Anaphylaxis Identification And Management

Anaphylaxis is a severe reaction within the spectrum of generalised immediate type hypersensitivity reactions. In its most critical form it is characterised by life threatening upper airway obstruction, bronchospasm and severe hypotension.

Clinical Presentation

Recognition

Table 1. Clinical Features of Anaphylaxis (Adapted from Brown 2006[1])

General	Anxiety, malaise, weakness, paresthesia, dry mouth
Cutaneous	Nasal congestion, rhinorrhoea, conjunctival erythema, tearing, itch, flushing, urticaria, angioedema
GIT	Nausea, vomiting, abdominal pain, diarrhoea
Respiratory	Upper airway oedema (difficulty speaking, swallowing, hoarse voice, stridor), chest or throat tightness, dyspnoea, bronchospasm, cough, hypoxaemia
CVS	Tachycardia associated with vasodilatation and hypotension, diaphoresis and circulatory failure, arrhythmias, cardiogenic shock and pulmonary oedema, cardiac arrest
CNS	Headache, dizziness, confusion, loss of consciousness

Most adult patients experiencing anaphylaxis will have some skin manifestation of mediator release. Can be transient however and some studies report they were lacking in 20% of cases[2,3]. Absent skin features may occur more frequently in the paediatric population where respiratory features predominate[3]. In adults sudden cardiovascular collapse and shock may occur prior to a rash[2,4].

CVS collapse is present in 90% of anaphylaxis cases, but may be the sole feature in 10%. Arrhythmias are common, supraventricular tachycardias being the most common. Cardiac arrest occurs in 11% of cases, with pulmonary oedema occurring in 3% (may be the sole feature). Initially there is an increase in cardiac output secondary to catecholamine release, which then leads to profound hypotension after catecholamine depletion.

Bronchospasm is present in 50% of cases and the sole feature in 3%. 12% have upper airway oedema. Bronchospasm may be the most difficult symptom to treat.

Anaphylaxis must be considered as a differential for any acute onset bronchospasm, respiratory distress, hypotension and cardiac arrest[1].

Differential Diagnosis

Other causes of shock need to be considered in the hypotensive patient. Other causes of rashes include acute and chronic urticaria, post viral syndromes. Isolated angioedema may be due to hereditary or acquired C1

esterase deficiency or induced by blockade or deficiency of angiotensin converting enzyme. Other conditions, which may be confused with anaphylaxis, include Scombroid fish poisoning, dystonic reactions and panic attacks[1].

Patterns of Organ Involvement

Lethal reactions to food occur at a median age of 22-24 years, have predominantly a respiratory component and appear to be more common in patients with asthma[5-7]. In contrast lethal reaction to insect venoms and drugs occur at median ages of 55-67 years, with cardiovascular collapse more likely to be the sole feature[6].

Severity Grading

Table 2. Severity Grading for Anaphylaxis (Adapted from Brown 2006[1])

Grade	Defined By
Mild (skin and subcutaneous tissue)	Generalised erythema, urticaria, periorbital oedema or angioedema
Moderate (features suggesting respiratory, CVS or GIT involvement)	Dyspnoea, stridor, wheeze, nausea, vomiting, dizziness, diaphoresis, chest or throat tightness, or abdominal pain
Severe (hypoxia, hypotension or neurological compromise)	Cyanosis or $SpO_2 \leq 92\%$, hypotension (SBP<90 mmHg in adults), confusion, collapse, LOC or incontinence

The above table helps to grade the severity of the reaction and correlates well with the use of adrenaline in the moderate and severe groups.

Aetiology

Table 3. Adapted from Brown 2006[1]

Venomous stings and bites	Ants, bees, wasps
Drugs	Penicillins, cephalosporins, cotrimoxazole, NSAIDs, narcotics, radiological contrast, ACE inhibitor, vaccines, gelofusin
Food	Sea food, nut, egg, monosodium glutamate, kiwi fruit
Idiopathic	
Other	Exercise induced, latex

Management (Adapted from Brown 2006[1])

Step 1

- Stop precipitant
- Assess reaction severity
- Call for help and 000 if required
- Give adrenaline IM (lateral thigh) 0.01mg/kg up to 0.5mg (i.e. up to 0.5 mls of 1:1000 or 5mls of 1:10000)
- Lie patient flat and elevate legs
- High flow oxygen, airway/ventilation support if required including intubation
- If hypotensive, insert large bore IV and give bolus of 20ml/kg of normal saline IV stat.

Step 2

- If there is an inadequate response or deterioration give further doses of adrenaline IV every 3-5 minutes as required (50 – 100mcg i.e. 0.5-1ml of 1:10000)
- **If the patient is arrested give 1mg increments (1ml of 1:1000, or 10mls of 1:10000) and commence Advanced Life Support**
- If the patient has bronchospasm consider giving continuous salbutamol nebulisers and or nebulised adrenaline (5mls of 1:1000)
- If the patient continues to be hypotensive give further boluses of fluid (10-20mls/kg up to 50mls/kg)
- Give atropine 0.02mg/kg if the patient has severe bradycardia
- Give IV glucagon if the patient is β-blocked and hypotensive (load with 1-5mg over 5 minutes)
- Give IV hydrocortisone 5mg/kg 6 hourly followed by oral prednisone 1mg/kg (max 50mg) for 4 days

Step 3

- All patients need transfer and observation in hospital for at least 4 hours
- Will need a serum mast cell tryptase on arrival to hospital
- Should be followed up by an immunologist and advised to carry an EpiPen and wear a Medic Alert bracelet

Antihistamines

H_1 blockade appears to be useful for mild allergic reactions confined to the skin. However there are no published trials examining utility during anaphylaxis. Histamine levels peak early then return rapidly to normal, suggesting that there may not be any benefit from antihistamines. One study found a small benefit of combined H_1 and H_2 blockade over H_1 blockade alone in mild allergic reactions. In a rat model pre-treatment with H_1 receptor

blockade with or without concurrent H_2 blockade worsens hypotension and decreases survival time[1]. Since in Australia the only IV formulation is promethazine, which is a potent vasodilator, it would be prudent to avoid it.

For the present non-sedating antihistamines should be limited for symptomatic relief of skin symptoms.

Steroids

There are no clinical trials of steroids in the treatment of anaphylaxis. Current recommendations for their use in brochospasm have been extrapolated from their use in asthma[8].

References

1. Brown SGA. Anaphylaxis: Clinical concepts and research priorities. Emergency Medicine Australasia (2006) 18, 155-169
2. Brown SGA. Clinical features and severity grading of anaphylaxis. J. Allergy Clin. Immunol. 2004; 114: 371-6
3. Braganza SC, Acworth JP, McKinnon DRL, Peake JE, Brown AFT. Paediatric emergency department anaphylaxis: different patterns from adults. Arch. Dis. Child. 2006; 91: 159-63
4. Fisher MM. Clinical observations on the pathophysiology and treatment of anaphylactic cardiovascular collapse. Anaesth. Intensive Care 1986; 14: 17-21
5. Pumphrey R. Anaphylaxis: can we tell who is at risk of a fatal reaction? Curr. Opin. Allergy Clin. Immunol. 2004; 4: 285-90
6. Pumphrey RS. Lessons for management of anaphylaxis from a study of fatal reactions. Clin. Exp. Allergy 2000; 30: 1144-50
7. Sampson HA, Mendelson L, Rosen JP. Fatal and near fatal anaphylactic reactions to food in children and adolescents. NEJM. 1992; 327: 380-4
8. Emergency medical treatment of anaphylactic reactions. Project Team of the Resuscitation Council (UK). Resuscitation 1999; 41: 93-9