Methanol poisoning by inhalation

Intoxicación por metanol inhalado

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Editor in Charge:
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To the Editor,

Methanol (CH_3-OH) or methyl alcohol is a type of alcohol with a wide range of uses. Although not suitable for consumption, it is used in industrial products, household cleaning agents, and as a denaturant for ethyl alcohol. It is a liquid that is colorless, flammable, and volatile. The degree of toxicity of this alcohol is related to the amount of toxic metabolites it produces.[1] Although methanol poisoning is an infrequent cause of intoxication in Spain, it is among the most dangerous. The magnitude of the damage produced depends on the dose, the time elapsed, and the route of exposure.[2] Most poisonings occur via the oral route, but inhalational exposure may also occur. Recently, a case of methanol poisoning through inhalation was reported in a patient who presented several days after exposure.[3] Because of the rarity of this route of entry, we consider it interesting to report a new case.

A 59-year-old woman, smoker, with moderate alcohol consumption and occasional cocaine use. Her past medical history included type 2 diabetes mellitus, hypertension, dyslipidemia, fibromyalgia, and multifactorial gait disorder. The patient presented with a 24-hour history of altered behavior—such as attempting to turn on the television using her glasses or trying to smoke a cookie. She exhibited no other accompanying symptoms. Upon arrival at the emergency department (ED), her blood pressure was 157/100 mmHg, heart rate 113 bpm, axillary temperature 35.3°C, respiratory rate 12 breaths/min, and oxygen saturation on room air 97%. She was conscious and oriented to person and place and partially oriented to time. Neurological examination showed difficulty with recall (4/5 fruits); the rest of the physical and neurological exams were unremarkable. ECG revealed sinus rhythm without repolarization abnormalities. Chest X-ray and cranial CT scan were both normal. Urine toxicology (enzyme immunoassay) tested positive for cocaine. Routine blood tests were within normal limits, but venous blood gas analysis revealed metabolic acidosis (pH 7.15; pCO_2 31.1 mmHg; HCO_3^- 10.7 mEq/L) with elevated anion and osmolal gaps. Determination of ethanol, methanol, and ethylene glycol levels was requested due to suspicion of intoxication, revealing methanol concentration of 110 mg/L. Upon further questioning, the patient reported that she had been cleaning with a product containing methanol 3 days prior. She received sodium bicarbonate and was started on IV ethanol and folic acid. The patient maintained a good level of consciousness, and metabolic acidosis corrected after treatment. After approximately 12 hours into therapy, and in the absence of neurological symptoms, IV ethanol was discontinued.

Acute methanol poisoning causes metabolic acidosis, neurological and visual impairment, and ultimately death.[4] Therapy consists of general supportive measures, symptomatic treatment, and administration of an antidote (ethanol or fomepizole). The use of IV ethanol could be considered debatable in this case, since levels were below the toxicity threshold; however, because of the symptoms and metabolic acidosis, antidotal treatment was initiated. Ethanol has an affinity for alcohol dehydrogenase (ADH) that is 10–20 times greater than methanol, and fomepizole has an even higher affinity. Treatment should be started without waiting for blood methanol levels if clinical suspicion exists. Methanol is metabolized by ADH, forming formic acid,[2] which causes acidosis and inhibits the mitochondrial function of the retina, leading to visual damage and metabolic acidosis.

The fact that 3 days had passed since methanol exposure may explain the low plasma methanol levels yet significant metabolic acidosis observed on admission. Of note, the positive cocaine result. The patient admitted using it the night before, but this use does not appear to explain the marked metabolic acidosis observed.

We emphasize the importance of maintaining diagnostic suspicion of intoxication in cases with atypical clinical signs and normal initial additional tests. In methanol poisoning, be-

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cause it is a rare but potentially lethal condition if not treated early, it is essential to identify warning signs and symptoms at first contact. When diagnostic suspicion arises, methanol levels should be requested and treatment initiated immediately with IV ethanol or fomepizole.[5]

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Respiratory distress syndrome secondary to *Streptococcus pyogenes* infection related to the use of e-cigarettes in a young immunocompromised man

Síndrome de distrés respiratorio secundario a infección por Streptococcus pyogenes *por el uso de cigarrillos electrónicos en un joven inmunodeprimido*

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Ethical Responsibilities:
All authors have confirmed their authorship, the nonexistence of external funding, and the maintenance of confidentiality and respect for patients' rights in the author's responsibilities document, publication agreement, and assignment of rights to Revista Española de Urgencias Emergencias.

Editor in Charge:
Guillermo Burillo-Putze.

To the Editor,

Electronic cigarettes are a recent form of inhaled consumption, particularly popular among young people. Their harmful health effects remain largely unknown, especially in the long term. However, cases of death due to severe acute complications from their use—such as EVALI (E-cigarette or vaping-associated lung injury)—have already been reported.[1]

This is the case of a 25-year-old man with Crohn's disease on adalimumab and a habitual e-cigarette user, who presented to the emergency department with a 48-hour history of dyspnea, cough, and sputum production following a significant increase in e-cigarette consumption after a social event. On initial examination, the patient appeared in fair general condition, tachypneic, with peripheral oxygen saturation (SpO_2) 92% on nasal cannula at 2 L/min, and diffuse crackles on lung auscultation. Laboratory tests showed elevated acute-phase reactants (C-reactive protein 486 mg/L, procalcitonin 94 ng/dL). Chest X-ray revealed an alveolar infiltrate in the right upper lobe (RUL) (Figure 1A). While the patient was being admitted to the Pulmonology Department, the rapidly deteriorated, developing hemoptysis and worsening respiratory failure, requiring oxygen with a Venturi mask (FiO_2 40%) to maintain SpO_2 90%. An emergency chest CT showed extensive involvement of the RUL parenchyma with a "crazy-paving" pattern and multiple pseudonodular ground-glass opacities in the remaining lobes (Figure 1B). Differential diagnosis included pulmonary hemorrhage, bacterial pneumonia, and acute respiratory distress syndrome (ARDS). The patient was admitted to the Intensive Care Unit (ICU). Bronchoscopy yielded hemorrhagic fluid, and *Streptococcus pyogenes* was isolated from sputum and bronchoaspirate cultures. Other microbiological studies and autoimmune tests were negative. Antibiotic treatment with benzylpenicillin and clindamycin was started, along with high-flow nasal oxygen therapy. After 24 hours, orotracheal intubation and invasive mechanical ventilation were required. Due to worsening hemodynamic status, he was transferred to *Hospital Reina Sofía* UCI (Córdoba, Spain), where venovenous extracorporeal membrane oxygenation (VV-ECMO) and ultraprotective mechanical ventilation were initiated. The patient improved progressively, with respiratory support withdrawn after 10 days, and subsequently had a favorable clinical outcome.

Electronic cigarettes have become increasingly popu-

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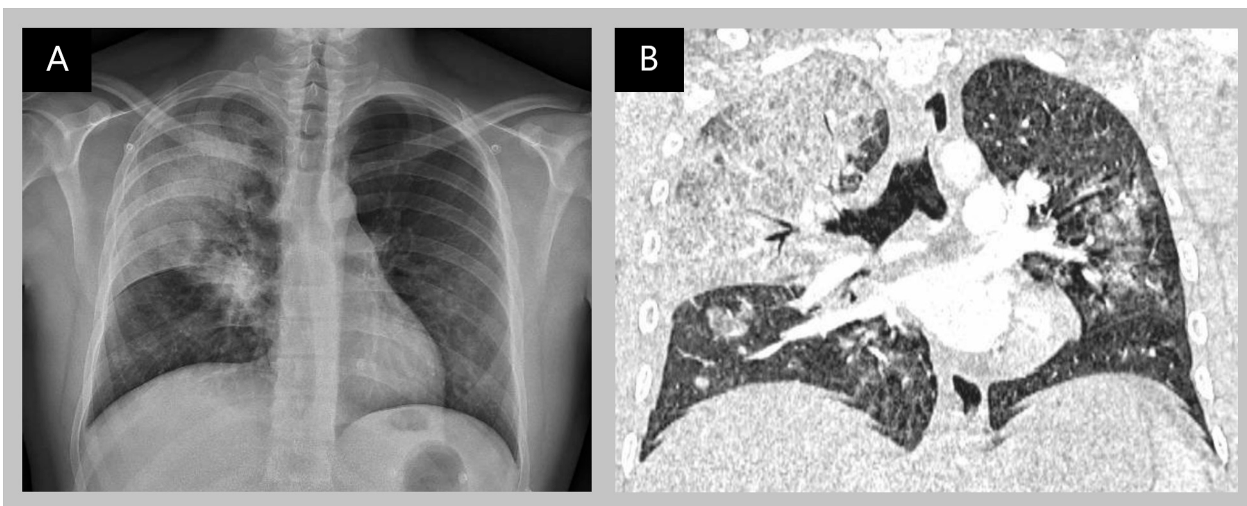

Figure 1. Chest X-ray obtained upon arrival to the emergency department showing alveolar infiltrate in the right upper lobe (RUL) and ipsilateral hilar enlargement. B: Chest CT scan showing a "crazy-paving" pattern in the RUL and areas of ground-glass opacity in the left upper and right lower lobes.

lar, especially among young people. Studies indicate that aerosol mixtures—composed of propylene glycol, glycerin, nicotine, and flavorings—can alter microbial growth and biofilm formation,[2] as well as lipid metabolism, compromising the epithelial barrier function of the upper respiratory tract. These effects can activate macrophages and type II pneumocytes, triggering inflammatory responses that may result in serious outcomes, as occurred in our case.[3]

S. pyogenes is part of the normal microbiota of the throat, genital mucosa, rectum, and skin. Many studies investigating the impact of e-cigarette use on the oropharyngeal microbiota focus on *Streptococcus* or *S. pneumoniae,* and some have shown increased colonization by these bacteria.[4] Recently, "vaping" devices different from e-cigs—which vaporize solid substances without nicotine that appear harmless—have become more common, but they can also be dangerous.[5] Moreover, especially among young people, there is an increase in the use of synthetic cannabinoids, which adds the toxic risk of these drugs, particularly via the pulmonary route.[6]

In our case, although not proven, it is plausible that the inhalation of nicotine-containing aerosols via e-cigs combined with the patient's immunosuppression converted this microorganism into a potential pathogen, leading to a severe infectious-inflammatory process. Therefore, it is essential to identify these risk factors in the emergency setting and warn users about the potential dangers, both in the general population[7] and particularly in vulnerable patients, as illustrated by this case.

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