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Early Detection and Integrated Management of Pediatric Celiac Disease: A Multidisciplinary Approach through general practitioner, nursing , laboratory and pediatric gastroenterologist

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Abstract

Celiac disease (CD) in children is a chronic, immune-mediated enteropathy precipitated by gluten exposure in genetically predisposed individuals, with a global prevalence approaching 1% and substantial underdiagnosis that delays treatment and increases the risk of growth failure, micronutrient deficiencies, bone demineralization, pubertal delay, neurocognitive and psychosocial sequelae, and coexisting autoimmune disease burdens. In the pediatric setting, where manifestations are protean and may evolve from "classical" malabsorption to extra-intestinal or subtle phenotypes, **early detection** hinges on vigilant **general practitioner (GP)** case-finding, intelligent **nursing-led** symptom surveillance and adherence support, **laboratory** accuracy in preanalytic, analytic, and interpretive steps of serology and genetics, and decisive, evidence-based **pediatric gastroenterology** leadership for diagnosis confirmation, biopsy-sparing eligibility assessment, complication screening, and structured follow-up. Contemporary guidance—particularly the ESPGHAN 2020 update—endorses standardized serologic algorithms emphasizing tissue transglutaminase IgA (tTG-IgA) with total IgA, reflex IgG-based assays when IgA deficiency is present, and carefully delimited no-biopsy pathways when tTG-IgA is ≥10× the upper limit of normal (ULN) with endomysial antibody (EMA) positivity on a second sample; these developments have simplified pathways, reduced invasive procedures, and reinforced the centrality of **quality-assured laboratory methods** in getting the diagnosis right the first time (1–6). Saudi Arabia contributes an important perspective: population-based and risk-group studies reveal a CD burden comparable to or higher than Western cohorts, especially among children with type 1 diabetes and autoimmune thyroid disease, while regional practice has increasingly adopted ESPGHAN-aligned strategies, highlighting opportunities to strengthen case-finding in primary care, expand dietetic access, and standardize gluten-free food labeling and school-based support (7–13). This review synthesizes epidemiology, pathophysiology, clinical spectrum, and the **multidisciplinary operational model**—detailing GP triggers for testing, nursing workflows, laboratory quality imperatives, and pediatric gastroenterologist decision-points—then proposes integrated metrics and implementation tactics (including Saudi-specific enablers) to accelerate diagnosis, improve adherence to the gluten-free diet (GFD), minimize complications, and optimize long-term quality of life.

Introduction

Pediatric celiac disease exemplifies modern challenges in population health: a common, lifelong condition with highly variable presentations, a definitive and non-pharmacologic therapy (the gluten-free diet) that succeeds only when the **system** supports families, and a diagnostic pathway that is straightforward on paper but vulnerable to failure at multiple small steps across the continuum of care. Prevalence estimates cluster near 1% in many populations, but rates vary between and within regions owing to genetic background, infant feeding practices, infections, and ascertainment intensity; importantly, the "prevalence gap" between seropositivity and biopsy-confirmed disease persists, implying that many affected children remain undiagnosed during critical growth windows (1–4,14–16). In Saudi Arabia and the broader Middle East, awareness has grown as pediatric cohorts in primary care, endocrine clinics, and gastroenterology services demonstrate seroprevalence and confirmed CD rates at least comparable to Europe and North America, with **risk-group**yields that rival or exceed those abroad (7–13,17). Clinically, the phenotype has shifted over recent decades away from the archetypal toddler with chronic diarrhea, distension, and failure to thrive toward a spectrum that includes iron-deficiency anemia resistant to therapy, short stature or growth deceleration, delayed puberty, dental enamel defects, elevated transaminases, chronic constipation, recurrent abdominal pain, headaches or "brain fog," and behavioral or school performance concerns—

features that often prompt GP visits and nurse-led triage long before subspecialty referral (2–3,5,18–22). The **stakes** of missing or delaying diagnosis are not trivial: beyond impaired growth and bone accrual, prolonged gluten exposure heightens risks of fractures, reduced peak bone mass, psychosocial impacts, and additional autoimmune conditions such as type 1 diabetes, autoimmune thyroiditis, and dermatitis herpetiformis, while a subset of patients experience refractory symptoms, poor quality of life, or (rarely) serious complications like enteropathy-associated T-cell lymphoma later in life (1–3,23–26).

Against this backdrop, guidelines from ESPGHAN and other authorities coalesced around a pragmatic pediatric algorithm that elevates **tTG-IgA** with **total IgA** as the optimal first-line serology, reflexes to **IgG-based** tests (tTG-IgG or deamidated gliadin peptide IgG) for children with selective IgA deficiency, and employs EMA-IgA as a confirmatory assay and gatekeeper for the **biopsy-sparing** diagnosis route when tTG-IgA reaches ≥10× ULN; while HLA typing (DQ2/DQ8) retains value as a rule-out tool in equivocal cases, it is no longer required to confirm no-biopsy diagnoses in children under ESPGHAN 2020 (1–6). The laboratory's performance—calibration to manufacturer-specific ULNs, attention to lot-to-lot variation, and rigorous external quality assurance—determines whether these thresholds are trustworthy, making lab-clinical dialogue essential whenever results and the child's clinical picture diverge (4–6,27–28). The GP's role is to **suspect and test early** when growth falters, iron deficiency persists, or risk conditions are present; the nurse's role is to **operationalize** consistent case-finding through symptom checklists and growth surveillance, to coach families on **gluten-containing diet during testing**, and to sustain **adherence** once the diagnosis is made; and the pediatric gastroenterologist's role is to **own** diagnostic confirmation (including the no-biopsy criteria), screen for associated conditions and deficiencies, coordinate dietetic care, and lead long-term surveillance with explicit stop points for escalation when serology or symptoms fail to normalize. The Saudi context emphasizes additional tasks for the system: ensure **equitable access** to high-quality serology and pediatric endoscopy, reinforce **dietitian capacity** with celiac-specific expertise, support **school meal planning** and **food labeling**, and maintain **registries** to monitor outcomes and the safety net for high-risk groups such as first-degree relatives and children with type 1 diabetes (7–13,17,29–30).

REVIEW

Pathophysiology and immunogenetics: why some children get celiac disease and how this informs practice

Celiac disease arises when dietary gluten peptides, rich in glutamine and proline residues, traverse the small-intestinal barrier and undergo deamidation by tissue transglutaminase (tTG), enhancing their affinity for HLA-DQ2 or HLA-DQ8 molecules on antigen-presenting cells; in genetically predisposed hosts, this presentation activates a Th1-skewed immune response, culminating in intraepithelial lymphocytosis, crypt hyperplasia, and villous atrophy with malabsorption and systemic effects (1–3,31–33). Genetic risk is necessary but not sufficient: although HLA-DQ2/DQ8 are present in almost all affected patients, these alleles are common in the general population, and only a minority develop CD, pointing to additional modulators such as non-HLA genes, early-life infections, microbiome composition, timing and amount of gluten introduction, and possibly breastfeeding practices, none of which consistently "prevent" CD but may shape age at onset and phenotype (31–35). For clinicians, two implications stand out: first, **HLA typing** is an excellent **rule-out** tool—absence of DQ2/DQ8 effectively excludes CD and is useful in complex cases or when a family seeks diet initiation before a complete workup; second, immunologic activity tracks imperfectly with mucosal injury and symptoms, which is why **structured follow-up** must integrate serology, growth, and well-being rather than rely on any single marker (3–6,33–36).

Epidemiology, risk groups, and the global–Saudi lens

Globally, pediatric CD prevalence approximates 1%, with higher yields in **risk groups**: first-degree relatives of affected individuals, children with **type 1 diabetes**, **autoimmune thyroiditis**, **selective IgA deficiency**, **Down syndrome**, **Turner syndrome**, **Williams syndrome**, and those with persistent iron-deficiency anemia, short stature, unexplained elevated transaminases, or recurrent abdominal pain and constipation resistant to standard therapy (2–3,14–16,18–22). These risk markers guide GP case-finding and justify **active screening** in endocrine clinics and genetics services. In **Saudi Arabia**, studies across schoolchildren and specialty cohorts consistently show meaningful seroprevalence and biopsy-confirmed disease rates, while pediatric **type 1 diabetes** clinics report CD prevalence often in the **high single to low double digits**, underscoring the value of **periodic serologic screening** after diabetes diagnosis and during follow-up, as seroconversion can occur years later (7–13,17,29). Regional practice has increasingly harmonized with ESPGHAN criteria, but challenges remain: geographic variability in access to pediatric endoscopy and dietetics, heterogeneity in school system awareness and gluten-free meal provision, and the need for **culturally adapted education** addressing staple foods, cross-contamination in extended family kitchens, and social occasions central to family life (7–13,29–30,37–38). These epidemiologic realities justify placing **GPs and nurses** at the forefront of early detection via growth monitoring, symptom algorithms, and risk-group registries that prompt scheduled testing, while **laboratories** ensure reproducible serology and clear interpretive comments that ease decision-making for busy primary care clinicians.

Diagnostic strategy in children: serology first, then confirm intelligently

The **serologic gateway** is built on two pillars: **tTG-IgA** and **total IgA** measured concurrently to detect selective IgA deficiency, a condition over-represented in CD and a common reason for false-negative IgA-based tests (1–6,27–28). When total IgA is normal, **tTG-IgA** is the recommended first-line assay owing to its high sensitivity and specificity; **EMA-IgA** is used to confirm high-titer results and arbitrate equivocal findings, especially because EMA is operator-dependent but exquisitely specific when performed by experienced laboratories (1–5,27–28). When selective IgA deficiency is present, **IgG-based** assays (tTG-IgG or deamidated gliadin peptide IgG) are indicated, recognizing that diagnostic performance is somewhat lower and thresholds for endoscopy should be correspondingly **lower** when clinical suspicion is high (1–5). The **biopsy-sparing** pathway—appropriate for children with **tTG-IgA ≥10× ULN** and **EMA-IgA positivity on a second sample**, with no mandatory requirement for HLA typing or symptom presence—has transformed pediatric practice by sparing many children sedation and invasive procedures while maintaining high diagnostic accuracy in settings where assay calibration and quality assurance are robust (1–4). However, this convenience should not eclipse **clinical judgment**: discordant scenarios (e.g., high tTG-IgA but negative EMA; positive serology with non-specific symptoms; borderline elevations) merit consultation with the pediatric gastroenterologist and often endoscopic confirmation to avoid **over-diagnosis**, especially in populations with high background HLA positivity (3–5,27–28). A crucial operational rule is that children must remain on a **gluten-containing diet** through testing; premature gluten restriction—common after internet searches or well-intentioned advice—can suppress serologic markers and mucosal injury, producing false reassurance and **diagnostic ambiguity** that later necessitates a burdensome gluten challenge (2–4,39). That is why **nursing scripts** and **GP counseling** should explicitly instruct families not to start a GFD before the diagnostic steps are complete, and why laboratories can add succinct report comments reminding clinicians of this principle when tTG testing is ordered.

Histology, when needed, and the limits of Marsh classification

When endoscopy is indicated—because serology does not meet no-biopsy criteria, results are discordant, or alternative diagnoses are suspected—the goal is to obtain **multiple biopsies** from the **distal duodenum** and at least one from the **duodenal bulb** to account for patchy lesions; meticulous orientation and prompt fixation are essential to avoid artifact that confounds villous assessment (3–4,40–41). Histology is reported by modified **Marsh–Oberhuber** grades, but clinicians should interpret grades in clinical context because **Marsh 1–2** changes (intraepithelial lymphocytosis with or without crypt hyperplasia) are not specific to CD and may reflect infections, medications, small intestinal bacterial overgrowth, or other inflammatory conditions; **Marsh 3** (villous atrophy with crypt hyperplasia) is more specific when supported by serology and clinical features (3–4,40–41). In equivocal cases, **HLA typing** is helpful as a **rule-out**, while repeat serology under a gluten-containing diet, attention to lab methodology and ULNs, and second-opinion pathology review can prevent misclassification. For Saudi centers scaling pediatric endoscopy capacity, **standardized biopsy protocols**, co-review between pathologists and gastroenterologists, and continuous education on artifacts (e.g., peptic duodenitis, bulb flattening) are pragmatic quality measures that reduce false positives and negatives (29–30,41–42)

The gluten-free diet: implementation, adherence, and the indispensable role of nursing and dietetics

The **gluten-free diet** is the cornerstone of treatment and, when implemented with **precision** and **support**, normalizes serology in most children within a year, heals mucosa in the majority over 12–24 months, restores growth trajectories, improves bone mineral accrual, and reduces long-term risks (2–3,23–26,43–45). Yet the GFD is deceptively complex: families must navigate label literacy, cross-contamination in mixed-diet households, school cafeterias, social gatherings, religious holidays, and travel—all settings where inadvertent gluten exposure is common. Here, **nurses and dietitians** are linchpins: they provide **structured education** at diagnosis (gluten sources, hidden ingredients, kitchen zoning, safe grains), develop **individualized meal plans** aligned with cultural preferences and socioeconomic realities, teach **reading of ingredient lists** and recognition of local certification marks, and deploy **behavioral strategies** that accommodate the child's age, autonomy, and peer dynamics (46–49). In Saudi Arabia, culturally adapted counseling addresses staples such as traditional breads and wheat-based dishes, clarifies labeling practices, highlights reputable gluten-free suppliers, and coordinates with **school health teams** to assure safe meal options and emergency plans for accidental exposures (29–30,37–38,50). Adherence benefits from **multimodal follow-up**—in-person or telehealth visits with nurses and dietitians, printed or digital checklists, growth and symptom diaries, and age-appropriate education for the child to become an informed partner in care. This is also the place to screen and support **psychosocial health**: anxiety, social isolation, and dietary burden are real and can be mitigated by peer groups, family counseling, and pragmatic problem-solving rather than perfectionism that undermines sustainability (47–49,51–52).

Monitoring after diagnosis: who does what, when, and how to act on results

Shared care models delineate responsibilities: **GPs** monitor interim growth, vaccination status (including pneumococcal and hepatitis B catch-up where indicated), iron and vitamin D supplementation, and intercurrent illnesses; **nurses**coordinate appointments, reinforce the GFD, and screen for psychosocial strain; **laboratories** deliver timely, consistent tTG-IgA (or appropriate IgG) measurements with the same assay where possible to allow trajectory assessment; and the **pediatric gastroenterologist** sets cadence (commonly at 3–6, 12, and 24 months, then annually), orders **nutritional panels** (iron studies, folate, vitamin B12 if appropriate, vitamin D, calcium, albumin), considers **thyroid function** in the presence of symptoms or risk, and determines when to escalate (2–4,23–26,43–45). A typical expectation is that **tTG-IgA**falls substantially by **6 months** and normalizes by **12–24 months** in adherent children, with growth velocity improvement evident on centile charts. **Red flags** that should trigger deeper review include persistently elevated or rising tTG, lack of growth catch-up, persistent symptoms, or new deficiencies; differential diagnoses include **ongoing gluten exposure**, slow mucosal healing, **concomitant conditions** (lactose intolerance, small intestinal bacterial overgrowth, functional disorders), **refractory CD** (exceedingly rare in children), and **incorrect original diagnosis** (2–4,43–45). In these scenarios, the pediatric gastroenterologist may order **dietetic re-assessment**, **repeat endoscopy**, **HLA typing** if it was never performed and doubt remains, or targeted testing for alternative explanations. Bone health merits specific attention: while routine DEXA for all children is not universally recommended, **selective** assessment is prudent in those with **severe malnutrition at diagnosis**, delayed puberty, fractures, persistent inflammation, or poor adherence; proactive **vitamin D and calcium** optimization is a low-risk, high-value strategy for all (23–26,53–55).

Special scenarios: infants and toddlers, IgA deficiency, type 1 diabetes, and asymptomatic relatives

In **very young children**, symptoms can be misleading, mixing functional gastrointestinal complaints with genuine malabsorption; nevertheless, the serologic paradigm holds, with careful attention to total IgA and judicious use of IgG assays when needed (1–5,18–22). In **selective IgA deficiency**, the diagnostic yield of **IgG-based** serology is lower and decision thresholds for endoscopy are correspondingly lower; once diagnosed, families should be counseled that **serologic monitoring** must use the **same IgG assay** longitudinally to interpret trends (1–5,27–28). Among children with **type 1 diabetes**, CD prevalence is high and **seroconversion** can occur years after diabetes onset; coordinated pathways with **endocrinology** and **nursing** teams can schedule **periodic tTG screening** (e.g., at diagnosis, 2–3 years, and 5 years, or sooner if symptoms arise), minimize duplicative phlebotomy by bundling labs, and address **glycemic variability** that accompanies gluten exposures or diet changes (7–13,56–58). For **asymptomatic first-degree relatives**, **opportunistic screening** is reasonable, with repeat testing at intervals in childhood since risk persists across development; counseling should discuss the implications of positive serology and the importance of remaining on gluten during evaluation (2–4,14–16,39).

Laboratory excellence: preanalytic realities, assay selection, and interpretive comments that prevent missteps

The power of the pediatric algorithm depends on the **laboratory** getting several quiet details right. **Preanalytic** variables—gluten consumption status, time of draw, sample handling, and prompt processing—matter less than sheer **gluten exposure** but can still affect borderline results (27–28). **Analytic** quality hinges on assay selection (well-validated **ELISA**for tTG-IgA with manufacturer-defined ULN, EMA performed by experienced personnel), participation in **external quality assurance**, and resistance to the temptation to substitute poorly validated point-of-care tests as primary diagnostics; if such tests are used in outreach or remote clinics, they must be **confirmatory** with lab-based assays before any diagnostic decisions (27–28,59–60). **Interpretive** excellence includes embedding standardized comments: when tTG-IgA is markedly elevated (≥10× ULN), reports can suggest EMA confirmation and **no-biopsy** eligibility if clinical context aligns; when total IgA is low, reports should direct clinicians toward **IgG-based** assays; and when tTG-IgA is borderline, reports can recommend **repeat testing** with continued gluten consumption or gastroenterology consultation, rather than reflexive diet initiation (1–6,27–28). In Saudi Arabia, scaling **regional laboratory networks** to standardize assays and ULNs across institutions would reduce confusion for GPs who see families moving between facilities; centralized education for clinicians on **how to read** celiac serology reports further closes the loop (29–30,37–38).

The pediatric gastroenterologist as pathway steward: diagnosis confirmation, complications, and long-term strategy

Pediatric gastroenterologists orchestrate the **decisive steps**: verifying that **no-biopsy** criteria are met or proceeding to endoscopy when they are not; ordering **baseline panels** (full blood count, ferritin and iron studies, folate, vitamin B12 as indicated, vitamin D, calcium, phosphate, albumin, transaminases, thyroid function when appropriate), and **screening for coexisting autoimmune disorders** according to risk; launching **dietetic care** with celiac-specific expertise; and setting a follow-up schedule with **serology milestones**, growth review, and psychosocial check-ins (2–4,23–26,43–45,53–55). They are also responsible for recognizing **non-responsive celiac disease**, differentiating **slow healers** from **ongoing gluten exposure** or **alternative diagnoses**, and for the rare child with **refractory disease**, coordinating a multidisciplinary evaluation that may include repeat histology, immunophenotyping of intraepithelial lymphocytes, advanced imaging, and collaboration with immunology and oncology in exceptional cases (43–45). In the Saudi context, pediatric gastroenterology leadership has an additional **systems** mandate: advocate for **dietitian workforce expansion**with celiac expertise, partner with **Ministry of Health** and **school health** programs to normalize gluten-free accommodation, and maintain **registries** that anchor quality improvement and research into regional phenotypes, adherence correlates, and long-term outcomes (7–13,29–30,37–38).

Multidisciplinary integration: a practical, scalable pathway

A functional pathway integrates four disciplines into an **operational script**. **At the GP/nurse front end**, every child with **growth deceleration**, **iron-deficiency anemia** refractory to iron, **chronic gastrointestinal symptoms** (including constipation resistant to standard therapy), **unexplained transaminase elevation**, **delayed puberty**, **dental enamel defects**, or belonging to **risk groups** (type 1 diabetes, autoimmune thyroiditis, Down syndrome, Turner syndrome, Williams syndrome, selective IgA deficiency, first-degree relative of CD) triggers **reflex testing**: order **tTG-IgA and total IgA**, explicitly instruct the family to **continue gluten**, and schedule a **follow-up** to review results; if IgA deficiency is found, add **tTG-IgG or DGP-IgG**. **Laboratories** return results with interpretive comments aligned to the pathway. **Decision rules** for the GP: if **tTG-IgA ≥10× ULN**, prompt **EMA** and **urgent, no-biopsy referral** to pediatric gastroenterology; if **tTG-IgA positive but <10× ULN** or discordant with symptoms, **refer for endoscopy**; if **serology negative** but suspicion remains high (e.g., severe growth faltering, strong family history, classic symptoms), **consult**gastroenterology for second-line strategies or **repeat serology** after a defined interval while maintaining gluten intake. **At the specialist node**, the pediatric gastroenterologist confirms diagnosis (no-biopsy or histology), initiates **dietetic counseling**, orders **baseline deficiency screens**, and sets the follow-up cadence; **nurses** coordinate visits, reinforce adherence, and troubleshoot school and social issues; **GPs** maintain surveillance between specialty appointments and manage routine childhood health, immunizations, and intercurrent illnesses with celiac-aware considerations; **laboratories** ensure consistent assays over time for valid trend analysis. **Joint metrics** across disciplines—time from GP test order to result, time from positive serology to dietetic counseling, serology normalization rates at 12 and 24 months, growth velocity improvements, and patient-reported outcome measures (fatigue, abdominal pain, school attendance, quality of life)—convert the pathway from a guideline to a **learning system** that continuously improves.

Implementation science: closing the practice–guideline gap globally and in Saudi Arabia
The literature is clear that **guidelines** alone do not equal **practice change**; therefore, implementation tactics must match identified barriers. Common obstacles include **low suspicion** among non-gastroenterology clinicians for extra-intestinal or subtle presentations; **premature gluten restriction** before testing; **assay confusion** and variable ULNs; **inconsistent interpretive comments**; **limited dietitian access**; and **social barriers** to adherence at school and family events (2–6,27–30,37–38,46–52). Practical tools that work include **growth-chart prompts** in EMRs that suggest tTG-IgA testing when centile crossing occurs; **reflex test bundles** (tTG-IgA + total IgA) that prevent single-analyte orders; **standardized lab comments** scaffolded by the pediatric gastroenterology service; **nurse-led group education** sessions for newly diagnosed families; **school care plans** codified with the education sector; and **telehealth** dietetic follow-up to reach families far from tertiary centers. In Saudi Arabia, **MOH-aligned protocols**, **national registries**, and collaboration with **Saudi celiac patient associations** can accelerate public awareness, improve food labeling reliability, and reinforce access to certified gluten-free products. Procurement initiatives that reduce the cost of gluten-free staples, combined with clear **subsidy pathways** for low-income families where available, address a frequently cited barrier to adherence. Lastly, **audit and feedback** that transparently report pathway metrics back to GPs, nurses, laboratories, and gastroenterologists create accountability and pride of ownership—crucial drivers of sustained improvement in any chronic-care model.

Discussion
Early detection of pediatric celiac disease is not simply a matter of ordering the "right test"; it is a **systems choreography**that begins in primary care and depends on the reliability of lab methods, the discipline of care teams to follow an agreed-upon script, and the practical wisdom of nurses and dietitians who help families turn abstract advice into daily routines that work at school, at home, and at social gatherings. The **serology-first** paradigm embodied in ESPGHAN's 2020 update has cleaned up much of the diagnostic ambiguity that plagued earlier eras, especially by formalizing a **no-biopsy**route for children with **very high** tTG-IgA and confirmatory EMA; at the same time, it has raised the stakes for **laboratory quality**, because a mis-calibrated ULN or inconsistent EMA technique would propagate errors. Accordingly, true

multidisciplinary integration is not rhetorical but operational: gastroenterology services should **co-design** lab report language and threshold tables, and labs should alert clinicians when assay kits or ULNs change. On the **clinical** side, GPs and nurses need a **short list of testing triggers**—growth deceleration, iron-deficiency anemia, transaminase elevation, risk conditions, and persistent functional GI complaints—to normalize early testing without over-testing. Equally, they need **scripts** to prevent the all-too-common error of starting a GFD before the evaluation is complete; nurses are especially effective at this task when empowered with handouts and checklists that families can take home.

Once diagnosed, success is overwhelmingly determined by **adherence**, and adherence is a function of **capacity** plus **support**. Families need accessible **dietitians** who understand local foods, **nurses** who can triage setbacks and reduce clinic friction, and **schools** that provide safe and stigma-free meal options. In Saudi Arabia and other regions with extended family structures and frequent communal meals, **family-wide education** reduces cross-contamination and ensures the child is not isolated; aligning with **patient associations** amplifies practical tips and mutual support. Measuring what matters—serology normalization at one year, growth velocity, vitamin D and ferritin correction, and quality-of-life indices—keeps the team focused on outcomes rather than checklists. Importantly, the discussion should not neglect **equity**: gluten-free foods can be expensive, clinics may be distant, and work schedules rigid; **telehealth**, **group education**, and **subsidized staples** are not luxuries but core enablers that ensure the GFD is a realistic therapy for all families, not only the well-resourced.

On controversies, the field continues to debate the precise role of **HLA typing** in the no-biopsy era (valuable as a rule-out in equivocal cases but not routinely required), the optimal **monitoring frequency** once children are stable (annual is common, but higher frequency may be warranted in adolescents who face adherence stressors), when to **repeat endoscopy** (selectively for non-response or diagnostic doubt rather than routinely), and how best to manage **persistent symptoms** with normalized serology (think broadly: constipation, functional pain, SIBO, lactose intolerance, and psychosocial contributors). Meanwhile, **emerging research** on the microbiome, non-dietary therapies, and enzyme or polymer strategies to degrade gluten is intriguing but not yet a replacement for the GFD in children; clinical teams should set realistic expectations and avoid therapeutic distraction. For now, the most impactful innovation remains **implementation**: leveraging EMR prompts, reflex testing, standardized lab comments, dietetic capacity, and school partnerships to deliver the care we already know works.

Conclusion

Pediatric celiac disease is common, consequential, and completely amenable to a non-pharmacologic therapy whose success depends on **early recognition** and **systemic support**. A **multidisciplinary model** that empowers **GPs** to test early in children with growth, hematologic, hepatic, or risk-group red flags; enables **nurses** to operationalize education, adherence monitoring, and family/school coordination; secures **laboratory** excellence with validated thresholds and clear interpretive comments; and places **pediatric gastroenterologists** as pathway stewards for diagnosis confirmation, deficiency correction, and long-term surveillance will narrow the diagnosis gap and optimize outcomes. The **ESPGHAN 2020** framework for serology and biopsy-sparing diagnosis provides a solid backbone, but the details—keeping children on gluten during testing, reflexing intelligently in IgA deficiency, aligning assays and ULNs, and planning follow-up milestones—make or break the pathway. In **Saudi Arabia**, where prevalence in key risk groups is high and awareness has risen, the agenda is clear: consolidate ESPGHAN-aligned protocols across regions, expand dietetic capacity and culturally attuned education, strengthen school partnerships and food labeling, and invest in registries and audit-feedback cycles that convert guidelines into **measurable, equitable** gains. When these pieces are in place, most children will experience normalization of serology, catch-up in growth, restoration of bone accrual, and improved quality of life—quiet successes that accrue from coordinated work by GPs, nurses, laboratories, and pediatric gastroenterologists aligned around a simple goal: diagnose early, treat precisely, and support the child and family for the long run.

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