Clinical Standards in Allergy diagnosis and management: A Comprehensive Guide for a Family Practice

Abstract

Allergic diseases represent a substantial and increasing burden in primary care, affecting approximately 20–30% of the global population and over 50% of paediatric cohorts. Family practitioners play a central role in the early identification, diagnosis, and longitudinal management of allergic conditions, including allergic rhinitis, asthma, atopic dermatitis, and food allergies. This document provides a comprehensive, evidence-based framework for allergy diagnosis and management in the primary care setting such as a family practice. Emphasis is placed on immunopathophysiology, structured clinical assessment, and the appropriate use of diagnostic modalities such as skin prick testing and serum-specific IgE assays. Management strategies are presented using a stepwise approach, incorporating allergen avoidance, pharmacotherapy, immunotherapy, and patient education. Particular focus is given to high-risk conditions such as food allergy and anaphylaxis, where prompt recognition and intervention are critical. Emerging preventive strategies, including early allergen introduction, and advances in biologic therapies are also discussed. This guide aims to standardize clinical practice, support decision-making, and improve patient outcomes in family medicine.

1. Background

Allergic diseases are among the most prevalent disorders encountered in primary care, affecting 20–30% of the population at large [1]. In a primary care setting, at least one atopic disease is often documented in over 50% of paediatric samples. The family physician therefore acts as the gatekeeper, identifying the the sequential progression from atopic dermatitis to food allergy, allergic rhinitis, and asthma. The rising incidence of allergic diseases over recent decades has been attributed to a complex interplay between genetic predisposition and environmental factors, including urbanisation, reduced microbial exposure (hygiene hypothesis), dietary changes, and increased pollution [2]. The family physician serves as the frontline clinician in the identification and management of allergic diseases. A key conceptual framework in understanding sequential development of allergic conditions is that of the Atopic March. This often begins with atopic dermatitis in infancy, followed by food allergies, allergic rhinitis, and ultimately asthma . Early recognition of this progression provides a critical window for intervention, with the potential to alter disease trajectory and reduce long-term morbidity [3]. Allergic diseases significantly impair quality of life, affecting sleep, cognitive performance, and productivity, while also contributing to substantial healthcare costs [4]. As such, the implementation of standardized clinical approaches in the primary care setting is essential.

2. Eitology and Pathophysiology

The core mechanism of allergic disease involves an overactive immune response to harmless antigens (allergens). These often include pollen, dust mites, animal dander, foods, and medications [5]. The sensation phase is the first phase of the allergenic response and occurs upon first contact with the allergen. Thereafter, antigen-presenting cells process and present the allergen to naïve T-helper (Th0) cells, which differentiate into Th2 lymphocytes. These cells release cytokines such as IL-4 and IL-13, promoting B-cell class switching to produce allergen-specific IgE antibodies. These IgE molecules bind to high-affinity receptors on mast cells and basophils, effectively sensitising the individual. During the effector phase, upon subsequent exposure, these antibodies trigger mast cell degranulation, releasing inflammatory mediators like histamine, which cause immediate symptoms such as swelling, itching, and mucus production [6]. Genetic and environmental contribution has become a strong predictor of allergy predisposition, therefore, parental history of atopy is the strongest predictor of a child developing an allergy [7]. According to the literature the risk of a child developing an allergy is 60–80% if both parents are affected, compared to 5–15% if neither are [7].

3. Diagnostic Framework

Accurate diagnosis of allergic disease in primary care is grounded in a thorough clinical history, supported by targeted diagnostic testing. A structured history should explore symptom onset, frequency, severity, and identifiable triggers, as well as any temporal relationship to environmental exposures or food intake. Family history of atopy and the impact of symptoms on quality of life should also be assessed. Physical examination should

be directed by presenting symptoms and may reveal characteristic findings such as pale, edematous nasal mucosa in allergic rhinitis, wheezing upon respiratory examination [8].

Diagnostic testing should be used to confirm clinical suspicion rather than as a screening tool. Skin prick testing remains the gold standard for identifying IgE-mediated sensitisation due to its high sensitivity, rapid results, and cost-effectiveness [9]. Serum-specific IgE testing provides an alternative when skin testing is contraindicated, although results must be interpreted cautiously, as sensitisation does not equate to clinical allergy [9]. Oral food challenges remain the definitive diagnostic modality for food allergy but carry inherent risks and are generally reserved for specialist settings. Lastly, a key principle in allergy diagnosis is the avoidance of indiscriminate testing, which can lead to false-positive results, overdiagnosis, and unnecessary dietary or lifestyle restrictions [10].

In recent years, In vitro allergy diagnostics have been significantly advanced by highly standardised platforms such as the Thermo Fisher Scientific ImmunoCAP™ system. This system is widely regarded as a reference method for serum-specific IgE quantification. These assays enable precise measurement of allergen-specific IgE antibodies using small serum volumes and offer extensive panels covering whole allergens, allergen mixtures, and individual allergen components. Component-resolved diagnostics (CRD), in particular, allow clinicians to distinguish between true primary sensitisation and cross-reactivity, thereby improving diagnostic accuracy and risk stratification. Such systems are especially valuable in clinical scenarios where skin prick testing is contraindicated, including patients with severe eczema, those taking antihistamines, or individuals at high risk of anaphylaxis. Additionally, multiplex testing platforms enable simultaneous evaluation of multiple allergen sensitivities, facilitating comprehensive allergy profiling in a single test. Despite their utility, results must always be interpreted within the clinical context, as the presence of allergen-specific IgE does not necessarily equate to clinically relevant allergy.

4. Management of Allergic Conditions with Pharmacological Agents

Pharmacological therapy forms the backbone of allergy management and should be individualised according to disease severity and phenotype. Second-generation H1 antihistamines, including Cetirizine, Loratadine, and Fexofenadine, are widely recommended as first-line agents due to their efficacy in controlling histamine-mediated symptoms and their favourable safety profile, with minimal sedation compared to first-generation agents [11]. First-generation antihistamines such as Diphenhydramine may still be used in acute settings but are limited by central nervous system and anticholinergic side effects [11]. Intranasal corticosteroids, including Fluticasone and Mometasone, are the most effective monotherapy for moderate to severe allergic rhinitis. Their mechanism involves suppression of multiple inflammatory pathways, leading to significant reduction in nasal congestion, rhinorrhoea, and mucosal inflammation [12]. Leukotriene receptor antagonists such as Montelukast may provide additional benefit, particularly in patients with concomitant asthma, although clinicians should remain aware of rare neuropsychiatric adverse effects [13]. In asthma, inhaled corticosteroids such as Budesonide are the cornerstone of long-term control, reducing airway inflammation and preventing exacerbations. Short-acting β2-agonists such as Salbutamol provide rapid symptom relief

but should not be used as monotherapy due to the risk of poor disease control [14]. Systemic corticosteroids, including Prednisone, are reserved for acute exacerbations due to their significant systemic side effect profile. In severe or refractory cases, biologic therapies such as Omalizumab and Dupilumab offer targeted treatment by modulating specific immune pathways, although their use is typically restricted to specialist care.

Table showing medication, type of allergy and clinical role

Medication/Class	Examples	Type of Allergy / Condition Treated	Clinical Role
Second-generation H1 antihistamines	Cetirizine, Loratadine, Fexofenadine	Allergic rhinitis, urticaria, mild IgE-mediated reactions	First-line for histamine-mediated symptoms (itching, sneezing, rhinorrhoea)
First-generation H1 antihistamines	Diphenhydramine	Acute allergic reactions, urticaria	Short-term symptomatic relief; limited by sedation and anticholinergic effects
Intranasal corticosteroids	Fluticasone, Mometasone	Allergic rhinitis (moderate–severe)	Most effective monotherapy; reduces nasal inflammation and congestion
Leukotriene receptor antagonists	Montelukast	Allergic rhinitis, asthma	Adjunct therapy; useful in patients with coexisting asthma
Inhaled corticosteroids (ICS)	Budesonide	Asthma (allergic and non-allergic)	First-line controller therapy; reduces airway inflammation
Short-acting β2-agonists (SABA)	Salbutamol	Acute asthma symptoms	Rapid bronchodilation for symptom relief ("rescue therapy")
Systemic corticosteroids	Prednisone	Severe asthma exacerbations, severe allergic reactions	Short-term use for acute inflammation control
Intramuscular adrenaline (epinephrine)	Epinephrine	Anaphylaxis (severe systemic allergy)	First-line, life-saving emergency treatment
Biologic anti-IgE therapy	Omalizumab	Severe allergic asthma, chronic urticaria	Targets IgE; used in refractory or severe disease
Biologic anti–IL-4/IL-13 therapy	Dupilumab	Atopic dermatitis, severe asthma	Modulates Th2-mediated inflammation
Allergen immunotherapy (SCIT/SLIT)	Pollen, dust mite extracts	Allergic rhinitis, allergic asthma	Disease-modifying therapy; induces immune tolerance

5. Dosing and Practical Prescribing Considerations

Effective management requires not only appropriate drug selection but also correct dosing and patient adherence. Second-generation antihistamines are typically administered once daily, with standard adult doses of 10 mg for Cetirizine and Loratadine, and 120–180 mg for Fexofenadine. Intranasal corticosteroids such as Fluticasone are generally prescribed as one to two sprays per nostril once daily, with optimal effects achieved after consistent use over several days. In asthma management, Salbutamol is used as needed for symptom relief, typically at doses of 100–200 micrograms every 4–6 hours. Maintenance therapy with inhaled corticosteroids such as Budesonide is initiated at low to moderate doses and

titrated according to symptom control. Montelukast is administered at 10 mg once daily, often in the evening. Short courses of systemic corticosteroids such as Prednisone (30–50 mg daily for 5–10 days) are used for acute exacerbations. In cases of anaphylaxis, intramuscular Epinephrine at a dose of 0.3–0.5 mg is administered promptly and may be repeated every 5–15 minutes if necessary. Dose adjustments should be considered in special populations, including paediatric, elderly, and comorbid patients.

Drug Class	Medication	Indication	Adult Dose	Route	Frequency	Key Notes / Adjustments	Guideline Source
Second-generation H1 antihistamine	Cetirizine	Allergic rhinitis, urticaria	10 mg	Oral	Once daily	Reduce dose in renal impairment; may cause mild sedation	ARIA, WAO
Second-generation H1 antihistamine	Loratadine	Allergic rhinitis, urticaria	10 mg	Oral	Once daily	Caution in hepatic impairment	ARIA, WAO
Second-generation H1 antihistamine	Fexofenadine	Allergic rhinitis	120 mg	Oral	Once daily	Non-sedating; avoid fruit juice interactions	ARIA
Second-generation H1 antihistamine	Fexofenadine	Chronic urticaria	180 mg	Oral	Once daily	Dose escalation possible in refractory cases	WAO
Intranasal corticosteroid	Fluticasone	Allergic rhinitis	1–2 sprays/nostril (50 µg/spray)	Intranasal	Once daily	Max effect after several days; correct technique essential	ARIA
Short-acting β2-agonist (SABA)	Salbutamol	Acute asthma symptoms	100–200 µg	Inhaled	Every 4–6 hrs PRN	Avoid SABA-only therapy in persistent asthma	GINA
Inhaled corticosteroid (ICS)	Budesonide	Asthma maintenance	200–400 µg/day (low)	Inhaled	Divided doses	Titrate to control; rinse mouth after use	GINA
Inhaled corticosteroid (ICS)	Budesonide	Asthma maintenance	400–800 µg/day (moderate)	Inhaled	Divided doses	Step-up therapy if uncontrolled	GINA
Inhaled corticosteroid (ICS)	Budesonide	Asthma maintenance	>800 µg/day (high)	Inhaled	Divided doses	Monitor for systemic effects	GINA
Leukotriene receptor antagonist	Montelukast	Asthma, allergic rhinitis	10 mg	Oral	Once daily (evening)	Monitor for neuropsychiatric effects	GINA, ARIA

Systemic corticosteroid	Prednisone	Acute asthma exacerbation	30–50 mg	Oral	Once daily (5–7 days typical)	No taper required if <10 days	GINA
Adrenergic agonist	Epinephrine	Anaphylaxis	0.3–0.5 mg	Intramuscular	Every 5–15 min PRN	Administer in anterolateral thigh; first-line therapy	WAO

6. Stepwise Management of Asthma

Asthma management in primary care follows a stepwise approach aimed at achieving optimal symptom control while minimizing exacerbation risk. Current strategies emphasise the early use of anti-inflammatory therapy rather than reliance on short-acting bronchodilators alone [15]. Patients with infrequent symptoms are managed with as needed low-dose inhaled corticosteroid–formoterol combinations, reflecting a paradigm shift away from short-acting β2-agonist monotherapy [15], [16].

As symptom frequency increases, daily controller therapy with inhaled corticosteroids is introduced. Patients with persistent symptoms require combination therapy with inhaled corticosteroids and long-acting β2-agonists, with regular assessment of adherence and inhaler technique. In cases of inadequate control, escalation to higher doses or the addition of adjunctive therapies is warranted.

Severe or treatment-resistant asthma necessitates specialist referral and consideration of advanced therapies, including biologics targeting specific inflammatory pathways. Regular follow-up is essential, with therapy adjusted according to disease control and patient response [17].

7. Management of Allergic Rhinitis

Allergic rhinitis (AR) is one of the most common allergic conditions encountered in primary care and is frequently underdiagnosed and undertreated. Often dismissed as a benign or permanent cold, AR is associated with substantial morbidity, including impaired sleep quality, reduced cognitive performance, absenteeism, and decreased overall quality of life. Importantly, AR and asthma are closely linked as part of a unified airway disease continuum, with up to 80% of patients with asthma also exhibiting symptoms of rhinitis. Consequently, optimal management of AR is essential not only for symptom control but also for improving asthma outcomes. AR is clinically classified based on symptom frequency (intermittent vs persistent) and severity (mild vs moderate–severe), which guides therapeutic decision-making. Management begins with allergen identification and avoidance strategies, although complete avoidance is often impractical. Environmental control measures, such as reducing indoor allergens (e.g., dust mites, pet dander) and limiting outdoor exposure during high pollen seasons, can provide adjunctive benefit [4], [8], [12], [13].

Pharmacotherapy remains the cornerstone of treatment. Intranasal corticosteroids, including Fluticasone and Mometasone, are considered the most effective first-line therapy for moderate to severe disease due to their potent anti-inflammatory effects across

multiple pathways. They significantly reduce nasal congestion, rhinorrhoea, sneezing, and itching. Their efficacy, however, is dependent on correct administration technique and consistent use, with maximal benefit often observed after several days to weeks. Second-generation oral antihistamines, such as Cetirizine and Loratadine, are effective for controlling sneezing, itching, and rhinorrhoea but are less effective for nasal congestion. Intranasal antihistamines may provide more rapid symptom relief and can be used in combination with intranasal corticosteroids for enhanced efficacy. Leukotriene receptor antagonists such as Montelukast may be considered in patients with coexisting asthma, although their overall efficacy in AR is modest compared to corticosteroids. In patients with persistent or severe disease refractory to pharmacotherapy, allergen immunotherapy (subcutaneous or sublingual) may be considered. This approach targets the underlying immune response and offers the potential for long-term disease modification. Referral to a specialist is recommended in such cases [4], [8], [12], [13].

8. Management of food allergies

Food allergies represent a significant clinical challenge in primary care, particularly in paediatric populations. Effective management requires accurate diagnosis, differentiation between immunological mechanisms, and comprehensive patient education. A critical distinction must be made between IgE-mediated and non-IgE-mediated food allergies. IgE-mediated reactions typically present within minutes to hours of allergen exposure and may involve cutaneous (urticaria, angioedema), gastrointestinal, respiratory, and cardiovascular systems. These reactions carry a risk of anaphylaxis and require urgent recognition. In contrast, non-IgE-mediated reactions are delayed, often presenting hours to days after ingestion, and are primarily gastrointestinal or dermatological in nature. Therefore, patients with IgE-mediated food allergies should be equipped with emergency action plans and prescribed adrenaline auto-injectors where appropriate. Regular follow-up is essential to reassess allergy status, as some food allergies may resolve over time [18].

Management of confirmed food allergy is centred on strict elimination of the offending allergen. This requires careful dietary counselling to ensure nutritional adequacy, particularly in children where growth and development may be impacted [18]. Patients and caregivers must be educated on label reading, cross-contamination risks, and recognition of early symptoms of allergic reactions. A major paradigm shift in recent years has been the emphasis on early allergen introduction as a preventive strategy. Evidence from large-scale trials has demonstrated that introducing allergenic foods, such as peanuts, in high-risk infants between 4–6 months of age can reduce the risk of developing food allergies by up to 80% [19]. This approach has been incorporated into international guidelines and represents a significant advancement in primary prevention.

9. Emergency Management: Anaphylaxis

Anaphylaxis is a severe, systemic hypersensitivity reaction that is rapid in onset and potentially fatal [20]. Prompt recognition and immediate intervention are critical to reducing morbidity and mortality. In the primary care setting, clinicians must maintain a

high index of suspicion, particularly in patients with known allergies presenting with multi-system involvement. Clinical manifestations of anaphylaxis typically involve the airway (laryngeal edema, stridor), respiratory system (bronchospasm, dyspnoea), cardiovascular system (hypotension, syncope), and skin (urticaria, angioedema). However, absence of cutaneous signs does not exclude the diagnosis [20].

The cornerstone of treatment is the immediate administration of intramuscular Epinephrine into the anterolateral thigh at a dose of 0.3–0.5 mg in adults. This should be administered promptly at the first sign of anaphylaxis, as delays are associated with worse outcomes [20]. Repeat dosing may be required every 5–15 minutes depending on clinical response. Supportive measures include positioning the patient supine with elevated legs to improve venous return, administering high-flow oxygen, and establishing intravenous access for fluid resuscitation in cases of hypotension. Adjunctive therapies, including antihistamines and corticosteroids, may be administered but should never delay or replace adrenaline. Following stabilization, patients should be monitored for biphasic reactions, which can occur hours after initial symptom resolution. Long-term management includes identification of triggers, prescription of adrenaline auto-injectors, and comprehensive patient education. Written emergency action plans and training in auto-injector use are essential components of care [20], [21].

10. Prevention and Future Directions

Prevention of allergic diseases is an evolving area of clinical research, with increasing emphasis on early-life interventions. Primary prevention strategies include early introduction of allergenic foods, avoidance of unnecessary dietary restrictions, and promotion of breastfeeding where possible. Environmental control measures, such as reducing exposure to tobacco smoke and indoor allergens also play a role. Secondary prevention focuses on early identification and optimal management of existing allergic conditions to prevent progression along the atopic march. This includes effective treatment of atopic dermatitis and allergic rhinitis to potentially reduce the risk of subsequent asthma development.

The future of allergy management is being transformed by advances in immunology and molecular medicine. Biologic therapies targeting specific immune pathways, including IgE and interleukins (IL-4, IL-5, IL-13), are increasingly used in severe asthma and atopic dermatitis. Agents such as Omalizumab and Dupilumab exemplify the shift toward precision medicine, where treatment is tailored to individual inflammatory profiles. Recently emerging research into the role of the microbiome, epigenetics, and immune tolerance induction holds promise for future preventive and therapeutic strategies. As these innovations evolve, primary care clinicians will need to remain informed and adapt practice accordingly.

11. Conclusion

Allergic diseases constitute a major and growing burden in primary care, requiring a structured, evidence-based approach to diagnosis and management. Family practitioners play a pivotal role in early recognition, initiation of therapy, and coordination of care across the disease spectrum. Effective management hinges on a thorough clinical assessment, judicious use of diagnostic testing, and implementation of stepwise treatment strategies tailored to disease severity and patient characteristics. Pharmacological therapy, combined with patient education and preventive strategies, forms the foundation of care. Timely recognition and management of high-risk conditions such as anaphylaxis are critical and can be lifesaving. Additionally, evolving approaches to prevention, including early allergen introduction, and advances in targeted therapies are reshaping the landscape of allergy management. Ultimately, improving outcomes in allergic disease requires not only adherence to clinical standards but also a patient-centred approach that emphasises education, empowerment, and continuity of care. As the prevalence of allergic diseases continues to rise, the role of the family practitioner will remain central in delivering high-quality, comprehensive care.

12. Reference list

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13. Addendum

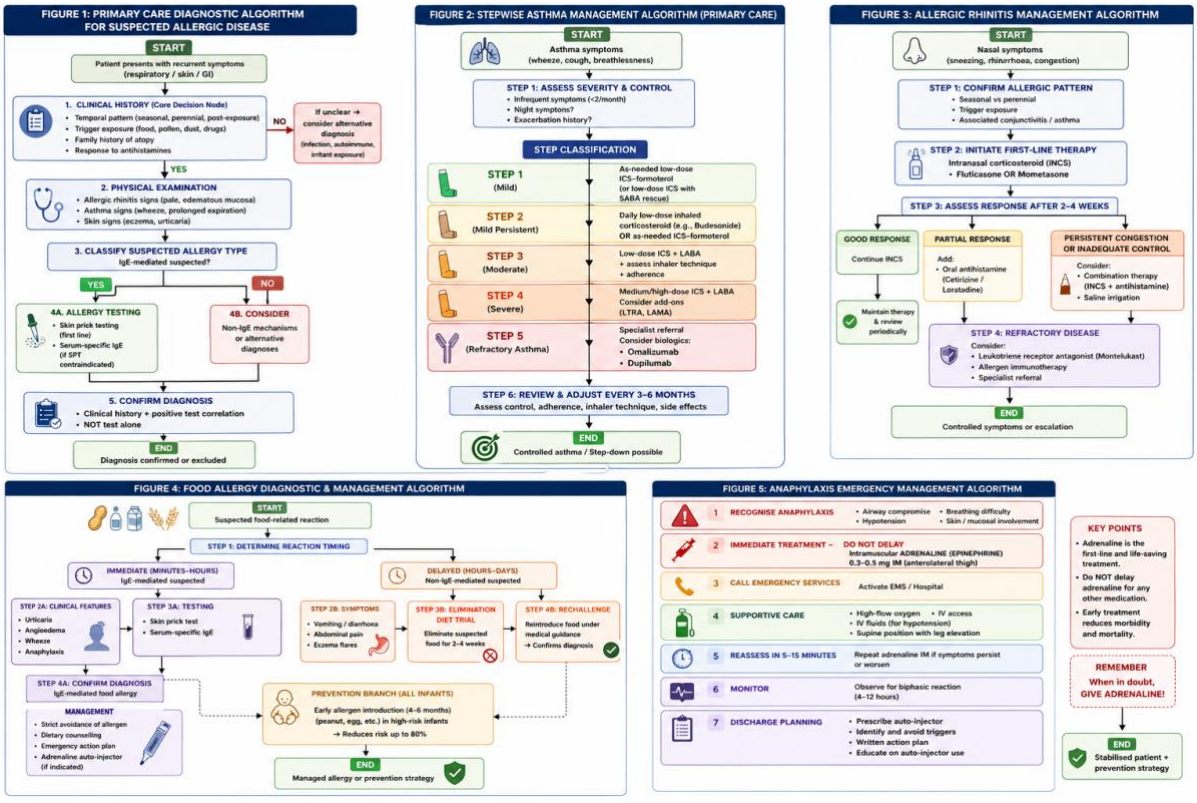
FIGURE 1: PRIMARY CARE DIAGNOSTIC ALGORITHM FOR SUSPECTED ALLERGIC DISEASE
START
Patient presents with recurrent symptoms (respiratory / skin / GI)
1. CLINICAL HISTORY (Core Decision Node)
Temporal pattern (seasonal, perennial, post-exposure)
Trigger exposure (food, pollen, dust, drugs)
Family history of atopy
Response to antihistamines
NO
If unclear → consider alternative diagnosis (infection, autoimmune, irritant exposure)
YES
2. PHYSICAL EXAMINATION
Allergic rhinitis signs (pale, edematous mucosa)
Asthma signs (wheeze, prolonged expiration)
Skin signs (eczema, urticaria)
3. CLASSIFY SUSPECTED ALLERGY TYPE
IgE-mediated suspected?
YES
NO
4A. ALLERGY TESTING
Skin prick testing (first line)
Serum-specific IgE (if SPT contraindicated)
4B. CONSIDER
Non-IgE mechanisms or alternative diagnoses
5. CONFIRM DIAGNOSIS
Clinical history + positive test correlation
NOT test alone
END
Diagnosis confirmed or excluded
FIGURE 2: STEPWISE ASTHMA MANAGEMENT ALGORITHM (PRIMARY CARE)
START
Asthma symptoms (wheeze, cough, breathlessness)
STEP 1: ASSESS SEVERITY & CONTROL
Infrequent symptoms (<2/month)
Night symptoms?
Exacerbation history?
STEP CLASSIFICATION
STEP 1 (Mild)
As-needed low-dose ICS–formoterol (or low-dose ICS with SABA rescue)
STEP 2 (Mild Persistent)
Daily low-dose inhaled corticosteroid (e.g., Budesonide) OR as-needed ICS–formoterol
STEP 3 (Moderate)
Low-dose ICS + LABA + assess inhaler technique + adherence
STEP 4 (Severe)
Medium/high-dose ICS + LABA Consider add-ons (LTRA, LAMA)
STEP 5 (Refractory Asthma)
Specialist referral Consider biologics:
Omalizumab
Dupilumab
STEP 6: REVIEW & ADJUST EVERY 3–6 MONTHS
Assess control, adherence, inhaler technique, side effects
END
Controlled asthma / Step-down possible
FIGURE 3: ALLERGIC RHINITIS MANAGEMENT ALGORITHM
START
Nasal symptoms (sneezing, rhinorrhoea, congestion)
STEP 1: CONFIRM ALLERGIC PATTERN
Seasonal vs perennial
Trigger exposure
Associated conjunctivitis / asthma
STEP 2: INITIATE FIRST-LINE THERAPY
Intranasal corticosteroid (INCS)
Fluticasone OR Mometasone
STEP 3: ASSESS RESPONSE AFTER 2–4 WEEKS
GOOD RESPONSE
Continue INCS
Maintain therapy & review periodically
PARTIAL RESPONSE
Add:
Oral antihistamine (Cetirizine / Loratadine)
PERSISTENT CONGESTION OR INADEQUATE CONTROL
Consider:
Combination therapy (INCS + antihistamine)
Saline irrigation
STEP 4: REFRACTORY DISEASE
Consider:
Leukotriene receptor antagonist (Montelukast)
Allergen immunotherapy
Specialist referral
END
Controlled symptoms or escalation
FIGURE 4: FOOD ALLERGY DIAGNOSTIC & MANAGEMENT ALGORITHM
START
Suspected food-related reaction
STEP 1: DETERMINE REACTION TIMING
IMMEDIATE (MINUTES–HOURS)
IgE-mediated suspected
DELAYED (HOURS–DAYS)
Non-IgE-mediated suspected
STEP 2A: CLINICAL FEATURES
Urticaria
Angioedema
Wheeze
Anaphylaxis
STEP 3A: TESTING
Skin prick test
Serum-specific IgE
STEP 4A: CONFIRM DIAGNOSIS
IgE-mediated food allergy
MANAGEMENT
Strict avoidance of allergen
Dietary counselling
Emergency action plan
Adrenaline auto-injector (if indicated)
STEP 2B: SYMPTOMS
Vomiting / diarrhoea
Abdominal pain
Eczema flares
STEP 3B: ELIMINATION DIET TRIAL
Eliminate suspected food for 2–4 weeks
STEP 4B: RECHALLENGE
Reintroduce food under medical guidance
→ Confirms diagnosis
PREVENTION BRANCH (ALL INFANTS)
Early allergen introduction (4–6 months) (peanut, egg, etc.) in high-risk infants
→ Reduces risk up to 80%
END
Managed allergy or prevention strategy
FIGURE 5: ANAPHYLAXIS EMERGENCY MANAGEMENT ALGORITHM
1 RECOGNISE ANAPHYLAXIS
Airway compromise
Breathing difficulty
Hypotension
Skin / mucosal involvement
2 IMMEDIATE TREATMENT – DO NOT DELAY
Intramuscular ADRENALINE (EPINEPHRINE) 0.3–0.5 mg IM (anterolateral thigh)
3 CALL EMERGENCY SERVICES
Activate EMS / Hospital
4 SUPPORTIVE CARE
High-flow oxygen
IV access
IV fluids (for hypotension)
Supine position with leg elevation
5 REASSESS IN 5–15 MINUTES
Repeat adrenaline IM if symptoms persist or worsen
6 MONITOR
Observe for biphasic reaction (4–12 hours)
7 DISCHARGE PLANNING
Prescribe auto-injector
Identify and avoid triggers
Written action plan
Educate on auto-injector use
KEY POINTS
Adrenaline is the first-line and life-saving treatment.
Do NOT delay adrenaline for any other medication.
Early treatment reduces morbidity and mortality.
REMEMBER
When in doubt, GIVE ADRENALINE!
END
Stabilised patient + prevention strategy