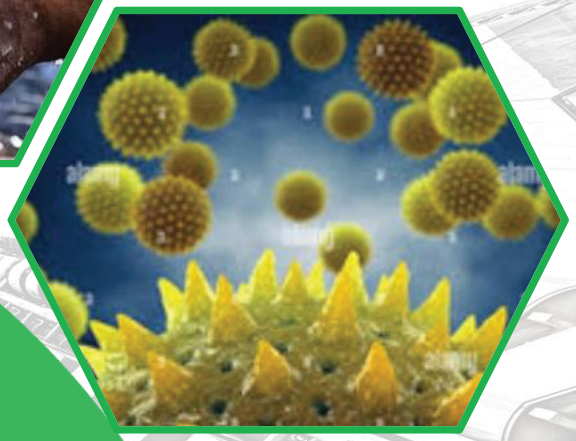
REPUBLIC OF KENYA

MINISTRY OF HEALTH

NATIONAL CLINICAL GUIDELINES FOR MANAGEMENT AND TESTING OF ALLERGIC CONDITIONS IN KENYA



STATE DEPARTMENT FOR MEDICAL SERVICES

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Foreword

Allergies are a global health concern affecting children and adults alike. Worldwide, atopic diseases affect up to 30-40% of general population. Within the last decades, the increase in allergy noted in some regions has been mostly attributed to changes in environment associated with urbanization. The spectrum of allergic disease spans from allergic rhinosinusitis, allergic conjunctivitis, asthma, atopic dermatitis, allergic gastro-enteropathies and acute reactions to medications, stings, bites and foods, with patients sometimes presenting with multiple manifestations of these conditions; and well recognized progression from one manifestation to another.

Allergic conditions carry with them a significant burden to the patient and their parents and/or caregivers. It carries significant morbidity and unfortunately, in some countries including Kenya, asthma and anaphylaxis also carry significant mortality due to poor management or misdiagnosis. The financial burden is significant as these chronic diseases require therapeutic interventions, hospital and physician appointments, investigations like allergy tests and in case of food allergies, dietary substitutes which are very expensive and difficult to find.

In response to the intricate dynamics of demographic shifts and epidemiological transitions, the Ministry of Health in Kenya has taken commendable steps to address the rising incidence of allergies and other Non-Communicable Diseases (NCDs). This commitment goes beyond a reaction to a health challenge; it represents a strategic response deeply rooted in global health goals and national policies.

The development of the National Guidelines for the Management and Testing of Allergic Conditions 2026 marks a pivotal moment in Kenya's ongoing health sector reforms. These Guidelines are more than just documents; they embody a standardized approach to allergic management, guiding healthcare providers in screening, diagnosing, and managing individuals with these conditions.

The significance of these Guidelines extends beyond the immediate challenges of allergies. They play a crucial role in realizing Universal Health Coverage (UHC), a key priority for the Ministry of Health (MoH). Emphasizing quality, equity, and financial protection, these Guidelines ensure the delivery of quality healthcare services to the Kenyan population.

As the healthcare system shifts towards primary health care (PHC), these Guidelines are indispensable tools, essential for achieving UHC and bringing healthcare services closer to the people where they

live and work. The Guidelines are strategically aligned with national policies, including the Kenya Health Policy 2012–2030, and the NCD Strategic Plan 2021-2026.

These Guidelines serve as a lifeline for those living with allergies, setting the standard for care and emphasizing the rights of access and quality for patients. To realize their desired impact, a collective effort is crucial, involving dissemination, synthesis into job aids, targeted training, a robust referral strategy, and investment in health products and technologies.

Recognizing the complexity of managing allergies, the MoH emphasizes the importance of a broad partnership involving public and private sectors, academia, professional associations, patients' groups, and non-state actors in developing, disseminating, and implementing these Guidelines.

In conclusion, effective allergic management requires a collective endeavor involving the commitment of healthcare providers, stakeholders, and the community at large. These Guidelines epitomize that commitment, providing a roadmap towards a healthier and more resilient Kenya.

Dr. Patrick Amoth, CBS

DIRECTOR GENERAL FOR HEALTH

Acknowledgement

The development of these National Guidelines for the Management and Testing of Allergic Conditions was a collaboration effort between the MOH, Allergy Society of Kenya (ASOK) and the National Technical Working Group (TWG) on Allergy. The process involved expert consultations, technical workshops, external peer review and validation. The MoH wishes to thank all the teams and institutions that were involved in the various stages of the development of these Guidelines. We also wish to thank ASOK for their efforts to develop the first version of the Guidelines and providing expert advisory on their development and structure. We thank the members of ASOK Allergy Guideline Committee for their input during the organization and review of the protocols included in the Guidelines. Special thanks to Anton Jansen (South Africa) for taking the time to join the TWG.

We thank Prof Carsten Flohr (MA MSc PhD FRCP FRCPCH), Chair in Dermatology & Population Health Science, St John's Institute of Dermatology, King's College London, the team at the World Health Organisation (WHO) for their appraisal of the document, as well as The Royal Children's Hospital, Melbourne, Australia, for permission to use their acute asthma clinical practice Guidelines.

We thank the Division of Clinical Services headed by Dr. Barded Saleh for providing support as the secretariat in the development of the Guidelines and organizing the technical development and review workshops.

We call upon all the stakeholders involved in the development of these Guidelines to continue collaborating with the MoH in their dissemination and implementation.

Dr. Andrew J. Toro, OGW

DIRECTOR, DIRECTORATE OF CURATIVE AND NURSING SERVICES

Abbreviations

AAI	Adrenaline Auto Injector
ACD	Allergic Contact Dermatitis
AD	Atopic Dermatitis
ADR	Adverse Drug Reaction
AGEP	Acute Generalised Exanthematous Polyposis
AKC	Allergic Keratoconjunctivitis
AKUH, N	Aga Khan University Hospital, Nairobi
APT	Atopy Patch Test
AR	Allergic Rhinitis
ASOK	Allergy Society of Kenya
BAT	Basophil Activation Test
BP	Blood Pressure
C1	Complement 1
C1 INH	Complement 1 Inhibitor
C4	Complement 4
CXR	Chest X Ray
DNA	Deoxyribonucleic Acid
DRESS	Drug Reaction with Eosinophilia and Systemic Symptoms
ECP	Eosinophilic Cationic Protein
ENT	Ears Nose and Throat
EOE	Eosinophilic Oesophagitis
FA	Food Allergy
FBOs	Faith Based Organisations
FeNO	Fractional Exhaled Nitric Oxide
FPC	Food Protein induced Constipation
FPE	Food Protein Enteropathy
FPGORD	Food Protein Gastroesophageal Reflux Disease
FPIP	Protein Induced Proctocolitis
FPIES	Food Protein Enterocolitis Syndrome
GSK	Glaxo SmithKline
HAE	Hereditary Angioedema
Hpf	High power film
HVA	Hymenoptera Venom Allergy
ICD	Irritant Contact Dermatitis
IDT	Intradermal Test
IgG	Immunoglobulin G
IgE	Immunoglobulin E
KNH	Kenyatta National Hospital
M&E	Monitoring and Evaluation
MDI	Metered Dose Inhaler
MOH	Ministry of Health
MTRH	Moi Teaching and Referral Hospital
NCDs	Non-Communicable Diseases
NRL	Natural Rubber Latex

NSAIDS	Non-Steroidal Anti -Inflammatory Drugs
OFC	Oral Food Challenge
PEF	Peak Expiratory Flow
PHC	Primary Health Care
PPD	Para Phenylenediamine
SABA	Short Acting Beta Agonists
SBM	Spontaneous Bowel Movements
SBP	Systolic Blood Pressure
SJS	Steven Johnson's Syndrome
SPT	Skin Prick Test
TEN	Toxic Epidermal Necrolysis
TWG	Technical Working Group
UHC	Universal Health Care
UoN	University of Nairobi
VIT	Venom Immunotherapy
WHO	World Health Organization
WOB	Work of Breathing

Chapter One: Introduction/ Background

1.1 Introduction

Allergic conditions such as asthma, allergic rhinoconjunctivitis and atopic eczema to name a few, are a global health concern affecting children and adults alike. Worldwide, atopic diseases affect up to 30-40% of the general population. There is paucity of large population-based data of the prevalence of the different allergic conditions in Kenya. It is currently estimated that 7.5 % of the Kenyan population has asthma. Adolescents aged 13-14 years had an increased prevalence of wheeze, rhinoconjunctivitis and atopic eczema symptoms 12 months before the study period of 13.9% -15.8%, 14.2-21.2% and 10.4-15.2% respectively in phase I (1992-1998) and III (1999-2004) of the International Study of Asthma and Allergy in Childhood (ISAAC). This data supports an increased need for allergy awareness and action in Kenya.

Within the last decades, the increase in allergy noted in some regions of the world has been mostly attributed to changes in environment associated with urbanization. Current thinking supports the view that allergies are both genetic and environmental in origin. Twin studies suggest that allergic diseases such as asthma and eczema are highly heritable, but their prevalence varies considerably by region, suggesting that environmental factors such as exposure to environmental tobacco smoke, pollution, allergens, infections and diet can influence allergic diseases especially in early life.

There is growing evidence that early life exposure to a diverse microbial environment is protective, while epigenetic changes caused by pollution and climate change, smoking etc. are harmful. Environmental and dietary factors are also at play in early life. Figure one shows the complicated relationship between an individual's genes, their lifestyle and the environment in which they live. The latter two exposures can result in changes in the supporting structure of an individual's DNA causing inheritable allergy risk for future generations.

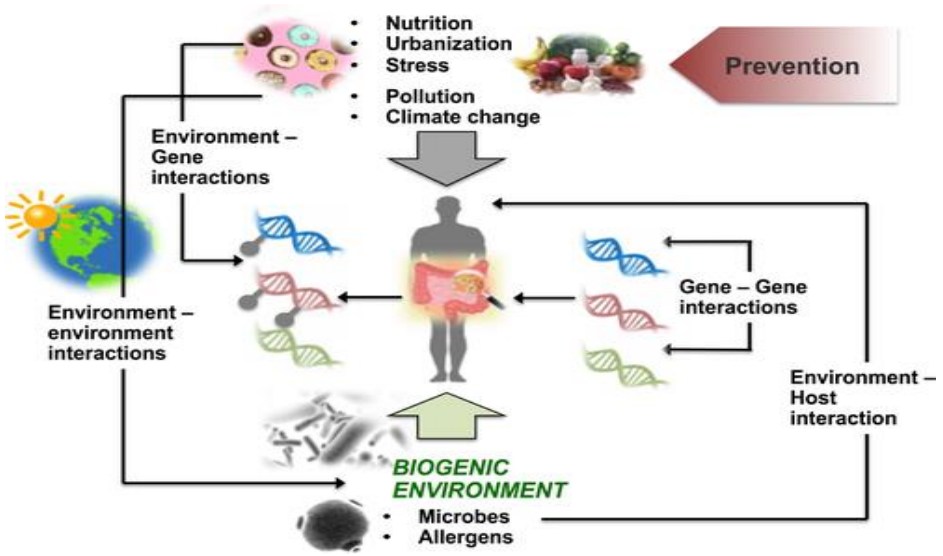


Figure 1: Anthropogenic environmental factors.

Image adapted from Gilles et al

1.2 Background

The spectrum of allergic disease spans from allergic rhinosinusitis, allergic conjunctivitis, asthma, allergic gastro-enteropathies and acute reactions to medications, stings, bites and foods, with patients sometimes presenting with multiple manifestations of these conditions; and well recognized progression from one manifestation to another. Atopic dermatitis/eczema is not considered an allergy *per se* though it is closely linked to allergic conditions due to the sensitization that occurs in those genetically predisposed. The atopic march represents the apparent progression seen in some young children over time, from atopic dermatitis and food allergy in early childhood phase to asthma and allergic rhinitis. Most of the burden of allergic conditions relates to quality-of-life impact. This can be lessened by making a correct diagnosis, avoiding troublesome allergens, and using anti-inflammatory treatments or desensitization when necessary. Allergic conditions can also cause fatal reactions, especially asthma. Fatal anaphylaxis is very rare, and most cases are caused by allergies to medications in the elderly, but also occasionally to insect stings or foods.

Allergy is a reaction where select individuals exhibit an immune response to a particular substance usually tolerated by majority of the population. This reaction is atypical as the substances or allergens recognized by the immune system are relatively harmless rendering allergies a form of immune dysregulation. Allergic patients are identified by the recurrence of signs and symptoms following exposure to their specific allergens.

Sensitization occurs when an individual's immune system develops specific immunoglobulin E (IgE) antibodies towards particular allergens. These allergens are usually protein in nature except for alpha gal or red meat allergy, where sensitization occurs towards carbohydrate moiety. Of note is that most substances in nature including fruits and vegetables contain protein moieties in their structure which can be identified by the immune system as an allergen. Sensitization is determined using skin prick, intradermal tests and serum specific IgE assays and does **NOT** denote allergy.

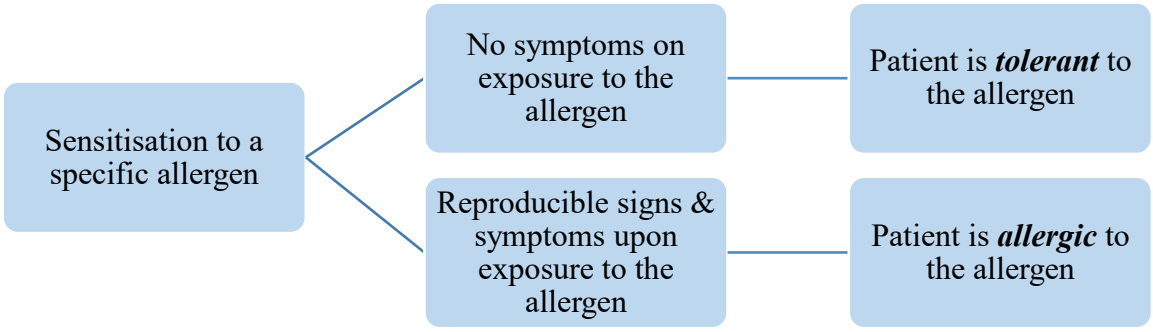


Figure 2: Sensitization to a specific allergen

Sensitization as shown in the image above is therefore always higher than true allergies. A 2014 study in South Africa found that whilst black children with atopic eczema had higher sensitization than those of mixed race, (69 % versus 61%) respectively, the proportion with food allergy was lower, (34 versus 46%). A 2018 population-based study of children aged 3 years and below in Cape Town found 11.4% of the participants were sensitized to at least one food whilst 2.5% had food allergy confirmed by oral challenge test. Similarly, a 2019 study in India reported that 19% of children had a positive specific IgE allergy test to at least one food, whereas only 0.14% of the same population of children had a food allergy.

Allergy testing is therefore more likely to identify a tolerant individual, especially when a detailed clinical history is not considered. This leads to unnecessary avoidance measures, worry and added expenses for the patient.

1.3 Purpose

This document serves to provide guidance on how to deliver standardized management to, and testing of patients with allergic conditions in Kenya.

1.4 Objectives

- To improve the clinical management of patients with allergic conditions in Kenya.
- To improve timely referrals for appropriate testing and specialised management in patients with allergic conditions in Kenya.
- To provide a framework for monitoring and evaluation of patients with allergic conditions in Kenya.

1.5 Rationale

Allergic conditions are increasingly common in our setting. The ratio of Allergists in Kenya is less than 1:11 million Kenyans, although specialist care for specific conditions is offered by ENT (Ear, Nose and Throat) Surgeons, Ophthalmologists, Dermatologists and Pulmonologists. Our current medical training in allergy is fragmented into these different specialties, which can lead to a fractured approach to management. However, majority of patients present with more than one allergic condition and require comprehensive evaluation and management of their allergies simultaneously. Healthcare practitioners are struggling with this concept of one stop allergy evaluation leading to different referrals for each patient, which has a cost implication both financially and, in the time taken before appropriate management is delivered. These Guidelines serve to inform health practitioners to assess associated allergies when they encounter allergy patients, initiate appropriate management at each level of health care and ensure timely referrals to the appropriate specialist.

Allergy testing has also been noted to be a source of confusion among both health care workers and the public. Most assume that allergic conditions have only one underlying mechanism and therefore only one method of testing. There is paucity of knowledge of which test is indicated in different allergic conditions as well as how to interpret them. Allergy testing is expensive and can lead to mismanagement when sensitization is confused with allergy status. These Guidelines serve to inform the practice of appropriate allergy testing among healthcare practitioners in Kenya.

1.6 Scope

These Guidelines will inform the care of patients with allergic conditions in levels two to six within the public and private health sector, including Faith Based Organizations (FBOs).

1.7 Target Audience

These Guidelines are aimed at all health care practitioners in Kenya.

1.8 Expected Outcomes

The use of these Guidelines are expected to increase access to the appropriate management for patients with allergic conditions and timely referrals for testing and more specialized care in the spirit of UHC.

1.9 Process of Guideline Development

A concept of the Guidelines was developed and refined into a zero and final draft by members of the National Technical Working Group (TWG) on Allergy during a three-day workshop in June 2024. These members were drawn from MoH and ASOK. The Guidelines were then submitted to a process of internal and external validation on 11th June 2025 and 13th August 2025 respectively before undergoing final editing and printing. The Guidelines were officially launched prior to dissemination to the counties. A monitoring and evaluation process was then implemented to inform the revision of the document in the next five years.

Chapter Two: Management of Common Allergic Conditions

1.10 Allergic Conjunctivitis

1.10.1 Definition

This is an immune mediated conjunctivitis that may present seasonally or without a specific pattern.

The grading is from mild to severe based on clinical signs.

1.10.2 Classification

a) Vernal Keratoconjunctivitis (VKC)

Presents in childhood and usually wanes in the late teens. Males are more commonly affected than females. Increased episodes noted in hot dry climates.

b) Atopic conjunctivitis (AKC)

Presents in adulthood and has a chronic and unremitting course. There is a strong association with allergic rhinitis, eczema and asthma.

c) Seasonal or perennial allergic conjunctivitis

Aggravated by various allergens:

- Seasonal- pollen and mould.
- Perennial- dust mites, animal dander and mould.

d) Giant papillary conjunctivitis

Not a true form of allergic conjunctivitis. It results from repetitive irritation of the ocular surface by contact lenses, prosthesis or exposed sutures.

1.10.3 Clinical features of Allergic Conjunctivitis

- Itching
- Tearing
- Eye redness
- Mucoid eye discharge
- Foreign body sensation
- Thickening, scaling, dryness and redness of eyelid skin (AKC).

1.10.4 Management

- Encourage the use of cool compresses.

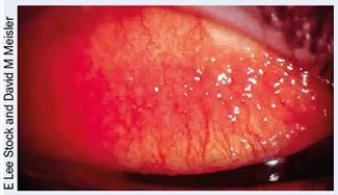
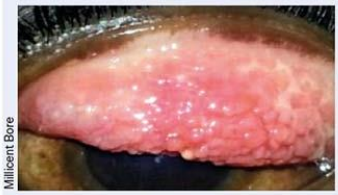
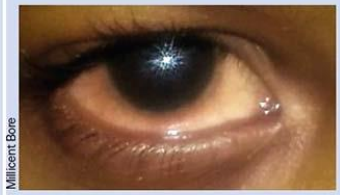
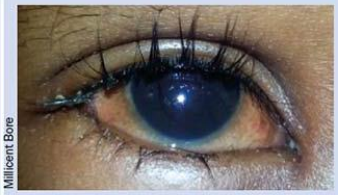
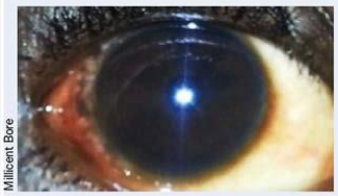
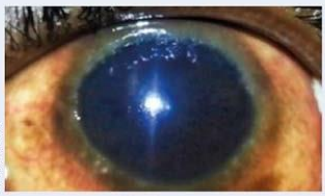
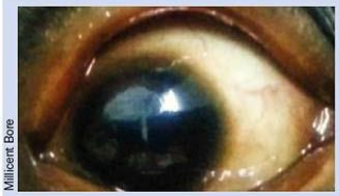
- Advice on avoidance of known allergens and eye rubbing.
- Prescribe a preservative free lubricant and multi-action drug such as olopatadine.

1.10.5 Recommendations/Referrals

Vernal keratoconjunctivitis and AKC can result in visual loss due to corneal scarring. Other sight threatening complications include keratoconus (due to excessive eye rubbing), cataracts and glaucoma (due to prolonged steroid use). As such, early referral to an ophthalmologist is key.

STEROID USE IN EYE DROPS SHOULD BE AVOIDED UNLESS PRESCRIBED BY AN OPHTHALMOLOGIST

Refer to the allergist for testing in severe cases.

Grade	Mild	Moderate	Severe
Papillae	 Millicent Bore Micro: <0.3mm	 E Lee Stock and David M Meisler • Macro: between 0.3 and 0.5mm • +/– Fibrosis	 Millicent Bore • Cobblestone papillae: >0.5mm but smaller than 1.0mm • Giant papillae: >1.0mm
Conjunctiva	 Millicent Bore Hyperemia	 Millicent Bore • Hyperemia • Diffuse thin chemosis	 Millicent Bore • Hyperemia • Cyst-like chemosis/scar • Conjunctivalisation of the cornea
Limbus (limbal oedema or Horner-Trantas dots)	 Erhardt Kidson No manifestations	 Millicent Bore <½ of limbal circumference affected	 Millicent Bore ½ or more of limbal circumference affected
Cornea	 Erhardt Kidson Clear	 Millicent Bore Superficial punctate keratitis	 Millicent Bore • Shield ulcer/epithelial erosion • Keratoconus +/– central leucoma

Note that patients diagnosed with vernal or atopic keraconjunctivitis should be treated as **'severe'** cases, whatever their presenting clinical signs.

Figure 3: Ocular allergy clinical grading guide developed for use in Kenya.

Grading is determined by the presence of the most severe sign in the most affected eye.

1.11 Allergic Rhinitis

1.11.1 Definition

Allergic Rhinitis (AR) is an allergen associated inflammation of the nasal mucosa. This allergic condition is a clinical diagnosis. It is commonly associated with other atopic conditions, especially asthma and allergic conjunctivitis.

1.11.2 Classification of allergic rhinitis

Allergic rhinitis can be classified based on:

Environment:
Seasonal - occurs during a certain time of the year e.g., with exposure to pollen Perennial - is continuous over the year e.g., due to dust mites or animal dander
Severity Mild symptoms - don't interfere with QoL
Moderate/ severe interfere with QoL: sleep disturbance, impairment of school or work performance, impairment of daily activities, leisure, and/or sport activities)
Frequency Intermittent < 4 days/week or < 4 consecutive weeks/year
Persistent > 4 consecutive weeks/ year

1.11.3 Clinical Features

Presence of two or more symptoms of runny nose, sneezing, nasal obstruction (blockage), nasal itchiness and eye symptoms (itching, redness or watery eyes) for most days of the week.

1.11.4 Clinical signs

Common signs include rubbing the nose (allergic salute), nasal crease and allergic shiners (darkening under the eyes).

1.11.5 Management of Allergic Rhinitis

- Avoid precipitating factors if known (e.g., grass pollen, dust, animal dander).
- Give second generation antihistamines for 5-7 days.
- Give a topical intranasal corticosteroid (e.g., fluticasone, mometasone) for minimum one month.

*Avoid xylometazoline, sodium cromoglycate, celestamine and piriton

*Montelukast should be prescribed by a specialist.

1.11.6 Recommendations/Referrals

Refer to ENT specialist if:

- Nasal polyposis
- Sino-nasal tumours
- Associated asthma
- Deviated nasal septum
- Sinusitis (frontal headaches, fevers, persistent rhinitis)
- Gross nasal obstruction (hypertrophied inferior turbinates)
- Adenoid hypertrophy (snoring, mouth breathing and disturbed sleep)
- Unilateral thick foul-smelling rhinorrhoea (possible foreign body nose)
- *Uncontrolled after using the topical nasal spray for 1 month without relief*

Refer to allergist for skin prick testing/serum specific IgE tests if poorly controlled or have associated asthma. Allergy testing will be guided by history.

1.12 Acute Asthma

Asthma is a heterogeneous clinical syndrome characterized by chronic airway inflammation to a variety of stimuli including irritants and allergens. This results in reversible airway obstruction of varying severity that responds to bronchodilator therapy. Salbutamol, a short acting beta agonist (SABA) is commonly used to demonstrate reversibility. In very severe cases, this reversibility is lost.

1.12.1 Clinical Features

Patients present with recurrent or chronic cough, shortness of breath, chest tightness and wheezing, the later may not be present in all patients.

1.12.2 Classification of acute asthma severity

Table 1: Classification of acute asthma severity.

Severity	**Mild**	**Moderate**	**Severe**	**Life-threatening**
Features	Mild increased work - of - breathing (WOB) Normal respiratory rate Alert and active O2 saturations in room air 90-95 % PEF > 50 % of personal best or predicted	Moderate increased WOB Increased respiratory rate Alert and active O2 saturations in room air 90-95 % PEF > 50 % of personal best or predicted	Markedly increased WOB Increased respiratory rate Agitated Pale Other signs of anaphylaxis may be present O2 saturations in room air < 90 % PEF ≤ 50 % of personal best or predicted	Maximal WOB Increased respiratory rate Confused, drowsy Silent chest Cyanosed Other signs of anaphylaxis may be present O2 saturations in room air 90-95 % PEF < 30% of personal best or predicted PCO_2 > 45 mm Hg PO_2 < 65 mm Hg

Adapted from The Royal Children's Hospital, Melbourne, Australia, Clinical Practice Guideline on Acute Asthma, [Internet, last updated July 2023); cited on 16th March 2026], Available from https://www.rch.org.au/clinicalguide/guideline_index/asthma_acute/ and Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2025. Updated May 2025. Available from: www.ginasthma.org.

1.12.3 Normal ranges of vital signs

Age group	Heart rate	Respiratory rate	Systolic BP
Preterm	120 - 180	50 - 70	40 - 60
Newborn (0-1 month)	100 - 160	35 - 55	50 - 70
Infant (1-12 months)	80 - 140	30 - 40	70 - 110
Toddler (1-3 years)	80 - 130	20- 30	70 - 110
Preschool (3-6 years)	80 - 110	20 - 30	80 - 110
School age (6-12 years)	70 - 100	18 - 24	80 - 110
Adolescents (> 12 years)	60 -9 0	14 - 22	100 -1 20

Table 2: Normal ranges of vital signs
Adapted from the Kenya Asthma Management Guidelines 2021.

1.12.4 Management of mild asthma

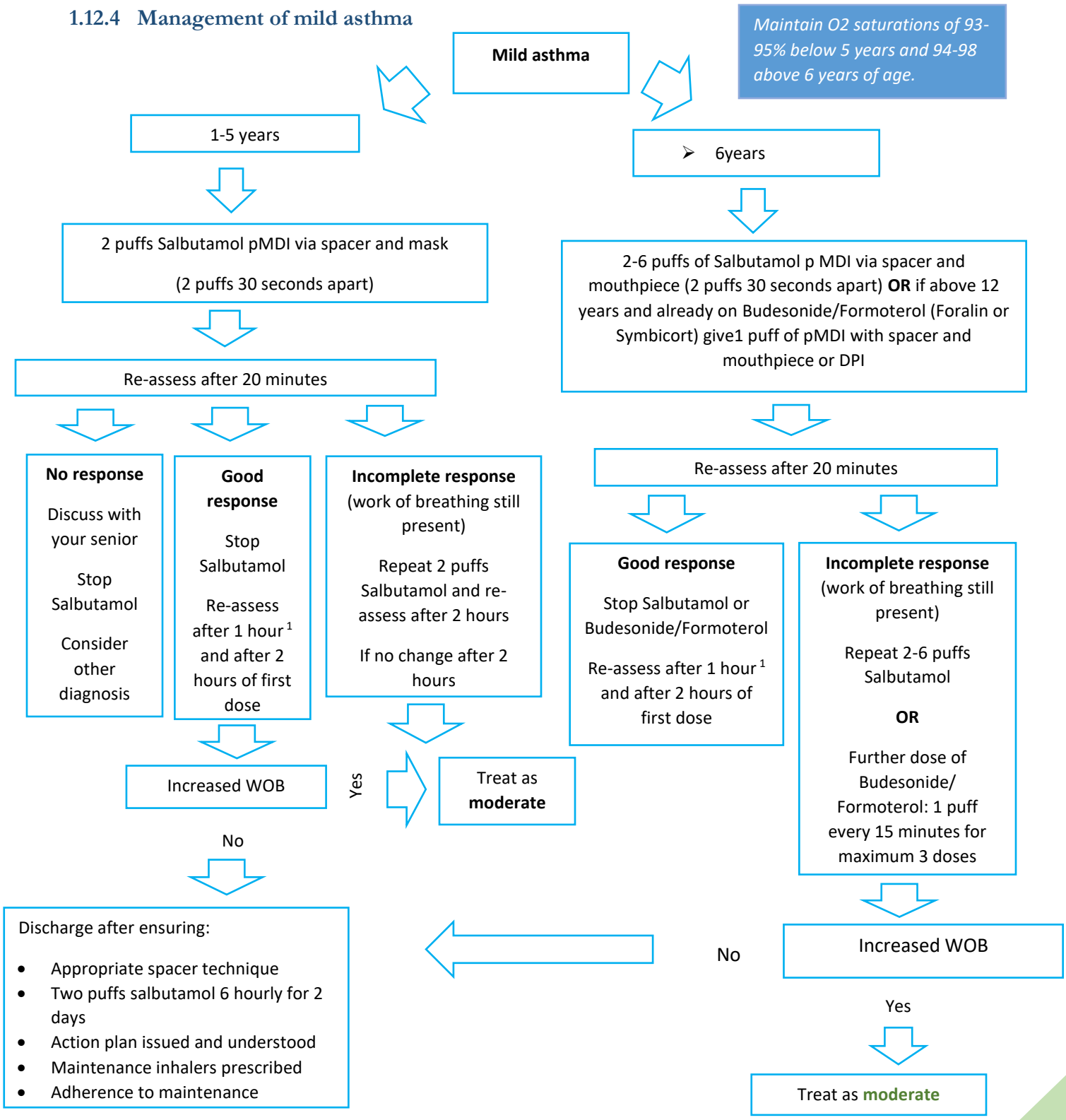


Figure 4: Acute mild asthma management algorithm

1.12.5 Management of moderate asthma

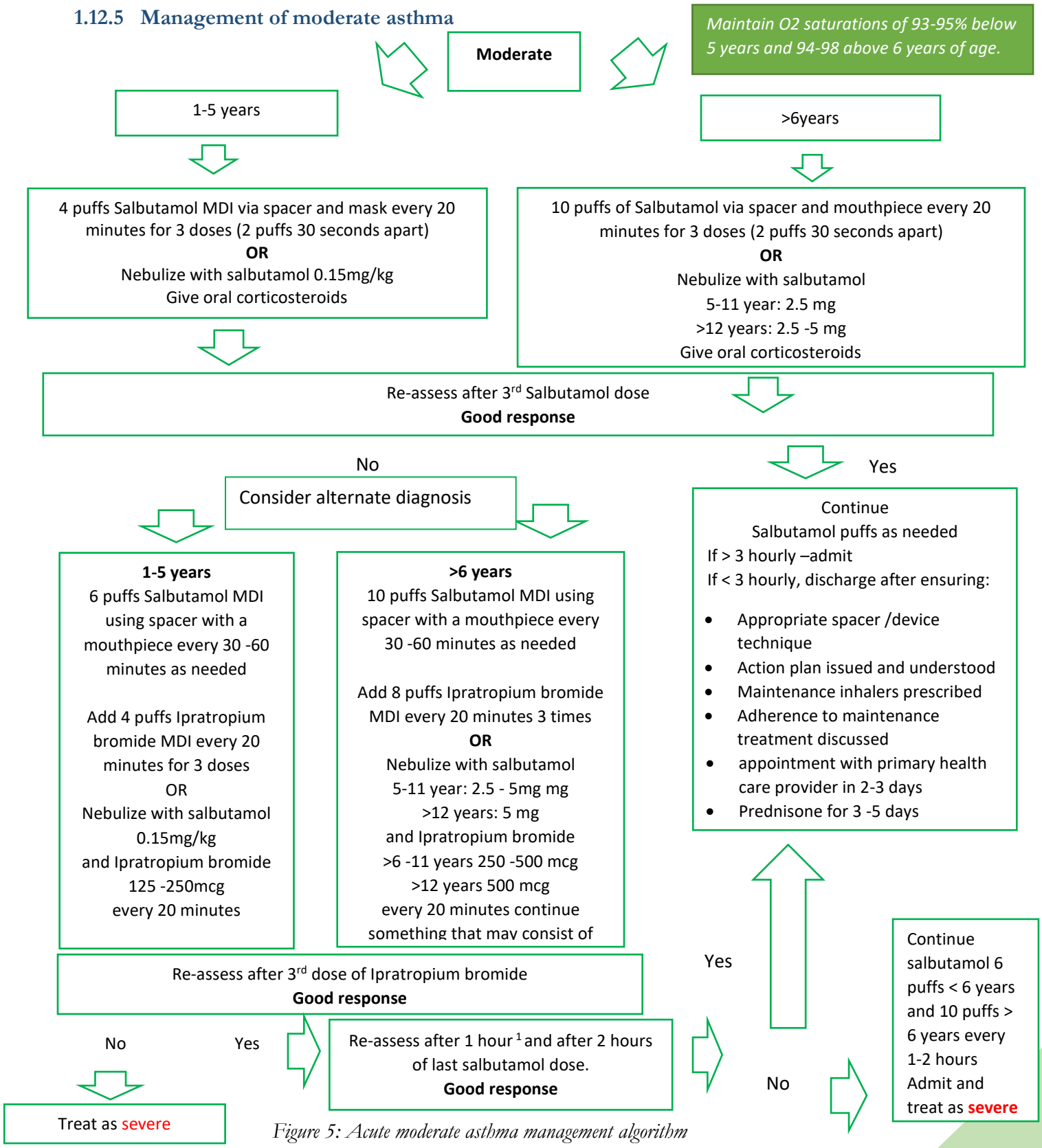


Figure 5: Acute moderate asthma management algorithm

1.12.6 Management of severe asthma

Severe

Maintain O2 saturations of 93-95% below 5 years and 94-98 above 6 years of age.

Admit to level 5 or 6 facility and involve specialist and subspecialists

1-5 years
6 puffs Salbutamol MDI using spacer with a mouthpiece every 20 minutes for 3 doses

Add 4 puffs Ipratropium bromide MDI every 20 minutes for 3 doses
OR
Nebulize with salbutamol 0.15mg/kg
& 125 -250 mcg Ipratropium bromide
every 20 minutes
Give systemic steroids *
Consider second line IV treatment early

>6 years
10 puffs Salbutamol MDI using spacer with a mouthpiece every 20 minutes for 3 doses

Add 8 puffs Ipratropium bromide MDI every 20 minutes for 3 doses
OR
Nebulize with salbutamol
5-11 year: 2.5 -5 mg
>12 years: 5 mg
and Ipratropium bromide
>6 -11 years 250 -500 mcg
>12 years 500 mcg

Re-assess after 3rd dose of Salbutamol and Ipratropium bromide
Increased WOB present?

Yes

1-5 years
6 puffs Salbutamol MDI using spacer with a mouthpiece every 20 minutes.
OR
Nebulize with salbutamol 0.15mg/kg
and 125 -250 mcg Ipratropium bromide
every 20 minutes
Reduce frequency if improving
If no improvement, treat as life threatening

>6 years
10 puffs Salbutamol MDI using spacer with a mouthpiece every 20 minutes.
OR
Nebulize with salbutamol
5-11 year: 2.5 -5 mg, >12 years: 5 mg
and Ipratropium bromide >6 -11 years 250 -500 mcg
>12 years 500 mcg every 20 minutes
Reduce frequency if improving
If no improvement, treat as life threatening

No

Continue salbutamol puffs or nebulization every 1-2 hours as an inpatient

Second line IV treatment
Magnesium sulphate:50mg/kg (max 2g) over 20 minutes
Aminophylline: 5-6 mg/kg (max 300mg) over 30-60 minutes then 5 mg/kg 6 hourly with ongoing cardiac monitoring

***Systemic steroids**
Prednisone 1-2 mg/kg (40-60 mg max) once daily, Methyl prednisone 1 mg/kg (600 mg max) 6 hourly **OR**
Hydrocortisone 4 mg/kg (max 100mg) 6 hourly **OR**
Dexamethasone 0.1-0.3 mg/kg (maximum 20 mg) in 2-3 divided doses via slow injection or infusion

Figure 6: Acute severe asthma management algorithm

1.12.7 Management of life-threatening asthma

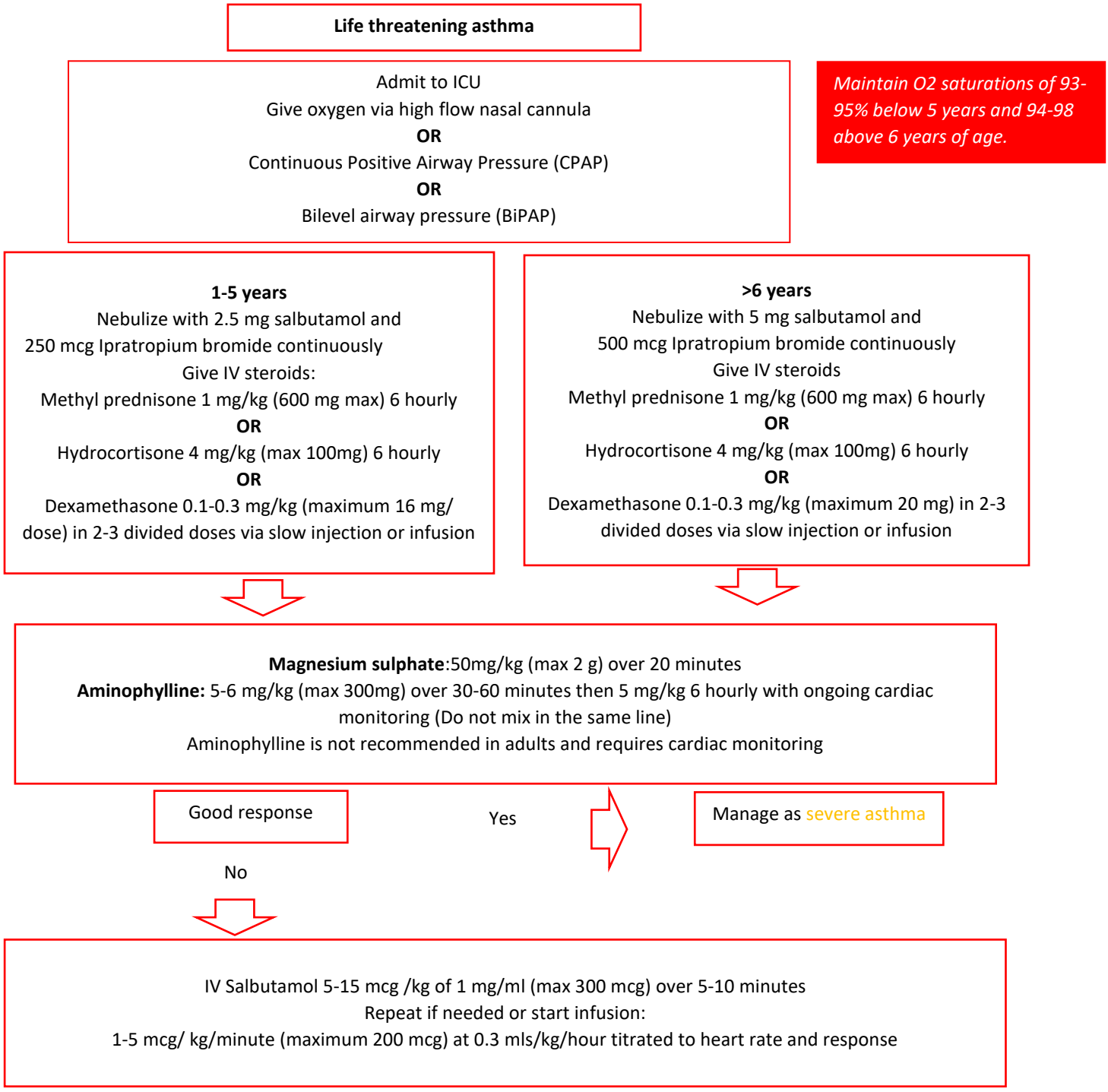


Figure 7: Acute life-threatening asthma management algorithm

The above acute asthma management algorithms were adapted with permission from the Royal Children's Hospital, Melbourne, Australia, Clinical Practice Guideline on Acute Asthma, [Internet, last updated July 2023); cited on 16th Mar 2026], Available from https://www.rch.org.au/clinicalguide/guideline_index/asthma_acute/

Note:

- Antibiotics are only needed if strong evidence of lung infections is present e.g. presence of fever, purulent sputum and signs of pneumonia on chest exam or CXR.
- Avoid the use of nebulized bronchodilators unless patient is hypoxic and requires oxygen or when giving both short acting bronchodilators and anticholinergics.

1.12.8 Testing in acute asthma patients:

- Mild to moderate exacerbations: pulse oximetry and PEF if available
- Severe or life-threatening asthma: pulse oximetry, arterial blood gases, +/- CXR and PEF is available.
- Uncontrolled asthma: pulse oximetry, PEF, spirometry, full blood count, assessment of sputum, environmental allergen assessment via skin prick or specific IgE testing.

1.12.9 Referrals

- Patients with acute severe or life-threatening asthma should be referred to health facilities level 4 and above for emergency management.
- Patients with history of frequent exacerbations > 4 in a year or one single severe or life-threatening exacerbations should be referred to pulmonology or allergy specialists for further management.

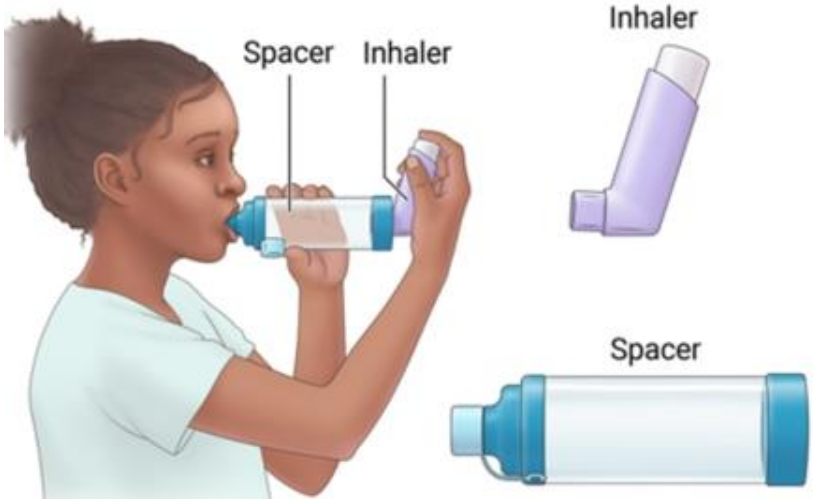


Figure 8: MDI and spacer with a mouthpiece

Suitable for patients above 6 years of age. Image credit myhealth.alberta.ca
https://share.google/RzeFTGzagF2r55P5h

Figure 9: Bottle spacer

This is made from 500 ml bottle that has a hole in the bottom with a hot knife for insertion of the MDI. Image credit – Marco Zampoli

Figure 10: Spacer and mask

Indicated for younger children, patients older than 6 years who are unable to follow instructions or have poor effort tolerance and therefore cannot do single breath technique with the mouthpiece.

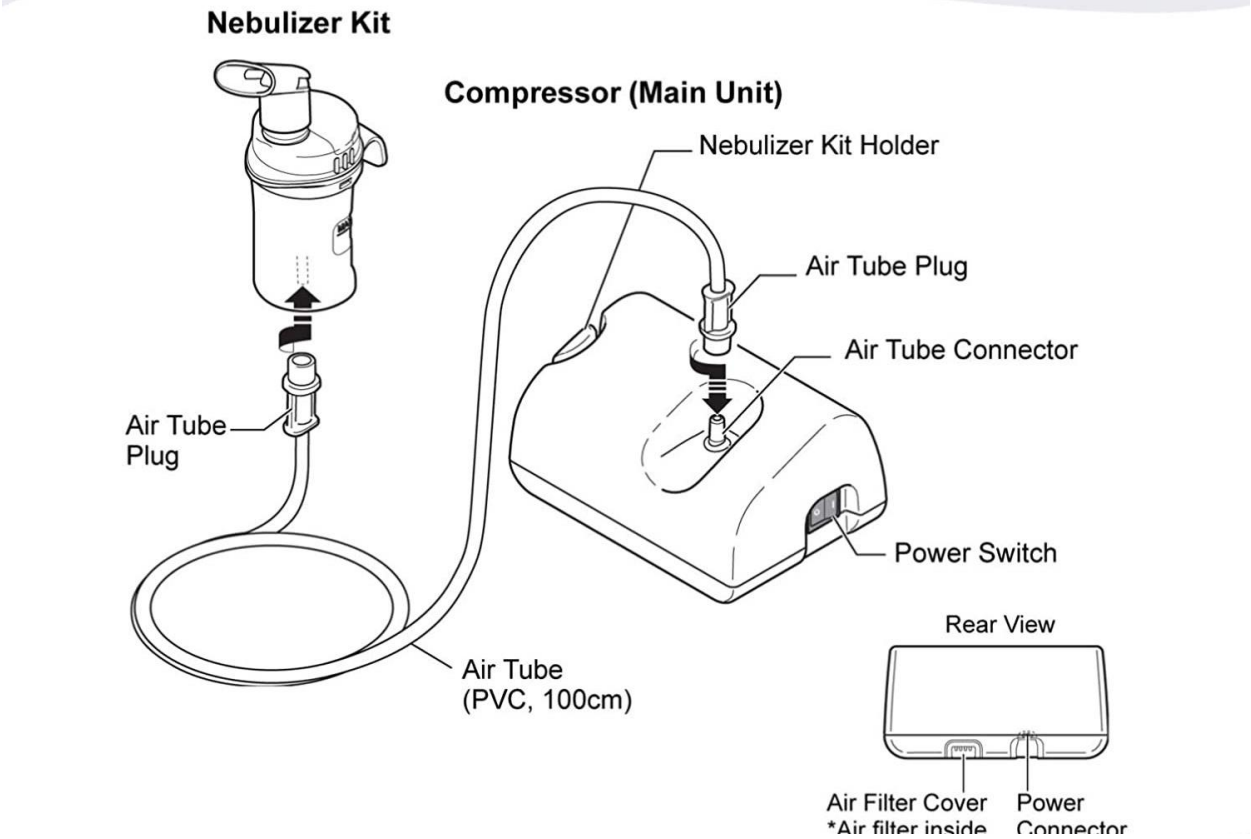


Figure 11: Example of a portable nebulizer machine

Image credit: https://harmainpharmacy.com/product/omron-ne-c28-compressor-nebulizer/

Follow-up of asthmatic patients requires assessment of control. The following tools can be used.

- The GINA assessment of control - https://ginasthma.org/
- The childhood asthma control test - https://www.asthmacontroltest.com/quiz/children-quiz/
- The asthma control test - https://www.asthmacontroltest.com/welcome/

- Take your controller medication every day whether you feel well or unwell.
- Visit the Doctor /Asthma Clinic twice a year even if asthma is well controlled.
- Take your medication / pumps / spacers with you to every doctors / nurses visit.
- Take this plan to each visit so it can be updated.
- Take the symptom or peak flow diary to each visit.

Doctors Phone No. .....

Hospital Phone No. .....

Date .....

Normal Peak Flow .....

Best Peak Flow .....

Asthma sufferers can:

- Have NO Symptoms.
- Have a normal lifestyle, play sport and sleep well.
- Have as few acute attacks as possible.
- Miss little or no school and work.
- Have your best possible peak flow.

What are the three zones?

Green Zone: *Your asthma is under control. This is where you want to be most of the time.*

Orange Zone - Caution: *Your asthma is not under control. The medication may need to be changed. Follow the advice in this plan and keep a symptom and medication diary. Make an appointment to see your doctor or asthma nurse.*

Red Zone – Red Alert: *Your asthma is critical! Follow the Red Zone Action and see a doctor immediately or go to the closest emergency room.*

Figure 12: Asthma action plan - a
Adapted from the National Asthma Education Program.

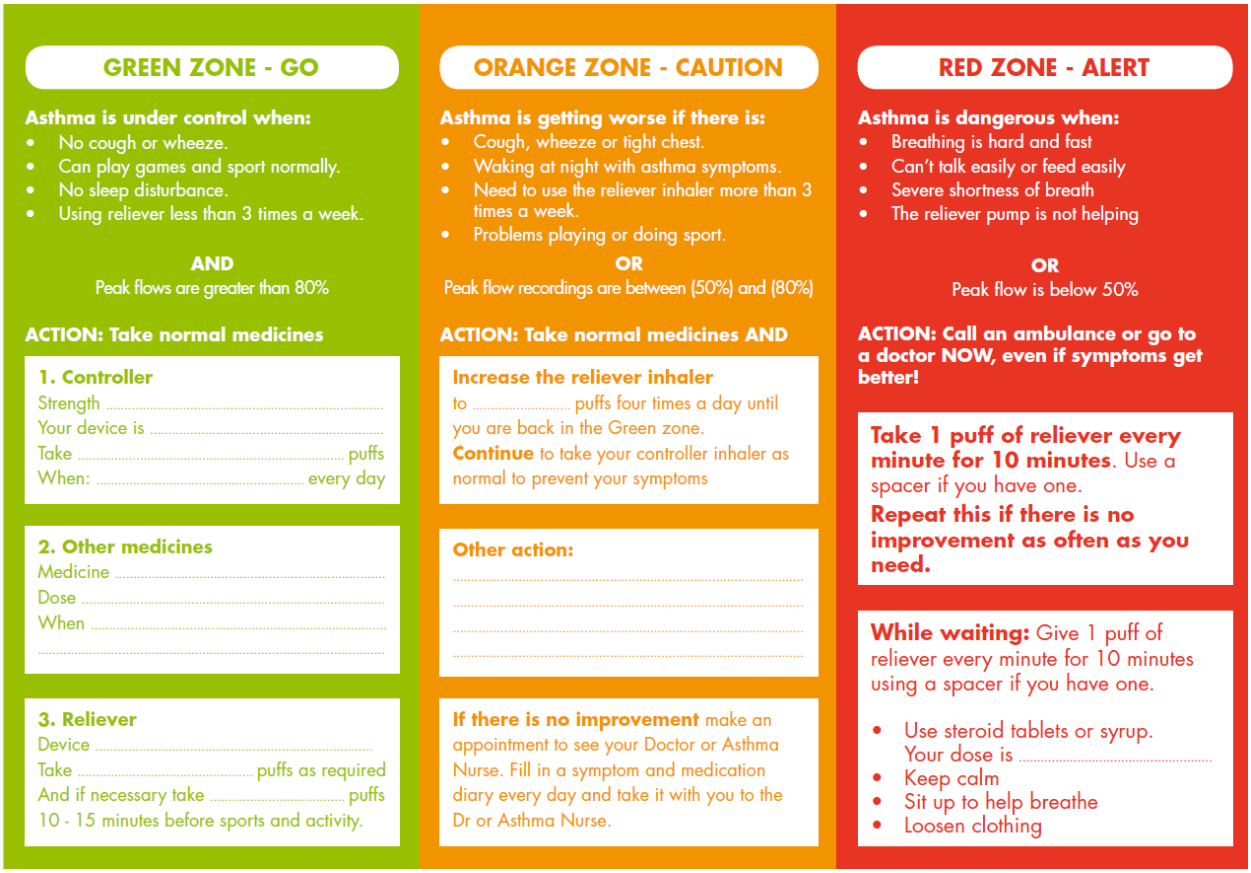


Figure 13: Asthma Action Plan - b

Adapted from the National Asthma Education Program.

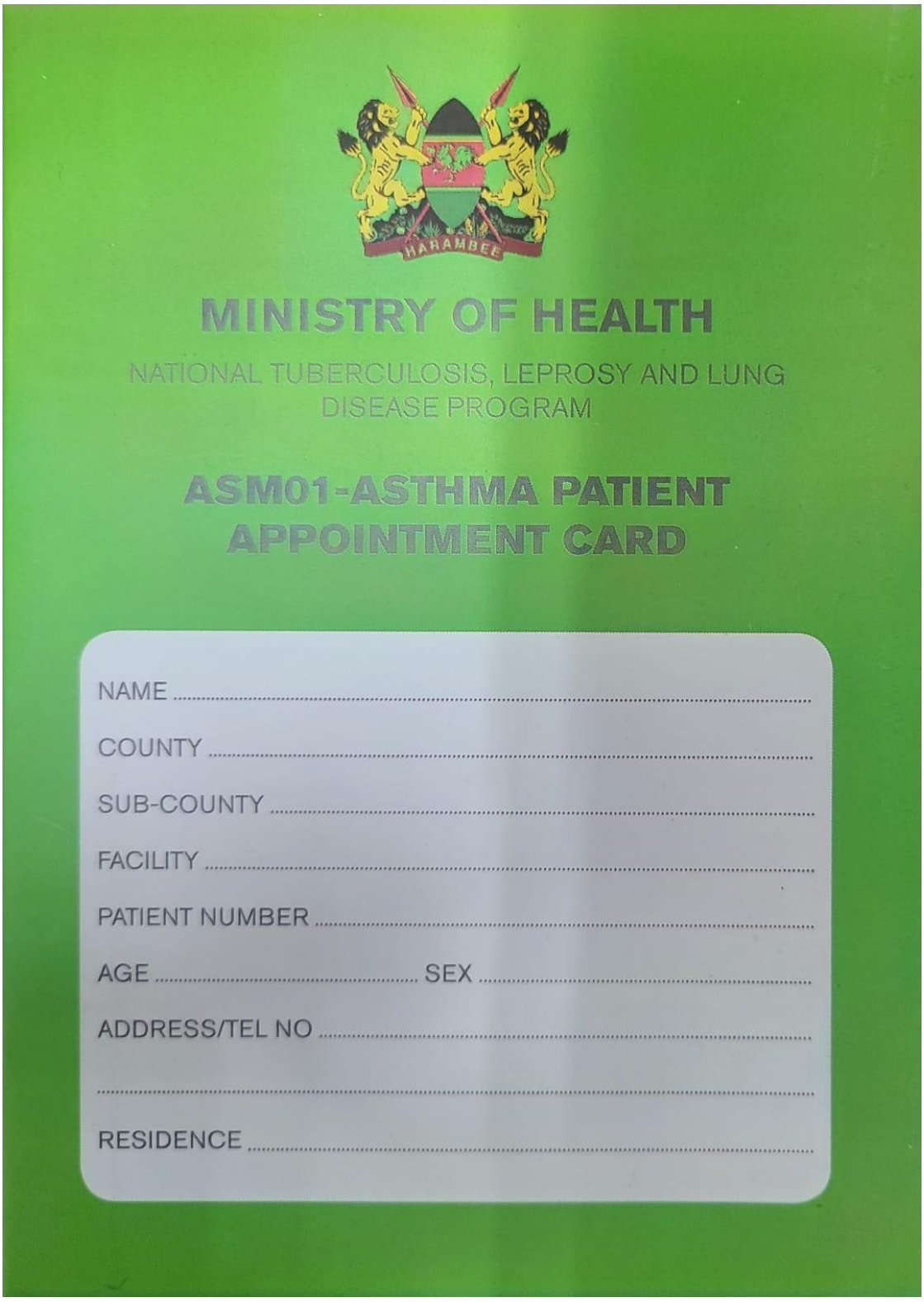


Figure 14: Appointment card for patient registration

1.13 Anaphylaxis

1.13.1 Definition

Anaphylaxis is a severe hypersensitivity reaction that may involve one vital or multiple systems. It is a potentially life-threatening reaction but is rarely fatal. Anaphylaxis is a clinical diagnosis.

1.13.2 Clinical Features

Symptoms are typically of rapid onset.

One of the 2 criteria below is required to make a diagnosis of anaphylaxis.

1. Sudden onset within *minutes to several hours* of mucocutaneous symptoms e.g., pruritus, flushing, generalized hives, swelling of the lips, tongue or uvula with at least one symptom of:
    - respiratory compromise; dyspnea, stridor, cough, wheeze, chest tightness, reduction in PEF or oxygen desaturation.
    - hypotension or reduction in end organ function; hypotonia / collapse, syncope or incontinence.
    - severe gastrointestinal symptoms (e.g., severe crampy abdominal pain, repetitive vomiting), especially after exposure to non-food allergens.
2. Acute onset of hypotension or bronchospasms or laryngeal involvement after exposure to a known or highly probable allergen for that patient (minutes to several hours), *even in the absence of typical skin involvement.*

Note:

- Hypotension is defined as a decrease in Systolic Blood Pressure (SBP) greater than 30% from that person's baseline, OR (Infants and children under 10 years: SBP less than (70 mmHg + [2 x age in years]) Adults and children over 10 years: systolic BP less than <90 mmHg.
- Excluding lower respiratory symptoms triggered by common inhalant allergens or food allergens perceived to cause "inhalational" reactions in the absence of ingestion.
- Laryngeal symptoms include stridor, vocal changes and odynophagia.

A detailed history of exposure and subsequent development of symptoms is required to determine the causative allergen. This is done after emergency treatment has been administered.

1.13.3 Causes/Triggers

IgE mediated anaphylaxis may be caused by various allergens such as:

- Foods: cows' milk, hens' egg, wheat, soya, fish, shellfish, peanuts, tree nuts. These are more common causes in children.
- Medications: antibiotics, Non-Steroidal Anti-inflammatory Drugs (NSAIDS), biologicals, immunotherapy. More common in adults.
- Environmental allergens are rare causes: pollen, animal dander
- Occupational allergens
- Insect venom
- Seminal fluid
- Latex
- Unknown

1.13.4 Management

EMERGENCY TREATMENT OF ANAPHYLAXIS Anaphylaxis is a life-threatening medical emergency. Prompt recognition of signs, symptoms and institution of appropriate management is critical.	
Step 1	Familiarize yourself with this protocol and rehearse it regularly.
Step 2	Remove exposure from trigger, if possible, e.g., disconnect from suspected IV medication
Step 3	Rapidly assess the patient's A, B, Cs and attach monitors
Step 4	Call for help, alert the senior physician If patient is decompensated call the resuscitation team: CODE BLUE
FIRST LINE TREATMENT	
Step 5	**Inject intramuscular undiluted 1mg/ml adrenaline 0.01ml/ kg (1:1000)** into the antero - lateral aspect of the thigh even through the clothes if necessary. < 6 months: 0.10 - 0.150 ml (0.1 – 0.15mg) 6 months - 6 years: 0.15 ml (0.15mg) Children 6 – 12 years: 0.3 ml (0.3mg) Above 12 years: 0.5 ml (0.5mg) Record time and dose given Repeat dose after 5 minutes if necessary. Most patients respond to one or two doses. If there is no response after two to three doses urgently contact the critical care team for intravenous adrenaline infusion (or refer as appropriate).
SECOND LINE TREATMENT	
Step 6	Place the patient in supine position and elevate lower extremities; fatality can occur within seconds if the patient stands or sits up suddenly. If in respiratory distress allow the patient to sit up but place in position outlined above if becomes dizzy or hypotensive. If unconscious, place in a recovery position and maintain the airway.

Step 7	Start supplemental oxygen if respiratory symptoms are present, the patient has prior history of asthma or repeated doses of adrenaline are required. If patient is wheezing, give salbutamol nebulization AFTER intramuscular adrenaline dose.
Step 8	Establish intravenous access with a large bore cannula if symptoms of shock are present and give a bolus of normal saline or Ringer's solution at *10 ml/kg* in children and *500-1000 ml* in adults then reassess the patient. (**AFTER** Intramuscular adrenaline dose or third spacing will occur*)*
Step 9	Initiate cardiorespiratory resuscitation if required at any time as per protocol
Step 10	Continuously evaluate and monitor the patient until condition improves.
Note that antihistamines and steroids are no longer recommended for the treatment of anaphylaxis	

Table 3: Emergency treatment of anaphylaxis

1.13.5 Admission

Admit in a level 4 and above health facility for a minimum of 24 hours to monitor possible biphasic reactions (a second delayed reaction which can occur 8-12 hours after initial reaction). While in the ward, assess for a recurrence of anaphylaxis. Monitor vital signs 4 hourly as well as urine output.

If anaphylaxis recurs, restart the protocol.

1.13.6 Recommendations/Referral

All patients with anaphylaxis should be referred to an allergy specialist for work up and continuation of care; education, allergy testing, adrenaline auto - injector (AAI) prescription, e.g. Epipen, and training on AAI use and registration for medical alert bracelet or chain.

1.14 Atopic dermatitis

1.14.1 Definition

Atopic dermatitis (AD) is a common inflammatory dermatological condition characterized by dry skin and pruritus (itching). It has a genetic predisposition (family history) and heralds the development of allergic conditions such as food allergies, asthma, allergic rhinitis and allergic conjunctivitis.

1.14.2 Clinical features

- Pruritus (cardinal feature)
- Acute changes include erythema, papules and vesicles with honey coloured crusting denoting a secondary infection.
- Chronic changes include lichenification (thickening and hyperpigmentation on the skin)
- Distribution of the lesions varies with age - infant (face especially cheeks and extensor surfaces); older children (flexures and skin creases). However, eczema can affect any part of the body.

1.14.3 Triggers

Below are the plausible causes of a ***flare*** in a patient with atopic dermatitis, arranged in no specific order:

- Climate - extremes of temperature, pollen season
- Use of hot or hard water when bathing
- Pets – animal dander or saliva
- House dust mite allergy
- Psychological stress
- Materials e.g., wool
- Soaps/ detergents
- Overdressing
- Infections:
    - Bacterial (Staphylococcus)
    - Viral (herpes simplex -eczema herpeticum, molluscum contangiosum warts)
    - Fungal skin infections.

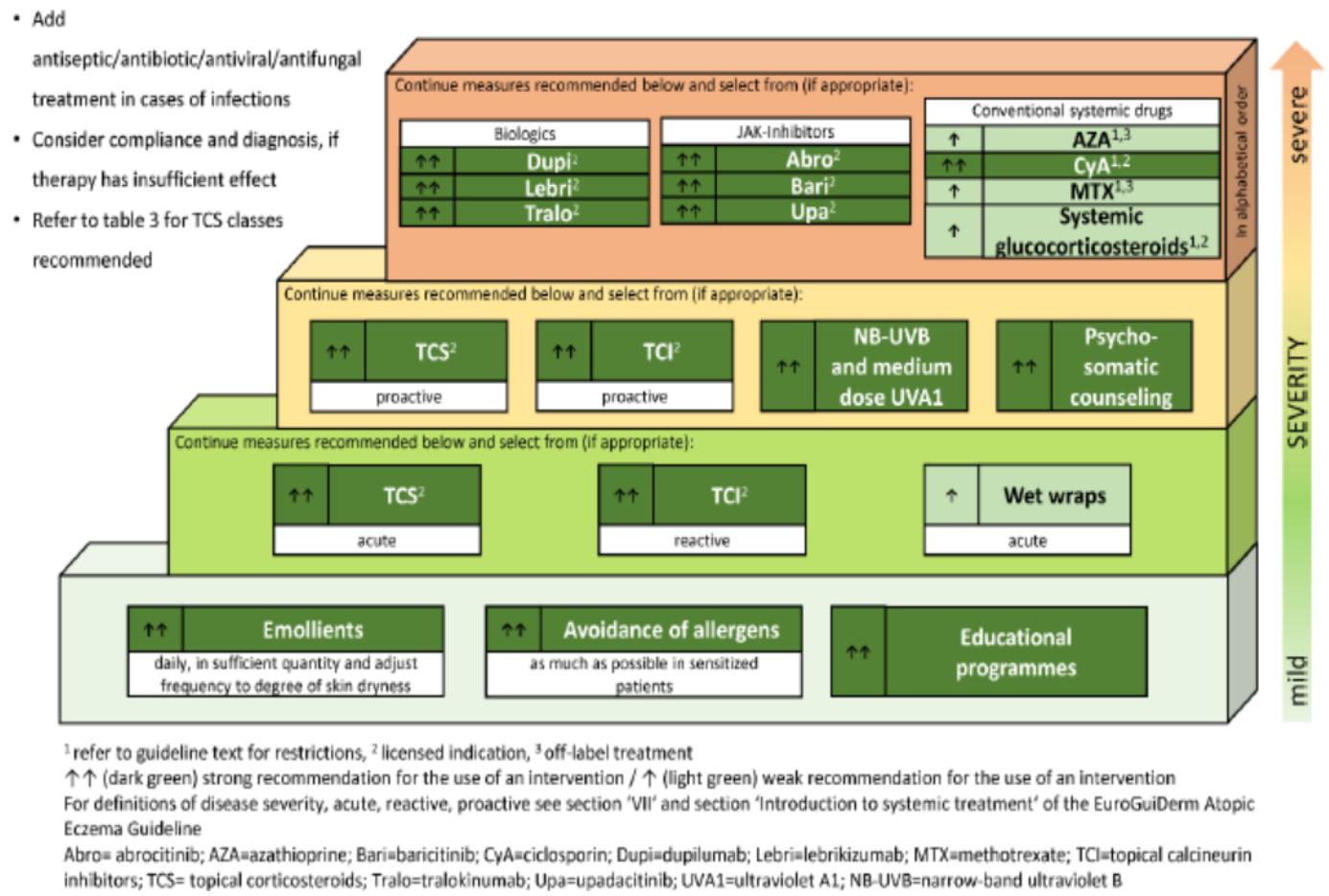
A patient who has periods of no AD while on their regular meal patterns is unlikely to have a food allergy. **As such, food avoidance without suggestive history will be detrimental to the child's growth. Food allergies can be considered for children with moderate to severe early onset AD,**

especially if they present with typical immediate reactions to a particular food. It can also be considered for refractory AD if history is suggestive.

*Note mimics of AD include fungal superficial *skin infections, scabies and psoriasis*.

1.14.4 Management

- Patient education on natural history and precipitating factors.
- Frequent use of moisturizers (such as emulsifying ointment) to keep the skin moist. *This is the mainstay of treatment.*
- Avoidance of precipitating factors.
- Avoid harsh (bar soaps)/medicated soaps. Use unscented mild soaps.
- Sedating antihistamines are used to induce sleep and not for the itch in AD unless they have acute flares mediated by Ig E.
- Use mild topical steroids (e.g., hydrocortisone cream for open skin or ointment for intact skin) for 1-2 weeks (see table for guidance).
- Treat any concurrent skin infections.



[1] refer to guideline text for restrictions, [2] licensed indication, [3] off-label treatment
↑↑ (dark green) strong recommendation for the use of an intervention / ↑ (light green) weak recommendation for the use of an intervention
For definitions of disease severity, acute, reactive, proactive see section 'VII' and section 'Introduction to systemic treatment' of the EuroGuiDerm Atopic Eczema Guideline
Abro= abrocitinib; AZA=azathioprine; Bari=baricitinib; CyA=ciclosporin; Dupi=dupilumab; Lebri=lebrikizumab; MTX=methotrexate; TCI=topical calcineurin inhibitors; TCS= topical corticosteroids; Tralo=tralokinumab; Upa=upadacitinib; UVA1=ultraviolet A1; NB-UVB=narrow-band ultraviolet B

Figure 15: EuroGuiDerm Guideline for AE. Stepped care plan for adults

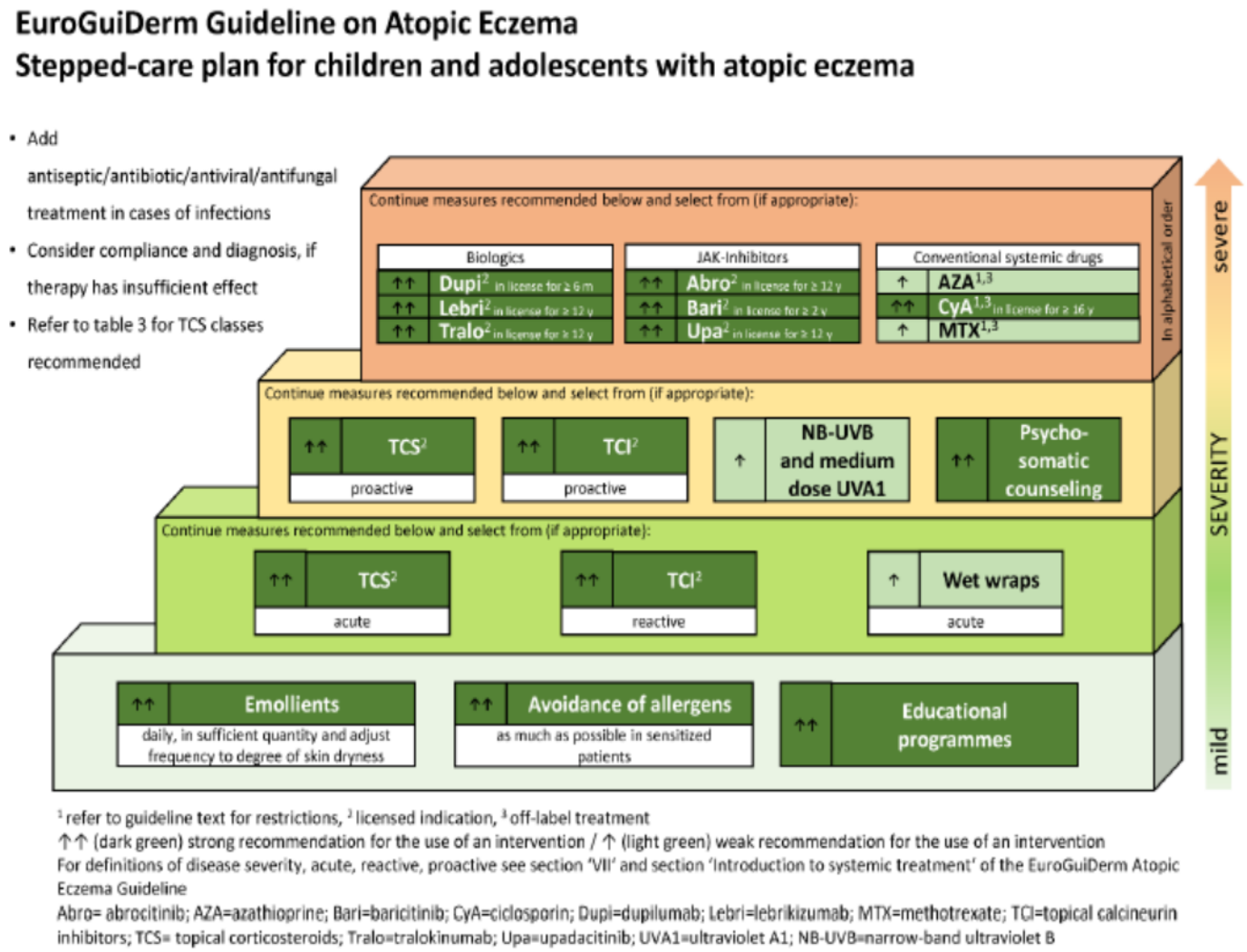


Figure 16: EuroGuiDerm Guideline for AE. Stepped care plan for children & adolescents

Table 4: List of available corticosteroids

Potency group	Corticosteroid	Concentration (%)	Formulation	Coopman classification
Group 1 (highest)	Augmented betamethasone dipropionate	0.05	Ointment, gel, lotion	D1
	Clobetasol propionate	0.05	Ointment, cream, gel, lotion	D1
	Fluocinonide	0.1	Cream	B
	Halobetasol propionate	0.05	Ointment, cream, lotion, foam	
Group 2 (high)	Amcinonide	0.1	Ointment	D2
	Betamethasone dipropionate	0.05	Ointment, augmented cream	D1
	Clobetasol propionate	0.025	Cream	D1
	Desoximetasone	0.25	Ointment, cream, spray	C
		0.05	Gel	
	Diflorasone diacetate	0.05	Ointment, cream	D1
	Fluocinonide	0.05	Ointment, cream, gel, solution	B
	Halcinonide	0.1	Ointment, cream, solution	B
	Halobetasol propionate	0.01	Lotion	
Group 3 (medium-high)	Amcinonide	0.1	Cream, lotion	B
	Betamethasone dipropionate	0.05	Cream	D1
	Betamethasone valerate	0.1	Ointment	D1
		0.12	Foam	
	Desoximetasone	0.05	Ointment, cream	C
	Diflorasone diacetate	0.05	Cream	D1
	Fluocinonide emollient	0.05	Cream	B
	Fluticasone propionate	0.005	Ointment	D1
	Mometasone furoate	0.1	Ointment	D1
	Triamcinolone acetonide	0.5	Ointment, cream	B
Group 4 (medium)	Betamethasone dipropionate	0.05	Spray	D1
	Clocortolone pivalate	0.1	Cream	C
	Fluocinolone acetonide	0.025	Ointment	B
	Fluticasone propionate	0.05	Cream	D1
	Hydrocortisone valerate	0.2	Ointment	A
	Mometasone furoate	0.1	Cream, lotion, solution	D1
	Triamcinolone acetonide	0.1	Ointment, cream	B
		0.05	Ointment	

Potency group		Corticosteroid	Concentration (%)	Formulation	Coopman classification
Group 5 (medium-low)		Betamethasone dipropionate	0.05	Lotion	D1
		Betamethasone valerate	0.1	Cream	D1
			0.05	Ointment	
		Desonide	0.05	Ointment	B
		Fluocinolone acetonide	0.025	Cream	B
		Fluticasone propionate	0.05	Lotion	D1
		Hydrocortisone butyrate	0.1	Ointment, cream, lotion, solution	D2
		Hydrocortisone valerate	0.2	Cream	D2
		Prednicarbate	0.1	Ointment, cream	D2
		Triamcinolone acetonide	0.025	Ointment	B
			0.1	Lotion	
		Alclometasone dipropionate	0.05	Ointment, cream	D1
		Betamethasone valerate	0.1	Lotion	D1
			0.05	Cream	
Group 6 (low)		Desonide	0.05	Cream, gel, lotion, foam	B
		Fluocinolone acetonide	0.01	Cream, solution, shampoo	B
		Triamcinolone acetonide	0.025	Cream, lotion	B
Group 7 (lowest)		Hydrocortisone base	2.5	Ointment, cream, solution	A
			2	Lotion	
			1 (OTC)	Ointment, cream, gel, lotion, spray	
			0.5 (OTC)	Ointment, cream	

Table 5:Atopic dermatitis impact and treatment.

Adapted from AAAAI/ACAAI JTF Atopic Dermatitis Panel, 2023.

Skin/Physical Severity Atopic eczema)		**Impact on quality of life**	**Treatment**
Clear	Normal skin, no evidence of active atopic eczema	None	None
Mild	Areas of **dry skin**, infrequent **itching**, (with or without small areas of **redness**)	Little impact on everyday activities, sleep and psychosocial well-being.	Emollients, mild topical corticosteroids
Moderate	Areas of dry skin, infrequent itching, redness (with or without excoriation and areas of **localized skin thickening**)	Moderate impact on everyday activities, psychosocial wellbeing. Frequently disturbed sleep.	Initiate treatment with emollients, moderate topical corticosteroids Refer to Dermatologist/ Allergist as detailed below.
Severe	Widespread areas of dry skin, incessant itching, redness (with or without excoriation, **extensive skin thickening, bleeding, oozing, cracking and alternation of pigmentation**)	Severe impact on everyday activities and psychosocial well-being. Nightly loss of sleep	Refer to Dermatologist/ Allergist

1.14.5 Recommendations/Referrals

Refer patients to a dermatologist if, in no specific order:

- Poor or no response to treatment after one month and/or
- Body surface involved is >30%.

- Worsening of symptoms at any time
- Profound impact on quality of life
- Recurrent skin infections
- Suspected contact allergy
- Diagnosis is uncertain

Appropriate referral avoids misuse of topical or systemic steroids, allows for evaluation of other differentials and optimizes care.

Refer to allergist for skin prick testing/specific IgE levels for:

- patients with moderate-severe eczema in whom optimized treatment has failed to control the disease and/or;
- patients presenting with associated immediate reactions e.g., urticaria/angioedema.

Note that patients with atopic eczema usually have elevated total IgE and positive sensitization tests (SPT and sIgE) which are not clinically relevant in majority of cases. Therefore, correlation with clinical history is mandatory.

1.15 Contact Dermatitis

1.15.1 Definition

Acute or chronic inflammation produced by substances encountering the skin and resulting in irritant or allergic (immune mediated) reactions at the area of contact.

1.15.2 Classification

- **Primary irritant contact dermatitis (ICD):** caused by substances like acids, alkalis, soaps, detergents, acetone, etc.
- **Allergic contact dermatitis (ACD):** topical drugs, plants (poison ivy), shoes (rubber), leather, clothing, metallic compounds (nickel), dyes (e.g. Para-phenylenediamine, PPD hair dye), glue, agrochemicals and cosmetics (including nail polish). Sensitivity to latex in gloves is a particular problem for many health workers, and sensitivity to latex condoms may preclude their use by some men.

1.15.3 Clinical Features

Lesions include acute vesicles, weeping subacute erythema, dry scaly papules or chronic lichenified (thickened) excoriated and hyper pigmented rashes. The lesions take the shape of contact with the offending item, for example shoes on the feet (termed moccasin distribution), watches cause lesions around the wrist, gloves on hands, etc., but may be asymmetric without any pattern.

Table 6: Differences between ICD and ACD

Feature	**ICD**	**ACD**
Frequency	80 %	20 %
Concentration of the agent	More critical	Less critical
Mechanism/trigger	Nonimmune; sensitization not required; damage to keratinocytes	Immune; sensitization required; interaction of antigen with primed T-cells
Atopic predisposition	↑	↓
Diagnostic test	None	Patch tests
Reaction delay after contact	Initially prolonged and/or repeated exposure	Many **hours** to 5 to 6 days (primary vs secondary)
Sharp demarcation	Often, **typical**	May occur
Time for clinical resolution	Frequently diminished by **96 hours**	Many **days**
Itching	Mild to moderate	Severe
Lesions beyond primary exposure site	Usually limited to exposure site	Can be widespread or generalized
Dryness and scaling	Present in early phase	Present in chronic phase

Hand dermatitis common	Dorsum and hand/webs of fingers	Palm involvement

1.15.4 Management

- Identify and remove causative agents (including affected clothing).
- Wash off the affected area with clean water.
- Drain large blisters but do not break or remove tops (roofs).
- Administer analgesics and/or antihistamines as needed.
- Apply a mild topical steroid (e.g. hydrocortisone) ointment for dry lesions and cream for wet lesions.

1.15.5 Recommendations/Referrals

Refer to dermatologist/allergist for patch test.

1.16 Drug Allergies

1.16.1 Definition

All medications have the potential to cause adverse drug reactions (ADR), which are unintended harmful events resulting from the use of a medicinal product. Adverse drug reactions are a common occurrence. Drug allergies are adverse drug reactions that are mediated by immunological mechanisms; either IgE-mediated (type 1) or non-IgE-mediated hypersensitivity reactions.

1.16.2 Classification

Adverse drug reactions are broadly classified as predictable and unpredictable. Majority are predictable; these are dose-dependent and caused by the pharmacologic actions of the drug. Unpredictable reactions are uncommon, independent of dose and unrelated to the pharmacologic effects of the drug. Unpredictable reactions include **drug intolerance, idiosyncratic reactions, drug allergies and other non-immunological drug hypersensitivities.**

1.16.3 Triggers/Causes of Drug Allergies

Common drugs associated with allergic reactions include beta-lactam antibiotics (penicillins, cephalosporins), and NSAIDs.

1.16.4 Clinical features

Table 7: Clinical features of drug allergies

Immediate, rapidly evolving reactions
Onset usually less than one hour after drug exposure (up to 6 hours) Manifestations: • IgE-mediated anaphylaxis • Urticaria or angioedema without systemic features • Exacerbation of asthma (e.g., with NSAIDs)
Non-immediate reactions without systemic involvement Onset 6-10 days after first drug exposure or within 3 days of second exposure Manifestations: • Delayed drug exanthem - generalized maculopapular rash appearing a few days after drug initiation and resolving a few days after discontinuation • Fixed drug eruptions - hyper pigmented plaques that recur at the same site with each exposure • Allergic contact dermatitis - dermatitis in an area of cutaneous contact with the medication that evolves over days, in a sensitized individual • Drug-induced lupus erythematosus with cutaneous symptoms: photo-distributed erythematous plaques

Non-immediate reactions with systemic involvement
• **Serum-sickness-like reaction**- urticarial rash, fever, arthralgia & lymphadenopathy one to three weeks after starting medication, or earlier in a sensitized individual. • **Stevens-Johnson Syndrome (SJS) or Toxic Epidermal Necrolysis (TEN)** - mucosal involvement, fever, cutaneous target and bullous lesions (SJS:10% epidermal detachment; **SJS/TEN overlap**: 10-30% epidermal detachment; **TEN: ≥ 30% epidermal detachment)**; liver, lungs or kidney involvement. Onset 7-14 days after first drug exposure or within 3 days after second exposure. • **Drug reaction with eosinophilia and systemic symptoms (DRESS)**: Fever, eosinophilia, lymphadenopathy, liver and/or renal dysfunction and cutaneous eruption. Onset 2-6 weeks and up to 12 weeks after first drug exposure or within 3 days of second exposure and persisting for weeks after stopping the medication. • **Acute generalized exanthematous pustulosis (AGEP)**: Widespread pustules, fever and neutrophilia. Onset 3-5 days after first drug exposure. • Sudden onset myalgia, fever, arthralgia, malaise. Onset several weeks after drug initiation. • Other Hematologic (cytopenia), hepatic (hepatitis, cholestatic jaundice), renal (interstitial nephritis), pulmonary (pneumonitis, fibrosis), vasculitis. Variable time of onset.

1.16.5 Diagnosis

Assessment of a patient presenting with a possible drug allergy begins with a medical history and physical examination. A review of the treatment sheet is also helpful. The information obtained may provide clues to aid in identification of the etiology of the reaction, as well as provide details suggesting the possible type of drug-induced allergic reaction.

Table 8: Diagnosis of drug allergies

The reaction is more likely to be caused by drug allergies if:	The reaction is less likely to be caused by drug allergies if:
It occurred during or after use of the drug The drug is known to cause that type of reaction The patient has previous history of a similar reaction to that drug or drug class	There is another possible explanation for the person's symptoms (e.g., has similar symptoms when not taking the drug) The person has only gastrointestinal symptoms.

1.16.6 Investigations

Routine laboratory tests (complete blood count, liver enzymes, renal function) may help determine organ involvement in the situation of severe, non-immediate reactions.

1.16.7 Management

- Avoid suspected offending drugs until the patient has been assessed by a dermatologist/allergist.
- Provide an appropriate alternative to the offending drug. However, if an alternative drug is unknown, consult the primary prescriber of that specialty or subspecialty e.g. a physician

1.16.8 Recommendations/Referral

SJS/TEN: immediate direct referral to dermatologist in a level 5 or 6 hospital.

Referral to dermatologist / allergy specialist for multidisciplinary team management is indicated for:

- Suspected anaphylactic reaction
- Severe non-immediate cutaneous reaction
- Diagnostic uncertainty or allergen drug not identified.

Document suspected or confirmed drug allergy in the patient's medical records, providing information on the drug name, the signs, symptoms and severity of the reaction and the date when the reaction occurred.

Discuss the suspected or confirmed drug allergy with the patient (and their family or caregiver) and provide information on drugs or drug classes to avoid in future while suggesting safe alternatives drugs that may be used.

De-labelling drug allergy is required when a suspected drug allergy has been ruled out and should be documented clearly in the patient's records.

1.17 Food Allergies

1.17.1 Definition

These are immune-mediated adverse reactions to specific foods. These are different from food intolerances which are caused by enzyme deficiencies or the characteristics of the food itself.

1.17.2 Classification

Food allergies (FA) fall into two groups:

- IgE-mediated
- Non-IgE mediated (mixed/cell-mediated).

1.17.3 Clinical Features

Below is a table showing signs and symptoms of possible food allergy.

Table 9: Signs and symptoms of possible food allergy

IgE mediated	Non-IgE mediated
The skin	
Acute urticaria – localized or generalized	Atopic eczema flares as delayed reactions
Acute angioedema – most commonly of the lips, face and around the eyes	
Atopic eczema flare can be triggered in people with eczema	
The Gastrointestinal system	
Itchy mouth/throat	***Food protein induced proctocolitis (FPIP)*** Bloody, mucoid stools in otherwise well infant. More common in formula fed infants < 3 months of age
Nausea/vomiting	***Food protein enteropathy (FPE)*** Abdominal discomfort, mucoid diarrhea and failure to thrive in infants
Abdominal pain	***Food protein induced proctocolitis (FPIP)*** Bloody, mucoid stools in otherwise well infant < 3 months of age
	Food protein induced enterocolitis (FPIES) Sudden onset of vomiting, usually multiple episodes followed by diarrhea within 2-4 and 5-10 hours of ingestion respectively in children 1-2 years of age. Results in dehydration or shock

	Eosinophilic esophagitis (EOE) As discussed in section 2.10
	Food protein induced gastroesophageal reflux disease (FPGORD) Episodes of vomiting associated with difficulty feeding, Sandifer syndrome (intermittent back and neck arching, recurrent cough in infants < 6 months
The respiratory system (usually in combination with one or more of the above symptoms and signs)	Vomiting, diarrhea or abdominal pain which only occur after consuming the relevant food allergen and don't occur on other occasions
Acute upper respiratory tract symptoms (nasal itching, sneezing, rhinorrhea or congestion [with or without conjunctivitis])	Faltering growth in an infant who is receiving cow's milk formula, where other causes of faltering growth have been excluded (e.g., insufficient intake)
Acute lower respiratory tract symptoms (cough, chest tightness, wheezing or shortness of breath)	
Other	
Signs or symptoms of anaphylaxis or other systemic allergic reactions	

1.17.4 Management

- A detailed history is the cornerstone for FA diagnosis. The following questions must be answered:
    - What is the time from exposure to development of symptoms?
    - *IgE mediated reactions occur within 2 hours of exposure and are usually obvious. Non-IgE mediated reactions occur later, most commonly 2-4 hours after exposure, and mainly cause gastrointestinal symptoms.*
    - What symptoms does the patient present with? *To be able to classify the likely mechanism.*
    - Does the patient develop symptoms with each exposure to that food in the original or other forms? *To determine reproducibility.*
    - How much of the food was ingested before symptoms occurred? *To determine if an allergy is likely. Allergies usually occur at normal or lower doses, tolerated by most of the population.*

- Physical location and activities involved in before symptoms occurred? *For possible food dependent exercise induced anaphylaxis or likely additive allergy if the same food is tolerated at home but not elsewhere.*

- Thorough physical examination also helps identify the consequences of food avoidance in children and adults
- If anaphylaxis is suspected - treat as per protocol. (See section on anaphylaxis).
- IV fluids for entities presenting with hypotension (e.g. Food Protein Induced Enterocolitis (FPIES).
- Mild – moderate immediate reactions are treated with 2nd generation antihistamines.

1.17.5 Recommendation/Referral

1. **Avoid blind food elimination diets unless guided by an allergist / paediatric gastroenterologist. This is to avoid negative impact on a child's growth and development as well as undernutrition in adults.**
2. In a breastfed infant or young child with immediate allergic reactions to specific foods, reactions to these foods in the mother's diet are uncommon, therefore, maternal dietary restriction is generally not necessary.
3. It is important to note that *results of allergy testing should be interpreted in the context of the history given.* Positive allergy test (skin prick tests and serum sIgE) results need to be interpreted with caution, since it may be clinically irrelevant when no symptoms are elicited within 2 hours of ingestion. Oral food challenges for diagnosis of IgE mediated food allergies should only be conducted by allergists and not in primary care or community settings.
4. The management of non-IgE mediated food allergy is based on the results of an allergy-focused clinical history. Trial elimination of the suspected allergen (usually for 2-6 weeks) is followed by re-introduction to confirm the suspected allergen. This should be done by an allergy specialist.
5. Allergen avoidance of known offending food and associated cross-reactive allergens is the mainstay of management. If unknown, refer to an allergist for allergy evaluation.
6. Referrals are required for patients with/who in no order.
    - Failure to thrive
    - Presumed multiple allergens
    - Require specialized allergy testing
    - Severe reactions – anaphylaxis or FPIES

- Suspected eosinophilic esophagitis (EOE)
- Difficulty identifying appropriate alternatives
- Patients with ongoing reactions despite elimination

1.18 Cow Milk Allergies

1.18.1 Definition

Cow milk allergy (CMA) is an immune mediated hypersensitivity reaction to cow milk proteins. It is both IgE and non-IgE mediated and most common in the first few years of life with a prevalence of 2-3%. Studies show that CMA is over diagnosed. Cow milk allergy was confirmed in less than 1% of infants whose diagnosis was based on history of vomiting (22%) and atopic eczema (43%).

1.18.2 Clinical features

The presentation is wide due to the different underlying mechanisms. Of importance is the true reproducibility of symptoms upon exposure within expected timelines based on the underlying mechanism.

1.18.3 Cause

Cow milk contains proteins in form of casein (80%) and whey (20%). The protein sequences in the casein fractions are linear and unchanged by heat, acidification or enzymatic action and are mostly responsible for reaction to all forms of dairy. Patients with primarily whey reactions can tolerate well-cooked dairy in form of biscuits or queen cakes/ muffins and are likely to outgrow their allergies earlier due to the conformational nature of their allergens that can be altered with exposure to heat, acidification or enzymatic action. The amount of cow milk protein in breast milk is rarely enough to cause IgE mediated reactions. *Cow milk forms an integral part of diet for young children and should not unnecessarily be withdrawn due to its nutritional benefits.*

1.18.4 Presentation

Below is a table showing signs and symptoms of possible cow milk allergy.

Table 10:Signs and symptoms of cow milk allergies

IgE mediated
Skin
Acute urticaria – localized or generalized
Acute angioedema – most commonly of the lips, face and around the eyes
Atopic eczema flare in eczema patients
Gastrointestinal system
Itchy mouth/throat
Nausea/vomiting
Abdominal pain
Respiratory system

Acute upper respiratory tract symptoms (nasal itching, sneezing, rhinorrhea or congestion [with or without conjunctivitis])
Acute lower respiratory tract symptoms (cough, chest tightness, wheezing or shortness of breath)
Other
Anaphylaxis as discussed in section 2.5
Non-IgE mediated
Skin
Atopic eczema flares as delayed reactions
Gastrointestinal system
Food protein induced proctocolitis (FPIP) Bloody, mucoid stools in otherwise well infant. More common in formula fed infants < 3 months of age
Food protein enteropathy (FPE) Abdominal discomfort, mucoid diarrhea and failure to thrive in infants
Food protein induced enterocolitis (FPIES) Sudden onset of vomiting, usually multiple episodes followed by diarrhea within 2-4 and 5-10 hours of ingestion respectively in children 1-2 years of age. Results in dehydration or shock
Eosinophilic esophagitis (EOE) As discussed in section 2.10
Food protein induced gastroesophageal reflux disease (FPGORD) Episodes of vomiting associated with difficulty feeding, Sandifer syndrome (intermittent back and neck arching, recurrent cough in infants < 6 months
Food protein induced constipation (FPC) At least 2 of the following ROME IV criteria for constipation for 1 month that does not respond to standard treatment. More common after 6 months of age. • Straining during more than ¼ (25%) of defecations • Lumpy or hard stools (Bristol Stool Form Scale 1-2) more than ¼ (25%) of defecations • Sensation of incomplete evacuation more than ¼ (25%) of defecations • Sensation of anorectal obstruction/blockage more than ¼ (25%) of defecations • Manual manoeuvres to facilitate more than ¼ (25%) of defecations (e.g., digital evacuation, support of the pelvic floor) • Fewer than three spontaneous bowel movements (SBM) per week • Loose stools are rarely present without the use of laxatives • Insufficient criteria for irritable bowel syndrome

1.18.5 Management

Determined by age and classification of CMA. Refer to section *2.8.4* and *2.8.5* for details on evaluation and approach to management of food allergies. Note that skin testing or sIgE in serum is not useful in Non-IgE mediated reactions.

Partially hydrolyzed formulas denoted HA are not suitable for patients with confirmed CMA. Only extensively and completely hydrolyzed (amino acid/ elemental) formulas are recommended. These are expensive, not widely available and unpalatable for most children and therefore should be used indiscriminately. Allergist support is usually required.

Goat milk/formulas are not acceptable options due to the high cross reactively among mammalian milks. Soya formula is an option for IgE mediated reactions. Camel and plant-based milks are more available and can be used though they contain less calories than cow milk, therefore careful follow up by the nutritionist is required to ensure adequate nutritional support. Plant-based milks are generally not recommended for children below two years of age, more so in infants, when appropriate nutritional follow-up is lacking. Mothers on elimination of dairy should be prescribed calcium supplements that offer at least 1 g of elemental calcium a day.

1.18.6 Recommendations

Refer to allergist for patients with (in no order):

- Failure to thrive
- More than one food allergy is suspected
- Severe reactions – anaphylaxis or FPIES
- Suspected eosinophilic esophagitis (EOE)
- Difficulty identifying appropriate alternatives
- Patients with ongoing reactions despite elimination

1.19 Eosinophilic Esophagitis

1.19.1 Definition

Eosinophilic esophagitis (EOE) is a chronic allergic condition with a genetic component. It is relatively rare, affecting about 1 in 3000 people and is most common in young adult males. It is rarer in children, and very rare indeed in young children and infants. Symptoms vary with age as shown below and most patients are identified after endoscopy. Definitive diagnosis requires endoscopy and biopsies of the esophagus.

1.19.2 Clinical Features

Symptoms vary with age as listed below in no specific order.

Children:

- Vomiting
- Dysphagia
- Food refusal
- Failure to thrive
- Chest/ epigastric pain
- Food impaction: take long to complete a meal or take small bits and drink fluids with meals

Adults:

- Globus sensation or food impaction with solids
- Retrosternal chest pain
- Upper abdominal pain
- Intractable heartburn

1.19.3 Causes/Triggers

Cow milk, soya, egg, wheat, fish, shellfish, peanuts and tree nuts. Exclude more common causes of feeding difficulties in children e.g., fussy eating, behavioral or sensory processing issues. Other differentials include gastro-esophageal reflux, coeliac disease, and inflammatory bowel disease.

1.19.4 Recommendation/Referral

- Refer to gastroenterologist for endoscopy
- Refer to allergist to guide further management of food elimination
- **Elimination diets must not be performed unless guided by a gastroenterologist or an allergist.**

1.20 Urticaria and Angioedema

1.20.1 Definition

Urticaria are superficial skin swellings, also known as hives/wheals/welts. **Angioedema** is a deeper tissue swelling. Patients may present with both lesions at the same time.

Clinical Features of Urticaria

Itchy, red, raised, flat lesions that:

- vary in size and shape
- typically resolve within 24 hours depending on the trigger.
- are migratory in nature and can affect any part of the body at any one time.

Acute hives occur for short durations usually < 6 weeks. Persistent lesions > 6 weeks are considered chronic, which may be recurrent over months or years.

1.20.2 Clinical Features of Angioedema

It is more painful than itchy and lasts longer than urticaria.

1.20.3 Causes/Triggers

1. **Allergens**: food, aeroallergens, therapeutics, insects, cosmetics, latex & occupational allergens can all cause acute urticaria, but are not usually relevant to chronic urticaria.
2. **Physical stimuli**, such as deep or light pressure, cold, heat, exercise, vibration, water or sun exposure are all relevant triggers for some people with inducible chronic urticaria.
3. **Infections**: bacterial, viral, parasitic can trigger acute infection-associated urticaria/angioedema, typically lasting 5-10 days.
4. **Autoimmune disorders**
5. **Idiopathic urticaria** is the commonest form of chronic urticaria and has no identifiable cause. It can be associated with the physical triggers as discussed above in the same individual.

1.20.4 Management

- Treat with second generation antihistamines.
- Investigations - haemogram, urinalysis, stool microscopy
- **Avoid use of systemic and topical steroids.**

1.20.5 Recommendations/Referrals

- Refer immediately to level 4 and above if symptoms of systemic involvement are present due to risk of anaphylaxis
- Refer to dermatologist/ allergist for those with poor response to treatment, requiring further investigations and management

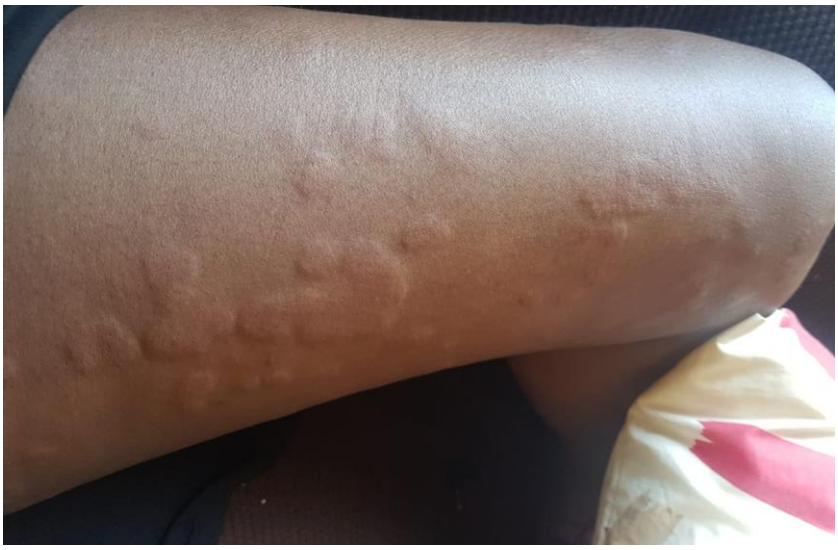


Figure 17: Hives or wheals

Image credit, Dr. Priya Bowry.

Figure 18: Lip angioedema

Image credit, Dr. Priya Bowry.

1.21 Hereditary Angioedema (HAE)

1.21.1 Definition

This is a non-allergic **bradykinin mediated** angioedema, unresponsive to antihistamines and steroids. It typically occurs in a person with a family history of angioedema. It is a rare autosomal dominant condition caused by deficient or dysfunctional complement 1 inhibitor (C1 Inhibitor). This results in the uncontrolled production of bradykinin resulting in massive swelling. **HAE will not respond to antihistamines of any kind (unlike allergic angioedema).**

1.21.2 Clinical Features

- Large painful swellings occurring gradually over days and are slow to resolve over several days, affecting any part of the body.
- Abdominal and genital symptoms frequent (swelling, vomiting and severe pain)
- **Laryngeal involvement presents as medical emergencies**
- Prodromal erythema marginatum rash may occur
- Family history of angioedema/sudden deaths

Note that:

- Urticaria and itching is usually absent
- Unresponsive to antihistamines/steroids/adrenaline

1.21.3 Classification

- HAE Type 1
- HAE Type 2
- HAE with normal C1 inhibitor

1.21.4 Triggers

- Stress
- Infections
- Physical trauma
- Surgery e.g. dental procedures
- Oral contraception/hormonal therapy/ menses

1.21.5 Management

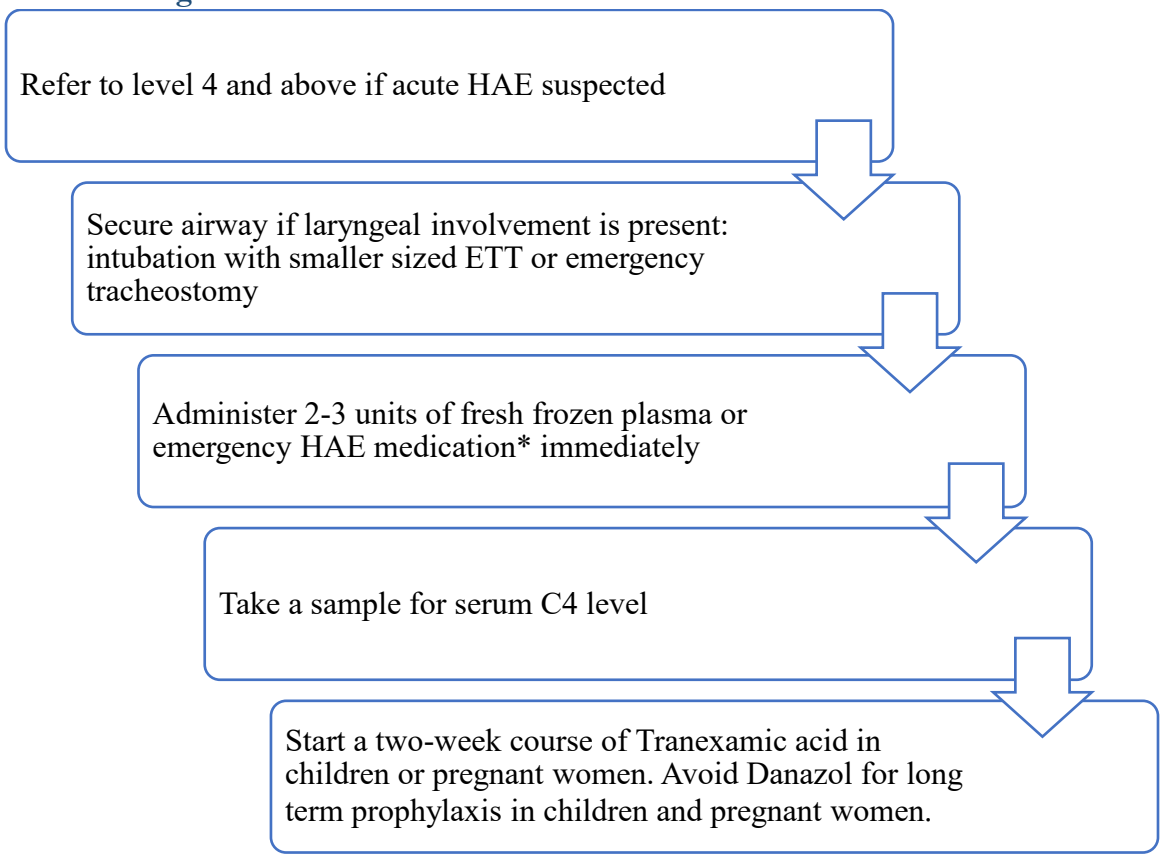


Figure 19: Management of Hereditary Angioedema (HAE)

*Emergency HAE medication includes C1 inhibitor concentrate or Icatibant where available.

1.21.6 Recommendation/Referral

Urgent two-week referral to allergist/dermatologist for further assessment, confirmatory investigation and management with more effective prophylaxis and treatment that is not widely available in the public sector but is life saving for the patient. (There are international organizations that assist the patient access these services)

1.22 Hymenoptera Venom Allergy

1.22.1 Definition

Hymenoptera venom allergy (HVA)/ insect sting venom allergy is one of the most serious IgE-mediated hypersensitivity reactions due to the high risk of severe and even fatal anaphylaxis. This condition is more common in adults than children.

1.22.2 Causes/Triggers

These insect stings include:

- bees (honey & bumble bees),
- wasps (paper wasps, yellow jacket,
- white faced hornet and yellow hornet)
- ants (fire ants).

1.22.3 Clinical features

Table 11: Clinical features of hymenoptera venom allergy (HVA)

Sting reaction in healthy individual	Pain and inflammation at bite or sting site (swelling, redness and itching)
Ig E mediated systemic reactions	Mild (generalized skin symptoms such as urticaria or angioedema) Moderate (e.g., dyspnea, gastrointestinal symptoms or dizziness) Severe (e.g., unconsciousness, anaphylactic shock, respiratory or cardiac arrest).
Unusual serious reactions	Serum sickness-like manifestations, thrombocytopenic purpura, hemolytic anemia, Guillain-Barre syndrome, vasculitis, glomerulonephritis and demyelination-related neurological complications

1.22.4 Management

- ABC - manage acute emergency.
- Antihistamines for mild symptoms.
- Education on allergen avoidance.
- Wear protective clothing.
- Avoid wearing strong floral perfumes.
- Avoid sweet drinks outdoors.

1.22.5 Recommendations/referrals

Patients with severe reactions require referral to an allergist for further investigations and management.

1.23 Occupational Allergy

1.23.1 Definition

This refers to an allergy caused by exposures to various allergens in the workplace. Median time to diagnosis is four years. This condition is largely under diagnosed and under reported.

1.23.2 Clinical features

It may manifest as:

- Asthma
- Rhinitis
- Eczema
- Urticaria
- Angioedema
- Conjunctivitis

Populations at risk include bakers, bankers, florists, flower farm workers, tea pickers, construction workers, healthcare workers, cleaners, cannabis growers amongst others.

1.23.3 Management

Treat the clinical condition based on presentation. Testing is based on patient history and occupational exposures. Tests include spirometry and skin prick testing, serum sIgE, patch tests and are usually done by the specialist.

1.23.4 Recommendation/referral

- Refer to occupational physician for assessment.
- Refer to an allergist/pulmonologist/dermatologist for further workup and management.

1.24 Latex Allergy

1.24.1 Definition

Latex allergy is an IgE-mediated immediate hypersensitivity response to natural rubber latex (NRL) protein.

1.24.2 Causes/Triggers

Risk factors are associated with high routine use of latex e.g. in gloves, condoms, balloons, rubber tubing etc.

1.24.3 Clinical presentation

Table 12: Clinical presentation for latex allergies

Clinical presentation			
Cutaneous	Respiratory	Systemic	Ocular
Contact urticaria Protein contact dermatitis (PCD) Allergic contact dermatitis (type IV hypersensitivity)	Occupational asthma & or rhinitis	Anaphylaxis	Conjunctivitis

1.25 Latex fruit allergy syndrome

1.25.1 Definition

This is a cross-reactive allergy between latex (which acts as the primary sensitizer) and fruits and vegetables where the patient reacts to the foods upon ingestion.

1.25.2 Causes/triggers and clinical features

Whereas patients allergic to fruits have an 11% risk of latex reactions, patients allergic to latex have a 35% risk of a reaction to fruits.

Table 13: Causes /triggers and clinical features of latex/fruit allergy syndrome

Foods cross reacting with latex	Clinical Features
Bananas Avocados Chestnuts Pawpaw Potatoes Tomatoes Apples Carrots Celery Melons	Oral itching Generalized pruritus Hives Angioedema Laryngospasms Nausea/Vomiting Dyspnea

1.25.3 Management

- If suspected, avoid latex products and refer those with moderate to severe reactions.
- For latex food syndrome, patients must avoid or change the form of cross-reactive foods.
- Symptomatic relief of symptoms with second generation antihistamines or topical steroids for the dermatitis as in the previous section.

1.25.4 Recommendation/referral

- Refer to allergist/ dermatologist for further testing/management of moderate to severe reactions.
- Refer to occupational physician for assessment of occupational exposure.

Chapter Three: Allergy Testing

1.26 Objectives

To guide health care workers on the different allergy tests available, their utility in the clinical setting and the level of health care the test is to be ordered to ensure appropriate use of allergy tests for the benefit of the patient.

Table 14: Allergy testing

CATEGORY	NAME OF TESTS	INDICATION / INTERPRETATION	WHO REQUESTS THE TEST?
1.27 In Vivo Testing	1.27.1 Skin Prick Test (SPT)	Diagnosis of Type 1 immediate hypersensitivity reactions that are responsible for IgE mediated allergic disease (food, inhalant, drug and latex allergens)	Allergist
		Interpretation: A wheal diameter above 3mm (or 3mm greater than negative control) is considered positive (sensitization)	
	1.27.2 Intradermal Skin Testing (IDT)	Diagnosis of type 1 immediate hypersensitivity reactions that are responsible for IgE mediated allergic disease. Its use is limited to drug, vaccine and venom allergies.	Allergist
		Interpretation: An increase of the wheal size of 3mm in diameter beyond the initial bleb is considered positive.	
	1.27.3 Atopy Patch Testing (APT)	Diagnosis of patients with delayed type IV hypersensitivity reactions responsible for allergic contact dermatitis and drug allergies as well as other occupational allergies.	Allergist / dermatologist
		Interpretation: Positive reactions are scored as follows: ?/+: doubtful (macular erythema), +: weak (papular erythema), ++: strong (edematous or vesicular), +++: extreme (bullous or ulcerative)	

CATEGORY	NAME OF TESTS	INDICATION / INTERPRETATION	WHO REQUESTS THE TEST?
1.28 Provocation Tests	**1.28.1 Oral Food Challenge (OFC)**	Gold standard confirmatory test. Diagnosis of type 1 immediate hypersensitivity IgE mediated allergic disease Gold standard for confirmation of IgE mediated food allergies. To establish the diagnosis of food allergy when clinical history is highly suggestive but initial testing is negative. To monitor for development of tolerance (outgrowing the allergy) To determine tolerance in sensitized patient who has never ingested the food before (this includes cross reactive foods) To identify the allergy threshold; usually before initiating oral immunotherapy	Allergist
		Interpretation: Patient is evaluated for development of allergic symptoms at set intervals.	
	1.28.2 Elimination – Reintroduction Challenge	This test is used to identify allergens in patients with non-IgE/ delayed food allergy as well as food intolerances.	Allergist / gastroenterologist
		Action: Documentation of symptoms by patient / caregiver	
	1.28.3 Drug Challenge	Not a first line test for the diagnosis of drug allergies. Used to: exclude hypersensitivity when history is not suggestive, or the symptoms are non-specific. to definitively diagnose allergy where history is suggestive, but the initial investigations are negative or equivocal to exclude cross-reactivity of related drugs where allergy has been proven. **Contraindication:** If the history is consistent with severe drug reactions.	Allergist / dermatologist
		Interpretation: Any positive drug challenge with reactions will guide drug avoidance. Document findings.	

CATEGORY	NAME OF TESTS	INDICATION / INTERPRETATION	WHO REQUESTS THE TEST?
1.29 In Vitro Allergy Tests	**1.29.1 Total Immunoglobulin E (Total IgE)**	Total IgE was initially used as a screening test for allergic disease, but it has limitations. However, it can give an indication of the degree of immune dysregulation. It is not recommended as a screening test for atopy, but could be useful in the following conditions: Allergic bronchopulmonary aspergillosis (ABPA) Hyper IgE Syndrome Severe asthma requiring anti IgE therapy	Allergist / pulmonologist
		Interpretation: Cut-off values based on age specific ranges Total IgE levels >2000 U/mL is indicative of Hyper IgE Syndrome	
	1.29.2 Serum Specific IgE	Diagnosis of type 1/ IgE mediated allergic disease: food, inhalant, venom, drugs allergens etc. This test measures the serum level of IgE to a specific allergen and indicates **sensitization**.	Allergist / specialist with allergy training
		Interpretation: **Single allergens:** all single allergen results ≥ 0.1 kUA/l are "positive", and true IgE antibodies are detected. **Mixed allergens:** ≥ 0.35 kUA/l are considered "positive". (4) Allergen specific IgE measures sensitisation, but not necessarily clinically relevant allergy. Tests should always be correlated with the clinical findings and the level of suspicion of allergy or a relevant specific allergen. Provocation test should be performed to confirm or exclude clinically relevant allergy in cases where sensitization was indicated by sIgE tests.	
	1.29.3 Basophil Activation Test (BAT)	Indicated in food allergies, reactions to colorants and preservatives as well as drug allergies. IgE as well as non-IgE mediated reactions are measured.	Allergist / Specialist with allergy training
		Interpretation: Results are interpreted based on the patient's background	

CATEGORY	NAME OF TESTS	INDICATION / INTERPRETATION	WHO REQUESTS THE TEST?
		stimulation and the % upregulation of the activation marker on the surface of the basophils. Results are interpreted according to different groups of allergens.	
	1.29.4 Serum Mast Cell Tryptase	The serum tryptase test can be useful as a marker of: Mast cell activation in the definitive diagnosis of anaphylaxis. Mastocytosis Risk stratification in Venom Immuno-Therapy (VIT)	Emergency physician / anesthesiologist / dermatologist / allergist
		Interpretation: **Anaphylaxis:** 11.4 µg/l is commonly used as upper reference value. The criteria for mast cell activation is an increase in total serum tryptase by at least 20% above baseline plus 2 µg/l during or within 4 hours after a symptomatic period. **Mastocytosis:** Transient increase in the tryptase level by at least 20%, above the baseline level, plus 2µg/l confirm mast cell activation. In patients with systemic mastocytosis levels of tryptase are, in general, persistently elevated above 20 µg/l.	
	1.29.5 Complement levels: C4 levels and C1-Inhibitor (C1-INH) function and levels	**Serum C4** level is the most available screening test for patients suspected to have hereditary angioedema (HAE). **C1-INHibitor level** for confirmatory diagnosis Type 1 HAE **Functional C1-INH** – for diagnosis of Type II HAE in case of normal (or even elevated) C1-INHibitor level.	Emergency physician / allergist
		Interpretation: Persistently low C4 levels in HAE. The condition can be screened by measuring reduced C4 levels between attacks and reduced to absent C4 levels during an attack. C1-INHibitor levels - confirmatory testing for Type 1 C1-INH deficiency	

CATEGORY	NAME OF TESTS	INDICATION / INTERPRETATION	WHO REQUESTS THE TEST?
		where levels are reduced. (80 – 85% of all HAE cases) Functional C1-INH - approximately 15–20% of HAE is type II in which C1-INH levels are normal (or increased) but the protein is dysfunctional.	
	1.29.6 Eosinophil Count	**Peripheral blood eosinophils:** used to evaluate the likely response to steroids or anti-eosinophilic monoclonal antibodies e.g. Mepoplizumab and Benralizumab in asthma treatment. **Interpretation:** The cut-off in adults - 300 cells/mcl children aged 6 months to 13 years: 500-700 cells/mcl **Sputum eosinophils:** Assessment of sputum inflammation for the diagnosis of asthma is currently ONLY recommended for children with SEVERE ASTHMA. **Interpretation:** Eosinophil count < 1% - not significant Eosinophil count > 1% - significant eosinophilia	Pulmonologist / allergist
	1.29.7 Serum Eosinophil Cationic Protein (ECP)	Estimating the severity of airway inflammation and following the course of disease in asthmatic patients. ECP can be used to monitor inflammation in asthma guide corticosteroid treatment in asthma identify non-compliant patients **Interpretation:** values over 15 µg/l should be considered elevated	Pulmonologist / allergist

CATEGORY	NAME OF TESTS	INDICATION / INTERPRETATION	WHO REQUESTS THE TEST?
1.30 Other Tests	**1.30.1 Exhaled Nitric Oxide (FeNO)**	to assess adherence to asthma therapy to assess the contribution of inflammation in patient with multiple risk factors like rhino sinusitis, obesity, gastroesophageal reflux disease (GERD) & continued allergen exposure (occupational) to determine likelihood of corticosteroid responsiveness	Pulmonologist / allergist
		Interpretation: **Adults:** A FeNO levels of ≥40 parts per billion (ppb) or more is a positive test and supportive of asthma. **Children:** A FeNO levels of ≥35 parts per billion (ppb) or more is a positive test and supportive of asthma.	
	1.30.2 Spirometry	To diagnose and manage respiratory problems **Indications** establish baseline lung function evaluate dyspnoea monitor disease progression establish objective diagnosis of asthma and assess effect of medication therapy pre-operative evaluation of lungs surveillance of occupational lung disease medico-legal issues / occupational health. insurance disability	Pulmonologist / allergist chest clinical officer (chest specialist)
		Interpretation: This requires a comparison of patient's values against reference values that are determined by the patient's age, gender, race/ ethnicity and height. Refer to the ASOK Allergy testing guidelines; https://allergysociety.or.ke/	
	1.30.3 Endoscopy/ Biopsy/ Histopathology	Diagnosis of eosinophilic oesophagitis / gastroenterological enteropathies	Gastroenterologist
		Interpretation: clinical symptoms of eosinophilic dysfunction ≥15 eosinophils/hpf on biopsy exclusion of other possible causes of eosinophilia	

CATEGORY	NAME OF TESTS	INDICATION / INTERPRETATION	WHO REQUESTS THE TEST?
1.31 Unvalidated Tests	• Allergen specific IgG • Cellular provocation-neutralization • Cytotoxic tests, muscle response testing (applied kinesiology) • Electrodermal testing, the "reaginic" pulse test and chemical analysis of body tissues including hair analysis	There are numerous tests purported to be of use in the diagnosis of IgE-mediated allergy; however, they lack evidence and rigorous validation to support their use. The use of such tests carries significant risks, namely unnecessary dietary restrictions which may be associated with dietary compromise, increased costs and reduced quality of life (QoL); and conversely, dietary liberation may be associated with potential exposure to culprit allergens. *The above unvalidated tests should NOT be requested due to lack of scientific evidence.*	Should not be requested

Chapter Four: Monitoring And Evaluation

1.31.1 Monitoring and Evaluation (M&E) basis

Monitoring and evaluating these Guidelines shall be embedded in a comprehensive National Strategic Plan, or Strategic Action Plan for allergic conditions in Kenya. The strategic documents shall be developed and reviewed every five years and will serve as a guide for its implementation and progress monitoring. Mid-term reviews shall be conducted every three years during the implementation period to ensure responsiveness to emerging health priorities and advances in evidence-based allergy management practices.

The indicators outlined in the Guidelines will be seamlessly integrated into the Kenya Health Information System (KHIS) and supported by digitized reporting tools. This structured approach will ensure the availability of high-quality data for informed decision-making and continuous improvement in the prevention, diagnosis, and treatment of allergic diseases. Table 13 outlines the broad M&E framework, which shall inform the strategies that are aimed at aiding in combating and managing allergic conditions in Kenya.

1.31.2 M&E Framework

Table 15: M&E Framework

S/NO.	KEY RESULT AREA	INDICATOR	INPUTS	OUTPUTS	RESPONSIBLE PARTY	BASELINE	OUTCOME
1	Training	Number of Allergists trained	Finances, human resource	Allergists trained	Ministry of Health. ASOK. Research & Training Institutions.	5 Allergists	Enhanced capacity to diagnose and treat allergic conditions.
		Number of Health care professionals trained in the use of guidelines		Health care professionals trained in the use of guidelines		-	
		Development of short online courses		Short online courses developed		-	
2	Access to Testing	Number of level 6 hospitals providing testing	Finances, human resource	Testing facilities established in level 6 hospitals	Ministry of Health. ASOK. Research & Training Institutions County Governments Partners	-	Improved access to diagnostic testing
		Number of testing equipment provided		Provision of peak flow meters, spirometers, nebulizers, and medications		-	
		Establish Referral Pathways		Referral system on allergic conditions established	Ministry of Health. ASOK. County Governments. Community Health Promoters.	-	Better treatment outcomes for patients
3	Curriculum development	Progress on curriculum development (stages completed, approvals obtained)	Finances, human resource	Developed and approved curriculum	Ministry of Health. ASOK. Research & Training Institutions.	-	Standardized training curriculum for allergic conditions.
		Number of institutions adopting the curriculum		Curriculum adopted by training institutions		-	
4	Capacity building	Number of guidelines disseminated	Finances, human resource	Guidelines disseminated	Ministry of Health.	-	Improved guideline adherence

S/NO.	KEY RESULT AREA	INDICATOR	INPUTS	OUTPUTS	RESPONSIBLE PARTY	BASELINE	OUTCOME
		Number of sensitization sessions conducted		Sensitization sessions conducted	ASOK. County Government. Partners.	-	and patient management
		Number of healthcare workers trained on allergy testing and management		Healthcare workers trained		-	
5	Research & Innovation	Number of surveys conducted	Finances, human resource	Quality data available	Ministry of Health. ASOK. WHO Research & Training Institutions.	-	Data-driven decision-making and future planning.
		Stakeholder forums created		Forums for information sharing		-	
		Monitoring & Evaluation (M&E) indicators established		MEAL framework developed		-	
		Regular assessment and revision of clinical guidelines	Finances, Human resource	Revised clinical guidelines after 3-5 years	Ministry of Health ASOK	-	Updated and effective strategies for managing allergic conditions.

1.31.3 Recording and reporting tools for allergic conditions

Appropriate recording and reporting of allergic conditions management:

- Asthma register
- Asthma Control Test card
- Asthma patient appointment card
- Allergic conditions management card
- Allergic conditions appointment card
- Heredity angioedema management card
- Register for common allergic conditions

1.31.4 Basic equipment for allergy testing and management at different levels of care

Table 16: Basic equipment for allergy testing and management at different levels of care

Equipment that should be available (✓) vs type of facility				
Equipment	**Facility level**			
	Level 2	Level 3	Level 4	Level 5 & 6
Diffusion capacity for carbon monoxide (DLCO)				✓
Fractional of exhaled nitric oxide (FENO)				✓
Spirometry			✓	✓
Bacterial viral filters		✓	✓	✓
Peak flow meter		✓	✓	✓
Weighing machine/ Height meter	✓	✓	✓	✓
Spacer	✓	✓	✓	✓
Short acting Beta agonists	✓	✓	✓	✓
Pulse oximeter	✓	✓	✓	✓
BP machine	✓	✓	✓	✓
Thermometer	✓	✓	✓	✓
Stethoscope		✓	✓	✓
Nebulization machine		✓	✓	✓
Mouthpiece with filter		✓	✓	✓
Oxygen cylinder	✓	✓	✓	✓
Skin prick test				✓
6-minute walk test			✓	✓

***Source**: Kenya Asthma Management Guideline 2021*

1.31.5 M&E matrix for testing and management of allergic conditions in Kenya.

The M&E key indicators that shall further inform the Kenya allergic conditions strategic documents for planning purposes are outlined below.

Table 17: M&E matrix for testing and management of allergic conditions in Kenya

SNO	Strategic Actions	Indicator	Data Source	Frequency	Responsible entity	Remarks
	Establish the prevalence of allergic diseases	Number of published baseline surveys	Reports from facilities	Bi-annually	Ministry of Health. Research & Training Institutions.	Collaborate with healthcare facilities, research institutions, and community health workers to collect relevant information
	Establish the risk factors associated with allergic diseases	Number of published nationwide surveys	Existing health records Survey reports	Annually	Ministry of Health. County Governments.	
	Enhance clinical evaluation & diagnostic capacity	Number of professionals trained to recognize allergic conditions & perform accurate diagnosis	Regulators' reports Human resource department report	Annually	Ministry of Health. KMPDC. County Governments.	Involve regulators to ensure continuous professional development.
		Number of diagnosed allergic cases	Hospital records (KHIS)	Monthly	Ministry of Health. Hospitals. Clinics.	Enhance diagnostic training
	Improve access to testing	Number of Level 6 hospitals providing testing	Facility records	Bi-annually	Ministry of Health. ASOK. Research & Training Institutions. County Governments Partners.	Increase testing coverage
		Number of testing equipment provided	Lab and clinic reports	Monthly	Ministry of Health. Health Facilities.	Continuously improve testing capacity
	Enhance diagnosis,	Number of healthcare providers trained	Training records	Annually	Ministry of Health.	Continuous professional education

SNO	Strategic Actions	Indicator	Data Source	Frequency	Responsible entity	Remarks
	management, and referral				Training Institutions.	
		Availability of allergy medications	Pharmacy inventory	Quarterly	Ministry of Health. KEMSA. Pharmacies. Hospitals.	Ensure steady supply.
		Referral system in place	Health facility reports	Monthly	Health Facilities	Keep updating the referral system based on availability of resources at each level.
	Monitoring and surveillance	National registry established	KHIS, Facility reports	Quarterly	Ministry of Health. Health facilities.	Regularly update data on disease incidence, prevalence, and severity. Monitor trends over time to identify emerging issues.
		Patient satisfaction rate	Patient surveys	Biannual	Ministry of Health. Healthcare providers.	Improve patient education
	Strengthen policy	National allergy testing and management guidelines developed	Policy documents	3-5 years	Ministry of Health.	Engage stakeholders
		Establishment of allergy registry	Registry data	Annually	Ministry of Health	Track incidence and trends

1.31.6 Key Notes:

1. **Responsible entity:** designated organizations or departments responsible for collecting, analyzing, and reporting data.

2. **Frequency:** indicates how often the data should be collected and reviewed.

3. **Remarks:** additional comments or actions needed.

2 Annexes

2.1 Annex Table 1: List of contributors

Name	Designation/Institution
Dr. Andrew J. Toro	Director Curative and Nursing Services (MoH)
Dr. Rebecca Kiptui	Former Ag. Director Curative and Nursing Services (MoH)
Dr. Saleh Bardad	Ag. Head, Division Clinical Services (MoH)
Mr. Manaseh Bocha	Clinical Services (MoH)
Dr Penny Muange	Ag. Head of Division Oral, Eye & Hearing (MoH)
Mr. Japheth Athanasio	Health System Strengthening Specialist (MOH)
Dr Judith Awinja	Ag. Director of Nursing Services (MOH)
Dr Sitienei Joseph	National Referral Services (MOH)
Marcyline Mokua	Nursing services (MOH)
Martha Njeri	Nursing services (MOH)
Mr. Anthony Komen	Monitoring and Evaluation/Statistician, (MOH)
Joyce Mokua	Clinical Services (MOH)
Dr. Anne Barasa	Pathologist (UoN, ASOK)
Mr. Anton Jensen	Scientific Affairs Manager, Thermofisher
Dr. Caroline Ndarathi	Dermatologist (KNH, ASOK)
Dr. Diana Marangu	Paediatric Pulmonologist (UoN, ASOK)
Dr. Elizabeth Kiragu	Paediatric Allergist (AKUH, N, ASOK)
Dr. Evelyne Nganga	Paediatric Allergist (The Paediatric Allergy Center. ASOK)
Dr. Jacqueline Kavete	Dermatologist (Mwai Kibaki Hospital, ASOK)
Prof. Kirtika Patel	Immunologist (Moi University, ASOK)
Dr. Maria Muthoka	ENT Surgeon (KU, ASOK)
Dr. Maureen Kiaraho	Ophthalmologist (Thika Level 5 Hospital, ASOK)
Ms. Joan Kagema	Nurse (KNH, ASOK)
Dr. Priya Bowry	Allergist (The Allergy Clinic, ASOK)
Dr. Ravi Sharma	General Practitioner (AKUH, N, ASOK)
Dr. Winnie Njenga	Dermatologist (Kiambu Level 5 Hospital, ASOK)

2.2 Annex Table 2: Key Messages for Primary Healthcare Providers

1. VKC and Atopic Conjunctivitis can result in visual loss due to corneal scarring. Other sight threatening complications include keratoconus (due to excessive eye rubbing), cataracts and glaucoma (due to prolonged steroid use). As such, early referral to an ophthalmologist is key. Topical steroids should NOT be prescribed without consultation with the ophthalmologist.
2. Allergic Rhinitis requires treatment with topical intranasal steroids. Avoid vasoconstrictors and oral steroids. Avoidance of known precipitating factors improves control. (e.g., grass pollen, house dust mite or animal dander).
3. Asthma: avoid frequent use of systemic steroids as every dose has an impact on a patient's overall morbidity and mortality. Early referral to pulmonologists or allergy specialists for those with more than 4 exacerbations is therefore required.
4. Avoid blind food elimination diets especially in patients with atopic eczema unless guided by an allergist / paediatric gastroenterologist. This is to avoid negative impact on a child's growth and development and undernutrition in adults.
5. In a breastfed infant or young child with immediate allergic reactions to specific foods, reactions to these foods in the mother's diet are uncommon, therefore, maternal dietary restriction is generally not necessary.
6. It is important to note that *results of allergy testing should be interpreted in the context of the history given.* Positive allergy test (skin prick tests and serum specific IgE) results need to be interpreted with caution, since it may be clinically irrelevant when no symptoms are elicited within 2 hours of ingestion. Oral food challenges for diagnosis of IgE mediated food allergies should only be conducted by allergists and not in primary care or community settings.
7. The management of non-IgE mediated food allergy is based on the results of an allergy-focused clinical history. Trial elimination of the suspected allergen (usually for 2-6 weeks) is followed by re-introduction to confirm the suspected allergen. This should be done by an allergy specialist.
8. Allergen avoidance of known offending food and associated cross-reactive allergens is the mainstay of management in food allergies. This is guided by allergy specialists.
9. Refer to allergist for allergy testing for patients with moderate-severe eczema in whom optimized treatment has failed to control the disease and/or patients presenting with associated immediate reactions e.g., urticaria/angioedema.

10. Avoid use of topical or systemic steroids in patients with urticaria or angioedema. Second generation antihistamines are the main stay of treatment.
11. Early recognition and referral of HAE patients to the allergy specialist is mandatory and lifesaving.
12. Early recognition and management of anaphylaxis with undiluted intramuscular adrenaline is lifesaving. Antihistamines only help with urticaria, and angioedema and systemic steroids are no longer indicated.

2.3 Annex Table 3: List of Stakeholders

1. Ministry of Health
2. Allergic Society of Kenya
3. Kenya Medical Practitioners & Dentists Council
4. Nursing Council
5. Clinical Officers Council
6. Aga Khan University Hospital, Nairobi
7. University of Nairobi
8. Kenyatta University
9. Moi University
10. Kenyatta National Hospital
11. Mwai Kibaki Hospital
12. Kiambu Level 5 Hospital
13. Thika level 5 Hospital
14. The Allergy Clinic
15. The Paediatric Allergy Centre

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