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 2006¹)

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

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 cases²,³. Absent skin features may occur more frequently in the paediatric population where respiratory features predominate³. In adults sudden cardiovascular collapse and shock may occur prior to a rash²,⁴.

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 arrest¹.

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.

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. 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.

Severity Grading

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

<table>
<thead>
<tr>
<th>Grade</th>
<th>Defined By</th>
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<tbody>
<tr>
<td>Mild (skin and subcutaneous tissue)</td>
<td>Generalised erythema, urticaria, periorbital oedema or angioedema</td>
</tr>
<tr>
<td>Moderate (features suggesting respiratory, CVS or GIT involvement)</td>
<td>Dyspnoea, stridor, wheeze, nausea, vomiting, dizziness, diaphoresis, chest or throat tightness, or abdominal pain</td>
</tr>
<tr>
<td>Severe (hypoxia, hypotension or neurological compromise)</td>
<td>Cyanosis or SpO₂ ≤ 92%, hypotension (SBP&lt;90 mmHg in adults), confusion, collapse, LOC or incontinence</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Venomous stings and bites</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ants, bees, wasps</td>
<td>Penicillins, cephalosporins, cotrimoxazole, NSAIDs, narcotics, radiological contrast, ACE inhibitor, vaccines, gelofusin</td>
</tr>
<tr>
<td>Food</td>
<td>Sea food, nut, egg, monosodium glutamate, kiwi fruit</td>
</tr>
<tr>
<td>Idiopathic</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>Exercise induced, latex</td>
</tr>
</tbody>
</table>
Management (Adapted from Brown 2006¹)

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

H₁ 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 H₁ and H₂ blockade over H₁ blockade alone in mild allergic reactions. In a rat model pre-treatment with H₁ receptor...
blockade with or without concurrent $H_2$ blockade worsens hypotension and decreases survival time. 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.

**References**