GUIDELINES

Early identification and management of chronic kidney disease in adults: summary of updated NICE guidance

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This is one of a series of *BMJ* summaries of new guidelines based on the best available evidence; they highlight important recommendations for clinical practice, especially where uncertainty or controversy exists.

Further information about the guidance and the supporting evidence statements are in the full version on thebmj.com

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Previous articles in this series

- Lipid modification and cardiovascular risk assessment for the primary and secondary prevention of cardiovascular disease: summary of updated NICE guidance (*BMJ* 2014;349:g4356)
- The management of atrial fibrillation: summary of updated NICE guidance (*BMJ* 2014;348:g3655)
- Prevention and management of pressure ulcers: summary of NICE guidance (*BMJ* 2014;348:g2592)
- Management of psychosis and schizophrenia in adults (*BMJ* 2014;348:g1173)
- Early management of head injury: summary of updated NICE guidance (*BMJ* 2014;348:g104)

The publication of an internationally accepted definition and classification of chronic kidney disease (CKD) 12 years ago precipitated a proliferation of research and literature surrounding the identification of CKD, risk factors for progression, and important prognostic factors.[1] It also stimulated continuing debate concerning the criteria used for definition of CKD and exactly how ageing influences CKD and its sequelae.[2 3] The National Institute for Health and Care Excellence (NICE) has modified its previous recommendations on the classification of CKD,[4] partly based on Kidney Disease Improving Global Outcomes (KDIGO)[5] and driven by prognostic data from large observational studies. NICE has also recognised a requirement for better identification of people at risk of adverse outcome and covers some key areas relating to the management of CKD, including frequency of monitoring, progression of CKD, acute kidney injury, and renin-angiotensin system blockade. This article summarises the most recent recommendations from NICE.[6]

Recommendations

NICE recommendations are based on systematic reviews of best available evidence and explicit consideration of cost effectiveness. When minimal evidence is available, recommendations are based on the Guideline Development Group's experience and opinion of what constitutes good practice. Evidence levels for the recommendations are in the full version of this article on thebmj.com.

Investigations for chronic kidney disease

- Clinical laboratories should:
  - Use the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) creatinine equation to estimate GFRcreatinine, using creatinine assays with calibration traceable to standardised reference material
  - Use creatinine assays that are specific (for example, enzymatic assays) and zero biased compared with isotope dilution mass spectrometry *and*
  - Participate in a UK national external quality assessment scheme for creatinine. (New recommendation.)
- Consider using eGFRcystatinC at initial diagnosis to confirm or rule out CKD in people with:
  - An estimated GFRcreatinine of 45-59 mL/min/1.73 m^2, sustained for at least 90 days *and*
  - No proteinuria (albumin:creatinine ratio <3 mg/mmol) or other marker of kidney disease. (New recommendation.)

Markers of kidney disease include albuminuria (albumin:creatinine ratio >3 mg/mmol), urine sediment abnormalities (haematuria, red blood cell casts, white blood cell casts, oval fat bodies or fatty casts, granular casts, and renal tubular epithelial cells), electrolyte and other abnormalities caused by tubular disorders, abnormalities detected by histology, structural abnormalities detected by imaging, and previous kidney transplantation. Cystatin C is an alternative endogenous filtration marker that can be used to estimate glomerular filtration rate. In people with GFR above 45 mL/min/1.73 m^2, GFR estimates based on cystatin C are more powerful predictors of clinical outcomes than is creatinine based estimated GFR, especially in people with no proteinuria.[7 8]

- Do not diagnose CKD in people with:
  - An estimated GFRcreatinine of 45-59 mL/min/1.73 m^2 *and*
  - An estimated GFRcystatinC of more than 60 mL/min/1.73 m^2 *and*
  - No other marker of kidney disease. (New recommendation.)
- To detect and identify proteinuria, use urine albumin:creatinine ratio in preference to protein:creatinine ratio, because it has greater sensitivity for low levels of proteinuria. For quantification and monitoring of high levels of proteinuria (albumin:creatinine ratio ≥70 mg/mmol), protein:creatinine ratio can be used as an alternative. Albumin:creatinine ratio is the recommended method for people with diabetes. (Updated recommendation.)
- Offer testing for CKD using estimated GFRcreatinine and albumin:creatinine ratio to people with any of the following risk factors:
  - Diabetes
  - Hypertension
  - Acute kidney injury
  - Cardiovascular disease (ischaemic heart disease, chronic heart failure, peripheral vascular disease, or cerebral vascular disease)
  - Structural renal tract disease, renal calculi, or prostatic hypertrophy
  - Multisystem diseases with potential kidney involvement—for example, systemic lupus erythematosus
  - Family history of end stage kidney disease (GFR category G5) or hereditary kidney disease
  - Opportunistic detection of haematuria. (Updated recommendation.)

Classification of chronic kidney disease

- Classify CKD by using a combination of GFR and albumin:creatinine ratio (ACR) categories (as illustrated in figure 1). Be aware that:
  - Increased albumin:creatinine ratio is associated with increased risk of adverse outcomes
  - Decreased GFR is associated with increased risk of adverse outcomes

– Increased albumin:creatinine ratio and decreased GFR in combination multiply the risk of adverse outcomes. (New recommendation.)

An incidental finding of clinically important proteinuria should always be considered in the context of GFR category.

Indications for renal ultrasound

- Offer a renal ultrasound scan to all people with CKD who:
  - Have accelerated progression of CKD (see "Defining progression of CKD")
  - Have visible or persistent invisible haematuria
  - Have symptoms of urinary tract obstruction
  - Have a family history of polycystic kidney disease and are aged over 20 years
  - Have a GFR of less than 30 mL/min/1.73 m^2 (GFR category G4 or G5)
  - Are considered by a nephrologist to require a renal biopsy. (Updated recommendation.)

Frequency of monitoring

- Use figure 2 to guide the frequency of GFR monitoring for people with or at risk of CKD, but tailor it to the person according to:
  - The underlying cause of CKD
  - Past patterns of estimated GFR and albumin:creatinine ratio (but be aware that progression of CKD is often non-linear)
  - Comorbidities, especially heart failure
  - Changes to their treatment (such as renin-angiotensin-aldosterone system antagonists, non-steroidal anti-inflammatory drugs, and diuretics)
  - Intercurrent illness
  - Whether they have chosen conservative management of CKD. (New recommendation.)
- Monitor people for the development or progression of CKD for at least two to three years after acute kidney injury, even if serum creatinine has returned to baseline. (New recommendation.)

Defining progression of CKD

- Define accelerated progression of CKD as:
  - A sustained decrease in GFR of 25% or more and a change in GFR category within 12 months *or*
  - A sustained decrease in GFR of 15 mL/min/1.73 m^2 per year. (New recommendation.)

Self management

- Ensure that systems are in place to:
  - Inform people with CKD of their diagnosis
  - Enable people with CKD to share in decision making about their care
  - Support self management (this includes providing information about blood pressure, smoking cessation, exercise, diet, and medicines) and enable people to make informed choices. (New recommendation.)

People should be encouraged to take exercise, achieve a healthy weight, stop smoking, and restrict salt intake to less than 6 g/day according to existing guidance (see www.nice.org.uk/guidance/PH25).

Indications for referral

- People with CKD in the following groups should normally be referred for specialist assessment:
  - GFR less than 30 mL/min/1.73 m^2 (GFR category G4 or G5), with or without diabetes*
  - Albumin:creatinine ratio 70 mg/mmol or more, unless known to be caused by diabetes and already appropriately treated
  - Albumin:creatinine ratio 30 mg/mmol (category A3) or more, together with haematuria
  - Sustained decrease in GFR of 25% or more and a change in GFR category or sustained decrease in GFR of 15 mL/min/1.73 m^2 or more within 12 months
  - Hypertension that remains poorly controlled despite the use of at least four antihypertensive drugs at therapeutic doses (see also NICE clinical guideline 127 on hypertension (www.nice.org.uk/Guidance/CG127))
  - Known or suspected rare or genetic causes of CKD
  - Suspected renal artery stenosis. (Updated recommendation.)

*Where this is a stable isolated finding, formal referral may not be indicated and advice may be all that is required. The aim is to avoid late referral of those people likely to progress to requirement for renal replacement therapy within one year.

- People with CKD and renal outflow obstruction should normally be referred to urological services, unless urgent medical intervention is required—for example, for the treatment of hyperkalaemia, severe uraemia, acidosis, or fluid overload.

Pharmacotherapy

Choice of antihypertensive agent and blood pressure control

- Offer a low cost renin-angiotensin system antagonist to people with CKD and:
  - Diabetes and an albumin:creatinine ratio of 3 mg/mmol or more (category A2 or A3)
  - Hypertension and an albumin:creatinine ratio of 30 mg/mmol or more (category A3)
  - An albumin:creatinine ratio of 70 mg/mmol or more (irrespective of hypertension or cardiovascular disease). (New recommendation.)

The evidence to support these criteria is limited in people aged over 70 years.

- Do not offer a combination of renin-angiotensin system antagonists to people with CKD. (New recommendation.)
- Follow the treatment recommendations in NICE clinical guideline 127 (www.nice.org.uk/Guidance/CG127) (on hypertension) for people with CKD, hypertension, and an albumin:creatinine ratio of less than 30 mg/mmol (categories A1 and A2) if they do not have diabetes. (New recommendation.)

Blood pressure control recommendations for people with CKD and diabetes and an albumin:creatinine ratio of greater than 3 mg/mmol (categories A2 and A3) and hypertension were not updated.

- In people with CKD, aim to keep the systolic blood pressure below 140 mm Hg (target range 120-139 mm Hg) and the diastolic blood pressure below 90 mm Hg.

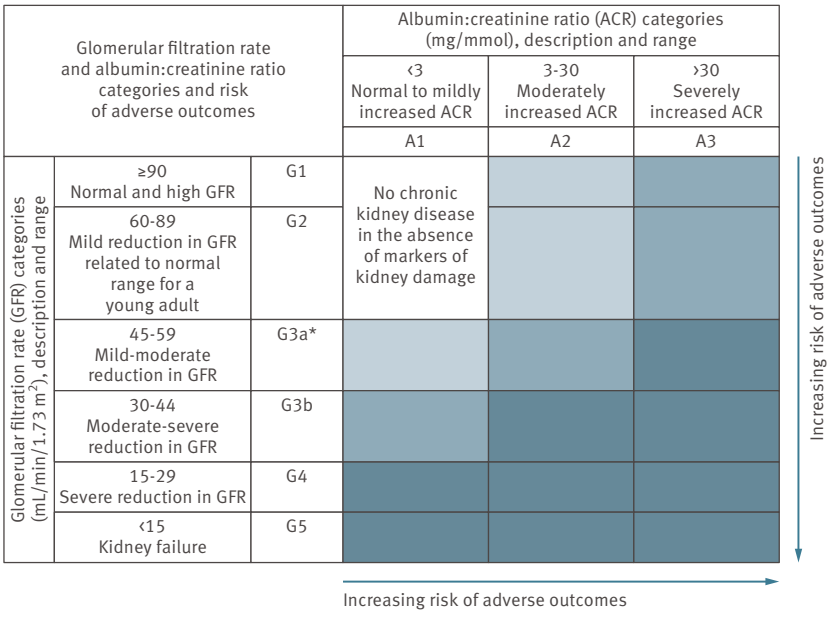

Fig 1 | Classification of chronic kidney disease by using glomerular filtration rate (GFR) and albumin:creatinine ratio categories. *Consider using estimated GFRcystatinC for people with CKD G3aA1. Adapted with permission from reference 5

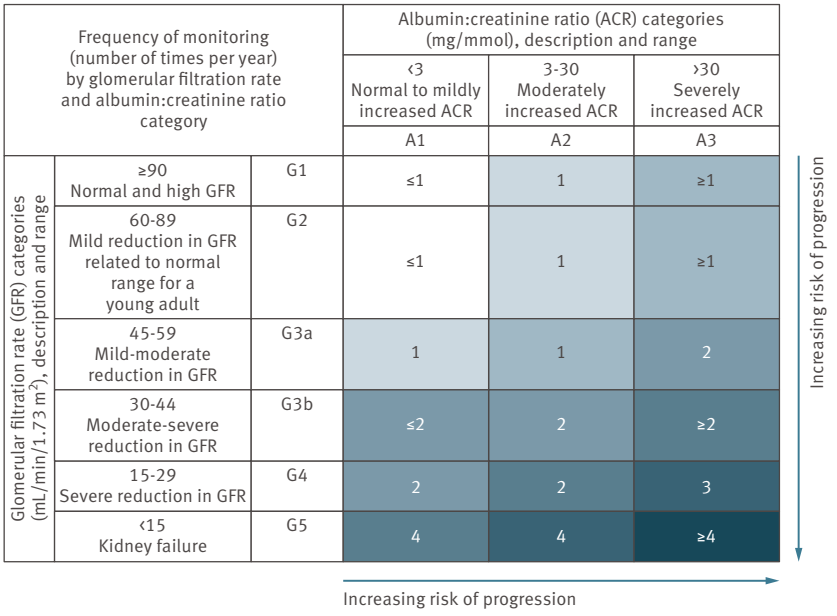

Fig 2 | Frequency of monitoring of glomerular filtration rate for people with or at risk of chronic kidney disease. Albumin:creatinine ratio is an important indicator of cardiovascular risk and progression. Intensity of shading reflects risk of progression. Adapted with permission from reference 5

- In people with CKD and diabetes, and also in people with an albumin:creatinine ratio of 70 mg/mmol or more (category A3), aim to keep the systolic blood pressure below 130 mm Hg (target range 120-129 mm Hg) and the diastolic blood pressure below 80 mm Hg.

Oral antiplatelets and anticoagulants

- Offer antiplatelet drugs to people with CKD for the secondary prevention of cardiovascular disease, but be aware of the increased risk of bleeding. (New recommendation.)

Overcoming barriers

The use of thresholds for diagnosis of CKD and the definition of progression remain contentious areas. In the whole adult population, the CKD-EPI creatinine equation reduces bias in GFR estimation, affords greater precision, and is better at categorising people at higher risk of all cause mortality, cardiovascular mortality, myocardial infarction, progression of CKD, and end stage kidney disease.[9 10] Categorisation by GFR in the new classification is complemented by categorisation of urinary albumin:creatinine ratio. Strong evidence shows that both lower GFR and greater albumin:creatinine ratio are independently related to mortality, cardiovascular events, progression to end stage kidney disease, and also acute kidney injury. This is important, although intervention trials in the different albumin:creatinine ratio categories are lacking, albuminuria increases the risk of acute kidney injury, and acute kidney injury predicts progression of CKD. The diagnosis of CKD in people without significant proteinuria in the GFR category 45-59 mL/min/1.73 m^2 (G3aA1), especially older people, continues to raise concerns regarding over-diagnosis. Use of cystatin C identifies those at higher risk of adverse outcomes, including end stage kidney disease, and overcomes some of the concerns relating to potential over-diagnosis and disease labelling. Another contentious area is the definition of progression of CKD. The recommendation is supported by more recent evidence and attempts to recognise both the inherent variability of serum creatinine in the assessment of GFR and the fact that progression of kidney disease is often not linear.[11-13] We used the term "accelerated progression" in the knowledge that reported decline in GFR among people with CKD may be lower (reviewed extensively in reference 5). Finally, categorisation by GFR and albumin:creatinine ratio also provides a template for frequency of monitoring in people with established CKD and aids decision making regarding referral.

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RATIONAL TESTING

Diagnosis of immediate food allergy

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This series of occasional articles provides an update on the best use of key diagnostic tests in the initial investigation of common or important clinical presentations. The series advisers are Steve Atkin, professor of medicine, Weill Cornell Medical College in Qatar, and Eric Kilpatrick, honorary professor, department of clinical biochemistry, Hull Royal Infirmary, Hull York Medical School. To suggest a topic for this series, please email us at practice@bmj.com.

A 10 month old girl with a history of eczema was brought to the emergency department with urticaria and angioedema without respiratory or circulatory features to suggest anaphylaxis (see glossary). Ten minutes before the symptoms appeared she had eaten some lightly cooked (scrambled) egg. The emergency department doctor administered oral antihistamines and instituted close clinical monitoring. Within 30 minutes symptoms had resolved, and the child was discharged after a six hour period of observation. The doctor explained the likely diagnosis of an egg allergy and recommended avoidance of foods containing egg. An appointment with the GP to consider further investigation or onward referral to a paediatric allergy service was advised. When consulting their GP the next day, the parents asked about tests to confirm egg allergy and whether an adrenaline auto-injector was necessary.

What is the next investigation?

Before allergy testing is carried out a focused history is essential (box 1). Urticaria and angioedema (see glossary) are compatible with an immediate hypersensitivity reaction to food but can also occur spontaneously without any allergic trigger. In immediate (IgE mediated) food allergy the allergen exposure should have a close temporal relation with the onset of symptoms.[1] Symptoms will typically begin within seconds/minutes of exposure to the allergen and resolve before 12 hours. In this case, symptoms began within 10 minutes and the total duration of symptoms was two hours.

The next appropriate investigation is to validate the clinical suspicion of egg allergy by performing skin prick or specific IgE testing. Access to these tests for generalists can vary between services and, in some instances, will first require referral to specialist allergy services. Though both methods of testing were assessed by the National Institute for Care and Health Excellence (NICE) in the 2011 guideline for the diagnosis and assessment of food allergy in children,[2] many of the studies reviewed were of poor quality. The guideline recommends these tests in the appropriate setting and when there is adequate competency in the interpretation of results. For many non-specialists this interpretation can require the advice of experienced allergy practitioners.

Box 1 | Key questions in an allergy focused history

Was there an exposure to a suspected allergen?
The most common allergens in children are egg, peanut, and milk. In adults, peanut, tree nut, fish, and shellfish are most common

How soon after exposure did symptoms begin?
Symptoms typically begin within minutes of exposure but can take up to two hours

What were the reported symptoms and were they suggestive of an allergic reaction?
Symptoms of an allergic reaction include immediate oral pruritus and swelling, urticaria, abdominal pain, vomiting, throat tightness, stridor, cough, respiratory distress, drowsiness, and collapse

Were there additional cofactors present?
Some allergic reactions need the addition of cofactors before the reaction is clinically apparent. Specific examples include infection, alcohol, exercise, and use of non-steroidal anti-inflammatory drugs (NSAIDs)

Has there been uneventful exposure to the potential allergen before or after the reaction?
Ask about how the food was prepared (raw versus cooked), and consider a dose dependent threshold for reactivity. Consider whether the implicated food has been correctly identified

Is a non-allergic explanation possible?
Did urticaria and angioedema persist despite avoidance of the reputed allergen? Has the same allergen been eaten since without adverse outcome? Spontaneous urticaria and angioedema should be considered in these circumstances

KEY POINTS

- Before you consider allergy testing, take a focused history: ask about the suspected allergen, symptoms, and their timing in relation to suspected allergen exposure
- Skin prick and specific IgE testing should be used to diagnose immediate food allergy only when the clinical history supports this
- Select only the specific allergens suspected in the clinical history. The use of specific IgE food panels or a blanket screening approach is likely to yield positive results that are difficult to interpret
- Positive results of specific IgE or skin prick testing without clinical reactivity denote sensitisation and dietary restriction is not recommended
- Unnecessary dietary restriction increases the risk of nutritional deficiencies and parental/patient anxiety

In our scenario, the GP measured specific IgE to both egg white and egg yolk, the results of which were 7.52 kU/L and 0.19 kU/L, respectively (reference range 0-0.35 kU/L), and did not request a "food panel" to five common food allergens offered by the local laboratory. Panel testing detects specific IgE to several of the most commonly implicated allergens. This screening approach is problematic and can give rise to unexpected positive results that can be difficult to interpret.

Specific IgE testing: limitations and difficulties in interpretation

Detectable specific IgE does not necessarily imply an allergy.[1] Patients can be sensitised, meaning that they have raised serum concentrations of specific IgE to an allergen without associated immediate symptoms when exposed. The higher the specific IgE, the more likely it is that the test result will be clinically relevant. Despite this, the level of specific IgE does not predict the severity of symptoms. Calculation of specific IgE concentrations with 95% positive predictive values (table 1) has been used to improve diagnostic accuracy.[1] These values are typically calculated from patients with confirmed food allergy and, if used rationally (guided by the history), can lead to an improved certainty of the clinical diagnosis. In the case above the specific IgE results suggest a greater than 95% certainty that the diagnosis of egg allergy considered before the tests is correct.

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Previous articles in this series

- Investigating hypophosphataemia (*BMJ* 2014;348:g3172)
- Using haemoglobin A1c to diagnose type 2 diabetes or to identify people at high risk of diabetes (*BMJ* 2014;348:g2867)
- Investigation of suspected urinary tract infection in older people (*BMJ* 2014;349:g4070)
- Interpreting raised serum prolactin results (*BMJ* 2014;348:g3207)
- Ordering and interpreting hepatitis B serology (*BMJ* 2014;348:g2522)

Table 1 | Specific IgE concentrations with 95% positive predictive values in children and infants*[1]

Allergen	At mean age 5	In infants aged <2
Egg	7 kUA/L	2 kUA/L
Milk	15 kUA/L	5 kUA/L
Peanut	14 kUA/L	Not clearly established

kUA/L=allergen specific KU/L

Table 2 | Advantages and disadvantages of skin prick and specific IgE testing of investigation of allergy*

Specific IgE testing	Skin testing
Sensitive	Often greater sensitivity than specific IgE
Widely available	Available in centres with appropriate reagents, equipment, and trained staff
Results can take days to weeks	Results in 15 minutes, visible to patients
No interference from drugs	Antihistamines, β blockers, and some antidepressants can interfere with results
Standardised laboratory assay	Observer dependent
Interference from high total IgE can cause positive results without clinical allergy	No interference from high total IgE

*Adapted from the ASCIA manual: Skin Prick Testing for the Diagnosis of Allergic Disease.[8]

The most common pitfall with specific IgE testing is the assumption that a positive test result confirms clinical allergy. Incorrect use of specific IgE as a screening test, without a compatible history, can lead to food allergy being erroneously diagnosed.[3] The consequences of incorrect diagnosis can include an unnecessarily restricted diet and patient/parental anxiety. Case reports have even described the loss of tolerance to foods because of prolonged inappropriate exclusion, leading to anaphylaxis on re-introduction.[4] Conversely, a negative specific IgE result can be used to inappropriately refute a diagnosis of allergy. A common example of this includes allergy to fruit and vegetables where the diagnostic performance characteristics of specific IgE testing are poor. In recent years, component resolved diagnostics, which detect specific IgE to epitopes (those sites of an allergen to which antibody binds, such as Gal d1 in egg), have been investigated as an additional diagnostic and prognostic tools.[5] The clinical utility and cost effectiveness have not yet been fully evaluated and remain controversial, and the use of component resolved diagnostics should be restricted to specialists. Requests for specific IgE testing must be tailored to the clinical history and involve an appreciation of its limitations.

Skin prick testing: limitations and difficulties in interpretation

Skin prick testing identifies in vivo sensitisation to allergens. It can be performed in an outpatient setting and involves the epicutaneous introduction of allergen extracts with a standardised lancet.[6] Typically the volar aspect of the forearm, or the back in small infants, is used. The site is then inspected 15 minutes later and compared with suitable positive and negative controls. A weal 3 mm greater in diameter than the negative control is considered a positive test result[4]; smaller yet still important weals can be observed in infants. Again, accurate interpretation of results requires an appropriate clinical history. Skin prick tests can fail to show sensitisation when antihistamines are used concurrently, and training in measurement and interpretation of results is required. In some cases, such as with fruit and vegetables, skin prick testing with fresh allergen is desirable as the performance of the distilled extract is poor. The choice of specific IgE or skin prick testing should be individualised to the patient and reflect the advantages and limitations of each test and the available expertise of the doctor involved (table 2).[7]

Oral food challenge testing

The ideal test to confirm or refute the diagnosis of immediate food allergy is the double blind placebo controlled food challenge carried out by specialist allergy services.[8] Blinded challenges allow confident identification of clinical allergy and exclude asymptomatic sensitisation and functional symptoms. This investigative strategy is particularly important when the history and test results are ambiguous. In practice, specialist centres routinely provide unblinded challenges, although access to these resource intensive investigations might be rationed in some healthcare systems. Various protocols exist to perform these tests, but all challenge protocols include the administration of increasing quantities of a proposed allergen under medical supervision. Direct mucosal exposure (allergen held to lip) is the first stage of the challenge before titrated oral ingestion. In certain cases, after risk stratification by experienced practitioners, home challenges might be recommended.[9] Failure to directly provoke symptoms will confidently exclude an allergy in most cases.

Outcome

Based on the history and specific IgE results, the GP diagnosed egg allergy. By avoiding blanket/panel allergy testing, the GP avoided the common pitfall of detecting multiple, clinically irrelevant, low level positive specific IgE results. She did not prescribe an adrenaline auto-injector given the absence of anaphylaxis or severe comorbid conditions—such as poorly controlled asthma—and the low incidence of life threatening reactions to egg.[9] For other allergens, an adrenaline auto-injector can be considered in the absence of life threatening features if there are comorbid conditions or the allergen is difficult to avoid or has a high associated risk of anaphylaxis (such as with peanut). A written management plan was agreed with the parents, clearly setting out the use of antihistamines for skin limited reactions after accidental exposure, and reasons to seek emergency help. Although this was the first exposure to scrambled egg (lightly cooked), the GP noted that the child had previously eaten baked egg (well cooked) uneventfully and thus advised avoiding raw and lightly cooked egg in the diet, but continuing previously tolerated well cooked egg. She discussed the question of onward referral with the local paediatric allergy service. They agreed that specialist assessment was not required at this stage as symptoms were limited to the skin, the child did not have asthma, and the diagnosis was clear (box 2). Referral for a home based challenge to less well cooked egg, when the child reached 3-4 years, was planned, with the parents' agreement.

Box 2 | Which patients with egg allergy should be referred to an allergy clinic?[10]

- Children with systemic symptoms consistent with a severe reaction
- Children who have poorly controlled asthma or use inhaled glucocorticoids regularly
- When the diagnosis is uncertain
- Persistent egg allergy (beyond age 6-8)
- Egg allergy with another major food allergy

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References are in the version on thebmj.com.