

Hemoptysis induced by vaping[☆]



Hemoptisis secundaria a vapeo

To the Editor:

In recent years, we have seen the emergence of different methods of smoking tobacco and other substances that are increasing in popularity, mainly among young people and adolescents. Despite the messages propagated among the population, the safety of the fumes and smoke generated by these systems and their usefulness in smoking cessation have not been demonstrated.¹

In this context, we present the case of a 33-year-old man, former smoker of 10 cigarettes/day from the age of 19 until 2 months previously, when he began to vape nicotine-free products (3 ml in 3 doses per day). He was receiving sertraline for an anxiety-depressive disorder, practiced sports regularly, and had no occupational risk exposure.

He presented in the emergency department with sudden onset of bloody expectoration after excessive consumption of vaping fluid (almost 30 ml, higher than usual) with a new flavoring agent. Upon arrival, the patient was hemodynamically stable, breathing normally, with a baseline oxygen saturation of 95%. Lab tests were performed, showing leukocytosis 12,230 cells/mm³. All other parameters were normal (platelets, coagulation, CRP, liver and renal profile, and later, autoimmune tests) and Ziehl staining in sputum was negative. Chest X-ray findings showed pathological changes. A CT scan was then performed, showing a ground glass infiltrate in the left upper lobe (Fig. 1). Under observation, the patient produced 40 ml bloody expectoration in 12 hours, but after admission to the ward, he produced 50 ml in 1 h. An arteriography was performed, and a tortuous right bronchial artery was observed, which was embolized, reducing the bleeding. Despite this procedure, the patient continued to expectorate blood in smaller amounts, so a bronchoscopy was performed, showing active diffuse capillary bleeding in both sides, with no clear source, which could not be controlled with cold serum or adrenaline. He was then admitted to the ICU for monitoring and was treated with low-flow oxygen therapy through nasal prongs, antibiotics (levofloxacin), and corticosteroids (methylprednisolone). Fortunately, his progress was favorable; he did not require intubation and was moved to the ward within 48 h. When the patient was stable, bronchoscopy was performed, which ruled out any endobronchial lesion. Abundant siderophages were observed in bronchoalveolar lavage, but no foamy macrophages were present and no microorganism was isolated. One month later, the patient was re-admitted to hospital-at-home, due to superinfection, for which he received another cycle of intravenous antibiotics. He subsequently remained asymptomatic until discharge from follow-up months later.

This is a case of special interest that can be added to the cases of lung damage associated with vaping, called e-cigarette or vaping product use associated lung injury (EVALI), that have recently been described. Nearly 3000 cases have been reported to the Centers for Disease Control in the United States, and the largest published cohort comprises 60 patients.² For this reason, in September 2019, the Centers for Disease Control published a guide collating the available scientific evidence.

The diagnosis of a “confirmed” case is based on 4 criteria: 1) electronic cigarette use in the previous 90 days; 2) pulmonary opacities; 3) exclusion of lung infection (respiratory virus, influenza PCR,



Fig. 1. CT scan showing infiltrate in the left upper lobe.

antigenuria for *Legionella* and *Streptococcus pneumoniae*: negative; blood cultures/sputum culture/bronchoalveolar lavage, detection of opportunistic germs, if performed: negative); and 4) no alternative diagnosis. A case would be designated as “probable” if no microbiological tests were carried out to rule out infection, or if any of these tests were positive but not considered clinically to be the only cause of the disease.³

Our patient is therefore a “probable” case of vaping-related lung disease, since the cause-effect relationship (use-hemoptysis) is very clear and only one sputum culture was available. Other causes of hemoptysis were ruled out by the laboratory tests: pulmonary-renal syndromes, bleeding disorders, and autoimmune diseases (normal labs), parenchymal lesions (infiltrate, tumors, cavities, bronchiectasis, etc., by CT scan), pulmonary thromboembolism, arteriovenous malformations, and endobronchial lesions (e.g., tumor or foreign body, by bronchoscopy).

It should be noted that no cases have been previously reported in Spain, and that our patient inhaled a flavoring substance that did not contain nicotine, tetrahydrocannabinol or cannabidiol, as was the case for all patients previously published.^{2,4-6}

The form of presentation with very acute hemoptysis after excessive consumption of the inhaled substance is also noteworthy, when compared with the mean 6-day duration of symptoms in other series (in the cohort of 53 patients). Hemoptysis is the least frequent of all symptoms presented (occurring in 11% of the series^{2,4}). Different forms of presentation have been described: organizing pneumonia, lipoid pneumonia, hypersensitivity pneumonitis, respiratory bronchiolitis, giant cell pneumonia,⁷ diffuse alveolar hemorrhage,⁸ and diffuse alveolar damage, among others.⁹⁻¹¹

In terms of treatment, antibiotics covering the most common microorganisms in community-acquired pneumonia have been prescribed empirically in most patients. Corticosteroids are not routinely recommended, except in life-threatening cases, in patients suffering from clinical deterioration and hypoxemia, and if eosinophilic or cryptogenic organizing pneumonia is observed, in line with standard care. The recommended treatment is methylprednisolone 0.5–1 mg/kg/day for 5–10 days, depending on the patient’s response and progress.⁴

With regard to disease course, series published so far (n = 53⁴ and n = 60²) report that more than half of the patients were admitted to the ICU, but less than one third required orotracheal intubation, and 3 deaths were recorded.^{2,4}

In short, as professionals we must be alert to new patterns of inhaled drug use, because of our profound lack of knowledge of the short- and long-term effects. Cases like ours are beginning to

[☆] Please cite this article as: Cano Aguirre M d P, Esperanza Barrios A, Martínez Muñoz F, Alonso Viteri S, Muñoz González F, Segoviano Mateo R, et al. Hemoptisis secundaria a vapeo. Arch Bronconeumol. 2021;57:505–506.

reveal some of the consequences of short-term consumption, but the pathophysiological mechanism by which the lung injury occurs, the substances responsible for it, and its correct treatment are still unknown.

A patient registry, as proposed by SEPAR, is now urgently needed to enable us to carry out a thorough analysis of the cases and shed light on this new entity. We must establish more precise diagnostic criteria, identify the substances responsible for lung damage, and produce solid scientific evidence to guide the proper management of these patients and to convey a clear message to the general population and health authorities about these devices.

References

- Signes-Costa J, de Granda-Orive JI, Ramos Pinedo Á, Camarasa Escrig A, de Higes Martínez E, Rábade Castedo C, et al. Official statement of the Spanish Society of Pulmonology and Thoracic Surgery (SEPAR) on Electronic Cigarettes and IQOS®. Arch Bronconeumol. 2019;55:581–6. <http://dx.doi.org/10.1016/j.arbres.2019.04.023>. Declaración Oficial de la Sociedad Española de Neumología y Cirugía Torácica (SEPAR) sobre cigarrillos electrónicos e IQOS®.
- Blagev DP, Harris D, Dunn AC, Guidry DW, Grissom CK, Lanspa MJ. Clinical presentation, treatment, and short-term outcomes of lung injury associated with e-cigarettes or vaping: a prospective observational cohort study. Lancet. 2019;394:2073–83. [http://dx.doi.org/10.1016/s0140-6736\(19\)32679-0](http://dx.doi.org/10.1016/s0140-6736(19)32679-0).
- Schier JG, Meiman JG, Layden J, Mikosz CA, Van Frank B, King BA, et al. Severe pulmonary disease associated with electronic-cigarette-product use – interim guidance. MMWR Morb Mortal Wkly Rep. 2019;68:787–90. <http://dx.doi.org/10.15585/mmwr.mm6836e2>.
- Layden JE, Ghinai I, Pray I, Kimball A, Layer M, Tenforde MW, et al. Pulmonary illness related to E-cigarette use in Illinois and Wisconsin – final report. N Engl J Med. 2020;382:903–16. <http://dx.doi.org/10.1056/NEJMoa1911614>.
- Kalininskiy A, Bach CT, Nacca NE, Ginsberg G, Marraffa J, Navarette KA, et al. E-cigarette, or vaping, product use associated lung injury (EVALI): case series and diagnostic approach. Lancet Respir Med. 2019;7:1017–26. [http://dx.doi.org/10.1016/s2213-2600\(19\)30415-1](http://dx.doi.org/10.1016/s2213-2600(19)30415-1).
- Triantafyllou GA, Tiberio PJ, Zou RH, Lamberty PE, Lynch MD, Kreit JW, et al. Vaping-associated acute lung injury: a case series. Am J Respir Crit Care Med. 2019;200:1430–1. <http://dx.doi.org/10.1164/rccm.201909-1809LE>.
- Henry TS, Kanne JP, Kligerman SJ. Imaging of vaping-associated lung disease. N Engl J Med. 2019;381:1486–7. <http://dx.doi.org/10.1056/NEJMc1911995>.
- Agustin M, Yamamoto M, Cabrera F, Eusebio R. Diffuse alveolar hemorrhage induced by vaping. Case Rep Pulmonol. 2018;2018. <http://dx.doi.org/10.1155/2018/9724530>, 9724530.
- Bakre SA, Al-Farra TS, Al-Farra S. Diffuse alveolar damage and e-cigarettes: case report and review of literature. Respir Med Case Rep. 2019;28:100935.
- Maddock SD, Cirulis MM, Callahan SJ, Keenan LM, Pirozz CS, Raman SM, et al. Pulmonary Lipid-Laden Macrophages and Vaping. N Engl J Med. 2019;381:1488–9. <http://dx.doi.org/10.1056/NEJMc1912038>.
- Butt YM, Smith ML, Tazelaar HD, Vaszar LT, Swanson KL, Cecchini MJ, et al. Pathology of vaping-associated lung injury. N Engl J Med. 2019;381:1780–1. <http://dx.doi.org/10.1056/NEJMc1913069>.

María del Puerto Cano Aguirre,^{a,e,*} Alba Esperanza Barrios,^{a,e} Francisco Martínez Muñoz,^a Soledad Alonso Viteri,^{a,e} Francisco Muñoz González,^b Rocío Segoviano Mateo,^c Ángela Algaba Calderón^{d,e}

^a Servicio de Neumología, Hospital Universitario de Torrejón, Torrejón de Ardoz, Madrid, Spain

^b Servicio de Neumología, Hospital Recoletas Campo Grande, Valladolid, Spain

^c Servicio de Medicina Interna, Hospital Universitario de Torrejón, Torrejón de Ardoz, Madrid, Spain

^d Servicio de Medicina Intensiva, Hospital Universitario de Torrejón, Torrejón de Ardoz, Madrid, Spain

^e Universidad Francisco de Vitoria, Madrid, Spain

Corresponding author.

E-mail address: mpcano@torrejonosalud.com (M.d.P. Cano Aguirre).

<https://doi.org/10.1016/j.arbr.2021.05.006>

1579-2129/ © 2020 SEPAR. Published by Elsevier España, S.L.U. All rights reserved.

Symptomatic azygous vein thrombosis: Clinical case and literature review[☆]



Trombosis sintomática de la vena ázigos: caso clínico y revisión de la literatura

To the Editor:

Azygos vein thrombosis (AVT) and hemiazygos vein thrombosis (HVT) are rare entities. The etiopathogenesis is unknown, but these thromboses have been associated with predisposing factors, such as underlying azygos vein aneurysm (AVA)¹ or other prothrombotic factors. AVT in a normal azygos and hemiazygos vein is exceptional.² The azygos system provides the superior (SVC) and inferior vena cava (IVC) with an alternative access to the right atrium, so AVT is usually asymptomatic, making it difficult to diagnose and treat early. It can be complicated by pulmonary embolism (PE), vena cava syndrome (VCS) and even stroke in the case of cardiac shunt, which confers a worse prognosis.³ This paper reports

an acute, symptomatic case of AVT and HVT and reviews the most relevant aspects of this rare entity.

A 60-year-old woman, former smoker, receiving chemotherapy for a diagnosis of ovarian adenocarcinoma, presented with sudden dyspnea, heaviness in the upper limbs, and syncope. Physical examination revealed blood pressure 110/60 mmHg, heart rate 110 bpm, oxygen saturation 95%, erythema and facial edema, and no signs of collateral circulation or lymphadenopathies. On laboratory tests, hemoglobin was only 8.6 g/dl. Chest CT-angiogram showed extensive thromboses in the azygos and hemiazygos veins, with no signs of PE or VCS (Fig. 1). No findings of interest were observed on lower limb ultrasound or echocardiogram. Thrombophilia tests, including antiphospholipid antibodies, were negative. The patient was given weight-adjusted tinzaparin treatment and anticoagulation continued until the end of chemotherapy (9 months), with complete resolution of her respiratory symptoms and no evidence of complications. Repermeabilization of the AVT was observed in the follow-up CT.

We performed a Pubmed search for original and review articles using the term “azygos vein thrombosis”. Original articles were eligible for inclusion if AVT and/or HVT were identified. Abstracts, animal studies, and articles written in languages other than English or Spanish were excluded. The references for each article included in this review were also analyzed to identify other studies of interest. We found a total of 216 eligible studies, published up to June 2020. Nineteen studies met the inclusion criteria and all reported

[☆] Please cite this article as: Lopez-Bauza A, Jara-Palomares L. Trombosis sintomática de la vena ázigos: caso clínico y revisión de la literatura. Arch Bronconeumol. 2021;57:506–509.