Cow's milk allergy verses cow's milk intolerance in infants

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ABSTRACT

Cow's milk allergy (CMA) and cow's milk intolerance (CMI) are common early-life feeding problems, but their overlap can obscure a crucial distinction: CMA is immune-mediated, whereas many forms of CMI are non-immune and often involve lactose malabsorption. Because eczema, vomiting, reflux-like symptoms, diarrhea, mucus in the stool, irritability, colic-like crying, and disrupted sleep can arise through more than one mechanism, management must avoid unsafe substitutions and distinguish dietary treatment from supportive care. The essay differentiates CMA from CMI, reviews diagnosis and treatment, and proposes a cautious 12-week adjunct chiropractic protocol focused on safety, interdisciplinary care, and measurable outcomes for a 12-week-old formula-fed infant with confirmed CMA and cross-reactivity to soy and goat milk.

Key words: Cow's milk allergy, cow's milk intolerance, chiropractic, probiotics, pediatric, infant, formula.

Introduction

CMA is an immune-mediated adverse reaction to cow's milk proteins. Important allergens include caseins and whey proteins such as beta-lactoglobulin and alpha-lactalbumin. It may be IgE-mediated, non-IgE-mediated, or mixed; these phenotypes differ in timing, severity, and diagnostic usefulness of tests such as specific IgE and skin prick testing.[1-3] IgE-mediated reactions are typically immediate, often appearing within minutes to two hours of exposure, and may include urticaria, angioedema, vomiting, wheeze, or anaphylaxis.

Non-IgE-mediated reactions are usually delayed, developing over several hours to days, and commonly present with gastrointestinal or skin symptoms such as proctocolitis, enteropathy-like symptoms, reflux-like distress, diarrhea, or eczema; because IgE testing is often negative, diagnosis depends more heavily on clinical history, elimination, and supervised re-challenge where appropriate. Mixed reactions involve both IgE and cellular immune mechanisms and may produce overlapping immediate and delayed features, making careful classification important for risk assessment and follow-up.[4,5]

By contrast, CMI is often used for non-immune adverse reactions to milk, most commonly lactose malabsorption due to lactase deficiency. It does not involve immune sensitization to cow's milk proteins, so management must be related to the mechanism rather than the broad label of "intolerance".[2,6] The clinical importance of this distinction is risk. IgE-mediated CMA can involve rapid systemic reactions, including urticaria, wheeze, or anaphylaxis, while non-IgE disease more often produces delayed gastrointestinal and skin symptoms.[2,3] Lactose intolerance more commonly produces gas, bloating, and diarrhea-like symptoms through osmotic and fermentative effects rather than allergy.

Recent literature confirms that milk-related symptoms should not be linked to lactose alone. Pal et al.[7] argue that A1 beta-casein may contribute to gastrointestinal symptoms, Darma et al.[8] distinguishes lactose intolerance from immune reactions to casein and beta-lactoglobulin, and Al-Beltagi et al.[9] frames cow's milk-related gastrointestinal disorders as mechanistically diverse. Together, these studies support this paper's central claim: similar digestive symptoms may arise from lactose malabsorption, immune responses to casein or whey proteins, or other protein-related mechanisms, so treatment must follow the underlying cause.

Because symptoms overlap, diagnosis should rely on structured history, elimination response, risk assessment, and medically supervised testing or food challenge where indicated. Cross-reactivity can be a central problem here, so substitution decisions must remain practitioner guided.[2,6] The updated ESPGHAN position paper reinforces short diagnostic elimination followed by challenge where appropriate and warns that over-diagnosis can also harm infants through unnecessary restriction.[10] In the case presented in this paper, a confirmed allergy and cross-reactivity move care away from trial-and-error feeding changes and toward structured risk management.

Some chiropractic frameworks describe reflex pathways between visceral input and somatic output. In this essay, gastrointestinal inflammation or discomfort may influence autonomic regulation, and lead to a viscerosomatic reflex resulting in somatic dysfunction.

METHODS

Utilized Chiro Index and Google Scholar for literature review including randomized control trials involving cow's milk allergy and cow's milk intolerance and the impact it may have on infants. Also, chiropractic pediatric textbooks were consulted. Informed consent was obtained from patient's guardian for publication.

CASE PARAMETERS AND OUTCOME MEASURES

- 12-week-old formula-fed infant with confirmed CMA and cross-reactivity to soy and goat milk.
- Primary management remains a practitioner-mediated elimination, hypoallergenic formula selection, and growth monitoring.
- Chiropractic care is typically considered adjunctive, but varies with advanced training, using gentle low-force approaches for suspected musculoskeletal discomfort and stopping if adverse responses occur.

Progress is tracked with a weekly diary covering GI symptoms, eczema, sleep, growth, handling/tummy-time tolerance, and adverse events. Outcomes should be interpreted as patterns: improved sleep matters only alongside stable feeding, normal growth, and no flare; improved positioning may reflect reduced discomfort, maturation, caregiver confidence, or manual-care effects rather than allergy improvement. The 12-week structure should therefore be understood as a pragmatic monitoring and reassessment window rather than a proven chiropractic dose-response schedule. It allows time for dietary elimination as directed by their medical physician, feeding assessment, growth review, and repeated functional observation while keeping manual care explicitly secondary to dietary and medical management as needed.

Treatment begins with strict avoidance of cow's milk protein and confirmed cross-reactive triggers, supported by hypoallergenic formula selection, growth monitoring, and attention to micronutrient adequacy. Extensively hydrolyzed formulas are common first-line options, while amino acid-based formulas are usually reserved for severe cases or intolerance of extensively hydrolyzed formulas. Goat milk cannot be assumed safe, and soy or rice-based alternatives require careful selection because allergenicity, cross-reactivity, nutritional adequacy, and regulatory suitability vary.[11]

Vanderhoof and Kleinman[12] add that infants with cow's milk protein allergy require careful iron consideration because cow's milk exposure, intestinal inflammation, and restrictive feeding patterns may increase risk for iron deficiency; therefore, formula choice, growth review, and dietary follow-up should include iron sufficiency rather than symptom control alone.

Evaluation should also include feeding mechanics: an IBCLC or feeding-trained clinician can assess oral function, bottle flow, aerophagia, tethered oral tissues, and feeding techniques that may mimic or worsen reflux-like symptoms. Restrictive tethered oral tissues can limit tongue mobility and may interfere with achieving or maintaining an effective seal and coordinated suck—swallow—breathe pattern, contributing to shallow or unstable latch, inefficient milk transfer, prolonged feeds, clicking, fatigue, and greater air intake.[13-15] Swallowed air and compensatory feeding patterns may contribute to burping, gas, abdominal discomfort, or reflux-like symptoms, although these findings are not specific to oral restriction and should not be confused with allergic gastrointestinal disease.

A 2024 systematic review and meta-analysis reported improvement in parent-reported infant reflux scores after restrictive oral-tissue release, but the evidence included predominantly observational studies and does not establish that release is appropriate for every infant with GI symptoms.[16] Current practice guidance therefore emphasizes diagnosis based on functional restriction rather than anatomy alone, multidisciplinary assessment, and individualized management with feeding support before and after any procedure.[17]

If symptoms persist after formula change, clinicians should reassess preparation technique, accidental exposure, feeding volume, stool and skin patterns, weight gain, and whether symptoms are allergic, feeding-related, or functional. In this particular case presentation, the chiropractor is also an IBCLC and an oral functional exam as well as a biomechanical evaluation is part of the initial evaluation, supporting a multidisciplinary approach to care.

Probiotics may be considered only as adjunctive, strain-specific interventions. Reviews propose mechanisms including microbiota modulation, barrier support, immune regulation, oral tolerance, and modification of allergenic epitopes.[18-21] Ezaki et al.[22] suggests probiotics may help selected high-risk neonates with dysbiosis after surgery and antibiotics, but probiotics should not replace elimination or formula management. Prebiotics remain promising but not universally established.[18]

Lactose-focused strategies require separate interpretation because they address a different mechanism from immune-mediated CMA. Kanabar, Randhawa and Clayton[23] found that lactase reduced breath hydrogen and crying time in some infants with colic, and Leis et al.[24] reported positive but heterogeneous probiotic findings in lactose intolerance. However, Pal et al.[7] and Darma et al.[8] caution that milk-related symptoms may also involve casein, beta-lactoglobulin, or other milk-protein mechanisms. Lactase or lactose reduction may help only when lactose

malabsorption is the problem; it should not be presented as treatment for confirmed CMA.

Vitamin D should be positioned as nutritional support rather than CMA treatment. Peng et al.[25] found that supplementation did not significantly prevent allergic diseases overall, including food allergy outcomes.

12-WEEK ADJUNCT CHIROPRACTIC PROTOCOL

Weeks 1—2: screen red flags, confirm medical alignment, establish baseline symptoms, and assess tolerance for positioning, head/neck movement, and gentle palpation. Low-force intervention is used only when clinically indicated and tolerated. Caregiver education is essential. The chiropractor should clarify that manual care does not desensitize the infant or replace dietary management. Parents should seek review for poor weight gain, blood in stool, persistent vomiting, lethargy, dehydration, respiratory symptoms, or signs of acute allergic reaction, if not addressed in the chiropractic office.

Weeks 3—8: provide weekly then reduced-frequency care focused on comfort, positional tolerance, gentle mobility, and caregiver-led home positioning. If GI flares or adverse responses occur, manual care is paused, dietary adherence is reviewed and referral to medical care as warranted. The middle phase remains flexible: if feeding and growth are stable, care may focus on spinal manipulation, fascial restriction, and overall well-being; if symptoms worsen, the priority is plan review, allergen-exposure assessment and medical review, not just increased chiropractic frequency.

Weeks 9—12: consolidate gains, reassess the full outcome set, and either discharge to caregiver-led strategies or coordinate further practitioner review if symptoms persist. Discharge depends on improved GI comfort, stable or reduced eczema, improved sleep or handling tolerance, acceptable growth, and no adverse events. By the final phase, success means stable improvement without dependence on practitioner-led care: confident caregiver routines, fewer distress signals during handling, and continued effectiveness of medical nutritional management. Inconsistent gains require cautious interpretation and possible medical, dietetic, feeding, or developmental reassessment.

DISCUSSION

Expected improvements in GI symptoms and eczema are most defensibly attributed to effective elimination and medical management.[2,6] When functionally restrictive tethered oral tissues or other feeding-mechanics problems are present, appropriately coordinated feeding support, in selected cases, oral-tissue release by a qualified clinician, may improve latch, seal, milk transfer, and aerophagia-related discomfort; these outcomes should be documented separately from changes in CMA.[16,17]

Chiropractic care may be used adjunctively to address identified musculoskeletal contributors to feeding position, comfort, and movement, but it should not be described as releasing oral restrictions or treating the allergic mechanism. Outcomes may also reflect diet, maturation, caregiver technique, and non-specific effects. Gentle, infant-appropriate manual care may support comfort and function. The medical team addresses the allergic mechanism, if it is not supported in the chiropractic office.

Hawk et al.[26] supports cautious pediatric chiropractic best-practice boundaries, and Bialosky et al.[27] frames manual therapy effects as neuromodulatory and functional rather than immune-curative. Chiropractic reports and pediatric texts provide clinical context, not proof of disease modification.[28-30]

CONCLUSION

In conclusion, CMA and CMI may look similar but require different clinical logic. CMA is immune-mediated and requires accurate diagnosis, cow's milk protein elimination, management of cross-reactive foods, and medical supervision. CMI is often non-immune, but it should not be reduced automatically to lactose intolerance. Evidence on A1 beta-casein, casein, and beta-lactoglobulin reinforces that similar gastrointestinal symptoms can arise from lactose malabsorption, immune milk-protein reactions, or other protein-related mechanisms.

A layered model is therefore safest: medical nutrition controls allergen exposure and protects growth; microbiome strategies remain promising but context-dependent; and lactase approaches apply only when lactose malabsorption is confirmed. Overall, the safest model is integrated care: medical nutrition often treats the disease process, while chiropractic care focuses on low-force, measurable outcomes and interdisciplinary review.

Chiropractors, with advanced training, are managing cases like the one presented in this paper, offering a more comprehensive treatment from chiropractic adjustments, oral evaluations, allergy and/or sensitivity testing, guidance on food elimination, and nutritional support for gut healing, as well as referrals to other practitioners as warranted. More research and case studies need to be completed to further evaluate the efficacy of chiropractic adjustments and specialized treatments.

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