Case Report

Access this article online
Quick Response Code:

Website:
https://eurasianjpulmonol.org
DOI:
10.14744/ejp.2026.01585

Vaping-triggered diffuse alveolar haemorrhage in a patient with advanced liver disease and severe coagulopathy: A causal inflection?

Benhur Joel Shadrach[1], Kunal Deokar[2], Lexmi Priya Raju[1], Vaibhav Patil[1], Priyanka Gaikwad[3], Jinish Doshi[4]

ORCID:
Benhur Joel Shadrach: 0000-0002-2314-2306
Kunal Deokar: 0000-0003-2603-1633
Lexmi Priya Raju: 0000-0002-2626-9644
Vaibhav Patil: 0000-0001-5796-9945
Priyanka Gaikwad: 0000-0002-8650-321X
Jinish Doshi: 0009-0003-7953-4506

Abstract:

Vaping-triggered diffuse alveolar haemorrhage (DAH) is rarely reported in the literature. We report the case of a 60-year-old man who presented to the emergency department with hemoptysis. He had a known history of chronic liver disease and presented with coagulopathy. He was an active smoker with a cumulative tobacco exposure of 12.5 pack-years and had been using nicotine-containing e-cigarettes for the past 10 months. Initial chest radiograph revealed bilateral infiltrates and high-resolution computed tomography (HRCT) of the thorax demonstrated bilateral, nodular, multifocal ground-glass opacities. Bronchoalveolar lavage (BAL) showed a progressively bloody return, consistent with diffuse alveolar haemorrhage (DAH). Coagulopathy was managed using targeted therapy guided by Rotational Thromboelastometry (ROTEM). Despite aggressive management, the patient succumbed to septic shock on the ninth day of admission. The pathogenesis of vaping-induced DAH may involve direct vascular injury from heated chemicals or an indirect mechanism via inflammatory cascades triggered by inhaled chemicals. In our patient with chronic liver disease complicated by coagulopathy, vaping triggered diffuse alveolar haemorrhage. This case highlights that vaping exposure may trigger DAH in patients with predisposing conditions such as severe coagulopathy due to chronic liver disease.

Keywords:

Bronchoscopy, diffuse alveolar haemorrhage, intensive care, respiratory failure, smoking, vaping

[1]Rela Hospital and Research institute, Chennai, Tamil Nadu, India, [2]All India Institute of Medical Sciences, Rajkot, Gujarat, India, [3]Pandit Deendayal Upadhyay Medical College (PDU), Rajkot, Gujarat, India, [4]Baptist Hospitals of Southeast Texas, Baeumont, Texas, USA

Address for correspondence:
Dr. Benhur Joel Shadrach, Rela Hospital and Research institute, Chennai, Tamil Nadu, India.
E-mail: benjoe6326@gmail.com

Received: 11-02-2026
Revised: 17-03-2026
Accepted: 24-03-2026
Published: 20-05-2026

Introduction

E-cigarettes were initially developed as a smoking cessation tool. However, their popularity increased rapidly among adolescents due to targeted marketing, the availability of flavored options, and the ease of access through online platforms. Over time, it became evident that ENDS (electronic nicotine delivery

How to cite this article: Shadrach BJ, Deokar K, Raju LP, Patil V, Gaikwad P, Doshi J. Vaping-triggered diffuse alveolar haemorrhage in a patient with advanced liver disease and severe coagulopathy: A causal inflection? Eurasian J Pulmonol 0000;00:1-5.

systems) are associated with various health-related hazards. There are several reported cases of EVALI (E cigarette or vaping associated lung injury).[1] However, vaping-triggered diffuse alveolar haemorrhage (DAH) is rarely reported in the literature.

Case Report

A 60-year-old male presented with an acute onset of cough and blood-tinged sputum. There was no history of fever, chest pain, orthopnea, or paroxysmal nocturnal dyspnea. There was no history suggestive of a connective tissue disease, such as joint pain, rash, myalgia, Raynaud's phenomenon, dry eye, or dry mouth. Travel and recreational drug use history were unremarkable. He had a 5-year history of chronic liver disease — non-alcoholic steatohepatitis (NASH). He was not taking any regular medications and did not report any drug intake in the past 2 months. He was an active smoker with a cumulative tobacco exposure of 12.5 pack-years and had been using nicotine containing e-cigarettes for the past 10 months.

On examination, he was conscious and oriented to time, place, and person. His vital signs were heart rate of 92 beats per minute, respiratory rate of 22 breaths per minute, and peripheral oxygen saturation (SpO_2) of 97 % on room air. Auscultation revealed fine end-inspiratory crepitations throughout both lung fields. The rest of the systemic examination was unremarkable.

Laboratory investigation revealed a hemoglobin level of 8.6 g/dl, a total leucocyte count of 10000/mm³, and a platelet count of 38000/mm³. Liver function tests showed a total bilirubin of 9.7 mg/dl and an international normalised ratio (INR) of 4.7. Serum creatinine was 1 mg/dl. The patient had a Child–Pugh score of 11 (Class C) and an estimated MELD 3.0 score of 34. Bedside 2D echocardiography showed a preserved left ventricular ejection fraction with no regional wall-motion abnormalities. The inferior vena cava (IVC) measured between 1.1 and 1.2 cm. Urinalysis did not reveal red blood cells.

The chest radiograph showed bilateral infiltrates [Fig. 1a]. The high-resolution computed tomography (HRCT) of the thorax showed bilateral, nodular, multifocal ground-glass opacities [Fig. 1b]. A nasopharyngeal swab for RT-PCR testing for H1N1, Influenza A, HMPV, and SARS COV-2 was negative. Sputum and blood cultures did not grow any pathogenic organisms.

Coagulopathy was addressed using ROTEM-guided targeted therapy. Despite correction of the coagulopathy, the patient's oxygen requirement progressively increased, and he eventually required 2 L/min of supplemental oxygen via nasal cannula to maintain an SpO_2 of 97%.

Autoimmune serologies, including antinuclear antibody, antineutrophil cytoplasmic antibodies (ANCA) — both cytoplasmic (c-ANCA) and perinuclear (p-ANCA) — were negative by indirect immunofluorescence. HIV screening using an immunochromatographic card test was non-reactive. A urinary drug screen for amphetamines, benzodiazepines, barbiturates, and cannabinoids was negative.

Flexible bronchoscopy, performed on day three, revealed blood clots at the carina [Fig. 1c]. Bronchoalveolar lavage (BAL) demonstrated a progressively bloody return, consistent with diffuse alveolar haemorrhage (DAH) [Fig. 1d]. Cytological examination of BAL fluid on hematoxylin and eosin-stained sections showed hemosiderin-laden macrophages [Fig. 1e]. Further confirmation of hemosiderin accumulation within the macrophages was achieved using Perls' Prussian blue stain, which specifically highlighted the hemosiderin pigment and demonstrated the presence of iron deposits [Fig. 1f]. BAL cultures were negative for infection. Despite correction of coagulopathy (INR less than 2), the patient's condition continued to deteriorate, with progressively increasing oxygen requirements, suggesting that correction of coagulation abnormalities alone did not fully reverse the pulmonary haemorrhage. A diagnosis of DAH secondary to coagulopathy in the context of chronic liver disease (non-alcoholic steatohepatitis, NASH) was made, with vaping identified as the likely trigger.

During the hospital stay, the patient developed a fever, septic shock, and acute kidney injury. Antibiotic therapy was escalated. Glucocorticoid therapy was planned after sepsis was controlled. The poor prognosis was explained to the patient's family. He required inotropic support to maintain blood pressure, and was started on continuous renal replacement therapy. The patient succumbed to septic shock on the ninth day of admission.

Discussion

E-cigarette or vaping product associated acute lung injury (EVALI) is a recognised complication of vaping.[1] Electronic nicotine delivery systems typically consist of

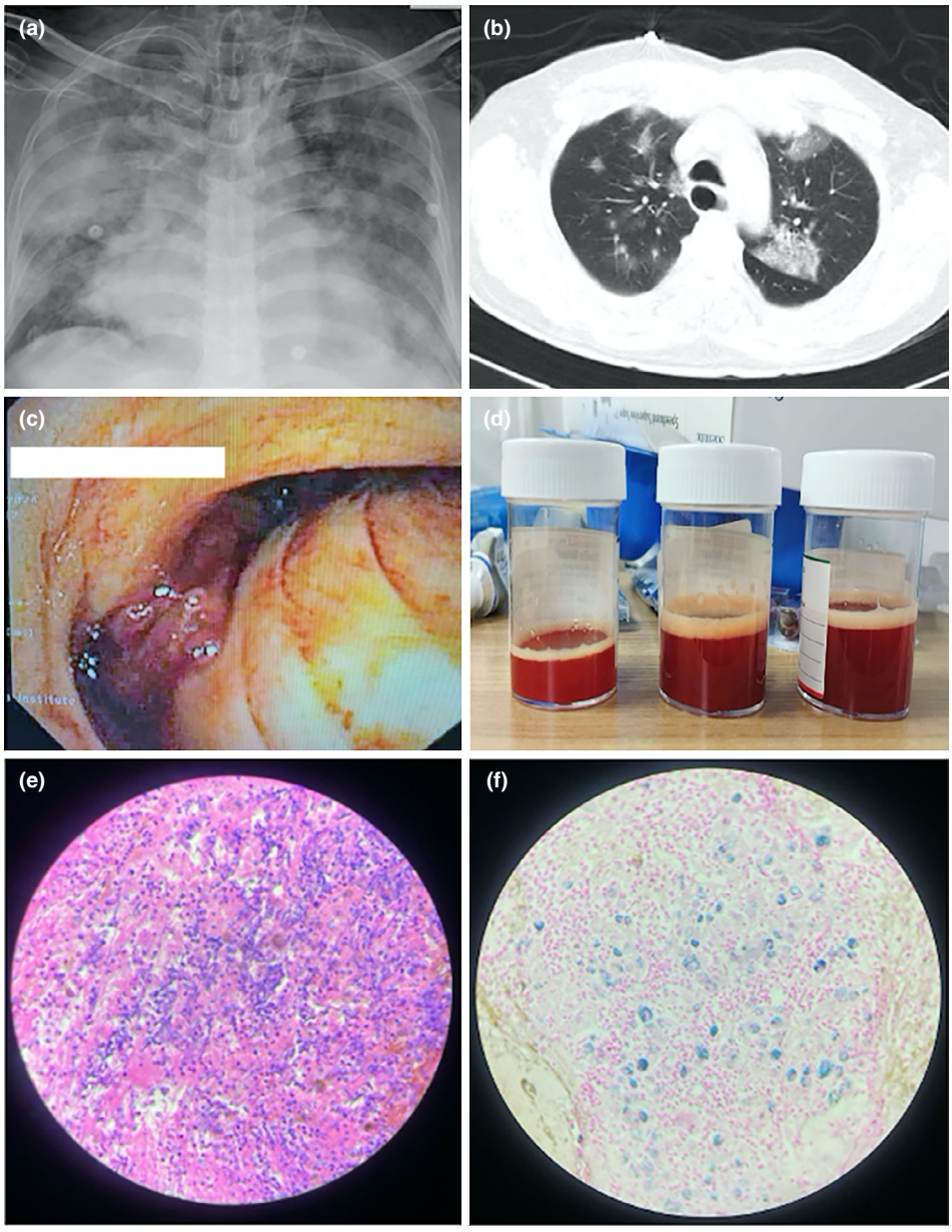

Figure 1: (a) Chest radiograph showed bilateral infiltrates. (b) The High resolution computed tomogram (HRCT) of thorax showing bilateral nodular, multifocal ground glass opacities. (c) Flexible bronchoscopy image showing blood clots at the carina. (d) Bronchoalveolar lavage (BAL) showing progressively bloody return consistent with diffuse alveolar haemorrhage (DAH). (e) BAL fluid showing hemosiderin-laden macrophages on the hematoxylin and eosin stained sections. (f) Perl's Prussian blue stain demonstrating the hemosiderin pigment

a cartridge containing a liquid, a chamber containing a heating element, and a battery-operated power source. E-cigarette liquid generally contains nicotine, propylene glycol, and added flavours. Vaping refers to using electronic cigarettes to vaporize nicotine and other flavoured liquids for inhalation.

EVALI is defined as the presence of bilateral lung opacities, the use of e-cigarettes within the previous 90 days, the exclusion of infection, and the absence of an alternative diagnosis. The clinical spectrum of e-cigarette-associated pulmonary disease is varied, ranging from mild pulmonary symptoms without associated lung injury to life-threatening acute lung injury. Our patient met several of the diagnostic criteria proposed for E-cigarette or vaping-associated lung injury (EVALI), including recent vaping within 90 days, bilateral pulmonary opacities on imaging, and exclusion of infectious causes. However,

Table 1: Cases of vaping induced diffuse alveolar haemorrhage reported in literature

Authors	Year of publication	Age (years)	Gender	Symptom onset after initiation of vaping	Predominant vaping product	Treatment
Agustin et al.[3]	2018	33	Male	2 months	Nicotine	Steroids
Wilhite et al.[4]	2019	22	Male	Not known	THC	Steroids
Edmonds et al.[5]	2020	31	Female	4 years	Nicotine	Only cessation of vaping
Singh et al.[6]	2020	22	Male	3-4 years	THC	Steroids
Shehata et al.[7]	2020	61	Female	2 weeks	THC	Steroids
Villeneuve et al.[8]	2020	28	Female	1 month	Nicotine	Steroids and Rituximab
Suhling et al.[9]	2020	21	Male	2 years	Nicotine	Steroids
Hoekstra et al.[10]	2021	17	Male	Not known	Nicotine and Cannabis oil	Observation

the clinical and bronchoscopic findings were most consistent with diffuse alveolar haemorrhage as the predominant pathological manifestation, rather than with the more typical organizing pneumonia or diffuse alveolar damage patterns described in EVALI. Therefore, this case is best interpreted as vaping-associated DAH within the broader EVALI spectrum, occurring in a patient with significant underlying coagulopathy.

Although vaping can cause various forms of acute lung injury, vaping-triggered DAH is particularly rare. In a retrospective multicentre cohort of 160 EVALI cases, DAH was reported in only six (3.8%) cases.[2] A PubMed search identified eight reported cases of DAH due to vaping (Table 1). Among these, four patients vaped only nicotine, three patients vaped only tetrahydrocannabinol (THC), and one patient vaped both nicotine and THC. All the patients except one was managed with steroids.[3–10] One patient received Rituximab in addition to steroids.[8] One patient improved only with smoking cessation as he was maintaining SpO_2 on room air.[5] Our patient differed from previously reported cases by presenting with advanced liver disease and severe coagulopathy, suggesting that vaping exposure may act as a trigger in such patients.

In our case, several alternative causes of diffuse alveolar haemorrhage were systematically evaluated. Pulmonary–renal syndromes were considered unlikely because of the absence of hematuria or renal dysfunction at presentation and because autoimmune serologies, including ANA and ANCA, were negative. Infectious etiologies were excluded through negative microbiological testing of respiratory samples and blood cultures. Portal hypertension–related pulmonary vascular complications such as hepatopulmonary syndrome and portopulmonary hypertension were also considered; however, echocardiography demonstrated preserved cardiac function without evidence of pulmonary hypertension, and the clinical presentation was not consistent with these conditions. These findings favored the diagnosis of DAH over other pulmonary complications of chronic liver disease.

The mechanism of vaping-induced DAH not fully understood and is proposed to be multifactorial. It may be due either to direct vascular injury from heated chemicals or to an inflammatory cascade triggered by the inhaled chemicals. In the present case, the patient had advanced chronic liver disease with significant coagulopathy (INR 4.7, thrombocytopenia). This likely created a vulnerable haemostatic environment within the pulmonary microvasculature. However, the temporal association with ongoing vaping exposure raises the possibility that inhaled chemical irritants may have acted as a trigger, potentially through direct endothelial injury or inflammatory activation. Thus, the clinical presentation may reflect a multifactorial process in which vaping exposure precipitated DAH in a patient with a pre-existing haemostatic vulnerability. There are no randomised controlled trials on the management of vaping-induced lung injury, so therapeutic regimens have not yet been confirmed. Currently, cessation of e-cigarette use and systemic glucocorticoid therapy are used in the management of vaping-induced DAH. Glucocorticoid therapy was planned, but its use was deferred in our patient due to the development of sepsis. The management focused primarily on correction of coagulopathy using targeted therapy, guided by rotational thromboelastometry (ROTEM), and supportive care.

Conclusion

This case highlights that vaping exposure may act as a potential trigger for diffuse alveolar haemorrhage in patients with predisposing conditions such as severe coagulopathy from chronic liver disease. Clinicians should consider a history of vaping when evaluating unexplained pulmonary haemorrhage in at-risk patients.

Ethics Committee Approval

This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent

Written informed consent was obtained from the patients relative.

Conflict of Interest

The authors have no conflicts of interest to declare.

Funding

The authors declared that this study received no financial support.

Use of AI for Writing Assistance

No use of AI-assisted technologies was declared by the authors.

Author Contributions

Concept – B.J.S., K.D.; Supervision – B.J.S.; Resource – B.J.S., L.P.R., V.P.; Materials – B.J.S., L.P.R., V.P.; Data Collection and/or Processing – B.J.S., L.P.R., V.P.; Analysis and/or Interpretation - K.D., P.G.; Literature Review – K.D., P.G., J.D.; Writing – K.D.; Critical Review – K.D., P.G., J.D.

Peer-review

Externally peer-reviewed.

References

1. Yayan J, Franke KJ, Biancosino C, Rasche K. Comparative systematic review on the safety of e-cigarettes and conventional cigarettes. Food Chem Toxicol 2024;185:114507. [CrossRef]
2. Kligerman SJ, Kay FU, Raptis CA, Henry TS, Sechrist JW, Walker CM, et al. CT Findings and Patterns of e-Cigarette or Vaping Product Use-Associated Lung Injury: A Multicenter Cohort of 160 Cases. Chest 2021;160(4):1492–511. [CrossRef]
3. Agustin M, Yamamoto M, Cabrera F, Eusebio R. Diffuse Alveolar Hemorrhage Induced by Vaping. Case Rep Pulmonol 2018;2018:9724530. [CrossRef]
4. Wilhite R, Patel T, Karle E, Shankar S, Krvavac A. Diffuse Alveolar Hemorrhage: An Uncommon Manifestation of Vaping-associated Lung Injury. Cureus 2019;11(12):e6519. [CrossRef]
5. Edmonds PJ, Copeland C, Conger A, Richmond BW. Vaping-induced diffuse alveolar hemorrhage. Respir Med Case Rep 2020;29:100996. [CrossRef]
6. Singh A, Tan Q, Saccone NM, Lindner DH. A Case of Vaping TCH Oil Leading to Vaping Associated Pulmonary Injury: Our Approach to Its Diagnosis, Management, and Recommendations. Case Rep Pulmonol 2020;2020:6138083. [CrossRef]
7. Shehata M, Kocher T. Vaping-associated diffuse alveolar hemorrhage - A case report. Respir Med Case Rep 2020;30:101038. [CrossRef]
8. Villeneuve T, Prevot G, Le Borgne A, Colombat M, Collot S, Ruiz S, et al. Diffuse alveolar haemorrhage secondary to e-cigarette "vaping" associated lung injury (EVALI) in a young European consumer. Eur Respir J 2020;56(1):2000143. [CrossRef]
9. Suhling H, Welte T, Fuehner T. Three Patients With Acute Pulmonary Damage Following the Use of E-Cigarettes-A Case Series. Dtsch Arztebl Int 2020;117(11):177–82. [CrossRef]
10. Hoekstra NE, Dannull KA, Weinman JP, Liptzin DR, Hinds DM. Vaping and diffuse alveolar hemorrhage: All EVALI is not created equal. Pediatr Pulmonol 2021;56(12):4057–9. [CrossRef]