Editorial

Current clinical management of smoke inhalation injuries. A reality check.

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To the Editor,

A major disaster is happening at the moment, as the Camp Fire, Woolsey Fire and Hill Fire are burning in California. Camp Fire in Northern California has already burned 546.3 km^2 and is the deadliest wildfire in the history of the state, with 48 fatalities and still counting (1). It was also only recently, in July 2018, when a fire entered the populated area of Mati, Greece, and created a wildland urban interface that caused 99 fatalities and numerous burns and smoke inhalation injuries. A few years ago, in August 2007, 67 people died in a megafire event in the Peloponnese region, Greece, which was created by 55 simultaneous large fires (based on size, intensity, environmental and socio-economic impact) (2). These and numerous other tragic incidents highlight the importance of our current clinical management in the victims of fire.

Burns are categorised according to depth, and their severity depends on the extent (percentage of total body surface area), age of the individual and accompanied smoke inhalation. Essential treatment for the burn victims remains the resuscitation with intravenous fluids to maintain optimal fluid balance, nutrition optimisation, wound coverage, and pain control (3). Smoke inhalation injury increases mortality and is the most common cause of death in the event of fire (4). This type of injury is defined as the "damage caused by breathing in harmful gases, vapours, and particulate matter contained in smoke" (4) and is categorised as: a) thermal, usually involving damage of the upper respiratory track by the high temperature of smoke and gases, b) chemical, usually involving damage of the lower respiratory system from the inhalation of irritant fire by-products and particulate matter deposition, c) asphyxiation and systemic toxicity, caused by inhalation of deleterious gases produced by combustion, usually carbon monoxide (CO) and hydrogen cyanide (HCN), or d) a combination of the above (5).

Diagnosis of smoke inhalation injury is based upon the history, signs and symptoms, and fibre-optic bronchoscopy, although parenchyma damage in some cases can be present even with negative bronchoscopy findings (6). Clinical indicators of smoke inhalation are: facial burns; lips, tongue, mouth, pharynx burns; or nasal mucosa singeing of nasal hair or eyebrows; oropharyngeal acute inflammatory changes; carbonaceous particles containing toxic chemicals (soot) in the oropharynx; and signs of airway obstruction, irritation or damage. A history of confusion, reduced level of consciousness or unconsciousness, and/or entrapment in a burning location, and carboxyhemoglobin (COHb) level of >10% are additional indicators. Symptoms can be present on hospital admission or develop up to 48 h post burn and they include dyspnoea, evidence of increased work of breathing, hoarseness, wheeze, stridor and ronchi, productive cough and soot stained/carbonaceous sputum (4). Bronchoscopy visualises the severity of inhalation injury, provides the grading system Abbreviated Injury Score (AIS), and uses washout fluid for pulmonary hygiene, microscopy and culture (7). Normal findings on chest radiography or oxygenation do not exclude injury (5).

Smoke inhalation injury can be critical and life-threatening, and there is a risk for rapid deterioration and admission to the intensive care unit (ICU) (4). Patients should be continuously monitored and assessed, particularly for upper airways obstruction, as oedema can be presented up to 36 h post burn (4, 5). The length of exposure, generated

temperatures and micro-environment of a fire, i.e. the scene, its materials, the availability of oxygen, and the nature of the combustion, dictate the composition of gases and severity of the injury (4). Chemical irritants, along with toxic by-products, damage the airway epithelium and introduce an inflammatory response with neutrophil infiltration and fibrinogen activation, which can lead to oedema and bronchospasm. Dead epithelial cells, inflammatory cells, mucous and fibrin formulate airway casts. These casts cause various degrees of mechanical airway obstruction, increase resistance and reduce lung compliance, therefore increase the work of breathing and generate ventilation-perfusion (V/Q) mismatch (4, 6).

To ensure airway patency, particularly in the development of tongue and tracheal oedema and stenosis, patients may require intubation by endotracheal tube or tracheostomy and mechanical ventilation (8). Ventilator-associated pneumonia (VAP) is a common complication and people with extensive burns may present pulmonary oedema or adult respiratory distress syndrome (ARDS). Since positive pressure mechanical ventilation is associated with ventilator-associated lung injury and ARDS, clinicians can apply lung protective strategies when this is compatible with the respiratory demands of the patient (5). Non-conventional ventilation strategies include the high-frequency percussive ventilation (HFPV), prone positioning and extracorporeal membrane oxygenation (ECMO), but evidence does not support their routine use (9-12). Patients in respiratory failure, during the weaning process or self-ventilated individuals with reduced chest wall elasticity and lung compliance, may require non-invasive ventilation (NIV) (13). In the application of the NIV, patient compliance and special contraindications to NIV such as facial burns, are considered (12).

Indeed, protecting gas exchange is paramount. The CO in the fire scene usually causes hypoxaemia, which in closed environments worsens from the fire's use of the oxygen. Additionally, patients with burns present hypermetabolic response, therefore high respiratory demands. Loss of the physiological hypoxic pulmonary vasoconstriction also results in V/Q mismatch. Restoring hypoxaemia reduces the V/Q mismatch and shifts the oxygen dissociation curve to the right, thus the increase of arterial partial pressure of oxygen (PaO_2) accelerates CO displacement from the haemoglobin in the tissue level (5). High fraction of inspired oxygen (FiO_2) is provided, initially 100% and then at the lowest acceptable level. The addition of humidification facilitates airway clearance, particularly in the presence of copious carbonaceous secretions (6). Inhaled nitric oxide (iNO) can be used to improve arterial oxygen partial pressure in acute respiratory failure, and exogenous surfactant to enhance the compliance of alveoli (14, 15). Furthermore, high flow oxygen serves as an "antidote" of inhaled toxic substances, such as to hydrogen cyanide poisoning. Hydroxocobalamin is the true antidote to hydrogen cyanide, but it must be administered immediately following the inhalation injury. For CO poisoning, hyperbaric oxygen therapy has been suggested, but is not routinely recommended (9, 16).

Airway clearance is another important part of the patient's management. Pain, drugs and artificial airways are common in patients with burns and can affect their breathing and cough effectiveness (17, 18). Mucus, inactivation of surfactant, sedation and anaesthesia for surgical procedures such as burn excision of the eschar and skin grafting, increase the risk of sputum retention, atelectasis and consequently pneumonia (6). The epithelial damage

triggers goblet cells to produce higher volume of secretions or foamy mucus, thus adversely affects the mucociliary clearance, and bronchial casts sloughing increases the risk of mucus plugging (19). Use of techniques such as positioning or postural drainage, manual techniques, and manual or ventilator hyperinflation have been suggested as respiratory physiotherapy treatment options. Positioning can promote airway clearance and facilitate optimisation of the V/Q matching, therefore improve oxygenation. Postural drainage can be part of positioning. This involves a group of gravity-assisted positions that promote mucus movement downwards (towards the mouth). Special contraindications for head-down positions are facial oedema and upper airway burns, which are usually nursed in the semi-upright position with a moderate elevation of the head and trunk (5). Physiotherapists may apply manual techniques such as percussion, and any other manual handling, whilst avoiding shearing forces close to newly grafted or escharotomy areas. In chest wall burns, clinicians consider the burn scar depth, graft type, healing and viability of the tissue in the area, before performing their treatment. Moreover, manual or mechanical hyperinflation aims to promote airway clearance, maintain thoracic expansion and mobility, and prevent atelectasis (20). In patients presenting weak and ineffective cough or those who are unable to perform the manoeuvre, suction can accompany mucus mobilisation, using the nasopharyngeal, oropharyngeal, endotracheal tube or tracheostomy routes (21). The airway clearance techniques follow the treatments for pain management and nebulisation of bronchodilators or hypertonic saline, whilst proceed to mucolitics such as N-acetylcysteine, inhaled anticoagulants such as heparin, or antibiotic medications (22-24). Still, prophylactic antibiotics and corticosteroids are not indicated in this group of patients (5).

Overall, treatments aim to maximise effectiveness and avoid fatigue, and are tailored to the individual clinical presentation and potential underlying respiratory diagnosis and cardiovascular instability. Some patients can present full recovery from smoke inhalation injury without remaining long-term respiratory symptoms, although this depends on factors such as the smoke composition or the definition of smoke inhalation (25-27). Mobilisation, early rehabilitation, activities of the daily living and cardiorespiratory fitness training are important and are prioritised in various stages of the clinical pathway (28). Patients may also require psychological support (29, 30). Smoke inhalation injury is a complex clinical condition that needs careful management from a multidisciplinary team. Still, evidence for its clinical management is relatively limited, despite the increasing number of publications throughout years (Figure 1). We base our current practice mainly on retrospective cohorts and case series, rather than large-scale clinical studies (12). Is this level of evidence or the principles of physiology enough to guide most of our clinical decisions? Real life shows us that we urgently need to move towards more research and get stronger evidence for clinical application in the area.

Conflict of interest

No conflict of interest.

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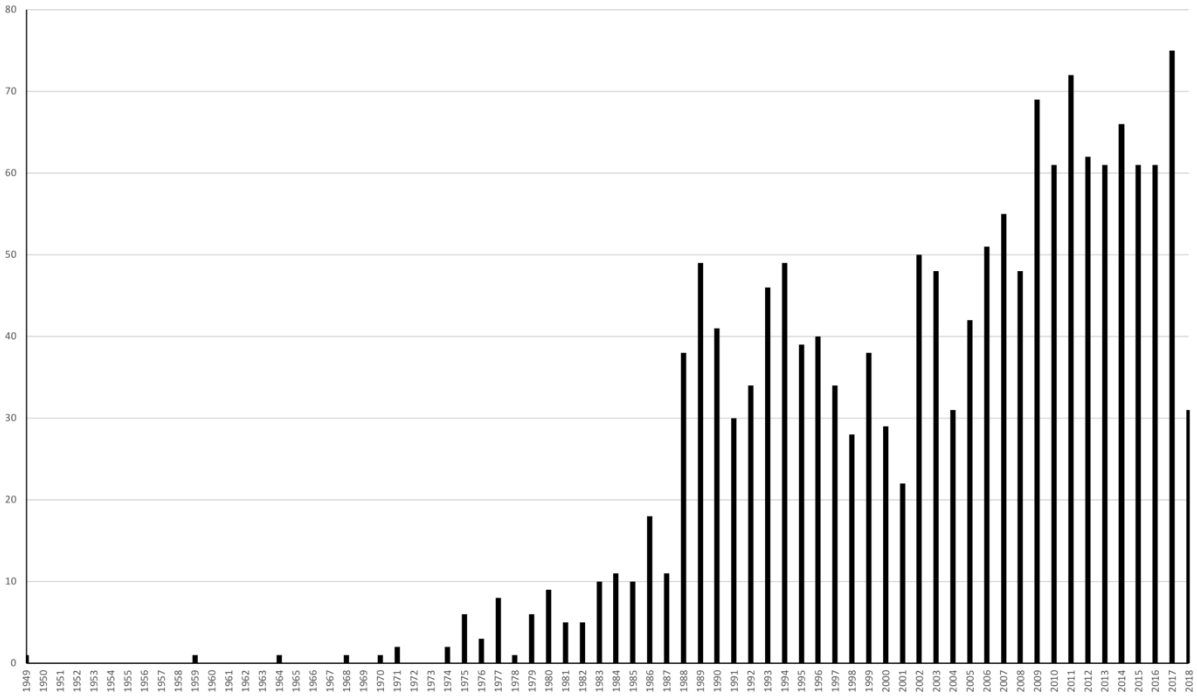

Figure 1. Number of PubMed findings per year, using the keywords “smoke inhalation injury”.
Data accessed October 2018.