NHS
London and South East
Burns Clinical Network

Burn Inhalation Injury

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DIAGNOSIS

History findings suggestive of BII

- Exposure to fire and smoke in an enclosed space
- Prolonged exposure to fire and smoke
- Loss of consciousness
- Requirement of CPR at the scene
- Known fatalities in the same incident

Examination findings suggestive of BII

- Significant facial or neck burns, including burns, blisters or oedema to the lips, tongue, oropharynx.
- Dyspnoea or accessory respiratory muscle use
- Stridor, cough, wheeze or hoarseness of voice
- Increased or carbonaceous secretions
- Altered consciousness
- Clinical hypoxaemia (SpO2 <94% on room air)

Diagnostic Tests

- Arterial blood gas with Carboxyhaemoglobin level
- Fibreoptic nasendoscopy and/or bronchoscopy if intubated.
- Diagnostic lavage and culture should be performed at bronchoscopy for all patients.
- Therapeutic lavage should be performed for moderate-severe BII only
- Chest radiograph or chest CT may play a role in prediction of injury severity and prognosis and should be performed at admission (note: may initially be normal).

Bronchoscopy

- Should be used to assess severity of BII in intubated patients within first 24 hours of injury, using the **Abbreviated Injury Score (AIS).**
- Should be used for initial therapeutic lavage for moderate-severe BII.
- Should be used for serial surveillance & therapeutic lavage in severe BII, (can consider in lesser grades).
- Note, in the absence of soot, severe vasoconstriction from hypovolaemia may initially mask injury.

MANAGEMENT

Airway Management

- Review by senior anaesthetist and consider early intubation if there is a suspicion of BII: early intubation may decrease pulmonary-related mortality in patients with inhalation injury.
- Initial intubation should be with a standard, uncut cuffed endotracheal tube of internal diameter ≥ 8.0mm.
- Chest physiotherapy daily with frequent airway clearance.
- Can consider airway adjuncts on an individual basis i.e. mucolytics (low evidence base, see Appendix 1)
- Respiratory bacterial surveillance with deep sampling (ideally bronchoscopic broncho-alveolar sampling or distal tracheo-bronchial aspirate) and appropriate antibiotic practice to be guided by clinical status and microbiological protocols (note that acute bacterial colonisation peaks 2-3 days after smoke inhalation).
- Tracheostomy should be considered in patients with BII, particularly those requiring repeated theatre trips.
- Extubate as soon as safe to do so using local protocols.
- Repeated extubation and re-intubation should be avoided.
- The use of hyperbaric oxygen therapy (HBOT) in the context of acute burn injury with smoke inhalation is no longer recommended due to logistics.

Invasive Mechanical Ventilation

- Lung protective ventilation strategies should be used (tidal volumes <6ml/kg ideal body weight, plateau pressure <30cmH2O).
- vvECMO should be considered for patients with refractory hypoxaemia, even if this requires transfer from a burns service to an alternate ICU (consider how to best manage the ongoing burns surgical and medical care if not co-located).
- Further measures including prone positioning, recruitment maneuvers, inhaled prostacyclin analogues, nitric oxide and neuromuscular blockade should be considered for patients with refractory hypoxaemia.

Pharmacotherapy

- Mild BII
  - No recommended inhaled therapy
- Moderate / Severe BII
  - 5000 IU nebulised heparin
  - Nebulised salbutamol*
  - Nebulised N-acetylcysteine*

*Beyond heparin, there is no inhaled adjunct that has been shown to have a short- or long-term benefit. This includes little evidence of benefit or harm for salbutamol, N-acetylcysteine, hypertonic saline or sodium bicarbonate. See Appendix 1 and 2.

MANAGEMENT CONT.

Frequency and course length:

- Nebulised heparin 5000 IU diluted with 3mls 0.9% saline ~4 hourly for 5-7 days (monitor coagulation daily and discontinue if APPT > 1.5).
- Nebulised salbutamol 2.5 – 5 mg ~4-6 hourly (if necessary and early withdrawal if feasible).
- Nebulised 20% acetylcysteine solution, 3mls ~4 hourly (may be irritant to airway, administer after salubutamol and should be discontinued if bronchospasm develops).
- Aim to stagger or alternate treatments to avoid prolonged gaps.

- Systemic prophylactic antibiotics (not including those at induction for surgery) and corticosteroids should not be used, regardless of BII severity.

Fluid management

- Patients with inhalation injury may require up to 40% higher fluid requirements for the same size burn, compared to those without inhalation injury.
- Fluids should be administered in response to clinical parameters and titrated to achieve physiological endpoints, not automatically increased in patients with BII.
- Excessive fluid administration should be avoided beyond resuscitation requirements.
- Cardiac monitoring (including invasive cardiac output monitoring) should be considered on an individual basis and as per local expertise.
- Urine output should not exceed 0.5-1ml/kg/hr.
- NG or NJ should be inserted on admission and feeds commenced immediately.

SYSTEMIC TOXICITY

1. Carbon Monoxide Intoxication

- Should be suspected in patients with:
  - Headache
  - Nausea
  - Irritability or agitation
  - Weakness
  - Tachypnoea
  - Dizziness or ataxia
  - Altered consciousness or coma
  - Metabolic acidosis
  - New neurological signs e.g. increased tone or upgoing plantars
  - Evidence of myocardial infarction or ischaemia
- Initial COHb gives an indication of severity of injury. Levels up to 10% may be normal in a smoker.
- Measure COHb levels if available eg from ABG
- High fractional inspired oxygen therapy should be used for treatment until CO levels <10% (if repeat testing available) or evidence of clinical improvement.

Typical clinical symptoms and signs relative to COHb (Normal = 0.5%)[9]

<10%	No symptoms, commonly found in smokers
10 – 20%	Nil or vague nondescript symptoms
30 – 40%	Headache, tachycardia, confusion, weakness, nausea, vomiting, collapse
50 – 60%	Coma, convulsions, Cheyne-Stokes breathing, arrhythmias, ECG changes
70 – 80%	Circulatory and ventilatory failure, cardiac arrest, death

2. Hydrogen Cyanide Intoxication

- Should be suspected in patients with:
  - Exposure to fire and smoke within an enclosed space
  - Metabolic acidosis (with raised anion gap)
  - Increased AA gradient (cherry red fingers)
  - Raised serum lactate (can be a marker of HCN intoxication but may be raised for other reasons)
  - Unexplained cardiac dysfunction
  - Altered consciousness
  - Seizures
  - Cardiac or respiratory arrest
- High fractional inspired oxygen therapy should be used as part of treatment.

- **Hydroxocobalamin ("Cyanokit")**
  - Hydroxocobalamin should be administered promptly and without laboratory confirmation in patients with high clinical suspicion of HCN toxicity such as heavy smoke exposure plus coma, seizures, cardiovascular collapse or significant lactic acidosis.
  - In these patients, untreated cyanide toxicity carries a very high risk of death within minutes to hours; the renal risk and lab interference are usually accepted trade-offs.
  - For hemodynamically stable, intubated burn patients with evidence of inhalation injury but normal or only mildly elevated lactate, preserved mental status before intubation or significant baseline renal impairment, the evidence does not show a survival advantage and cumulative data indicate a real increase in AKI. In this group, many centres use a more tailored approach based on lactate, GCS, haemodynamics and the extent of closed-space exposure.
  - The standard dose of hydroxocobalamin is 5g IV over 15 minutes, repeated for a second dose if there is a poor clinical response or severe toxicity.[10]
  - Monitor renal function for at least 7 days after administration given the increased risk of AKI.
  - See Appendix 3.

SYSTEMIC TOXICITY CONT.

3. **Lithium Battery Fire Fumes / Hydrofluoric Acid Vapour**

- Hydrofluoric Acid (HF) vapour is found in large amounts in the plume from some Lithium battery fires.
- There are case reports of this causing severe inhalation injury
- The toxic effects of HF are mainly due to fluoride ions, which penetrate tissues and bind intracellular calcium and magnesium, in addition to it's acidic pH.
- HF inhalation injury should be suspected in patients with:
    - Exposure to lithium-ion battery fires in enclosed space, or other source of HF vapour
    - Upper airway irritation, dyspnoea or stridor
    - Hypocalcaemia, hypomagnesaemia or hyperkalaemia
- Watch for clinical signs of hypocalcaemia including paraesthesia, tetany, seizures, diarrhoea and vomiting, decreased myocardial contractility and arrhythmias.
- High fractional inspired oxygen therapy should be used as part of treatment.
- Use continuous ECG monitoring and check plasma electrolytes frequently.
- Intravenous or topical calcium gluconate should be used for treatment of concurrent dermal HF burns as per normal protocols.

- **Nebulised 2.5% calcium gluconate** should be considered.
- Note there is limited evidence for this, but a theoretical benefit, no evidence of harm and it is recommended in many industry-based guidelines:
  1ml 10% calcium gluconate + 3ml 0.9% sodium chloride = 4ml 0.25% calcium gluconate
  This can be administered 4 hourly for 48h from the time of inhalation injury.

FOLLOW UP

- Patients should be followed up in a clinic with specific reference to the patient's respiratory status following BII, irrespective of their standard burns follow up care.
- This should be done according to clinical need and within 3-6 months.
- NICE follow up guidance recommends 2-3 months ideally.

Supporting Evidence:

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