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Hydrofluoric acid inhalation injury after electric bike battery fire

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Summary *Background:* The rapid increase in electric bike (e-bike) use has led to a rise in lithium-ion battery fires, which present significant hazards. Beyond thermal injury, these fires emit toxic gases such as hydrogen fluoride (HF), capable of causing severe chemical inhalation injury. The pulmonary effects of inhaled hydrofluoric acid are not well characterised in the literature.
Cases: Two cases of severe lung injury occurred following indoor e-bike battery fires. The first patient sustained 32% total body surface area (TBSA) burns and developed acute respiratory distress syndrome with radiological evidence of chemical pneumonitis, necessitating prolonged mechanical ventilation and resulting in persistent pulmonary impairment. The second patient sustained 44% TBSA burns and experienced rapidly progressive respiratory failure that was disproportionate to typical smoke inhalation injury. Despite maximal supportive therapy, including extracorporeal membrane oxygenation, the patient died from catastrophic pulmonary failure.
Conclusion: Lithium-ion battery fires present both thermal and chemical hazards, especially in enclosed environments. These cases highlight the importance of maintaining a high index of suspicion for toxic inhalation injury, promptly recognising disproportionate respiratory failure, and monitoring for biochemical indicators of hydrofluoric acid exposure as the prevalence of lithium-ion battery use increases.

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The e-bike market is growing rapidly. Lithium-ion batteries pose new safety risks, particularly fires that generate heat and toxic gases, complicating burn and inhalation injury management.[1]

In addition to asphyxiant gases such as carbon monoxide, lithium-ion battery fires release hydrogen fluoride (HF), which, upon contact with water, forms hydrofluoric acid. Although the systemic toxicity of hydrofluoric acid is well established, the pulmonary effects of inhaled hydrofluoric acid remain poorly characterised, and clinical guidance for managing these exposures is limited.[2]

This report presents two cases of severe lung injury following enclosed space lithium-ion battery fires, highlighting the risk of chemical inhalation injury and the need for clinical awareness.

Case presentation

Case 1

A man in his thirties sustained 32% TBSA burns and inhalation injury after his e-bike's lithium-ion battery ignited while charging. He was intubated prophylactically and transferred to a National Burns Unit.

Bronchoscopy showed carbonaceous deposits and early airway oedema, consistent with inhalation injury. Initial findings included respiratory acidosis (pH 7.26, pCO_2 6.6 kPa), a mildly reduced serum calcium (2.1 mmol/L) and derangement of coagulation indices (prothrombin time [PT] 15.1, activated partial thromboplastin time [aPTT] 40.3). His burns were debrided and grafted. During the first 5 days his gas exchange deteriorated, and serial chest X-rays (CXR) showed worsening bilateral infiltrates consistent with acute respiratory distress syndrome (ARDS), (Supplementary Figure 1).

CT of the thorax on day 13 showed widespread ground-glass opacities, pneumatoceles, bronchial wall thickening, and traction bronchiectasis, indicating alveolar wall destruction and severe chemical pneumonitis, (Figure 1). He required mechanical ventilation for 45 days due to persistent gas exchange impairment. His exercise tolerance remains significantly reduced at two-year follow-up.

Case 2

A 37-year-old man sustained 44% TBSA flame burns with an inhalation injury after a lithium-ion e-bike battery ignited while charging. He escaped the building and was transferred to a local hospital. He had an initial carboxyhaemoglobin level of 5.6%, and a mild hypocalcaemia of 2.02 mmol/l. His blood gas showed mixed metabolic and respiratory acidosis (pH 7.31, pO2 7.34 kPa, pCO2 5.24 kPa, HCO3- 19.5 mmol/l) along with coagulation derangement (PT 23, aPTT 66.8).

Given his facial burns, he was electively intubated. At the time, bronchoscopy showed carbonaceous material in his upper airways, with his distal airways relatively preserved. He was transferred to a National Burns Unit and underwent bilateral upper-limb escharotomies.

His burn was debrided and grafted on days 1 and 3 of his admission. He underwent a surgical tracheostomy on day 6 of admission. Despite early burn debridement, he remained acidotic and at one week developed severe hypercapnic respiratory failure (FiO_2 70%, pCO_2 23 kPa, pH 6.8, SaO_2 80%). CXR showed extensive bilateral airspace opacification, consistent with ARDS. (Supplementary Figure 2).

Various ventilatory strategies were trialled including high-frequency oscillatory ventilation, prone positioning, and inhaled nitric oxide without improvement. He was commenced on veno-venous extracorporeal membrane oxygenation (VV-ECMO) on day 8 of admission. Repeat bronchoscopy revealed hyperaemic, grossly oedematous airways. After one week of ECMO he failed to wean to mechanical ventilation. CT thorax imaging on day 21 demonstrated complete bilateral pulmonary opacification with poor parenchymal preservation and extensive pulmonary destruction with no aeration beyond the main bronchi (Figure 2). Following 18 days of VV-ECMO, he passed away. His burn was fully healed.

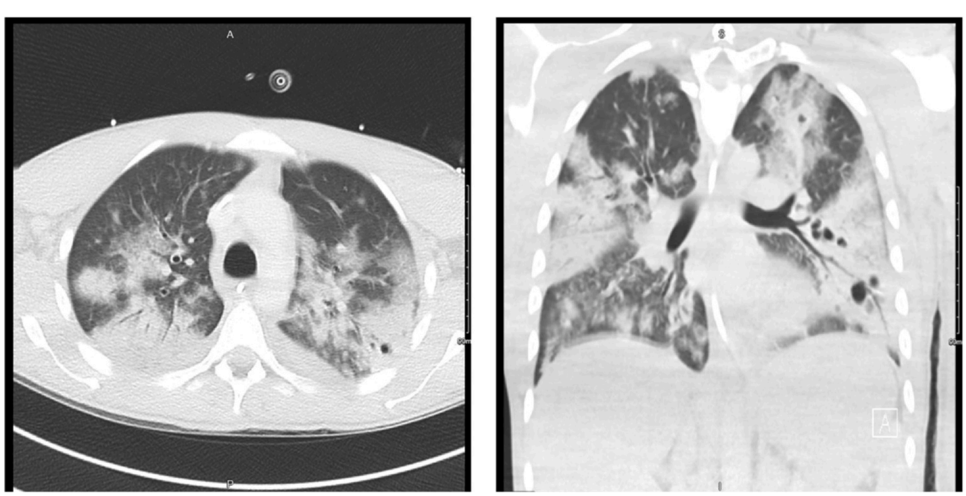

Figure 1 Chest CT performed on day 13 of admission demonstrating extensive perihilar ground-glass opacification with superimposed dense bilateral bibasal consolidations. Traction bronchiectasis and multiple pneumatoceles are present. Overall radiological appearance is consistent with alveolar destruction.

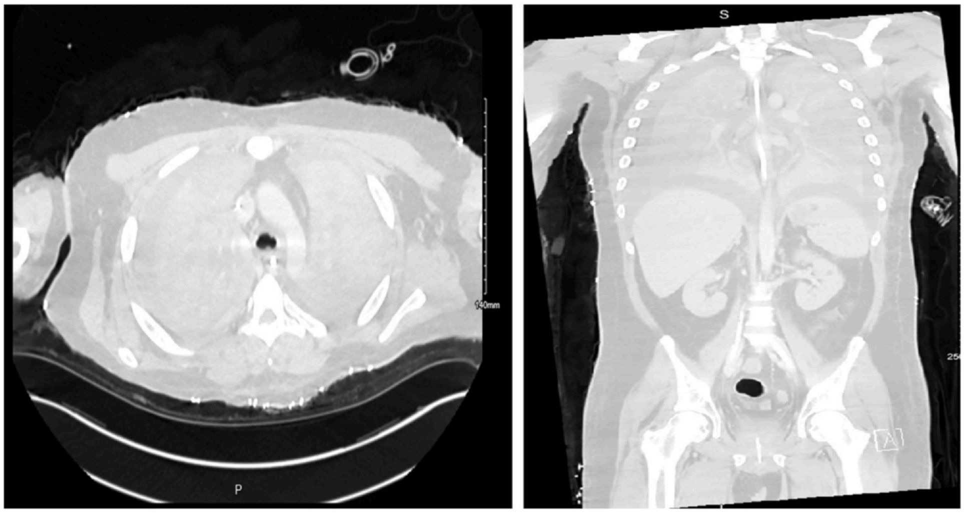

Figure 2 Contrast-enhanced computed tomography (CT) of the thorax obtained on day 21 of admission while the patient was supported on veno-venous extracorporeal membrane oxygenation (VV-ECMO). Axial (left) and coronal (right) views demonstrate complete bilateral pulmonary opacification with moderate pleural effusions. There is absence of aeration of the bronchial tree beyond the main bronchi at the level of the hila bilaterally, findings supported by bronchoscopy which showed extensive sloughing of the mucosa and complete airway destruction.

Discussion

Thermal and chemical injuries from lithium-ion battery fires are emerging challenges in burn care. Engineering literature documents battery hazards, but medical consequences, especially inhalation injuries, are less understood.

Combustion of lithium-ion batteries release asphyxiants as well as fluorinated compounds that decompose to produce hydrogen fluoride (HF). HF and hydrofluoric acid are particularly hazardous due to their ability to penetrate tissues, bind calcium, and induce both local tissue destruction and systemic toxicity.[3]

During battery failure, HF release can reach 20–200 mg/Wh. As e-bike batteries store 250–3000 Wh, concentrations can rapidly exceed the immediately dangerous to life or health threshold of 25 mg/m^3. Which may quickly prove leathal particularly in confined rooms underscoring the risk of indoor charging.[3]

The cases presented illustrate the combined thermal and chemical hazards associated with lithium-ion battery fires. Both patients developed severe respiratory failure despite early respiratory support and aggressive burn management. One patient survived prolonged mechanical ventilation with pulmonary morbidity, while the other exhibited clinical and biochemical features consistent with hydrofluoric acid inhalation, resulting in catastrophic lung injury. Important early clinical findings common to both cases were mild hypocalcaemia, coagulation derangement and bronchoscopic oedema. Serial calcium monitoring was performed, however, trajectories were confounded by fluid resuscitation, electrolyte management and renal replacement therapy, precluding meaningful interpretation. Respiratory impairment out of proportion to bronchoscopic findings is an important early indicator of potential HF inhalation exposure.

No established management protocol exists for HF inhalation injury. We advocate early calcium monitoring and intensive respiratory support. Although direct evidence is lacking, nebulised calcium gluconate, used in cutaneous HF burns to chelate free fluoride, may be mechanistically appropriate and warrants consideration.

Conclusion

These cases highlight the need for clinical suspicion of toxic inhalation injury following lithium-ion battery fires and underscore the importance of early recognition, monitoring for biochemical indicators of HF exposure, and consideration of chemical pneumonitis in this emerging injury pattern.

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Declaration of Competing Interest

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.bjps.2026.04.035.

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