



## Case 4.1 Wasp venom anaphylaxis

A 69-year-old woman was fit and well until one August when she was stung on the back of her right hand by a wasp. She had previously been stung on several occasions, the last time 2 weeks earlier. Within 5 min, she felt faint, followed shortly by a pounding sensation in her head and tightness of her chest. She collapsed and lost consciousness and, according to her husband, became grey and made gasping sounds. After 2–3 min, she regained awareness but lost consciousness immediately when her husband and a friend tried misguidedly to help her to her feet. Fortunately, a doctor neighbour arrived in time to prevent her being propped up in a chair: he laid her flat, administered intramuscular epinephrine (adrenaline) and intravenous antihistamines and ordered a paramedic ambulance. She had recovered fully by the next day.

She had *clinical wasp venom anaphylaxis*. Her total serum IgE was 147 IU/ml (NR <120 IU/ml). Her antigen-specific IgE antibody level to wasp venom was 21 U/ml [radioallergosorbent test (RAST) class 4], but that to bee venom was 0.3 U/ml (RAST class 0). The patient was a candidate for specific allergen injection immunotherapy (hyposensitization). The slight but definite risk of desensitization was explained and balanced against the major risk of anaphylaxis should she be stung again. The first injection consisted of 0.1 ml of 0.0001 µg/ml of wasp venom vaccine given subcutaneously. No reaction occurred. Over the next 12 weeks, gradually increasing doses were given without adverse effects. Over this period, she tolerated injections of 100 µg venom. She then continued on a maintenance regimen of 100 µg of venom per month for 3 years.

At the age of 76 years, she was stung by a wasp that had come into her bathroom. She remained calm, lay down and experienced no significant systemic reaction.



**Case Figure 4.1** Although a local reaction did not occur in this case, they are common. This image shows localized reaction after venom desensitization.



## Case 4.2 Latex-induced anaphylaxis

A 38-year-old woman was referred for investigation following an anaphylactic reaction whilst visiting a relative in hospital. She gave a 5-year history of recurrent conjunctival oedema and rhinitis when blowing up balloons for her children's birthday parties. In the year prior to admission, three successive visits to her dentist triggered marked angioedema of her face on the side opposite to that requiring dental treatment. The swellings took 48 h to subside.

On the day of admission, she visited a critically ill relative in hospital. The patient was being reverse barrier nursed and visitors were required to wear gown and gloves. About 20 min after putting on the gloves her face and eyes became swollen, she felt wheezy and developed a pounding heart beat and light-headedness. Her tongue started to swell and she was taken to the Emergency Department where she was given intramuscular epinephrine (adrenaline) and intravenous hydrocortisone (inappropriately as it transpired). She recovered rapidly but was kept under observation overnight.

She had no history of atopy or other allergies. Ten years earlier she had undergone a series of operations for ureteric reflux and in the preceding 2 years had received colposcopic laser treatment for cervical intra-epithelial neoplasia (CIN-III).

Skin-prick testing to a crude latex extract produced a very strong reaction and her antigen-specific IgE antibody level to latex was significantly elevated at 57 U/ml (RAST class 5).

The diagnosis was that of *latex-induced anaphylaxis*. She was advised to avoid contact with all materials containing latex, and warned that she could react to certain foods (see Table 4.3). It was suggested that she wears a medical alert bracelet, in case she required future emergency surgery, and carry a self-injectable form of epinephrine. The diagnosis has important implications for any further dental, surgical or anaesthetic procedures. Hospitals now have written procedures for latex-free surgery for emergency and planned operations.

**Table 4.3** Key features of latex rubber allergy

### *High-risk groups*

- Patients with spina bifida or multiple urological procedures (10–50% risk)
- Healthcare workers (5–10% risk)
  - Operating theatre staff
  - Females
  - Atopics
- Rubber industry workers (5–10% risk)

### *High-risk latex products*

- Surgical latex gloves
- Latex rubber gloves for home use
- Balloons
- Catheters and enema tubes
- Condoms
- Teats and dummies (pacifiers)

### *Cross-reactivity with food allergies*

- Kiwi fruit, banana, avocado, melon, chestnut



**Case Figure 4.2a** Medic Alert™ bracelet.



**Case Figure 4.2b** EpiPen.



### Case 4.3 Drug-induced reaction

A 77-year-old woman was referred from the Accident and Emergency Department, having been admitted overnight because of sudden onset of massive angioedema of her tongue associated with laryngeal stridor. She was treated with intravenous hydrocortisone only. This was her fifth such episode: an anaphylactoid attack 2 months earlier was severe enough for her to be intubated and ventilated on the Intensive Care Unit.

She had no history of previous allergy and no family history of atopy. A drug history revealed that, in addition to oral prednisolone prescribed in the Accident and Emergency Department, she was taking oral furosemide and captopril. Captopril is an angiotensin-converting enzyme (ACE) inhibitor and this group of drugs is known to cause severe episodes of angioedema. Captopril was discontinued and her mild hypertension was managed with alternative medication. The attacks have not recurred.

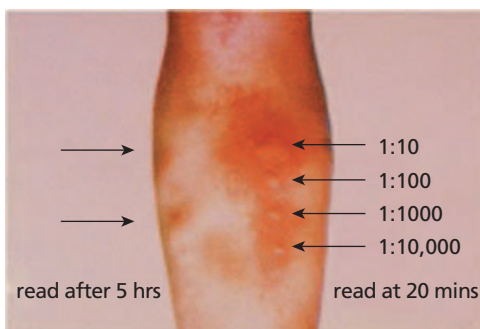
#### Case 4.4 Seasonal allergic conjunctivitis

A 7-year-old boy developed itchy eyes and swollen lids after playing tennis in the garden. Because his mother had hay fever, the boy's symptoms were also presumed to be an allergy to grass pollen. After several episodes of increasing severity, medical help was sought. He was skin tested; a weal-and-flare reaction appeared 5–15 min after prick testing with extracts of grass pollens, cat fur and house dust mite. The speed and nature of the reaction confirmed *immediate (type I) hypersensitivity* to these antigens, and he was told to try to avoid exposure to high concentrations of grasses in the pollen season. He developed similar reactions the following summer, particularly in June and July; they were sometimes accompanied by sneezing and rhinorrhoea. He was therefore started on prophylactic eye drops containing sodium cromoglycate, which helped control his *seasonal allergic conjunctivitis*.

#### Case 4.5 Perennial allergic rhinitis

A 29-year-old doctor developed intense itching of her eyes and nose and a tickling sensation in her ears and palate, followed by sneezing and rhinorrhoea. These symptoms developed within 15 min of visiting an elderly patient who kept four cats. The symptoms settled down over the next 2 h but started to recur whenever home visits were made to houses where cats were present. Symptoms occurred even though the cats were excluded from the interview room. Each episode took slightly longer to resolve and some were accompanied by a dry cough.

The doctor had suffered from asthma in childhood and her non-atopic parents had a cat. During her years in medical school and in hospital posts, she had no respiratory symptoms. The move into general practice and exposure to cat dander had triggered perennial allergic rhinitis. On investigation, she had strongly positive skin tests to cat dander, house dust mite and grass pollen. She started prophylactic use of a nasal spray and eye drops containing sodium cromoglycate, with abolition of most of her symptoms. Sometimes, she also needed to use a local antihistamine spray to relieve breakthrough attacks of rhinitis. The value of hyposensitization (antigen-specific immunotherapy) to cat dander was discussed because occupational exposure was unavoidable, but not undertaken while her symptoms continued to be controllable. It remains a therapeutic option.



**Case Figure 4.5** Skinprick tests with grass pollens. Roitt & Rabson.

#### Case 4.6 Allergic asthma

A 15-year-old girl presented with a prolonged wheezing attack which had come on suddenly 36 h earlier. She had experienced several episodes of 'wheezy bronchitis' as a child and eczema as an infant. She was a non-smoker. Her father suffered from hay fever but there was no family history of asthma. On examination, she was tired and unwell, with a rapid respiratory rate and tachycardia (140/min). There were bilateral expiratory wheezes on chest auscultation. Investigations showed a normal haemoglobin but a raised white cell count ( $14 \times 10^9/l$ ). A chest X-ray was normal but lung function tests showed reversible airways obstruction. The clinical diagnosis was asthma and the family history and skin tests later suggested this was *allergic asthma*. She continues to have periodic attacks of asthma, although they are controlled, in part, by prophylactic inhaled steroids and  $\beta_2$ -adrenergic stimulants (salbutamol) as needed.



### Case 4.7 Oral allergy syndrome

A 40-year-old man knew he had longstanding 'hay fever', although his symptoms were worse in March and April each year rather than in summer months. For the previous 4 years he had noticed that eating certain fruits, particularly apples, pears and peaches, produced tingling, burning and swelling of his lips and gums. These symptoms occurred within seconds of starting to eat these fruits and lasted about 30 min, but were never associated with vomiting, urticaria, bronchospasm or circulatory collapse. He found that he could eat cooked or preserved apples without any reactions. He was worried that these reactions heralded an increasing potential to develop anaphylaxis to fruit.

He was skin-prick tested to a variety of allergens: he showed strongly positive reactivity to tree pollen and peach but a negative reaction to the commercial apple solution. However, when the skin test lancet was first pricked into a fresh apple and then into the patient's skin – so-called 'prick-prick' testing – a strongly positive immediate reaction developed.

He has the *oral allergy syndrome*.



### Case 4.8 Nut allergy

A 15-year-old schoolgirl was admitted to hospital as an emergency whilst on holiday. Her parents believed her to be allergic to nuts. At the age of 5 years, she vomited about 1 min after eating a bar of chocolate containing nuts. Three years later, she developed marked angioedema of her face, lips and tongue, followed by tightness of her throat and vomiting: this occurred 2–3 min after friends of her brother decided to test her allergic status by pushing peanuts into her mouth and holding her jaws shut! Less severe attacks had followed inadvertent ingestion of hazelnuts and almonds. As a consequence, she avoided peanuts and tree nuts wherever possible.

The emergency admission occurred following a single lick of a vanilla ice cream. Within seconds, she developed angioedema of her lips and tongue, difficulty in breathing, and felt light-headed. Following an emergency call, she was injected with intramuscular adrenaline (and intravenous hydrocortisone inappropriately) by the paramedical service, and admitted to hospital overnight. She made a rapid and uneventful recovery. Her parents later recalled that one ice-cream scoop was used by the vendor to dispense all flavours: the customer immediately before the patient had been served a nut-flavoured ice cream.

On investigation, she had a grade 6 RAST (see Chapter 19) to peanut with significant but lesser (grade 2) reactivity to hazelnut, almonds and brazil nuts. She was also atopic, with strongly positive RASTs to grass pollen (grade 4) and cat dander (grade 3).

The management of her *nut allergy* comprised advice on strict avoidance of peanuts and tree nuts, with particular attention to 'hidden' nuts in food. She was advised to wear a medical alert bracelet as a warning to emergency personnel of a possible cause of sudden collapse, and to carry with her at all times a self-injectable form of epinephrine (adrenaline).



Case Figure 4.8a Medic Alert™ bracelet.



Case Figure 4.8b EpiPen.



### Case 4.9 Is this food allergy?

A 38-year-old woman presented with a 2-year history of abdominal bloating, cramping abdominal pains and loose stools. Attacks occurred every 7–10 days but lasted only 2–3h. One attack occurred about 8h after a meal of pasta in a local restaurant and led her to believe her symptoms were food-related. She initially eliminated wheat-based products from her diet and then, because the attacks continued, dairy products as well. She was referred to a gastroenterologist and investigated extensively: gastroduodenal endoscopy, duodenal biopsy, barium meal, colonoscopy and pancreatic function tests were all normal.

She continued to believe her symptoms were food related: vague muscular pains, headaches, poor concentration and fatigue were attributed to other foods, which were then eliminated from her diet. She was referred to an allergy clinic for further assessment.

Physical examination showed an undernourished woman with no other abnormal findings. As this was a second opinion, a wide range of tests were done. Her haemoglobin and erythrocyte sedimentation rate were normal. Her serum immunoglobulins were normal and her serum IgE was only 8IU/ml. She had no detectable antibodies to tissue transglutaminase, endomysium or gliadin (see Chapter 19). While waiting for her outpatient appointment, she had responded to an advertisement in a health food shop and undergone electrodermal or 'Vega' testing. The report listed 24 foods to which she was allergic, many of which she had felt able to tolerate previously. Her diet had become increasingly restricted: expert dietetic assessment showed her diet to be nutritionally unsound, with deficient intake of protein, fat, fat-soluble vitamins and trace elements. The diagnosis was that of *psychological food aversion* and irritable bowel syndrome. She was reluctant to accept this diagnosis and asked her GP for referral to another specialist.



## Case 4.10 Chronic urticaria and angioedema

A 25-year-old joiner presented with a 12-month history of an intensely itchy 'nettle rash' on his chest and back (see Fig. 4.9). The lesions appeared suddenly and lasted for 6 to 12h, to be replaced by new lesions at other sites. The lesions varied in size from a few millimetres to several centimetres. Attacks occurred two to three times each week. In addition, he had experienced four episodes of sudden swelling of lips that took 48h to subside. He said he looked as though he had been punched (see Fig. 4.10). He was unaware of any triggering factors and there was no personal or family history of atopy. His general health was excellent and he was not taking any medications. On examination, the lesions consisted of raised, red, irregular patches, some with white centres, and were typically urticarial. General examination was entirely normal.

Laboratory investigations showed a normal haemoglobin and white cell count, with no eosinophilia. His complement C4 and C1 inhibitor levels were normal, excluding hereditary angioedema (see Case 11.5).

The urticaria was fairly well controlled by a long-acting antihistamine (levocetirizine) but he was reluctant to take these tablets on a long-term basis. Three years later, his urticarial lesions are still present, although less severe; their cause is unknown.

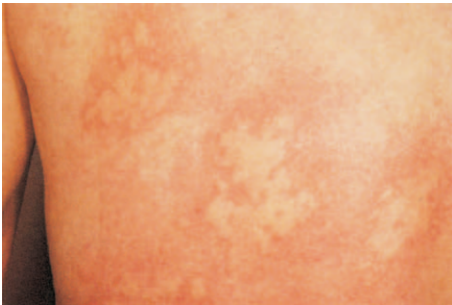


Fig. 4.9 Typical urticaria.



Fig. 4.10 Angioedema of the lower lip.



Case Figure 4.10 Urticaria – marked wheals with surrounding erythema.



## Case 4.11 Atopic eczema

Sam was born at full term, after a normal pregnancy, and weighed 3.4kg. He was breast-fed. At the age of 4 weeks, he was admitted with a 2-day history of screaming attacks, loose motions and rectal bleeding. He was treated conservatively, but 3 days after discharge his symptoms recurred, together with patches of eczema on his arms and trunk. On detailed questioning, it transpired that a health visitor had told Sam's mother that her breast milk was of 'poor quality' and had advised her to 'top up' each feed with cow's milk. His mother had been following this advice from the time Sam was 2 weeks old. When investigated at the age of 6 weeks, strongly positive IgE-specific antibodies to cow's milk were present on RAST testing. His mother returned to exclusive breast-feeding and excluded dairy products from her own diet, to eliminate any possibility that cow's milk antigens might be excreted in her breast milk. Within 2–3 days, the screaming attacks stopped.

At the age of 10 months he was referred back to hospital with extensive atopic eczema. It had recurred behind his knees at the age of 7 months, when solids were first introduced into his diet, and steadily worsened. The areas affected were the popliteal and antecubital fossae, arms and abdomen. He scratched the eczematous lesions, especially at night, with the result that his and the family's sleep was badly disturbed. Sam had a strong family history of atopic disease; his mother and maternal grandmother both suffered from asthma. On examination, his height and weight were around the 50th centile. He was covered in extensive eczema, involving 60% of the total skin area.

Laboratory investigations showed a normal haemoglobin (123g/l) with a raised white cell count ( $16.0 \times 10^9/l$ ) including an absolute ( $650/mm^3$ ) eosinophilia. His total serum IgE level was markedly raised at 4600IU/ml (NR for age <50IU/ml) with strongly positive RASTs to grass pollen, cat epithelium, dog dander, house dust mite, cow's milk, wheat and peas. Skin-prick testing was not considered in the presence of such widespread eczema. Samples of dust from his home showed very high levels of house dust mite in the carpet and on several toys.

He was treated with antihistamines at night and liberal applications of an emollient cream to his skin lesions. Environmental control of antigen exposure was also attempted: the mite count was lowered by changing carpets, and covering the mattress, pillows and duvet with covers impermeable to mite allergens, and the cat was found a new home. Sam was put on a diet free of cow's milk, wheat, oats, peas, beans, nuts, food preservatives and food colourings. Over the following 3 months, there was only partial improvement in the severity of his eczema and topical pimecrolimus was then used, successfully (see Chapter 7).



Case Figure 4.11 Eczema on trunk of male infant.