia are the four patterns. Acute eosinophilic pneumonia is distinguished by areas of ground-glass opacification, interlobular septal thickening, and pleural effusions caused by fast eosinophilia within the lung parenchyma. Diffuse alveolar injury, commonly known as idiopathic acute interstitial pneumonia, is characterized by bilateral ground-glass opacification with late-phase honeycombing. The mechanism of injury that frequently leads in the clinical presentation of ARDS is diffuse alveolar destruction. Furthermore, organized pneumonia frequently manifests as migrating zones of localized subpleural and peribronchovascular opacification that resolve and recur on a random basis. Lipoid pneumonia develops when lipids accumulate in the alveoli and a low attenuating consolidation builds in the dependent lung parenchyma. The triggering event in all of the aforementioned kinds of acute lung injury is inflammatory, resulting in a type of pneumonitis that may be linked to vaping [29].

Based on a study by Werner et al, Acute respiratory distress syndrome and its sequelae may be more likely to occur in patients with EVALI who also have several chronic illnesses. When treating patients with severe cases of EVALI, clinicians should take evidence-based principles regarding the management of acute respiratory distress syndrome into consideration [30]. This is because traditional mechanical ventilation can exacerbate lung injury in patients with acute lung injury or acute respiratory distress syndrome, increasing the risk of nonpulmonary organ or system failure.²⁰ Werner et al, on his study state that obesity is a major risk for respiratory disease and can worsen the condition of patients with EVALI. Obesity can aggravate respiratory conditions, alter pulmonary physiologic parameters, and make it more difficult to get appropriate mechanical ventilation [31].

According to Akkanti's Case Report, the patient in their case had ARDS due to a complex influenza-related severe lung injury and rhabdomyolysis further complicated by vaping of a mixture of Vitamin-E and THC. Chronic exposure to vapor from electronic nicotine delivery systems (ENDS) or e-cigarettes did not result in pulmonary inflammation or emphysema in an experimental murine model, but it did produce aberrant phospholipids in alveolar macrophages and increased surfactant-associated phospholipids in the airways. Exposure to ENDS vapor reduced innate immunity against viral infections in local macrophages [32]. Furthermore, animals infected with influenza that were exposed to ENDS vapor showed increased lung inflammation and tissue damage. Alterations in innate immunity have also been linked to chronic vaping-related tracheobronchial and pulmonary damage in individuals [33].

4. CONCLUSION

The 2019 EVALI epidemic showed the swift and occasionally fatal effects of acute inhalation injury and the ensuing inflammatory damage. However, it is crucial to remember that EVALI is primarily caused by liquids that contain THC and vitamin E acetate rather than commercial vaping juices. There is substantial correlative evidence for people who use excessively and regularly for weeks or months, implying an elevated risk of VAPI and the possibility of ARDS complications in the absence of fast and aggressive care.

ACKNOWLEDGEMENTS

The author would like to thank for all the help and support provided until the article was completed and published.

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