The Management of Coeliac Disease in Infants and Children in the UK

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Coeliac disease (CeD) is a chronic, auto-immune mediated enteropathy precipitated by gluten ingestion (present in wheat, barley and rye) in genetically susceptible individuals. CeD affects around 1% of the UK population, with an estimated 70% of these being undiagnosed. This is likely due to a huge variation in the clinical presentation and symptoms of CeD, including some patients being asymptomatic upon diagnosis.[1,2] It is also estimated that this 1% prevalence may be an underestimation of the true prevalence, and prevalence ratings may differ slightly globally.[3]

Diagnosis and management of CeD in the paediatric setting is guided by the 2015 National Institute for Health and Care Excellence (NICE) guidance and the 2020 European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) recommendations. Shifts in care have led to a biopsy-sparing diagnostic pathway for appropriately presenting children, with structured dietetic care and successful dietetic-led practice in many centres with multidisciplinary long-term follow up.

This article reviews contemporary evidence and UK relevant guidance on diagnosis, dietary treatment, nutrition and bone health monitoring, vaccinations, psychosocial support, and the role of human leukocyte antigen (HLA) typing.

Epidemiology & pathophysiology

CeD is triggered by ingestion of gluten (wheat, barley, rye), causing immune-mediated small intestinal injury in genetically predisposed individuals, predominantly those carrying HLA DQ2 or DQ8 haplotypes.[4] It is prudent to adopt a low threshold for testing for CeD in children and teenagers who present with any symptoms or a family history of CeD. It is cost-effective to carry out serum testing for CeD in a patient presenting with symptoms of unknown cause, as earlier diagnosis can help prevent years of significant medical intervention, psychological distress, poor educational outcomes, and ongoing or worsening symptoms impacting quality of life.

Serum testing for CeD must be carried out when sufficient gluten is being ingested (at least one meal containing gluten daily for 6 weeks before testing). When serum tissue transglutaminase (TTG) testing is carried out without sufficient gluten in the diet, the results have the potential to return a false negative.[3]

HLA susceptibility is necessary but not sufficient to cause CeD; around 30-40% of the general population carry the specific HLA genotypes associated with CeD but not all of these people go on to develop CeD. Whereas >99% of patients with CeD have positive DQ2.5, DQ8 or DQ2.2, making HLA testing useful mainly to exclude CeD when these results return negative.[4] To test for genetic predisposition to CeD does not require gluten to be consumed. Therefore, HLA tissue typing can be useful in instances where gluten has already been removed from the child's diet before testing was carried out appropriately and it would be too problematic, or clinically unsafe, to reintroduce gluten at the required amount to ensure a reliable result.

Diagnosis

Symptoms of CeD are vast and display significant heterogeneity, even within the same family. It is important to note that severity of symptoms does not correlate to severity of disease, and some children and young people with CeD will be asymptomatic but have total villous atrophy on biopsy. First line tests are usually serum levels of total IgA and IgA anti tissue transglutaminase (tTG IgA). Where a child or young person has IgA deficiency, the IgG tTG should also be tested and often these patients will require further investigation with endoscopy with duodenal biopsy.

The NICE guidelines (2015)[3] recommend testing children with classical symptoms such as persistent gastrointestinal symptoms (e.g. chronic diarrhoea, abdominal pain), unexplained faltering growth, unexplained iron/folate deficiency, prolonged fatigue, and those with associated conditions (e.g. type 1 diabetes, autoimmune thyroid disease) or first degree relatives of people with CeD.[1]

The NICE guidelines[3] also recommend testing children and young people with non-classical symptoms of CeD, such as peripheral neuropathy and unexplained ataxia. Other non-classical symptoms that should indicate a CeD screen as part of diagnostic work-up may include headache, brain fog, poor sleep, behavioural difficulties and migraine.

Cutting edge research has yielded promising scope to test for whole-blood assay interleukin (IL-2) presence in a serum sample of a patient with suspected CeD who is not eating dietary gluten. Although not yet part of mainstream practice, this is a keystone change to the diagnostic pathways for all patients with suspected CeD.[5]

ESPGHAN 2020 biopsy sparing criteria

ESPGHAN's 2020 guideline supports a no biopsy diagnosis in children when both (a) tTG IgA ≥10× upper limit of normal using a validated assay, and (b) endomysial antibody (EMA IgA) positivity is confirmed in a second sample; HLA typing and the presence of symptoms are not required for this serology based pathway. Otherwise, upper GI endoscopy, with multiple duodenal biopsies (≥4 distal, ≥1 bulb), is recommended. The approach is widely adopted in UK services and reaffirmed in local NHS paediatric guideline updates.

Dietary management: The lifelong gluten-free diet

Gluten-free diet

A strict, lifelong gluten-free diet (GFD) remains the only established therapy for CeD and requires comprehensive education from specialist paediatric dietitians. The strict avoidance of wheat, barley and rye is necessary to avoid stimulating the immune cascade response that occurs in individuals with CeD.

Patients with CeD need to remove all traces of gluten to maintain a diet with gluten levels less than 20 ppm, which is deemed currently to be safe for patients with CeD.[3] It is important to note that this numerical threshold differs globally and is currently under review.

To maintain a diet less than 20 ppm, patients with CeD must adhere to strict practices to remove traces of cross contamination of gluten in the home and when eating out, and avoid products that state a 'may contain' gluten warning. Patients are advised to use separate toasters, butter and condiments, and eat from clean chopping boards, cutlery, plates and saucepans, etc. Sharing spoons between cooking water of gluten and gluten-free foods is not allowed. Careful use of clean oven trays and air fryers must be part of education.

Patients are advised to use reliable sources of information from Coeliac UK, who produce a venue guide and app to help identify suitable gluten-free (GF) foods depending on the individual's dietary settings added.[6]

The inclusion of pure/GF oats, which are not contaminated with gluten from milling in a factory alongside wheat, rye and barley, is generally considered safe for most children with CeD and can improve diet quality due to the high nutritional value of oats. Practice differs nationally on whether the inclusion of GF oats can begin at the start of treatment upon diagnosis or await normalised tTG and resolution of symptoms. Evidence from randomised trials and systematic reviews indicates no adverse effect on mucosal healing or serology, though individual sensitivity can vary and for children with ongoing symptoms and consistently high TTG despite the GFD, GF oats should be considered as a cause with a trial for removal.

Recent immunological data continue to refine our understanding of avenin responses in children with CeD and may inform future guidance. Hardy *et al.*[7] investigated the immunological effects of incorporating GF oats into the diets of individuals with CeD. While avenin induced an immunological response in 38% of participants and symptomatic reactions in 59%, both responses diminished with prolonged exposure. Furthermore, this exposure did not induce enteropathy.

Dietetic support & adherence

NICE recommends access to specialist dietetic advice at diagnosis and follow up, including education regarding label reading, cross-contamination avoidance (home, school, eating out), and ensuring nutritional adequacy (kcals, protein, iron, folate, calcium, zinc, vitamin D, fibre).[3]

Adherence to the diet can fluctuate depending on age and life stage of the patient. This is particularly challenging during adolescence and transition. Family and community support, peer support, group sessions, and attendance to community events such as GF food festivals and Coeliac UK meet ups, can all support young people to reduce these feelings of isolation. Young people can benefit from structured dietary education, eating out and travel guidance, treating nutrient deficiencies, accessing digital resources, school liaison to ensure safe and varied GF food is available, and family-centred counselling to ensure that the whole family gain understanding of the severity and necessary steps.

It can be necessary in clinical practice to reiterate the risks related to poor adherence to the GFD. Poor dietary adherence is linked to complications such as poor growth, poor academic performance, osteoporosis, infertility, and increased risk of intestinal lymphoma, as well as a higher risk of mortality.[8]

Monitoring after diagnosis

Clinical & serological follow up

NICE recommends periodic review to assess symptoms, growth, dietary adherence, and need for investigations; tTG IgA (and EMA IgA if needed) is used to monitor response, expecting normalisation over time with a strict GFD.[3] UK paediatric pathways commonly schedule reviews at 6-12 months post diagnosis and annually thereafter, with additional visits for non-responsive patients or dietary lapses. It is important to note that patients with CeD who are IgA deficient and are thus reliant on IgG serology to monitor GF compliance may find that their IgG markers often take significantly longer to normalise than patient's who use tTG-IgA to monitor their GF compliance despite following a strict GFD.

Nutritional status & bone health

Children with CeD are at risk of nutritional deficiencies (iron, folate, B12, vitamin D) and low bone mineral density (BMD) at diagnosis due to malabsorption secondary to villous atrophy and inflammation; vitamin D should be measured and corrected, and calcium intake optimised. It should be noted that serum calcium measurements are not reliable to diagnose calcium deficiency; a normal serum calcium does not rule out a deficient intake and careful dietary assessment should take place to ensure calcium requirements are met.

Routine DEXA (dual-energy X-ray aAbsorptiometry) scanning in all children is not universally recommended; UK tertiary centres increasingly reserve DEXA for patients in high-risk subgroups (e.g. poor growth, delayed diagnosis, persistent seropositivity, fractures, prolonged dietary non-adherence).[9, 10]

Non-responsive & refractory disease

Persistent symptoms despite a strict GFD (that is free from all oats) warrant re-evaluation for hidden gluten exposure, alternative diagnoses (e.g. lactose intolerance, small intestinal bacterial overgrowth), with medical review and multidisciplinary team discussion. Paediatric refractory CeD is rare but can occur and should be managed by a specialist team with experience in refractory CeD.[3]

Vaccinations & functional hyposplenism

Adults with CeD have a recognised prevalence of functional hyposplenism; this is less common in children, but UK practice stresses individual risk assessment. The UK Green Book guidance provides details on the immunisation of people with splenic dysfunction (including additional pneumococcal, meningococcal, and influenza vaccination).[11] Coeliac UK's Health Advisory Council recommends pneumococcal vaccination for people with CeD (with booster intervals in adults).[12] Infants are already covered by the routine vaccination programme and paediatric decisions should align with national vaccination schedules and local specialist advice.

Psychosocial aspects & quality of life

Living a strict GF life affects every aspect of daily living. Children experience a significant change in their social participation (school meals, parties, travel), and the long-term nature of the condition can influence wellbeing and adherence, especially in adolescents. A recent systematic review in children and adolescents highlighted mixed findings on health-related quality of life but consistent challenges with social functioning, supporting integration of psychosocial care into routine follow up.[13]

Prescribing of gluten-free products

The cost of GF foods from the supermarket are significantly higher than the equivalent gluten-containing foods. The GFD results in a significant financial burden for families having a significant impact on adherence to the GFD and also availability of nutritional quality and variation. This should be taken into account wherever possible, with practical advice enabling families to eat foods that are naturally GF, including potatoes, rice, vegetables, meats, fish, cheese, milk, nuts and beans. In some areas of the country there are still GF items available on prescription for eligible patients. However, many local authorities are deciding to remove these services in line with cost-saving pressures to the NHS overall.

Dermatitis herpetiformis (DH)

Children can present with DH, although this may be misdiagnosed as atopic dermatitis. In children with DH, a diagnosis of CeD can be made clinically with dermatology input and a skin biopsy is needed to diagnose DH. Management of DH aligns with CeD – a strict GFD, with or without dapsone for symptom control per specialist advice.[3]

Practical management checklist

1. **Before testing:** Ensure the child is eating a gluten-containing diet (gluten in >1 meal/day for ≥6 weeks).
2. **Initial tests:** Total IgA + tTG IgA; consider IgG-based assays (tTG/EMA/DGP) if IgA deficient.
3. **Diagnosis:** Apply ESPGHAN 2020 criteria[2] for no biopsy pathway (tTG IgA ≥10×ULN + second sample EMA IgA); otherwise proceed to endoscopic biopsy.
4. **At diagnosis:** Refer to specialist paediatric dietitian; screen for nutritional deficiencies (full blood count, ferritin, folate, B12, vitamin D), plot growth centiles, discuss school care plans and cross contamination. Careful assessment of calcium intake versus requirements.
5. **Follow up:** Review at ~6–12 months, then annually; monitor symptoms, growth, adherence, and tTG IgA; address diet quality and micronutrients; reserve DEXA for at risk cases.
6. **Vaccinations:** Ensure routine UK schedule; assess for hyposplenism in selected cases and follow the 'Green Book'[11] advice for splenic dysfunction if present.
7. **Check if patient is eligible for GF prescribable products.**
8. **Signpost to naturally GF foods in the supermarket;** provide practical advice.
9. **Consider inclusion of GF oats when children are well with good symptom improvement and normalisation of TTG.**
10. **Psychosocial care:** Screen for adherence barriers and wellbeing; offer/signpost to age-appropriate psychological resources.

Conclusions

For infants and children in the UK, management of CeD centres on precise, evidence-based diagnosis (with validated no biopsy pathways), immediate initiation of a strict GFD supported by specialist dietetic care, targeted nutritional and bone health monitoring. Careful consideration should be given to the quality of life and significant social impact of the GFD, and integrated psychosocial support where possible/available. Follow-up must take into account the likelihood of fluctuating adherence depending on life stage; clinicians must be aware of the significant impact a diagnosis of CeD has on a young person.

Ongoing UK efforts to raise awareness and standardise diagnostics (including HLA reporting) and to address psychological and quality of life factors will hopefully enhance adherence and long-term outcomes for infants, children and young adults diagnosed and following a lifelong GFD.

References: **1.** Downey L, *et al.* (2015). Recognition, assessment, and management of coeliac disease: summary of updated NICE guidance. BMJ.; 351: h4513. **2.** Husby S, *et al.* (2020). European Society for Paediatric Gastroenterology, Hepatology and Nutrition guidelines for diagnosing coeliac disease. J Pediatr Gastroenterol Nutr.; 70(1): 141–156. **3.** National Institute for Health and Care Excellence (NICE) (2015). Coeliac disease: recognition, assessment and management (NG20). Accessed online: www.nice.org.uk/guidance/ng20 (May 2026). **4.** Pritchard D, *et al.* (2024). Laboratory testing and clinical interpretation of HLA genotyping results supporting the diagnosis of coeliac disease. Int J Immunogenet.; 51(Suppl 1): 3–20. **5.** Moscatelli OG, *et al.* (2025). Blood-based T-cell diagnosis of celiac disease. Gastroenterol; 169(6): 1253–1267. **6.** Coeliac UK (2026). Accessed online: www.coeliac.org.uk/ (May 2026). **7.** Hardy MY, *et al.* (2025). Purified oat protein can trigger acute symptoms linked to immune activation in coeliac disease patients but not histological deterioration. Gut; 74(6): 906–917. **8.** Catassi C, *et al.* (2022). Coeliac disease. Lancet; 399(10344): 2413–2426. **9.** Duerksen D, *et al.* (2018). Management of bone health in patients with celiac disease. Can Fam Phys.; 64(6): 433–438. **10.** Khweir S, Kiparissi F, Gaynor E (2020). Monitoring bone health in children and young people with coeliac disease – a clinical audit. Arch Dis Child.; 105(Suppl 2): A8.3-A8. **11.** UK Health Security Agency (2020). Guidance: Immunisation of individuals with underlying medical conditions: the green book, chapter 7. Accessed online: www.gov.uk/government/publications/immunisation-of-individuals-with-underlying-medical-conditions-the-green-book-chapter-7 (May 2026). **12.** Coeliac UK (2026). Vaccinations. Accessed online: www.coeliac.org.uk/healthcare-professionals/vaccinations/ (May 2026). **13.** Ho WHJ, Atkinson EL, David AL (2023). Examining the psychosocial well-being of children and adolescents with coeliac disease: a systematic review. J Pediatr Gastroenterol Nutr.; 76(1): e1–e14.