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Asthma: Management of Severe and Life-threatening Disease

Link to Flow Sheet

Aim

The aim of this document is to provide a guideline for the management of children with a severe or life-threatening exacerbation of asthma.

Definition of terms

AnaConDa Device, Anaesthetic Conserving Device; BPM, Beats per Minute; CO, Cardiac Output; ECLS, Extracorporeal Life Support; ETT, Endotracheal Tube; PEEP, Positive End Expiratory Pressure; PICU, Paediatric Intensive Care Unit; PIP, Peak Inspiratory Pressure; RCT, Randomised Control Trial; RR, Respiratory Rate; VT, Tidal Volume; V/Q, Ventilation/ Perfusion

Target patient population

This evidence summary applies to children aged 2 to 16 years with acute, severe or life threatening exacerbation of asthma.

Target users

This guide is directed towards health-care professionals engaged in the care of critically ill infants and children.

Assessment

Medical history to identify risk factors for PICU admission and death: Previous PICU admission; previous intubation; persistent and poorly controlled disease; SpO2<91% at presentation.[1, 2]

Clinical signs correlate poorly with the severity of airways obstruction.

Initial acute

SEVERITY[3,4]	AGE		
	< 5 years		> 5 years
Severe	SpO2 < 92%		SpO2 < 92%
	Too breathless to talk		Too breathless to talk
	Too breathless to feed		
	RR > 50		RR > 30
	Pulse > 130bpm		Pulse > 120bpm
	Significant respiratory distress		Use of accessory muscles
Life threatening	**ANY AGE**	Silent Chest	Fatigue or exhaustion
	Cyanosis	Poor respiratory effort	Agitation or reduced level of consciousness

Referral to intensive care

General indications for referral include a patient requiring ventilatory support and those with severe acute or life threatening asthma who are failing to respond to appropriate therapy. However, data to support specific criteria for referral to intensive care services are lacking.[5]

Investigations

All children with severe or life-threatening asthma require continuous SpO2 monitoring, a CXR (to identify collapse, consolidation, pneumothorax) and a blood gas (a normal or raised PCO2 suggests worsening disease).[3]

Management

First line

- **Supplemental O2** to maintain SpO2 94-98%
- **Salbutamol Nebule** 2.5mg if <6y or 5mg if >6y combined with Ipratropium Bromide Nebule 250micrograms and given every 20 min - 30 min for three doses. (250 micrograms/dose mixed with 2.5mg or 5 mg of salbutamol solution in the same nebuliser) Thereafter, wean Salbutamol to 1-2hrly and Ipratropium to 4-6hrly.[3]
- **Systemic steroids** should be given early. Intravenous Hydrocortisone 4mg/kg (max. 100mg per dose) 6hrly initially then convert to enteral Prednisolone 1mg/kg/day (max dose 60mg). A 3 day course is usually sufficient. There is no need to wean unless course exceeds 14 days. Do not use nebulised steroids in place of systemic steroids.[3]

Second line

- **Fluid bolus** of 10ml/kg IV (0.9%w/v Sodium Chloride or Compound Sodium Lactate Solution) should be considered and repeated as required to achieve euvolaemia (aim is to improve V/Q match and CO).

- **Magnesium Sulphate**
    - Magnesium sulphate injection 50% w/v 0.2mmol/kg (50mg/kg) Max dose 8mmol (2g) intravenous infusion over 20min. Monitor BP closely.[3, 6, 7, 8]
    - Evidence for the use of nebulized Magnesium Sulphate are lacking.[3, 9]

- **Aminophylline**
    - Load with 5mg/kg intravenously over 20min (unless on maintenance oral theophylline) with ECG monitoring. Continuous infusion: 1 month – 9 years: 1mg/kg/hour by continuous IV infusion.
      9 – 16 years: 0.7mg/kg/hour by continuous IV infusion. Consider reducing to 0.5mg/kg/hour after 12 hours of treatment.
      16 – 18 years: 0.5mg/kg/hour by continuous IV infusion.
    - Monitor serum levels in those already receiving oral theophylline treatment or if duration of treatment prolonged.[3, 10, 11]

- **Salbutamol (intravenous)**
    - Load with 5 micrograms/kg/min for 1h followed by continuous infusion at a rate of 1-5micrograms/kg/min. Continuous ECG monitoring and at least 12 hourly electrolytes. Data supporting continuation of nebulised Salbutamol are weak.[12, 13]

- **High Flow Nasal Cannula** at a flow rate 2L/kg/min have shown to improve work of breathing in observational studies but further research and clinical trials are needed.[14, 15, 16]

- **Non-invasive positive pressure ventilation** of PEEP 5 and PS 8cmH2O has been demonstrated to reduce clinical asthma score and improve oxygenation.[17, 18] Judicious use of sedation may be required. Ensure NG sited and on free drainage. In-line nebulisation is preferable to interruption of NIV.

Rescue therapies

- **Adrenaline** nebulised does not appear to confer benefit over nebulised selective beta 2 agonists.[19] In the setting of acute asphyxial asthma **IM adrenaline 1:1000 10micrograms/kg** should be considered as **anaphylaxis** may be the underlying mechanism.[20] Its use as an adjunct in severe paediatric asthma exacerbations outside this setting is currently the subject of a RCT.[21] Investigate suspected anaphylaxis as per hospital guidelines.

- **Ketamine**. Use should be reserved for those with expertise in airway management and under continuous observation. A Ketamine bolus (1-2mg/kg) followed by infusion (15-33 microgram/kg/min or 1-2mg/kg/h) to improve clinical measures of severity is supported by poor quality data.[22,23] In a single RCT lower dose Ketamine (bolus of 0.2mg/kg followed by infusion of 8 microgram/kg/min or 0.5mg/kg/h) in addition to "standard therapy" did not prove beneficial.[24]

- **Magnesium Sulphate** by continuous infusion may be administered targeting a serum Magnesium of 2.5mmol/L.[4] However, we recommend this target is more safely achieved by repeated boluses if desired.

- **Intubation** is required based on a clinical judgement of progressive fatigue, worsening hypoxia or following a respiratory arrest. Intubation of such patients is high risk and should ideally be performed by a Consultant Intensivist or Anaesthetist. The approach to airway management and ventilation is dictated by the pathophysiology of air trapping leading to V/Q mismatch and reduced cardiac preload. A fluid bolus should be administered. Rapid sequence induction using **Ketamine** 2mg/kg (bronchodilator) with **Midazolam** 100microgram/kg (to prevent dysphoria) and Suxamethonium 1-2mg/kg is recommended.[25] However, we typically use non-depolarising muscle relaxants. **Rocuronium**, **Vecuronium**, **Pancuronium** and **Cisatracurium** are considered safe, while Atracurium and Mivacurium are associated with histamine-mediated bronchoconstriction. Use a **cuffed ETT** as high PIP may be required. Once the airway is secure a sedative infusion should be commenced. Once sufficiently sedated most patients will not require a continuation of paralysis.

- **Invasive Ventilation**. The goals of ventilation are to reverse hypoxia and rest the respiratory muscles whilst maintaining adequate CO through avoidance of hyperinflation. A strategy of **permissive hypercapnia** (pH > 7.2) with low tidal volumes (5-7ml/kg) and respiratory rates (15-20 BPM) and long expiratory times is optimal.[26,27] Sodium Bicarbonate 1mmol/kg may prove beneficial even in the setting of hypercapnia.[28] The application of PEEP in the absence of spontaneous patient effort is not widely advocated due to the risk of worsening of air-trapping, however, some may benefit.[29,30] A PEEP just below the level of intrinsic PEEP can decrease the work of breathing and improve synchrony for the spontaneously breathing patient.[31] There are no compelling data to support a particular mode of ventilation. However, PRVC has theoretical advantages due to the high initial flow rates and ability to limit pressure whilst delivering consistent VT's.[25] Pressure should be limited initially to < 35cmH2O, however due to high airway resistance greater Paw may be required and PIP's up to 50cmH2O have been safely applied.[26]

- **Inhalational Anaesthetics**. Volatile anaesthetic agents exert a direct dilatory effect on airway smooth muscle and inhibit release of inflammatory mediators.[32] There use in the paediatric population is supported by case series in which **Sevoflurane** delivered with a peak concentration ranging from 1 to 8% and **Isoflurane** delivered at 0.5 to 1.5% have been demonstrated to be effective and safe. [33,34,35] Hypotension is common and pressor support is likely to be required.[35]
- **Helium/Oxygen** or Heliox may improve pulmonary function in patients with the most severe acute asthma but aside from one study in children,[36] there is little evidence base to support its use.[37 - 40] Heliox's use as a rescue agent in intubated patients requiring high pressures is supported by case series.[41]

- **ECLS**. Extracorporeal support has been utilised for those with asthma refractory to conventional measures with survival rates >90%. Veno-Venous ECLS has potential advantages over Veno-Arterial.[42] ECLS should be considered where severe respiratory acidosis persists despite optimal medical management and there is failure to respond to less invasive rescue therapies.

Special Considerations

- Delivery of Heliox will require the early involvement of clinical engineering.

- Delivery of Isoflurane using an AnaConDa device is optimal for longer term therapy, however, Sevoflurane may be delivered via an anaesthetic machine as a bridge or where an AnaConDa device is unavailable.

Companion Documents

- Link to nursing guideline on the set up and management of the AnaConDa Device
- **Link to SOP for delivery of Heliox via the Maquet Servo I (Pending)**
- **Link to Parent Information Sheet (Pending)**
- Link to References
- Link to Literature Search Strategy

Links to useful websites

www.picu.ie

www.nasccrs.ie