Guidelines for referral of patients to the allergy clinic

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ANAPHYLAXIS

Any patient with a history of anaphylaxis\(^1\) should be referred to the clinic.

\(^1\): This implies that the patient has suffered from an acute collapse with any combination of bronchospasm, laryngeal / pharyngeal oedema or hypotension. This is usually, but not always, accompanied by other visible features such as flushing, urticaria, or angioedema.

URTICARIA AND ANGIOEDEMA

Urticaria is very common and angioedema is often an extension of this. The aims of these guidelines are:

- to avoid referral before simple measures have been tried
- to avoid unnecessary investigation, while promoting the few relevant investigations that might alter management
- to promote referral of refractory cases, or cases with pathology that needs specialist input

Background and general principles: allergic or non-allergic?

Urticaria (hives, weals or ‘nettle rash’) and angioedema (soft tissue swelling) can occur alone or together. They can be allergic or non-allergic in aetiology. Contrary to popular perception, these symptoms are very commonly non-allergic and, in that situation, investigation is usually unrevealing and prolonged maintenance treatment is often necessary.

Many patients with urticaria and/or angioedema are referred as cases of possible food allergy ‘to find out what they are allergic to’. In the majority of cases, food allergy is not the cause and can be excluded on the basis of the clinical history alone.

Features suggesting that symptoms are not due to allergy:

- there is no consistent relationship to a particular allergen trigger\(^2\), or the timing is not right for allergy\(^3\)
- symptoms are spontaneous, without any apparent triggering factor, or come on overnight or first thing in the morning (before the patient has eaten anything)
- symptoms have physical triggers, such as minor trauma or local pressure, temperature change, sweating or exposure to water
- the patient develops angioedema while on treatment with an ACE inhibitor\(^4\)
Based on these principles, you will be able to decide whether your patient’s symptoms are likely to be allergic or non-allergic. Having made this initial decision, you will be able to access the appropriate advice on management (below).

2: An important exception is urticaria / angioedema / anaphylaxis due to an unusual wheat allergy, in which exercise is an important cofactor for symptoms (discussed further in the recommendations below). In this situation, because wheat will often be tolerated in the absence of exercise, patients may not make the connection between wheat consumption and symptoms.

3: Patients frequently examine what foods they have eaten the previous day in the search for a connection with their symptoms. However, this is largely irrelevant as, in genuine food allergy, symptoms usually occur within an hour of exposure to a single food, or well-defined group of foods (a delay of more than 2 hours is rare, and more than 4 hours is very rare).

4: Angioedema is a well-documented adverse side-effect of ACE inhibitors, can occur for the first time even after prolonged treatment, and persist for several weeks after the treatment is withdrawn.

Suggestions for management

The following management plans are suggested for patients presenting with urticaria/angioedema.

When the history does not suggest allergy:

Decide where your patient lies on the following flow chart and then refer to the appropriate set of recommendations below (each is on a separate page, so that you can print off only the recommendations that apply to your patient).
SET 1: patients with both urticaria and angioedema —

- check that symptomatic episodes have not typically followed ingestion of a non-steroidal anti-inflammatory drug, such as aspirin\(^5\) (in any case, warn the patient that these drugs might provoke symptoms)

- if symptoms appear to be related to physical exertion on at least some occasions, consider wheat-dependent, exercise-induced allergy\(^6\)

- if the patient is on an ACE inhibitor, it is probably best to change to an alternative treatment, if reasonably possible\(^7\) (although ACE inhibitors are less likely to be implicated when urticaria and angioedema occur together)

- give a potent oral antihistamine:\(^8\) cetirizine, levocetirizine, loratadine, desloratadine or fexofenadine at standard dose (daily, or prn, if symptoms are infrequent)

- if necessary after 2 weeks, *increase* dose to 2 × standard dose (e.g. loratadine 10 mg twice daily)\(^8\)

- if necessary after another 2 weeks, *increase* dose to 4 × standard dose (e.g. loratadine 20 mg twice daily)\(^8\)

- if necessary after another 2 weeks, *add* montelukast 10 mg daily

- if necessary, an H\(_2\) blocker can be added (but the evidence for these is poor)

- if the angioedema has involved the tongue, pharynx or upper respiratory tract, consider prescribing adrenaline auto-injectors\(^9\) and advise to seek medical attention if this recurs; refer if recurrences are not largely abolished by the maintenance treatment described above

- *infrequent* courses of low-dose prednisolone (e.g. 20 mg daily for 3–5 days) are acceptable

- consider attempting to wean off treatment if there have been no symptoms for 3–6 months, but re-institute treatment if they recur (having to do this does not indicate a need to refer)

- refer only if symptoms remain intolerable despite these measures

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5: there is usually no benefit in stopping treatment in patients who have been stabilised on long term maintenance NSAID therapy at constant dosage, and almost never any benefit in stopping low-dose aspirin being used for antiplatelet effect. Sporadic, intermittent use of NSAIDs is more likely to cause problems.

6: this is strongly associated with IgE antibodies to \(\omega5\)-gliadin (omega-5-gliadin) – ask for a total IgE plus specific IgE to \(\omega5\)-gliadin. (Note: this test is completely unrelated to the IgA anti-gliadin test for coeliac disease.) If there is IgE to \(\omega5\)-gliadin, at least partial wheat avoidance (within 4–6 hours of exercise) may be necessary to avoid symptoms.

7: our current policy is to change patients to an angiotensin II receptor antagonist – the majority of patients can tolerate these.

8: in pregnancy / lactation, no antihistamine is guaranteed to be safe, as no trials have been done to assess this. However, there is little or no evidence of harm from the preparations cited above. Current recommendations are: avoid in the first trimester if possible; otherwise, use loratadine as the drug of choice (but do not exceed the standard dose).

9: *EpiPen* or *Jext* can be recommended. Patient information can be obtained from the product websites. There is no consensus on how many injectors a patient should have access to – two at any given time seems reasonable (it may sometimes be necessary to take two doses and sometimes a device may fail or, more likely, be incorrectly deployed), while patients going on trips to isolated places may require additional injectors.

Prescribe cautiously in uncontrolled hypertension, severe cardiovascular disease, or patient on treatment with a \(\beta\)-blocker or tricyclic antidepressant (are alternatives feasible?). Do not prescribe if patient on treatment with a monoamine oxidase inhibitor (these are now very rarely encountered).
SET 2: patients with urticaria but no angioedema —

- check that symptomatic episodes have not typically followed ingestion of a non-steroidal anti-inflammatory drug, such as aspirin\(^5\) (in any case, warn the patient that these drugs might provoke symptoms)

- if symptoms appear to be related to physical exertion on at least some occasions, consider wheat-dependent, exercise-induced allergy\(^6\)

- give a potent oral antihistamine:\(^8\) cetirizine, levocetirizine, loratadine, desloratadine or fexofenadine at standard dose (daily, or prn, if symptoms are infrequent)

- if necessary after 2 weeks, increase dose to 2 × standard dose (e.g. loratadine 10 mg twice daily)\(^8\)

- if necessary after another 2 weeks, increase dose to 4 × standard dose (e.g. loratadine 20 mg twice daily)\(^8\)

- if necessary after another 2 weeks, add montelukast 10 mg daily

- if necessary, an H\(_2\) blocker can be added (but the evidence for these is poor)

- infrequent courses of low-dose prednisolone (e.g. 20 mg daily for 3–5 days) are acceptable

- consider attempting to wean off treatment if there have been no symptoms for 3–6 months, but reinstitute treatment if they recur (having to do this does not indicate a need to refer)

- refer only if symptoms remain intolerable despite these measures

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\(^5\): there is usually no benefit in stopping treatment in patients who have been stabilised on long term maintenance NSAID therapy at constant dosage, and almost never any benefit in stopping low-dose aspirin being used for antiplatelet effect. Sporadic, intermittent use of NSAIDs is more likely to cause problems.

\(^6\): this is strongly associated with IgE antibodies to ω5-gliadin (omega-5-gliadin) – ask for a total IgE plus specific IgE to ω5-gliadin. (Note: this test is completely unrelated to the IgA anti-gliadin test for coeliac disease.) If there is IgE to ω5-gliadin, at least partial wheat avoidance (within 4–6 hours of exercise) may be necessary to avoid symptoms.

\(^8\): in pregnancy / lactation, no antihistamine is guaranteed to be safe, as no trials have been done to assess this. However, there is little or no evidence of harm from the preparations cited above. Current recommendations are: avoid in the first trimester if possible; otherwise, use loratadine as the drug of choice (but do not exceed the standard dose).
Set 3: patients with angioedema but no urticaria and taking an ACE inhibitor —

- stop the ACE inhibitor\(^7\) (regardless of how long the patient has been on it)
- warn that symptoms may recur for up to 6 weeks (possibly as long as 3 months)
- give high-dose antihistamine treatment during attacks (e.g. loratadine 20 mg twice daily)\(^8\)
- unless contraindicated,\(^10\) also give tranexamic acid during attacks (1 g four times daily); additionally, 1 g at night may be effective prophylaxis if symptoms tend to be nocturnal
- if there has been involvement of the tongue, pharynx or upper respiratory tract, consider prescribing adrenaline auto-injectors\(^9\) (however, it should be noted that adrenaline is less effective in this type of angioedema) and advise to seek medical attention if this occurs
- if the patient still has severe symptoms after 6 weeks, or any symptoms after 3 months: refer

7: our current policy is to change patients to an angiotensin II receptor antagonist – the majority of patients can tolerate these.

8: in pregnancy / lactation, no antihistamine is guaranteed to be safe, as no trials have been done to assess this. However, there is little or no evidence of harm from the preparations cited above. Current recommendations are: avoid in the first trimester if possible; otherwise, use loratadine as the drug of choice (but do not exceed the standard dose).

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Prescribe cautiously in uncontrolled hypertension, severe cardiovascular disease, or patient on treatment with a \(\beta\)-blocker or tricyclic antidepressant (are alternatives feasible\(^?\)). Do not prescribe if patient on treatment with a monoamine oxidase inhibitor (these are now very rarely encountered).

10: essentially, history of thromboembolic disease or known thrombotic diathesis (we take this to include ischaemic heart disease).
Set 4: patients with angioedema but no urticaria and not taking an ACE inhibitor —

- check that symptomatic episodes have not typically followed ingestion of a non-steroidal anti-inflammatory drug, such as aspirin\(^5\) (in any case, warn the patient that these drugs might provoke symptoms)
- if symptoms appear to be related to physical exertion on at least some occasions, consider wheat-dependent, exercise-induced allergy\(^6\)
- exclude C1-inhibitor deficiency\(^1\)
- give a potent oral antihistamine:\(^8\) cetirizine, levocetirizine, loratadine, desloratadine or fexofenadine at standard dose (daily, or prn, if symptoms are infrequent)
- if necessary after 2 weeks, increase dose to 2 × standard dose (e.g. loratadine 10 mg twice daily)\(^8\)
- if necessary after another 2 weeks, increase dose to 4 × standard dose (e.g. loratadine 20 mg twice daily)\(^8\)
- if there has been involvement of the tongue, pharynx or upper respiratory tract, consider prescribing adrenaline auto-injectors\(^9\) and advise to seek medical attention if this recurs; refer the patient
- also refer if:
  - unresponsive to high-dose antihistamine, or
  - there is evidence of C1-inhibitor deficiency

5: there is usually no benefit in stopping treatment in patients who have been stabilised on long term maintenance NSAID therapy at constant dosage, and almost never any benefit in stopping low-dose aspirin being used for antiplatelet effect. Sporadic, intermittent use of NSAIDs is more likely to cause problems.

6: this is strongly associated with IgE antibodies to \(\omega_5\)-gliadin (omega-5-gliadin) — ask for a total IgE plus specific IgE to \(\omega_5\)-gliadin. (Note: this test is completely unrelated to the IgA anti-gliadin test for coeliac disease.) If there is IgE to \(\omega_5\)-gliadin, at least partial wheat avoidance (within 4–6 hours of exercise) may be necessary to avoid symptoms.

8: in pregnancy / lactation, no antihistamine is guaranteed to be safe, as no trials have been done to assess this. However, there is little or no evidence of harm from the preparations cited above. Current recommendations are: avoid in the first trimester if possible; otherwise, use loratadine as the drug of choice (but do not exceed the standard dose).

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Prescribe cautiously in uncontrolled hypertension, severe cardiovascular disease, or patient on treatment with a \(\beta\)-blocker or tricyclic antidepressant (are alternatives feasible?). Do not prescribe if patient on treatment with a monoamine oxidase inhibitor (these are now very rarely encountered).

11: a complement C4 is an efficient screening test — it is markedly reduced in the majority of untreated patients with hereditary or acquired C1-inhibitor deficiency. It is likely to be a sufficient investigation when the index of suspicion is relatively low. **SAMPLE = PLAIN CLOTTED TUBE (i.e. serum or serum gel)**

When the index of suspicion is high — angioedema starting before the age of 20 and / or positive family history — ask for a C4 + C1-inhibitor + functional C1-inhibitor. **SAMPLE = PLAIN CLOTTED TUBE + INR TUBE**
When the history suggests that symptoms are due to allergy:

- advise avoidance of the likely allergen
- prescribe a potent, low-sedative oral antihistamine (e.g. cetirizine, loratadine, fexofenadine) — chew and swallow 2 tablets at the onset of symptoms (and twice daily until the episode settles)
- prescribe adrenaline auto-injectors if the perceived risk of anaphylaxis merits this, the patient wants them, and cautions / contraindications have been considered (patient instructions are always necessary)
- if necessary, refer for diagnostic testing and further advice on management

9: EpiPen or Jext can be recommended. Patient information can be obtained from the product websites. There is no consensus on how many injectors a patient should have access to — two at any given time seems reasonable (it may sometimes be necessary to take two doses and sometimes a device may fail or, more likely, be incorrectly deployed), while patients going on trips to isolated places may require additional injectors.

Prescribe cautiously in uncontrolled hypertension, severe cardiovascular disease, or patient on treatment with a β-blocker or tricyclic antidepressant (are alternatives feasible?). Do not prescribe if patient on treatment with a monoamine oxidase inhibitor (these are now very rarely encountered).
**RHINITIS**

Rhinitis can be allergic or non-allergic, although both forms can coexist.

Allergic rhinitis is likely if symptoms obviously coincide with exposure to known aero-allergens, such as pollens and animal danders. Allergic rhinitis due to house dust mite antigen (which is relatively ubiquitous and tends to cause perennial symptoms) is less easy to distinguish clinically from non-allergic rhinitis. To a variable degree, patients with allergic rhinitis can also have ocular symptoms (sometimes these are the main feature).

**Nasal polyposis is not, in itself, an allergic phenomenon,** though it is associated with pharmacological hypersensitivity to aspirin/NSAIDs, and it can be useful to know whether there is an additional allergic component to the rhinitis.

Referral of patients with rhinitis to the allergy clinic is justified if it is:

- to confirm the agent(s) responsible for allergic rhinitis, so that advice can be given on avoidance. If the allergen is already clinically obvious (e.g. pollen or cats), referral for purely diagnostic reasons may not be necessary — requesting a specific IgE level against the suspected allergen(s) might, if positive, give you all the information you reasonably need for diagnosis (although negative tests do not completely exclude allergy).
- to advise on treatment\(^{12}\)
- to offer allergen desensitization therapy\(^{13}\)

12: it is reasonable to optimise medical treatment as much as possible in the GP setting:

- for allergic rhinitis, see appendix 1.
- for nasal polyps — if these do not respond adequately to topical steroid (e.g. betamethasone or fluticasone drops for 2–3 weeks, followed by maintenance with a steroid spray), referral to ENT might be indicated. Aspirin desensitization can sometimes be considered for patients with nasal polyps and aspirin sensitivity, but this is rarely of more than modest benefit.

13: allergen desensitization can be considered for patients whose symptoms of allergic rhinitis are not adequately controlled by standard treatment, or for those who genuinely cannot tolerate nasal steroids.

Exclusion criteria:

- patients with perennial asthma requiring regular treatment are precluded from having injection desensitization therapy but can be considered for sublingual desensitization. (This does not affect patients with purely seasonal asthma.)
- patients who must remain on \(\beta\)-blocker or ACE-inhibitor therapy.

It should be noted that desensitization is not appropriate in cases where the allergen could reasonably be avoided without this having a significant adverse effect on the patient’s daily activities or employment.

**ASTHMA**

Testing of asthmatics for allergy can be a useful guide to advice on allergen avoidance. However, referral to the allergy clinic should not be used to access routine asthma management.

Unfortunately, desensitization therapy is not currently recommended as a treatment for asthma.
**Eczema**

Patients with troublesome eczema are often keen to discover an avoidable dietary cause of their symptoms. Unfortunately, although trials of exclusion diets are sometimes of clinical benefit in cases of eczema, prior allergy testing does not reliably predict the response. Indeed, many patients with eczema will demonstrate positive allergy tests to dietary antigens which they know they can tolerate — this can lead to unnecessary food avoidance.

If the history suggests contact eczema, skin patch testing may be worthwhile — this requires a referral to dermatology.

In the absence of other symptoms of food allergy, there is little to be gained by referring patients with eczema beyond the dermatology clinic.

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**Suspected Drug Allergy**

Antibiotics are the commonest cause of suspected drug allergy. When considering whether to refer a patient for investigation of possible drug allergy, please note that investigation is only justified if there is (or is likely to be) a convincing clinical indication for giving the relevant drug (or related compound) again. This is because adequate investigation will often necessitate challenge or provocation testing.

Although NSAIDs can cause symptoms indistinguishable from those of type I hypersensitivity (up to and including anaphylaxis), these reactions are generally not IgE-mediated. There is no diagnostic test (apart from challenge), so the diagnosis is almost always clinical, and these drugs should be avoided if there is a strong index of suspicion. Only if there is a pressing clinical need for treatment with one of these drugs, should patients be referred for challenge testing. Most patients can take paracetamol without difficulty.

ACE-inhibitors are well known to cause or exacerbate angioedema (see section on urticaria and angioedema). Again, this reaction is not immunologically mediated and there is no diagnostic test. Patients who have had angioedema, from whatever cause, should avoid ACE inhibitors, while most can tolerate angiotensin II receptor antagonists.

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**Allergy to Bee or Wasp Venom**

Any patient with a history of respiratory symptoms or a systemic allergic reaction (generalised urticaria, angioedema, bronchospasm or anaphylaxis) following a bee or wasp sting should be referred for assessment. Prescription of EpiPens is appropriate, and some will benefit from allergen desensitization.

It is not necessary to refer patients:
- for “screening”, because they have other allergies
- who have only had local reactions to stings (even though these can be quite intense)
- because another family member is allergic to bee or wasp venom

14: if there is anxiety because of these situations, send blood for circulating specific IgE against bee and wasp venom. If these are undetectable, the patient is unlikely to be at high risk.
CLINICAL SITUATIONS UNRELATED TO ALLERGY

Patients frequently attribute a wide variety of symptoms to allergy. This perception has often been reinforced by items in the popular media, and by the results of privately available, ‘alternative’, diagnostic tests, which lack inherently plausibility or established scientific validity. In many cases, the patients’ belief that their symptoms are due to allergy are deeply entrenched and resistant to rational debate.

The following should be borne in mind:

- weight loss, weight gain, headache/migraine, confusion, depression, lack of concentration, vertigo, tiredness/chronic fatigue and hair loss can never be explained in terms of allergy.
- The same is almost always true of isolated vague abdominal symptoms such as bloating (these can, however, be due to irritable bowel syndrome, undiagnosed coeliac disease or other organic pathologies). Even if “food intolerance” (e.g. to wheat) does exist as a diagnostic entity, there are no validated diagnostic tests (or therapies, other than avoidance).
- there is no good evidence that “systemic candidiasis syndrome” or “multiple chemical sensitivity syndrome” exist as objective physical entities, or that they are actually due to those agents from which they derive their name. There are no validated diagnostic tests or therapies.
- some substances are simply not recognised allergens, e.g. tap water, sugar, caffeine, dental amalgam. Patients who consider themselves intolerant of various scents or odours (e.g. bleach, “chemical smells”, pot pourri, air freshener, perfumes, etc.) are likely to be suffering from learned behavioural responses to perceived noxious stimuli (this is sometimes associated with laryngeal dysfunction).
- positive results obtained by unorthodox techniques, such as electrodermal testing (e.g. the Vega test), are irrelevant to the diagnosis of allergy and should be ignored — they do not justify referral to the allergy service. In addition, IgG antibodies to food allergens, as measured by some private laboratories, have not been shown to be of diagnostic value.

We do not normally accept referrals of patients in these categories.

C1-INHIBITOR DEFICIENCY

This rare but important problem, which can be hereditary or acquired, is likely to present as angioedema (see URTICARIA AND ANGIOEDEMA). However, it deserves special mention as the presentation may be atypical.

The diagnosis should be considered in patients with:

- unexplained episodic angioedema (without urticaria) and / or episodic abdominal pain
- a family history of these symptoms (or of known hereditary angioedema)

Investigation:

- a complement C4 is an efficient screening test — it is markedly reduced in the majority of untreated patients with hereditary or acquired C1-inhibitor deficiency. It is likely to be a sufficient investigation when the index of suspicion is relatively low.

SAMPLE = PLAIN CLOTTED TUBE (i.e. serum or serum gel)

- when the index of suspicion is high — angioedema starting before the age of 20 and / or positive family history — ask for a C4 + C1-inhibitor + functional C1-inhibitor.

SAMPLE = PLAIN CLOTTED TUBE + INR TUBE

We strongly recommend that all cases of C1-inhibitor deficiency be referred to the clinic for specialist follow-up.

15: C1-inhibitor deficiency does not cause urticaria, so there is no need to investigate patients with urticaria for this condition.
TREATMENT OF ALLERGIC RHINITIS

STAGE 1:

Potent, low-sedative oral antihistamine, e.g.

cetirizine (Zirtek) 10 mg daily, or levocetirizine (Xyzal) 5 mg daily, OR
fexofenadine (Telfast) 180 mg daily, OR
loratadine (Clarityn) 10 mg or desloratadine (Neoclarityn) 5 mg daily

Doses can be doubled to twice daily, if necessary (except if pregnant / lactating) — warn about possible additional sedation.

We do not recommend older, short-acting, sedative antihistamines, such as chlorphenamine (Piriton).

1: if possible, avoid antihistamines in the first trimester. Otherwise, loratadine is the preparation of choice in pregnancy.

STAGE 2:

Add a topical steroid, e.g.

Fluticasone (Flixonase) or mometasone (Nasonex): 1 spray each nostril twice a day (reducing to once a day, if/when symptoms are controlled).

When symptoms are severe, steroid drops may be more effective, and these can usually be changed to a steroid spray after 3–4 weeks:

Betamethasone (Betnesol): 3 drops each nostril 3 times a day for one week, then reducing to twice a day for 2–3 weeks,

OR

Fluticasone (Flixonase) nasules: 6 drops each nostril twice daily for 2 weeks, then once daily for 1 or 2 weeks

THEN changing to e.g. Flixonase or Nasonex spray, as above

Patients using drops should be advised to take them as follows:

When using drops, you should lie flat on the bed with your head tilted back over the edge so that your nostrils point upwards — put in the drops and try to maintain this position for 30 seconds. Then turn your head to one side, and then the other, for 30 seconds each. Having to do this is rather inconvenient, but it does allow the treatment to act more effectively. (NB: if you have arthritis in your neck, it may be unwise for you to adopt this position — let the doctor know this.) When you return to a normal upright position, some of the drops will run out of your nose or down the back of your throat — that is normal and does not mean that the treatment will not be effective.

For patients known to have problematic hayfever, stages 1 & 2 can be started simultaneously. Patients must be advised to take the treatments regularly, even on days when symptoms are less severe.

STAGE 3:

Add leukotriene inhibitor

Montelukast (Singulair) 10 mg daily.
STAGE 4:

**i.** If a runny nose (rhinorrhoea) is still a problem: **add ipratropium bromide, e.g.**

   *Rinatec*, 2 sprays each nostril 2–3 times a day.

   OR

**ii.** If nasal congestion is still a problem: **add sodium cromoglicate, e.g.**

   *Rynacrom*, 1 spray each nostril 2–4 times a day.

   OR

**iii.** If symptom control remains poor, or to cover important events (exams, etc.): **add a brief course of oral steroid, e.g.**

   prednisolone 20 mg daily for maximum of 1 week

We do not recommend the use of depot steroid injections.

EYE SYMPTOMS:

In rhinocojunctivitis, ocular symptoms sometimes improve when the nose is treated as above. If necessary, however, nedocromil sodium and / or antihistamine eye drops can be prescribed.

**Over-the-counter decongestants**

When symptoms are severe at the start of treatment, a sympathomimetic decongestant spray, containing oxymetazoline or xylometazoline (occasionally phenylephrine), may reduce congestion and rhinorrhoea, making the nasal mucosa accessible to other treatments. **Use should be limited to a maximum of three times daily (preferably once or twice) for not more than one week.** Prolonged use of these preparations is associated with a significant rebound effect and worse symptoms (rhinitis medicamentosa).

   Sympathomimetic agents should not be used at all by patients taking a monoamine oxidase inhibitor antidepressant.